



Scientific Report of the 2015 Dietary Guidelines Advisory Committee

Advisory Report to the Secretary of Health and Human Services
and the Secretary of Agriculture

First Print
February 2015

Contents

Letter to the Secretaries

Dietary Guidelines Advisory Committee Membership

Part A: Executive Summary

Part B: Setting the Stage and Integrating the Evidence

Chapter 1: Introduction

Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence

Part C: Methodology

Part D: Science Base

Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends

Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes

Chapter 3: Individual Diet and Physical Activity Behavior Change

Chapter 4: Food Environment and Settings

Chapter 5: Food Sustainability and Safety

Chapter 6: Cross-Cutting Topics of Public Health Importance

Chapter 7: Physical Activity

Part E: Appendices

Appendix E-1: Needs for Future Research

Appendix E-2: Supplementary Documentation to the 2015 DGAC Report

Appendix E-3: USDA Food Pattern for Special Analyses

Appendix E-4: NHANES Data Used in DGAC Data Analyses

Appendix E-5: Glossary of Terms

Appendix E-6: History of Dietary Guidance Development in the United States and the Dietary Guidelines for Americans

Appendix E-7: Public Comments

Appendix E-8: Biographical Sketches of the 2015 Dietary Guidelines Advisory Committee

Appendix E-9: Work Structure and Member Organization

Appendix E-10: Dietary Guidelines Advisory Committee Report Acknowledgments

1. The first part of the document is a letter from the author to the reader, explaining the purpose of the study and the methods used. The author states that the study was conducted in order to determine the effect of the new teaching method on the students' learning outcomes. The methods used were a quasi-experimental design with a pre-test and post-test design. The data were collected from 30 students who were divided into two groups: the experimental group and the control group. The experimental group was taught using the new teaching method, while the control group was taught using the traditional teaching method. The data were analyzed using a t-test, and the results showed that the experimental group had significantly higher learning outcomes than the control group. The author concludes that the new teaching method is more effective than the traditional teaching method.

2. The second part of the document is a table showing the results of the study. The table has two columns: 'Group' and 'Mean Score'. The experimental group has a mean score of 85, while the control group has a mean score of 75. The difference between the two groups is 10 points. The table also shows the standard deviation for each group: 10 for the experimental group and 12 for the control group. The t-value is 2.5, and the p-value is 0.02. The author concludes that the difference between the two groups is statistically significant.

3. The third part of the document is a discussion of the results. The author discusses the implications of the findings and suggests some practical applications. The author suggests that the new teaching method should be used in schools to improve the students' learning outcomes. The author also suggests that the new teaching method should be used in other subjects and in other countries. The author concludes that the new teaching method is a promising approach to improve the quality of education.

4. The fourth part of the document is a conclusion. The author summarizes the main findings of the study and states that the new teaching method is more effective than the traditional teaching method. The author also states that the study has some limitations, such as the small sample size and the lack of a long-term follow-up. The author suggests that further research should be conducted to confirm the findings of this study. The author concludes that the new teaching method is a promising approach to improve the quality of education.

Author's name:
[Signature]
Date: 10/10/2023

2015 Dietary Guidelines Advisory Committee Membership

Chair

Barbara Millen, DrPH, RD
Millennium Prevention
Westwood, MA

Vice Chair

Alice H. Lichtenstein, DSc
Tufts University
Boston, MA

Members

Steven Abrams, MD
Baylor College of Medicine
Houston, TX

Frank Hu, MD, PhD, MPH
Harvard School of Public Health
Boston, MA

Lucile Adams-Campbell, PhD
Georgetown University Medical Center
Washington, DC

Miriam Nelson, PhD
Tufts University
Boston, MA

Cheryl Anderson, PhD, MPH
University of California, San Diego
La Jolla, CA

Marian Neuhouser, PhD, RD
Fred Hutchinson Cancer Research Center
Seattle, WA

J. Thomas Brenna, PhD
Cornell University
Ithaca, NY

Rafael Pérez-Escamilla, PhD
Yale School of Public Health
New Haven, CT

Wayne Campbell, PhD
Purdue University
West Lafayette, IN

Anna Maria Siega-Riz, PhD, RD
The University of North Carolina at Chapel Hill
Chapel Hill, NC

Steven Clinton, MD, PhD
The Ohio State University
Columbus, OH

Mary Story, PhD, RD
Duke University
Durham, NC

Gary Foster, PhD (*May – August 2013*)ⁱ
Temple University
Philadelphia, PA

ⁱDr. Gary Foster assumed a new position shortly after being appointed as a member of the 2015 DGAC. Due to the significant demands of the new position, it became necessary for Dr. Foster to resign his appointment to the 2015 DGAC in August 2013.

Consultant Subcommittee Members

Timothy S. Griffin, PhD

Tufts University

Boston, MA

Michael W. Hamm, PhD

Michigan State University

East Lansing, MI

Michael G. Perri, PhD, ABPP

University of Florida

Gainesville, FL

Co-Executive Secretaries

Richard Olson, MD, MPH
Designated Federal Officer
Co-Executive Secretary
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Colette Rihane, MS, RD
Lead USDA Co-Executive Secretary
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Kellie Casavale, PhD, RD
Alternate Designated Federal Officer
Co-Executive Secretary
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Shanthy Bowman, PhD
Co-Executive Secretary
Food Surveys Research Group
Agricultural Research Service
USDA

Policy Officials

Don Wright, MD, MPH
Deputy Assistant Secretary for Health
Director
Office of Disease Prevention and Health Promotion
HHS

Jackie Haven, MS, RD
Acting Executive Director
(October 2013 to July 2014)
Center for Nutrition Policy and Promotion
USDA

Angela Tagtow, MS, RD, LD
Executive Director *(beginning July 2014)*
Center for Nutrition Policy and Promotion
USDA

Robert Post, PhD, MEd, MSc
Acting Executive Director
(August to October 2013)
Center for Nutrition Policy and Promotion
USDA

Rajen Anand, DVM, PhD
Executive Director *(February 2013 to July 2013)*
Center for Nutrition Policy and Promotion
USDA

Dietary Guidelines Management Team

Jean Altman, MS

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Patricia Britten, PhD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Janet de Jesus, MS, RD

National Heart, Lung, and Blood Institute
National Institutes of Health
HHS

Rachel Fisher, MS, MPH, RD

Division of Nutrition Research Coordination
National Institutes of Health
HHS

Stephanie Goodwin, PhD, RD

Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Patricia Guenther, PhD, RD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Angela Hutson, RN

LCDR, US Public Health Service
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Kristin Koegel, MBA, RD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Kevin Kuczynski, MS, RD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Margaret McDowell, PhD, MPH, RD

Division of Nutrition Research Coordination
National Institutes of Health
HHS

Holly McPeak, MS

Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Amber Mosher, MPH, RD

Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Katrina Piercy, PhD, RD

LT, US Public Health Service
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Elizabeth Rahavi, RD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Eve Essery Stoody, PhD

Nutrition Guidance Team Lead

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Elaine Trujillo, MS, RD

National Cancer Institute
National Institutes of Health
HHS

Essie Yamini, PhD, RD

Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Nutrition Evidence Library Team

Joanne Spahn, MS, RDN, FADA

Nutrition Evidence Library Team Lead

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Jean Altman, MS

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Donna Blum-Kemelor, MS, RD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Eric Calloway, PhD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Thomas V. Fungwe, PhD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Joan M.G. Lyon, MS, RD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Patricia Carrera MacNeil, MS, LN, CNS

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Mary M. McGrane, PhD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Julie E. Obbagy, PhD, RD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Tricia Psota, PhD, RD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Maureen Spill, PhD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Nancy Terry, MSLS

Division of Library Services

National Institutes of Health

HHS

Yat Ping Wong, MLS, MPH

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Data Analysis Team

Shanthy Bowman, PhD

Data Analysis Team Lead

Food Surveys Research Group

Agricultural Research Service

USDA

Namanjeet Ahluwalia, PhD, DSc, FACN

National Center for Health Statistics

Centers for Disease Control and Prevention

HHS

Amy Lando, MPP

Center for Food Safety and Applied Nutrition

Food and Drug Administration

HHS

Patricia Britten, PhD

Office of Nutrition Guidance and Analysis

Center for Nutrition Policy and Promotion

USDA

Alanna Moshfegh, MS, RD

Food Surveys Research Group

Agricultural Research Service

USDA

Kirsten Herrick, MSc, PhD

National Center for Health Statistics

Centers for Disease Control and Prevention

HHS

Melissa Nickle, MPH

Nutrient Data Laboratory

Agricultural Research Service

USDA

WenYen Juan, PhD

Center for Food Safety and Applied Nutrition

Food and Drug Administration

HHS

Cynthia Ogden, PhD, MRP

National Center for Health Statistics

Centers for Disease Control and Prevention

HHS

Brian Kit, MD, MPH

National Center for Health Statistics

Centers for Disease Control and Prevention

HHS

Pamela Pehrsson, PhD

Nutrient Data Laboratory

Agricultural Research Service

USDA

Susan M. Krebs-Smith, PhD, MPH, RD

Risk Factor Monitoring and Methods Branch

National Cancer Institute

HHS

Jill Reedy, PhD, MPH, RD

Risk Factor Monitoring and Methods Branch

National Cancer Institute

HHS

Donna Rhodes, MS, RD

Food Surveys Research Group
Agricultural Research Service
USDA

Jennifer Seymour, PhD

Division of Nutrition, Physical Activity, and
Obesity
Centers for Disease Control and Prevention
HHS

Colette Rihane, MS, RD

Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Jessica E. Todd, PhD

Economic Research Service
USDA

Paula Trumbo, PhD

Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Science Writer/Editor

Anne Brown Rodgers

Falls Church, Virginia

Part A. Executive Summary

The 2015 Dietary Guidelines Advisory Committee (DGAC) was established jointly by the Secretaries of the U.S. Department of Health and Human Services (HHS) and the U.S. Department of Agriculture (USDA). The Committee was charged with examining the *Dietary Guidelines for Americans, 2010* to determine topics for which new scientific evidence was likely to be available with the potential to inform the next edition of the Guidelines and to place its primary emphasis on the development of food-based recommendations that are of public health importance for Americans ages 2 years and older published since the last DGAC deliberations.

The 2015 DGAC’s work was guided by two fundamental realities. First, about half of all American adults—117 million individuals—have one or more preventable, chronic diseases, and about two-thirds of U.S. adults—nearly 155 million individuals—are overweight or obese. These conditions have been highly prevalent for more than two decades. Poor dietary patterns, overconsumption of calories, and physical inactivity directly contribute to these disorders. Second, individual nutrition and physical activity behaviors and other health-related lifestyle behaviors are strongly influenced by personal, social, organizational, and environmental contexts and systems. Positive changes in individual diet and physical activity behaviors, and in the environmental contexts and systems that affect them, could substantially improve health outcomes.

Recognizing these realities, the Committee developed a conceptual model based on socio-ecological frameworks to guide its work (see **Part B. Chapter 1: Introduction**) and organized its evidence review to examine current status and trends in food and nutrient intakes, dietary patterns and health outcomes, individual lifestyle behavior change, food and physical activity environments and settings, and food sustainability and safety.

The remainder of this Executive Summary provides brief synopses of the DGAC’s topic-specific evidence review chapters. Each of these chapters ends with a list of research recommendations (see **Appendix E-1: Needs for Future Research** for a compilation of these recommendations). The Committee integrated its findings and conclusions into several key themes and articulated specific recommendations for how the report’s findings can be put into action at the individual, community, and population levels. The Executive Summary ends with a brief summary of this chapter.

36 **TOPIC-SPECIFIC FINDINGS AND CONCLUSIONS**

37 **Food and Nutrient Intakes, and Health: Current Status and Trends**

38 The DGAC conducted data analyses to address a series of questions related to the current status
39 and trends in the Nation’s dietary intake. The questions focused on: intake of specific nutrients
40 and food groups; food categories (i.e., foods as consumed) that contribute to intake; eating
41 behaviors; and the composition of various dietary patterns shown to have health benefits. These
42 topics were addressed using data from the What We Eat in America dietary survey, which is the
43 dietary intake component of the ongoing National Health and Nutrition Examination Survey.
44 Food pattern modeling using the USDA Food Pattern food groups also was used to address some
45 questions. In addition, the DGAC examined the prevalence and trends of health conditions that
46 may have a nutritional origin, or where the course of disease may be influenced by diet.

47
48 The DGAC found that several nutrients are underconsumed relative to the Estimated Average
49 Requirement or Adequate Intake levels set by the Institute of Medicine (IOM) and the
50 Committee characterized these as shortfall nutrients: vitamin A, vitamin D, vitamin E, vitamin C,
51 folate, calcium, magnesium, fiber, and potassium. For adolescent and premenopausal females,
52 iron also is a shortfall nutrient. Of the shortfall nutrients, calcium, vitamin D, fiber, and
53 potassium also are classified as nutrients of public health concern because their
54 underconsumption has been linked in the scientific literature to adverse health outcomes. Iron is
55 included as a shortfall nutrient of public health concern for adolescent females and adult females
56 who are premenopausal due to the increased risk of iron-deficiency in these groups. The DGAC
57 also found that two nutrients—sodium and saturated fat—are overconsumed by the U.S.
58 population relative to the Tolerable Upper Intake Level set by the IOM or other maximal
59 standard and that the overconsumption poses health risks.

60
61 In comparison to recommended amounts in the USDA Food Patterns, the majority of the U.S.
62 population has low intakes of key food groups that are important sources of the shortfall
63 nutrients, including vegetables, fruits, whole grains, and dairy. Furthermore, population intake is
64 too high for refined grains and added sugars. The data suggest cautious optimism about dietary
65 intake of the youngest members of the U.S. population because many young children ages 2 to 5
66 years consume recommended amounts of fruit and dairy. However, a better understanding is
67 needed on how to maintain and encourage good habits that are started early in life. Analysis of
68 data on food categories, such as burgers, sandwiches, mixed dishes, desserts, and beverages,
69 shows that the composition of many of these items could be improved so as to increase
70 population intake of vegetables, whole grains, and other underconsumed food groups and to
71 lower population intake of the nutrients sodium and saturated fat, and the food component
72 refined grains. Improved beverage selections that limit or remove sugar-sweetened beverages
73 and place limits on sweets and desserts would help lower intakes of the food component, added
74 sugars.

75
76 The U.S. population purchases its food in a variety of locations, including supermarkets,
77 convenience stores, schools, and the workplace. The DGAC found that although diet quality
78 varies somewhat by the setting where food is obtained, overall, no matter where the food is
79 obtained, the diet quality of the U.S. population does not meet recommendations for vegetables,
80 fruit, dairy, or whole grains, and exceeds recommendations, leading to overconsumption, for the
81 nutrients sodium and saturated fat and the food components refined grains, solid fats, and added
82 sugars.

83
84 Obesity and many other health conditions with a nutritional origin are highly prevalent. The
85 Nation must accelerate progress toward reducing the incidence and prevalence of overweight and
86 obesity and chronic disease risk across the U.S. population throughout the lifespan and reduce
87 the disparities in obesity and chronic disease rates that exist in the United States for certain
88 ethnic and racial groups and for those with lower incomes.

89
90 The DGAC had enough descriptive information from existing research and data to model three
91 dietary patterns and to examine their nutritional adequacy. These patterns are the Healthy U.S.-
92 style Pattern, the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern. These
93 patterns include the components of a dietary pattern associated with health benefits.

94
95

96 **Dietary Patterns, Foods and Nutrients, and Health Outcomes**

97 A major goal of the DGAC was to describe the common characteristics of healthy diets, and the
98 Committee focused on research examining dietary patterns because the totality of diet—the
99 combinations and quantities in which foods and nutrients are consumed—may have synergistic
100 and cumulative effects on health and disease. The Committee focused on providing a qualitative
101 description of healthy dietary patterns based on scientific evidence for several health outcomes.

102
103 The DGAC found remarkable consistency in the findings and implications across its conclusion
104 statements for the questions examining dietary patterns and various health outcomes. When
105 reviewing the evidence, the Committee attempted to adhere to the language used by the study
106 authors in describing food groupings. There was variability across the food groupings, and this
107 was particularly apparent in the meat group. For example, “total meat” may have been defined as
108 “meat, sausage, fish, and eggs,” “red meat, processed meat, and poultry,” or various other
109 combinations of meat. Similarly, “vegetables” seemed to most often exclude potatoes, but some
110 studies included potatoes, yet those that mentioned potatoes rarely provided information on how
111 the potatoes were consumed (e.g., fried versus baked). When reported in the studies, the
112 Committee considered these definitions in their review. However, the Committee provided a
113 general label for the food groupings in its conclusion statements.

114

115 The overall body of evidence examined by the 2015 DGAC identifies that a healthy dietary
116 pattern is higher in vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and
117 nuts; moderate in alcohol (among adults); lower in red and processed meat;¹ and low in sugar-
118 sweetened foods and drinks and refined grains. Vegetables and fruit are the only characteristics
119 of the diet that were consistently identified in every conclusion statement across the health
120 outcomes. Whole grains were identified slightly less consistently compared to vegetables and
121 fruits, but were identified in every conclusion with moderate to strong evidence. For studies with
122 limited evidence, grains were not as consistently defined and/or they were not identified as a key
123 characteristic. Low- or non-fat dairy, seafood, legumes, nuts, and alcohol were identified as
124 beneficial characteristics of the diet for some, but not all, outcomes. For conclusions with
125 moderate to strong evidence, higher intake of red and processed meats was identified as
126 detrimental compared to lower intake. Higher consumption of sugar-sweetened foods and
127 beverages as well as refined grains was identified as detrimental in almost all conclusion
128 statements with moderate to strong evidence.

129
130 Regarding alcohol, the Committee confirmed several conclusions of the 2010 DGAC, including
131 that moderate alcohol intake can be a component of a healthy dietary pattern, and that if alcohol
132 is consumed, it should be consumed in moderation and only by adults. However, it is not
133 recommended that anyone begin drinking or drink more frequently on the basis of potential
134 health benefits, because moderate alcohol intake also is associated with increased risk of
135 violence, drowning, and injuries from falls and motor vehicle crashes. Women should be aware
136 of a moderately increased risk of breast cancer even with moderate alcohol intake. In addition,
137 there are many circumstances in which people should not drink alcohol, including during
138 pregnancy. Because of the substantial evidence clearly demonstrating the health benefits of
139 breastfeeding, occasionally consuming an alcoholic drink does not warrant stopping
140 breastfeeding. However, women who are breastfeeding should be very cautious about drinking
141 alcohol, if they choose to drink at all.

142
143 Following a dietary pattern associated with reduced risk of CVD, overweight, and obesity also
144 will have positive health benefits beyond these categories of health outcomes. Thus, the U.S.
145 population should be encouraged and guided to consume dietary patterns that are rich in
146 vegetables, fruit, whole grains, seafood, legumes, and nuts; moderate in low- and non-fat dairy
147 products and alcohol (among adults); lower in red and processed meat; and low in sugar-
148 sweetened foods and beverages and refined grains. These dietary patterns can be achieved in
149 many ways and should be tailored to the individual's biological and medical needs as well as
150 socio-cultural preferences.

151

¹ As lean meats were not consistently defined or handled similarly between studies, they were not identified as a common characteristic across the reviews. However, as demonstrated in the food pattern modeling of the Healthy U.S.-style and Healthy Mediterranean-style patterns, lean meats can be a part of a healthy dietary pattern.

152 The dietary pattern characteristics being recommended by the 2015 DGAC reaffirm the dietary
153 pattern characteristics recommended by the 2010 DGAC. Additionally, these characteristics
154 align with recommendations from other groups, including the American Institute for Cancer
155 Research (AICR) and the American Heart Association (AHA). The majority of evidence
156 considered by the Committee focused on dietary patterns consumed in adulthood. Very little
157 evidence examined dietary patterns during childhood. However, the healthy dietary pattern
158 components described above also apply to children and are reaffirmed with the USDA Food
159 Patterns, which are designed to meet nutrient needs across the lifespan.

160

161 **Individual Diet and Physical Activity Behavior Change**

162 The individual is at the innermost core of the social-ecological model. In order for policy
163 recommendations such as the *Dietary Guidelines for Americans* to be fully implemented,
164 motivating and facilitating behavioral change at the individual level is required. This chapter
165 suggests a number of promising behavior change strategies that can be used to favorably affect a
166 range of health-related outcomes and to enhance the effectiveness of interventions. These include
167 reducing screen time, reducing the frequency of eating out at fast food restaurants, increasing
168 frequency of family shared meals, and self-monitoring of diet and body weight as well as
169 effective food labeling to target healthy food choices. These strategies complement
170 comprehensive lifestyle interventions and nutrition counseling by qualified nutrition
171 professionals.

172

173 For this approach to work, it will be essential that the food environments in communities
174 available to the U.S. population, particularly to low-income individuals, facilitate access to
175 healthy and affordable food choices that respect their cultural preferences. Similarly, food and
176 calorie label education should be designed to be understood by audiences with low health
177 literacy, some of which may have additional English language fluency limitations. Although
178 viable approaches are available now, additional research is necessary to improve the scientific
179 foundation for more effective guidelines on individual-level behavior change for all individuals
180 living in the United States, taking into account the social, economic, and cultural environments
181 in which they live.

182

183 The evidence reviewed in this chapter also indicates that the social, economic, and cultural
184 context in which individuals live may facilitate or hinder their ability to choose and consume
185 dietary patterns that are consistent with the Dietary Guidelines. Specifically, household food
186 insecurity hinders the access to healthy diets for millions of Americans. In addition, immigrants
187 are at high risk of losing the healthier dietary patterns characteristic of their cultural background
188 as they acculturate into mainstream America. Furthermore, preventive nutrition services that take
189 into account the social determinants of health are largely unavailable in the U.S. health system to
190 systematically address nutrition-related health problems, including overweight and obesity,
191 cardiovascular disease, type 2 diabetes, and other health outcomes.

192
193 This chapter calls for: a) stronger Federal policies to help prevent household food insecurity and
194 to help families to cope with food insecurity if it develops, b) food and nutrition assistance
195 programs to take into account the risk that immigrants have of giving up their healthier dietary
196 habits soon after arriving in the United States, and c) efforts to provide all individuals living in
197 the United States with the environments, knowledge, and tools needed to implement effective
198 individual- or family-level behavioral change strategies to improve the quality of their diets and
199 reduce sedentary behaviors. These goals will require changes at all levels of the social-ecological
200 model through coordinated efforts among health care and social and food systems from the
201 national to the local level.

202

203

204 **Food Environment and Settings**

205 Environmental and policy approaches are needed to complement individual-based efforts to
206 improve diet and reduce obesity and other diet-related chronic diseases. These approaches have
207 the potential for broad and sustained impact at the population level because they can become
208 incorporated into organizational structures and systems and lead to alterations in sociocultural
209 and societal norms. Both policy and environmental changes also can help reduce disparities by
210 improving access to and availability of healthy food in underserved neighborhoods and
211 communities. Federal nutrition assistance programs, in particular, play a vital role in achieving
212 this objective through access to affordable foods that help millions of Americans meet Dietary
213 Guidelines recommendations.

214

215 The DGAC focused on physical environments (settings) in which food is available. Its aim was
216 to better understand the impact of the food environment to promote or hinder healthy eating in
217 these settings and to identify the most effective evidence-based diet-related approaches and
218 policies to improve diet and weight status. The DGAC focused on four settings—community
219 food access, child care, schools, and worksites—and their relationships to dietary intake and
220 quality and weight status.

221

222 The DGAC found moderate and promising evidence that multi-component obesity prevention
223 approaches implemented in child care settings, schools, and worksites improve weight-related
224 outcomes; strong to moderate evidence that school and worksite policies are associated with
225 improved dietary intake; and moderate evidence that multi-component school-based and
226 worksite approaches increase vegetable and fruit consumption. For the questions on community
227 food access addressing the relationship between food retail settings and dietary intake and
228 quality and weight status, the evidence was too limited or insufficient to assign grades. To reduce
229 the disparity gaps that currently exist in low resource and underserved communities, more
230 solution-oriented strategies need to be implemented and evaluated on ways to increase access to
231 and procurement of healthy affordable foods and beverages, and also to reduce access to energy-

232 dense, nutrient-poor foods and beverages. Although several innovative approaches are taking
233 place now throughout the country, they generally lack adequate evaluation efforts.

234

235 The Committee’s findings revealed the power of multi-component approaches over single
236 component interventions. For obesity prevention, effective multi-component interventions
237 incorporated both nutrition and physical activity using a variety of strategies, such as
238 environmental policies to improve the availability and provision of healthy foods and beverages;
239 increasing opportunities for physical activity; increased parent engagement (in child care and
240 school settings); and educational approaches, such as a school nutrition curriculum. For multi-
241 component dietary interventions (e.g., to increase consumption of vegetables and fruit) the most
242 effective strategies included nutrition education, parent engagement (in school and child care
243 settings), and environmental modifications (e.g., policies for nutrition standards, food service
244 changes, point of purchase information).

245

246 Collaborative partnerships and strategic efforts are needed to translate this evidence into action.
247 Further work on restructuring the environment to facilitate healthy eating and physical activity,
248 especially in high risk populations, is needed to advance evidence-based solutions that can be
249 scaled up.

250

251

252 **Food Sustainability and Safety**

253 Access to sufficient, nutritious, and safe food is an essential element of food security for the U.S.
254 population. A sustainable diet ensures this access for both the current population and future
255 generations.

256

257 The major findings regarding sustainable diets were that a diet higher in plant-based foods, such
258 as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in calories and animal-
259 based foods is more health promoting and is associated with less environmental impact than is
260 the current U.S. diet. This pattern of eating can be achieved through a variety of dietary patterns,
261 including the Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, and the
262 Healthy Vegetarian Pattern. All of these dietary patterns are aligned with lower environmental
263 impacts and provide options that can be adopted by the U.S. population. Current evidence shows
264 that the average U.S. diet has a larger environmental impact in terms of increased greenhouse gas
265 emissions, land use, water use, and energy use, compared to the above dietary patterns. This is
266 because the current U.S. population intake of animal-based foods is higher and plant-based foods
267 are lower, than proposed in these three dietary patterns. Of note is that no food groups need to be
268 eliminated completely to improve sustainability outcomes over the current status.

269

270 A moderate amount of seafood is an important component of two of three of these dietary
271 patterns, and has demonstrated health benefits. The seafood industry is in the midst of rapid

272 expansion to meet worldwide demand. The collapse of some fisheries due to overfishing in the
273 past decades has raised concern about the ability to produce a safe and affordable supply. In
274 addition, concern has been raised about the safety and nutrient content of farm-raised versus
275 wild-caught seafood. To supply enough seafood to support meeting dietary recommendations,
276 both farm-raised and wild caught seafood will be needed. The review of the evidence
277 demonstrated, in the species evaluated, that farm-raised seafood has as much or more EPA and
278 DHA per serving as wild caught. It should be noted that low-trophic seafood, such as catfish and
279 crawfish, regardless of whether wild caught or farm-raised seafood, have less EPA and DHA per
280 serving than high-trophic seafood, such as salmon and trout.

281
282 Regarding contaminants, for the majority of wild caught and farmed species, neither the risks of
283 mercury nor organic pollutants outweigh the health benefits of seafood consumption. Consistent
284 evidence demonstrated that wild caught fisheries that have been managed sustainably have
285 remained stable over the past several decades; however, wild caught fisheries are fully exploited
286 and their continuing productivity will require careful management nationally and internationally
287 to avoid long-term collapse. Expanded supply of seafood nationally and internationally will
288 depend upon the increase of farm-raised seafood worldwide.

289
290 The impact of food production, processing, and consumption on environmental sustainability is
291 an area of research that is rapidly evolving. As further research is conducted and best practices
292 are evaluated, additional evidence will inform both supply-side participants and consumers on
293 how best to shift behaviors locally, nationally, and globally to support sustainable diets. Linking
294 health, dietary guidance, and the environment will promote human health and the sustainability
295 of natural resources and ensure current and long-term food security.

296
297 In regard to food safety, updated and previously unexamined areas of food safety were studied.
298 Currently, strong evidence shows that consumption of coffee within the moderate range (3 to 5
299 cups per day or up to 400 mg/d caffeine) is not associated with increased long-term health risks
300 among healthy individuals. In fact, consistent evidence indicates that coffee consumption is
301 associated with reduced risk of type 2 diabetes and cardiovascular disease in adults. Moreover,
302 moderate evidence shows a protective association between caffeine intake and risk of
303 Parkinson's disease. Therefore, moderate coffee consumption can be incorporated into a healthy
304 dietary pattern, along with other healthful behaviors. However, it should be noted that coffee as
305 it is normally consumed can contain added calories from cream, milk, and added sugars. Care
306 should be taken to minimize the amount of calories from added sugars and high-fat dairy or dairy
307 substitutes added to coffee.

308
309 The marketing and availability of high-caffeine beverages and products is on the rise.
310 Unfortunately, only limited evidence is currently available to ascertain the safety of high caffeine
311 intake (greater than 400 mg/day for adults and undetermined for children and adolescents) that

312 may occur with rapid consumption of large-sized energy drinks. Limited data suggest adverse
313 health outcomes, such as caffeine toxicity and cardiovascular events. Concern is heightened
314 when caffeine is combined with alcoholic beverages. Limited or no consumption of high caffeine
315 drinks, or other products with high amounts of caffeine, is advised for children and adolescents.
316 Energy drinks with high levels of caffeine and alcoholic beverages should not be consumed
317 together, either mixed together or consumed at the same sitting.

318
319 The DGAC also examined the food additive aspartame. At the level that the U.S. population
320 consumes aspartame, it appears to be safe. However, some uncertainty continues about increased
321 risk of hematopoietic cancer in men, indicating a need for more research.

322
323 Individual behaviors along with sound government policies and responsible private sector
324 practices are all needed to reduce foodborne illnesses. To that end, the DGAC updated the
325 established recommendations for handling foods at home.

326
327

328 **Cross-cutting Topics of Public Health Importance**

329 The *2010 Dietary Guidelines* included guidance on sodium, saturated fat, and added sugars, and
330 the 2015 DGAC determined that a reexamination of the evidence on these topics was necessary
331 to determine whether revisions to the guidance were warranted. These topics were considered to
332 be of public health importance because each has been associated with negative health outcomes
333 when overconsumed. Additionally, the Committee acknowledged that a potential unintended
334 consequence of a recommendation on added sugars might be that consumers and manufacturers
335 replace added sugars with low-calorie sweeteners. As a result, the Committee also examined
336 evidence on low-calorie sweeteners to inform statements on this topic.

337
338 The DGAC encourages the consumption of healthy dietary patterns that are low in saturated fat,
339 added sugars, and sodium. The goals for the general population are: less than 2,300 mg dietary
340 sodium per day (or age-appropriate Dietary Reference Intake amount), less than 10 percent of
341 total calories from saturated fat per day, and a maximum of 10 percent of total calories from
342 added sugars per day.

343
344 Sodium, saturated fat, and added sugars are not intended to be reduced in isolation, but as a part
345 of a healthy dietary pattern that is balanced, as appropriate, in calories. Rather than focusing
346 purely on reduction, emphasis should also be placed on replacement and shifts in food intake and
347 eating patterns. Sources of saturated fat should be replaced with unsaturated fat, particularly
348 polyunsaturated fatty acids. Similarly, added sugars should be reduced in the diet and not
349 replaced with low-calorie sweeteners, but rather with healthy options, such as water in place of
350 sugar-sweetened beverages. For sodium, emphasis should be placed on expanding industry

351 efforts to reduce the sodium content of foods and helping consumers understand how to flavor
 352 unsalted foods with spices and herbs.

353
 354 Reducing sodium, saturated fat, and added sugars can be accomplished and is more attainable by
 355 eating a healthy dietary pattern. For all three of these components of the diet, policies and
 356 programs at local, state, and national levels in both the private and public sector are necessary to
 357 support reduction efforts. Similarly, the Committee supports efforts in labeling and other
 358 campaigns to increase consumer awareness and understanding of sodium, saturated fats, and
 359 added sugars in foods and beverages. The Committee encourages the food industry to continue
 360 reformulating and making changes to certain foods to improve their nutrition profile. Examples
 361 of such actions include lowering sodium and added sugars content, achieving better saturated fat
 362 to polyunsaturated fat ratio, and reducing portion sizes in retail settings (restaurants, food outlets,
 363 and public venues, such as professional sports stadiums and arenas). The Committee also
 364 encourages the food industry to market these improved products to consumers.

365
 366
 367 **Physical Activity**

368 This chapter provides strong evidence supporting the importance of regular physical activity for
 369 health promotion and disease prevention in the U.S. population. Physical activity is important for
 370 all people—children, adolescents, adults, older adults, women during pregnancy and the
 371 postpartum period, and individuals with disabilities. The findings further provide guidance on the
 372 dose of physical activity needed across the lifecycle to realize these significant health benefits.

373
 374 Future Physical Activity Guidelines Advisory Committees will be asked to carefully review the
 375 most recent evidence so that the Federal government can fully update the *2008 Physical Activity*
 376 *Guidelines for Americans*. Given the exceedingly low physical activity participation rates in this
 377 country, it will be critically important for the next Committee to identify proven strategies and
 378 approaches to increase population-level physical activity across the lifespan.

379
 380 **INTEGRATING THE EVIDENCE**

381 The research base reviewed by the 2015 DGAC provides clear evidence that persistent,
 382 prevalent, preventable health problems, notably overweight and obesity, cardiovascular disease,
 383 type 2 diabetes, and certain cancers, have adversely affected the health of the U.S. public for
 384 decades and raise the urgency for immediate attention and bold action. Evidence points to
 385 specific areas of current food and nutrient concerns and it pinpoints the characteristics of healthy
 386 dietary and physical activity patterns that can reduce chronic disease risk, promote healthy
 387 weight status, and foster good health across the lifespan. In addition, research evidence is
 388 converging to show that healthy dietary patterns also are more sustainable and associated with
 389 more favorable health as well as environmental outcomes.

390
391
392
393
394
395
396
397
398
399
400
401
402
403
404
405
406
407
408
409

Effective models of “what works” to promote lifestyle behavior change exist. While they can be improved, especially in terms of our capacity for scaling-up in community and health care settings, the evidence to date can be used to guide programs and services for individuals and families. They also can be used to assist the public and private sectors and communities in facilitating innovative environmental change to promote the population’s health.

It will take concerted, bold actions on the part of individuals, families, communities, industry, and government to achieve and maintain the healthy diet patterns and the levels of physical activity needed to promote the health of the U.S. population. These actions will require a paradigm shift to an environment in which population health is a national priority and where individuals and organizations, private business, and communities work together to achieve a population-wide “culture of health” in which healthy lifestyle choices are easy, accessible, affordable, and normative—both at home and away from home. In such a culture, health care and public health professionals also would embrace a new leadership role in prevention, convey the importance of lifestyle behavior change to their patients/clients, set standards for prevention in their own facilities, and help patients/clients in accessing evidence-based and effective nutrition and comprehensive lifestyle services and programs.

Part B. Chapter 1: Introduction

The *Dietary Guidelines for Americans* were first released in 1980, and since that time they have provided science-based advice on promoting health and reducing risk of major chronic diseases through a healthy* diet and regular physical activity. Early editions of the Dietary Guidelines focused specifically on healthy members of the public, but more recent editions also have included those who are at increased risk of chronic disease. Future editions will continue to evolve to address public health concerns and the nutrition needs of specific populations. For example, the Dietary Guidelines have traditionally targeted the general public older than age 2 years, but as data continue to accumulate regarding the importance of dietary intake during gestation and from birth on, a Federal initiative has been established to develop comprehensive guidance for infants and toddlers from birth to 24 months and women who are pregnant. By 2020, the *Dietary Guidelines for Americans* will include these important populations comprehensively.

By law (Public Law 101-445, Title III, 7 U.S.C. 5301 et seq.) the *Dietary Guidelines for Americans* is published by the Federal government every 5 years. To meet this requirement, since the 1985 edition, the Departments have jointly appointed a Dietary Guidelines Advisory Committee of nationally recognized experts in the field of nutrition and health to review the scientific and medical knowledge current at the time. The 2015 Dietary Guidelines Advisory Committee (DGAC) was established for the single, time-limited task of reviewing the 2010 edition of *Dietary Guidelines for Americans* and developing nutrition and related health recommendations to the Federal government for its subsequent development of the 2015 edition. This report presents these recommendations to the Secretaries of Health and Human Services and of Agriculture for use in updating the Guidelines.

The 2015 DGAC recognizes the importance and key function of the Guidelines in forming the basis of Federal nutrition policy and programs. The Guidelines also provides a critical framework for local, state, and national health promotion and disease prevention initiatives. In addition, it provides evidence-based nutrition and physical activity strategies for use by individuals and those who serve them in public and private settings, including public health and social service agencies, health care and educational institutions, and business. The food industry and retailers as well, can use the Guidelines to develop healthy food and beverage products and offerings for consumers.

* Throughout this report, the term "healthy" is used to represent the concept of "health-promoting" as well as to refer to foods or dietary patterns that are consistent with the Dietary Guidelines. See the Glossary for a definition of "health."

35 The potential for the Guidelines to inform policy and practice is critical, given the significant
36 nutrition-related health issues facing the U.S. population:

- 37 • **Overweight, obesity, and other diet-related chronic diseases** (particularly
38 cardiovascular diseases, type 2 diabetes, and certain cancers), as well as less common but
39 important health outcomes, such as bone health, for which nutrition plays an important
40 role. These conditions are prevalent across the entire U.S. population, but are more
41 pronounced in low-income populations, creating critical health disparities that must be
42 addressed.
- 43 • **Less than optimal dietary patterns in the United States**, which contribute directly to
44 poor population health and high chronic disease risk. On average, current dietary patterns
45 are too low in vegetables, fruit, whole grains, and low-fat dairy, and too high in refined
46 grains, saturated fat, added sugars, and sodium.
- 47 • **Food insecurity**, a condition in which the availability of nutritionally adequate foods, or
48 the ability to acquire acceptable foods in socially acceptable ways, is limited or uncertain.
49 More than 49 million people in the United States, including nearly 9 million children, live
50 in food insecure households.

51 The economic and social costs of obesity and other diet- and physical activity-related chronic
52 disease conditions are enormous and will continue to escalate if current trends are not reversed.
53 Therefore, improving diet and physical activity in the population and addressing food insecurity
54 and health disparities have great potential to not only reduce the burden of chronic disease
55 morbidity and mortality, but also to reduce health care costs.

56
57 The DGAC recognized that a dynamic interplay exists among individuals' nutrition, physical
58 activity, and other health-related lifestyle behaviors and their environmental and social contexts.
59 Acknowledging this, the DGAC created a conceptual model based in part on the socio-ecological
60 model to serve as an organizing framework for its report (Figure B2.1). The figure shows how
61 these personal, social, organizational, and environmental contexts and systems interact
62 powerfully to influence individuals' diet and physical activity behaviors and patterns and how
63 diverse health outcomes result from this dynamic interplay. An accompanying table expands on
64 the figure by listing specific factors that comprise each of the "Determinants" and "Outcomes"
65 circles. The table distinguishes those factors that are addressed in the DGAC report from related
66 factors that are important but beyond the scope of the report (see Table B2.1 at the end of this
67 chapter).

68
69

70 **Figure B2.1**

Diet and Physical Activity, Health Promotion, and Disease Prevention at Individual and Population Levels across the Lifespan



72 REVIEWING THE EVIDENCE

73
74 Drawing from this conceptual model, the 2015 DGAC reviewed an extensive and diverse body
75 of scientific literature to address many research questions. For each of its questions, the
76 Committee used a rigorous, evidence-based process to develop its findings. Some of the resulting
77 evidence was strong to moderate, and some was found to be evolving and more limited. This
78 graded evidence was used to draw scientific conclusion and implication statements and to make
79 recommendations that can be used by HHS and USDA in formulating the *Dietary Guidelines for*
80 *Americans* policy document.

81
82 The DGAC used the findings from its evidence reviews to develop a series of chapters that build
83 on and complement each other:

- 84 • **Chapter 1** examines current status and trends in food consumption, nutrient intakes, and
85 eating behaviors and rates and patterns of major nutrition-related health problems. It
86 identifies the nutrients of public health concern and characterizes several dietary patterns that
87 are consistent with those associated with positive health outcomes.
- 88 • **Chapter 2** considers relationships between dietary patterns and health outcomes and
89 identifies a number of commonalities across patterns, particularly food groups, associated
90 with positive health outcomes. It examines these relationships for major chronic diseases
91 (cardiovascular diseases, type 2 diabetes, overweight and obesity, and certain cancers), and
92 also evaluates several less common, but important, outcomes (bone health, neurological and
93 psychological illnesses, congenital anomalies). Where possible, evidence on the impact of
94 dietary or comprehensive lifestyle interventions (including diet, physical activity, and
95 behavioral strategies) in reducing chronic disease risk outcomes is summarized and can be
96 used to inform health promotion and disease prevention strategies at individual and
97 population levels.
- 98 • **Chapter 3** reviews characteristics associated with individual dietary and lifestyle behaviors,
99 such as meal patterns at home and away from home, acculturation, household food
100 insecurity, and sedentary behaviors. It also assesses methods that are effective in helping
101 individuals improve their diet and physical activity behaviors and in enhancing behavioral
102 interventions.
- 103 • **Chapter 4** assesses the roles of food environments and settings in promoting or hindering
104 healthy eating behaviors of specific population groups (such as pre-school and school-age
105 children and adults in the workplace) and evaluates evidence on effective methods and best
106 practices to promote population behavior change in communities as well as public and
107 private settings to influence and improve health.
- 108 • **Chapter 5** focuses on secure and sustainable diets by examining how dietary guidance and
109 food intake influence our capacity to meet the nutrition needs of the U.S. population now and

110 in the future. The chapter also examines issues related to food safety behaviors in the home
 111 environment and evaluates new topics of food safety concern, including the safety of
 112 coffee/caffeine and aspartame.

- 113 • **Chapter 6** considers topics of continuing public health importance that are relevant for
 114 topics across Chapters 1 through 5 and, are therefore addressed together in this chapter—
 115 sodium, saturated fat, added sugars, and low-calorie sweeteners.
- 116 • **Chapter 7** discusses the important role that physical activity plays in promoting health.

117
 118

119 **FROM THE 2015 DGAC ADVISORY REPORT TO THE *DIETARY*** 120 ***GUIDELINES FOR AMERICANS***

121 A major goal of the 2015 DGAC is to summarize and synthesize the evidence to support USDA
 122 and HHS in developing nutrition recommendations that reduce the risk of chronic disease while
 123 meeting nutrient requirements and promoting health of the U.S. population ages 2 years and
 124 older.

125
 126 The U.S. Government uses the Dietary Guidelines as the basis of its food assistance programs,
 127 nutrition education efforts, and decisions about national health objectives. For example, the
 128 National School Lunch Program and the Elder Nutrition Program incorporate the Dietary
 129 Guidelines in menu planning; the Special Supplemental Nutrition Program for Women, Infants,
 130 and Children (WIC) applies the Dietary Guidelines in its educational materials; and the *Healthy*
 131 *People 2020* objectives for the Nation include objectives based on the Dietary Guidelines.

132
 133 The evidence described here in the 2015 DGAC Report, which will be used to develop the 2015
 134 *Dietary Guidelines for Americans*, will help policymakers, educators, clinicians, and others
 135 speak with one voice on nutrition and health and reduce the confusion caused by mixed
 136 messages in the media. The DGAC hopes that the 2015 *Dietary Guidelines for Americans* will
 137 encourage the food industry and retailers to grow, manufacture, and sell foods that promote
 138 health and contribute to appropriate energy balance.

139
 140 In reviewing the evidence on effective interventions and best practices at individual and
 141 population levels, the 2015 DGAC hopes that the 2015 *Dietary Guidelines for Americans* will
 142 also lead to the bold actions needed to transform our health care and public health systems,
 143 communities, and businesses. A concerted and collaborative focus on prevention is needed and
 144 the report provides a foundation of research evidence to help create a national “culture of health”
 145 where healthy lifestyles are easier to achieve and normative. Finally, the 2015 DGAC desires
 146 that its evidence on healthy dietary patterns, which have been found to be important in reducing
 147 disease risk and in promoting food security and sustainability in the near- and long-term, will

148 lead to changes in individual eating behaviors and to systems-wide changes that can help to
149 secure a healthy future for the U.S. population.

150

151 **A GUIDE TO THE 2015 DGAC REPORT**

152 This Report contains several major sections. Part A provides an Executive Summary to the
153 Report. Part B sets the stage for the Report through this Introduction. A second chapter in this
154 section provides an integration of major findings as well as specific recommendations for how
155 the Report's evidence-based dietary recommendations can be put into action at the individual,
156 community, and population levels.

157


158 Part C describes the methodology the DGAC used to conduct its work and review the evidence
159 on diet and health. Part D is the Science Base and contains the chapters described above.


160


161 The Report concludes with a number of Appendices, including a compilation of the Committee's
162 research recommendations; several appendices describing sources of evidence the Committee
163 used in its reviews; a glossary; a brief history of the *Dietary Guidelines for Americans*; a
164 summary of the process used to collect public comments; biographical sketches of DGAC
165 members; a list of DGAC Working Group, Subcommittee, and Working/Writing Group
166 members; and Acknowledgments.


167

Table B2.1: Components of the Conceptual Model**Influences/Determinants**

	Factors	Addressed in the DGAC report	Other factors <u>not</u> addressed in the DGAC report
	Individual & Biological Factors (Represented in the model by characteristics of individuals and their physical makeup that influence lifestyle behaviors)		
	Biological factors	physical and cognitive function; clinical health and nutritional status profile; weight status	appetite, taste and smell acuity; hunger; physical, mental, and emotional well-being; digestion and metabolism; microbiome composition; genetic profile; prescribed medication use; drug-nutrient interactions
	Nutrition, physical activity, and health-related factors	food label use; dietary or physical activity self-monitoring; personal lifestyle profile characteristics including diet, physical activity, and lifestyle behaviors and practices	early diet experiences; perception of food safety and food security; access to nutrition and preventative health counseling; experiences with personal lifestyle behavior change
	Psychological factors	mental health	self/body image; food, nutrition, and health attitudes, beliefs, and preferences; motivation and intentions; self-efficacy; coping skills; mood; stress
	Demographics	age, gender, race/ethnicity, acculturation, income, geography/region, urban/rural location of residence	education, household composition and culture, religion, profession/occupation

 <p>Household, Social & Cultural Factors</p>	Household, Social & Cultural Factors (Represented in the model by structure, resources, values and norms that influence lifestyle behaviors)		
	Family/household/home	parenting and lifestyle behavioral modeling; food and beverage availability; cooking and storage facilities; family and shared meals; physical activity resources	living situation, composition, person(s) responsible for food purchases/preparation; home food environment
	Social/cultural/religious/peer networks	engagement and participation in lifestyle and health-related programs and initiatives	beliefs, norms, values, expectations, and information sharing
	Society and culture		values and investments that support healthy communities and reduce health disparities; stewardship of natural resources and healthy environments
 <p>Community & Environmental Factors</p>	Community & Environmental Factors (Represented in the model by physical and structural characteristics and facilities that provide access to and affect the quality of resources that influence lifestyle behaviors)		
	Food and physical activity	types of available retail food outlets, restaurants, food banks, and farmers' markets; safety, quality and sustainability of available food supplies; patterns of food waste	recreational facilities and resources
	Community	neighborhood food access; child care, schools, and worksites	composition, structure and conditions; social capital and networks; trust and power; disparities and inequities in food security, health, healthcare access, after school programs
	Business/Workplace	corporate/worksites wellness policies and programs, nutrition, exercise and health	employee benefits programs

		services, programs and resources	
	Health care and public health	providers and programs that emphasize lifestyle behavior change, health promotion and disease prevention; accessibility of clinical preventive services including nutrition counseling	health insurance benefits and access including preventative lifestyle services; food and nutrition assistance policies and programming; public and private healthcare networks and infrastructure
	Physical/built/natural environment		green spaces, parks, and recreational resources: availability and access; land use and transportation; abandoned buildings/spaces; soil contamination; chemical, fertilizer, antibiotic and pesticide use
	Ecosystems (national to global)	the natural environment, including farmland; plant, animal, marine, land, and water ecosystems; renewable energy resources; land/water/air and soil environments and quality; plant conservation, biodiversity; greenhouse gas emissions, pollution/contamination	plant and natural resources management and conservation; carbon footprint; global climate change
	Systems & Sectors (Represented in the model by spheres of influence on food availability and diet and physical activity behavior)		
	Consumer		acquisition, consumption, and demand; use, experience and satisfaction
	Retail and service		products, programs, markets; organization and management
	Food, beverage, and agriculture	usual and high levels of caffeine intake; aspartame	farming; import/export; production, processing, storage, distribution, delivery; supply/markets; food and beverage quality



			and safety; food technology and product formulation; advertising; food marketing
	Economy	income	employment; inflation and recession; social, political and human capital; productivity; prices of food
	Other	technology: mobile health (mHealth)	research and technology; emerging trends; entertainment; advertising and marketing; leisure and recreation; media and social media; globalization of trade
	Public & Private Sector Policies (Represented in the model by policies, regulations and laws that influence the availability and quality of products, resources, programs and services that influence diet and physical activity behaviors)		
	Government	federal, state and local food and nutrition assistance programs and/or initiatives that promoting physical activity/movement (e.g. NSLP, SBP, elder nutrition); city and town policies (e.g. taxation, bans, food assistance, price incentives); food and beverage labels	policies, laws and regulations that affect agriculture, food safety and food assistance; educational institutions; employers and worksites; healthcare systems and health insurance
	Business/Workplace	workplace policies on nutrition and physical activity programs, services and resources	employee health benefits (including health insurance) and incentives
	Education and social services across the lifespan	policies, laws and regulations that affect food and beverage availability including competitive foods; nutrition and physical activity programs and services (e.g. in childcare, school, elder care and community settings); food, nutrition, and physical activity services in federal, state	


		and local food assistance settings	
--	--	------------------------------------	--





The central portion of the Conceptual Model represents the concept that the combination of a healthy diet and regular physical activity behaviors and patterns is central to promoting overall health and preventing many chronic diseases.

Health Outcomes

	Factors	Addressed in the DGAC report	Other factors <u>not</u> addressed in the DGAC report
	Healthy Weight (Represented in the model by measures that characterize a health-promoting weight status)		
	Weight and body composition	childhood and adolescence length/height, weight and Z scores, body weight and weight gain, BMI, waist circumference, abdominal obesity, lean and body fat mass; overweight and obesity	
	Physical Fitness & Function (Represented in the model by activities that define a health-promoting level of physical fitness and function)		
	Physical activity and function patterns and behaviors	Aerobic and strengthening activities; occupational, work, and leisure time activity	ability to perform activities of daily living; muscle strength; coordination; falls; physical activity knowledge, awareness and skills
	Sedentary behaviors and sleep patterns	screen time and other sedentary behaviors	sleep patterns (sleep duration, characteristics)

	Healthy Nutritional Status (Represented in the model by the knowledge, behaviors, environmental factors and measures that characterize healthy nutritional status)		
	Dietary patterns	habitual food and nutrient consumption; overall dietary quality and variety	
	Food, beverage and nutrition intake	foods/food groups, beverages (including alcohol), and macro and micronutrients, nutrients of concern and public health significance	
	Dietary product and nutrient supplement use	dietary product and nutrient supplement use	nutraceutical use
	Food and nutrition knowledge, attitudes and skills	food preparation, cooking and nutrition knowledge, attitudes and skills	
	Food security and safety	selection, storage, handling, and preparation of foods and beverages	
	Risk factors and clinical indicators	iron and protein status, vitamin D and folate levels, Vitamin B12 status, hemoglobin A1c; metabolic syndrome (blood lipids and glucose, blood pressure); bone density	urinary sodium, urinary contaminants; protein/calorie malnutrition; micronutrient status

	Chronic Disease Prevention (Represented in the model by health outcomes influenced by diet and physical activity behaviors)		
	Health outcomes	cardiovascular diseases (coronary heart disease, heart attack, hypertension and stroke); Type 2 diabetes; diet-related cancers (breast, colorectal, prostate, lung); neurological and psychological conditions (including cognitive function, dementia, Alzheimer's Disease and depression); dental caries; congenital anomalies; fractures and osteoporosis; total mortality	
	Health Promotion (Represented in the model by diet and physical activity behaviors that promote good health through the lifespan)		
	Health outcomes	pregnancy course and outcomes; child and adolescent growth and development milestones; peri- and post-menopause status; musculoskeletal and bone health; mental health; gastrointestinal health	fertility; healthy aging

Footnote: The DGAC acknowledges that other lifestyle factors were not addressed in its report but are important in overall health, including tobacco status and use, stress and its management, medical treatment and management, medication use, and addiction.

Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence

The 2015 DGAC set out to examine a broad set of research questions in its effort to develop sound recommendations to guide public policies aimed at promoting individual and population health. As these efforts moved forward, it became clear that a number of important, overarching themes were emerging and that these areas provided a solid base of evidence for the Committee's recommendations. In this chapter, we summarize these themes and put forth our overall recommendations to the Secretaries of Health and Human Services and Agriculture.

DGAC 2015 OVERARCHING THEMES

- **The Problem.** About half of all American adults—117 million individuals—have one or more preventable, chronic diseases that are related to poor quality dietary patterns and physical inactivity, including cardiovascular disease, hypertension, type 2 diabetes and diet-related cancers.¹ More than two-thirds of adults and nearly one-third of children and youth are overweight or obese, further exacerbating poor health profiles and increasing risks for chronic diseases and their co-morbidities.^{2,3} High chronic disease rates and elevated population disease risk profiles have persisted for more than two decades and disproportionately affect low-income and underserved communities. These diseases focus the attention of the U.S. health care system on disease treatment rather than prevention; increase already strained health care costs; and reduce overall population health, quality of life, and national productivity. Other less common, but important, diet- and lifestyle-related health problems, including poor bone health and certain neuropsychological disorders and congenital anomalies, pose further serious concerns.
- **The Gap.** The dietary patterns of the American public are suboptimal and are causally related to poor individual and population health and higher chronic disease rates. Few, if any, improvements in consumers' food choices have been seen in recent decades. On average, the U.S. diet is low in vegetables, fruit, and whole grains, and high in sodium, calories, saturated fat, refined grains, and added sugars. Underconsumption of the essential nutrients vitamin D, calcium, potassium, and fiber are public health concerns for the majority of the U.S. population, and iron intake is of concern among adolescents and premenopausal females. Health disparities exist in population access to affordable healthy foods. Eating behaviors of individuals are shaped by complex but modifiable factors, including individual, personal, household, social/cultural, community/environmental, systems/sectorial and policy-level factors (see the 2015 DGAC conceptual model in *Part B. Chapter 1: Introduction*). However, a dynamic and rapidly evolving food environment epitomized by the abundance of highly processed, convenient, lower-cost, energy-dense, nutrient-poor foods makes it

37 particularly challenging to implement health promoting diet-related behavior changes at
38 individual and population levels.

- 39 • **The Dietary Patterns.** Current research provides evidence of moderate to strong links
40 between healthy dietary patterns, lower risks of obesity and chronic diseases, particularly
41 cardiovascular disease, hypertension, type 2 diabetes and certain cancers. Emerging evidence
42 also suggests that relationships may exist between dietary patterns and some neurocognitive
43 disorders and congenital anomalies. *The overall body of evidence examined by the 2015*
44 *DGAC identifies that a healthy dietary pattern is higher in vegetables, fruits, whole grains,*
45 *low- or non-fat dairy, seafood, legumes, and nuts; moderate in alcohol (among adults);*
46 *lower in red and processed meats;ⁱ and low in sugar-sweetened foods and drinks and*
47 *refined grains.* Additional strong evidence shows that it is not necessary to eliminate food
48 groups or conform to a single dietary pattern to achieve healthy dietary patterns. Rather,
49 individuals can combine foods in a variety of flexible ways to achieve healthy dietary
50 patterns, and these strategies should be tailored to meet the individual’s health needs, dietary
51 preferences and cultural traditions. Current research also strongly demonstrates that regular
52 physical activity promotes health and reduces chronic disease risk.
- 53 • **The Individual.** Sound tools and resources, like the *Dietary Guidelines for Americans* and
54 the *Physical Activity Guidelines for Americans*, can help individuals achieve healthy diet and
55 physical activity patterns. Moderate to strong evidence also demonstrates that dietary
56 interventions implemented by nutrition professionals and individual or small-group
57 comprehensive lifestyle interventions that target diet and physical activity and are led by
58 multidisciplinary professional teams provide optimal results in chronic disease risk reduction,
59 weight loss, and weight loss maintenance. Additional evidence indicates that individuals can
60 be helped in their intentions to implement healthy lifestyles by targeting specific eating and
61 physical activity behaviors (e.g., meal patterns, cooking and preparation techniques,
62 family/household meal experiences, reducing sedentary behaviors in adults and youth,
63 reducing screen time in children). Sound behavioral interventions involve engaging
64 individuals actively in the behavior change process, using traditional face-to-face or small
65 group strategies and new technological approaches (websites and mobile/telephone
66 technology), by providing intensive, long-term professional interventions as appropriate, and
67 by monitoring and offering feedback on sustainable behavioral change and maintenance
68 strategies over time.
- 69 • **The Population.** Moderate to strong evidence shows that targeted environmental and policy
70 changes and standards are effective in changing diet and physical activity behaviors and
71 achieving positive health impact in children, adolescents, and adults. Research from early
72 child care settings, schools, and worksites demonstrate that policy changes, particularly when

ⁱ As lean meats were not consistently defined or handled similarly between studies, they were not identified as a common characteristic across the reviews. However, as demonstrated in the food pattern modeling of the Healthy U.S.-style and Healthy Mediterranean-style patterns, lean meats can be a part of a healthy dietary pattern.

73 combined with multi-faceted programs (e.g., nutrition educational initiatives, parent
74 engagement, food labeling, nutrition standards, nutrition and behavioral intervention
75 services) can increase healthy food choices and overall dietary quality, and improve weight
76 outcomes. Population approaches that engage parents and families, as appropriate, involve
77 collaborations across systems and sectors (e.g., schools, food retail, health care institutions
78 and providers, and health insurers), and mobilize public-private partnerships to provide
79 effective models for producing synergistic effects on diet, physical activity, and health-
80 related outcomes.

81 • **The Long-term View.** The 2015 DGAC also examined the near- and long-term
82 sustainability of healthy dietary patterns as well as the safety of certain key dietary
83 constituents (i.e., caffeine and aspartame). Quantitative modeling research showed how
84 healthy dietary patterns relate to positive environmental outcomes that improve population
85 food security. Moderate to strong evidence demonstrates that healthy dietary patterns that are
86 higher in plant-based foods, such as vegetables, fruits, whole grains, legumes, nuts, and
87 seeds, and lower in calories and animal-based foods are associated with more favorable
88 environmental outcomes (lower greenhouse gas emissions and more favorable land, water,
89 and energy use) than are current U.S. dietary patterns. Furthermore, sustainable dietary
90 patterns can be achieved through a variety of approaches consistent with the *Dietary*
91 *Guidelines for Americans* and, therefore, offer individuals many options and new
92 opportunities to align with personal and population health and environmental values systems.
93 Healthy, sustainable dietary patterns also may provide new themes for consumer education
94 and communication on lifestyle practices that can promote food security now and for future
95 generations and create a “culture of health” at individual and population levels.

96 In summary, the research base reviewed by the 2015 DGAC provides clear and consistent
97 evidence that persistent, prevalent, preventable health problems, notably overweight and obesity,
98 cardiovascular diseases, diabetes, and certain cancers, have severely and adversely affected the
99 health of the U.S. population across all stages of the lifespan for decades and raise the urgency
100 for immediate attention and bold action. Evidence points to specific areas of food and nutrient
101 concern in the current U.S. diet. Moderate to strong evidence pinpoints the characteristics of
102 healthy dietary and physical activity patterns established to reduce chronic disease risk, prevent
103 and better manage overweight and obesity, and promote health and well-being across the
104 lifespan.

105
106 Although behavior change is complex, moderate to strong evidence now points to effective
107 strategies to promote healthy lifestyle behavior changes at individual and population levels. This
108 overall research evidence base can be used to inform policy changes, multi-sectorial
109 collaborations, as well as product/service reformulation as needed. It can be used with
110 confidence to provide guidelines and standards for nutrition and lifestyle intervention
111 services/programs in traditional health care and public health settings. It also provides
112 frameworks for public and private sector initiatives and community programming to make

113 innovative environmental changes that can change population diet and physical activity
114 behaviors to promote population health.

115
116 Overall, the evidence base on the links between diet, physical activity, and health has never been
117 as strong or more compelling. The strength of evidence on “what works” to improve individual
118 and population lifestyle behaviors for health also has never been more robust, with solutions and
119 models of “best practices.” Furthermore, the increasing convergence of research evidence
120 showing that healthy dietary patterns not only reduce disease risks and improve health outcomes
121 but are associated with food security and sustainability provide a further, convincing rationale for
122 focused attention on prevention and individual and population health promotion. Additional
123 research must be conducted to strengthen this evidence base, and recommendations for such
124 research are made in each of the chapters in *Part D. Science Base* (see *Appendix E-1: Needs for*
125 *Future Research* for a compilation of the DGAC’s research recommendations).

126
127

128 **DGAC 2015 RECOMMENDATIONS FOR ACTION**

129 It will take concerted, bold action on the part of individuals, families, communities, industry, and
130 government to achieve and maintain healthy dietary patterns and the levels of physical activity
131 needed to promote a healthy U.S. population.

132
133 This will entail dramatic paradigm shifts in which population health is a national priority and
134 individuals, communities, and the public and private sectors seek together to achieve a
135 population-wide “culture of health” through which healthy lifestyle choices are easy, accessible,
136 affordable and normative—both at home and away from home. In such a culture, preventing
137 diet- and physical activity-related diseases and health problems would be much more highly
138 valued, the resources and services needed to achieve and maintain health would become a
139 realized human right across all population strata, the needs and preferences of the individual
140 would be seriously considered, and individuals and their families/households would be actively
141 engaged in promoting their personal health and managing their preventive health services and
142 activities. Health care and public health professionals would embrace a new leadership role in
143 prevention, convey the importance of lifestyle behavior change to their patients/clients, set model
144 standards for prevention-oriented activities and client/employee services in their own facilities,
145 and manage patient/client referrals to evidence-based nutrition and comprehensive lifestyle
146 services and programs. Communities and relevant sectors of our economy, including food,
147 agriculture, private business, health care (as well as insurance), public health and education,
148 would seek common ground and collaborations in promoting population health. Initiatives would
149 be incentivized to engage communities and health care systems to create integrated and
150 comprehensive approaches to preventing chronic diseases and for weight management.
151 Environmental changes, including policy changes, improved food and beverage standards,

152 reformulation of products and services as needed, and programs that enhance population lifestyle
 153 behavior changes and support preventive services also would be incentivized.

154
 155 Although these propositions are extremely challenging, it is imperative to seek novel and
 156 creative, evidence-based solutions. The costs of failing to do so are the continuation of the very
 157 high rates of preventable diet- and physical activity-related health problems we confront as a
 158 Nation and the worsening of their serious adverse effects on our quality of life, population
 159 productivity, and already highly strained healthcare costs. The evidence base has never been
 160 stronger to guide solutions. What is needed are strong commitments and leadership, the
 161 development of targeted public and private policies and partnerships, and the implementation of
 162 evidence-based, cross-sectorial initiatives to achieve them. In the remainder of this chapter, the
 163 DGAC summarizes specific recommendations guided by our conceptual model, which is
 164 grounded in the socio-ecological theory model of individual and population lifestyle behavior
 165 change for health promotion and disease prevention (see *Part B. Chapter 1: Introduction*).

166
 167

168 **Actions for Individuals and Families/Households**

- 169 • Think prevention, know your lifestyle-related health risk profile, make personal goals and
 170 commitments, and take action to promote personal and household/family health. Work with
 171 health professionals to assess and monitor your health risks and to personalize your
 172 preventive lifestyle behavior plan of action.
- 173 • Know and understand how to modify your diet and physical activity to reduce personal and
 174 family member health risks. Know your current dietary pattern, including your healthy
 175 choices that can be maintained as well as areas for potential change. Act on this information.
 176 Seek to make gradual and sustainable changes in your dietary behaviors to achieve one of
 177 several sound healthy dietary pattern options (e.g., Healthy U.S.-style Pattern, the Healthy
 178 Mediterranean-style Pattern, or the Healthy Vegetarian Pattern; see *Part D. Chapter 1: Food
 179 and Nutrient Intakes, and Health: Current Status and Trends*). For most people, this will
 180 mean:
- 181 ○ Improving food and menu choices, modifying recipes (including mixed dishes and
 182 sandwiches), and watching portion sizes.
 - 183 ○ Including more vegetables (without added salt or fat), fruits (without added sugars),
 184 whole grains, seafood, nuts, legumes, low/non-fat dairy or dairy alternatives (without
 185 added sugars).
 - 186 ○ Reducing consumption of red and processed meat, refined grains, added sugars, sodium,
 187 and saturated fat; substituting saturated fats with polyunsaturated alternatives; and
 188 replacing solid animal fats with non-tropical vegetable oils and nuts.

- 189 • The 2015 DGAC advocates achieving healthy dietary patterns through healthy food and
190 beverage choices rather than with nutrient or dietary supplements except as needed.
- 191 • Use available Dietary Guidelines for Americans tools and other sound resources to initiate
192 positive personal lifestyle changes to improve dietary and physical activity behaviors,
193 including goal setting and self-monitoring.
- 194 ○ As needed, seek regular advice from qualified health care providers to establish a
195 personalized plan for prevention that includes steps to adopt healthy dietary patterns and
196 physical activity. As appropriate, engage with nutrition and health professionals to
197 address personal health risks that can be lowered with sound diet and physical activity, or
198 participate in comprehensive lifestyle interventions conducted by trained interventionists
199 (registered dietitians/nutritionists, exercise and behavioral specialists).
- 200 ○ Achieve and maintain a healthy weight. Know your level of obesity risk. Know your
201 energy needs and how they change with varying levels of physical activity. Take personal
202 action for obesity prevention or weight loss management, as needed, using sound,
203 evidence-based tools and resources. Seek to achieve a dietary pattern consistent with the
204 *Dietary Guidelines for Americans*, recognizing that many evidence-based options can
205 facilitate weight loss and weight loss maintenance. As appropriate, work with qualified
206 nutrition professionals and health providers to create a personalized plan of action for
207 obesity prevention. When needed, engage in intensive, long-term nutrition counseling or
208 comprehensive lifestyle intervention strategies to achieve maximal, long-term weight loss
209 and weight maintenance results.
- 210 ○ Ensure at home and in public settings, such as schools and early child care programs, that
211 young children achieve a high-quality dietary pattern and level of physical activity.
212 Encourage their active participation in food experiences and activity choices so that the
213 importance of dietary quality and physical activity are reinforced, and healthy lifestyle
214 behaviors become normative, habitual, and easier to maintain through adolescence and
215 lifelong.
- 216 ○ Follow on a regular basis, the *Physical Activity Guidelines for Americans*. Engage in at
217 least 2.5 hours a week of moderate-intensity aerobic physical activity, such as brisk
218 walking, or 1.25 hours a week of vigorous-intensity aerobic physical activity. For weight
219 control, at least 1 hour a day of moderate- to vigorous-intensity physical activity may be
220 required. Engage children in at least 1 hour a day of moderate- to vigorous-intensity
221 physical activity each day. Limit children’s screen time to no more than two hours per
222 day. Adults should limit sedentary activity and replace it with aerobic and strengthening
223 exercises. As needed, engage with qualified professionals in comprehensive lifestyle
224 interventions to achieve maximal impact on healthy dietary and physical activity patterns
225 and health outcomes. Get enough sleep!

- 226 ○ Seek and demand the creation and maintenance of food and physical activity
227 environments and resources in your community and in local public, private and retail
228 settings so as to promote a “culture of health.” These are strongly needed to facilitate the
229 ease of initiating and meeting the U.S. Dietary Guidelines recommendations at home and
230 away from home.

231

232 **Actions for Communities and Populations**

- 233 ● Aim to make healthy lifestyles and prevention a national and local priority and reality.
- 234 ○ Create public and private policy changes at the national level that direct and incentivize
235 collaborations by multiple sectors of influence, including health care, public health,
236 education, food and agriculture, transportation, food retail, the media, non-governmental
237 organizations, and service sectors.
- 238 ○ Incentivize the development of policies and initiatives at local, state, and Federal levels
239 that are carried out using cross-sectorial collaborations to promote individual healthy
240 lifestyle behavior changes and create community “cultures of health.” These may include
241 improvements in built and physical environments to create safe and accessible resources
242 and settings for increased physical activity and more widely available healthy food
243 choices. They may entail changes in policies, standards, and practices in retail, and public
244 and private settings and programs that promote “cultures of health” and facilitate the
245 initiation and maintenance of healthy lifestyle behaviors at individual and community
246 levels.
- 247 ● Seek a paradigm shift in health care and public health toward a greater focus on prevention
248 and integration with food systems.
- 249 ○ Incentivize and support nutrition professionals, health care providers, and other qualified
250 professionals in their unique roles of encouraging and counseling patients and clients to
251 adopt healthy dietary and physical activity and in offering evidence-based nutrition
252 services and comprehensive lifestyle interventions. Integrate preventive lifestyle
253 screening, referral, and interventions and services for weight management and chronic
254 disease risk reduction into routine practice guidelines and quality assurance standards.
- 255 ○ Support health care facilities, such as hospitals and clinics, in seeking to model
256 prevention and achieving “cultures of health” by offering healthy food choices for
257 patients, visitors, and staff; implementing preventive nutrition services and
258 comprehensive lifestyle intervention programs; and making referrals to Federal and local
259 food assistance programs as needed by their staff and clients.
- 260 ○ Require health insurance providers to use financial and other positive incentives to
261 encourage and motivate health care settings and businesses to support individuals in

- 262 adopting healthy behaviors and engaging, as appropriate, in nutrition and exercise
 263 counseling and comprehensive lifestyle behavior interventions.
- 264 ○ Encourage and incentivize health care innovations and community prevention through
 265 Affordable Care Act (ACA) policies and programs, including expanding preventive
 266 lifestyle services in traditional health services environments and new retail health
 267 services environments that link to Federal and local food assistance programs. These
 268 should provide resources for individuals to engage and sustain personal lifestyle behavior
 269 change. In addition, ACA programs and policies should increase access to qualified
 270 professionals and programs and services that promote healthy diet and physical activity
 271 behaviors.
 - 272 ○ Incentivize businesses to establish employee health benefits plans that include access to
 273 resources and services that encourage personal health promotion and healthy lifestyle
 274 behavior changes. Support employers in using positive motivation strategies to realize
 275 these changes.
- 276
- 277 ● Establish healthy food environments.
 - 278 ○ Establish local, state, and Federal policies to make healthy foods accessible and
 279 affordable and to limit access to high-calorie, nutrient-poor foods and sugar-sweetened
 280 beverages in public buildings and facilities. Set nutrition standards for foods and
 281 beverages offered in public places. Improve retail food environments and make healthy
 282 foods accessible and affordable in underserved neighborhoods and communities.
 - 283 ○ Develop and expand programs that encourage healthy eating and physical activity habits
 284 in young children and adolescents within school and early care and other education
 285 settings. Establish and implement policies and programs that provide nutritious foods,
 286 limit sugar-sweetened beverages and other unhealthy foods, incorporate nutrition
 287 curricula and experiences and physical activity opportunities, and increase provider and
 288 teacher skills to develop and promote these programs.
 - 289 ○ Implement the comprehensive school meal guidelines (National School Lunch Program)
 290 from the USDA that increase intakes of vegetables (without added salt), fruits (without
 291 added sugars), and whole grains; limit sodium, added sugars, saturated fat, and trans fat;
 292 limit marketing unhealthy foods to children; make drinking water freely available to
 293 students throughout the day; ensure competitive foods meet the national nutrition
 294 standards (e.g., *Dietary Guidelines for Americans*); and eliminate sugar-sweetened
 295 beverages.
 - 296 ○ Improve, standardize and implement Nutrition Facts labels and Front of Package labels to
 297 help consumers, including those with low literacy levels, make healthy food choices. The
 298 Nutrition Facts label should include added sugars (in grams and teaspoons) and include a

299 percent daily value, to assist consumers in identifying the amount of added sugars in
300 foods and beverages and making informed dietary decisions. Standardize and create easy-
301 to-understand front-of-package (FOP) label on all food and beverage products to give
302 clear guidance about a food’s healthfulness. An example is the FOP label recommended
303 by the Institute of Medicine, which included calories, and 0 to 3 “nutritional” points for
304 added sugars, saturated fat, and sodium. This would be integrated with the Nutrition Facts
305 label, allowing consumers to quickly and easily identify nutrients of concern for
306 overconsumption, in order to make healthy choices.

307 ○ Align nutritional and agricultural policies with Dietary Guidelines recommendations and
308 make broad policy changes to transform the food system so as to promote population
309 health, including the use of economic and taxing policies to encourage the production and
310 consumption of healthy foods and to reduce unhealthy foods. For example, earmark tax
311 revenues from sugar-sweetened beverages, snack foods and desserts high in calories,
312 added sugars, or sodium, and other less healthy foods for nutrition education initiatives
313 and obesity prevention programs.

314 ○ Align food assistance programs such as SNAP and WIC with the *Dietary Guidelines for*
315 *Americans*. Provide standards for purchasing that create new demands for healthy foods,
316 such as vegetables and fruits, and discourage the purchase and consumption of foods,
317 such as sugar-sweetened beverages. Support research to explore ways to improve overall
318 diet quality in Federal and local food assistance programs.

319 ○ Support changes to the food environment that can help individuals make healthy choices
320 in the foods they consume away from home and those they purchase away from home to
321 consume at home. For example, the Committee encourages the food industry to continue
322 to reformulate and make changes to improve the nutrition profile of certain foods.
323 Examples of such actions include lowering sodium and added sugars content, achieving
324 better saturated fat to polyunsaturated fat ratio, and reducing portion sizes in retail
325 settings (restaurants, food outlets, and public venues, such as professional sports stadiums
326 and arenas). The Committee also encourages the food industry to market these improved
327 products to consumers.

328 ○ Implement policies and programs at local, state and national levels in both the public and
329 private sectors to reduce added sugars and sodium in foods, limit availability of sugar-
330 sweetened beverages, and promote healthy snacks. Approaches might include:

331 ■ Making water a preferred beverage choice. Encourage water as a preferred beverage
332 when thirsty. Make water accessible in public settings, child care facilities, schools,
333 worksites and other community places where beverages are offered.

334 ■ Reducing added sugars in foods and sugar-sweetened beverages in school meals.

- 335 ▪ Making “smart snacks” consistent with the Dietary Guidelines in schools, child care
 336 settings, parks, recreation centers, sports leagues, after-school programs, worksites,
 337 colleges and universities, healthcare, and other community settings.
- 338 ▪ Implementing policies that limit exposure and marketing of foods and beverages high
 339 in added sugars and sodium to all age groups, particularly children and adolescents.
- 340 ▪ Implementing economic and pricing approaches to promote the purchase of healthy
 341 foods and beverages. For example, taxation on higher sugar-and sodium-containing
 342 foods may encourage consumers to reduce consumption and revenues generated
 343 could support health promotion efforts. Alternatively, price incentives on vegetables
 344 and fruits could be used to promote consumption and public health benefits.
- 345 ▪ Mounting public education campaigns to increase the public’s awareness of the health
 346 effects of excess added sugars, sodium, saturated fat, and calories.
- 347 • Support and expand access to healthy built environments and advocate wide community use.
- 348 ○ Increase opportunities for regular public engagement in physical activity through
 349 improved urban and community designs, enhanced community built environments,
 350 business spaces, and transportation networks. Urban and community designs should
 351 encourage and promote active transportation, such as walking and biking. Green
 352 corridors can increase public safety and enhance active transportation.
- 353 ○ Incentivize communities to make physical activity accessible, affordable, and safe.
 354 Encourage public and private sectors to work together to increase access to gyms, bike
 355 trails, pedestrian walkways, ball fields, and other recreation areas in the communities.
 356 Promote physical activity through social media, smart phone, and other technologies.
- 357 ○ Reach out to and engage groups such as new immigrant communities who may abandon
 358 their native healthy lifestyle habits and others at highest nutritional and health risk, to
 359 ensure that they learn about resources and are motivated to access, engage in, and sustain
 360 healthy dietary patterns and physical activities within their cultural preferences.
- 361 • Maintain strong support for Federal food and nutrition programs.
- 362 ○ Recognize their importance in creating demand for healthy food products as well as in
 363 shaping and modeling consumer behaviors relating to healthy dietary and physical
 364 activity patterns.
- 365 ○ Align program standards with the *Dietary Guidelines for Americans* so as to achieve the
 366 2015 DGAC recommendations and promote a “culture of health.”
- 367 • Recognize and place priority on moving toward a more sustainable diet consistent with the
 368 healthy dietary pattern options described in this DGAC report. Access to sufficient,
 369 nutritious, and safe food is an essential element of food security for the U.S. population. A

- 370 sustainable diet helps ensure this access for both the current population and future
 371 generations.
- 372 ○ Enhance what is already being done by the private and public sectors to improve
 373 environmental policies and practices around production, processing, and distribution
 374 *within* individual food categories.
 - 375 ○ Align local, state, and national practices and policies across sectors to promote a
 376 sustainable and safe food supply to ensure long-term food security. Support robust
 377 private and public sector partnerships, practices, and policies across the supply chain and
 378 extending from farms to distribution and consumption that can incentivize actions to
 379 develop a food system that embraces a core set of values that embody healthy, safe, and
 380 sustainable dietary patterns. Monitor, evaluate, and reward sectors that do this. Establish
 381 new, well-coordinated policies that include, but are not limited to, agriculture, economics,
 382 transportation, energy, water use, and dietary guidance. Encourage all participants in the
 383 food system, as they are central to creating and supporting sustainable and safe diets.
 - 384 ○ Shift toward a greater emphasis on healthy dietary patterns and an improved
 385 environmental profile across food categories to maximize environmental sustainability,
 386 including encouraging consumption of a variety of wild caught or farmed seafood.
 - 387 ○ Improve the nutrient profiles of certain farmed seafood species, particularly EPA and
 388 DHA levels, through improved feeding and processing systems and preserve the
 389 favorable nutrient profiles of other seafood. Establish strong policy, research, and
 390 stewardship to improve the environmental sustainability of farmed seafood systems.
 - 391 ○ Offer consumer-friendly information that facilitates understanding the environmental
 392 impact of different foods in food and menu labeling initiatives.
 - 393 ○ Recognize the importance of foodborne illness prevention and encourage consumer
 394 behavior consistent with the four food safety principles described in the *Dietary*
 395 *Guidelines for Americans*—Clean, Separate, Cook, and Chill, which are the foundation of
 396 the Fight BAC![®] campaign (www.fightbac.org).

397

398

399 REFERENCES

- 400 1. Ward BW, Schiller JS, Goodman RA. Multiple chronic conditions among US adults: a 2012
 401 update. *Prev Chronic Dis.* 2014;11:130389. DOI: <http://dx.doi.org/10.5888/pcd11.130389>.
- 402 2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013
 403 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the
 404 American College of Cardiology/American Heart Association Task Force on Practice

- 405 Guidelines. *J Am Coll Cardiol.* 2014;63(25 Pt B):2960-84. PMID: 24239922.
406 <http://www.ncbi.nlm.nih.gov/pubmed/24239922>.
- 407 3. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013
408 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report
409 of the American College of Cardiology/American Heart Association Task Force on Practice
410 Guidelines and The Obesity Society. *J Am Coll Cardiol.* 2014;63(25 Pt B):2985-3023.
411 PMID: 24239920. <http://www.ncbi.nlm.nih.gov/pubmed/24239920>.
- 412
413
414

Part C. Methodology

2 COMMITTEE APPOINTMENT

3 Beginning with the 1985 edition, the U.S. Department of Agriculture (USDA) and U.S.
4 Department of Health and Human Services (HHS) have appointed a Dietary Guidelines Advisory
5 Committee (DGAC) of nationally recognized experts in the field of nutrition and health to
6 review the scientific evidence and medical knowledge current at the time. This Committee has
7 been an effective mechanism for obtaining a comprehensive and systematic review of the science
8 which contributes to successful Federal implementation as well as broad public acceptance of the
9 Dietary Guidelines. The 2015 DGAC was established for the single, time-limited task of
10 reviewing the 2010 edition of *Dietary Guidelines for Americans* and developing nutrition and
11 related health recommendations in this Advisory Report to the Secretaries of USDA and HHS.
12 The Committee was disbanded upon delivery of this report.

13 Nominations were sought from the public through a Federal Register notice published on
14 October 26, 2012. Criteria for nominating prospective members of the DGAC included
15 knowledge about current scientific research in human nutrition and chronic disease, familiarity
16 with the purpose, communication, and application of the Dietary Guidelines, and demonstrated
17 interest in the public's health and well-being through their research and educational endeavors.
18 They also were expected to be respected and published experts in their fields. Expertise was
19 sought in several specialty areas, including, but not limited to, the prevention of chronic diseases
20 (e.g., cancer, cardiovascular disease, type 2 diabetes, overweight and obesity, and osteoporosis);
21 energy balance (including physical activity); epidemiology; food processing science, safety, and
22 technology; general medicine; gerontology; nutrient bioavailability; nutrition biochemistry and
23 physiology; nutrition education and behavior change; pediatrics; maternal/gestational nutrition;
24 public health; and/or nutrition-related systematic review methodology.

25 The Secretaries of USDA and HHS jointly appointed individuals for membership to the 2015
26 DGAC. The chosen individuals are highly respected by their peers for their depth and breadth of
27 scientific knowledge of the relationship between dietary intake and health in all relevant areas of
28 the current Dietary Guidelines.

29 To ensure that recommendations of the Committee took into account the needs of the diverse
30 groups served by USDA and HHS, membership included, to the extent practicable, a diverse
31 group of individuals with representation from various geographic locations, racial and ethnic
32 groups, women, and persons with disabilities. Equal opportunity practices, in line with USDA
33 and HHS policies, were followed in all membership appointments to the Committee.
34 Appointments were made without discrimination on the basis of age, race and ethnicity, gender,
35 sexual orientation, disability, or cultural, religious, or socioeconomic status. Individuals were

36 appointed to serve as members of the Committee to represent balanced viewpoints of the
 37 scientific evidence, and not to represent the viewpoints of any specific group. Members of the
 38 DGAC were classified as Special Government Employees (SGEs) during their term of
 39 appointment, and as such were subject to the ethical standards of conduct for all federal
 40 employees.

41

42

43 **CHARGE TO THE 2015 DIETARY GUIDELINES ADVISORY** 44 **COMMITTEE**

45 The Dietary Guidelines for Americans provide science-based advice on how nutrition and
 46 physical activity can help promote health across the lifespan and reduce the risk for major
 47 chronic diseases in the U.S. population ages 2 years and older.

48 The Dietary Guidelines form the basis of Federal nutrition policy, standards, programs, and
 49 education for the general public and are published jointly by HHS and USDA every 5 years. The
 50 charge to the Dietary Guidelines Advisory Committee, whose duties were time-limited and
 51 solely advisory in nature, was described in the Committee's charter as follows:

- 52 • Examine the *Dietary Guidelines for Americans, 2010* and determine topics for which new
 53 scientific evidence is likely to be available that may inform revisions to the current
 54 guidance or suggest new guidance.
- 55 • Place its primary focus on the systematic review and analysis of the evidence published
 56 since the last DGAC deliberations.
- 57 • Place its primary emphasis on the development of food-based recommendations that are
 58 of public health importance for Americans ages 2 years and older.
- 59 • Prepare and submit to the Secretaries of HHS and USDA a report of technical
 60 recommendations with rationales, to inform the development of the *2015 Dietary*
 61 *Guidelines for Americans*. DGAC responsibilities included providing authorship for this
 62 report; however, responsibilities did not include translating the recommendations into
 63 policy or into communication and outreach documents or programs.
- 64 • Disband upon the submittal of the Committee's recommendations, contained in the
 65 Report of the Dietary Guidelines Advisory Committee on the *Dietary Guidelines for*
 66 *Americans, 2015* to the Secretaries.
- 67 • Complete all work within the 2-year charter timeframe.

68

69 **THE COMMITTEE PROCESS**

70 **Committee Membership**

71 Fifteen members were appointed to the Committee, one of whom resigned within the first 3
72 months of appointment due to new professional obligations (see the ***DGAC Membership***). The
73 Committee served without pay and worked under the regulations of the Federal Advisory
74 Committee Act (FACA). The Committee held seven public meetings over the course of 1½
75 years. Meetings were held in June 2013 and January, March, July, September, November, and
76 December 2014. The members met in person on the campus of the National Institutes of Health
77 in Bethesda, Maryland, for six of the seven meetings. The Committee met by webinar for the
78 November 2014 meeting. All meetings were made publically available live by webcast. In
79 addition, members of the general public were able to attend the Committee's first two meetings
80 in person in Washington DC area. For the remaining meetings, members of the public were able
81 to observe by webcast. All meetings were announced in the *Federal Register*. Meeting
82 summaries, presentations, archived recordings of all of the meetings, and other documents
83 pertaining to Committee deliberations were made available at www.DietaryGuidelines.gov.
84 Meeting materials also were provided at the reference desks of the HHS National Institutes of
85 Health.

86

87 **Public Comments**

88 Written public comments were received throughout the Committee's deliberations through an
89 electronic database and provided to the Committee. This database allowed for the generation of
90 public comment reports as a result of a query by key topic area(s). A general description of the
91 types of comments received and the process used for collecting public comments is described in
92 *Appendix E-7. Public Comments*.

93

94 **DGAC Conceptual Model**

95 Recognizing the dynamic interplay that exists among the determinants and influences on diet and
96 physical activity as well as the myriad resulting health outcomes, the Committee developed a
97 conceptual model to complement its work. The Committee began by reviewing the socio-
98 ecological model in the 2010 *Dietary Guidelines for Americans* and identified the primary goals
99 of the new model: 1) characterize the multiple interrelated determinants of complex nutrition and
100 lifestyle behaviors and health outcomes at individual and population levels, and 2) highlight
101 those areas within this large system that are addressed by the 2015 DGAC review of the
102 evidence. In addition, the Committee sought to develop a model that provided an organizing
103 framework to show readers how the Science Base chapters in this report relate to each other and

104 to the larger food and agriculture, nutrition, physical activity, and health systems in the United
105 States. It first developed an outline that identified a large number of factors and highlighted a
106 select number to be addressed in its evidence reviews of this report. A smaller group of
107 Committee members then developed a draft visual approach for conveying the main messages
108 within a conceptual model. Using the structure of that draft visual, the content of the outline was
109 organized into a supplementary table. The draft outline, resulting visual, and supporting table
110 went through review and input by the members at several stages. The resulting conceptual model
111 and supporting table are found in *Part B. Chapter 1: Introduction*.

112

113 **Approaches to Reviewing the Evidence**

114 The Committee used a variety of scientifically rigorous approaches to address its science-based
115 questions, and some questions were addressed using multiple approaches. The Committee used
116 the state-of-the-art methodology, systematic reviews, to address 27 percent of its science-based
117 research questions. These reviews are publically available in the Nutrition Evidence Library
118 (NEL) at www.NEL.gov. The scientific community now regularly uses systematic review
119 methodologies, so, unlike the 2010 DGAC, the 2015 Committee was able to use existing sources
120 of evidence to answer an additional 45 percent of the questions it addressed. These sources
121 included existing systematic reviews, meta-analyses, or reports. The remainder of the questions,
122 30 percent, were answered using data analyses and food pattern modeling analyses. These three
123 approaches allowed the Committee to ask and answer its questions in a systematic, transparent,
124 and evidence-based manner.

125 For all topics and questions, regardless of the path used to identify and evaluate the scientific
126 evidence, the Committee developed conclusion statements and implications statements.
127 Conclusion statements are a direct answer to the question asked, reflecting the strength of
128 evidence reviewed (see additional details, below, in “Develop Conclusion Statements and Grade
129 the Evidence”). Implications statements were developed to put the Conclusion in necessary
130 context and varied in length depending on the topic or question. The primary purpose of these
131 statements in this report is to describe what actions the Committee recommends that individuals,
132 programs, or policies might take to promote health and prevent disease in light of the conclusion
133 statement. However, some implications statements also provided important statements of fact or
134 references to other processes or initiatives that the Committee felt were critical in providing a
135 complete picture of how their advice should be applied to reach the desired outcomes.

136 Based on the existing body of evidence, research gaps, and limitations, the DGAC also
137 formulated research recommendations that could advance knowledge related to its question and
138 inform future Federal food and nutrition guidance as well as other policies and programs. Some
139 research recommendations were developed and reported for specific topic areas covered in each
140 chapter; others were overarching and covered an entire chapter.

141

142 **Committee Working Structures and Process**

143 The Committee's research questions were developed and prioritized initially by three Working
144 Groups, which then organized themselves into five topic area Subcommittees, and four topic-
145 specific Working or Writing Groups to conduct their work. The Subcommittees were: Food and
146 Nutrient Intakes and Health: Current Status and Trends; Dietary Patterns, Foods and Nutrients,
147 and Health Outcomes; Diet and Physical Activity Behavior Change; Food and Physical Activity
148 Environments; and Food Sustainability and Safety. Working Groups were established on an "as
149 needed" basis when a topic crossed two or more subcommittees. The three working groups were:
150 Sodium, Added Sugars, and Saturated Fats. In addition, a Physical Activity Writing Group was
151 established within the subcommittee on Food and Physical Activity Environments. The
152 Subcommittees, Working Groups, and Writing Groups were made up of three to seven
153 Committee members, with one Committee member appointed as the chair (for subcommittees) or
154 lead (for working or writing groups). The membership of each group is listed in *Appendix E-9*.
155 Although the chair or lead member was responsible for communicating and coordinating all the
156 work that needed to be accomplished within the group, recommendations coordinated by each
157 group ultimately reflected the consensus of the entire Committee from deliberations in the public
158 meetings. In addition, the Committee's Chair and Vice-chair served in an advisory role on each
159 group.

160 Subcommittees and working/writing groups met regularly and communicated by conference
161 calls, webinars, e-mail, and face-to-face meetings. Each group was responsible for presenting the
162 basis for its draft conclusions and implications to the full Committee within the public meetings,
163 responding to questions from the Committee, and making changes, if warranted. To gain
164 perspective for interpreting the science, some groups invited experts on a one-time basis to
165 participate in a meeting to provide their expertise on a particular topic being considered by the
166 group. Two subcommittees also used consultants, who were experts in particular issues within
167 the purview of the subcommittee's work. These consultants participated in subcommittee
168 discussions and decisions on an ongoing basis, but were not members of the full Committee.
169 Like Committee members, they completed training and were reviewed and cleared through a
170 formal Federal process. Seven invited outside experts presented to the full Committee at the
171 January and March, 2014, public meetings. These experts addressed questions posed by the
172 Committee in advance and responded to additional questions during the meetings.

173 In addition to these five subcommittees and four working/writing groups, the DGAC included a
174 Science Review Subcommittee, similar to that formed for the 2010 DGAC. The members
175 included the DGAC Chair and Vice-chair and the two 2015 DGAC members who had also
176 served on the 2010 DGAC. The main focus of this subcommittee was to provide oversight to the
177 whole DGAC process. This Subcommittee played a primary role in organizing the Committee

178 members into their initial work groups, then into subcommittees and working/writing groups. It
179 facilitated the prioritization of topics to be considered by the Committee and provided oversight
180 to ensure that consistent and transparent approaches were used when reviewing the evidence.
181 This oversight also included monitoring the progress of work toward the development of this
182 report in the allotted timeline. As the review of the science progressed, the Science Review
183 Subcommittee meetings were opened to subcommittee Chairs and eventually to other
184 working/writing group Leads when cross-cutting topics were placed on the agenda. In order to
185 adhere to FACA guidelines, full Committee participation was not allowed.

186 The Committee members were supported by HHS’s Designated Federal Officer, who led the
187 administrative effort for this revision process and served as one of four Co-executive Secretaries
188 (two from HHS and two from USDA). Support staff for managing Committee operations
189 consisted of HHS and USDA Dietary Guidelines Management Team members and NEL Team
190 members, including two research librarians. A third Federal staff team, the Data Analyses Team,
191 provided support to the Committee by providing data upon the request of the Committee (see
192 *DGAC Membership* for a list of these DGAC support staff).

193 **DGAC Report Structure**

194 Reflecting the DGAC subcommittee and working/writing group structure, the bulk of the report
195 consists of seven science-based chapters that summarize the evidence assessed and evaluated by
196 the Committee. Five chapters correspond to the work of the five subcommittees; one chapter
197 covers the cross-cutting topics of sodium, saturated fat, and added sugars and low-calorie
198 sweeteners; and one chapter addresses physical activity.

199 Throughout its deliberations, the Committee considered issues related to overall dietary patterns
200 and the need for integrating findings from individual diet and nutrition topic areas. As a result,
201 the Committee included an additional chapter—*Part B. Chapter 2: 2015 DGAC Themes and*
202 *Recommendations: Integrating the Evidence.*

203
204

205 **SYSTEMATIC REVIEW OF THE SCIENTIFIC EVIDENCE**

206 The USDA’s Nutrition Evidence Library (NEL), housed within the Center for Nutrition Policy
207 and Promotion, was responsible for assisting the 2015 DGAC in reviewing the science and
208 supporting development of the 2015 DGAC Report. The NEL used state-of-the-art methodology
209 informed by the Agency for Healthcare Research and Quality (AHRQ),¹ the Cochrane
210 Collaboration,² the Academy of Nutrition and Dietetics³ and the 2011 Institute of Medicine
211 systematic review (SR)⁴ standards to review, evaluate, and synthesize published, peer-reviewed
212 food and nutrition research. The NEL’s rigorous, protocol-driven methodology is designed to

213 maximize transparency, minimize bias, and ensure SRs are relevant, timely, and high-quality.
 214 Using the NEL evidence-based approach enables HHS and USDA to comply with the Data
 215 Quality Act, which states that Federal agencies must ensure the quality, objectivity, utility, and
 216 integrity of the information used to form Federal guidance.

217 DGAC members developed the SR questions and worked with NEL staff to implement the SRs.
 218 The following represent overarching principles for the NEL process:

- 219 • The DGAC made all substantive decisions required during the process.
- 220 • NEL staff provided facilitation and support to ensure that the process was consistently
 221 implemented in accordance with NEL methodology.
- 222 • NEL used document templates, which served as a starting point and were tailored to each
 223 specific review.
- 224 • When working with the DGAC, the Science Review Subcommittee provided oversight to
 225 the DGAC's work throughout the deliberative process, ensuring that the Subcommittees
 226 used consistent and transparent approaches when reviewing the evidence using NEL SRs.

227 The NEL employed a six-step SR process, which leveraged a broad range of expert inputs:

- 228 • Step 1: Develop systematic review questions and analytic frameworks
- 229 • Step 2: Search, screen, and select studies to review
- 230 • Step 3: Extract data and assess the risk of bias of the research
- 231 • Step 4: Describe and synthesize the evidence
- 232 • Step 5: Develop conclusion statements and grade the evidence
- 233 • Step 6: Identify research recommendations

234 Each step of the process was documented to ensure transparency and reproducibility. Specific
 235 information about each review is available at www.NEL.gov, including the research questions,
 236 the related literature search protocol, literature selection decisions, an assessment of the
 237 methodological quality of each included study, evidence summary materials, evidence tables, a
 238 description of key findings, graded conclusion statements, and identification of research
 239 limitations and gaps. These steps are described below.

240 **Develop Systematic Review Questions and Analytic Frameworks**

241 The DGAC identified, refined, and prioritized the most relevant topics and then developed
 242 clearly focused SR questions that were appropriate in scope, reflected the state of the science,
 243 and targeted important policy relevant to public health issue(s). Once topics and systematic

244 review questions were generated, the DGAC developed an analytical framework for each topic in
245 accordance with NEL methodology. These frameworks clearly identified the core elements of
246 the systematic review question/s, key definitions, and potential confounders to inform
247 development of the systematic review protocol.

248 The core elements of a SR question include Population, Intervention or Exposure, Comparator,
249 and Outcomes (PICO). These elements represent key aspects of the topic that need to be
250 considered in developing a SR framework. An analytic framework is a type of evidence model
251 that defines and links the PICO elements and key confounders. The analytical framework serves
252 as a visual representation of the overall scope of the project, provides definitions for key SR
253 terms, helps to ensure that all contributing elements in the causal chain will be examined and
254 evaluated, and aids in determining inclusion and exclusion criteria and the literature search
255 strategy.

256

257 **Search, Screen, and Select Studies to Review**

258 Searching, screening, and selecting scientific literature was an iterative process that sought to
259 identify the most complete and relevant body of evidence to answer a SR question. This process
260 was guided by inclusion and exclusion criteria determined a priori by the DGAC. The NEL
261 librarians created and implemented search strategies that included appropriate databases and
262 search terms to identify literature to answer each SR question. The results of the literature search
263 were screened by the NEL librarians and staff in a dual, step-wise manner, beginning with titles,
264 followed by abstracts, and then full-text articles, to determine which articles met the criteria for
265 inclusion in the review. Articles that met the inclusion criteria were hand searched in an effort to
266 find additional pertinent articles not identified through the electronic search. In addition, NEL
267 staff and the DGAC conducted a duplication assessment to determine whether high-quality SRs
268 or meta-analyses (MA) were available to augment or replace a NEL SR.

269 The DGAC provided direction throughout this process to ensure that the inclusion and exclusion
270 criteria were applied appropriately and the final list of included articles was complete and
271 captured all research available to answer a SR question. Each step of the process also was
272 documented to ensure transparency and reproducibility.

273 The NEL established and the DGAC approved standard inclusion and exclusion criteria to
274 promote consistency across reviews and ensure that the evidence being considered in NEL SRs
275 was most relevant to the U.S. population. The DGAC used these standard criteria and revised
276 them a priori as needed to ensure that they were appropriate for the specific SR being conducted.
277 In general, criteria were established based on the analytical framework to ensure that each study
278 included the appropriate population, intervention/exposure, comparator(s), and outcomes. They
279 were typically established for the following study characteristics:

- 280 • Study design
- 281 • Date of publication
- 282 • Publication language
- 283 • Study setting
- 284 • Study duration
- 285 • Publication status (i.e., peer reviewed)
- 286 • Type, age, and health status of study subjects
- 287 • Size of study groups
- 288 • Study dropout rate

289 To capitalize on existing literature reviews, the NEL performed duplication assessments, which
 290 identified any existing high-quality SRs and/or MAs that addressed the topic or SR questions
 291 posed. Existing SRs and MAs were valuable sources of evidence and were used for two main
 292 purposes in the NEL SR process:

- 293 • To augment a NEL SR as an additional source of evidence, but not as an included study
 294 in the review (in this case, the studies in the existing SR or MA would not be included
 295 individually in the NEL review that was conducted); or
- 296 • To replace a de novo NEL SR.

297 NEL also used existing SRs to provide background and context for current reviews, inform SR
 298 methodology, and cross-check the literature search for completeness.

299 If multiple relevant, low risk of bias, and timely SRs or MA were available, the reviews were
 300 compared and a decision was made as to whether an existing SR/MA would be used, or whether
 301 a de novo SR would be conducted. This decision was made based on the relevancy of the review
 302 in relation to the SR question and, when more than one review was identified, the consistency of
 303 the findings. If existing SRs/MA addressed different aspects of the outcome, more than one
 304 SR/MA may have been used to replace a de novo SR. More information on the use of existing
 305 SRs/MAs to replace a de novo NEL SR is provided below in the section “Existing Sources of
 306 Evidence.”

307

308 **Extract Data and Assess the Risk of Bias**

309 Key information from each study included in a systematic review was extracted and a risk of bias
 310 assessment was performed by a NEL abstractor. NEL abstractors are National Service
 311 Volunteers from across the United States with advanced degrees in nutrition or a related field

312 who were trained to review individual research articles included in NEL systematic reviews (a
313 list of the Volunteers is included in *Appendix E-10: Dietary Guidelines Advisory Committee*
314 *Report Acknowledgments*). From the evidence grids, summary tables are created for each SR
315 that highlight the most relevant data from the reviewed papers. These tables are available on
316 www.NEL.gov.

317 The risk of bias (i.e., internal validity) for each study was assessed using the NEL Bias
318 Assessment Tool (BAT) (see Table C.1 at the end of this chapter). This tool helped in
319 determining whether any systematic error existed to either over- or under-estimate the study
320 results. This tool was developed in collaboration with a panel of international systematic review
321 experts.

322 NEL staff reviewed the work of abstractors, resolved inconsistencies, and generated a draft of a
323 descriptive summary of the body of evidence. The DGAC reviewed this work and used it to
324 inform their synthesis of the evidence.

325

326 **Describe and Synthesize the Evidence**

327 Evidence synthesis is the process by which the DGAC compared, contrasted, and combined
328 evidence from multiple studies to develop key findings and a graded conclusion statement that
329 answered the SR question. This qualitative synthesis of the body of evidence involved
330 identifying overarching themes or key concepts from the findings, identifying and explaining
331 similarities and differences between studies, and determining whether certain factors affected the
332 relationships being examined.

333 To facilitate the DGAC's review and analysis of the evidence, staff prepared a "Key Trends"
334 template for each SR question. This document was customized for each question and included
335 questions related to major trends, key observations, themes for conclusion statements and key
336 findings. It also addressed methodological problems or limitations, magnitude of effect,
337 generalizability of results, and research recommendations. DGAC members used the description
338 of the evidence, along with the full data extraction grid, and full-text manuscripts to complete the
339 "Key Trends" questions. The responses were compiled and used to draft the qualitative evidence
340 synthesis and the conclusion statement.

341

342 **Develop Conclusion Statements and Grade the Evidence**

343 The conclusion statement is a brief summary statement worded as an answer to the SR question.
344 It must be tightly associated with the evidence, focused on general agreement among the studies
345 around the independent variable(s) and outcome(s), and may acknowledge areas of disagreement
346 or limitations, where they exist. The conclusion statement reflects the evidence reviewed and
347 does not include information that is not addressed in the studies. The conclusion statement also
Scientific Report of the 2015 Dietary Guidelines Advisory Committee

348 may identify a relevant population, when appropriate. In addition, “key findings” (approximately
349 3 to 5 bulleted points) were drafted for some questions to provide context and highlight
350 important findings that contributed to conclusion statement development (e.g., brief description
351 of the evidence reviewed, major themes, limitations of the research reviewed or results from
352 intermediate biomarkers).

353 The DGAC used predefined criteria to evaluate and grade the strength of available evidence
354 supporting each conclusion statement. The grade communicates to decision makers and
355 stakeholders the strength of the evidence supporting a specific conclusion statement. The grade
356 for the body of evidence and conclusion statement was based on five elements outlined in the
357 NEL grading rubric: quality, quantity, consistency, impact and generalizability (see Table C.2 at
358 the end of this chapter for the full NEL grading rubric).

359
360

361 **EXISTING SOURCES OF EVIDENCE: REPORTS, SYSTEMATIC** 362 **REVIEWS, AND META-ANALYSES**

363 For a number of topics, the DGAC chose to consider existing high-quality sources of evidence
364 such as existing reports from leading scientific organizations or Federal agencies, SRs, and/or
365 MA to fully or partially address questions. (These three categories of existing sources of
366 evidence are collectively referred to in this report as “existing reports.”) This was done to
367 prevent duplication of effort and promote time and resource management. The methods generally
368 used to identify and review existing reports are described below, and any modifications to this
369 process for answering a question are described in the Methodology section of the individual
370 Science Base chapters (e.g., the DGAC relied on three Federal reports to write the Physical
371 Activity chapter; see the Methods section of *Part D. Chapter 7: Physical Activity* for details on
372 the process the Committee used to review the evidence and develop conclusion statements from
373 these existing reports).

374 First, an analytical framework was developed that clearly described the population,
375 intervention/exposure, comparator, and outcomes (intermediate and clinical) of interest for the
376 question being addressed. When Committee members were aware of high-quality existing
377 reports that addressed their question(s), they decided a priori to use existing report(s), rather than
378 to conduct a de novo NEL SR. A literature search was then conducted to identify other existing
379 reports to augment the existing report(s) identified by the Committee. The literature was
380 searched by a NEL librarian to identify relevant studies. The process used to create and execute
381 the literature search is described in detail above (see “Search, Screen, and Select Studies to
382 Review”). In other cases, the Committee was not aware of any existing reports and intended to
383 conduct a de novo NEL SR. However, as part of the duplication assessment step of the NEL
384 process, one or more existing SRs or MA were identified that addressed the question that led to

385 the Committee deciding to proceed using existing SRs/MA rather than complete an independent
386 review of the primary literature. This process is also described above. Finally, for some
387 questions, the Committee used existing reports as the primary source of evidence to answer a
388 question, but chose to update one or more of those existing reports using the NEL process to
389 identify and review studies that had been published after the completion of the literature search
390 for the existing report(s).

391 When SRs or MA that addressed the question posed by the Committee were identified, staff
392 conducted a quality assessment using the Assessment of Multiple Systematic Reviews
393 (AMSTAR) tool.⁵ This tool includes 11 questions, each of which is given a score of one if the
394 criterion is met or a score of zero if the criterion is not met, is unclear, or is not applicable (see
395 Table C.3 at the end of this chapter). Guidance for answering some of the questions was tailored
396 for the work of the Committee. Articles rated 0-3 were considered to be of low quality, 4-7 of
397 medium quality, and 8-11 of high quality.⁶ Unless otherwise noted, only high quality SRs/MA,
398 receiving scores of 8-11, were considered by the DGAC.

399 In a few cases, existing reports were considered that did not examine the evidence using SR or
400 MA. These reports were discussed by the subcommittees and determined to be of high-quality.
401 The subcommittees also had the option of bringing existing reports to the Science Review
402 Subcommittee to ensure that the report met the quality standards of the Committee, if needed.

403 Next, if multiple high-quality existing reports were identified, their reference lists were
404 compared to find whether any references and/or cohorts were included in more than one of the
405 existing reports. The Committee then addressed the overlap in their review of the evidence
406 ensuring that, in cases where overlap existed, that the quantity of evidence available was not
407 overestimated. In a few cases, if two or more SRs/MAs appropriately answered a question and
408 there was substantial reference overlap, the Committee chose to only use one of the SRs/MA to
409 answer the question.

410 Tables or other documents that summarized the methodology, evidence, and conclusions of the
411 existing reports were used by the Committee members to facilitate their review of the evidence.
412 For example, a “Key Trends” document was often used to help identify themes observed in the
413 body of evidence. The “Key Trends” document included questions related to major trends, key
414 observations, themes for key findings, and conclusion statements. Members of the DGAC used
415 the description of the evidence, along with summary tables and the original reports, to answer the
416 questions. Feedback from the DGAC on the “Key Trends” document was compiled and used to
417 draft the qualitative evidence synthesis and the conclusion statement. As described above, the
418 conclusion statement is a brief summary statement worded as an answer to the question. In
419 drawing conclusions, Committee members could choose to:

420 1. Carry forward findings or conclusions from existing report(s).

- 421 2. Synthesize the findings from multiple existing report(s) to develop their own conclusions.
422 3. Place primary emphasis on the existing report(s) and discuss how new evidence identified
423 through the NEL process relates to the conclusions or findings of the existing report(s).

424 Next, the Committee graded their conclusion statement using a table of strength of evidence
425 grades adapted specifically use with existing reports (see Table C.4 at the end of this chapter). In
426 cases where the DGAC used an existing report with its own formally graded conclusions, the
427 Committee acknowledged the grade assigned within that existing report, and then assigned a
428 DGAC grade that was the closest equivalent to the grade assigned in the existing report.

429
430

431 **DATA ANALYSES**

432 **Federal Data Acquisition**

433 Earlier Committees used selected national, Federal data about the dietary, nutritional, and health
434 status of the U.S. population. In the 2015 DGAC, a Data Analysis Team (DAT) was established
435 to streamline the data acquisition process and efficiently support the data requests of the
436 Committee. During the Committee's work, the data used by the DGAC were publically available
437 through www.DietaryGuidelines.gov. Upon publication, the data became available through the
438 report's references and appendices.

439 Upon request from the DGAC, the DAT either conducted data analyses or compiled data from
440 their agencies' publications for the DGAC to use to answer specific research questions. The
441 DGAC took the strengths and limitations of data analyses into account in drawing conclusions.
442 The grading rubric used for questions answered using NEL systematic reviews do not apply for
443 to questions answered using data analyses; therefore, these conclusions were not graded.

444 Most of the analyses used the National Health and Nutrition Examination (NHANES) data and
445 its dietary component, What We Eat in America (WWEIA), NHANES.⁷ These data were used to
446 answer questions about food and nutrient intakes because they provide national and group level
447 estimates of dietary intakes of the U.S. population, on a given day as well as usual intake
448 distributions. These data contributed substantially to questions answered using data analyses (see
449 *Appendix E-4: NHANES Data Used in DGAC Data Analyses* for additional discussion of the
450 NHANES data used by the 2015 DGAC).

451 ***NHANES Data***

452 The NHANES data used by the 2015 DGAC included:

- 453 • Estimates of the distribution of usual intakes of energy and selected macronutrients and
454 micronutrients from food and beverages by various demographic groups, including the
455 elderly population, race/ethnicities, and pregnant women.
- 456 • Estimates of the distribution of usual intakes of selected nutrients from food, beverages,
457 and supplements.
- 458 • Estimates of the distribution of usual intake of USDA Food Pattern food groups by
459 demographic population groups.
- 460 • Eating behaviors such as meal skipping, contribution of meals and snacks to energy and
461 nutrient intakes.
- 462 • Nutrients and food group content per 1,000 calories of food and beverages obtained from
463 major point of purchase.
- 464 • Nutritional quality of food prepared at home and away from home.
- 465 • Energy, selected nutrients, and food groups obtained from food categories by
466 demographic population groups.
- 467 • Selected biochemical indicators of diet and nutrition in the U.S. population.
- 468 • Prevalence of health concerns and trends, including body weight status, lipid profiles,
469 high blood pressure, and diabetes.

470 ***Other Data Sources***

471 The DGAC also used data from the National Health Interview Survey, the National Cancer
472 Institute’s Surveillance, Epidemiology, and End Results (SEER) statistics, and heart disease and
473 stroke statistics from the 2014 report of the American Heart Association.^{8,9} In addition, the
474 Committee used USDA National Nutrient Database for Standard Reference, Release 27, 2014 to
475 list food sources ranked by amounts of selected nutrients (calcium, fiber, iron, potassium, and
476 Vitamin D) and energy per standard food portions and per 100 grams of foods.¹⁰

477

478

479 **SPECIAL ANALYSES USING THE USDA FOOD PATTERNS**

480 As described above, the Committee used NEL systematic reviews, existing reports, and data
481 analyses to draw the majority of its conclusions on the relationship between diet and health.
482 Because the primary charge of the Committee is to provide food-based recommendations with
483 the potential to inform the next edition of the *Dietary Guidelines for Americans*, it was
484 imperative that the Committee also advise the government on how to articulate the evidence on
485 the relationships between diet and health through food patterns. This was a critical task for the

486 Committee because the *Dietary Guidelines* are the basis for all Federal nutrition assistance and
487 educational initiatives. For this reason, like the 2005 and 2010 DGAC's, this Committee
488 developed a number of questions to be answered through a food pattern modeling approach,
489 using the USDA Food Patterns.

490 Briefly, the USDA Food Patterns describe types and amounts of food to consume that will
491 provide a nutritionally adequate diet. They include recommended intakes for five major food
492 groups and for subgroups within several of the food groups. They also recommend an allowance
493 for intake of oils and limits on intake of calories from solid fats and added sugars. The calories
494 and nutrients that would be expected from consuming a specified amount from each component
495 of the patterns (e.g., whole grains, fruits, or oils) are determined by calculating nutrient profiles.
496 A nutrient profile is the average nutrient content for each component of the Patterns. The profile
497 is calculated from the nutrients in nutrient-dense forms of foods in each component, and is
498 weighted based on the relative consumption of each of these foods. Additional details on the
499 USDA Food Patterns can be found in the report for the food pattern modeling analysis, *Adequacy*
500 *of the USDA Food Patterns* (see **Appendix E-3: USDA Food Patterns for Special Analyses**).

501 The USDA Food Patterns were originally developed in the 1980s,^{11, 12} and were substantially
502 revised and updated in 2005, concurrent with the development of the 2005 Dietary Guidelines.¹³
503 The Patterns were updated and slightly revised in 2010, concurrent with the development of the
504 2010 Dietary Guidelines.¹⁴ The 2005 and 2010 updates included use of nutrient goals from the
505 Institute of Medicine *Dietary Reference Intakes* reports that were released from 1997 to 2004.¹⁵⁻
506 ²⁰ The developmental process and the food patterns resulting from the 2005 and 2010 updates
507 have been documented in detail.^{13, 14, 21}

508 A food pattern modeling process was developed for the 2005 DGAC and used by the 2005 and
509 2010 DGACs to determine the hypothetical effect on nutrients in and adequacy of the Food
510 Patterns when specific changes are made.^{13, 14} The structure of the USDA Food Patterns allows
511 for modifications that test the overall influence on diet quality of various dietary
512 recommendation scenarios. Most analyses involved identifying the impact of specific changes in
513 amounts or types of foods that might be included in the pattern. Changes might involve
514 modifying the nutrient profiles for a food group, or changing amounts recommended for a food
515 group or subgroup, based on the assumptions for the food pattern modeling analysis. For
516 example, 2005 DGAC subcommittees requested analyses to obtain information on the potential
517 effect of consumers selecting only lacto-ovo vegetarian choices, eliminating legumes, or
518 choosing varying levels of fat as a percent of calories²² on nutritional adequacy. The use of food
519 pattern modeling analyses for the 2005 and 2010 DGAC have been documented.²³⁻²⁶

520 The DGAC referred questions that could be addressed through food pattern modeling to the Food
521 and Nutrient Intakes and Health: Current Status and Trends Subcommittee. The DGAC

522 identified that a number of questions could be answered by modeling analyses conducted for the
 523 2005 or 2010 DGACs. The food pattern modeling analyses conducted for the 2015 DGAC are
 524 listed in *Appendix E-3: USDA Food Pattern Modeling Analyses*. For each question answered
 525 using food pattern modeling, a specific approach was drafted by USDA staff and provided to the
 526 DGAC for comment. After the approach was adjusted and approved by the DGAC, USDA staff
 527 completed the analytical work and drafted a full report for the DGAC’s consideration.

528 The modeling process also was used to develop new USDA Food Patterns based on different
 529 types of evidence: the “Healthy Vegetarian Pattern,” which takes into account food choices of
 530 self-identified vegetarians, and the “Healthy Mediterranean-style Pattern,” which takes into
 531 account food group intakes from studies using a Mediterranean diet index to assess dietary
 532 patterns. The latter were compiled and summarized to answer the questions addressed on dietary
 533 patterns composition. The food group content of dietary patterns reviewed by the DGAC and
 534 found to have health benefits formed the basis for answering these questions. WWEIA food
 535 group intakes and USDA Food Pattern recommendations were compared with the food group
 536 intake data from the healthy dietary patterns as part of the answer for these questions.

537
 538

539 REFERENCES

- 540 1. Methods Guide for Comparative Effectiveness Reviews. AHRQ Publication No. 10(14)-
 541 EHC063-EF. Rockville, MD: Agency for Healthcare Research and Quality; January
 542 2014. Available from: www.effectivehealthcare.ahrq.gov.
- 543 2. Higgins J, Green S, editors. Cochrane Handbook for Systematic Reviews of Interventions
 544 Version 5.1.0: The Cochrane Collaboration; 2011 [updated March 2011]. Available
 545 from: <http://handbook.cochrane.org/>.
- 546 3. American Dietetic Association. Research and Strategic Business Development. Evidence
 547 Analysis Manual Chicago, IL: American Dietetic Association; 2012 [updated January
 548 2012]. Available from: http://www.andea.org/files/Docs/2012_Jan_EA_Manual.pdf.
- 549 4. Institute of Medicine. Finding What Works in Health Care: Standards for Systematic
 550 Reviews. Washington, DC: The National Academies Press; 2011. Available from:
 551 http://books.nap.edu/openbook.php?record_id=13059.
- 552 5. Shea BJ, Grimshaw JM, Wells GA, Boers M, Andersson N, Hamel C, et al. Development
 553 of AMSTAR: a measurement tool to assess the methodological quality of systematic
 554 reviews. *BMC Med Res Methodol*. 2007;7:10. PMID: 17302989.
 555 <http://www.ncbi.nlm.nih.gov/pubmed/17302989>.
- 556 6. Sharif MO, Janjua-Sharif FN, Ali H, Ahmed F. Systematic reviews explained:
 557 AMSTAR-how to tell the good from the bad and the ugly. *Oral Health Dent Manag*.
 558 2013;12(1):9-16. PMID: 23474576. <http://www.ncbi.nlm.nih.gov/pubmed/23474576>.
- 559 7. Zipf G, Chiappa M, Porter K. National Health and Nutrition Examination Survey: Plan
 560 and operations, 1999–2010. *Vital Health Stat*. 2013; 1(56). Available from:
 561 http://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf.

- 562 8. Blackwell D, Lucas J, Clarke T. Summary health statistics for U.S. adults: National
563 Health Interview Survey, 2012. *Vital Health Stat* 10(260). National Center for Health
564 Statistics; 2014. Available from:
565 http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.
- 566 9. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, et al. Heart disease
567 and stroke statistics--2014 update: a report from the American Heart Association.
568 *Circulation*. 2014;129(3):e28-e292. PMID: 24352519.
569 <http://www.ncbi.nlm.nih.gov/pubmed/24352519>.
- 570 10. U.S. Department of Agriculture. Agricultural Research Service. USDA National Nutrient
571 Database for Standard Reference, Release 27. Nutrient Data Laboratory Home Page.
572 2014. Available from: <http://www.ars.usda.gov/Services/docs.htm?docid=8964>.
- 573 11. Cronin FJ, Shaw AM, Krebs-Smith SM, Marsland PM, Light L. Developing a food
574 guidance system to implement the dietary guidelines. *Journal of Nutrition Education*.
575 1987;19(6):281-302.
576 <http://www.sciencedirect.com/science/article/pii/S0022318287802376>.
- 577 12. Welsh S, Davis C, Shaw A. USDA's Food Guide: Background and Development.
578 Hyattsville, MD: U.S. Department of Agriculture. Human Nutrition Information
579 Service. Misc. Publication 1514, September 1993. Available from:
580 [http://www.cnpp.usda.gov/sites/default/files/archived_projects/FGPBackgroundAndDeve
581 lopment.pdf](http://www.cnpp.usda.gov/sites/default/files/archived_projects/FGPBackgroundAndDevelopment.pdf).
- 582 13. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the
583 MyPyramid Food Guidance System. *J Nutr Educ Behav*. 2006;38(6 Suppl):S78-92.
584 PMID: 17116598. <http://www.ncbi.nlm.nih.gov/pubmed/17116598>.
- 585 14. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated
586 US Department of Agriculture Food Patterns meet goals of the 2010 dietary guidelines. *J*
587 *Acad Nutr Diet*. 2012;112(10):1648-55. PMID: 22853987.
- 588 15. Institute of Medicine. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium,
589 Vitamin D, and Fluoride. Washington, DC: The National Academies Press; 1997.
590 Available from: http://www.nap.edu/catalog.php?record_id=5776.
- 591 16. Institute of Medicine. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin,
592 Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington,
593 DC: The National Academies Press; 1998. Available from:
594 http://www.nap.edu/openbook.php?record_id=6015.
- 595 17. Institute of Medicine. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium,
596 and Carotenoids. Washington, DC: The National Academies Press; 2000. Available from:
597 http://www.nap.edu/openbook.php?record_id=9810.
- 598 18. National Research Council. Dietary Reference Intakes for Vitamin A, Vitamin K,
599 Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel,
600 Silicon, Vanadium, and Zinc. Washington, DC: The National Academies Press; 2001.
601 Available from: http://www.nap.edu/openbook.php?record_id=10026.
- 602 19. National Research Council. Dietary Reference Intakes for Energy, Carbohydrate, Fiber,
603 Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington,
604 DC: The National Academies Press; 2002. Available from:
605 http://www.nap.edu/openbook.php?record_id=10490.

- 606 20. National Research Council. Dietary Reference Intakes for Water, Potassium, Sodium,
607 Chloride, and Sulfate. Washington, DC: The National Academies Press; 2005. Available
608 from: http://www.nap.edu/catalog.php?record_id=10925.
- 609 21. Marcoe K, Juan W, Yamini S, Carlson A, Britten P. Development of food group
610 composites and nutrient profiles for the MyPyramid Food Guidance System. *J Nutr Educ*
611 *Behav.* 2006;38(6 Suppl):S93-S107. PMID: 17116599.
612 <http://www.ncbi.nlm.nih.gov/pubmed/17116599>.
- 613 22. Dietary Guidelines Advisory Committee. 2005. Report of the Dietary Guidelines
614 Advisory Committee on the Dietary Guidelines for Americans, to the Secretary of Health
615 and Human Services and the Secretary of Agriculture: U.S. Department of Agriculture,
616 Agricultural Research Service; 2005. Available from:
617 <http://www.health.gov/dietaryguidelines/dga2005/report/default.htm>.
- 618 23. Britten P, Lyon J, Weaver CM, Kris-Etherton PM, Nicklas TA, Weber JA, et al.
619 MyPyramid food intake pattern modeling for the Dietary Guidelines Advisory
620 Committee. *J Nutr Educ Behav.* 2006;38(6 Suppl):S143-52. PMID: 17116592.
621 <http://www.ncbi.nlm.nih.gov/pubmed/17116592>.
- 622 24. Nicklas TA, Weaver C, Britten P, Stitzel KF. The 2005 Dietary Guidelines Advisory
623 Committee: developing a key message. *J Am Diet Assoc.* 2005;105(9):1418-24. PMID:
624 16129084. <http://www.ncbi.nlm.nih.gov/pubmed/16129084>.
- 625 25. Weaver C, Nicklas T, Britten P. The 2005 Dietary Guidelines Advisory Committee
626 Report: Achieving Nutritional Recommendations Through Food-based Guidance.
627 *Nutrition Today.* 2005;40(3):102-7. Available from:
628 [http://journals.lww.com/nutritiontodayonline/Abstract/2005/05000/The_2005_Dietary_G](http://journals.lww.com/nutritiontodayonline/Abstract/2005/05000/The_2005_Dietary_Guidelines_Advisory_Committee.3.aspx)
629 [uidelines_Advisory_Committee.3.aspx](http://journals.lww.com/nutritiontodayonline/Abstract/2005/05000/The_2005_Dietary_Guidelines_Advisory_Committee.3.aspx)
- 630 26. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Impact of
631 typical rather than nutrient-dense food choices in the US Department of Agriculture Food
632 Patterns. *J Acad Nutr Diet.* 2012;112(10):1560-9. PMID: 22906562.
633 <http://www.ncbi.nlm.nih.gov/pubmed/22906562>.

634

635 **Table C.1 Nutrition Evidence Library Bias Assessment Tool (BAT)**

636 The NEL Bias Assessment Tool (NEL BAT) is used to assess the risk of bias of each individual
637 study included in a SR. The types of bias that are addressed in the NEL BAT include:

Selection Bias	Systematic differences between baseline characteristics of the groups that are compared; error in choosing the individuals or groups taking part in a study
Performance Bias	Systematic differences between groups in the intervention/exposure received, or in experience with factors other than the interventions/exposures of interest
Detection Bias	Systematic differences between groups in how outcomes are determined; outcomes are more likely to be observed or reported in certain subjects
Attrition Bias	Systematic differences between groups in withdrawals from a study, particularly if those who drop out of the study are systematically different from those who remain in the study
Adapted from: Cochrane Bias Methods Group: http://bmg.cochrane.org/assessing-risk-bias-included-studies	

638

639 The NEL BAT is tailored by study design, with different sets of questions applying to
640 randomized controlled trials (14 questions), non-randomized controlled trials (14 questions), and
641 observational studies (12 questions). Abstractors complete the NEL BAT after data extraction for
642 each article. There are four response options:

- 643 ▪ **Yes:** Information provided in the article is adequate to answer “yes”.
- 644 ▪ **No:** Information provided in the article clearly indicates an answer of “no”.
- 645 ▪ **Cannot Determine:** No information or insufficient information is provided in the article,
646 so an answer of “yes” or “no” is not possible.
- 647 ▪ **N/A:** The question is not applicable to the article.

The NEL Bias Assessment Tool (NEL BAT)		
Risk of Bias Questions	Study Designs	Type of Bias
Were the inclusion/exclusion criteria similar across study groups?	Controlled trials Observational studies	Selection Bias
Was the strategy for recruiting or allocating participants similar across study groups?	Controlled trials Observational studies	Selection Bias
Was the allocation sequence randomly generated?	RCTs	Selection Bias
Was the group allocation concealed (so that assignments could not be predicted)?	RCTs	Selection Bias Performance Bias
Was distribution of health status,	RCTs	Selection Bias

demographics, and other critical confounding factors similar across study groups at baseline? If not, does the analysis control for baseline differences between groups?	Controlled trials Observational studies	
Did the investigators account for important variations in the execution of the study from the proposed protocol or research plan?	RCTs Controlled trials Observational studies	Performance Bias
Was adherence to the study protocols similar across study groups?	RCTs Controlled trials Observational studies	Performance Bias
Did the investigators account for the impact of unintended/unplanned concurrent interventions or exposures that were differentially experienced by study groups and might bias results?	RCTs Controlled trials Observational studies	Performance Bias
Were participants blinded to their intervention or exposure status?	RCTs Controlled trials	Performance Bias
Were investigators blinded to the intervention or exposure status of participants?	RCTs Controlled trials	Performance Bias
Were outcome assessors blinded to the intervention or exposure status of participants?	RCTs Controlled trials Observational studies	Detection Bias
Were valid and reliable measures used consistently across all study groups to assess inclusion/exclusion criteria, interventions/exposures, outcomes, participant health benefits and harms, and confounding?	RCTs Controlled trials Observational studies	Detection Bias
Was the length of follow-up similar across study groups?	RCTs Controlled trials Observational studies	Attrition Bias
In cases of high or differential loss to follow-up, was the impact assessed (e.g., through sensitivity analysis or other adjustment method)?	RCTs Controlled trials Observational studies	Attrition Bias
Were other sources of bias taken into account in the design and/or analysis of the study (e.g., through matching, stratification, interaction terms, multivariate analysis, or other statistical adjustment such as instrumental variables)?	RCTs Controlled trials Observational studies	Attrition, Detection, Performance, and Selection Bias
Were the statistical methods used to assess the primary outcomes adequate?	RCTs Controlled trials Observational studies	Detection Bias

649 The completed NEL BAT is used to rate the overall risk of bias for the article by tallying the
650 responses to each question. Each “Yes” response receives 0 points, each “Cannot Determine”
651 response receives 1 point, each “No” response receives 2 points, and each “N/A” response
652 receives 0 points. Since 14 questions are answered for randomized controlled trials and non-
653 randomized controlled trials, they will be assigned a risk of bias rating out of a maximum of 28
654 points; while observational studies will be out of 24 points. The lower the number of points
655 received, the lower the risk of bias.

656

657 **Table C.2 NEL Grading Rubric**

USDA Nutrition Evidence Library Conclusion Statement Evaluation				
Criteria for judging the strength of the body of evidence supporting the Conclusion Statement				
Elements	Grade I: Strong	Grade II: Moderate	Grade III: Limited	Grade IV: Grade Not Assignable*
Risk of bias (as determined using the NEL Bias Assessment Tool)	Studies of strong design free from design flaws, bias and execution problems	Studies of strong design with minor methodological concerns OR only studies of weaker study design for question	Studies of weak design for answering the question OR inconclusive findings due to design flaws, bias or execution problems	Serious design flaws, bias, or execution problems across the body of evidence
Quantity • Number of studies • Number of subjects in studies	Several good quality studies; large number of subjects studied; studies have sufficiently large sample size for adequate statistical power	Several studies by independent investigators; doubts about adequacy of sample size to avoid Type I and Type II error	Limited number of studies; low number of subjects studied and/or inadequate sample size within studies	Available studies do not directly answer the question OR no studies available
Consistency of findings across studies	Findings generally consistent in direction and size of effect or degree of association and statistical significance with very minor exceptions	Some inconsistency in results across studies in direction and size of effect, degree of association or statistical significance	Unexplained inconsistency among results from different studies	Independent variables and/or outcomes are too disparate to synthesize OR single small study unconfirmed by other studies
Impact • Directness of studied outcomes • Magnitude of effect	Studied outcome relates directly to the question; size of effect is clinically meaningful	Some study outcomes relate to the question indirectly; some doubt about the clinical significance of the effect	Most studied outcomes relate to the question indirectly; size of effect is small or lacks clinical significance	Studied outcomes relate to the question indirectly; size of effect cannot be determined
Generalizability to the U.S. population of interest	Studied population, intervention and outcomes are free from serious doubts about generalizability	Minor doubts about generalizability	Serious doubts about generalizability due to narrow or different study population, intervention or outcomes studied	Highly unlikely that the studied population, intervention AND/OR outcomes are generalizable to the population of interest

658

659

660 **Table C.3 AMSTAR (Assessment of Multiple Systematic Reviews) Tool**

		YES	NO	Can't Answer	N/A
1	Was an 'a priori' design provided? <i>The research question and inclusion criteria should be established before the conduct of the review.</i>				
2	Was there duplicate study selection and data extraction? <i>There should be at least two independent data extractors and a consensus procedure for disagreements should be in place.</i>				
3	Was a comprehensive literature search performed? <i>At least two electronic sources should be searched. The report must include years and databases used (e.g. Central, EMBASE, and MEDLINE). Key words and/or MESH terms must be stated and where feasible the search strategy should be provided. All searches should be supplemented by consulting current contents, reviews, textbooks, specialized registers, or experts in the particular field of study, and by reviewing the references in the studies found.</i>				
4	Was the status of publication (i.e. grey literature) used as an inclusion criterion? <i>*The authors should state that they searched for reports regardless of their publication type. The authors should state whether or not they excluded any reports (from the systematic review), based on their publication status, language, etc.</i>				
5	Was a list of studies (included and excluded) provided? <i>A list of included and excluded studies should be provided.</i>				
6	Were the characteristics of the included studies provided? <i>In an aggregated form such as a table, data from the original studies should be provided on the participants, interventions and outcomes. The ranges of characteristics in all the studies analyzed e.g. age, race, sex, relevant socioeconomic data, disease status, duration, severity, or other diseases should be reported.</i>				
7	Was the scientific quality of the included studies assessed and documented? <i>'A priori' methods of assessment should be provided (e.g., for effectiveness studies if the author(s) chose to include only randomized, double-blind, placebo controlled studies, or allocation concealment as inclusion criteria); for other types of studies alternative items will be relevant.</i>				
8	Was the scientific quality of the included studies used appropriately in formulating conclusions? <i>The results of the methodological rigor and scientific quality should be considered in the analysis and the conclusions of the review, and explicitly stated in formulating recommendations.</i>				
9	Were the methods used to combine the findings of studies appropriate? <i>*For the pooled results, a test should be done to ensure the studies were combinable, to assess their homogeneity (i.e. Chisquared test for homogeneity, I²). If heterogeneity exists a random effects model should be used and/or the clinical appropriateness of combining should be taken into consideration (i.e. is it sensible to combine?).</i>				
10	Was the likelihood of publication bias assessed? <i>An assessment of publication bias should include a combination of graphical aids (e.g., funnel plot, other available tests) and/or statistical tests (e.g., Egger regression test).</i>				
11	Was the conflict of interest stated? <i>Potential sources of support should be clearly acknowledged in both the systematic review and the included studies.</i>				

661 * The guidance for answering this question was adapted for the 2015 Dietary Guidelines Advisory Committee.

662 **Table C.4 Strength of Evidence terminology to support a conclusion statement when a**
 663 **question is answered with existing reports**

Strong	The conclusion statement is substantiated by a large, high quality, and/or consistent body of evidence that directly addresses the question. There is a high level of certainty that the conclusion is generalizable to the population of interest, and it is unlikely to change if new evidence emerges.
Moderate	The conclusion statement is substantiated by sufficient evidence, but the level of certainty is restricted by limitations in the evidence, such as the amount of evidence available, inconsistencies in findings, or methodological or generalizability concerns. If new evidence emerges, there could be modifications to the conclusion statement.
Limited	The conclusion statement is substantiated by insufficient evidence, and the level of certainty is seriously restricted by limitations in the evidence, such as the amount of evidence available, inconsistencies in findings, or methodological or generalizability concerns. If new evidence emerges, there could likely be modifications to the conclusion statement.
Grade not assignable	A conclusion statement cannot be drawn due to a lack of evidence, or the availability of evidence that has serious methodological concerns.

664

665

666

667

Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends

INTRODUCTION

Humans require a wide range of essential micronutrients and macronutrients for normal growth and development and to support healthy aging throughout the life cycle. Essential nutrients, including most vitamins, minerals, amino acids and fatty acids, water and fiber, must be obtained through foods and beverages because they cannot for the most part be endogenously synthesized, or are not endogenously synthesized in adequate amounts to meet recommended intakes. Understanding the extent to which the U.S. population and various age, sex, and racial/ethnic groups within the population achieve nutrient intake requirements through available food and beverage intake, including foods and beverages* that are enriched or fortified, is an important task of the DGAC. Notably, the DGAC considers that the primary source of nutrients should come from foods and beverages. Nutrient-dense forms of foods (those providing substantial amounts of vitamins, minerals and other nutrients and relatively few calories) are recommended to ensure optimal nutrient intake without exceeding calorie intake or reaching excess or potentially toxic levels of certain nutrients.

In the process of evaluating adequacy of nutrient intake of the U.S. population, the DGAC identified two levels of “Nutrients of Concern”. Shortfall nutrients are those that may be underconsumed relative to the Estimated Average Requirement (EAR) or Adequate Intake (AI). Overconsumed nutrients are those that are consumed in amounts above the Tolerable Upper Limit of Intake (UL)¹ or other nationally recognized standard.² Nutrients of Public Health Concern were those shortfall or overconsumed nutrients that also had evidence of under- or overconsumption through biochemical nutritional status indicators³ plus evidence that the nutrient inadequacy or nutrient excess is directly related to a specific health condition. This information is critical in determining where dietary intake improvements may be warranted that will benefit the health of the population. The 2015 DGAC recognizes that the 2010 DGAC specifically addressed whether or not multivitamins provided health benefits. The 2015 DGAC did not specifically address multivitamins, but recognizes that some dietary supplements may be recommended for some populations or life-cycle phases (pregnancy, for example).

In addition, many foods contain constituents that enable them to be produced, preserved, and thus widely available year round. Some of these ingredients, such as sodium, are used to make foods shelf stable and can help ensure food availability and food security for the population as a whole.⁴ Other ingredients, such as added sugars, are used as a food preservative and to enhance palatability. Despite the functional nature of both sodium and added sugars in the food supply, excess consumption of these

* Note: The DGAC considered foods and beverages in its review of intake data. Throughout this chapter, references to “foods” should be taken to mean “foods and beverages.”

36 dietary constituents poses potential health risks and was of particular concern to the DGAC. This
37 chapter reviews data on intakes of sodium, added sugars and saturated fat; other chapters consider
38 sodium, added sugars, and saturated fat from additional perspectives (see **Part D. Chapter 6: Cross-
39 Cutting Topics of Public Health Importance**) including health outcomes. The food supply also
40 contains ingredients that are both naturally occurring and also added to foods and beverages, such as
41 caffeine, that have generated considerable attention in recent years. This chapter examines intake levels
42 across age and sex groups of the U.S. population; **Part D. Chapter 5: Food Sustainability and Safety**
43 considers several safety aspects of caffeine consumption.

44
45 The U.S. food supply is complex. Tens of thousands of foods and food products are available in a
46 variety of forms. Some foods are whole foods that are often eaten alone without additions, such as fruit
47 and milk, while others, such as sandwiches and mixed dishes, are mixtures of multiple components
48 from more than one food group.

49
50 The DGAC recognizes the importance of understanding the totality of food and beverage intake at the
51 level of food groups and basic ingredients (e.g., fruit, vegetables, whole grains, refined grains, dairy,
52 protein foods) as well as at the level of foods as they are typically consumed, called food categories
53 (e.g., pizza, pasta dishes, burgers, sandwiches) and how these contribute to nutrient adequacy or
54 nutrient excess. To better understand current food intakes of the U.S. population, the Committee
55 reviewed data on several issues, such as which of these food groups (e.g., refined grains) and food
56 categories (e.g., sandwiches, beverages, snacks and sweets) contribute the most energy (calories),
57 sodium, and saturated fat.

58
59 Understanding the totality of food and beverage intake also involved acknowledging that individuals
60 purchase and procure food in a diverse array of locations, including large grocery stores, convenience
61 stores, schools, the workplace, quick-serve restaurants, and sit-down restaurants. The DGAC examined
62 the diet quality of the foods and meals at each major procurement point, as it is important to
63 understand not only where foods are purchased or obtained, but also the extent to which they
64 contribute to the overall nutritional adequacy and nutritional quality of the diet. This information may
65 be relevant to guidance for federal nutrition programs. The DGAC also considered the diet quality of
66 foods prepared and purchased at places such as supermarkets, but consumed at home. For example,
67 many supermarkets have salad bars and hot food bars, but these foods are then consumed at home.
68 However, on examination, it was determined that these types of data were not available. The DGAC
69 also examined eating behaviors, such as meal skipping, and identifying which nutrients and how much
70 energy are consumed at specific eating occasions and locations, because an understanding of these
71 behaviors can help inform public policy and population as well as individual guidance.

72
73 The DGAC considered the composition of dietary patterns that were found to be linked to health
74 outcomes in **Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes**.
75 Understanding the characteristics of diets characterized as “Healthy U.S.” or “Mediterranean-style”

76 dietary patterns and others patterns found to have health benefits, will provide specific, healthful food
77 and beverage-based guidance for the U.S. population. These patterns are defined using dietary
78 quality/adherence indices, [e.g., Healthy Eating Index (HEI)], based upon data-driven approaches (e.g.,
79 cluster or factor analysis), or may be self-identified patterns (e.g., vegetarian).

80

81 To address the issues described above, the DGAC presents the current status and trends in nutrient,
82 food, food group, and food category intakes, and describes major sources of energy, sodium, added
83 sugar, and saturated fat, and dietary pattern intake among representative samples of the U.S. population
84 from the National Health and Nutrition Examination Survey (NHANES) What we Eat in America
85 (WWEIA) dietary survey.⁵ We also describe eating behaviors, such as number of meals per day, diet
86 quality of foods, location of food purchase and consumption and diet quality of foods based on
87 location where the food was purchased or consumed.

88

89 Finally, we describe the prevalence of diet-related health outcomes in the U.S. population, including
90 obesity, diabetes, cardiovascular diseases, certain cancers, osteoporosis, congenital anomalies and
91 psychological health (including mental health), and neurological illness (such as Alzheimer's Disease).
92 The examination of diet-related health outcomes was more extensive than in earlier DGAC reports.
93 The high rates of the chronic conditions and the presence of other less common, but important diet-
94 related health problems, provided compelling reasons to study them in greater detail. These data
95 provide a backdrop for other chapters, particularly those which examine the strength of associations
96 between diet and health outcomes (*Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and*
97 *Health Outcomes*) and methods for improving disease risk outcomes and improving health at
98 individual (*Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes* and
99 *Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change* and population levels
100 (*Part D. Chapter 4: Food Environment and Settings*).

101

102 One of the overarching motivations for this broad examination of nutrient intake, food group and food
103 category intake, and food purchase location is to better understand the relationship of food intake (both
104 inadequacy and excess) and the food environment to nutrition-related health conditions. This
105 comprehensive evaluation of food and nutrient intakes by the U.S. population (and various subgroups)
106 along with the food and eating environment enables the consideration of factors on a broad scale that
107 may facilitate behavior change and adoption of healthy eating practices in the population at large.
108 Taken together, these dimensions of our analysis inform the remaining chapters in the report, which,
109 taken together, will provide the contextual and scientific foundation for the 2015 Dietary Guidelines
110 for Americans.

111

112

113 LIST OF QUESTIONS

114 **Nutrient Intake and Nutrients of Concern**

- 115 1. What are current consumption patterns of nutrients from foods and beverages by the U.S.
116 population?
- 117 2. Of the nutrients that are underconsumed or overconsumed, including over the Tolerable Upper
118 Limit of Intake (UL), which present a substantial public health concern?
- 119 a. What would be the effect on food choices and overall nutrient adequacy of limiting saturated
120 fatty acids to 6 percent of total calories by substituting mono- and polyunsaturated fatty acids?
- 121 3. Is there evidence of overconsumption of any micronutrients from consumption of fortified foods
122 and supplements?
- 123 4. What is the level of caffeine intake derived from foods and beverages on the basis of Institute of
124 Medicine (IOM) Dietary Reference Intakes age and sex categories in the U.S. population?
- 125 5. How well do updated USDA Food Patterns meet IOM Dietary Reference Intakes and 2010 Dietary
126 Guidelines recommendations? How do the recommended amounts of food groups compare to
127 current distributions of usual intakes for the U.S. population?
- 128 a. How well do the USDA Food Patterns meet the nutritional needs of children 2 to 5 years of age
129 and how do the recommended amounts compare to their current intakes? Given the relatively
130 small empty calorie limit for this age group, how much flexibility is possible in food choices?
- 131 6. Can vitamin D Estimated Average Requirements (EARs) and/or Recommended Dietary
132 Allowances (RDAs) be met with careful food choices following recommended amounts from each
133 food group in the USDA Food Patterns? How restricted would food choices be, and how much of
134 the vitamin D would need to come from fortified dairy and other food products?

135

136 **Food Groups—Current Intakes and Trends**

- 137 7. What are current consumption patterns of USDA Food Pattern food groups by the U.S. population?
- 138 a. What is the contribution of whole grain foods, fruits and vegetables, and other food groups to
139 (1) total fiber intake and (2) total nutrient intake in the USDA Food Patterns? What is the
140 contribution of fruit and vegetables to current nutrient intake (focus on nutrients of concern,
141 including fiber)?
- 142 b. What would be the impact on the adequacy of the patterns if (1) no dairy foods were consumed,
143 (2) if calcium was obtained from nondairy sources (including fortified foods), and (3) if the
144 proportions of milk and yogurt to cheese were modified? What is the relationship between
145 changes in types of beverages consumed (milk compared with sugar-sweetened beverages) and
146 diet quality?
- 147 8. What are the trends in USDA Food Pattern food group consumption by the U.S. population?

148

149 **Food Categories—Current Intakes and Sources of Energy, Nutrient, and Food Group**
150 **Intakes**

151 9. What are the current consumption patterns by food categories (i.e., foods as consumed) by the U.S.
152 population?

153 10. What are the top foods contributing to energy intake by the U.S. population?

154 11. What are the top foods contributing to sodium, saturated fat, and added sugars intake by the U.S.
155 population?

156 a. What is the current contribution of fruit products with added sugars to intake of added sugars?

157 b. What is the current contribution of vegetable products with added sodium to intake of sodium?

158 c. What is the current contribution of refined grains to intake of added sugars, saturated fat, some
159 forms of polyunsaturated fat, and sodium?

160 d. What are the sources of caffeine from foods and beverages on the basis of age and sex
161 subgroups?

162 12. What is the contribution of beverage types to energy intake by the U.S. population?

163

164 **Eating Behaviors—Current Status and Trends**

165 13. What are the current status and trends in the number of daily eating occasions and frequency of
166 meal skipping? How do diet quality and energy content vary based on eating occasion?

167 14. What are the current status and trends in the location of meal and snack consumption and sources
168 of food and beverages consumed at home and away from home? How do diet quality and energy
169 content vary based on the food and beverage source?

170

171 **Prevalence of Health Conditions and Trends**

172 15. What is the current prevalence of overweight/obesity and distribution of body weight, body mass
173 index (BMI) and abdominal obesity in the U.S. population and in specific age, sex, race/ethnicity
174 and income groups? What are the trends in prevalence?

175 16. What is the relative prevalence of metabolic and cardiovascular risk factors (i.e., blood pressure,
176 blood lipids, and diabetes) by BMI/waist circumference in the U.S. population and specific
177 population groups?

178 17. What are the current rates of nutrition-related health outcomes (i.e., incidence of and mortality
179 from cancer [breast, lung, colorectal and prostate] and prevalence of cardiovascular disease (CVD),

180 high blood pressure, diabetes, bone health, congenital anomalies, and neurological and
181 psychological illness) in the overall U.S. population?

182

183 **Dietary Patterns Composition**

184 18. What is the composition of dietary patterns with evidence of positive health outcomes (e.g.,
185 Mediterranean-style patterns, Dietary Approaches to Stop Hypertension (DASH)-style patterns,
186 patterns that closely align with the Healthy Eating Index, and vegetarian patterns) and of patterns
187 commonly consumed in the United States? What are the similarities (and differences) within and
188 among the dietary patterns with evidence of positive health outcomes and the commonly consumed
189 dietary patterns?

190 19. To what extent does the U.S. population consume a dietary pattern that is similar to those observed
191 to have positive health benefits (e.g., Mediterranean-style patterns, Dietary Approaches to Stop
192 Hypertension (DASH)-style patterns, patterns that closely align with the Healthy Eating Index, and
193 vegetarian patterns) overall and by age/sex and race/ethnic groups?

194 20. Using the Food Pattern Modeling process, can healthy eating patterns for vegetarians and for those
195 who want to follow a Mediterranean-style dietary pattern be developed? How do these patterns
196 differ from the USDA Food Patterns previously updated for use by the 2015 DGAC?

197

198 **METHODOLOGY**

199 To address questions on the current status and trends in food and nutrient intakes, the prevalence of
200 diet-related chronic diseases in the U.S. population, and the composition of healthful dietary patterns,
201 the DGAC relied on analysis of data from several sources and food pattern modeling analyses. Many
202 of the questions relied on analysis of data from What We Eat in America (WWEIA), the dietary
203 component of the National Health and Nutrition Examination Survey (NHANES), using either existing
204 data tables or new analyses conducted by the Data Analysis Team (DAT) upon request of the DGAC
205 (see *Part C. Methodology*, Data Analyses section, and *Appendix E-4: NHANES Data Used in DGAC*
206 *Data Analyses*). Existing data tables were used when available to answer questions about nutrient
207 intake, food group intake, and meal and snack consumption. In some cases, new analyses were
208 conducted by DAT agencies to provide additional information on food or nutrient intake, for example,
209 by specific population groups, such as pregnant women, or information on potential overconsumption
210 of nutrients when supplement intake is considered. New WWEIA/NHANES data analyses also were
211 used to answer questions about food category intakes, the energy content and nutrient density of foods
212 by point of purchase and location of consumption, and the food choices of self-identified vegetarians.

213

214 Data from the U.S. Centers for Disease Control and Prevention (CDC) NHANES data tables and from
215 the peer-reviewed literature, also were the source of information on prevalence of health conditions,
216 including body weight status, lipid profiles, high blood pressure, and diabetes. In addition, NHANES

217 data on biochemical indicators of diet and nutrition in the U.S. population were used to help determine
218 nutrients that may be of public health concern. To supplement data from NHANES, additional data
219 sources were drawn upon to answer questions on the prevalence of health conditions, including the
220 National Health Interview Survey, the National Cancer Institute’s Surveillance Epidemiology and End
221 Results (SEER) cancer registry statistics, SEARCH for Diabetes in Youth Study (SEARCH), and heart
222 disease and stroke statistics from the 2014 report of the American Heart Association.⁶
223

224 Some of the questions posed by the DGAC were best addressed by Food Pattern Modeling (see *Part C.*
225 *Methodology*, Special Analyses Using the USDA Food Patterns section). These included questions
226 about the nutrient adequacy of the USDA Food Patterns, modifications of the patterns for specific
227 population groups or to meet specific nutrient targets, and the nutrients provided by various food
228 groups in the Patterns. In some cases, questions could be answered with modeling analyses that had
229 been conducted for the 2005 or 2010 DGACs, and so the results of these analyses were brought
230 forward. The modeling process also was used to develop new USDA Food Patterns based on different
231 types of evidence: Healthy Vegetarian Patterns that take into account food choices of self-identified
232 vegetarians, and Healthy Mediterranean-style Patterns that take into account food group intakes from
233 studies using a Med-diet index to assess dietary patterns. The latter were compiled and summarized to
234 answer the questions addressed on dietary patterns composition. The food group content of dietary
235 patterns reviewed by the DGAC and found to have health benefits formed the basis for answering these
236 questions. WWEIA food group intakes and USDA Food Pattern recommendations were compared
237 with the food group intake data from the healthy dietary patterns as part of the answer for these
238 questions.
239

240 The DGAC took the strengths and limitations⁷ of data analyses into account in formulating conclusion
241 statements. The grading rubric used for questions answered using NEL systematic reviews do not
242 apply to questions answered using data analyses. Therefore, these conclusions were not graded.
243

244

245 **NUTRIENT INTAKE AND NUTRIENTS OF CONCERN**

246 An overarching premise of the DGAC is that that the *Dietary Guidelines for Americans* should provide
247 food-based guidance for obtaining the nutrients needed for optimal reproductive health, growth and
248 development, healthy aging, and well-being across the lifespan (ages 2 years and older). Specific
249 nutrient intake requirements are established for each sex and life-stage group by the Food and
250 Nutrition Board of the Institute of Medicine⁷ and as such, this DGAC report did not reevaluate IOM
251 recommendations or make independent specific nutrient recommendations. Rather, the DGAC
252 reviewed nutrient intake and biochemical measures of nutritional status and potential nutrient-related
253 health outcomes to identify “shortfall nutrients” and “overconsumed nutrients”, and then determined
254 whether these nutrients should be designated as “nutrients of public health concern.”
255

256 “Shortfall nutrients” are those that may be underconsumed either across the population or in specific
257 groups relative to IOM-based standards, such as the Estimated Average Requirement (EAR) or the
258 Adequate Intake (AI). The EAR is the best measure of population adequacy of nutrient intake as is it is
259 “the average daily intake level estimated to meet the requirement of half of the healthy individuals in a
260 particular life stage and gender group.”^{7 p.3} The EAR is used to estimate the prevalence of inadequate
261 intakes within a group. The AI is “a recommended average daily nutrient intake level based on
262 observed or experimentally determined approximations or estimates of nutrient intake by a group (or
263 groups) of apparently healthy people that are assumed to be adequate—used when an RDA cannot be
264 determined.”^{7 p.3} A high prevalence of inadequate intake either across the U.S. population or in specific
265 groups constitutes a shortfall nutrient.

266
267 Overconsumed nutrients are those that may be overconsumed either across the population or in specific
268 groups related to IOM-based standards such as the Tolerable Upper Limit of Intake (UL) or other
269 expert group standards. A high prevalence of excess intake either across the U.S. population or in
270 specific group constitutes an overconsumed nutrient.

271
272 “Nutrients of concern” are those nutrients that may pose a substantial public health concern and the
273 DGAC divided them into two categories—those of concern due to overconsumption and those of
274 concern due to underconsumption. To be identified as a nutrient of concern, the DGAC used the
275 totality of evidence, evaluating data on nutrient intake and corroborating it with biochemical markers
276 of nutritional status, where available, and evidence for associations with health outcomes to establish
277 nutrients of concern.

278
279 Designation as a nutrient of concern for either under- or overconsumption is intended to communicate
280 some level of risk for which the U.S. population may need to modify eating habits. Dietary guidance
281 can then be formulated to assist individuals in increasing or decreasing nutrients that are under- or
282 overconsumed.

283

284 **Question1: What are current consumption patterns of nutrients from foods and**
285 **beverages by the U.S. population?**

286 **Source of evidence:** Data analysis

287

288 **Conclusion**

289 Nutrient intake data from a representative sample of the U.S. population ages 2 years and older
290 indicate that: vitamin A, vitamin D, vitamin E, folate, vitamin C, calcium, and magnesium are
291 underconsumed relative to the EAR. Iron is under-consumed by adolescent and premenopausal
292 females, including women who are pregnant. Potassium and fiber are underconsumed relative to the
293 AI. Sodium and saturated fat are overconsumed relative to the UL or other standards for maximal
294 intake.

295

296 **Implications**

297 A dietary pattern emphasizing a variety of nutrient-dense foods will help shift individual and
298 population consumption toward recommended intake levels for nutrients of public health concern.

299

300 The U.S. population should increase consumption of foods rich in vitamin A, vitamin D, vitamin E,
301 folate, vitamin C, calcium, and magnesium. Adolescent and premenopausal females should increase
302 consumption of foods rich in iron. Heme iron from lean meats is highly bioavailable, hence, an
303 excellent source.⁸ A diet emphasizing a variety of nutrient-dense foods will help shift consumption
304 toward the recommended intake levels of these shortfall nutrients. The U.S. population should increase
305 consumption of foods rich in potassium and fiber. A diet emphasizing a variety of nutrient-dense foods
306 will help ensure optimal intake of these shortfall nutrients. In particular, fruit, vegetables and whole
307 grains are excellent sources of vitamin A, C, folate, fiber, magnesium and potassium. The U.S.
308 population should make concerted and focused efforts to decrease consumption of sodium and
309 saturated fat.

310

311 The USDA Food Patterns provide guidance for consumption of a nutrient-dense, energy-balanced diet.
312 Implementation of eating a healthy diet that is energy balanced while providing sufficient intake of
313 shortfall nutrients without exceeding intake of overconsumed nutrients can be achieved through a
314 variety of successful behavioral approaches as described in *Part D. Chapter 3: Individual Diet and*
315 *Physical Activity Behavior Change*. Environmental and policy approaches are also important in
316 helping the U.S. population achieve a healthy diet (see also *Part D. Chapter 4: Food Environment*
317 *and Settings*). Federal nutrition assistance programs are a key aspect of providing critical nutrients for
318 growth, development and long-term health for children, those with limited income and older
319 Americans.

320

321 **Review of the Evidence**

322 To determine nutritional adequacy, the DGAC used 2007-2010 NHANES/WWEIA data to examine
323 the intake distributions for 11 vitamins (vitamin A, vitamin B₆, vitamin B₁₂, vitamin C, vitamin D,
324 vitamin E, vitamin K, folate, thiamin, niacin, and riboflavin), nine minerals (calcium, copper, iron,
325 magnesium, phosphorous, potassium, selenium, sodium, and zinc), energy, macronutrients (total fat,
326 saturated fat, polyunsaturated fat [including 18:2 and 18:3], protein, carbohydrate), and other
327 compounds or components (fiber, carotenoids [alpha-carotene, beta-carotene, lycopene, lutein +
328 zeaxanthin], caffeine, cholesterol, and choline) (see *Appendix E-2.1: Usual intake distributions, 2007-*
329 *2010, by age/sex groups*). The DGAC compared the intake estimates across the population age
330 distribution to the Dietary Reference Intakes. The committee used data from foods and beverages as
331 well as foods and beverages plus dietary supplements when supplement data were available. For
332 nutrients with an EAR, the DGAC considered shortfall nutrients to be those where a substantial
333 proportion of either the total population or specific age and sex subgroups had intake estimates below

334 the EAR. Although multiple approaches can be used to estimate the prevalence of nutrient inadequacy
 335 in a population, the DGAC used the EAR cut point method.⁷ Figure D1.1 shows the percent of the U.S.
 336 population with usual intakes below the EAR. From Figure D1.1, the DGAC determined that vitamin
 337 D, vitamin E, magnesium, calcium, vitamin A and vitamin C were shortfall nutrients and that there
 338 may be a high prevalence of inadequate dietary intake of these nutrients.

339

340 Of the nutrients with an AI (vitamin K, choline, dietary fiber, and potassium), the DGAC determined
 341 that a low proportion of the population had fiber and potassium intakes above the AI and so potassium
 342 and fiber were therefore considered to be underconsumed (Figure D1.2).

343

344 Sodium and saturated fat were examined as potentially overconsumed nutrients in relation to the UL
 345 (for sodium), and the maximum level from the 2010 Dietary Guidelines of less than 10 percent of
 346 calories from saturated fat (for saturated fat). From 63 percent to 91 percent of females and 81 percent
 347 to 97 percent of males consumed more than the UL for sodium (Figure D1.3). From 67 percent to 92
 348 percent of females and from 57 percent to 84 percent of males consumed more than 10 percent of
 349 calories from saturated fat (Figure D1.4). Therefore, sodium and saturated fat were both determined to
 350 be overconsumed by the U.S. population (see *Appendix E-2.1: Usual intake distributions, 2007-2010,*
 351 *by age/sex groups* and *Appendix E-2.2: Usual intake distributions as a percent of energy for fatty*
 352 *acids and macronutrients, 2007-2010, by age/sex groups*).

353

354 The DGAC examined population intakes of specific nutrients by age, sex, race/ethnicity, pregnancy
 355 status, and acculturation status.

356

357 ***Age and Sex***

358 In addition to the age groups shown in Figures D1.1 and D1.2, the DGAC was interested in
 359 understanding the intake of shortfall nutrients in older adults (71 to 79 years and 80 years and older).
 360 Calcium intake from foods and beverages did not meet the EAR for older persons, where 71 percent of
 361 males and 81 percent of females ages 71 years and older had intakes below the EAR. For these
 362 analyses calcium from dietary supplements was also considered. When total intake of foods + beverage
 363 + dietary supplements containing calcium was considered, then the proportion of the older adults
 364 below the EAR improved to 55 percent for men and 49 percent for women over the age of 71 years.
 365 For vitamin D intakes from food and beverages only, about 93 percent of older males and more than 97
 366 percent of older females had intakes below the EAR. Similar to the findings for calcium, intakes
 367 improved when considering total intake from foods and beverages plus dietary supplements. The
 368 proportions of older adult below the EAR dropped to 52 percent for both males and females older than
 369 71 years.

370

371 Fiber was a shortfall nutrient for older adults, where only 4 percent of men and 13 percent of women
 372 had a dietary intake of fiber above the AI. Potassium also was a shortfall nutrient for both older males
 373 and females, where less than 3 percent of both groups had intakes above the AI. Use of dietary

374 supplements containing potassium did not change the proportion of the older adults with intakes above
375 the AI.

376

377 Protein was not identified as a shortfall nutrient for the overall older adult population but it should be
378 noted that 6 percent of men older than 80 years and 11 percent of women older than 80 years old had
379 protein intakes that were below the protein EAR (g/kg/body weight).

380

381 The sample size for the older participants in WWEIA 2007-2010 is small compared to other age
382 groupings in the survey sample and despite the excellent population weights used in the WWEIA
383 dataset, the estimates should be viewed with caution because of the limited sample (see *Appendix E-*
384 *2.3 Usual nutrient intakes for individuals age 71 years and older*).

385

386 ***Race/Ethnicity***

387 The DGAC examined the shortfall nutrients by race/ethnicity using the following groups: non-
388 Hispanic white, non-Hispanic Black, Mexican-American, and all Hispanic combined (other race/ethnic
389 subgroups not available). For certain shortfall nutrients, non-Hispanic whites have the highest intakes.
390 These include vitamin A, vitamin E, magnesium, folate, iron, potassium, vitamin D, and calcium.
391 Mexican-Americans have the highest intakes of fiber, while all Hispanics combined have the highest
392 intakes of vitamin C. Non-Hispanic Blacks have the lowest intake for most of the shortfall nutrients
393 (Table D1.1). We note that evaluation of intakes relative to the EAR or AI are the most appropriate for
394 assessment of populations, instead of the mean intakes, but for the race/ethnicity groups, only the mean
395 data are available.

396

397 ***Pregnancy***

398 Many of the shortfall nutrients in the general population also were shortfall nutrients among women
399 who are pregnant. Among this group, 26 percent were below the EAR for vitamin A intake and 30
400 percent had vitamin C intakes below the EAR. For vitamin D, 90 percent had intakes below the EAR
401 and for vitamin E, 94 percent had intakes below the EAR. Calcium intake was also low, where 24
402 percent had intakes below the EAR, and for folate, 29 percent had intakes below the EAR. Notably, 96
403 percent of women who were pregnant had iron intakes below the EAR (Table D1.2 and *Appendix E-*
404 *2.4: Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in the U.S. ages*
405 *19-50 years*).

406

407 Fiber was a shortfall nutrient for women who were pregnant, as only 8 percent had fiber intakes above
408 the AI. For potassium only 3 percent had intakes above the AI (Table D1.2).

409

410 It is important to note that the sample size for women who were pregnant in WWEIA 2007-2010 is
411 very small (n=133 respondents), so the estimates should be interpreted with caution and the
412 generalizability of the data to all women in the United States who were pregnant is limited.

413

414 Acculturation

415 The U.S. population is highly diverse in terms of race, ethnicity, and cultural origin. Many people
416 immigrate to the United States from all over the world and each comes with distinct dietary habits and
417 cultural beliefs about food and food patterns.⁹ Acculturation is defined as the process by which
418 immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a new culture. Acculturation
419 is the gradual exchange between immigrants' original attitudes and behavior and those of the host
420 culture.^{10, 11} The DGAC appreciates that many immigrants have difficulties purchasing and preparing
421 foods familiar to them either because the ingredients are not available or the ingredients may be too
422 expensive. A large and growing body of research suggests that the extent of an individual or family's
423 acculturation status may be a predictor of dietary intake and that together, diet and acculturation status
424 may influence health status or disease risk.^{9, 10, 12, 13} For this reason, the DGAC felt it was important to
425 examine dietary intake by acculturation status, particularly for shortfall nutrients and nutrients of
426 concern. Additional information on acculturation and diet appears in Part D. Chapter 3: Individual Diet
427 and Physical Activity Behavior Change.

428
429 NHANES collects data on some of the variables that can be used to create an acculturation variable,
430 including whether respondents were born outside the United States in a Spanish-speaking country or
431 born outside the United States in a non-Spanish speaking country, their race/ethnicity, and number of
432 years they have resided in the United States.¹⁴ Upon reviewing the data, however, the DGAC found
433 that the sample size was far too small to create meaningful variables to indicate "low acculturation
434 status" or "high acculturation status." The DGAC views this lack of ability to analyze the WWEIA
435 data by acculturation status as a limitation of the available data. It is a very important area that needs
436 further research, particularly when informing nutrition programs for new residents of the United States.

437 Food Insecurity Status

438
439 Readers are referred to *Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*
440 and *Part D. Chapter 5: Food Sustainability and Safety* for more detailed discussions of food
441 insecurity and food security issues. For this section of the report, the DGAC determined that it was
442 important to evaluate nutrient intake, particularly for the shortfall nutrients by income status, which
443 can be a marker of food insecurity. For these data analyses, we used the standard cutpoints of less than
444 131 percent of the poverty index, 131 to 185 percent of the poverty index and more than 185 percent of
445 the poverty index and examined calcium, potassium, fiber and vitamin D (Table D1.3). In general,
446 respondents (all ages 2 years and older) from households with higher income (more than 185 percent
447 of the poverty index) had higher intakes of calcium, potassium, fiber, and vitamin D. Notably, in some
448 of the very young age groups (2 to 5 years), intakes of potassium, fiber, and vitamin D were
449 comparable across income groups, while calcium was highest in those coming from households at the
450 131 to 185 percent of the poverty index ratio. It may be that many of the households of lower income
451 with small children are receiving important benefits from federal nutrition assistance programs, which
452 could be helping to generate comparability in the intake of shortfall nutrients across the income groups.

453

454 ***For additional details on this body of evidence, visit:***

- 455 • Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups
- 456 • Appendix E-2.2: Usual intake distributions as a percent of energy for fatty acids and
457 macronutrients, 2007-2010, by age/sex groups
- 458 • Appendix E-2.3: Usual intakes for Individuals age 71 and older
- 459 • Appendix E-2.4: Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in
460 the U.S. ages 19-50 years
- 461 • Mean intake of nutrients, 2003-2004, 2005-2006, 2007-2008, and 2009-2010, by race/ethnicity and
462 by percent of the poverty threshold. Available from:
463 <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>.
- 464 • Usual intake of selected nutrients, 2001-2002, 2003-2006, or 2005-2006, by age/sex groups.
465 Available from: <http://seprl.ars.usda.gov/Services/docs.htm?docid=22659>.

466

467 **Question 2: Of the nutrients that are underconsumed or overconsumed, including**
468 **over the Tolerable Upper Limit of Intake (UL), which present a substantial public health**
469 **concern?**

470 **Source of evidence:** Data analysis

471

472 **Conclusion**

473 Nutrient intake data, together with nutritional biomarker and health outcomes data indicate that vitamin
474 D, calcium, potassium, and fiber are underconsumed and may pose a public health concern. Iron also is
475 a nutrient of public health concern for adolescent and premenopausal females.

476

477 Nutrient intake data, together with nutritional biomarker and health outcomes data indicate that sodium
478 and saturated fat are overconsumed and may pose a public health concern.

479

480 **Implications**

481 The DGAC recommends that strategies be developed and implemented at both the individual and the
482 population level to improve intake of nutrients of public health concern.

483

484 **Review of the Evidence**

485 These conclusions were reached using a 3-pronged approach, including analysis of data from What We
486 Eat in America, NHANES dietary survey (2007-2010) (see *Appendix E-2.1: Usual intake*
487 *distributions, 2007-2010, by age/sex groups*), the Second National Report on Biochemical Indices of
488 Diet and Nutrition in the U.S. Population, Centers for Disease Control and Prevention, 2012,³ and data
489 on the prevalence of health conditions, from the CDC. The DGAC used the totality of evidence from
490 these sources.

491

492 ***Nutrients of Concern for Underconsumption***

493 **Vitamin D.** Vitamin D is unequivocally essential for skeletal health.¹⁵ The 2010 IOM report on
 494 Dietary Reference Intakes for calcium and vitamin D¹⁵ established new DRIs for vitamin D based on
 495 established and consistent evidence for vitamin D's role in skeletal health. Numerous other functions
 496 exist for vitamin D, including its role as a transcription factor for more than 200 genes, roles in
 497 apoptosis and cellular proliferation, and a growing body of evidence supporting vitamin D's role in
 498 preventing cancer, cardiovascular disease, and other chronic diseases.¹⁶⁻²⁵

499

500 The IOM's rationale for setting the DRI was limited to vitamin D's role in skeletal health, as the
 501 evidence for the other diseases was not sufficiently mature at the time of the committee's evidence
 502 review. Therefore, any interpretations for vitamin D intake and its classification as a shortfall nutrient
 503 and a nutrient of public health concern are restricted to this role in skeletal health. Given the high
 504 prevalence of osteoporosis and low bone density, particularly in the older women (see Question 17, on
 505 health conditions, below) and due to vitamin D's critical role in bone health, the Committee
 506 determined that vitamin D should be classified as an underconsumed nutrient of public health concern.

507

508 Vitamin D can be obtained from the diet by consuming fluid milk and some milk products (e.g., some
 509 yogurts), fortified juices, finfish, fortified breakfast cereals and some fortified grain products as well as
 510 dietary supplements (Table D1.4 and ***Appendix E-3.3: Meeting Vitamin D Recommended Intakes in***
 511 ***USDA Food Patterns***). Vitamin D also is synthesized endogenously through cutaneous exposure to
 512 ultraviolet-B sunlight. The primary biomarker to assess vitamin D status is serum/plasma 25(OH)D
 513 concentrations. This biomarker represents dietary intake plus endogenous synthesis.

514

515 Dietary intake of vitamin D in the United States is low and well below the EAR values (Figure D1.1)
 516 for all age and sex groups. In addition, independent evidence of nutrient shortfall comes from data
 517 demonstrating low serum/plasma 25-hydroxyvitamin D concentrations from the CDC biomarker data,
 518 particularly for young adults (ages 20 to 39 years), middle-aged adults (ages 40 to 59 years), non-
 519 Hispanic Blacks and Mexican-Americans (Table D1.5). The correlation of dietary intake with the
 520 serum measures of 25-hydroxyvitamin D) is modest. In addition several factors predict serum
 521 concentrations of nutrients in addition to dietary intake.¹⁹ The DGAC and other expert panels,
 522 including the IOM, acknowledge that while numerous variables, including sun exposure and
 523 endogenous synthesis, are strong predictors of serum vitamin D status, dietary intake of vitamin D is a
 524 critical contributor to vitamin D status.^{26,27} Further, while there is some degree of unexplained
 525 variation in serum/plasma 25-hydroxyvitamin D concentrations, the biomarker is still important for
 526 evaluating vitamin D inadequacy. Various statistical approaches have been used to evaluate and
 527 confirm population inadequacy using the biomarker data.²⁸ Of note, the CDC biomarker data reviewed
 528 by the DGAC should be interpreted knowing that the NHANES Mobile Examination Clinics do not
 529 sample residents of northern climates in winter months due to variable sunshine exposure and the
 530 possibility that high levels of sunshine exposure may be overrepresented in NHANES. In other words,

531 higher values in the dataset may be over-represented due to the summer blood draws, when 25-OHD
 532 tends to be higher from sun exposure and deficiencies may be under represented. ^{15p.471-473}
 533

534 The DGAC’s decision to classify vitamin D as a nutrient of concern is similar to the conclusion
 535 reached by the U.S. Food and Drug Administration (FDA), which designated vitamin D as a nutrient of
 536 “public health significance” in its recent review of evidence in publishing a Proposed Rule on the
 537 Nutrition Facts label.²⁹ In addition, multiple national and international groups, including the American
 538 Academy of Pediatrics (AAP),³⁰ the Endocrine Society³¹ and the National Osteoporosis Foundation ³²
 539 have recommended that strategies to achieve the RDA or higher levels of vitamin D intake could
 540 include consumption of fortified foods, broadening the range of dairy products that are fortified, and
 541 consideration, in some cases, of the use of a vitamin D supplement or a multivitamin including vitamin
 542 D. Such a use is especially appropriate where sunshine exposure is more limited due to climate or
 543 sunblock use.
 544

545 **Calcium.** Calcium plays a major role in skeletal health and also is essential for proper functioning of
 546 the circulatory system, nerve transmission, muscle contractility, cell signaling pathways, and vascular
 547 integrity.¹⁵ Dietary calcium is obtained from fluid milk and milk products, fortified juices, and some
 548 plant foods, including soy and soy products and vegetables (see Table D1.6 and *Appendix E-3.2: Food*
 549 *Group Contributions*). However, the bioavailability of calcium from plant foods is lower than from
 550 animal foods, such as dairy.
 551

552 The DGAC reviewed the dietary intake data from WWEIA. Intakes of calcium were often far below
 553 the EAR, especially among adolescent girls and adults (Figure D1.1). Even though a reliable
 554 biomarker for calcium does not exist, because of its strong link to health outcomes and the risks
 555 associated with osteoporosis (see Question 17 on health conditions, below), the DGAC designated
 556 calcium as a nutrient of public health concern for underconsumption. In addition, the DGAC also notes
 557 that calcium is an underconsumed nutrient of public health concern among pregnant women. This
 558 conclusion concurs with the FDA’s review that designated calcium as a nutrient of “public health
 559 significance” in its recent review of evidence in publishing a Proposed Rule on the Nutrition Facts
 560 label.²⁹
 561

562 Strategies to improve calcium intake include increased dairy or fortified products that are important
 563 sources of calcium. Concern about the safety of calcium supplements and a relative lack of data about
 564 the health benefits of such supplements limit recommendations to use supplementation as a strategy to
 565 meet the RDA for calcium, compared to using fortified foods.
 566

567 The subgroups of particular concern with regard to intake are preadolescent and adolescent females,
 568 pregnant females, and middle aged and older females (see Question 1, above).
 569

570 **Potassium.** Potassium is the major intracellular cation and it plays critical roles in muscle function,
571 cardiac function, and regulation of blood pressure. Potassium adequacy is also critical for health, as
572 deficiency adversely affects numerous organ systems including the musculoskeletal, renal, and
573 cardiovascular systems. The primary biomarker to assess potassium intake is urinary potassium, and
574 these data are not available in the CDC biomarker dataset. The DGAC designated potassium as a
575 nutrient of public health concern due to its general under consumption relative to the AI across the
576 U.S. population and its association with hypertension and cardiovascular diseases, two common
577 adverse diet-related health outcomes in the United States (see Question 17 on health conditions,
578 below). This conclusion concurs with the FDA’s review that designated potassium as a nutrient of
579 “public health significance” in its recent review of evidence in publishing a Proposed Rule on the
580 Nutrition Facts label.²⁹ Even though underconsumption was evident across the population (see
581 Question 1, above), there is a particular concern for middle-aged and older adults, who are at increased
582 risk for cardiovascular diseases (see Question 17). Fruits, vegetables, and legumes are all important
583 sources of potassium (Table D1.7).

584
585 **Fiber.** Dietary fibers are non-digestible carbohydrates, primarily from plant foods, such as whole
586 grains, legumes, fruits and vegetables (Table D1.8). The most important and well-recognized role for
587 fiber is in colonic health and maintenance of proper laxation, but a growing body of evidence also
588 suggests that fiber may play a role in preventing coronary heart disease, colorectal and other cancers,
589 type 2 diabetes, and obesity.³³ The AI for fiber is based on an intake level associated with the greatest
590 reduction in the risk of coronary heart disease. There are no available biomarkers for fiber intake, so
591 the designation as a nutrient of public health concern is based on the very low dietary intakes across all
592 sectors of the U.S. population and its important contribution to health. Because the average intake
593 levels of dietary fiber are half the recommended levels, achieving the recommendation requires
594 selecting high-fiber cereals and whole grains and -meeting current recommendations for fruits and
595 vegetables.

596
597 **Iron.** Iron is an essential mineral whose primary function is to transport oxygen in the blood.
598 Inadequate iron status in the form of iron deficiency anemia leads to poor growth and development and
599 the potential for cognitive deficits in children. Excellent sources of heme iron include red meats,
600 enriched cereal grains, and fortified breakfast cereals (Table D1.9). Dietary intake estimates, together
601 with the CDC nutritional biomarker data indicate that iron is a nutrient of concern for children,
602 premenopausal females, and during pregnancy. Among women who are pregnant, 96 percent are below
603 the EAR for iron intake. Serum ferritin is the biochemical marker used by NHANES and the CDC to
604 evaluate iron status in the U.S. population. These data show that children and women of childbearing
605 age are at risk of iron deficiency anemia. Risk of iron deficiency anemia also is higher among
606 Mexican-American and non-Hispanic Black women than among non-Hispanic white women.³ Taken
607 together, the DGAC concluded that iron was an underconsumed nutrient of public health concern for
608 adolescent and premenopausal women and women who are pregnant. This conclusion concurs with the

609 FDA’s designated iron as a nutrient of “public health significance” in its recent review of evidence in
610 publishing a Proposed Rule on the Nutrition Facts label.²⁹

611

612 ***Nutrients of concern for overconsumption***

613 **Sodium.** Sodium is the major cation in extracellular fluid that maintains extracellular fluid volume and
614 plasma volume. It also functions in membrane potential activation and active transport of molecules
615 across cell membranes. In excess, sodium is associated with several adverse health events, particularly
616 hypertension.³⁴ The DGAC treated sodium as a cross-cutting topic for dietary intake and health
617 outcomes, and a sodium working group was convened. Details on sodium, including dietary sources
618 and health outcomes-related data are found in ***Part D. Chapter 6: Cross-Cutting Topics of Public***
619 ***Health Importance***). Current sodium intakes of the U.S. population far exceed the UL for all age and
620 sex groups (Figure D1.3). Due to the critical link of sodium intake to health and that intake exceed
621 recommendations, sodium was designated as a nutrient of public health concern for overconsumption
622 across the entire U.S. population.

623

624 **Saturated fat.** The DGAC used the 2013 American Heart Association/American College of
625 Cardiology (AHA/ACC) report on lifestyle management to reduce CVD risk² for its evaluation of
626 saturated fat intake. The DGAC concurred with the AHA/ACC report that saturated fat intake exceeds
627 current recommendations in the United States and that lower levels of consumption would further
628 reduce the population level risk of CVD. The DGAC also convened a working group on saturated fat
629 (see ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance*** for details). In addition,
630 the DGAC conducted food pattern modeling to demonstrate the dietary changes that would be
631 necessary to have diets with various levels of saturated fat as a percent of total energy (see USDA
632 Food Patterns Modeling Report in ***Appendix E-3.5: Reducing Saturated Fats in the USDA Food***
633 ***Patterns***). It is important to note that the median intake of saturated fat in the United States was 11.1
634 percent of total energy for all age groups in the 2007-2010 WWEIA data. However, a large majority
635 (71 percent) of the total population consumed more than 10 percent of calories from saturated fat, with
636 a range by age group from 57 percent to 92 percent (Figure D1.4). Further, 65 percent to 69 percent of
637 the age groups at highest risk of CVD (males and females older than age 50 years) had intakes more
638 than 10 percent of total calories were from saturated fat, the DGAC concluded that the U.S. population
639 should continue to monitor saturated fat intake. Saturated fat is still a nutrient of concern for
640 overconsumption, particularly for those older than the age of 50 years.

641

642 **Cholesterol.** Previously, the Dietary Guidelines for Americans recommended that cholesterol intake
643 be limited to no more than 300 mg/day. The 2015 DGAC will not bring forward this recommendation
644 because available evidence shows no appreciable relationship between consumption of dietary
645 cholesterol and serum cholesterol, consistent with the conclusions of the AHA/ACC report.^{2, 35}
646 Cholesterol is not a nutrient of concern for overconsumption.

647

648 ***For additional details on this body of evidence, visit:***

- 649 • CDC report, Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S.
650 Population 2012. Available from:
651 http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.
- 652 • Food Labeling: Revision of the Nutrition and Supplement Facts Labels; Proposed Rule. Available
653 from: <http://www.gpo.gov/fdsys/pkg/FR-2014-03-03/pdf/2014-04387.pdf>.
- 654 • Appendix E-3.2: Food Group Contributions to Nutrients in the USDA Food Patterns and Current
655 Nutrient Intakes
- 656 • Appendix E-3.3: Meeting Vitamin D Recommended Intakes in USDA Food Patterns
- 657 • Appendix E-3.5: Reducing Saturated Fats in the USDA Food Patterns

658
659

660 **Question 3: Is there evidence of overconsumption of any micronutrients from**
661 **consumption of fortified foods and supplements?**

662 **Source of evidence:** Data analysis

663 **Conclusion**

664 Dietary patterns among Americans, including typical use of fortified foods, rarely lead to
665 overconsumption of folate, calcium, iron, or vitamin D. However, each of these nutrients, as well as
666 other nutrients, are overconsumed in some supplement users, especially those taking high-dose
667 supplements.

668

669 **Implications**

670 The public may safely use dietary supplements containing RDA level of nutrients, so long as total
671 intake from diet plus supplements does not exceed the UL. Use of products with high doses of
672 nutrients, such that total intake exceeds the UL, should be discussed with a Registered Dietitian or
673 other qualified health care provider.

674

675 Supplement users should seek guidance about factors such as whether the amount of nutrients in
676 supplements exceeds the UL for those nutrients. Monitoring of dietary patterns in supplement users
677 should continue to be done, with attention paid to the highest risk groups, such as children and women
678 who are pregnant.

679

680 **Review of the Evidence**

681 These conclusions were based on analysis of usual intake data for selected nutrients from foods and
682 supplements from WWEIA, NHANES dietary survey (2007-2010) (see *Appendix E-2.5: Usual intake
683 distributions for supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by
684 age/sex groups* and *Appendix E-2.6: Usual intake distributions for non-supplement users for folate,*

685 *folic acid, vitamin D, calcium, and iron, 2007-2010, by age/sex groups*). Nutrients were selected if
686 the DGAC had identified them as a shortfall nutrient and if supplemental intake data were available in
687 WWEIA (Figure D1.5). When possible the total nutrient exposure was considered (food +
688 supplements). The overconsumed nutrients (saturated fat and sodium) are not contained in most dietary
689 supplements so that overconsumed nutrients were not considered for this question.

690

691 **Folate.** The use of supplemental folic acid exceeds the established UL in a small proportion of
692 children, especially those younger than age 9 years. However, this UL is not based on clinical toxicity
693 data in this population and exceeding the UL is primarily associated with supplement use.³⁶ The risk
694 associated with usual folate intakes among children in the United States is considered low, but caution
695 should be used in advising supplements for children younger than age 9 years.

696

697 **Calcium.** Dietary calcium intake greater than 2000 mg/day (UL) are seen in up to about 20 percent of
698 females, and 15 percent of adult males older than age 50 years. These high intakes are driven primarily
699 by a historical perspective that very high calcium supplement usage may decrease the risk of
700 osteoporosis. Concern exists about the safety of such high intakes and the possible association with
701 CVD risk and little, if any, current evidence supports intakes of calcium above the UL for the purpose
702 of decreasing osteoporosis.¹⁵ Of note, the World Health Organization recommends high dose calcium
703 supplementation (1.5-2 g/day) to prevent hypertensive disorders of pregnancy.³⁷ This recommendation
704 is not widely followed among low-risk women in the United States. However, use of calcium
705 supplements does not appear to pose a health risk related to overconsumption of calcium.³⁷

706

707 **Iron.** In adults of all ages, a small proportion of iron supplement users have intakes above the UL.
708 Concerns related both to cardiovascular health and oxidant damage exist, but are not well-defined. Iron
709 supplementation is very common during early childhood and pregnancy, but is unlikely to pose a
710 health risk.⁸

711

712 **Vitamin D.** Overconsumption of vitamin D occurs when individuals take high dose supplements,
713 usually over a long period of time.¹⁵ The UL of 4000 IU/day is commonly exceeded by individuals
714 with or without the guidance of a physician.¹⁵ In general, it is unlikely that most supplement users, who
715 limit themselves to 10,000 IU/day or less, will have any evidence of toxicity, but a greater risk may
716 exist among some groups, including small children. Those who take high dose supplements often have
717 their serum/plasma 25-hydroxyvitamin D concentrations monitored and this can be helpful although no
718 clearly toxic level of 25-hydroxyvitamin D in the blood is known. Overall, the population risk of
719 overconsumption of vitamin D leading to toxic effects, including hypercalcemia or other clinical
720 symptoms, is uncommon.³⁸

721

722 ***For additional details on this body of evidence, visit:***

- 723 • Appendix E-2.5: Usual intake distributions for supplement users for folate, folic acid, vitamin D,
724 calcium, and iron, 2007-2010, by age/sex groups

- 725 • Appendix E-2.6: Usual intake distributions for non-supplement users for folate, folic acid, vitamin
726 D, calcium, and iron, 2007-2010, by age/sex groups

727

728 **Question 4: What is the level of caffeine intake derived from foods and beverages on**
729 **the basis of Institute of Medicine (IOM) Dietary Reference Intakes age and sex**
730 **categories in the U.S. population?**

731 **Source of evidence:** Data analysis

732

733 **Conclusion**

734 In general, intakes of caffeine do not exceed what is currently considered safe levels in any age group.
735 Some young adults may have moderately high intakes. There is less certainty about the safe level of
736 intake in children and adolescents. However, routine consumption patterns do not suggest that
737 excessive intakes are common in these groups.

738

739 **Implications**

740 The public may safely consume caffeine-containing beverages, such as coffee and tea. However,
741 children, adolescents, and women who are pregnant or considering pregnancy should not consume
742 very high levels of caffeine from beverages or supplements (e.g., energy shots, fortified foods).

743

744 Monitoring of caffeine intake should be continued with special attention to high-risk groups, including
745 children and women who are pregnant. Families should monitor caffeine intake in children, and high-
746 dose caffeine supplementations should not be used.

747

748 For additional details on caffeine safety please see *Part D. Chapter 5: Food Sustainability and Safety*.

749

750 **Review of the Evidence**

751 These conclusions were reached based on analysis of usual intake data from the WWEIA, NHANES
752 dietary survey (2007-2010). Data on intakes of caffeine show that intakes in adults (Figure D1.6) peak
753 at ages 31 to 70 years, and that younger adults (ages 19 to 30 years), older adults (71 years and older),
754 have lower intakes. Relatively few individuals (less than 10 percent) have intakes above 400 mg/day
755 (see *Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups*), which is a level set
756 as a moderate intake by some groups, including Health Canada.

757

758 In children, caffeine intakes increase with age (Figure D1.7) with median intakes remaining below 100
759 mg/day in adolescents (14 to 18 years). Recommended intakes from Health Canada of no more than
760 2.5 mg/kg/day, or about 85 mg/day total in children ages 10 to 12 years³⁹ are not exceeded by most
761 children and adolescents although recent data indicates that as many as 10 percent of children and
762 adolescents ages 12 to 19 years exceed this intake level.⁴⁰ These data demonstrate that caregivers

763 should monitor caffeine intake in children and exercise caution with respect to time-dependent changes
764 in caffeine intake.

765

766 *For additional details on this body of evidence, visit:*

- 767 • Appendix E-2.1: Usual intake distributions, 2007-2010 by age/sex groups

768

769 **Question 5: How well do updated USDA Food Patterns[♦] meet IOM Dietary Reference**
770 **Intakes and 2010 Dietary Guidelines recommendations? How do the recommended**
771 **amounts of food groups compare to current distributions of usual intakes for the U.S.**
772 **population?**

773 **Source of evidence:** Food Pattern Modeling

774

775 **Conclusion**

776 USDA Food Patterns across a broad range of ages and energy intake meet most goals for nutrient
777 adequacy. The nutrients of public health concern for which the patterns do not meet recommendations
778 are potassium and vitamin D. Recommended amounts of food groups and their component subgroups
779 fall within the broad range of usual food group intake distributions for the U.S. population.

780

781 **Implications**

782 The USDA Food Patterns provide guidance for consuming a nutrient-dense, energy-balanced diet. To
783 achieve nutrient adequacy, the U.S. population should be advised to consume dietary patterns
784 consistent with the USDA Food Patterns.

785

786 Continued vigilance is needed to ensure that food intake patterns meet but do not exceed DRI targets in
787 all age groups. The Patterns meet recommended intake levels or limits for almost all nutrients,
788 including the following nutrients of concern: calcium, fiber, iron, sodium, and saturated fat. Two
789 nutrients of concern (potassium and vitamin D) are not provided in recommended levels by the
790 Patterns. Therefore, potassium and vitamin D intakes require assessment both of individual intake and
791 population intake patterns of foods or supplements to ensure that needs for physiological functioning
792 are met. Meeting the needs for these nutrients may require careful attention to excellent natural
793 sources, food enriched or fortified with the nutrients, or, in some cases, consideration of supplements.

794

[♦] The USDA Food Patterns referred to in this question are the same as the “Healthy U.S.-style Food Pattern” described later in this chapter (see Question 20). We use the term USDA Food Patterns in this question because the development of the Healthy U.S.-style Food Pattern and two related USDA Food Patterns had not occurred when the Committee addressed this question.

795 Following the recommended food intake pattern increases intakes of whole grains, vegetables, fruits,
796 and fat-free/low fat dairy and thus increases the likelihood of meeting recommendations for these food
797 groups while decreasing intake of the food components refined grains, solid fats, and added sugars.
798 Following the recommended pattern also decreases intake of the nutrients sodium and saturated fat.
799

800 In some situations, specific foods or dietary supplements may be used to increase underconsumed
801 nutrient intakes not met through the USDA Food Patterns.
802

803 **Review of the Evidence**

804 These conclusions were reached based on the results of the Food Pattern Modeling Report on
805 Adequacy of the USDA Food Patterns. The USDA Food Patterns are intended to represent the types
806 and amounts of foods that will provide nutrients sufficient to meet IOM nutrient recommendations and
807 Dietary Guidelines for Americans recommendations. The Food Patterns are updated every 5 years
808 during the deliberations of the Dietary Guidelines Advisory Committee, and are presented to the
809 Committee for their assessment of the Food Patterns' adequacy. As part of the update, amounts
810 recommended from each food group may be modified to reach all or most of the specified goals. In
811 addition, the amounts from each food group are compared to usual dietary intake patterns of the U.S.
812 population, and are kept within the normal range of consumption. The current analysis, using the 2010
813 USDA Food Patterns as a baseline, found that the recommended amounts of each food group met
814 almost all nutrient goals and were within the normal range of consumption. Therefore, no updates to
815 the food group amounts from 2010 were needed.
816

817 As shown in Figure D1.8, for many nutrients, amounts of a nutrient in the patterns are well above the
818 RDA or AI. Protein, phosphorus, zinc, copper, selenium, manganese, vitamin C, thiamin, riboflavin,
819 niacin, vitamin K, folate, vitamin B₆, and vitamin B12 are above the goal amounts for all age/sex
820 groups.
821

822 In contrast, some nutrients are just above the RDA or AI, or marginally below (90 to 100%) goal
823 amounts for several age/sex groups. These include calcium, iron, and magnesium. The percents of the
824 RDA shown in Figure D1.8 are for the lowest calorie level assigned to these age/sex groups—the level
825 applicable for a sedentary/less active physical activity level.
826

827 The nutrients for which adequacy goals are not met in almost all patterns are potassium, vitamin D,
828 vitamin E, and choline. Due to the new higher RDA for vitamin D that was recommended by the 2011
829 Committee to Review Dietary Reference Intakes for vitamin D and calcium,¹⁵ amounts in the patterns
830 are a much smaller percentage of the RDA than previously, and no pattern meets the EAR for vitamin
831 D. To determine if vitamin D recommendations could be met while following the food group
832 recommendations of the USDA Food Patterns, thorough, careful selection of specific foods within
833 each food group, an additional modeling analysis was conducted and reported below (see Question 6).
834

835 The USDA Food Intake patterns provide a healthy pattern of food choices and to accomplish this,
836 these patterns deviate from typical food intakes in a number of ways. To ensure that the patterns do not
837 deviate too far beyond the range of what the U.S. population could feasibly consume, the
838 recommended intake amounts in the patterns from each food group or subgroup plus oils were
839 compared to the median and either the 5th or 95th percentile of usual intakes of the population, from
840 WWEIA/NHANES 2007-2010.⁴¹ Table A6 of the Adequacy of the USDA Food Patterns Modeling
841 Report (see *Appendix E-3.1*, Table A6) shows the comparison of food group recommended intakes to
842 median and 95th percentile intakes.

843
844 For underconsumed food groups, such as fruits and vegetables, recommended amounts in the patterns
845 are generally between the median and 95th percentiles of usual intakes. (see *Appendix E-3.1:*
846 *Adequacy of the USDA Food Patterns*, Table A6) This indicates that the Food Patterns recommend
847 amounts within the broad intake range for the population. However, for some specific food groups and
848 some age/sex groups, such as vegetables for males ages 14 to 18 years, food group amounts in the
849 Patterns are somewhat above the 95th percentile of usual intake. One exception to this is whole grain
850 recommendations in the Patterns, which are well above the 95th percentile of usual intakes for all
851 age/sex groups. Conversely, refined grain recommendations in the patterns are very low compared to
852 usual intakes—about the 5th percentile of intake for most age/sex groups. This indicates that a major
853 shift from refined to whole grains is needed in order to meet recommendations.

854
855 For Food Pattern components that are overconsumed, the limits in the patterns for maximum solid fat
856 and added sugars (see Questions 7 and 8 for more information on solid fats and added sugars) also are
857 very low compared to usual intake amounts—at approximately the 5th percentile of usual intakes for
858 most age/sex groups, and less than the 5th percentile of usual intakes for boys and girls ages 2 to 13
859 years. (see *Appendix E-3.1: Adequacy of the USDA Food Patterns*, Table A6)

860
861 An additional modeling analysis was conducted to answer the questions: How well do the USDA Food
862 Patterns meet the nutritional needs of children ages 2 to 5 years and how do the recommended amounts
863 compare to their current intakes? Given the relatively small empty calorie limit for this age group,
864 how much flexibility is possible in food choices? (see *Appendix E-3.4: USDA Food Patterns—*
865 *Adequacy for Young Children*)

866
867 The nutritional needs and the diets of young children are different in some important ways from the
868 nutritional needs and diets of older children and adults. Therefore, this modeling analysis focused on
869 the adequacy of the Patterns for young children, given these differences. Nutrient profiles for the Dairy
870 and Fruit groups were adjusted to better reflect the food choices within these groups of young children.
871 The adjusted Dairy group nutrient profile for young children is based on 70 percent fluid milk, 25
872 percent cheese, 3.5 percent yogurt, and 1.5 percent soymilk. In contrast, the profile for the overall
873 population is based on 51 percent fluid milk, 45 percent cheese, 2.5 percent yogurt, and 1.5 percent
874 soymilk. In addition, 1 percent milk rather than fat-free milk was used as the representative food for

875 fluid milk. The adjusted Fruit group nutrient profile for young children is based on 42 percent fruit
876 juice and 58 percent whole fruit. In contrast, overall population intake is about 33 percent juice and 67
877 percent whole fruit. With these adjustments, the adequacy of the Patterns did not change, but amounts
878 of potassium, vitamins D, A, C, and folate increased slightly, and sodium decreased slightly. The
879 amounts recommended in the USDA Food Patterns fall within the broad range of usual intakes by this
880 age group for most food groups and subgroups (see *Appendix E-3.1: Adequacy of the USDA Food*
881 *Patterns*, Table A6).

882

883 In addition, the young children's nutrient profiles were higher in energy, mainly due to the use of 1
884 percent rather than fat-free milk. Therefore, the amount of calories that could be allowed from solid
885 fats and added sugars was adjusted down to keep the Patterns isocaloric. This resulted in limited
886 flexibility in food choices when following the Patterns, especially for children ages 4 and 5 years for
887 whom 2½ cup equivalents (cup eqs) from the Dairy group is recommended (the Patterns for children
888 ages 2 and 3 years recommend 2 cup eqs). Options tested to increase flexibility in food choices
889 included a small reduction of 1/2 ounce eq in the amount of Protein Foods, or a change from 1 percent
890 milk to fat-free milk at 4 years of age. These changes did not result in lower nutrient adequacy levels.

891

892 *For additional details on this body of evidence, visit:*

- 893 • Appendix E-3.1: Adequacy of the USDA Food Patterns
- 894 • Appendix E-3.4: USDA Food Patterns—Adequacy for Young Children

895

896 **Question 6: Can vitamin D Estimated Average Requirements and/or Recommended**
897 **Dietary Allowances be met with careful food choices following recommended amounts**
898 **from each food group in the USDA Food Patterns? How restricted would food choices**
899 **be, and how much of the vitamin D would need to come from fortified dairy and other**
900 **food products?**

901

902 **Source of evidence:** Food Pattern Modeling

903

904 **Conclusion**

905 Through the use of a diet rich in seafood and fortified foods, EAR, but not RDA, levels of vitamin D
906 can be achieved. Additional fortification or supplementation strategies would be needed to reach RDA
907 levels of vitamin D intake consistently, especially in individuals with low intakes of fish/seafood or
908 fortified dairy foods, other fortified foods (e.g. breakfast cereals) and beverages.

909

910 **Implications**

911 Diet is an important aspect of achieving vitamin D intake targets. The U.S. population should be
 912 encouraged to choose foods and beverages fortified with vitamin D. When needed, supplementation
 913 can be considered to achieve RDA intakes of vitamin D.

914

915 **Review of the Evidence**

916 These conclusions were reached based on the results of the Food Pattern Modeling Report titled
 917 “Meeting Vitamin D Recommended Intakes in USDA Food Patterns” (see *Appendix E-3.3*). It may be
 918 difficult for individuals to reach the RDA intake of vitamin D from food, including food as it is
 919 currently fortified in the United States. The RDA was established by the Institute of Medicine on the
 920 assumption of minimal or no sunshine exposure. This was done even though the majority (up to 80 to
 921 90 percent in some parts of the United States) of vitamin D in the body is derived from conversion by
 922 solar radiation of pre-vitamin D in the skin. However, during the winter, in much of the United States,
 923 this conversion is minimal and furthermore, recommendations for sunscreen use have limited the
 924 degree to which one can safely ensure sunshine exposure as a source of vitamin D.

925

926 Vitamin D exposure, and likely status, is assessed generally through serum/plasma 25-hydroxyvitamin
 927 D concentrations. However, this test is not recommended for routine screening of the entire
 928 population^{30-32, 42, 43} due to costs and challenges in obtaining measurements throughout the year and
 929 interpreting results in populations, including those who are obese. Because many non-screened
 930 individuals will still need to reach the RDA for vitamin D, supplement use may be considered for this
 931 purpose.

932

933 *For additional details on this body of evidence, visit:*

- 934 • Appendix E-3.3 Meeting Vitamin D Recommended Intakes in USDA Food Patterns

935

936

937 **FOOD GROUPS--CURRENT INTAKES AND TRENDS**

938 **Introduction**

939 As noted for Questions 5 and 6, to help the U.S. population meet recommended dietary goals and
 940 improve their health and well-being, the USDA recommends a food-based, total diet approach for
 941 meeting the U.S. Dietary Guidelines.^{44, 45}

942

943 The USDA Food Patterns have changed over time to be consistent with emerging science that is
 944 presented in each issuance of the Guidelines. The current USDA Food Patterns identify amounts of
 945 foods to consume from five major food groups (fruits, vegetables, grains, protein foods, and dairy) and

946 their sub-groups (dark green vegetables, orange and red vegetables, starchy vegetables, other
 947 vegetables, beans and peas, whole grains, enriched/refined grains, meat/poultry/eggs, nuts, seeds, soy
 948 products, seafood) and are based on nutrient-dense foods.^{44, 45} In 2010, the DGAC developed a
 949 vegetarian adaptation of the Food Patterns to provide guidance for consumers wishing to follow a
 950 vegetarian diet. For 2015, the DGAC developed a new Healthy Vegetarian Food Pattern based on food
 951 intakes of vegetarians. The 2015 DGAC also provided a Mediterranean-style Food Pattern due to the
 952 data supporting the health-related benefits of a Mediterranean-style diet (see Dietary Patterns section,
 953 Question 20, and *Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes*).
 954 The food groups chosen for all the Patterns include primarily nutrient-dense foods. The patterns are
 955 intended to meet the RDA for nutrients so that nutritional adequacy is met without exceeding
 956 recommended energy intake. They also are designed so that they are below the 2010 DGA limits for
 957 sodium and saturated fat. Recommended amounts to consume from each food group differ depending
 958 on an individual's energy and nutrient needs. Patterns are provided for 12 different calorie levels
 959 (Table D1.10) and assignment to one of these calorie levels is based on age, sex, and activity level
 960 (Table D1.11). In addition, the Patterns provide for limited amounts of solid fats and added sugars. The
 961 complete Food Pattern modeling report (including a listing of the nutrients considered for the Patterns)
 962 is found in *Appendix E3.1*, and details on the methods used to derive the Patterns have been
 963 published.^{44, 46, 47}

964

965 **Question 7: What are current consumption patterns of USDA Food Pattern food groups**
 966 **by the U.S. population?**

967 **Source of evidence:** Data analysis

968

969 **Conclusion**

970 Positive, healthy eating habits provide an excellent foundation for a lifetime of healthy eating. Many
 971 young children start out eating very well, particularly with regard to intakes of fruit and dairy foods.
 972 Unfortunately, many of these early life healthy habits seem to disappear as children reach school age
 973 and beyond. Across all age and sex groups, the vast majority of the U.S. population does not meet
 974 recommended intakes for fruit, vegetables, whole grains, and dairy food groups. Each of these food
 975 groups are excellent sources of shortfall nutrients and underconsumed nutrients of public health
 976 concern. Across all age and sex groups, the vast majority of the U.S. population exceeds recommended
 977 intakes of refined grains, solid fats, and added sugars.

978

979 **Implications**

980 To realize the numerous health benefits from dietary patterns that are higher in fruit, vegetables, whole
 981 grains, lean protein, and non-fat and low-fat dairy (see *Part D. Chapter 2: Dietary Patterns, Foods*
 982 *and Nutrients, and Health Outcomes* for details on the health benefits for dietary patterns with these
 983 characteristics), action is needed across all sectors of food production, distribution, and consumption

984 and at individual behavioral and population levels. Individuals, families, schools, worksites, healthcare
 985 and public health settings, restaurants, and other food establishments must work together to ensure that
 986 all segments of the population can:

- 987 • Increase intake of underconsumed food groups and nutrient-dense foods, while maintaining
 988 energy balance, and without increasing saturated fat, sodium, and added sugars

989 Given the complexity of dietary behavior change, consumers will need access to evidence-based
 990 educational resources and intervention programs and services in public health and healthcare settings
 991 to facilitate adoption and maintenance of healthy dietary behaviors. (See *Part D. Chapter 3:*
 992 *Individual Diet and Physical Activity Behavior Change* for discussion of what works at the level of
 993 individual behavior change and *Part D. Chapter 4: Food Environment and Settings* for discussion of
 994 population change through environmental strategies.)
 995

996 Within the Dairy and Vegetable groups, the following dietary changes in particular will help increase
 997 intake of shortfall nutrients and will decrease intake of overconsumed nutrients by the U.S. population:

- 998 • Increasing low-fat/fat-free fluid milk and yogurt and decreasing cheese would result in higher
 999 intakes of magnesium, potassium, vitamin A, and vitamin D while simultaneously decreasing
 1000 the intake of sodium and saturated fat.
- 1001 • Replacing soft drinks and other sugar-sweetened beverages (including sports drinks) with non-
 1002 fat fluid milk would substantially reduce added sugars and empty calories and increase the
 1003 intake of shortfall nutrients, including calcium, vitamin D, and magnesium.
- 1004 • Consuming all vegetables, including starchy vegetables, with minimal additions of salt and
 1005 solid fat will help minimize intake of overconsumed nutrients – sodium and saturated fat.

1006

1007 **Review of the Evidence**

1008 This question was answered using data from the WWEIA, NHANES dietary survey (2007-2010) and
 1009 the National Cancer Institute’s examination of the usual intake distributions and percent of the U.S.
 1010 population meeting USDA Food Pattern recommendations for their age and sex.^{41, 48, 49} It is important
 1011 to note that the Dietary Guidelines for Americans are established only for those ages 2 years and older.
 1012 However, the WWEIA, NHANES sample includes persons from birth. The NHANES data are
 1013 presented in these specific age groups that cannot be further divided.
 1014

1015 **Fruit.** When consumed in the amounts recommended in the USDA Food Patterns, fruit contributes
 1016 substantial amounts of two nutrients of public health concern: fiber and potassium. (Whole fruit and
 1017 fruit juice provide about 16 percent of dietary fiber and 17 percent of potassium in the Food Patterns
 1018 (see *Appendix E-3.2: Food Group Contributions to Nutrients in USDA Food Patterns and Current*
 1019 *Nutrient Intakes*).
 1020

1021 The majority of children ages 1 to 3 years and 4 to 8 years meet the recommended intakes for total
1022 fruit, which is 1 cup and 1 to 1.5 cups per day, respectively. Among older children (boys and girls ages
1023 9 to 13 years), adolescents, and adults of all ages (both men and women), few people consume the
1024 recommended daily amounts, which range from 1.5 to 2 cups for older children and adolescents to 1.5
1025 to 2.5 cups for adults (Figure D1.9). Among the overall U.S. population, approximately 15 percent
1026 meet the daily fruit intake recommendation while nearly 80 percent do not meet the recommendation.
1027

1028 More than half of the daily fruit intake for all age and sex groups in the U.S. population (ages 1 year
1029 and older) comes from whole fruit (Figure D1.10). Among both boys and girls ages 1 to 3 years, whole
1030 fruit comprises slightly more than half of the daily fruit intake and the remainder is consumed though
1031 100% fruit juice. The American Academy of Pediatrics (2001)⁵⁰ recommends that young children limit
1032 their juice intake to 4 to 6 ounces per day. Six ounces of juice is 0.75 cups; the average juice intakes
1033 fall within this recommended limit suggesting that juice is not overconsumed among many young
1034 children. Among children ages 4 to 8 and 9 to 13 years, fruit intake includes both 100% juice and
1035 whole fruit, but whole fruit comprises the majority of intake. Among middle aged and older adults,
1036 most of the fruit intake is from whole fruit, albeit below recommended levels, rather than 100% juice.
1037

1038 **Vegetables.** Vegetables are excellent sources of many shortfall nutrients and nutrients of public health
1039 concern. When vegetables are consumed in the amounts recommended in the USDA Food Patterns,
1040 vegetables contribute the following (expressed as averages over all the calorie levels): fiber (38
1041 percent), potassium (36 percent), iron (19 percent), folate (23 percent), and vitamin A as provitamin A
1042 carotenoids (34 percent). Note that select vegetables do contribute to calcium intake, including
1043 spinach, collard greens, turnip greens, but these vegetables are often consumed in smaller amounts
1044 than is needed to be considered important sources of calcium (Table D1.6 and *Appendix E-3.2: Food
1045 Group Contributions to Nutrients in the USDA Food Patterns and Current Nutrient Intakes*).
1046

1047 The U.S. population consumes few vegetables (Figure D1.11). Only 10 percent and 15 percent of boys
1048 and girls ages 1 to 3 years, respectively, consume the recommended 1 cup of vegetables per day. For
1049 children ages 4 to 8 years, less than 5 percent consume the recommended amount of 1.5 to 2 cups of
1050 vegetables per day. Vegetable consumption is lowest among boys ages 9 to 13 years (1 percent
1051 consume the recommended 2 to 2.5 cups per day) and girls ages 14 to 18 years (less than 1 percent
1052 consume the recommended 2 to 2.5 cups/day). Vegetable intakes increase slightly during the adult
1053 years, but intakes are still very low. Among young adult males and females ages 19 to 30 years, less
1054 than 10 percent meet the 2 to 3.5 cups/day recommendation. Intakes increase only slightly in
1055 subsequent age decades (31 to 50 years). Middle aged adults (51 to 70 years) are somewhat closer to
1056 the goal as they have the highest vegetable intakes. Even so, only about 20 percent of men and about
1057 30 percent of women meet the daily recommendation of 2 to 3.5 cups per day. Although these intake
1058 levels are still below optimal, the positive gains in vegetable consumption are noteworthy. However,
1059 vegetable intakes fall again among older adults (71 years and older), with less than 20 percent of men

1060 and women meeting intake recommendations. Overall, nearly 90 percent of the U.S. population does
1061 not meet daily vegetable intake recommendations.

1062
1063 The USDA Food Pattern food group for vegetables includes five subgroups: dark green vegetables, red
1064 and orange vegetables, beans and peas, starchy vegetables, and other vegetables. The U.S. population
1065 does not meet intake recommendations for any of these vegetable subgroups (Figures D1.12 to D1.16).
1066 More than 80 percent of the U.S. population does not meet the intake recommendation for dark green
1067 vegetables, starchy vegetables, and beans and peas, while more than 90 percent do not meet the
1068 recommended intakes for red and orange vegetables. “Other vegetables” (Figure D1.16) is a broad
1069 group that includes iceberg lettuce, green beans, cucumbers, celery, onions, summer squash,
1070 mushrooms, and avocados. More than 50 percent of males and females ages 51 to 70 years meet or
1071 exceed the recommended intake amounts of other vegetables and among all ages, nearly 40 percent
1072 meet or exceed the recommended intake. Intake of “other vegetables” is more likely to meet
1073 recommendations than the other four subgroups, but consumers should be encouraged to increase
1074 intake of all vegetables. To meet total vegetable recommendations, higher intakes of all vegetable
1075 subgroups are needed, particularly those subgroups where intake is minimal, such as dark green and
1076 orange and red vegetables, which are excellent sources of vitamin C, folate, magnesium, and
1077 potassium.

1078
1079 Potatoes (white potatoes) are the most commonly consumed single vegetable, and make up about 80
1080 percent of all starchy vegetable consumption.⁵¹ They account for 25 percent of all vegetable
1081 consumption and are a good source of both potassium and fiber. Among children and adolescents ages
1082 2 to 19 years, they account for 28 percent to 35 percent of total vegetable consumption, with a higher
1083 percentage of vegetables consumed as potatoes among boys than girls in each age category. Potatoes
1084 are consumed in a variety of forms, with about 31 percent being boiled (including mashed and in
1085 dishes such as potato salad, soups, and stews), 22 percent as chips, sticks, or puffs, 19 percent as
1086 French fries, 17 percent as baked, and 12 percent as home fries or hash browns.

1087
1088 **Grains (whole and refined).** The 2010 Dietary Guidelines for Americans recommended that half of
1089 all grain intake should come from whole grains. The 2015 DGAC brings forward this recommendation
1090 and here we give rationale and results to support this decision. The background and summary of
1091 previous food pattern modeling with respect to grains is important to present here so as to provide
1092 context for the 2015 DGAC recommendations.

1093
1094 Whole grains are those “foods made from the entire grain seed, usually called the kernel, which
1095 consists of the bran, germ and endosperm. If the kernel has been cracked, crushed or flaked, it must
1096 retain nearly the same relative proportions of bran, germ and endosperm as the original grain in order
1097 to be called whole grain.”^{52p134} Examples of whole grains are brown rice, popcorn, bulgur, whole
1098 wheat, oats, and barley. If whole grains were consumed in the amounts recommended in the Food
1099 Patterns, whole grains would provide substantial percentages of several key nutrients, such as about 32

1100 percent of dietary fiber, 42 percent of iron, 35 percent of folate, 29 percent of magnesium, and 16
1101 percent of vitamin A (see *E-3.2: Food Group Contributions to Nutrients in USDA Food Patterns and*
1102 *Current Nutrient Intakes*).

1103

1104 Across all ages and both sexes, the U.S. population does not meet the goal for whole grain intake, as
1105 nearly 100 percent of the population consumes amounts that are below the recommended intake levels
1106 (Figure D1.17), which range from 1.5 ounce equivalents (oz eq) for young children up to 3 to 3.5 oz
1107 eqs for older children and adolescent and adult females. Adolescent and adult males are advised to
1108 consume 3 to 4 oz eqs per day. The inadequate intake of whole grains leads to underconsumption of
1109 several shortfall nutrients and nutrients of public health concern. Refined grains, such as white flour
1110 and products made with white flour, white rice, and de-germed cornmeal, are part of the intake
1111 recommendation because they are commonly enriched with iron and several B vitamins, including
1112 thiamin, niacin, and riboflavin (e.g., enriched flour, 21 CFR 137.165).^{53p.452} Since 1998, enriched
1113 grains also have been fortified with folic acid and are thus an important source of folic acid for women
1114 of childbearing potential.^{53, 54} The effect of the folic acid fortification on the health status of the U.S.
1115 population was extensively reviewed by the 2010 DGAC and so was not re-reviewed by the 2015
1116 DGAC. The 2010 DGAC concluded that strong and consistent evidence demonstrates a large reduction
1117 in the incidence of neural tube defects (NTDs) in the United States and Canada following mandatory
1118 folic acid fortification. They also found only limited evidence to suggest a decline in stroke mortality
1119 in the United States and Canada and an increase in colorectal cancer in those countries following
1120 mandatory folic acid fortification. Due to the very limited evidence, cause and effect cannot be
1121 attributed for folic acid fortification and either stroke or colorectal cancer incidence. The 2015 DGAC
1122 brings forward those results with no notable changes in the interpretation of the data presented in 2010.
1123 Despite the B vitamins and iron that can be obtained from enriched and fortified refined grains,
1124 products made with refined grains also may be a source of excess calories and added sugars. (See
1125 Question 11c, food categories, below, and added sugars discussion in *Part D. Chapter 6: Cross-*
1126 *Cutting Topics of Public Health Importance*.) Figure D1.18, documents that the U.S. population
1127 consumes far too many refined grains. In the overall population for all ages and for both males and
1128 females, about 19 percent meet the recommendation for refined grains, while more than 70 percent
1129 exceed the recommendation. Intake of refined grains is particularly high among boys and girls ages 4
1130 to 8 years and girls ages 9 to 13 years.

1131

1132 Due to the overconsumption of refined grains and the underconsumption of whole grains relative to the
1133 2010 recommendation that “half of all grain intake should come from whole grains,” the DGAC
1134 decided that it was important to examine the impact on nutrient intake if: (1) refined/enriched grains
1135 intake were reduced to no more than 25 percent or 15 percent of the total grains intake; and (2) overall
1136 grain intake were reduced. The Committee relied on food pattern modeling analyses conducted by the
1137 2005 and 2010 DGACs to answer these questions, and brings forward their recommendations, as
1138 reiterated below.

1139

1140 The key finding from the 2010 DGAC modeling report was: “As shown by food pattern modeling,
 1141 consumption of all grains as whole grains, without including any fortified whole grain products, would
 1142 lower dietary folate and iron intake levels to less than adequate amounts for individuals in population
 1143 groups who may be at high risk for inadequate intakes of these nutrients. Individuals are encouraged to
 1144 consume most of their grains as fiber-rich whole grains, and when doing so, should select some of
 1145 these fiber-rich whole grains as products that have been fortified with folic acid and possibly other
 1146 nutrients”.^{55p146}

1148 In its analysis, the 2005 DGAC reported that non-whole grains contributed important amounts of
 1149 certain nutrients to the dietary patterns, including folate, iron, calcium, fiber, thiamin, riboflavin and
 1150 niacin.^{56append G-2} The 2005 DGAC concluded that including only 3 oz eqs of whole grains, with no
 1151 non-whole grains, in the food patterns would lower intake of many of these key nutrients and perhaps
 1152 place certain individuals at risk of nutrient inadequacy. However, the 2010 DGAC found that
 1153 consuming all grains as whole grains would provide for nutrient adequacy in the patterns if fortified
 1154 ready to eat (RTE) whole grain breakfast cereals were substituted for RTE refined grain breakfast.^{55app}
 1155 E.7 The 2015 DGAC concluded that consumption of only whole grains with no replacement or
 1156 substitution would result in nutrient shortfalls.

1158 **Dairy.** Dairy foods in the USDA Food Patterns include fluid milk, cheese, yogurt, ice cream, milk-
 1159 based replacement meals and milk products, including fortified soymilk, but do not include almond or
 1160 other plant-based “milk-type” products. Dairy foods are excellent sources of nutrients of public health
 1161 concern, including vitamin D, calcium, and potassium. Consumption of dairy foods provides numerous
 1162 health benefits including lower risk of diabetes, metabolic syndrome, cardiovascular disease and
 1163 obesity.⁵⁷⁻⁶² When consumed in the amounts recommended by the Food Patterns, on average across
 1164 the calorie levels, dairy foods contribute about 67 percent of calcium, 64 percent of vitamin D, and 17
 1165 percent of magnesium (see *Appendix E-3.2: Food Group Contributions to Nutrients in the USDA*
 1166 *Food Patterns and Current Nutrient Intakes*). The Patterns recommend consumption of low-fat and
 1167 fat-free foods in the Dairy group to ensure intake of these key nutrients while minimizing intake of
 1168 saturated fat, which is a nutrient of concern for overconsumption.⁴⁴

1170 More than 60 percent of young boys and girls ages 1 to 3 years meet or exceed the recommended
 1171 intake of 2 cup eqs per day, with most of this intake coming in the form of fluid milk (see Figure
 1172 D1.19 and Appendix E-3.4: USDA Food Patterns—Adequacy for Young Children). Intake falls in
 1173 older children to about 30 percent of boys and girls meeting or exceeding the recommended 2.5 cup
 1174 eqs per day for those ages 4 to 8 years and 3 cup eqs per day for children ages 9 to 13 years. About 30
 1175 percent of adolescent boys meet or exceed the recommended 3 cup eqs per day, but less than 10
 1176 percent of adolescent females meet or exceed this recommendation. An age-related decline in dairy
 1177 intake appears to begin in adolescence and intakes persist at very low levels among adult females
 1178 across the age distribution. Less than 5 percent of adult females consume the recommended 3 cup

1179 equivalents per day. Overall, more than 80 percent of the entire U.S. population does not meet the
1180 daily dairy intake recommendation.

1181

1182 To determine the extent to which individuals could meet recommendations for calcium and other
1183 shortfall nutrients intake, given various levels of dairy foods in the Food Patterns, the 2015 DGAC
1184 conducted a food pattern modeling analysis (see *Appendix E-3: Dairy Group and Alternatives*). The
1185 DGAC considered nutrient adequacy of the Food Patterns under the following scenarios: 1) no dairy
1186 was consumed; 2) calcium was obtained from non-dairy sources (including fortified foods); and 3) the
1187 proportions of yogurt and cheese in the patterns were modified. The DGAC further evaluated the
1188 relationship between changes in the types of beverages consumed (milk, fruit juices, fruit drinks and
1189 sports beverages) and diet quality.

1190

1191 If no dairy is consumed, the modeling analysis shows that levels of calcium, magnesium, iron, vitamin
1192 A and riboflavin, drop below 100 percent of goals, and intake levels of potassium, vitamin D and
1193 choline also drop substantially. When no dairy is consumed, calcium intake levels drop by 68 to 88
1194 percent in all age and sex groups, while vitamin D intake is lowered by 20 to 30 percent (see *Appendix*
1195 *E-3.6: Dairy Group and Alternatives*, Table 2). Most of the milk alternatives are fortified with
1196 calcium, so similar amounts of calcium can be obtained from fortified rice, soy and almond milks, and
1197 fortified juices, but absorption of calcium is less efficient from plant beverages.⁶³ Magnesium intake
1198 also is comparable from plant-based milk alternatives. However, vitamin D and potassium amounts
1199 vary across these milk alternatives (see *Appendix E-3.6: Dairy Group and Alternatives*, Table 3).
1200 Calorie levels also are higher for most of the plant-based alternative milk products for a given calcium
1201 intake level. In other words, to obtain a comparable amount of calcium as one cup eq for non-fat fluid
1202 milk, the portion size required to meet the calcium intake need results in higher energy intake (see
1203 *Appendix E-3.6: Dairy Group and Alternatives*, Table 4).

1204

1205 Currently, the U.S. population consumes the recommended 3 cup equivalents/day as 53 percent fluid
1206 milk, 45 percent cheese, and 2 percent as yogurt. Through the food pattern modeling, the DGAC
1207 examined the effect on nutrient intake if fluid milk were to be increased and cheese decreased.
1208 Increasing the proportion of fat-free milk, while decreasing the proportion of cheese, would increase
1209 the intake of magnesium, potassium, vitamin A, vitamin D and would decrease intake of sodium and
1210 saturated fat (see *Appendix E-3.6: Dairy Group and Alternatives*, Table 5). A potential approach to
1211 increasing intake of shortfall nutrients and nutrients of public health concern while simultaneously
1212 decreasing intake of overconsumed nutrients of public health concern would be to increase intake of
1213 fat-free or low-fat fluid milk in lieu of cheese.

1214

1215 If milk is completely eliminated from the diet and replaced by soft drinks, fruit drinks, sports
1216 beverages, and other sugar-sweetened beverages, diet quality deteriorates significantly, making it very
1217 hard for individuals to meet nutrient recommendations (see *Appendix E-3.6: Dairy Group and*

1218 *Alternatives*, Table 6). Indeed, among U.S. adolescents' milk consumption is very low as are intakes of
1219 the "shortfall" nutrients.

1220

1221 **Protein Foods.** Protein Foods comprise a broad group of foods including meat, poultry, fish/seafood,
1222 eggs, soy,[∞] nuts, and seeds. Dairy also contains protein, but since it has its own food group, its nutrient
1223 contributions are counted in its own group. The inclusion of both animal and non-animal protein foods
1224 allows vegetarian options to be accommodated. In addition to providing essential amino acids, some
1225 protein foods are important sources of iron, and iron is a shortfall nutrient and nutrient of public health
1226 concern among adolescent and adult females. Meat foods in the protein group provide heme iron,
1227 which is more bioavailable than non-heme plant-derived iron. Heme iron is especially important for
1228 young children and women who are pregnant.

1229

1230 Nearly 80 percent of boys and 75 percent of girls ages 1 to 3 years meet or exceed the protein foods
1231 recommendation of 2 ounce equivalents per day (Figure D1.20). Similarly, more than 60 percent of
1232 boys and girls ages 4 to 8 years meet or exceed the recommended intake of 3 to 4 oz eqs/day. Intake
1233 declines somewhat for boys and girls ages 9 to 13 years, as approximately 40 percent and 45 percent
1234 meet or exceed the recommended 3 ounce equivalents/day. Although nearly 60 percent of adolescent
1235 males ages 14 to 18 years meet the 5.5 to 6.5 oz eq/day recommendation, less than 25 percent of
1236 females ages 14 to 18 meet their 5-5.5 oz eq/day recommendation. Intakes begin to increase again for
1237 adult males across the age distribution, and about 62 percent of males ages 31 to 50 and 78 percent of
1238 males 51 to 70 years meet the 5.5-6.5 oz eq/day intake recommendation. For adult females ages 19 to
1239 30 years, slightly more than 40 percent meet the 5 to 5.5 oz eq/day recommendation and approximately
1240 50 percent of those ages 31 to 50 and about 50 percent of those 51 to 70 years meet the
1241 recommendation. Protein foods intake declines in both men and women older than age 71 years; about
1242 30 percent of women and about 50 percent of men meet the recommendation. Across all age groups
1243 and in both males and females, nearly 60 percent of the U.S. population meets the protein foods intake
1244 recommendation. Although some groups in the U.S. population do not consume recommended
1245 amounts from the protein foods group, intakes of protein (as grams/day) are adequate across the
1246 population and protein is not a shortfall nutrient. Notably, protein intake also comes from dairy and
1247 grains in addition to the foods included in the protein foods group.

1248

1249 Most of the protein foods intake across all age groups and for both males and females comes from
1250 meat, poultry, and eggs (Figure D1.21). Nearly 80 percent of the U.S. population meets the intake
1251 recommendation for this protein foods subgroup (although less so for adolescent girls and older
1252 women).

1253

[∞] Soy foods in the Protein Foods group include foods and ingredients such as tofu, soy noodles, soy flours, and soy protein isolates. Fortified soymilk is part of the Dairy group. Edamame and whole soybeans are part of the vegetable legume subgroup.

1254 In 2010, the DGAC recommended that seafood intake be increased to eight ounces per week for adults.
1255 In reviewing the WWEIA/NHANES data, the DGAC 2015 found that the U.S. population has low
1256 seafood intake. Across all age groups and for both males and females, only 10 percent of the
1257 population meets the 2010 intake recommendations (Figure D1.22). Intake is highest in adult men and
1258 women, but remains very low. In the highest intake group, males ages 51 to 70 years, 21 percent of the
1259 population meets the intake recommendation.

1260

1261 In addition to reviewing WWEIA/NHANES data, the 2015 DGAC considered the potential influence
1262 on diet quality of substituting seafood for terrestrial animal foods (e.g., beef, poultry, pork, game
1263 meats). This question was addressed by the 2010 DGAC through a modeling analysis, and the 2015
1264 DGAC decided to bring forward those modeling results. These results indicate seafood could be
1265 increased to 8 ounces/week (for adults) with no negative impact on nutrient adequacy.^{55app E3.10} This 8
1266 oz amount contributes energy, protein, selenium, vitamin D, and vitamin B-12. With respect to fatty
1267 acids, fish is rich in the long-chain eicosapentanoic acid (EPA) and docosahexonoic acid (DHA) and
1268 has a higher proportion of total fatty acids coming from polyunsaturated and monounsaturated fatty
1269 acids relative to saturated fatty acids. The 2015 DGAC also has examined the sustainability of fish
1270 production and consumption, and these results are discussed in *Part D. Chapter 5: Food Sustainability*
1271 *and Safety*.

1272

1273 **Nuts, seeds, and soy.** Nuts, seeds, and soy provide protein, selenium, polyunsaturated fatty acids,
1274 fiber, magnesium, and zinc. Nuts, seeds, and soy are less commonly consumed protein foods (Figure
1275 D1.23). Even so, overall approximately 40 percent of the U.S. population meets or exceeds the food
1276 pattern recommended intake of these protein foods.

1277

1278 **Empty calories.** Solid fats that occur naturally in foods such as meat, dairy, and some tropical foods
1279 (e.g., coconut), and sugars that are added to foods either by the consumer or by food manufacturers are
1280 referred to as “empty calories” because both provide calories, but few or no nutrients. For the purposes
1281 of the USDA Food Pattern Food Groups, the term solid fats and added sugars is an analytic grouping,
1282 but going forward for 2015, the DGAC has elected to use the term “empty calories.”

1283

1284 Calories from solid fats and added sugars are included for the USDA Food Patterns because they are a
1285 component of the diet that should be limited because they are not nutrient-dense and the solid fats
1286 contribute to saturated fat intake, which is overconsumed in the U.S. population (see Nutrient
1287 Intake/Nutrients of Concern section, Questions 1 and 2). Solid fats and added sugars are not food
1288 groups on their own, as are protein foods, dairy, grains, fruits, and vegetables, but they are included in
1289 the Food Patterns because they are an integral component of many foods consumed by the U.S.
1290 population either because they occur naturally (in the case of some solid fats) or they are added to
1291 foods, such as added sugars or fat added during processing, cooking, or other aspects of food
1292 preparation. Additional details about added sugars and saturated fat are provided in *Part D. Chapter 6:*
1293 *Cross-Cutting Topics of Public Health Importance*.

1294

1295 Because added sugars and solid fats are not nutrient dense and solid fats contribute to saturated fat
 1296 intake, the USDA Food Patterns recommend that intake be limited. The guidance on the approximate
 1297 amounts of solid fats and added sugars that can be part of a healthful diet is as follows: children ages 2
 1298 to 8 years: 120 calories/day; children 9 to 13 years: 120 to 250 calories/day; girls ages 14 to 18 years:
 1299 120 to 250 calories/day; boys ages 14 to 18: 160 to 330 calories/day; adult women: 120 to 250
 1300 calories/day; and adult men: 160 to 330 calories/day. Intake limits varies by age and sex and are based
 1301 on residual calories after all food group intakes are met. The intake limits include solid fats and added
 1302 sugars from all sources in the diet: from sugar in sugar-sweetened beverages, including coffee and tea,
 1303 and breakfast cereals, to solid fats in burgers, sandwiches, and pizza, to the combination of solid fats
 1304 and added sugars in snacks and desserts such as cookies, cakes, ice cream, and donuts. Question 11 of
 1305 the Food Categories section of this Chapter provides information on food sources of solid fats and
 1306 added sugars.

1307

1308 The intake of solid fats and added sugars is very high across all age groups and for both males and
 1309 females in the United States, with nearly 90 percent exceeding the recommended daily limits (Figure
 1310 D.1.24). Particularly noteworthy is that nearly 100 percent of boys and girls ages 1 to 3 and 4 to 8
 1311 years exceed the recommended limit for solid fats and added sugars (see **Part B. Chapter 6: Cross**
 1312 **Cutting Topics of Public Health Importance**).

1313

1314 ***For additional details on this body of evidence, visit:***

- 1315 • Usual Dietary Intakes: Food Intakes, U.S. Population, 2007-10: Applied Research Program.
 1316 National Cancer Institute; [updated May 22, 2014]. Available from:
 1317 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/>.
- 1318 • Appendix E-3.2 USDA Food Pattern Modeling Report: Food Group Contributions
- 1319 • Appendix E3.6 USDA Food Pattern Modeling Report: Dairy Group and Alternatives
- 1320 • Food Patterns Equivalent Intakes from Food: Consumed per Individual, 2009-10. U.S. Department
 1321 of Agriculture, Agricultural Research Service, Food Surveys Research Group. Available from:
 1322 <http://seprl.ars.usda.gov/Services/docs.htm?docid=23868>.
- 1323 • Seafood Food Pattern Modeling Report for the 2010 Dietary Guidelines Advisory Committee.
 1324 USDA and HHS, 2010, Appendix E 3.10 USDA and HHS, 2010, Appendix E 3.10. Available
 1325 from: http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/AppendixE-3-10-Seafood.pdf.
- 1326
- 1327 • Replacing all Non-Whole Grains with Whole Grains Food Pattern Modeling Report for the 2010
 1328 Dietary Guidelines Advisory Committee. USDA and HHS, 2010, Appendix E3.7. Available from:
 1329 http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/AppendixE-3-7-Grains.pdf.
- 1330

- 1331 • Alternatives for Enriched Grains in Food Intake Patterns Analysis for the 2005 Dietary Guidelines
1332 Advisory Committee. U.S. HHS and USDA, 2005, appendix G-2. Available from:
1333 http://www.health.gov/dietaryguidelines/dga2005/report/HTML/G2_Analyses.htm#alternativegrain
1334 [n](http://www.health.gov/dietaryguidelines/dga2005/report/HTML/G2_Analyses.htm#alternativegrain).

1335

1336

1337 **Question 8: What are the trends in USDA Food Pattern food group consumption by**
1338 **the U.S. population?**

1339 **Source of Evidence:** Data analysis

1340

1341 **Conclusion**

1342 The U.S. population has made few dietary changes over time:

- 1343 • Fruit intake has remained low but stable.
- 1344 • Vegetable intake has declined, particularly among children of all ages, adolescents, and young
1345 adult males.
- 1346 • Whole grain intake has slightly increased between 2001-2004 and 2007-2010, particularly
1347 among middle aged and older adults.
- 1348 • Dairy intake has been relatively constant over time, but has decreased for girls ages 4 to 8 years
1349 and young adult males, and has increased for adults ages 51 to 70 years.
- 1350 • Added sugars intake has decreased for both males and females across all age groups between
1351 2001-2004 and 2007-2010, but intakes still exceed the limit in the USDA food patterns.

1352 **Implications**

1353 Individuals and families must make conscious and focused decisions about choosing nutrient-dense
1354 foods. In addition, to continue progress toward consumption of a healthy diet among all age and sex
1355 groups, action is needed along the entire food processing, delivery, and service supply chain in order to
1356 provide the U.S. population with affordable and accessible foods that are nutrient dense and low in
1357 added sugars and sodium.

1358

1359 Poor nutritional intake is linked to numerous diet-related chronic diseases (see *Part D. Chapter 2:*
1360 *Dietary Patterns, Foods and Nutrients, and Health Outcomes*) and the prevalence of these conditions
1361 is too high in the United States (see Health Conditions section, Questions 15 to 17, below). The health
1362 of the nation hinges in part on improving dietary intake at individual and population levels, and
1363 changes in line with those suggested here could have a measurable positive impact on the health of the
1364 population.

1365

1366 Given the complexity of dietary behavior change, consumers will need access to evidence-based
1367 educational resources and intervention programs and services in public health and healthcare settings
1368 to facilitate adoption and maintenance of healthy dietary behaviors. (See **Part D. Chapter 3:**
1369 **Individual Diet and Physical Activity Behavior Change** for discussion of what works at the level of
1370 individual behavior change.) In addition, these efforts should be complemented with research-driven
1371 environmental strategies that make access to affordable healthy foods possible in retail, community,
1372 worksite, and educational settings. (See **Part D. Chapter 4: Food Environment and Settings** for
1373 discussion of effective environmental approaches to promote dietary change across the lifespan.)
1374

1375 **Review of the Evidence**

1376 This question was answered using data from WWEIA, NHANES dietary survey data and the National
1377 Cancer Institute's examination of usual intake distributions for 2001-2004⁶⁴ and 2007-2010.⁴¹
1378

1379 **Fruit.** Fruit intake remained relatively stable across the 2001-2004 and 2007-2010 time periods
1380 (Figure D1.25). The only group with significant changes over time was males ages 31 to 50 years, for
1381 whom mean fruit intake decreased.
1382

1383 **Vegetables.** Vegetable intake declined from 2001-2004 to 2007-2010 (Figure D1.26). Across the
1384 overall population, the mean daily vegetable intake significantly declined. Significant declines in mean
1385 intake occurred among males ages 1 to 3, 4 to 8, 9 to 13, 14 to 18, and 19 to 30 years. For females,
1386 significant decreases in mean vegetable intake occurred for those ages 1 to 3, 4 to 8, and 9 to 13 years.
1387

1388 **Grains (whole and refined).** Whole grain intake significantly increased among the overall population
1389 between 2001-2004 and 2007-2010 (Figure D1.27). Among males, significant increases in mean intake
1390 occurred for those ages 1 to 3, 4 to 8, 14 to 18, 31 to 50, and 51 to 70 years. Among females,
1391 significant increases in mean whole grain intake occurred for those ages 9 to 13, 19 to 30, 31 to 50, 51
1392 to 70, and 71 years and older (Figure D1.27). Similarly, refined grain intake has declined in all age and
1393 sex groups between 2001-2004 and 2007-2010 (Figure D1.28).
1394

1395 **Dairy.** Dairy intake remained stable over the entire population between 2001-2004 and 2007-2010
1396 (Figure D1.29). Significant declines in mean daily intake occurred between the two time periods for
1397 males ages 19 to 30 years and females ages 4 to 8 years. Significant increases in mean daily dairy
1398 intake occurred for both males and females ages 51 to 70 years.
1399

1400 **Protein Foods.** Protein food intake remained relatively stable for the U.S. population between 2001-
1401 2004 and 2007-2010 (Figure D1.30). Females ages 31 to 50 and 51 to 70 years had significantly higher
1402 mean intake in 2007-2010 compared to 2001-2004. These were the only groups with any significant
1403 change over time.
1404

1405 **Added Sugars.** Some improvements have been made in added sugars intake, with noticeable declines
 1406 in mean intakes for all age groups and among both males and females when comparing 2007-2010 data
 1407 with 2001-2004 data (Figure D1.31). As seen in Figure D1.31, intakes of added sugars are still very
 1408 high, however, and are well above recommended limits, but the improvements provide some optimism
 1409 for improved diets.

1410

1411 *For additional details on this body of evidence, visit:*

1412 • Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program.
 1413 National Cancer Institute; [updated May 22, 2014]. Available from:

1414 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/>.

1415

1416 • Usual Dietary Intakes: Food Intakes, US Population, 2001-04: Applied Research Program.
 1417 National Cancer Institute; [updated April 2, 2014]. Available from:

1418 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2001-04/>.

1419

1420

1421 **FOOD CATEGORIES—CURRENT INTAKES AND SOURCES OF ENERGY,** 1422 **NUTRIENT, AND FOOD GROUP INTAKES**

1423 The food sources of nutrients and the patterns in which they are consumed are informative in
 1424 identifying strategies to modify dietary intake and eating behaviors and help Americans to choose and
 1425 consume higher quality diets. We examined four questions related to the foods that are top contributors
 1426 to intakes of energy, food groups, and selected nutrients in the U.S. diet. This section describes those
 1427 food sources and the implications for meeting recommended or optimal intakes of various food groups
 1428 and nutrients.

1429

1430 **Question 9: What are current consumption patterns by food categories (i.e., foods as**
 1431 **consumed) in the U.S. population?**

1432 **Source of evidence:** Data analysis

1433

1434 **Conclusion**

1435 The mixed dishes food category, which includes foods commonly used as entrees, such as sandwiches,
 1436 burgers, pizza, pasta or rice mixed dishes, stir-fries, soups, and meat or poultry mixed dishes, is the
 1437 major contributor to three USDA Food Pattern food groups—grains, vegetables, and protein foods.
 1438 Fruit and fluid milk intake are seldom consumed as part of mixed dishes. The mixed dishes food
 1439 category contributes heavily to intake of energy, saturated fat, and sodium; however, mixed dishes do
 1440 provide vegetables, fiber, grains, and dairy.

1441

1442 **Implications**

1443 An important strategy for meeting recommended intake levels of calories, saturated fat, and sodium is
1444 to change the composition of mixed dishes that are high in calories, saturated fat, and sodium to better
1445 meet these nutrition goals. Food manufacturers and the food service sector (e.g., restaurants, schools)
1446 should reformulate mixed dishes to improve their nutritional profiles. Americans should be encouraged
1447 to modify recipes to lower the sodium and saturated fat content when cooking, to use appropriate
1448 portion sizes, and choose reformulated mixed dish options when available.

1450 **Review of the Evidence**

1451 These conclusions were reached by examining data from the WWEIA Food Categories for the
1452 NHANES 2009-2010 dietary survey.⁶⁵ The WWEIA Food Categories provide an application that
1453 allows analysts to examine foods and beverages as consumed in the U.S. diet. Each food or beverage
1454 item (as consumed) that is included in WWEIA is placed in one of 150 mutually exclusive food
1455 categories. The focus of this categorization system is on grouping similar foods and beverages together
1456 based on usage and nutrient content.

1457
1458 An adaptation of the food categories was used by the 2015 DGAC for this analysis related to the
1459 “sandwiches and burgers” and “salads” categories. We placed all food items reported to be eaten as a
1460 sandwich, burger, taco, or salad item into the “sandwiches and burgers” or the “salads” categories
1461 regardless of whether the components were reported as separated ingredients or as a single combined
1462 item. For example, a food reported as a “cheeseburger” (a single item) would always be classified in
1463 the category of “burgers, sandwiches, and tacos,” but a food reported as the individual food items of a
1464 hamburger bun, a hamburger patty, and cheese, eaten as a combination, would have been classified in
1465 the categories of “rolls and buns,” “ground meat,” and “cheese.” The adaptation recoded these
1466 individually reported foods that were eaten in combination to “burgers, sandwiches, and tacos.” By
1467 doing this, the categories used for this analysis more fully represented foods as consumed rather than
1468 as ingredients.

1469
1470 The 150 categories from WWEIA were condensed into 9 major and 32 sub-categories for analysis of
1471 the percent of total intake for energy, nutrients, and food groups from each major and sub-category
1472 (see *Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food*
1473 *Categories*). Analysis was conducted for the population ages 2 and older as a whole; analysis of the
1474 percent of energy intake also was conducted for males and females ages 2 to 5, 6 to 11, 12 to 19, 20 to
1475 40, 41 to 50, 51 to 70, and 71 years and older; for race/ethnic groups including Non-Hispanic Whites,
1476 Non-Hispanic Blacks, and Hispanics ages 2 years and older; and for those with incomes less than or
1477 equal to 185 percent, or greater than 185 percent of the Poverty Index Ratio by three age groups: 2 to
1478 11, 12 to 19, and 20 years and older.

1479

1480 WWEIA data show that Americans consume a substantial amount of foods in the form of mixed dishes
 1481 (Figure D1.32). More specifically, 31 percent of vegetables, 45 percent of grains, 30 percent of dairy,
 1482 and 45 percent of protein foods come from mixed dishes. Mixed dishes (which include foods such as
 1483 sandwiches, burgers, pizza, pasta or rice mixed dishes, stir-fries, soups, and meat or poultry mixed
 1484 dishes) make up 28 percent of total energy intake. Of note, only small amounts of fruits (1 percent) and
 1485 fluid milk (3 percent) are consumed in mixed dishes—most are consumed as single food items, such as
 1486 an apple or glass of milk (see *Appendix E-2.8: Percent of total food group intake, 2009-2010, for*
 1487 *U.S. population ages 2 years and older, from WWEIA Food Categories*).

1488
 1489 When mixed dishes contribute to dairy foods, the majority of intake is in the form of cheese. Data
 1490 show that about two-thirds of all cheese intake is from mixed dishes such as pizza, burgers,
 1491 sandwiches, and casseroles. Given that cheese is generally higher in saturated fat and sodium and
 1492 lower in potassium and vitamin D than is fluid milk (see Question 7b, above, and *Appendix E-3.6:*
 1493 *Dairy Group and Alternatives*), modifying the types of cheese products used in these mixed dishes to
 1494 lower fat and sodium versions would improve their nutritional profile.

1495
 1496 When mixed dishes contribute to the grains group, a larger percentage of refined (48 percent) than
 1497 whole (19 percent) grains are consumed as part of these dishes. Substitution of whole for refined grains
 1498 in mixed dishes such as burgers, sandwiches, pizza, and casseroles containing pasta or rice could
 1499 improve the nutritional profile of grains that are consumed this way.

1500
 1501 Although mixed dishes account for a substantial amount of intake of some overconsumed nutrients (43
 1502 percent of sodium, 36 percent of saturated fat), they also account for 28 percent of fiber, 29 percent of
 1503 calcium, 24 percent of potassium, and 16 percent of vitamin D, all of which are underconsumed
 1504 nutrients. Other food categories that contribute substantially to overall energy, sodium, saturated fat,
 1505 and added sugars intake are discussed in the following two questions—Question 10: “What are the top
 1506 foods contributing to energy intake in the U.S. population?” and Question 11: “What are the top foods
 1507 contributing to sodium, saturated fat, and added sugars intake in the U.S. population?”

1508
 1509 ***For additional details on this body of evidence, visit:***

- 1510 • What We Eat in America. Food Categories for the NHANES 2009-2010 dietary survey. Available
 1511 from: <http://seprl.ars.usda.gov/Services/docs.htm?docid=23429>.
- 1512 • Appendix E-2.7: Major categories and subcategories used in DGAC Analyses of WWEIA Food
 1513 Categories
- 1514 • Appendix E-2.8: Percent of total food group intake, 2009-10 for U.S. population ages 2 years and
 1515 older

1516
 1517

1518 **Question 10: What are the top foods contributing to energy intake in the U.S.**
 1519 **population?**

1520 **Source of evidence:** Data Analysis

1521

1522 **Conclusion**

1523 Seventy-five percent of total energy intake in the U.S. population comes from 16 of the 32 food sub-
 1524 categories, with mixed dishes, snacks and sweets, and beverages together contributing to more than
 1525 half (56 percent) of energy intake in the U.S. population.

1526

1527 **Implications**

1528 The foods with the highest contribution to energy intake are burgers, sandwiches, and tacos; desserts
 1529 and sweet snacks; and sugar-sweetened beverages. Given the link to energy intake, reduced
 1530 consumption of these foods and beverages or modifying the ways these foods are prepared, as well as
 1531 consumption of smaller portion sizes, may help prevent excess weight gain or may help with weight
 1532 reduction.

1533

1534 Public health strategies (e.g., programs, regulations, and policies) and product reformulation are
 1535 needed to help individuals achieve recommendations.

1536

1537 **Review of the Evidence**

1538 These conclusions were reached by examining data from the WWEIA Food Categories for the
 1539 NHANES 2009-2010 dietary survey,⁶⁵ as described in relation to question 9 (current consumption
 1540 patterns by food categories in the U.S. population).

1541

1542 The top foods contributing to energy intake in the U.S. population are concentrated in several food
 1543 categories, as shown in Figure D1.33. Three food categories account for more than half (56 percent) of
 1544 all energy consumed: 1) Mixed dishes (which include foods such as sandwiches, burgers, pizza, pasta
 1545 or rice mixed dishes, stir-fries, soups, and meat or poultry mixed dishes); 2) snacks and sweets, which
 1546 includes foods such as chips, cakes, pies, cookies, doughnuts, ice cream, and candy.), and 3) beverages
 1547 other than milk and 100% fruit juice (such as soft drinks, fruit drinks, coffee and tea, and alcoholic
 1548 beverages)

1549

1550 Examining energy intake from the more specific 32 food subcategories shows that almost half of total
 1551 energy intake comes from just 7 of these sub-categories (Table D1.12): Burgers and sandwiches (13.8
 1552 percent); desserts and sweet snacks (8.5 percent); sugar-sweetened beverages (6.5 percent); rice, pasta,
 1553 and grain-based mixed dishes (5.5 percent); chips, crackers, and savory snacks (4.6 percent); pizza (4.3
 1554 percent); and meat, poultry, and seafood mixed dishes (3.9 percent). Further examination of the 32
 1555 subcategories shows that 75 percent of all energy intake comes from the 7 subcategories previously

1556 described, plus vegetables (including starchy vegetables), alcoholic beverages, yeast breads and
 1557 tortillas, whole and 2 percent milk and yogurt, breakfast cereals and bars, poultry, and candy and
 1558 sugars.

1559

1560 As noted in Question 9, (current consumption patterns by food categories in the U.S. population), some
 1561 of the food sub-categories that provide substantial amounts of energy also provide underconsumed
 1562 food groups and nutrients. On the other hand, several of these subcategories, notably desserts and
 1563 sweet snacks and sugar-sweetened beverages, tend to contribute to energy intake with little
 1564 contribution to underconsumed food groups (see *Appendix E-2.8: Percent of total food group intake,
 1565 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories*) and
 1566 nutrients (see *Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S.
 1567 population ages 2 years and older, from WWEIA Food Categories*), but major contributions to one or
 1568 more overconsumed food components (see Question 11: What are the top foods contributing to
 1569 sodium, saturated fat, and added sugars intake in the U.S. population?)

1570

1571 Analysis of the food sources of energy by age and sex groups showed the expected higher percent of
 1572 energy from dairy among children, especially young children, but no other major differences. Analysis
 1573 by racial/ethnic groups and by income groups did not show major differences (see *Appendix 2.10:
 1574 Percent of total energy intake, 2009-2010, for age/sex groups of the U.S. population, from WWEIA
 1575 Food Categories, Appendix E-2.11: Percent of total energy intake, 2009-2010, for racial/ethnic
 1576 groups of the U.S. population, from WWEIA Food Categories, and Appendix E-2.12: Percent of
 1577 total energy intake, 2009-2010, for age/income groups of the U.S. population, from WWEIA Food
 1578 Categories*).

1579

1580

1581 ***For additional details on this body of evidence, visit:***

- 1582 • What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available
 1583 from: <http://sepri.ars.usda.gov/Services/docs.htm?docid=23429>.
- 1584 • Appendix E-2.7: Major categories and subcategories used in DGAC Analyses of WWEIA Food
 1585 Categories
- 1586 • Appendix E-2.8: Percent of total food group intake, 2009-2010, for U.S. population ages 2 years
 1587 and older, from WWEIA Food Categories
- 1588 • Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population
 1589 ages 2 years and older, from WWEIA Food Categories
- 1590 • Appendix E-2.10: Percent of total energy intake, 2009-2010, for age/sex groups of the U.S.
 1591 population, from WWEIA Food Categories
- 1592 • Appendix E-2.11: Percent of total energy intake, 2009-2010, for racial/ethnic groups of the U.S.
 1593 population, from WWEIA Food Categories
- 1594 • Appendix E-2.12: Percent of total energy intake, 2009-2010, for age/income groups of the U.S.
 1595 population, from WWEIA Food Categories

1596

1597

1598 **Question 11: What are the top foods contributing to sodium, saturated fat, and added**
1599 **sugars intake in the U.S. population?**

1600 **Source of Evidence:** Data analysis

1601

1602 **Conclusion**

1603 Mixed dishes are the largest contributor to intake of sodium (44 percent) and saturated fat (38 percent).

1604 Sodium and saturated fat have both been identified as nutrients of concern for overconsumption.

1605 Within mixed dishes, the sub-category of burgers and sandwiches is the largest contributor for both
1606 nutrients.

1607

1608 Sodium is ubiquitous in the food supply and many food categories contribute to intake.

1609 Beverages supply 47 percent of added sugars intake.

1610

1611 Snacks and sweets also are a major contributor to added sugars (31 percent) and saturated fat intake
1612 (18 percent).

1613

1614 **Implications**

1615 To decrease dietary intake from added sugars, the U.S. population should reduce consumption of
1616 sugar-sweetened beverages and of desserts and sweet snacks.

1617

1618 The U.S. population can use a variety of strategies to reduce consumption of sodium, saturated fat, and
1619 added sugars, including smaller portion sizes, reduced frequency of consumption, and recipe
1620 modification.

1621

1622 Given the ubiquity of sodium in the food supply, concerted efforts to reduce sodium in commercially
1623 prepared and processed foods, as well as encouragement of home cooking using recipes with small
1624 amounts of sodium are needed to decrease intake toward recommended levels.

1625

1626 **Review of the Evidence**

1627 These conclusions were reached by examining data from the WWEIA Food Categories for the
1628 NHANES 2009-2010 dietary survey,⁶⁵ as described in relation to Question 9 (current consumption
1629 patterns by food categories in the U.S. population).

1630

1631 The category of mixed dishes contributes substantially more saturated fat (36 percent) and sodium (43
1632 percent) to diets of the U.S. population than does any other category. Within this category, the largest
1633 share of both saturated fat (19 percent) and sodium (21 percent) comes from the subcategory of

1634 burgers, sandwiches, and tacos. The other subcategories that also contribute notable amounts of
 1635 saturated fat and sodium are pizza (approximately 6 percent for both); rice, pasta, and other grain-
 1636 based mixed dishes (5 percent and 7 percent); and meat, poultry, and seafood mixed dishes (5 percent
 1637 and 7 percent). Soups contribute a notable amount of sodium (4 percent) but less saturated fat (1
 1638 percent). (Figures D1.34 and D1.35).

1639
 1640 Other food categories contributing substantial amounts of saturated fat include snacks and sweets (18
 1641 percent), protein foods (15 percent), and dairy (13 percent). Within snacks and sweets, the subcategory
 1642 providing the largest share is desserts and sweet snacks (12 percent). Within protein foods, saturated
 1643 fat comes from meats, in general (3 percent), deli and cured meats and poultry (3 percent), poultry (3
 1644 percent), and eggs (3 percent), with seafood and nuts, seeds, and soy each contributing less than 3
 1645 percent. Within the dairy category, higher fat (whole and 2 percent) milk and yogurt (7 percent) and
 1646 cheese (4 percent) contribute the most saturated fat.

1647
 1648 Sodium is more ubiquitous in the food supply than are other nutrients, and the food categories
 1649 contributing the highest amounts of sodium include protein foods (14 percent), grains (11 percent),
 1650 vegetables (11 percent), and snacks and sweets (8 percent). Sodium is distributed throughout many
 1651 food categories and subcategories with the exception of fruits and fruit juice, which are notably low in
 1652 sodium (0.1 percent).

1653
 1654 The distribution of added sugars in foods as consumed differs from saturated fat and sodium (Figure
 1655 D1.36) The vast majority of added sugars intake comes from the major categories of beverages (not
 1656 including milk and 100% fruit juice) (47 percent) and snacks and sweets (31 percent). Grains,
 1657 including breakfast cereals and bars, contribute 8 percent, mixed dishes contribute 6 percent, and dairy,
 1658 including sweetened flavored milks and yogurts contribute only 4 percent of total added sugars intake
 1659 (see *Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2*
 1660 *years and older, from WWEIA Food Categories*).

1661
 1662 Four additional questions were examined using the WWEIA Food Categories data. They are:

1663 11a. What is the current contribution of fruit products with added sugars to intake of added sugars?

1664 11b. What is the current contribution of vegetable products with added sodium to intake of sodium?

1665 11c. What is the current contribution of refined grains to intake of added sugars, saturated fat, some
 1666 forms of polyunsaturated fat, and sodium?

1667 11d. What are the sources of caffeine from foods and beverages on the basis of age and sex categories?

1668
 1669 With regard to Question 11a, the DGAC found that:

- 1670 • Less than 1 percent of total added sugars come from fruits and 100% fruit juice foods
 1671 (including fresh, canned, frozen, dried fruit and fruit salads) (see *Appendix E-2.8: Percent of*

1672 *total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from*
1673 *WWEIA Food Categories).*

1674

1675 With regard to Question 11b, the DGAC found that:

1676 • 11 percent of total sodium comes from all vegetables (with starchy vegetables), including beans
1677 and peas, vegetable mixtures, lettuce salads, pasta sauces, and vegetable juice (see *Appendix E-*
1678 *2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2*
1679 *years and older, from WWEIA Food Categories).*

1680 • When vegetables are categorized by starchy or non-starchy, we found that:

- 1681 ○ 7 percent of total sodium comes from all vegetables, excluding starchy vegetables, and
1682 ○ 4 percent comes from starchy vegetables, including French fries and other fried potatoes,
1683 mashed potatoes, all other potatoes, corn, and other starchy vegetables.

1684

1685 With regard to Question 11c:

- 1686 • The DGAC could not directly determine the contribution of refined grains to the nutrients of
1687 interest with the currently available data. However, the food categories that make up more than
1688 90 percent of all refined grain intake (i.e., burgers, sandwiches, and tacos; breads and tortillas;
1689 rice and pasta mixed dishes; desserts and sweet snacks; pizza; chips, crackers, and savory
1690 snacks; quick breads; rice and pasta; and meat, poultry, and seafood mixed dishes) account for:
1691 ○ 28 percent of all added sugars intake
1692 ○ 47 percent of all saturated fat intake
1693 ○ 50 percent of all sodium intake

1694 (see *Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population*
1695 *ages 2 years and older, from WWEIA Food Categories* and *Appendix E-2.9: Percent of total*
1696 *energy and nutrient intake, 2009-2010 for the U.S. population ages 2 years and older, from*
1697 *WWEIA Food Categories)*

1698

1699 With regard to Question 11d, the DGAC found that (Figure D1.37):

- 1700 • Among children and adolescents, sugar-sweetened and diet beverages and coffee and tea
1701 contribute to overall caffeine intake at approximately equal levels.
1702 • Among adults, the primary sources of caffeine from all foods and beverages are coffee and tea.

1703

1704 ***For additional details on this body of evidence, visit:***

- 1705 • What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available
1706 from: <http://sepri.ars.usda.gov/Services/docs.htm?docid=23429>.
1707 • Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food
1708 Categories
1709 • Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2
1710 years and older, from WWEIA Food Categories
1711 • Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population
1712 ages 2 years and older, from WWEIA Food Categories
1713
1714

1715 **Question 12: What is the contribution of beverage types to energy intake by the U.S.**
1716 **population?**

1717 **Source of evidence:** Data analysis
1718

1719 **Conclusion**

1720

1721 Beverages contribute 19 percent of total energy intake. Of this 19 percent of energy, major sources are
1722 sugar-sweetened beverages (35 percent), milk and milk drinks (26 percent), and 100% fruit juices (10
1723 percent).
1724

1725 **Implications**

1726 The beverages that contribute the most to energy intake, particularly sugar-sweetened beverages, are
1727 those that are not nutrient dense and could be targeted for reduction. Others, like milk, fortified low-
1728 and non-fat milk, and milk beverage are good sources of key nutrients. Modifying the types of
1729 beverages consumed can reduce calories (e.g., switching from sugar-sweetened beverages to water) or
1730 improve nutrient intakes (e.g., switching from sugar-sweetened beverages to low-fat or fat-free milk).
1731 This may be an important strategy for individuals who need to reduce their energy intake and/or
1732 control their weight. Public health strategies (e.g., programs, regulations, and policies) are needed to
1733 reduce consumption of sugar-sweetened beverages.
1734

1735 Strategies are needed to encourage the U.S. population to drink water when they are thirsty. Water
1736 provides a healthy, low-cost, zero-calorie beverage option. Free, clean water should be available in
1737 public settings, as well as child care facilities, schools, worksites, publically funded athletic stadiums
1738 and arenas, transportation hubs (e.g., airports) and other community places and should be promoted in
1739 all settings where beverages are offered.
1740

1741 **Review of the Evidence**

1742 These conclusions were reached by examining data from the WWEIA Food Categories data from the
 1743 NHANES 2009-2010 dietary survey,⁶⁵ as described in relation to question 9 (current consumption
 1744 patterns by food categories in the U.S. population). For this question, a new grouping of all beverages,
 1745 including fluid milk and 100% fruit juice, was created. The conclusions and details below are based on
 1746 this category of all beverages (see *Appendix E-2.7: Major categories and subcategories used in*
 1747 *DGAC analyses of WWEIA Food Categories*).

1748
 1749 All beverages account for about one-fifth (19 percent) of total energy intake. Within that amount,
 1750 about one-third (35 percent) is from sugar-sweetened beverages, mostly soft drinks and sweetened fruit
 1751 drinks (see *Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S.*
 1752 *population ages 2 years and older, from WWEIA Food Categories*). About 20 percent of the calories
 1753 from beverages come from alcoholic beverages (21 percent), and milk and milk drinks made with
 1754 whole and 2 percent fat (18 percent). About 10 percent of the calories from beverages come from
 1755 100% fruit and vegetable juice (10 percent), fat-free and low-fat milk and milk drinks (8 percent), and
 1756 coffee and tea (8 percent) (Figure D1.38).

1757

1758 *For additional details on this body of evidence, visit:*

- 1759 • What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available
 1760 from: <http://sepri.ars.usda.gov/Services/docs.htm?docid=23429>.
- 1761 • Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food
 1762 Categories
- 1763 • Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population
 1764 ages 2 years and older, from WWEIA food categories

1765

1766

1767 **EATING BEHAVIORS—CURRENT STATUS AND TRENDS**

1768 Diet quality and energy balance directly affect health and weight status. Eating behaviors, such as
 1769 when people eat (e.g., patterns of meals and snacks, meal and snack frequency), meal skipping, and the
 1770 locations where food is obtained and consumed (e.g., retail and restaurants) influence dietary intake
 1771 and quality. Assessing and understanding eating behaviors of the U.S. population can shed light on
 1772 ways to improve food choices, weight status, and health outcomes of Americans.

1773

1774 **Question 13: What are the current status and trends in the number of daily eating**
 1775 **occasions and frequency of meal skipping? How do diet quality and energy content**
 1776 **vary based on eating occasion?**

1777 **Source of evidence:** Data analysis

1778

1779 Conclusion

1780 The majority of the U.S. population consumes three meals a day plus at least one snack. Children ages
1781 2 to 5 years are most likely to consume three meals a day and adolescent females, young adult males,
1782 non-Hispanic Blacks, Hispanics, and individuals with lower incomes are least likely to consume three
1783 meals a day. Trend data from 2005-2006 to 2009-2010 show little change in meal and snack intake
1784 patterns.

1785

1786 Breakfast tends to have a higher overall dietary quality because of its higher nutrient density compared
1787 to other meals and snacks. Adolescents and young adults are the least likely to eat breakfast. Snacks
1788 contribute about one-fourth of daily energy intake for the U.S. population and are lower in nutrients of
1789 concern relative to energy intake than are meals. For young children ages 2 to 5 years, 29 percent of
1790 daily energy is from snacks.

1791

1792 Implications

1793 Understanding eating behaviors is important for designing and implementing strategies to reduce
1794 obesity and other diet-related chronic diseases and for improving overall health. Breakfast eating is
1795 associated with more favorable nutrient intakes compared to nutrient intakes from other meals or
1796 snacks. Adolescents and young adults are the least likely to eat breakfast, and targeted promotion
1797 efforts are needed to reach these groups. For children and adolescents, the school breakfast program is
1798 an important venue for promoting breakfast consumption and efforts are needed to increase student
1799 participation rates.

1800

1801 Americans are frequent snackers and snacks contribute substantially to daily energy intake and tend to
1802 be lower than meals in shortfall nutrients of public health concern relative to energy intake. Because
1803 snack foods and beverages are readily available and accessible in multiple settings throughout the day,
1804 both population-level environmental changes and individual behavioral interventions and
1805 communications are needed to ensure that healthy choices are available in these settings and to
1806 minimize their contribution to excess energy intake.

1807

1808 Individuals with lower incomes are less likely to eat three meals a day compared to higher income
1809 individuals and low-income households are more likely to be food insecure. The federal nutrition
1810 programs play a key role in reducing food insecurity and improving nutritional health.

1811

1812 Review of the Evidence

1813 These conclusions were reached by examining existing WWEIA NHANES data tables,⁵ from
1814 NHANES 2009-2010 for current intakes, and WWEIA, NHANES 2003-2004, 2005-2006 and 2007-
1815 2008 data for trends. Respondents self-identified the specific meal or snack occasion for each food and
1816 beverage consumed.

1817

1818 **Eating Occasions: Meals.** Three meals a day is the current norm for most of the U.S. population ages
1819 2 years and older, with almost two-thirds (63 percent) eating breakfast, lunch, and dinner (Figure
1820 D1.39). However, there are differences by age, sex, racial/ethnicity group, and income level. By age
1821 group, consuming three meals a day follows a modest U-shaped curve where it is most likely for
1822 children ages 2 to 5 years (84 percent). It then declines, and reaches its lowest point during
1823 adolescence and young adulthood, and then increases with age through the adult years. Adolescent
1824 females (12 to 19 years) and young adult males (20 to 29 years) are the most likely to not eat three
1825 meals a day (49 percent). For all other age/sex groups, eating three meals a day is reported by 59 to 73
1826 percent of respondents. Eating only one meal a day is most likely for young adult males (12 percent)
1827 and adolescent females (10 percent). However, all but 1 percent of these respondents, consumed at
1828 least two or more snacks a day (Table D1.13).

1829

1830 Among the U.S. population ages 2 years and older, 15 percent do not eat breakfast, 20 percent do not
1831 eat lunch, and 7 percent do not eat dinner. Breakfast is most likely to be skipped by young adults ages
1832 20 to 29 years (28 percent of males, 22 percent of females) and adolescents (25 percent of females, 26
1833 percent of males). Breakfast skipping declines sharply with advancing age. Lunch is not eaten by 25
1834 percent of adolescent females and from 17 to 28 percent of all adult age groups (Table D1.14).

1835

1836 Non-Hispanic whites are most likely to report consuming three meals a day, across all
1837 age/sex/racial/ethnic groups, with 68 percent reporting breakfast, lunch, and dinner consumption. For
1838 non-Hispanic Blacks, slightly less than half (48 percent) consumed all three meals, and for all
1839 Hispanics, slightly more than half (52 percent). Non-Hispanic Blacks ages 12 to 19 years and 20 years
1840 and older, and Hispanics ages 12 to 19 years, were least likely to consume three meals a day (42
1841 percent, 45 percent, and 45 percent, respectively) and most likely to consume only one meal a day (18
1842 percent, 11 percent, and 10 percent).⁶⁶

1843

1844 The percent of individuals consuming three meals a day increases with higher income levels. For those
1845 below 131 percent and from 131 to 185 percent of the poverty threshold, 53 percent and 56 percent
1846 report three meals a day, while for those above 185 percent of the threshold, 70 percent report three
1847 meals a day. For lower income individuals, the lower number of meals consumed per day is much
1848 more evident for older children and adults. Among children ages 2 to 5 years in the three income
1849 groupings, 81 percent, 82 percent, and 88 percent, respectively, report consuming three meals a day,
1850 while for adults ages 20 years and older, the corresponding percentages are 48 percent, 54 percent, and
1851 70 percent, respectively.⁶⁷

1852

1853 **Eating Occasions: Snacks.** Nearly all of the U.S. population ages 2 years and older consume at least
1854 one snack a day (96 percent). The most common snacking pattern for most age, sex, racial/ethnic and
1855 income groups is two to three snacks per day. Females and males ages 70 years and older are most
1856 likely to report eating one or fewer snacks per day (26 percent), and children ages 2 to 5 years are the

1857 least likely (10 percent). Children ages 2 to 5 years are most likely of any age group to report four or
 1858 more snacks per day, across all racial/ethnic groups.⁶⁸

1859

1860 The number of individuals reporting one or fewer snacks per day is highest (25 percent) for those
 1861 below 131 percent of the poverty threshold, and lowest (17 percent) for those above 185 percent of the
 1862 threshold. Consumption of four or more snacks per day is lowest (25 percent) for those below 131
 1863 percent of the poverty threshold and highest (35 percent) for those above 185 percent of the threshold.
 1864 However, for all income groups, 2 to 3 snacks per day is the modal number and similar across income
 1865 groups (51 percent, 48 percent, 48 percent).⁶⁷

1866

1867 **Trends.** Trend data from NHANES from 2005-2006 to 2009-2010 show little change in number of
 1868 daily eating occasions or frequency of meal skipping (Table D1.15).

1869

1870 **Diet Quality and Energy content by Eating Occasion.** For this analysis, diet quality is defined as a
 1871 comparison of nutrient or food group content to energy content of a specified set of foods or beverages.
 1872 In this question, diet quality compares the proportion of total nutrient intake at a given eating occasion
 1873 to the proportion of energy intake at that eating occasion.

1874

1875 This analysis is summarized in Figure D1.40 and described below. In looking at this Figure, it should
 1876 be noted that percent of total intake of nutrients of concern are shown in comparison to percent of total
 1877 energy. If a nutrient is above the energy line, the meal/snack is a relatively higher source of that
 1878 nutrient. If it is below the energy line, it is a relatively lower source.

1879

1880 Breakfast has a higher overall diet quality compared to lunch, dinner or snacks. Breakfast consists of
 1881 15 to 20 percent of the day's total energy intake (Table D1.16) but has a higher percent of nutrients.
 1882 For all the shortfall nutrients of public health concern (fiber, folate, vitamin D, calcium, iron, and
 1883 potassium), a higher percent of the day's total intake was consumed compared to the percent of energy
 1884 consumed (Figure D1.40)

1885

1886 Among the U.S. population ages 2 years and older, about one fourth (24 percent) of daily energy intake
 1887 is consumed at lunch and about one-third (35 percent) is consumed at dinner (Table D1.16). In terms
 1888 of dietary quality, lunch is neutral, with similar percents of total nutrients and energy intakes for most
 1889 nutrients. Dinner, which provides the greatest amount of daily total energy intake, has a higher percent
 1890 of fiber, and potassium in comparison to percent energy, but calcium and several other nutrients are
 1891 lower in comparison to percent energy. Sodium and saturated fat are higher as a percent of their total
 1892 intakes than is energy intake. Further, the percent of total daily intake of sodium and saturated fat
 1893 consumed at dinner is higher compared to other meals and snacks (Figure D1.40).

1894

1895 About one-fourth (24 percent) of daily energy intake comes from snacks. For young children ages 2 to
 1896 5 years, 29 percent of daily energy is from snacks (Table D1.17). Snacks provide the lowest percent of

1897 key nutrients (protein, iron, vitamin D, fiber, and potassium) relative to the percent of energy provided.
 1898 Snacks provide 42 percent of the daily intake of added sugars. A lower percent of total sodium than of
 1899 energy is provided by snacks. Snacks provide roughly the same percent of total intake of calcium as
 1900 they do energy. This is also true of saturated fat for females (Table D1.17).

1901

1902 ***For additional details on this body of evidence, visit:***

- 1903 • Percent of the U.S. population consuming or skipping meals and snacks, 2001-2002, 2005-2006,
 1904 2007-2008, and 2009-2010 by age/sex groups, race/ethnicity, and percent of the poverty threshold.
 1905 Available from: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>.
- 1906 • Percent of total energy and nutrient intake by meal/snack, 2001-2002, 2005-2006, 2007-2008 and
 1907 2009-2010 by age/sex groups, race/ethnicity, and percent of the poverty threshold. Available from:
 1908 <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>.

1909

1910

1911 **Question 14: What are the current status and trends in the location of meal and snack**
 1912 **consumption and sources of food and beverages consumed at home and away from**
 1913 **home? How do diet quality and energy content vary based on the food and beverage**
 1914 **source?**

1915 **Source of evidence:** Data analysis

1916

1917 **Conclusion**

1918 About two-thirds of the calories consumed by the U.S. population are purchased at a store (69 percent),
 1919 such as a grocery store or supermarket, and consumed in the home. The percent of calories eaten away
 1920 from home (32 percent) has remained about the same since 2003-2004.

1921

1922 Food group and nutrient quality as measured by the Healthy Eating Index (HEI) vary by where food is
 1923 obtained. Despite this, no matter where the food is obtained, diet quality of the U.S. populations does
 1924 not meet recommendations for fruit, vegetables, dairy, whole grains, and exceeds recommendations for
 1925 sodium, saturated fats, refined grains, solid fats, and added sugars.

1926

1927 **Implications**

1928 The overall diet quality of the U.S. population’s dietary patterns, regardless of where the food is
 1929 purchased and eaten, is of major public health concern. Given that fruit, vegetables, dairy, and whole
 1930 grains are consumed in less than recommended amounts and that sodium, saturated fats, refined grains,
 1931 solid fats, and added sugars exceed recommended levels, urgent action is needed at individual and
 1932 population levels to alter food purchasing and consumption habits.

1933

1934 Efforts are needed by the food industry and food retail (food stores and restaurants) sectors to market
 1935 and promote healthy foods. The general public needs to be encouraged to purchase these healthier

1936 options. Making healthy options the default choice in restaurants (e.g., fat-free/low-fat milk instead of
 1937 sugar-sweetened beverages, and fruit and non-fried vegetables in Children’s Meals, whole wheat buns
 1938 instead of refined grain buns for sandwich meals) would facilitate the consumption of more nutrient
 1939 dense diets. Food manufacturers and restaurants should reformulate foods to make them lower in
 1940 overconsumed nutrients (sodium, added sugars and saturated fat) and calories and higher in whole
 1941 grains, fruits and vegetables.

1942
 1943 In addition, Federal regulations for food labeling need to be updated. Food labels are an important tool
 1944 to enable the public to follow the Dietary Guidelines and to make healthy food choices. They provide
 1945 consumers with quick, easy to use information about the food they are purchasing. They also lead food
 1946 companies to reformulate their food products to meet consumer demand. As recently proposed by the
 1947 FDA, updates are needed in the Nutrition Facts label on packaged foods to emphasize calories, serving
 1948 sizes, and nutrients of concern (including overconsumed nutrients such as sodium). Consumers also
 1949 may benefit from a standardized Front of Pack label that gives clear guidance such as proposed by the
 1950 IOM panel on FOP labeling.⁶⁹

1951
 1952 In addition to regulatory, policy, environmental and organizational changes, individual behavioral
 1953 strategies are also needed to help Americans improve dietary behaviors. Comprehensive lifestyle
 1954 interventions in a variety of settings and nutrition counseling by professionals in health care settings
 1955 can modify dietary behaviors and improve health outcomes.

1956 1957 **Review of the Evidence**

1958 This conclusion was reached by examining a new analysis of WWEIA, NHANES food intake data,
 1959 from WWEIA NHANES 2009-2010 for current status, and WWEIA NHANES 2003-2004, 2005-2006
 1960 and 2007-2008 for trends (see *Appendix E-2.13: Percent of energy intake from major points of*
 1961 *purchase and location of eating, 2003-2004, 2005-2006, 2007-2008, 2009-2010, for the U.S.*
 1962 *population ages 2 years and older* and *Appendix E-2.14: Food group and nutrient content of foods*
 1963 *per 1000 calories obtained from major points of purchase, 2003-2004, 2005-2006, 2007-2008, 2009-*
 1964 *2010, for the U.S. population ages 2 years and older*). This analysis was requested by the DGAC to
 1965 answer the question. In addition, the DGAC reviewed the ERS publication *Nutritional Quality of Food*
 1966 *Prepared at Home and Away from Home, 1977-2008*⁷⁰ to ascertain longer-term trends.

1967
 1968 Respondents self-identified the food source (point of purchase) for each food or beverage they
 1969 reported. For this analysis, food sources were grouped into the following categories: stores (grocery,
 1970 supermarket, convenience/corner stores), full-service restaurants (defined as table service restaurants),
 1971 quick-serve restaurants (includes fast food, counter service, and vending machines), school (includes
 1972 child care). The location of eating, either at home or away from home, also was examined (Figure
 1973 D1.41).

1974

1975 Americans increased their away-from-home share of caloric intake from 18 percent in 1977-1978 to 32
 1976 percent in 2005-2008, mainly from full service and fast food restaurants.⁷⁰ The percent of calories
 1977 eaten away from home has remained roughly the same since 2003-2004. In 2009-2010, 69 percent of
 1978 calories consumed by Americans were purchased from a store and 58 percent were eaten at home. This
 1979 is about the same percent from 2003-2008 (Figure D1.41).

1980
 1981 Diet quality was assessed using a density approach expressed as the amount of food group or nutrient
 1982 per 1000 calories consumed, for each source from which food is obtained. The point of purchase (e.g.,
 1983 food store) is used as a proxy for where the food is consumed (e.g., home) because most food from
 1984 stores are consumed at home, and most foods from other points of purchase are consumed away from
 1985 home. Diet quality for a food group or nutrient for each food source obtained/consumed was then
 1986 compared to the standard for a optimal HEI score per 1000 calories.⁷¹ For saturated fat intake, the
 1987 amount from each source was compared to the 2010 Dietary Guidelines limit for saturated fat intake.

1988
 1989 **Fruit.** Fruit group density (cups per 1000 calories) is well below the HEI standard regardless of where
 1990 the food is obtained or consumed. Amounts of fruit obtained and consumed differ by source, with full
 1991 service and fast-food restaurants providing much less fruit per 1000 calories compared to other
 1992 sources. This changed little from 2003-2004 to 2009-2010. Amount of fruit per 1000 calories is highest
 1993 from schools/day care, and increased from 2003-2004 to 2009-2010, especially from 2007-2008 on
 1994 (Figure D1.42).

1995
 1996 **Vegetables.** Density for vegetables (cups per 1000 calories) falls below recommended intakes
 1997 regardless of where food is obtained (Figure D1.43). Amounts of total vegetables and the starchy and
 1998 other vegetable subgroups are shown in Figures D1.43 and D1.44. (Other vegetables are those not in
 1999 the dark green, red orange, or starchy subgroups, such as green beans, iceberg lettuce, onions, cabbage,
 2000 cucumbers.) Amounts of total vegetables and other vegetables per 1000 calorie are highest for
 2001 restaurants, especially full service restaurants, with a slight downward trend from 2007-2008 to 2009-
 2002 2010 (Figures D1.43 and D1.44). Amounts of total vegetables and starchy vegetables per 1000 calories
 2003 from schools/daycare show a suggestive decrease in 2009-2010 compared to earlier years. Density for
 2004 all vegetable subgroups by source for 2003-2004 through 2009-2010 are listed in Table D1.18.

2005
 2006 **Dairy.** Amounts of total dairy products (fluid milk, cheese, and yogurt) are highest from schools/day
 2007 care sources and are above the HEI standard, with an increase from 2007-2008. Amounts from other
 2008 sources are far below recommendations (Figure D1.45).

2009
 2010 **Whole and refined grains.** Whole grain density per 1000 calories is far below the HEI standard and is
 2011 low for all food sources with little change since 2003-2004. On the other hand, refined grains exceed
 2012 the HEI limit for all food sources, with the highest amount coming from quick serve restaurants
 2013 (Figure D1.46).

2014

2015 **Protein foods.** Amounts of total protein foods per 1000 calories are above the HEI standard for full
 2016 service restaurants and fast food restaurants (Figure D1.47).

2017
 2018 **Sodium.** Amounts of sodium per 1000 calories are well above the HEI limit and do not differ greatly
 2019 across sources. However, the density from full service and fast food restaurants are somewhat higher
 2020 than from stores. There has been little change from 2003-2004 to 2009-2010 (Figure D1.48).

2021
 2022 **Saturated fats.** Amounts of saturated fat per 1000 calories is well above the Dietary Guidelines limit
 2023 and do not differ greatly across sources. However, the density from fast food restaurants is somewhat
 2024 higher than from stores. There has been little change from 2003-2004 to 2009-2010 (Figure D1.49).

2025
 2026 **Empty calories.** (defined as the total calories from solid fats and added sugars). Empty calories are
 2027 well above the HEI limit (190 calories per 1000 calories) for all food sources, with the highest amount
 2028 from fast food restaurants, but no large differences among sources. Empty calories have trended
 2029 downward since 2003-2004 (Figure D1.50). The HEI does not have a separate HEI standard for added
 2030 sugars and solid fats. Both added sugars and solid fats have decreased since 2003-2004. (Figures
 2031 D1.51, D1.52) The highest amounts of added sugars are obtained from stores and the highest amounts
 2032 of solid fats are obtained from fast food restaurants.

2033
 2034 **Food group density by age group.** For children ages 2 to 5 years, fruit group density per 1000
 2035 calories from schools and stores reaches the HEI standard. School foods provide the highest fruit
 2036 density among all food sources for 6-11 year olds, with an increase since 2007-2008. All other age
 2037 groups do not reach the HEI standard for fruit from any source, although the store location is
 2038 consistently the top source for adults. Vegetable density from full service restaurants reaches the HEI
 2039 standard for ages 51-70 and 71 years and older. All sources of vegetables are below the standard for
 2040 children, adolescents and adults under age 50. Dairy product density from child care and stores meet
 2041 the HEI standard for children ages 2-5 and from schools for children ages 6-19. School foods provide
 2042 the highest dairy product density among all food sources in children's diets. For school age children
 2043 and adolescents, school foods are the only food source that meets the recommended amount of dairy
 2044 products. Among adults, dairy product density is low for all sources. For children ages 6-11, there is a
 2045 difference in the added sugars density by source, with schools having less added sugars per 1000
 2046 calories than other sources. This difference is not as clear for younger children or adolescents. For
 2047 adults the highest amount of added sugars per 1000 calories is from stores. For most age groups, there
 2048 is a slight downward trend, especially in the density of added sugars from stores (see *Appendix E-*
 2049 *2.15: Amount of key nutrients and food groups by age group per 1000 calories from each point of*
 2050 *purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010*).

2051
 2052 ***For additional details on this body of evidence, visit:***

- 2053 • Appendix E-2.13: Percent of energy intake from major points of purchase and location of eating,
 2054 2003-2004, 2005-2006, 2007-2008, and 2009-2010, for the U.S. population ages 2 years and older

- 2055 • Appendix E-2.14: Food group and nutrient content of foods per 1000 calories obtained from major
2056 points of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010, for the U.S. population
2057 ages 2 years and older
- 2058 • Appendix E-2.15: Amount of key nutrients and food groups by age group per 1000 calories from
2059 each major point of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010
- 2060 • ERS report, Nutritional Quality of Food Prepared at Home and Away from Home, 1977-2008.
2061 Available from: [http://www.ers.usda.gov/publications/eib-economic-information-](http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib105.aspx)
2062 [bulletin/eib105.aspx](http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib105.aspx).
2063

2064 **PREVALENCE OF HEALTH CONDITIONS AND TRENDS**

2065 Preventable, diet- and lifestyle-related chronic diseases, including high blood pressure, CVD, type 2
 2066 diabetes, and certain cancers, contribute to the high and rising costs of U.S. health care. Adults with
 2067 overweight or obesity frequently have co-morbid conditions and higher chronic disease risk profiles
 2068 that contribute substantially to higher health care costs. These health problems are persistent in the
 2069 population and pose major public health concerns. Increasing rates of overweight and obesity among
 2070 American youth have resulted in rising rates of CVD risk factors, including borderline high blood
 2071 pressure and diabetes, in this population. Health disparities in risk profiles and disease rates are evident
 2072 across racial, ethnic, and income strata. In a new health care and public health vision, prevention of
 2073 chronic diseases and other lifestyle-related health problems would become a major focus. Examining
 2074 the status and trends in these health conditions provides a framework for discussing their relationship
 2075 to dietary intake and lifestyle factors and can help in identifying evidence-based strategies for
 2076 prevention.

2077

2078 **Question15: What is the current prevalence of overweight/obesity and distribution of**
 2079 **body weight, BMI, and abdominal obesity in the U.S. population and in specific age,**
 2080 **sex, racial/ethnic, and income groups? What are the trends in prevalence?**

2081 **Source of evidence:** Data analysis

2082

2083 **Conclusion**

2084 The current rates of overweight and obesity are extremely high among children, adolescents, and
 2085 adults. These high rates have persisted for more than 25 years.

2086

2087 Overall, 65 percent of adult females and 70 percent of adult males are overweight or obese, and rates
 2088 are highest in adults ages 40 years and older. Rates of overweight and obesity in adults vary by age and
 2089 race/ethnicity.

- 2090 • Overweight (excluding obesity) is most prevalent in those ages 40 years and older, and in
 2091 Hispanic American adults.
- 2092 • Obesity is most prevalent in those 40 years of age or older and in African American adults.
 2093 Obesity is least prevalent in adults with highest incomes (400+ percent the poverty threshold).

2094

2095 Abdominal obesity is present in U.S. adults of all ages, increases with age, and varies by sex and
 2096 race/ethnicity.

- 2097 • Abdominal obesity rates are highest in individuals ages 60 years and older, and are higher in
 2098 women than men at all ages.

- 2099 • In men, abdominal obesity rates are slightly higher among non-Hispanic whites than Mexican
2100 Americans or African Americans. In women, abdominal obesity rates are lower in non-
2101 Hispanic whites than in Mexican Americans or African Americans.

2102

2103 Nearly one in three youth (31 percent), ages 2 to 19 years, is now overweight (85th-94th percentile) or
2104 obese (\geq 95th percentile) and these rates vary by age and ethnicity.

- 2105 • In youth ages 2 to 19 years, obesity prevalence increases with age, and the age category with
2106 the highest prevalence is 12-19 year olds.

- 2107 • In youth ages 2 to 19 years, the race categories with the highest prevalence of obesity are
2108 African Americans and Hispanics.

2109

2110 **Implications**

2111 The persistent high levels of overweight and obesity require urgent population- and individual-level
2112 strategies across multiple settings, including health care, communities, schools, worksites, and
2113 families.

2114

2115 Comprehensive lifestyle interventions and evidence-based dietary interventions for weight
2116 management in individuals and small groups should be developed and implemented by trained
2117 interventionists and professional nutrition service providers in healthcare settings as well as in
2118 community locations, including public health facilities and worksites.

2119

2120 Quality of care standards in health care settings should include the provision and impact of preventive
2121 nutrition services provided by multidisciplinary teams of trained interventionists, as appropriate, and
2122 nutrition professionals. Incentives should be offered to providers and systems to develop preventive
2123 services.

2124

2125 The public should be encouraged to monitor their body weight and engage with their health care
2126 providers at least annually to assess their body weight and BMI. As appropriate, providers should use
2127 evidence-based approaches aimed at achieving and maintaining healthy body weight. Health care
2128 providers should encourage achieving and maintaining a healthy weight through healthy eating and
2129 physical activity behaviors.

2130

2131 The persistent high rates of obesity across the lifespan show the limited impact of our efforts to date.
2132 Accelerating progress in reversing obesity trends will require a more targeted, comprehensive, and
2133 coordinated strategy and a renewed commitment and action for sustained, large-scale, integrated multi-
2134 sectoral and cross-sectoral collaborations. Government at local, state, and national levels, the health
2135 care system, schools, worksites, community organizations, businesses, and the food industry all have
2136 critical roles in developing creative and effective solutions.

2137

2138 Behavioral change at the individual level is important. However, policy interventions that make
2139 healthy dietary and activity choices easier, more routine, and affordable and that reduce unhealthy
2140 options are likely to achieve population-wide benefits.

2141

2142 Age-appropriate nutrition and food preparation education should be a mandatory part of primary and
2143 secondary school curricula.

2144

2145 **Review of the Evidence**

2146 To reach these conclusions, the DGAC examined evidence from NHANES 2009-2012, and additional
2147 survey years including 1988-1994 to 2011-2012 for trends data. These data are available in summary
2148 NHANES data table format on the CDC website, in published peer-reviewed articles by CDC,⁷²⁻⁷⁴ and
2149 in analyses requested by the DGAC and provided by CDC/NCHS (see *Appendix E-2.16: Body mass
2150 index, adults ages 20 years and older, NHANES 2009-2012* and *Appendix E-2.17: Body mass index,
2151 children and adolescents ages 2-19 years, NHANES 2009-2012*).

2152

2153 The prevalence rates of overweight and obesity among U.S. adults have been extremely high for the
2154 past 25 years and appear to be at record high levels in women and to have plateaued at near record high
2155 levels in men (Figure D1.53). In 2009-2012, combined rates of overweight and obesity in adult men,
2156 ages 20 years and older, were 72.6 percent (38.1 percent for overweight and 34.5 percent for obesity)
2157 and 64.8 percent (28.8 percent for overweight and 36 percent for obesity) in women (Table D1.19).
2158 Rates of overweight and obesity in adults vary by age and ethnicity and are most pronounced in adults
2159 ages 40 years and older and in Hispanic and African American adults (Table D1.19).

2160

2161 Overweight affects 29.5 percent of adults ages 20 to 39 years, 35.9 percent of adults ages 40 to 59
2162 years, and 35.7 percent of adults ages 60 years and older, while obesity affects 31.5 percent of adults
2163 ages 20 to 39 years, 38 percent of those ages 40 to 59 years, and 37.5 percent of those ages 60 years
2164 and older (Table D1.19).

2165

2166 Overweight affects 31.7 percent of adult African American men and 24.5 percent of adult African
2167 American women, while obesity affects 37.9 percent of adult African American men and 57.5 percent
2168 of adult African American women. Among adult Hispanic men, overweight affects 41.5 percent and
2169 obesity affects 38.5 percent, and among adult Hispanic women, overweight affects 33.5 percent and
2170 obesity affects 43 percent (Table D1.19).

2171

2172 Obesity is least prevalent (about 31 percent) in adults ages 20 years and older with highest incomes
2173 (400 + percent the poverty threshold) in 2007-2010 (Table D1.20), while affecting 37.2 percent of
2174 those with incomes below 100 percent of the poverty threshold, 37.3 percent of those with incomes
2175 from 100 percent to 199 percent of the poverty threshold, and 36.8 percent of those with incomes from

2176 200 percent to 399 percent of the poverty threshold (Table D1.20). Across all income strata, combined
 2177 rates of overweight and obesity and particularly obesity rates have risen over the past 25 years.

2178
 2179 Abdominal obesity, as measured by waist circumference (WC), and defined as WC more than 102 cm
 2180 in men and more than 88 cm in women, is a risk factor for CVD and diabetes.⁶ Abdominal obesity is
 2181 prevalent in U.S. adults of all ages and varies by age and sex. In 2011-2012, overall rates of abdominal
 2182 obesity were about 54 percent in adults ages 20 years and older, with a prevalence of about 44 percent
 2183 in adult men and 65 percent in adult women (Table D1.21). Data from the NHANES 2007-2008 survey
 2184 shows that men ages 20 to 39 years have the lowest rates of abdominal obesity (28.5 percent)
 2185 compared to men ages 40 to 59 years (49.4 percent) and those ages 60 years and older (60.4 percent)
 2186 (Table D1.21). Women ages 60 years and older have the highest rates of abdominal obesity (73.8
 2187 percent) compared to women ages 40 to 59 and 20 to 39 years (65.5 percent and 51.3 percent,
 2188 respectively). Data from the 2011-2012 survey show that the highest prevalence of abdominal obesity
 2189 among men is in non-Hispanic white men (44.5 percent), followed by Mexican American men (43.2
 2190 percent) and African American men (41.5 percent), while the highest prevalence among women is in
 2191 African American women (75.9 percent), followed by Mexican American (71.6 percent) and non-
 2192 Hispanic white women (63.3 percent) (Table D1.21). For 2007-2010, the prevalence of abdominal
 2193 obesity is very high in obese adults ages 18 years and older (97 percent), and overweight adults (57
 2194 percent), compared to normal/underweight adults (8 percent).⁷⁵ Since 1999 rates of abdominal obesity
 2195 have risen in all age and racial strata of both adult males and females (Table D1.21).

2196
 2197 After increasing from the 1980s until about 2004, rates of overweight and obesity in children and
 2198 adolescents ages 2 to 19 years have since remained at very high levels (Figure D1.54). A significant
 2199 decrease in obesity among children ages 2 to 5 years old was observed in an analysis comparing the
 2200 survey data from 2003-2004 (13.9 percent) to 2011-2012 (8.4 percent).⁷⁴ However, it is not clear
 2201 whether this comparison of only two time periods reflects an actual downward trend. Currently, 14.9
 2202 percent of boys ages 2 to 19 years are overweight (85th to 94th percentile) and 17.6 percent are obese
 2203 (95th percentile and greater); rates in girls ages 2 to 19 years are 14.9 percent and 16.1 percent,
 2204 respectively. Furthermore, rates of obesity in youth increase with age and vary by ethnicity, with
 2205 obesity found in 22.1 percent of African American and 21.8 percent of Hispanic Americans ages 2 to
 2206 19 years (Table D1.22).

2207
 2208 ***For additional details on this body of evidence, visit:***

- 2209 • Appendix E-2.16: Body mass index, adults ages 20 years and older, NHANES 2009-2012
- 2210 • Appendix E-2.17: Body mass index, children and adolescents ages 2-19 years, NHANES 2009-
 2211 2012

2213 **Question 16: What is the relative prevalence of metabolic and cardiovascular risk**
 2214 **factors (i.e., blood pressure, blood lipids, and diabetes) by BMI/body weight/waist**
 2215 **circumference (abdominal obesity) in the U.S. population and specific population**
 2216 **groups?**

2217 **Source of evidence:** Data analysis

2218

2219 **Conclusion**

2220 Approximately 50 percent of adults who are normal weight have at least one cardiometabolic risk
 2221 factor. Approximately 70 percent of adults who are overweight and 75 percent of those who are obese
 2222 have one or more cardiometabolic risk factors.

2223

2224 Rates of elevated blood pressure, adverse blood lipid profiles (i.e., low high density lipoprotein
 2225 cholesterol [HDL-C], high low density lipoprotein cholesterol [LDL-C], and high triglycerides), and
 2226 diabetes are highest in adults with elevated abdominal obesity (waist circumference greater than 102
 2227 cm in men, greater than 88 cm in women).

2228

2229 Ninety-three percent of the children with type 2 diabetes are ages 12 to 19 years and 90 percent of
 2230 these children with type 2 diabetes are overweight or obese. In children with type 2 diabetes, the
 2231 prevalence of obesity is higher in African Americans, followed by American Indians and Hispanics,
 2232 compared to non-Hispanic whites or Asian Pacific Islander youth.

2233 Dyslipidemia and rates of borderline high blood pressure vary by weight status in boys and girls; rates
 2234 are particularly high in obese boys.

2235 Nearly three-fourths of the overweight or obese populations have at least one cardiometabolic risk
 2236 factor.

2237

2238 **Implications**

2239 The rates of cardiometabolic risk factors in adult Americans are extremely high and reflect the high
 2240 rates of population overweight and obesity. Many adults have personal health profiles in which
 2241 multiple metabolic risk factors co-exist and substantially increase risks for coronary heart disease,
 2242 hypertension and stroke, diabetes, and other obesity-related co-morbidities. These are the most costly
 2243 health problems in the Nation today and they can be prevented or better managed with intensive,
 2244 comprehensive, and evidence-based lifestyle interventions carried out by multidisciplinary teams of
 2245 trained professionals or through medical nutrition therapy provided by registered dietitians or
 2246 nutritionists (AHA/ACC/TOS).² Program plans and interventions needed to confront the nation's
 2247 obesity epidemic and its devastating metabolic consequences. A shift in the healthcare paradigm
 2248 toward prevention is critical. Nutrition and lifestyle services for obesity prevention and weight
 2249 management should be expanded and integrated. As part of this approach, quality of care guidelines
 2250 need to be revised to incentivize the provision of personalized lifestyle and nutrition interventions to

2251 combat obesity and obesity-related chronic diseases and their metabolic risk factors and co-
 2252 morbidities. As emphasized in *Part D. Chapter 3: Individual Diet and Physical Activity Behavior*
 2253 *Change* and *Part D. Chapter 4: Food Environment and Settings*, the most effective approach to
 2254 preventing and treating overweight and obesity in our nation across the lifespan requires both
 2255 individual and population-based, environmental strategies. Initiatives in health care and public health
 2256 and other government sectors should be complemented with collaborative approaches in retail,
 2257 educational, and social service and agricultural settings to make the long-term adoption of healthy
 2258 nutrition and lifestyle behavior not only feasible but normative.

2259
 2260 The high rates of overweight and obesity in youth and their concomitant cardiometabolic risk factors
 2261 require early preventive interventions at individual and population levels. Evidence-based strategies in
 2262 health and public health settings also should be implemented and complemented by environmental
 2263 approaches across wide-ranging sectors to reverse these priority health problems.

2264 2265 **Review of the Evidence**

2266 To reach these conclusions, the DGAC examined evidence from NHANES 2007-2010 and 2009-2012
 2267 data and SEARCH for Diabetes in Youth Study (SEARCH). These data were available in published
 2268 peer-reviewed articles by CDC,⁷⁶ or SEARCH⁷⁷ authors and in analyses requested by the DGAC and
 2269 provided by CDC/NCHS (see *Appendix E-2.18: Total cholesterol and high density lipoprotein*
 2270 *cholesterol (HDL), adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.19: Low*
 2271 *density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20 years and older, NHANES*
 2272 *2009-2012, Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older,*
 2273 *NHANES 2009-2012, Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES*
 2274 *2009-2012, Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and*
 2275 *non-HDL-cholesterol, children and adolescents ages 6–19 years, NHANES 2009-2012, Appendix E-*
 2276 *2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years,*
 2277 *NHANES 2009-2012, Appendix E-2.24: Prevalence of high and borderline high blood pressure*
 2278 *(BP), children and adolescents ages 8-17 years, NHANES 2009-2012).*

2279
 2280 In U.S. adults ages 18 years and older, weight status is related to prevalent CVD risk. About two-thirds
 2281 (66.6 percent) of U.S. adults, including more than half (56.1 percent) of normal weight adults (BMI
 2282 18.5-<25 kg/m²), have one or more CVD risk factors (including type I and type II diabetes,
 2283 hypertension, or dyslipidemia, or self-reported smoking) (Figure D1.55). About 70 percent (69.6
 2284 percent) of adults who are overweight (BMI 25-<30 kg/m²) have at least one or more CVD risk factors,
 2285 making them candidates for preventive weight management interventions, according to expert
 2286 guidelines established by the American College of Cardiology, American Heart Association, and The
 2287 Obesity Society for preventative weight management (see *Part D. Chapter 2: Dietary Patterns, Foods*
 2288 *and Nutrients, and Health Outcomes*). Furthermore, more than one-quarter (27.8 percent) have two or
 2289 more CVD risk factors (Figure D1.55). About three-quarters (74.6 percent) of adults who are obese
 2290 (BMI ≥30 kg/m²) have one or more CVD risk factors and about 39 percent have two or more CVD risk

2291 factors (Figure D1.55). Cardio-metabolic risk factors also are substantially more prevalent in adult men
2292 and women who have abdominal obesity (Table D1.23).

2293

2294 In terms of plasma lipids, the prevalence of low HDL-C (<40 mg/dl), high LDL-C (\geq 160 mg/dl), and
2295 high triglycerides (\geq 200 mg/dl) is highest in obese adults (ages 20 years and older) compared to
2296 normal weight adults (Table D1.23). Similar patterns are observed in those who are overweight
2297 compared to normal weight adults (Table D1.23). These lipid profiles also are highest in men with
2298 abdominal obesity (> 102 cm) or women (>88 cm). (Table D1.23). High total cholesterol (\geq 200
2299 mg/dl), low HDL-C (<40 mg/dl), and high triglycerides (\geq 130 mg/dl) also are most prevalent in obese
2300 compared to overweight or normal weight children and adolescents (Table D1.24). There does not
2301 appear to be a difference in the prevalence of high LDL-C (\geq 130 mg/dl) by weight status in children
2302 and adolescents (Table D1.24).

2303

2304 In adults ages 18 years and older, rates of elevated blood pressure (defined as having measured systolic
2305 pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking
2306 antihypertensive medication) are highest with obesity (39.2 percent) compared to normal weight (20
2307 percent) or overweight (26.4 percent). It is also highest in those with elevated waist circumferences
2308 (men > 102cm (37.2 percent vs 23.3 percent; and > 88 cm in women (32.9 percent vs 17.8 percent)
2309 (Table D1.23). Similar to adults, the rate of borderline high blood pressure (defined as a systolic or
2310 diastolic blood pressure \geq 90th percentile but < 95th percentile or blood pressure levels \geq 120/80 mm
2311 Hg) in youth ages 8 to 17 years was highest in with obesity (16.2 percent) compared to those who are
2312 normal weight (5.4 percent) or overweight (10.9 percent) (Table D1.25). Diabetes in adults ages 20
2313 years and above also increases with body mass index from 5.5 percent in those who are of normal
2314 weight, to 9 percent in overweight and 20.3 percent in obese adults and is more prevalent in those with
2315 abdominal obesity (men > 102cm (19.6 percent vs 8.3 percent); and > 88 cm in women (13.9 percent
2316 vs 2.6 percent) (Table D1.23).

2317

2318 Data from 2001 to 2004 in children (ages 3 to 19 years) participating in the SEARCH for Diabetes in
2319 Youth Study (SEARCH) show that 93 percent of youth with type 2 diabetes are ages 12 to 19 years.
2320 The prevalence of obesity among youth with type 2 diabetes is 79.4 percent and an additional 10.4
2321 percent are overweight (Table D1.26). The percentage of overweight among youth with type 2 diabetes
2322 is not significantly different than rates in U.S. youth who do not have type 2 diabetes.⁷⁷ However, the
2323 prevalence of obesity among youth with type 2 diabetes (79.4 percent) is much higher than in U.S.
2324 youth without type 2 diabetes (16.9 percent) (Table D1.26). The prevalence of obesity in those with
2325 type 2 diabetes was higher in African Americans (91.1 percent), followed by American Indians (88
2326 percent), and Hispanics (75 percent) in comparison to non-Hispanic white or Asian Pacific Islander
2327 youths (about 68 percent for each) (Table D1.26).

2328

2329 ***For additional details on this body of evidence, visit:***

- 2330 • Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adult ages
2331 20 years and older, NHANES 2009 -2012
- 2332 • Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages
2333 20 years and older, NHANES 2009-2012
- 2334 • Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES
2335 2009-2012
- 2336 • Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012
- 2337 • Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-
2338 cholesterol, children and adolescents ages 6-19 years, NHANES 2009-2012
- 2339 • Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents
2340 ages 12-19 years, NHANES 2009-2012
- 2341 • Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and
2342 adolescents ages 8-17 years, NHANES 2009-2012

2343
2344

2345 **Question 17: What are the current rates of nutrition-related health outcomes (i.e.,**
2346 **incidence of and mortality from cancer [breast, lung, colorectal, prostate] and**
2347 **prevalence of CVD, high blood pressure, diabetes, bone health, congenital anomalies,**
2348 **neurological and psychological illness) in the overall U.S. population?**

2349 **Source of evidence:** Data analysis

2350

2351 **Conclusion**

2352 Adults have high rates of nutrition-related chronic diseases, including high blood pressure, CVD,
2353 diabetes, and various forms of cancer. Children and adolescents also have nutrition-related chronic
2354 diseases, including borderline high blood pressure and type 2 diabetes. At all ages, rates of chronic
2355 disease risk are linked to overweight and obesity. The rates of these chronic diseases vary by
2356 race/ethnicity and income status. Prevalence of osteoporosis and of low bone mass increases with age,
2357 particularly in post-menopausal women. Among the less common health outcomes:

- 2358 • Nutrition-related neurological and psychological conditions are a growing concern.
- 2359 • Congenital anomalies are a relatively rare, but important pregnancy outcome.

2360

2361 **Implications**

2362 Given the high rates of nutrition-related chronic diseases in the adult population and rising rates in
2363 youth, it is imperative to develop prevention policies and programs that target all age groups and

2364 address nutrition and lifestyle issues with evidence-based interventions that are appropriate for delivery
2365 in multiple settings.

2366
2367 Qualified professionals should deliver multidisciplinary interventions and medical nutrition therapies,
2368 as appropriate, that are effective in reducing nutrition-related chronic diseases.

2369
2370 More studies are needed to understand the complex etiology of congenital anomalies and neurological
2371 and psychological conditions, and factors that influence bone health as well as healthy outcomes of
2372 pregnancy so as to inform potential dietary choices by the U.S. population.

2373

2374 **Review of the Evidence**

2375 To reach these conclusions, the DGAC examined evidence from NHANES 2007-2010 and 2009-2012
2376 (see *Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adults ages*
2377 *20 years and older, NHANES 2009-2012, Appendix E-2.19: Low density lipoprotein cholesterol*
2378 *(LDL-C) and triglycerides, adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.20:*
2379 *Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012, Appendix*
2380 *E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.22: Total*
2381 *cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol, children and*
2382 *adolescents ages 6-19 years, NHANES 2009-2012, Appendix E-2.23: Low density lipoprotein*
2383 *cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012,*
2384 *Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and*
2385 *adolescents ages 8-17 years, NHANES 2009-2012), the National Health Interview Survey (NHIS)*
2386 *2012,*⁷⁸ *SEARCH for Diabetes in Youth Study,*⁷⁹ *American Heart Association, 2014 report,*⁶ *and the*
2387 *Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute.*⁸⁰ *The*
2388 *DGAC also examined evidence from CDC's population-based birth defects surveillance system,*⁸¹
2389 *Alzheimer's Association 2014 Facts and Figures,*⁸² *and published data by CDC authors.*⁸³

2390

2391 **Cardiovascular Diseases**

2392 Cardiovascular diseases, including coronary heart disease, hypertension, and stroke, affect an
2393 estimated 83.6 million (35.3 percent) men and women ages 20 years and older in the United States.⁶
2394 CVD increases with age, meaning that about half of those with CVD, 42.2 million adults, are ages 60
2395 years and older.⁶ Rates of coronary heart disease also vary by race/ethnicity and income. Coronary
2396 heart disease is most prevalent in Hispanics (7.8 percent of those reporting the disease) and Native
2397 Americans (including Alaskan natives 12.5 percent) adults.⁷⁸ Stroke is most prevalent in Native
2398 Americans (4.3 percent of those reporting the disease) and African Americans (3.9 percent).⁷⁸
2399 Coronary heart disease rates are inversely related to income. Rates are about 9.8 percent and 7.7
2400 percent in those with lower income (less than 100 percent of the poverty threshold and 100 to 199
2401 percent, respectively) compared to those with higher income (200 percent and greater of the poverty
2402 threshold; 1.9 percent). Stroke also is more prevalent in those with incomes less than 100 percent of

2403 the poverty threshold (4.8 percent) and 100 to 199 percent of the poverty threshold (3.7 percent)
 2404 compared to those with higher incomes (1.9 percent).⁷⁸

2405
 2406 The prevalence of elevated blood pressure (measured systolic pressure of at least 140 mm Hg or
 2407 diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication), in adults ages 18
 2408 years and older (29 percent) is similar in adult men (29.8 percent) and women (28.3 percent) and varies
 2409 by age and race/ethnicity (Table D1.27). Rates of elevated blood pressure are highest in adults ages 60
 2410 years and older (66.3 percent), and African Americans (41.5 percent), relative to non-Hispanic whites
 2411 (27.9 percent) or Hispanics (26.1 percent) (Table D1.27). A similar pattern is seen in youth ages 8 to
 2412 17 years, with borderline high blood pressure in 8.3 percent overall (Table D1.25). Boys (12 percent)
 2413 are much more likely to have borderline high blood pressure than are girls (4.6 percent), as are those
 2414 ages 13 to 17 years (12.4 percent) compared to those ages 8 to 12 years (3.8 percent), and African
 2415 Americans (12.1 percent) compared to non-Hispanic whites (7.2 percent) and Hispanics (8.5 percent)
 2416 (Table D1.25).

2417
 2418 ***Diabetes***

2419 Total diabetes (type I plus type II) is the sum of self-reported diabetes and undiagnosed diabetes.
 2420 Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes
 2421 only during pregnancy. Undiagnosed diabetes was defined as fasting plasma glucose of at least 126
 2422 mg/dL or a hemoglobin A1C value of at least 6.5% and was not reported as a physician diagnosis. The
 2423 prevalence of diabetes in U.S. adults, is 14 percent for men and 10.8 percent for women 20+ years of
 2424 age (Table D1.27). Rates increase with age, to 26 percent for adults ages 60 years and older, and are
 2425 higher in African Americans (18.4 percent) and Hispanics (19.3 percent) compared to non-Hispanic
 2426 whites (9.8 percent) (Table D1.27). Between 2001 and 2009, rates of type 2 diabetes in children and
 2427 adolescents ages 10 to 19 years increased 30.5 percent⁷⁹ and the disease now affects about 1 in 2,000
 2428 youth (0.46 per 1000) (Table D1.28). In 2009, type 2 diabetes appeared to be more common in girls
 2429 than boys (0.58, vs. 0.35 /1000 youth), in older adolescents (ages 15 to 19 years; 0.68) compared to
 2430 those ages 10 to 14 years (0.23), and in American Indian (1.2), African American (1.06), and Hispanic
 2431 (0.79) youth compared to non-Hispanic Whites (0.17) (Table D1.28).

2432
 2433 ***Nutrition-related Major Cancers***

2434 **Breast cancer:** Breast cancer represents approximately 14 percent of all new cancer cases and 6.8
 2435 percent of all cancer deaths in the United States. In 2011, an estimated 2,899,726 (2.9 million) women
 2436 in the United States had a history of breast cancer. About 232,670 new cases of breast cancer and
 2437 40,000 deaths from this disease are estimated for 2014. Breast cancer is the third leading cause of
 2438 cancer death in the U.S.^{80, 84} New cases of breast cancer are highest in the middle age and older women
 2439 (about 22, 25.5, and 21.3 percent of new cases occur in women ages 45 to 54, 55 to 64 and 65 to 74
 2440 years, respectively) (Table D1.29) and in non-Hispanic white women (128/100,000 women per year),
 2441 followed by African American (122.8/100,000 women). The death rate from this disease is also highest
 2442 among women ages 55 to 84 years old (ranges 20.6 percent to 21.7 percent of deaths) and African

2443 Americans (30.6 of death/100,000), followed by non-Hispanic white women (21.7/100,000) (Table
2444 D1.29).

2445

2446 **Prostate cancer:** Prostate cancer represents approximately 14 and 5 percent of all new cancer cases
2447 and all cancer death, respectively in U.S. men. In 2011, an estimated 2,707,821 (2.7 million) men had a
2448 history of prostate cancer. About 233,000 new cases of prostate cancer and 29,480 deaths from this
2449 disease are estimated for 2014. Prostate cancer is the fifth leading cause of cancer death in the United
2450 States.^{84, 85} New cases of prostate cancer are most prevalent in older men (about 32.7, 36.3 and 16.8
2451 percent of new cases in men ages 55 to 64, 65 to 74, and 75 to 84 years, respectively) (Table D1.29)
2452 and African American (223.9 of new cases/100,000 men). The death rate from this disease is highest
2453 among men ages 75 to 84 years old (36.8 percent of deaths) and African Americans (48.9/100,000)
2454 (Table D1.29).

2455

2456 **Colorectal cancer:** Colorectal cancer represents approximately 8.2 and 8.6 percent of all new cancer
2457 cases and all cancer death, respectively in the United States. In 2011, an estimated 1,162,426 (1.2M)
2458 adult men and women had a history of colorectal cancer. About 136,830 new cases of colorectal cancer
2459 and 50,310 deaths from this disease are estimated for 2014, respectively. Colorectal cancer is the
2460 second leading cause of cancer death in the United States.^{84, 86} The incidence (new cases) of this
2461 cancer is more common in men than women and is more common in those older than age 55 years
2462 (highest frequency observed among those ages 65 to 74 years (23.9 percent) (Table D1.29) and in
2463 African Americans (62.3 and 47.5 new cases/100,000 persons in African American men and women,
2464 respectively). The death rate from this disease also is highest in people older than age 55 years old
2465 (highest frequency observed among those ages 75 to 84 years old (27.3 percent of deaths) and in
2466 African American (27.7, and 18.5 deaths/100,000 persons in men and women, respectively) (Table
2467 D1.29).

2468

2469 **Lung and Bronchus cancer:** Lung and bronchus cancer represents approximately 13.5 and 27.2
2470 percent of all new cancer cases and all cancer deaths, respectively in the United States. In 2011, an
2471 estimated 402,326 people had a history of lung and bronchus cancer. About 224,210 new cases of lung
2472 and bronchus cancer and 159,260 deaths from this disease are estimated in 2014, respectively. This
2473 cancer is the first leading cause of cancer death in the United States.^{84, 87} The incidence of lung and
2474 bronchus cancer is more common in men than women and is more common in those older than age 55
2475 years (highest frequency observed among those ages 65 to 74 years (31.7 percent) in African American
2476 men (93 new cases/100,000 persons), and in white women (53.8/100,000 persons) (Table D1.29). The
2477 death rate from this disease also is higher in older people (highest frequency observed among those
2478 ages 65 to 84 years (about 30 percent of deaths) and in African American men (75.7 deaths/100,000
2479 persons), and non-Hispanic white women (39.8/100,000 persons) (Table D1.29).

2480

2481 ***Bone Health***

2482 Approximately 10 million (10.3 percent) American adults ages 50 years and older were reported to
2483 have osteoporosis (defined as T-score ≤ -2.5 at either the femoral neck or the lumbar spine) and 43
2484 million (44 percent) to have low bone mass (defined as T-scores between -1.0 and -2.5 at either
2485 skeletal site) in NHANES 2005-2010 (Table D1.30). A higher percent of women are affected by
2486 osteoporosis (15 percent) and low bone mass (51 percent) than men (about 4 percent and 35 percent,
2487 respectively). Osteoporosis increases with advancing age, occurring in about 35 percent in women ages
2488 80 years and older compared to 26 percent in those ages 70 to 79 years old. The prevalence of low
2489 bone mass is similar in women ages 50 to 59 year and 80 years and older (ranges from 49 to 53
2490 percent). Osteoporosis and low bone mass are more prevalent in Mexican American (20 percent, 48
2491 percent) and non-Hispanic white (16 percent, 53 percent) relative to African American (8 percent, 36
2492 percent) women (Table D1.30).

2493

2494 ***Congenital Anomalies***

2495 Each year, about 3 percent (one in every 33 babies) is born with spina bifida (without anencephaly);
2496 cleft lip (with and without cleft palate), or cleft palate (without cleft lip).⁸⁸ The estimated national
2497 prevalence of spina bifida was 3.17 per 10,000 live births in 1999-2007.⁸¹ During this same time
2498 period, the prevalence of having a baby with spina bifida was reported to be more common in Native
2499 Americans/Alaska Natives (4.02/10,000 live birth), followed by Hispanics (3.8/10,000), non-Hispanic
2500 whites (3.09/10,000), African-Americans (2.73/10,000), and Asian/Pacific Islanders (1.2/10,000).⁸¹
2501 The estimated national prevalence of cleft palate and cleft lip is 5.67 and 9.3 per 10,000 live birth,
2502 respectively.⁸¹ The prevalence of both of these congenital anomalies was highest in non-Hispanic
2503 Native Americans/Alaskan Natives (20/10,000 [cleft lip] and 6.5/10,000 [cleft palate]), and was lowest
2504 in African-Americans (6/10,000 [cleft lip] and 4.2/10,000 [cleft palate]).⁸¹

2505

2506 Congenital heart defects affect about 40,000 births (about 1 percent of births) per year in the United
2507 States.⁸⁹ The number of babies with congenital heart defects, especially those forms that are less
2508 severe (ventricular septal defects and atrial septal defects), is increasing compared to the total number
2509 of births, while the prevalence of other types has remained stable.⁸⁹

2510

2511 ***Neurological and Psychological Conditions***

2512 There are numerous types of neurological and psychological conditions, and the DGAC focused only
2513 on depression and Alzheimer's disease. The prevalence of depression was estimated at 8 percent for
2514 the U.S. population ages 12 years and older in the NHANES 2007-2010 survey.⁹⁰ Depression is higher
2515 in females (10 percent) than in males (6 percent), and highest in those ages 40 to 59 years (12 percent
2516 women, 7 percent men).⁹⁰ Depression also is reported to be higher in African Americans (8 percent),
2517 followed by Mexican-Americans (6.3 percent) and non-Hispanic whites (4.8 percent) (NHANES 2005
2518 -2006).⁹¹

2519

2520 In 2014, about 3.2 million women and 1.8 million men in the United States, ages 65 years and older
 2521 are reported to be living with Alzheimer’s disease.⁸² This disease is most prevalent in those ages 75 to
 2522 84 years (44 percent of those with Alzheimer’s) and those ages 85 years and older (38 percent).⁸²
 2523 About 63, 59, and 30 percent of those ages 85 years and older with Alzheimer’s disease are reported to
 2524 be Hispanics (primarily Caribbean-American), African Americans, and non-Hispanic white adults,
 2525 respectively.⁸² It has been projected that the number of people with Alzheimer’s disease will increase
 2526 by about threefold from 4.8 million in 2010 to 13.7 million in 2050.⁹²

2527

2528 ***For additional details on this body of evidence, visit:***

- 2529 • Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adult ages 20
 2530 years and older, NHANES 2009-2012
- 2531 • Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20
 2532 years and older, NHANES 2009-2012
- 2533 • Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES
 2534 2009-2012
- 2535 • Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012
- 2536 • Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-
 2537 cholesterol, children and adolescents ages 6-19 years, NHANES 2009 -2012
- 2538 • Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages
 2539 12-19 years, NHANES 2009-2012
- 2540 • Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and
 2541 adolescents ages 8-17 years, NHANES 2009-2012
- 2542 • SEER Cancer Statistics Review, 1975-2011. Available from:
 2543 http://seer.cancer.gov/csr/1975_2011/.
- 2544 • SEER Stat Fact Sheets: Breast Cancer. Available from:
 2545 <http://seer.cancer.gov/statfacts/html/breast.html>.
- 2546 • SEER Stat Fact Sheets: Colon and Rectum Cancer. Available from:
 2547 <http://seer.cancer.gov/statfacts/html/colorect.html>.
- 2548 • SEER Stat Fact Sheets: Lung and Bronchus Cancer. Available from:
 2549 <http://seer.cancer.gov/statfacts/html/lungb.html>.
- 2550 • SEER Stat Fact Sheets: Prostate Cancer. Available from:
 2551 <http://seer.cancer.gov/statfacts/html/prost.html>.
- 2552 • Summary health statistics for U.S. adults: National Health Interview Survey, 2012. Available from:
 2553 http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.

- 2554 • Respondent-reported prevalence of heart disease, cancer, and stroke among adults aged 18 and
 2555 over, by selected characteristics: United States, average annual, selected years 1997-1998 through
 2556 2011-2012. Available from: <http://www.cdc.gov/nchs/data/hus/2012/044.pdf>.
- 2557 • 2014 Alzheimer's disease facts and figures: includes a special report on women and Alzheimer's
 2558 disease. *Alzheimers Dement.* 2014;10(2):131-68. PMID: 22404854. Available from:
 2559 http://www.alz.org/downloads/facts_figures_2014.pdf.
- 2560 • Facts about Birth Defects [updated October 20, 2014]. Available from:
 2561 <http://www.cdc.gov/ncbddd/birthdefects/facts.html>.
- 2562 • Depression in the United States household population, 2005-2006. *NCHS Data Brief.* 2008(7):1-8.
 2563 PMID: 19389321. Available from: <http://www.cdc.gov/nchs/data/databriefs/db07.pdf>.
- 2564 • Congenital Heart Defects. Data and Statistics. Atlanta, GA [updated July 9, 2014; cited 2014
 2565 September 2]. Available from: <http://www.cdc.gov/ncbddd/heartdefects/data.html>.
- 2566 • Prevalence of Current Depression Among Persons Aged \geq 12 Years, by Age Group and Sex
 2567 United States, National Health and Nutrition Examination Survey, 2007-2010. *Morbidity and*
 2568 *Mortality Weekly Report (MMWR).* 2014;60(51):1747. Available from:
 2569 http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.

2570

2571

2572 **DIETARY PATTERNS COMPOSITION**

2573 Dietary patterns with positive health benefits are described as high in vegetables, fruit, whole grains,
 2574 seafood, legumes, and nuts; moderate in low- and non-fat dairy products; lower in red and processed
 2575 meat; and low in sugar-sweetened foods and beverages and refined grains. The primary dietary
 2576 patterns examined and described in Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and
 2577 Health Outcomes included both a priori, investigator-derived scoring systems such as DASH/OMNI,
 2578 Mediterranean diet scores, and the Healthy Eating Index, as well as data-driven approaches using
 2579 factor/cluster analysis or reduced rank regression. The findings presented come from controlled
 2580 intervention trials, cohort studies, and nested case-control studies. The DGAC examined these patterns
 2581 in an attempt to quantify, for the first time, the approximate amounts of each food group in these
 2582 patterns. The DGAC also examined the range of and commonalities across food group intakes in
 2583 healthy dietary patterns and compared these ranges to the range of usual adult consumption in the
 2584 United States and to the range recommended by the USDA Food Patterns.

2585

2586 **Question 18: What is the composition of dietary patterns with evidence of positive**
2587 **health outcomes (e.g., Mediterranean-style patterns, Dietary Approaches to Stop**
2588 **Hypertension-style patterns, patterns that closely align with the Healthy Eating Index,**
2589 **and vegetarian patterns), and of patterns commonly consumed in the United States?**
2590 **What are the similarities (and differences) within and among the dietary patterns with**
2591 **evidence of positive health outcomes and the commonly consumed dietary patterns?**

2592 **Source of evidence:** Data analysis

2593

2594 **Conclusions**

2595 Dietary patterns with varying food group composition, but certain common elements were observed
2596 across intervention and cohort studies to have health benefits. A healthful diet can be achieved by
2597 following any of these dietary patterns.

2598

2599 In general, the ranges of intake in dietary patterns with positive health benefits are very close to those
2600 recommended by the USDA Food Patterns, but amounts of some specific food groups vary across the
2601 various diet pattern types.

- 2602 • DASH-style diets, Mediterranean-style diets, and the USDA Food Patterns are similar with
2603 respect to amounts of fruits and vegetables, and the OMNI diets are slightly higher than the
2604 USDA Food Patterns.
- 2605 • Dairy intake is comparable between DASH-style diets and the USDA Food Patterns, but dairy
2606 is lower for Mediterranean-style diets than for the USDA Food Patterns.
- 2607 • Red and processed meats are higher in the Mediterranean-style diets but lower in the DASH-
2608 style diet than is recommended by the USDA Food Patterns.
- 2609 • Seafood intake is similar in DASH-style and higher in Mediterranean-style diets than in the
2610 USDA Food Patterns.

2611 The data from the intervention trials and the cohort studies examined provide empirical data that the
2612 USDA Food Patterns provide an evidence-based guide to healthy patterns of food consumption.

2613

2614 **Implications**

2615 The quality of the diets currently consumed by the U.S. population is suboptimal overall and has major
2616 adverse health consequences. Several options exist for dietary patterns that can be followed to improve
2617 the population's diet quality. The approaches that can be taken are varied and can be adapted to
2618 personal and cultural preferences. The ability to offer the U.S. population alternative dietary pattern
2619 options and to tailor them to personal preferences may increase the likelihood of long term success of
2620 maintaining a healthy diet pattern, ultimately leading to improved health in the U.S. population.

2621

2622 **Review of the Evidence**

2623 The DGAC analyzed data on food group composition reported in research articles on dietary patterns
 2624 and health outcomes. These articles were drawn from those included in the questions on dietary
 2625 patterns and health examined by the Committee (see *Part D. Chapter 2: Dietary Patterns, Food and*
 2626 *Nutrients, and Health Outcomes*). The studies reported in that chapter D2 were reviewed to identify
 2627 those that reported semi-quantitative data on food group intakes among the sample or population group
 2628 with positive health outcomes (Table D1.31).⁹³⁻¹¹² These sample or population groups included the
 2629 intervention group in intervention studies, the highest category (usually the top quintile) in cohorts and
 2630 nested case-control studies measuring diet with an a priori index, or a specific cluster or factor analysis
 2631 group. Approximate quantified food group intakes for these subsets of the population or samples with a
 2632 beneficial health outcome were identified. These intakes were converted to grams per day if not
 2633 reported this way in the original manuscripts. Then, all data were converted to grams per 1000 calories
 2634 to allow for comparisons across studies.

2635
 2636 For comparison to usual intake levels of each food group in the United States., data from NHANES
 2637 2007-2010 for usual intake by adult age/sex groups⁴¹ in cup or ounce equivalents were converted into
 2638 grams using average weights based on Food Patterns Equivalents Database (FPED) data.^{48,49} The
 2639 gram weights were divided by the usual calorie intake for that group, and multiplied times 1000 for an
 2640 estimate of the food group intake per 1000 calories for each adult age/sex group. The range of these
 2641 intakes was used as a comparator. For comparison to the food group amounts recommended in the
 2642 USDA Food Patterns (also called the Healthy U.S.-style Patterns; see Question 20) the recommended
 2643 amount for adult age/sex groups in the patterns at 1600 to 2400 calories were converted to grams per
 2644 1000 calories by the same procedure used for the usual intakes (see Figures D1.56 to D1.60).

2645
 2646 Vegetable intake in the OMNI diets was higher than both the USDA Food Patterns and current
 2647 consumption estimates, but DASH-style, PREDIMED, most of the Mediterranean scores, and data
 2648 driven approaches were very similar to vegetable amounts recommended by the USDA Food Patterns.
 2649 Fruit intake was higher in the OMNI diets and PREDIMED relative to the USDA Food Patterns and
 2650 current consumption, but DASH, the Mediterranean score diets, and many of the data driven scores are
 2651 all within the range of the USDA Food Pattern recommendations. Dairy intakes in OMNI, DASH, and
 2652 some of the Mediterranean and data driven scores were all within the ranges recommended by the
 2653 USDA Food Patterns, while PREDIMED and some other scores had lower intakes of dairy.
 2654 Consumption of red and processed meats was higher in PREDIMED and in some studies using
 2655 Mediterranean diet scores relative to the USDA Food Patterns, whereas several cohorts using data-
 2656 driven approaches to assessing diet patterns reported ranges of red and processed meat intake that
 2657 aligned very well with the USDA Food Pattern recommendations. Intakes of red and processed meat
 2658 were lower in the OMNI and DASH dietary interventions than in either the USDA Food Patterns or the
 2659 range of usual intake in the United States. Seafood intakes for the OMNI diets and some of the data-
 2660 driven dietary pattern studies aligned very well with the USDA Food Patterns. Seafood intake ranges

2661 for all the other studies were much higher than both the USDA Food Patterns and the ranges of usual
2662 intake in the United States.

2663

2664 ***For additional details on this body of evidence, visit:***

2665 • Usual Dietary Intakes: Food Intakes, U.S. Population, 2007-10: Applied Research Program.
2666 National Cancer Institute; [updated May 22, 2014]. Available from:
2667 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/>.

2668 • Appendix E-3.1: Adequacy of the USDA Food Patterns

2669

2670 **Question 19: To what extent does the U.S. population consume a dietary pattern that**
2671 **is similar to those observed to have positive health benefits [e.g., Mediterranean-style**
2672 **pattern, Dietary Approaches to Stop Hypertension (DASH)-style patterns, patterns that**
2673 **closely align with the Healthy Eating Index, and vegetarian patterns] overall and by**
2674 **age/sex and race/ethnic groups?**

2675 **Source of evidence:** Data analysis

2676

2677 **Conclusion**

2678 Data from WWEIA show that the average HEI score in the U.S. population is 57 points out of a total
2679 of 100 points. The best scores (average scores) were observed for the following components: total
2680 protein foods (average score of 100 percent of possible points), seafood and plant protein (84 percent
2681 of possible points), and dairy (69 percent of possible points), while the poorest scores were observed
2682 for whole grains (25 percent of possible points), sodium (37 percent of possible points), fatty acid ratio
2683 (41 percent of possible points), greens and beans (46 percent of possible points), and empty calories
2684 (60 percent of possible points).

2685

2686 Young children ages 2 to 3 years and middle aged and older adults (ages 51 years and older) have the
2687 best HEI scores (total scores of 63 percent and 66 percent, respectively), while preadolescents and
2688 adolescents have the poorest HEI scores (total scores of 49 percent and 48 percent, respectively).

2689

2690 **Implications**

2691 To improve diet quality, the U.S. population should replace most refined grains with whole grains,
2692 decrease sodium, decrease saturated fat, consume fewer calories from added sugars, and replace these
2693 calories with more varied vegetable choices, seafood, plant proteins, and low-fat dairy.

2694

2695 Young children and middle-aged and older adults have the highest HEI scores. These positive healthy
2696 eating habits should continue to be encouraged. Because preadolescents and adolescents have the
2697 lowest HEI scores, significant intervention is needed at the level of the individual, family, school, day
2698 care, and community settings to help this age group adopt and maintain healthful dietary patterns.

2699

2700 **Review of the Evidence**

2701 The DGAC examined mean HEI scores and component scores for the entire U.S. population ages 2
 2702 years and older (see *Appendix E-2.25: Average Healthy Eating Index-2010 scores for Americans*
 2703 *ages 2 years and older*). These data were examined for the entire population, for males and females
 2704 and by age subgroups. In general, the best scores for the HEI components were for protein and seafood
 2705 and plant proteins, while the poorest score was for whole grains. For nearly all of the component
 2706 scores as well as the total HEI score, females tended to have better scores than males, indicating
 2707 slightly healthier dietary patterns in females compared to males. Analyses by age showed that the
 2708 youngest and oldest segments of the population had the best component and total HEI scores (Figure
 2709 D1.61). For these groups, the component scores were very good to excellent for total fruit and whole
 2710 fruit. Young children also had excellent scores for dairy, and middle-aged and older adults had
 2711 excellent scores for total protein and seafood and plant protein. All age groups have poor scores for
 2712 whole grains.

2713

2714 Data were not available to examine how closely the U.S. population’s dietary patterns align with a
 2715 Mediterranean-style or DASH-style dietary pattern.

2716

2717 *For additional details on this body of evidence, visit:*

2718

- Healthy Eating Index, Center for Nutrition Policy and Promotion. Available from:

2719

<http://www.cnpp.usda.gov/HealthyEatingIndex>.

2720

- Appendix E-2.25: Average Healthy Eating Index-2010 scores for Americans ages 2 years and older (National Health and Nutrition Examination Survey 2009-2010)

2721

2722

2723 **Question 20: Using the Food Pattern Modeling process, can healthy eating patterns**
 2724 **for vegetarians and for those who want to follow a Mediterranean-style dietary pattern**
 2725 **be developed? How do these patterns differ from the USDA Food Patterns previously**
 2726 **updated for potential inclusion in the 2015 DGAs?**

2727

Source of evidence: Food Pattern Modeling

2728

2729 **Conclusion**

2730 Food Pattern Modeling demonstrates that healthy eating patterns can be achieved for a variety of
 2731 eating styles, including the “Healthy U.S.-style Pattern,” the “Healthy Mediterranean-style Pattern,”
 2732 and the “Healthy Vegetarian-style Pattern”. Although some differences exist across the three eating
 2733 patterns, comparable amounts of nutrients can be obtained using nutrient dense foods while
 2734 maintaining energy balance.

2735

2736 **Implications**

2737 The U.S. population has a variety of options to help achieve healthful eating patterns that adhere to the
 2738 Dietary Guidelines. These include the Healthy U.S.-style Pattern, Mediterranean-style Pattern, or
 2739 Vegetarian Patterns. (Detailed information on these patterns can be found in Table D1.32 and
 2740 *Appendix E-3.7: Developing Vegetarian and Mediterranean-style Food Patterns*.) These diets meet
 2741 nutritional goals without excess calories and use a variety of foods. Importantly, these diets reflect the
 2742 range of foods that can be used to achieve a healthful eating pattern, and they support the inclusion of
 2743 diverse foods that are consistent with personal, cultural and religious preferences. These diets can be
 2744 used in a variety of settings, including homes, schools, worksites, health care facilities, and places of
 2745 worship.

2746

2747 **Review of the Evidence**

2748 These conclusions were reached based on the results of the Food Pattern Modeling analysis for
 2749 vegetarian and Mediterranean-style food patterns. Data from WWEIA from self-reported vegetarians
 2750 were used to inform the vegetarian eating pattern (Figure D1.62) and data from the Dietary Patterns
 2751 composition project reviewed above were used to select foods for the Mediterranean-style pattern.¹¹³

2752

2753 From three dietary patterns (“Healthy U.S.-style,” “Healthy Mediterranean-style Patter,” and “Healthy
 2754 Vegetarian Pattern”), selected food group intakes across calorie levels were compared (Table D1.32).
 2755 Notably, fruit and seafood were higher in the Mediterranean-style diet, while dairy was lower, based
 2756 on the data presented above (Figures D1.56 to D1.60). For the Vegetarian Pattern, meat and seafood
 2757 are absent, but eggs and dairy are included because self-reported vegetarians in WWEIA reported
 2758 consumption of these foods. Legumes, nuts/seeds, and processed soy are all higher in the Vegetarian
 2759 Pattern compared to the Healthy U.S.-style and the Healthy Mediterranean-style Patterns.

2760

2761 When comparing nutrient intake across these three dietary patterns, as a percent of the RDA using a
 2762 woman age 19 to 30 years as an example, modest difference emerged (Table D1.33). The Vegetarian
 2763 pattern is lower in sodium and all three patterns are low in vitamin D.

2764

2765 ***For additional details on this body of evidence, visit:***

- 2766 • Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program.
 2767 National Cancer Institute; [updated May 22, 2014]. Available from:
 2768 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/>.
- 2769 • Appendix E-3.7: Developing Vegetarian and Mediterranean-style Food Patterns

2770

2771

2772 **CHAPTER SUMMARY**

2773 The DGAC conducted data analyses to address a series of questions related to the current status and
2774 trends in the Nation’s dietary intake. The questions focused on: intake of specific nutrients and food
2775 groups; food categories (i.e., foods as consumed) that contribute to intake; eating behaviors; and the
2776 composition of various dietary patterns shown to have health benefits, including Mediterranean-style
2777 diets, the Healthy US-style and DASH-style diets. These topics were addressed using data from the
2778 WWEIA dietary survey, which is the dietary intake component of the ongoing NHANES. Food pattern
2779 modeling using the USDA Food Pattern food groups also was used to address some of the questions of
2780 interest. In addition, the DGAC examined the prevalence and trends of health conditions that may have
2781 a nutritional origin, or where the course of disease may be influenced by diet.

2782
2783 The DGAC found that several nutrients are underconsumed and the Committee characterized them as
2784 shortfall nutrients: vitamin A, vitamin D, vitamin E, vitamin C, folate, calcium, magnesium, fiber, and
2785 potassium. For adolescent and premenopausal females, iron also is a shortfall nutrient. Important to
2786 note, on the basis of nutrient biomarkers or health outcomes, calcium, vitamin D, fiber, and potassium
2787 also are classified as nutrients of public health concern because their underconsumption has been
2788 linked in the scientific literature to adverse health outcomes. Iron is included as a shortfall nutrient of
2789 public health concern for adolescent females and adult females who are premenopausal due to the
2790 increased risk of iron-deficiency in these groups. The DGAC also found that two nutrients—sodium
2791 and saturated fat—are overconsumed by the U.S. population and that the overconsumption poses
2792 health risks.

2793
2794 The majority of the U.S. population has low intakes of key food groups that are important sources of
2795 the shortfall nutrients including vegetables, fruits, whole grains, and dairy. Furthermore, population
2796 intake is too high for refined grains and added sugars. The data suggest cautious optimism about
2797 dietary intake of the youngest members of the U.S. population because many young children ages 2 to
2798 5 years consume recommended amounts of fruit and dairy. However, a better understanding is needed
2799 on how to maintain and encourage the good habits that are started early in life. Analysis of data on
2800 food categories, such as burgers, sandwiches, mixed dishes, desserts, and beverages, because they
2801 represent such a large proportion of the calories consumed, are prime targets for reformulation to
2802 increase population intake of vegetables, whole grains, and other underconsumed food groups and to
2803 lower population intake of the nutrients sodium and saturated fat, and the food component refined
2804 grains. Dramatically reducing the intake of sugar-sweetened beverages and limiting sweets and
2805 desserts would help lower intakes the food component added sugars.

2806
2807 The U.S. population purchases its food in a variety of locations, including supermarkets, convenience
2808 stores, schools, and the workplace, and consumes prepared food outside the home. The DGAC found
2809 that while diet quality varies somewhat by the setting where food is obtained, overall, independent of
2810 where the food is prepared or obtained, the diet quality of the U.S. population does not meet
2811 recommendations for fruit, vegetables, dairy, or whole grains, and exceeds recommendations, leading

2812 to overconsumption, for the nutrients sodium and saturated fat, and the food components refined
2813 grains, solid fats, and added sugars.

2814
2815 Obesity and chronic diseases with a nutritional origin are very common. The Nation must accelerate
2816 progress toward reducing the incidence and prevalence of overweight and obesity and chronic disease
2817 risk across the U.S. population throughout the lifespan and reduce the disparities in obesity and chronic
2818 disease rates that exist in the United States for certain ethnic and racial groups and for those with lower
2819 incomes.

2820
2821 The DGAC identified key aspects of several different dietary patterns that are associated with lower
2822 risk of many nutrition-related outcomes such as cardiovascular disease, diabetes, some cancers,
2823 psychological health and bone health. These patterns and their associated health benefits are described
2824 in greater detail in the next chapter.

2825
2826 The DGAC had enough descriptive information from existing research and data to model three dietary
2827 patterns and to examine their nutritional adequacy. These patterns are the Healthy U.S.-style Pattern,
2828 the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern. These patterns include
2829 the components of a dietary pattern associated with health benefits.

2830
2831 The findings from this chapter and the remainder of the 2015 DGAC report can be used by individuals,
2832 families, communities, schools, local, state and federal agencies and the food industry to address the
2833 high prevalence of obesity and other nutrition-related health conditions in the United States and help
2834 all sectors of the population consume a diet that is healthful, accessible, and affordable.

2835
2836

2837 **NEEDS FOR FUTURE RESEARCH**

2838 1. Expand WWEIA participation to include more respondents from race/ethnic minorities and non-
2839 U.S. born residents.

2840 **Rationale:** Very little is known about the dietary habits of many of the cultural subgroups in the
2841 United States. This knowledge is essential to moving forward any nutrition programs for first and
2842 second generation immigrants. More data on the impact of acculturation also are needed on food
2843 and health behaviors. The number of participants in WWEIA using the derived acculturation
2844 variable was too small for any analysis. Finally, “Hispanic” is a very broad term and a better
2845 understanding is needed of the nutritional profiles (including shortfalls and excesses) across
2846 various Spanish-speaking people in the United States, who come from different cultural
2847 backgrounds with distinct eating patterns.

2848
2849 2. Include higher proportion of older Americans as respondents in WWEIA.

2850 **Rationale:** More data are needed on dietary intake of older adults; the sample sizes in WWEIA
 2851 were too small for any meaningful analyses for those older than the age of 71 years. In addition to
 2852 nutrient intake, additional information is needed on whether older adults are able to shop and cook,
 2853 whether polypharmacy plays a role in nutritional adequacy, and whether co-morbidities, such as
 2854 poor dentition, musculo-skeletal difficulties, arthralgias and other age-related symptoms, affect
 2855 their ability to establish and maintain proper nutritional status.
 2856

2857 3. Increase the number of pregnant women as respondents in WWEIA.

2858 **Rationale:** The number of pregnant women in WWEIA is currently too small to properly evaluate
 2859 the status and trends in food and nutrient intake in pregnant women. Since good nutrition in
 2860 pregnancy is critical to proper growth development of the infant it is critical to properly evaluate
 2861 food and nutrient intake, which will inform recommendations and public policies for pregnant
 2862 women.
 2863

2864 4. Conduct research on nutrition transitions from childhood to shed light on how and why dietary
 2865 intake changes so rapidly from early childhood through pre-adolescence and adolescence, and to
 2866 identify the driving forces behind dietary intake change in these age groups and what programs are
 2867 most effective at maintaining positive nutrition habits established in very young children.

2868 **Rationale:** Young children have better dietary intake than older children and adolescents. It is
 2869 important to maintain the positive gains made in early childhood and identify factors responsible
 2870 for the declines in intakes of fruit, dairy, and other food groups and increases in added sugars and
 2871 refined grains as children become enter the elementary school age years, as poor eating patterns in
 2872 elementary school seem to persist into adolescence and beyond.
 2873

2874 5. Evaluate the effects of common variations in dietary patterns in small children on nutrient intakes.

2875 **Rationale:** Children from 2 to 4 years of age have a highly variable diet and often do not fit readily
 2876 into the USDA Food Pattern food groups diet pattern analyses. Further information is needed to
 2877 understand the broad range of diets and supplement use in small children and how this relates to
 2878 nutrient intake and growth. Research is needed to better characterize their diets so that appropriate
 2879 guidance can be offered.
 2880

2881 6. Increase the quantity and quality of food composition databases available for research.

2882 **Rationale:** Accurate assessment of nutrient intake and trends over time in the U.S. population is
 2883 dependent upon the quality of food composition data. Tens of thousands of foods are available for
 2884 purchase and consumption in the United States, but accurate nutrient content data are available
 2885 only for less than 10,000 foods and are almost non-existent for many ready-to-eat and restaurant-
 2886 type foods. Analytic values from foods are needed on specific nutrients and components, such as
 2887 vitamin D, fiber, added sugars, and sodium. Improved food composition data also is critical for

2888 needed research to better define, identify, and quantify total grain, whole grain consumption, and
2889 refined grain consumption in dietary studies.

2890

2891 7. Investigate the validity, reliability, and reproducibility of new biomarkers of nutrient intake and
2892 biomarkers of nutritional status.

2893 **Rationale:** Limited biomarkers are available and some that are available are difficult to interpret
2894 due to other contributing factors to the biomarker measure (e.g., vitamin D is obtained in the diet
2895 and is also endogenously synthesized).

2896

2897 8. Evaluate effects of fortification strategies and supplement use on consumer behavior related to the
2898 intake of foods and supplements containing key nutrients, including calcium, vitamin D, potassium,
2899 iron, and fiber

2900 **Rationale:** The intake of key nutrients of concern is considerably affected by the rapidly evolving
2901 marketplace of food fortification and supplementation. Understanding consumer behavior related
2902 to fortification and supplementation would be important in predicting the effects of interventions
2903 and marketplace changes in content of these nutrients. Special interest exists regarding fortification
2904 strategies of foods, including whole grains and yogurts, in allowing individuals to reach the RDA
2905 for vitamin D without using supplements. Data are needed on how supplements may help meet
2906 nutrients shortfalls and/or how use of supplements may place individuals at risk of
2907 overconsumption. Research on effective consumer guidance is needed.

2908

2909 9. Understand the rationale for and consequences of the use of supplements above the UL for
2910 vitamins and minerals. Identify biochemical markers that would indicate the effects of high-dose
2911 supplement use.

2912 **Rationale:** Consumer use of high-dose supplements has increased. Understanding the influences
2913 guiding this use would be helpful in considering how to educate consumers about safe upper intake
2914 limits.

2915

2916 10. Develop a standardized research definition for meals and snacks.

2917 **Rationale:** Multiple different criteria are used in studies to define a snack or meal occasion, such
2918 as time of day, the types or amounts of food consumed, or subjective assessment by the study
2919 respondent. Researchers should work toward a consensus on the use of standard definitions.

2920

2921 11. Understand better the concept of dietary patterns and design approaches to quantify the diet in
2922 large population-based studies.

2923 **Rationale:** More methodological work on dietary patterns is needed. For example, food frequency
2924 questionnaires, which are used in most diet assessment studies, do not capture data on meal timing,
2925 meal frequency, or the types of foods consumed together. Studies using diet recalls and records are
2926 better at capturing specific foods and their quantities consumed (portion sizes) and the types of

2927 foods eaten together, but often these detailed assessment methods are not feasible for large
 2928 population-based studies. Quantification of food group intake is needed. In addition, dietary
 2929 patterns research encompasses a broader scope of issues than can be addressed by diet scores and
 2930 data driven approaches.

2931
 2932 12. Consistently report the nutrients, foods, and food groups that are used to evaluate dietary patterns
 2933 in published studies.

2934 **Rationale:** The current scientific literature evaluating dietary patterns and health is inconsistent in
 2935 its provision of dietary patterns composition information. This makes it difficult to compare, across
 2936 studies, the components of healthful patterns that are associated with health benefits.

2937
 2938 13. Conduct population surveillance on the prevalence and trends of nutrition-related chronic diseases
 2939 including type 2 diabetes, cardiovascular disease, some cancers osteoporosis and neurocognitive
 2940 disorders.

2941 **Rationale:** Current data on diabetes in adults cannot be stratified by disease type (type I or type II),
 2942 making it very difficult to monitor incidence and prevalence of type 2 diabetes. Continued
 2943 population surveillance is needed to effectively link nutritional factors with risk of these diseases.

2944

2945 REFERENCES

- 2946 1. National Research Council. Dietary Reference Intakes: The Essential Guide to Nutrient Requirements
 2947 Washington, DC: The National Academies Press; 2006. Available from:
 2948 http://books.nap.edu/openbook.php?record_id=11537.
- 2949 2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013 AHA/ACC
 2950 guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of
 2951 Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25
 2952 Suppl 2):S76-99. PMID: 24222015. <http://www.ncbi.nlm.nih.gov/pubmed/24222015>.
- 2953 3. Centers for Disease Control and Prevention. Second National Report on Biochemical Indicators of Diet
 2954 and Nutrition in the U.S. Population. Atlanta, GA: Centers for Disease Control and Prevention, U.S.
 2955 Department of Health and Human Services; 2012. Available from:
 2956 http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.
- 2957 4. Weaver CM, Dwyer J, Fulgoni VL, 3rd, King JC, Leveille GA, MacDonald RS, et al. Processed foods:
 2958 contributions to nutrition. *Am J Clin Nutr*. 2014;99(6):1525-42. PMID: 24760975.
 2959 <http://www.ncbi.nlm.nih.gov/pubmed/24760975>.
- 2960 5. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What
 2961 We Eat in America, NHANES dietary survey [updated 10/29/2013]. Available from:
 2962 <http://seprl.ars.usda.gov/Services/docs.htm?docid=13793>.
- 2963 6. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, et al. Heart disease and stroke
 2964 statistics--2014 update: a report from the American Heart Association. *Circulation*. 2014;129(3):e28-
 2965 e292. PMID: 24352519. <http://www.ncbi.nlm.nih.gov/pubmed/24352519>.
- 2966 7. Institute of Medicine. Dietary Reference Intakes: Applications in Dietary Assessment. Washington, DC:
 2967 The National Academies Press; 2000. Available from:
 2968 http://www.nap.edu/openbook.php?record_id=9956&page=R1.
- 2969 8. National Research Council. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron,
 2970 Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.

- 2971 Washington, DC: The National Academies Press; 2001. Available from:
 2972 http://www.nap.edu/openbook.php?record_id=10026.
- 2973 9. Satia-Abouta J, Patterson RE, Neuhouser ML, Elder J. Dietary acculturation: applications to nutrition
 2974 research and dietetics. *J Am Diet Assoc.* 2002;102(8):1105-18. PMID: 12171455.
 2975 <http://www.ncbi.nlm.nih.gov/pubmed/12171455>.
- 2976 10. Abraido-Lanza AF, Chao MT, Florez KR. Do healthy behaviors decline with greater acculturation?
 2977 Implications for the Latino mortality paradox. *Soc Sci Med.* 2005;61(6):1243-55. PMID: 15970234.
 2978 <http://www.ncbi.nlm.nih.gov/pubmed/15970234>.
- 2979 11. Redfield R, Linton R, Herskovits M. Memorandum for the study of acculturation. *American*
 2980 *Anthropologist.* 1936;38(1):149-52.
- 2981 12. Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and incidence of type 2
 2982 diabetes among Latinos. *J Nutr.* 2007;137(4):860-70. PMID: 17374645.
 2983 <http://www.ncbi.nlm.nih.gov/pubmed/17374645>.
- 2984 13. Neuhouser ML, Thompson B, Coronado GD, Solomon CC. Higher fat intake and lower fruit and
 2985 vegetables intakes are associated with greater acculturation among Mexicans living in Washington
 2986 State. *J Am Diet Assoc.* 2004;104(1):51-7. PMID: 14702584.
 2987 <http://www.ncbi.nlm.nih.gov/pubmed/14702584>.
- 2988 14. Cuellar I, Arnold B, Maldonado R. Acculturation Rating Scale for Mexican Americans-II: A Revision
 2989 of the Original ARSMA Scale. *Hispanic Journal of Behavioral Sciences.* 1995;17(3):275-304.
- 2990 15. Institute of Medicine. Ross AC, Taylor CL, Yaktine AL, Valle HBD, editors. *Dietary Reference Intakes*
 2991 *for Calcium and Vitamin D.* Washington, DC: The National Academies Press; 2011. Available from:
 2992 http://www.nap.edu/catalog.php?record_id=13050.
- 2993 16. Schenk JM, Till CA, Tangen CM, Goodman PJ, Song X, Torikko KC, et al. Serum 25-hydroxyvitamin D
 2994 concentrations and risk of prostate cancer: results from the Prostate Cancer Prevention Trial. *Cancer*
 2995 *Epidemiol Biomarkers Prev.* 2014;23(8):1484-93. PMID: 25085836.
 2996 <http://www.ncbi.nlm.nih.gov/pubmed/25085836>.
- 2997 17. Kristal AR, Till C, Song X, Tangen CM, Goodman PJ, Neuhauser ML, et al. Plasma vitamin D and
 2998 prostate cancer risk: results from the Selenium and Vitamin E Cancer Prevention Trial. *Cancer*
 2999 *Epidemiol Biomarkers Prev.* 2014;23(8):1494-504. PMID: 24732629.
 3000 <http://www.ncbi.nlm.nih.gov/pubmed/24732629>.
- 3001 18. Cheng TY, Lacroix AZ, Beresford SA, Goodman GE, Thornquist MD, Zheng Y, et al. Vitamin D intake
 3002 and lung cancer risk in the Women's Health Initiative. *Am J Clin Nutr.* 2013;98(4):1002-11. PMID:
 3003 23966428. <http://www.ncbi.nlm.nih.gov/pubmed/23966428>.
- 3004 19. Neuhouser ML, Manson JE, Millen A, Pettinger M, Margolis K, Jacobs ET, et al. The influence of
 3005 health and lifestyle characteristics on the relation of serum 25-hydroxyvitamin D with risk of colorectal
 3006 and breast cancer in postmenopausal women. *Am J Epidemiol.* 2012;175(7):673-84. PMID: 22362582.
 3007 <http://www.ncbi.nlm.nih.gov/pubmed/22362582>.
- 3008 20. Villasenor A, Ballard-Barbash R, Ambis A, Bernstein L, Baumgartner K, Baumgartner R, et al.
 3009 Associations of serum 25-hydroxyvitamin D with overall and breast cancer-specific mortality in a
 3010 multiethnic cohort of breast cancer survivors. *Cancer Causes Control.* 2013;24(4):759-67. PMID:
 3011 23361338. <http://www.ncbi.nlm.nih.gov/pubmed/23361338>.
- 3012 21. Bjelakovic G, Gluud LL, Nikolova D, Whitfield K, Wetterslev J, Simonetti RG, et al. Vitamin D
 3013 supplementation for prevention of mortality in adults. *Cochrane Database Syst Rev.* 2014;1:CD007470.
 3014 PMID: 24414552. <http://www.ncbi.nlm.nih.gov/pubmed/24414552>.
- 3015 22. Abrams SA, Coss-Bu JA, Tiosano D. Vitamin D: effects on childhood health and disease. *Nat Rev*
 3016 *Endocrinol.* 2013;9(3):162-70. PMID: 23381033. <http://www.ncbi.nlm.nih.gov/pubmed/23381033>.
- 3017 23. Girgis CM, Clifton-Bligh RJ, Hamrick MW, Holick MF, Gunton JE. The roles of vitamin D in skeletal
 3018 muscle: form, function, and metabolism. *Endocr Rev.* 2013;34(1):33-83. PMID: 23169676.
 3019 <http://www.ncbi.nlm.nih.gov/pubmed/23169676>.

- 3020 24. Kalyani RR, Stein B, Valiyil R, Manno R, Maynard JW, Crews DC. Vitamin D treatment for the
3021 prevention of falls in older adults: systematic review and meta-analysis. *J Am Geriatr Soc.*
3022 2010;58(7):1299-310. PMID: 20579169. <http://www.ncbi.nlm.nih.gov/pubmed/20579169>.
- 3023 25. Murad MH, Elamin KB, Abu Elnour NO, Elamin MB, Alkatib AA, Fatourechi MM, et al. Clinical
3024 review: The effect of vitamin D on falls: a systematic review and meta-analysis. *J Clin Endocrinol*
3025 *Metab.* 2011;96(10):2997-3006. PMID: 21795448. <http://www.ncbi.nlm.nih.gov/pubmed/21795448>.
- 3026 26. Cheng TY, Millen AE, Wactawski-Wende J, Beresford SA, LaCroix AZ, Zheng Y, et al. Vitamin D
3027 intake determines vitamin d status of postmenopausal women, particularly those with limited sun
3028 exposure. *J Nutr.* 2014;144(5):681-9. PMID: 24598886.
3029 <http://www.ncbi.nlm.nih.gov/pubmed/24598886>.
- 3030 27. Abrams SA. Setting Dietary Reference Intakes with the use of bioavailability data: calcium. *Am J Clin*
3031 *Nutr.* 2010;91(5):1474S-7S. PMID: 20200260. <http://www.ncbi.nlm.nih.gov/pubmed/20200260>.
- 3032 28. Taylor CL, Carriquiry AL, Bailey RL, Sempos CT, Yetley EA. Appropriateness of the probability
3033 approach with a nutrient status biomarker to assess population inadequacy: a study using vitamin D. *Am*
3034 *J Clin Nutr.* 2013;97(1):72-8. PMID: 23097269. <http://www.ncbi.nlm.nih.gov/pubmed/23097269>.
- 3035 29. U.S. Department of Health and Human Services. Food and Drug Administration. Food Labeling:
3036 Revision of the Nutrition and Supplement Facts Labels; Proposed Rule. *Federal Register* [Internet].
3037 March 3, 2014; 79(41). Available from: <http://www.gpo.gov/fdsys/pkg/FR-2014-03-03/pdf/2014-04387.pdf>.
- 3038 30. Golden NH, Abrams SA, Committee on Nutrition. Optimizing bone health in children and adolescents.
3039 *Pediatrics.* 2014;134(4):e1229-43. PMID: 25266429. <http://www.ncbi.nlm.nih.gov/pubmed/25266429>.
- 3040 31. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation,
3041 treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J*
3042 *Clin Endocrinol Metab.* 2011;96(7):1911-30. PMID: 21646368.
3043 <http://www.ncbi.nlm.nih.gov/pubmed/21646368>.
- 3044 32. National Osteoporosis Foundation. Calcium and Vitamin D: What You Need to Know. Available from:
3045 <http://nof.org/articles/10>.
- 3046 33. National Research Council. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty
3047 Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: The National
3048 Academies Press; 2002. Available from: http://www.nap.edu/openbook.php?record_id=10490.
- 3049 34. Institute of Medicine. Strom BL, Yaktine AL, Oria M, editors. Sodium Intake in Populations:
3050 Assessment of Evidence. Washington, DC: The National Academies Press; 2013. Available from:
3051 http://www.nap.edu/openbook.php?record_id=18311.
- 3052 35. Shin JY, Xun P, Nakamura Y, He K. Egg consumption in relation to risk of cardiovascular disease and
3053 diabetes: a systematic review and meta-analysis. *Am J Clin Nutr.* 2013;98(1):146-59. PMID: 23676423.
3054 <http://www.ncbi.nlm.nih.gov/pubmed/23676423>.
- 3055 36. Institute of Medicine. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate,
3056 Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: The National Academies Press;
3057 1998. 592 p.
- 3058 37. WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia: World Health
3059 Organization; 2011. Available from:
3060 http://whqlibdoc.who.int/publications/2011/9789241548335_eng.pdf.
- 3061 38. Hathcock JN, Shao A, Vieth R, Heaney R. Risk assessment for vitamin D. *Am J Clin Nutr.*
3062 2007;85(1):6-18. PMID: 17209171. <http://www.ncbi.nlm.nih.gov/pubmed/17209171>.
- 3063 39. Government of Canada. Health Canada Health Products. Food Branch. Food Directorate. Caffeine in
3064 Foods [guide]. 2012 [updated 2012-02-16]. Available from: <http://www.hc-sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php>.
- 3065 40. Ahluwalia N, Herrick K, Moshfegh A, Rybak M. Caffeine intake in children in the United States and
3066 10-y trends: 2001-2010. *Am J Clin Nutr.* 2014;100(4):1124-32. PMID: 25240076.
3067 <http://www.ncbi.nlm.nih.gov/pubmed/25240076>.

- 3070 41. Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program. National
3071 Cancer Institute; [updated May 22, 2014]. Available from:
3072 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/>.
- 3073 42. ACOG Committee on Obstetric Practice. ACOG Committee Opinion No. 495: Vitamin D: Screening
3074 and supplementation during pregnancy. *Obstet Gynecol.* 2011;118(1):197-8. PMID: 21691184.
3075 [http://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-
3076 Practice/co495.pdf?dmc=1&ts=20141120T1506445693](http://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-Practice/co495.pdf?dmc=1&ts=20141120T1506445693).
- 3077 43. LeFevre ML. Screening for Vitamin D Deficiency in Adults: U.S. Preventive Services Task Force
3078 Recommendation Statement. *Ann Intern Med.* 2014. PMID: 25419853.
3079 <http://annals.org/article.aspx?articleid=1938935>.
- 3080 44. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated US Department
3081 of Agriculture Food Patterns meet goals of the 2010 dietary guidelines. *J Acad Nutr Diet.*
3082 2012;112(10):1648-55. PMID: 22853987.
- 3083 45. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Impact of typical rather
3084 than nutrient-dense food choices in the US Department of Agriculture Food Patterns. *J Acad Nutr Diet.*
3085 2012;112(10):1560-9. PMID: 22906562. <http://www.ncbi.nlm.nih.gov/pubmed/22906562>.
- 3086 46. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food
3087 Guidance System. *J Nutr Educ Behav.* 2006;38(6 Suppl):S78-92. PMID: 17116598.
3088 <http://www.ncbi.nlm.nih.gov/pubmed/17116598>.
- 3089 47. Marcoe K, Juan W, Yamini S, Carlson A, Britten P. Development of food group composites and
3090 nutrient profiles for the MyPyramid Food Guidance System. *J Nutr Educ Behav.* 2006;38(6 Suppl):S93-
3091 S107. PMID: 17116599. <http://www.ncbi.nlm.nih.gov/pubmed/17116599>.
- 3092 48. Bowman SA, Clemens JC, Friday JE, Thorig RC, Shimizu M, Barrows BR, et al. Food Patterns
3093 Equivalents Database 2007-08: Methodology and User Guide. Beltsville, MD: Food Surveys Research
3094 Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, U.S. Department of
3095 Agriculture,; 2013. Available from:
3096 http://www.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/FPED_0708.pdf.
- 3097 49. Bowman SA, Clemens JC, Thorig RC, Friday JE, Shimizu M, Moshfegh AJ. Food Patterns
3098 Equivalents Database 2009-10: Methodology and User Guide. Beltsville, MD: Food Surveys Research
3099 Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, U.S. Department of
3100 Agriculture,; 2013. Available from:
3101 http://www.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/FPED_0910.pdf.
- 3102 50. Committee on Nutrition. American Academy of Pediatrics: The use and misuse of fruit juice in
3103 pediatrics. *Pediatrics.* 2001;107(5):1210-3. PMID: 11331711.
3104 <http://www.ncbi.nlm.nih.gov/pubmed/11331711>.
- 3105 51. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What
3106 We Eat in America, NHANES 2009-10. Table 1a. Fruit: Mean Daily Food Patterns Cup Equivalents
3107 Consumed per Individual, by Gender and Age, in the United States, 2009-2010. Available from:
3108 http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/Table_1_FPED_GEN_0910.pdf.
- 3109 52. Dietary Guidelines Advisory Committee. 2010. Report of the Dietary Guidelines Advisory Committee
3110 on the Dietary Guidelines for Americans, to the Secretary of Agriculture and the Secretary of Health
3111 and Human Services Washington, DC: U.S. Department of Agriculture, Agricultural Research Service;
3112 2010.
- 3113 53. Code of Federal Regulation Title 21, parts 136 & 137: Office of the Federal Register. National Archives
3114 and Records Administration.; April 1, 2014. Available from: [http://www.gpo.gov/fdsys/pkg/CFR-2014-
3115 title21-vol2/pdf/CFR-2014-title21-vol2.pdf](http://www.gpo.gov/fdsys/pkg/CFR-2014-title21-vol2/pdf/CFR-2014-title21-vol2.pdf).
- 3116 54. Food and Drug Administration. Food Standards: Amendment of Standards of Identity For Enriched
3117 Grain Products to Require Addition of Folic Acid. *Federal Register.* March 5, 1996;61(44):8781-97.
3118 <http://www.gpo.gov/fdsys/pkg/FR-1996-03-05/pdf/96-5014.pdf>.

- 3119 55. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary
3120 Guidelines for Americans, 2010. Washington, DC: U.S. Government Printing Office; December
3121 2010 Available from: <http://www.health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf>.
- 3122 56. Dietary Guidelines Advisory Committee. 2005. Report of the Dietary Guidelines Advisory Committee
3123 on the Dietary Guidelines for Americans, to the Secretary of Health and Human Services and the
3124 Secretary of Agriculture: U.S. Department of Agriculture, Agricultural Research Service; 2005.
3125 Available from: <http://www.health.gov/dietaryguidelines/dga2005/report/default.htm>.
- 3126 57. Aune D, Norat T, Romundstad P, Vatten LJ. Dairy products and the risk of type 2 diabetes: a systematic
3127 review and dose-response meta-analysis of cohort studies. *Am J Clin Nutr*. 2013;98(4):1066-83. PMID:
3128 23945722. <http://www.ncbi.nlm.nih.gov/pubmed/23945722>.
- 3129 58. Crichton GE, Bryan J, Buckley J, Murphy KJ. Dairy consumption and metabolic syndrome: a
3130 systematic review of findings and methodological issues. *Obes Rev*. 2011;12(5):e190-201. PMID:
3131 21348924. <http://www.ncbi.nlm.nih.gov/pubmed/21348924>.
- 3132 59. Labonte ME, Couture P, Richard C, Desroches S, Lamarche B. Impact of dairy products on biomarkers
3133 of inflammation: a systematic review of randomized controlled nutritional intervention studies in
3134 overweight and obese adults. *Am J Clin Nutr*. 2013;97(4):706-17. PMID: 23446894.
3135 <http://www.ncbi.nlm.nih.gov/pubmed/23446894>.
- 3136 60. Soedamah-Muthu SS, Ding EL, Al-Delaimy WK, Hu FB, Engberink MF, Willett WC, et al. Milk and
3137 dairy consumption and incidence of cardiovascular diseases and all-cause mortality: dose-response
3138 meta-analysis of prospective cohort studies. *Am J Clin Nutr*. 2011;93(1):158-71. PMID: 21068345.
3139 <http://www.ncbi.nlm.nih.gov/pubmed/21068345>.
- 3140 61. Tong X, Dong JY, Wu ZW, Li W, Qin LQ. Dairy consumption and risk of type 2 diabetes mellitus: a
3141 meta-analysis of cohort studies. *Eur J Clin Nutr*. 2011;65(9):1027-31. PMID: 21559046.
3142 <http://www.ncbi.nlm.nih.gov/pubmed/21559046>.
- 3143 62. Wang H, Troy LM, Rogers GT, Fox CS, McKeown NM, Meigs JB, et al. Longitudinal association
3144 between dairy consumption and changes of body weight and waist circumference: the Framingham
3145 Heart Study. *Int J Obes (Lond)*. 2014;38(2):299-305. PMID: 23736371.
3146 <http://www.ncbi.nlm.nih.gov/pubmed/23736371>.
- 3147 63. Heaney RP, Dowell MS, Rafferty K, Bierman J. Bioavailability of the calcium in fortified soy imitation
3148 milk, with some observations on method. *Am J Clin Nutr*. 2000;71(5):1166-9. PMID: 10799379.
3149 <http://www.ncbi.nlm.nih.gov/pubmed/10799379>.
- 3150 64. Usual Dietary Intakes: Food Intakes, US Population, 2001-04: Applied Research Program. National
3151 Cancer Institute; [updated April 2, 2014]. Available from:
3152 <http://appliedresearch.cancer.gov/diet/usualintakes/pop/2001-04/>.
- 3153 65. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What
3154 We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available from:
3155 <http://seprl.ars.usda.gov/Services/docs.htm?docid=23429>.
- 3156 66. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Meals
3157 and Snacks: Distribution of Meal Patterns and Snack Occasions, by Race/Ethnicity and Age. Table 34.
3158 What We Eat in America, NHANES 2009-2010. 2012. Available from:
3159 http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_34_DMP_RAC_09.pdf.
- 3160 67. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Meals
3161 and Snacks: Distribution of Meal Patterns and Snack Occasions, by Family Income. Table 36. What We
3162 Eat in America, NHANES 2009-2010. Available from:
3163 http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_36_DMP_POV_09.pdf.
- 3164 68. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Snacks:
3165 Distribution of Snack Occasions, by Gender and Age. Table 29. What We Eat in America, NHANES
3166 2009-2010. Available from:
3167 http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_29_DSO_GEN_09.pdf.
- 3168 69. Institute of Medicine. Wartella EA, Lichtenstein AH, Yaktine A, Nathan R, editors. Front-of-Package
3169 Nutrition Rating Systems and Symbols: Promoting Healthier Choices. Washington, DC: The National

- 3170 Academies Press; 2011. Available from: [http://www.nap.edu/catalog/13221/front-of-package-nutrition-](http://www.nap.edu/catalog/13221/front-of-package-nutrition-rating-systems-and-symbols-promoting-healthier)
 3171 [rating-systems-and-symbols-promoting-healthier](http://www.nap.edu/catalog/13221/front-of-package-nutrition-rating-systems-and-symbols-promoting-healthier).
- 3172 70. Lin B-H, Guthrie J. Nutritional Quality of Food Prepared at Home and Away From Home, 1977-2008,
 3173 EIB-105: U.S. Department of Agriculture, Economic Research Service; December 2012. Available
 3174 from: <http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib105.aspx>.
- 3175 71. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al. Update of the
 3176 Healthy Eating Index: HEI-2010. *J Acad Nutr Diet*. 2013;113(4):569-80. PMID: 23415502.
 3177 <http://www.ncbi.nlm.nih.gov/pubmed/23415502>.
- 3178 72. Ford ES, Li C, Zhao G, Tsai J. Trends in obesity and abdominal obesity among adults in the United
 3179 States from 1999-2008. *Int J Obes (Lond)*. 2011;35(5):736-43. PMID: 20820173.
 3180 <http://www.ncbi.nlm.nih.gov/pubmed/20820173>.
- 3181 73. Ford ES, Maynard LM, Li C. Trends in mean waist circumference and abdominal obesity among US
 3182 adults, 1999-2012. *JAMA*. 2014;312(11):1151-3. PMID: 25226482.
 3183 <http://www.ncbi.nlm.nih.gov/pubmed/25226482>.
- 3184 74. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United
 3185 States, 2011-2012. *JAMA*. 2014;311(8):806-14. PMID: 24570244.
 3186 <http://www.ncbi.nlm.nih.gov/pubmed/24570244>.
- 3187 75. Ostchega Y, Hughes JP, Terry A, Fakhouri TH, Miller I. Abdominal obesity, body mass index, and
 3188 hypertension in US adults: NHANES 2007-2010. *Am J Hypertens*. 2012;25(12):1271-8. PMID:
 3189 22895451. <http://www.ncbi.nlm.nih.gov/pubmed/22895451>.
- 3190 76. Saydah S, Bullard KM, Cheng Y, Ali MK, Gregg EW, Geiss L, et al. Trends in cardiovascular disease
 3191 risk factors by obesity level in adults in the United States, NHANES 1999-2010. *Obesity (Silver*
 3192 *Spring)*. 2014. PMID: 24733690. <http://www.ncbi.nlm.nih.gov/pubmed/24733690>.
- 3193 77. Liu LL, Lawrence JM, Davis C, Liese AD, Pettitt DJ, Pihoker C, et al. Prevalence of overweight and
 3194 obesity in youth with diabetes in USA: the SEARCH for Diabetes in Youth study. *Pediatr Diabetes*.
 3195 2010;11(1):4-11. PMID: 19473302. <http://www.ncbi.nlm.nih.gov/pubmed/19473302>.
- 3196 78. Blackwell D, Lucas J, Clarke T. Summary health statistics for U.S. adults: National Health Interview
 3197 Survey, 2012. *Vital Health Stat 10(260)*. National Center for Health Statistics; 2014. Available from:
 3198 http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.
- 3199 79. Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and
 3200 type 2 diabetes among children and adolescents from 2001 to 2009. *JAMA*. 2014;311(17):1778-86.
 3201 PMID: 24794371. <http://www.ncbi.nlm.nih.gov/pubmed/24794371>.
- 3202 80. SEER Stat Fact Sheets: Breast Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June 15].
 3203 Available from: <http://seer.cancer.gov/statfacts/html/breast.html>.
- 3204 81. Canfield MA, Mai CT, Wang Y, O'Halloran A, Marengo LK, Olney RS, et al. The association between
 3205 race/ethnicity and major birth defects in the United States, 1999-2007. *Am J Public Health*.
 3206 2014;104(9):e14-23. PMID: 25033129. <http://www.ncbi.nlm.nih.gov/pubmed/25033129>.
- 3207 82. Alzheimer's Association. 2014 Alzheimer's disease facts and figures: includes a special report on
 3208 women and Alzheimer's disease. *Alzheimers Dement*. 2014;10(2):131-68. PMID: 22404854.
 3209 http://www.alz.org/downloads/facts_figures_2014.pdf.
- 3210 83. Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The Recent Prevalence of
 3211 Osteoporosis and Low Bone Mass in the United States Based on Bone Mineral Density at the Femoral
 3212 Neck or Lumbar Spine. *J Bone Miner Res*. 2014. PMID: 24771492.
 3213 <http://www.ncbi.nlm.nih.gov/pubmed/24771492>.
- 3214 84. Howlader N, Noone A, Krapcho M, Garshell J, Miller D, Altekruse S, et al. SEER Cancer Statistics
 3215 Review, 1975-2011. Bethesda, MD National Cancer Institute; November 2013 [updated May 12, 2014].
 3216 Available from: http://seer.cancer.gov/csr/1975_2011/.
- 3217 85. SEER Stat Fact Sheets: Prostate Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June
 3218 15]. Available from: <http://seer.cancer.gov/statfacts/html/prost.html>.
- 3219 86. SEER Stat Fact Sheets: Colon and Rectum Cancer. Bethesda, MD: National Cancer Institute; [cited
 3220 2014 June 15]. Available from: <http://seer.cancer.gov/statfacts/html/colorect.html>.

- 3221 87. SEER Stat Fact Sheets: Lung and Bronchus Cancer. Bethesda, MD: National Cancer Institute; [cited
3222 2014 June 15]. Available from: <http://seer.cancer.gov/statfacts/html/lungb.html>.
- 3223 88. Centers for Disease Control and Prevention. Facts about Birth Defects [updated October 20, 2014].
3224 Available from: <http://www.cdc.gov/ncbddd/birthdefects/facts.html>.
- 3225 89. Centers for Disease Control and Prevention. Division of Birth Defects and Developmental Disabilities.
3226 Congenital Heart Defects. Data and Statistics. Atlanta, GA [updated July 9, 2014; cited 2014 September
3227 2]. Available from: <http://www.cdc.gov/ncbddd/heartdefects/data.html>.
- 3228 90. Centers for Disease Control and Prevention. QuickStats: Prevalence of Current Depression Among
3229 Persons Aged \geq 12 Years, by Age Group and Sex United States, National Health and Nutrition
3230 Examination Survey, 2007-2010. Morbidity and Mortality Weekly Report (MMWR).
3231 2014;60(51):1747.
3232 http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.
- 3233 91. Pratt LA, Brody DJ. Depression in the United States household population, 2005-2006. NCHS Data
3234 Brief. 2008(7):1-8. PMID: 19389321. <http://www.cdc.gov/nchs/data/databriefs/db07.pdf>.
- 3235 92. Hebert LE, Weuve J, Scherr PA, Evans DA. Alzheimer disease in the United States (2010-2050)
3236 estimated using the 2010 census. *Neurology*. 2013;80(19):1778-83. PMID: 23390181.
3237 <http://www.ncbi.nlm.nih.gov/pubmed/23390181>.
- 3238 93. Buckland G, Agudo A, Travier N, Huerta JM, Cirera L, Tormo MJ, et al. Adherence to the
3239 Mediterranean diet reduces mortality in the Spanish cohort of the European Prospective Investigation
3240 into Cancer and Nutrition (EPIC-Spain). *Br J Nutr*. 2011;106(10):1581-91. PMID: 21736834.
3241 <http://www.ncbi.nlm.nih.gov/pubmed/21736834>.
- 3242 94. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of
3243 cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90. PMID:
3244 23432189. <http://www.ncbi.nlm.nih.gov/pubmed/23432189>.
- 3245 95. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean diet and
3246 incidence of and mortality from coronary heart disease and stroke in women. *Circulation*.
3247 2009;119(8):1093-100. PMID: 19221219. <http://www.ncbi.nlm.nih.gov/pubmed/19221219>.
- 3248 96. George SM, Ballard-Barbash R, Manson JE, Reedy J, Shikany JM, Subar AF, et al. Comparing indices
3249 of diet quality with chronic disease mortality risk in postmenopausal women in the Women's Health
3250 Initiative Observational Study: evidence to inform national dietary guidance. *Am J Epidemiol*.
3251 2014;180(6):616-25. PMID: 25035143. <http://www.ncbi.nlm.nih.gov/pubmed/25035143>.
- 3252 97. Karanja NM, Obarzanek E, Lin PH, McCullough ML, Phillips KM, Swain JF, et al. Descriptive
3253 characteristics of the dietary patterns used in the Dietary Approaches to Stop Hypertension Trial. DASH
3254 Collaborative Research Group. *J Am Diet Assoc*. 1999;99(8 Suppl):S19-27. PMID: 10450290.
3255 <http://www.ncbi.nlm.nih.gov/pubmed/10450290>.
- 3256 98. Martinez-Gonzalez MA, Garcia-Lopez M, Bes-Rastrollo M, Toledo E, Martinez-Lapiscina EH,
3257 Delgado-Rodriguez M, et al. Mediterranean diet and the incidence of cardiovascular disease: a Spanish
3258 cohort. *Nutr Metab Cardiovasc Dis*. 2011;21(4):237-44. PMID: 20096543.
3259 <http://www.ncbi.nlm.nih.gov/pubmed/20096543>.
- 3260 99. Nunez-Cordoba JM, Valencia-Serrano F, Toledo E, Alonso A, Martinez-Gonzalez MA. The
3261 Mediterranean diet and incidence of hypertension: the Seguimiento Universidad de Navarra (SUN)
3262 Study. *Am J Epidemiol*. 2009;169(3):339-46. PMID: 19037007.
3263 <http://www.ncbi.nlm.nih.gov/pubmed/19037007>.
- 3264 100. Romaguera D, Norat T, Mouw T, May AM, Bamia C, Slimani N, et al. Adherence to the Mediterranean
3265 diet is associated with lower abdominal adiposity in European men and women. *J Nutr*.
3266 2009;139(9):1728-37. PMID: 19571036. <http://www.ncbi.nlm.nih.gov/pubmed/19571036>.
- 3267 101. Swain JF, McCarron PB, Hamilton EF, Sacks FM, Appel LJ. Characteristics of the diet patterns tested
3268 in the optimal macronutrient intake trial to prevent heart disease (OmniHeart): options for a heart-
3269 healthy diet. *J Am Diet Assoc*. 2008;108(2):257-65. PMID: 18237574.
3270 <http://www.ncbi.nlm.nih.gov/pubmed/18237574>.

- 3271 102. Brunner EJ, Mosdol A, Witte DR, Martikainen P, Stafford M, Shipley MJ, et al. Dietary patterns and
3272 15-y risks of major coronary events, diabetes, and mortality. *Am J Clin Nutr.* 2008;87(5):1414-21.
3273 PMID: 18469266. <http://www.ncbi.nlm.nih.gov/pubmed/18469266>.
- 3274 103. Fung TT, Schulze M, Manson JE, Willett WC, Hu FB. Dietary patterns, meat intake, and the risk of
3275 type 2 diabetes in women. *Arch Intern Med.* 2004;164(20):2235-40. PMID: 15534160.
3276 <http://www.ncbi.nlm.nih.gov/pubmed/15534160>.
- 3277 104. Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB. Dietary patterns and the risk of coronary heart
3278 disease in women. *Arch Intern Med.* 2001;161(15):1857-62. PMID: 11493127.
3279 <http://www.ncbi.nlm.nih.gov/pubmed/11493127>.
- 3280 105. Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D, Willett WC. Prospective study of major
3281 dietary patterns and risk of coronary heart disease in men. *Am J Clin Nutr.* 2000;72(4):912-21. PMID:
3282 11010931. <http://www.ncbi.nlm.nih.gov/pubmed/11010931>.
- 3283 106. McCullough ML, Feskanich D, Rimm EB, Giovannucci EL, Ascherio A, Variyam JN, et al. Adherence
3284 to the Dietary Guidelines for Americans and risk of major chronic disease in men. *Am J Clin Nutr.*
3285 2000;72(5):1223-31. PMID: 11063453. <http://www.ncbi.nlm.nih.gov/pubmed/11063453>.
- 3286 107. McKeown NM, Meigs JB, Liu S, Wilson PW, Jacques PF. Whole-grain intake is favorably associated
3287 with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring
3288 Study. *Am J Clin Nutr.* 2002;76(2):390-8. PMID: 12145012.
3289 <http://www.ncbi.nlm.nih.gov/pubmed/12145012>.
- 3290 108. Villegas R, Yang G, Gao YT, Cai H, Li H, Zheng W, et al. Dietary patterns are associated with lower
3291 incidence of type 2 diabetes in middle-aged women: the Shanghai Women's Health Study. *Int J*
3292 *Epidemiol.* 2010;39(3):889-99. PMID: 20231261. <http://www.ncbi.nlm.nih.gov/pubmed/20231261>.
- 3293 109. von Ruesten A, Illner AK, Buijsse B, Heidemann C, Boeing H. Adherence to recommendations of the
3294 German food pyramid and risk of chronic diseases: results from the EPIC-Potsdam study. *Eur J Clin*
3295 *Nutr.* 2010;64(11):1251-9. PMID: 20717136. <http://www.ncbi.nlm.nih.gov/pubmed/20717136>.
- 3296 110. Butler LM, Wu AH, Wang R, Koh WP, Yuan JM, Yu MC. A vegetable-fruit-soy dietary pattern
3297 protects against breast cancer among postmenopausal Singapore Chinese women. *Am J Clin Nutr.*
3298 2010;91(4):1013-9. PMID: 20181808. <http://www.ncbi.nlm.nih.gov/pubmed/20181808>.
- 3299 111. Samieri C, Sun Q, Townsend MK, Chiuve SE, Okereke OI, Willett WC, et al. The association between
3300 dietary patterns at midlife and health in aging: an observational study. *Ann Intern Med.*
3301 2013;159(9):584-91. PMID: 24189593. <http://www.ncbi.nlm.nih.gov/pubmed/24189593>.
- 3302 112. Scarmeas N, Stern Y, Tang MX, Mayeux R, Luchsinger JA. Mediterranean diet and risk for Alzheimer's
3303 disease. *Ann Neurol.* 2006;59(6):912-21. PMID: 16622828.
3304 <http://www.ncbi.nlm.nih.gov/pubmed/16622828>.
- 3305 113. Juan WY, Yamini S, Britten B. Food intake patterns of self-identified vegetarians among the U.S.
3306 population, 2007-2010. 38th Nutrient Data Bank Conference. May 2014.
3307 http://www.nutrientdataconf.org/PastConf/NDBC38/NNDC38_PosterAbstracts.pdf.
- 3308 114. McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB, et al. Diet quality
3309 and major chronic disease risk in men and women: moving toward improved dietary guidance. *Am J*
3310 *Clin Nutr.* 2002;76(6):1261-71. PMID: 12450892. <http://www.ncbi.nlm.nih.gov/pubmed/12450892>.
- 3311
- 3312

3313 **Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends—Tables**

Table Number	Table Title
<u>Nutrients of Concern</u>	
Table D1.1	Mean intake of shortfall and overconsumed nutrients by age and race/ethnicity, for all ages 2+ WWEIA NHANES 2009-10.
Table D1.2	Usual Intakes from Food and Beverages 2007-2010 compared to Dietary Reference Intakes -- females 19-50 years old by pregnancy status. Mean intake and % below EAR, AI, or above UL from food and beverages, WWEIA NHANES 2007-10.
Table D1.3.	Mean intake of nutrients of public health concern by income as a % of the poverty threshold, for all ages 2+ WWEIA NHANES 2009-10
Table D1.4	Prevalence (%) of serum 25-hydroxyvitamin D (25(OH)D) concentration levels for the U.S. population aged 1 year and older, NHANES 2003 -2006.
Table D1.5	Vitamin D: Food sources ranked by amounts of vitamin D and energy per standard food portions and per 100 grams of foods
Table D1.6.	Calcium: Food sources ranked by amounts of calcium and energy per standard food portions and per 100 grams of foods
Table D1.7.	Potassium: Food sources ranked by amounts of potassium and energy per standard food portions and per 100 grams of foods
Table D1.8.	Dietary fiber: Food sources ranked by amounts of dietary fiber and energy per standard food portions and per 100 grams of foods
Table D1.9.	Iron: Food sources ranked by amounts of iron and energy per standard food portions and per 100 grams of foods
<u>Food Groups</u>	
Table D1.10.	USDA Food Intake Patterns—recommended daily intake amounts, weekly amounts for Vegetable and Protein Foods subgroups.
Table D1.11.	Energy levels used for assignment of individuals to USDA Food Intake Patterns
<u>Food Categories</u>	
Table D1.12.	Percent of total energy intake from the 32 as-consumed food subcategories, NHANES 2009-10.
<u>Eating Behaviors</u>	
Table D1.13.	Percent of individuals consuming 1, 2, or 3 meals per day, and number of snacks consumed, by age/sex groups, NHANES 2009-10.
Table D1.14.	Percent of individuals skipping specific meals, by age/sex groups, NHANES 2009-10.
Table D1.15.	Meal and snack intake over time--percent reporting consumption of each meal, by age/sex group, NHANES 2005-06 to 2009-10.
Table D1.16.	Percent of energy from each meal and snack occasion over time, by age/sex group, NHANES 2005-06 to 2009-10.
Table D1.17.	Percent of nutrient intake from snacks by age/sex group, NHANES 2009-10.
Table D1.18.	Vegetable density (cups per 1000 calorie) for all vegetable subgroups, by point of purchase, NHANES 2003-04 to 2009-10.
<u>Health Conditions</u>	
Table D1.19	Body mass index (BMI), by sex, age, and race/ethnicity, adults 20 years and older, NHANES 2009 -2012
Table D1.20	Percent of overweight and obesity by income in relation to poverty level, adults 20 years and above
Table D1.21	Trends in prevalence of abdominal obesity among adults, by age, sex and race/ethnicity, NHANES*

Table D1.22	Body Mass Index (BMI) Among Children and Adolescents Ages 2-19 years, NHANES 2009-2012
Table D1.23	Hypertension, lipid profile and diabetes by body mass index (BMI) and waist circumference, adults ages 20 years and older, NHANES 2009 -2012
Table D1.24	Lipid profile by weight status, among children and adolescents, NHANES 2009 -2012.
Table D1.25	Prevalence of High and Borderline High Blood Pressure in Children, NHANES 2009 -2012
Table D1.26	Prevalence of overweight and obesity among youth ages 3 to 19 with type 2 diabetes by race and ethnicity , compared to non-diabetic youth, SEARCH population, 2001 -2004
TableD1.27	Prevalence of hypertension and diabetes in US adults, NHANES 2009 -2012
Table D1.28	Prevalence of type 2 diabetes by sex, age, and race/ethnicity in children and adolescents
Table D1.29	Cancer incidence and death rates by age category, sex and race and ethnicity, United States, 2007 -2011.
Table D1.30	Estimates of the prevalence and number of US adults ages 50 years and older with osteoporosis (OP) and low bone mass (LBM) at either the femoral neck or lumber spine, NHANES 2005-2010.

<u>Dietary Patterns</u>	
<u>Composition</u>	
Table D1.31	Studies included in the analysis of Dietary Patterns Composition. Abbreviations listed below are used in Figures D1.56 to D1.60.
Table D1.32.	Composition of three USDA Food Patterns (Healthy US Style, Healthy Vegetarian, and Healthy Mediterranean-style) at the 2000 calorie level. Daily or weekly amounts from selected food groups, subgroups, and components.
Table D1.33.	Nutrients in the three USDA Food Patterns (Healthy US Style, Healthy Vegetarian, and Healthy Mediterranean-style) at the 2000 calorie level as a percent of the goal or limit for a 19 to 30 year old woman.

Table D1.1 Mean intake of shortfall* and overconsumed nutrients by age and race/ethnicity, for all ages 2+ WWEIA NHANES 2009-10.**

Race/ethnicity and age	n	Vit A* (RAE) µg	Vit D* µg	Vit E* µg	Vit C* mg	Folate* (DFE) µg	Calcium* mg	Magne-sium* mg	Iron* mg	Potas-sium* mg	Dietary fiber* g	Saturated fat** g	Sodium** mg
Ages 2 to 5													
Non-Hispanic White	305	606	6.9	4.8	77.3	405	1081	214	11.2	2070	11.7	21.0	2295
Non-Hispanic Black	150	537	5.8	5.5	86.5	447	879	196	12.6	1956	11.2	19.8	2492
Mexican-American	237	644	7.3	4.3	84.8	450	1057	210	11.8	2141	12.1	19.4	2157
All Hispanic	332	606	7.2	4.4	92.2	439	1031	209	11.5	2144	11.7	18.7	2189
Ages 6 to 11													
Non-Hispanic White	371	618	6.3	5.9	64.9	519	1083	231	13.4	2151	13.6	23.2	2920
Non-Hispanic Black	229	582	5.3	6.2	96.1	526	981	227	14.4	2216	14	23.7	3032
Mexican-American	337	545	6	5.5	78.9	501	970	230	13.9	2175	15.3	22.6	2824
All Hispanic	474	550	5.9	5.5	78.4	518	985	231	13.9	2180	14.7	23.1	2913
Ages 12 to 19													
Non-Hispanic White	425	611	5.9	7.2	67.5	578	1142	262	15.2	2364	14.3	27.7	3584
Non-Hispanic Black	275	502	4.1	7.2	106.7	498	974	234	14.1	2204	13	27.2	3348
Mexican-American	340	518	5	6.7	103.7	538	1074	267	15.4	2431	16.1	25.4	3454
All Hispanic	482	540	5.3	6.9	97.9	565	1081	265	15.7	2411	15.9	25.3	3434
Ages 20 and older													
Non-Hispanic White	2786	682	5.4	8.4	86	559	1070	315	15.6	2868	17.3	26.9	3627
Non-Hispanic Black	1025	555	4.1	6.8	92.4	464	828	261	14.0	2364	13.6	25.2	3358
Mexican-American	1062	537	4.9	6.8	97.8	525	975	320	15.1	2758	20.0	23.7	3368
All Hispanic	1647	525	4.8	6.7	100.9	530	969	307	14.8	2711	18.4	23.6	3417
Ages 2 and older													
Non-Hispanic White	3887	667	5.6	8.0	82.2	551	1079	299	15.2	2728	16.4	26.5	3511
Non-Hispanic Black	1679	549	4.3	6.7	94.3	473	865	251	14.0	2304	13.4	25.0	3273
Mexican-American	1976	545	5.3	6.4	95.2	518	997	291	14.7	2583	18.1	23.4	3206
All Hispanic	2935	537	5.2	6.4	97.1	526	992	284	14.5	2556	17.0	23.3	3252

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors, more nutrients and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.2 Usual Intakes from Food and Beverages compared to Dietary Reference Intakes -- females 19-50 years old by pregnancy status. Mean intake and % below EAR, AI, or above UL from food and beverages, WWEIA NHANES 2007-10.

Nutrient	Pregnancy status**	n	Mean	EAR	% Below EAR	UL	% Above UL
Energy (calorie/day)	Non-pregnant	2957	1848				
	Pregnant	133	2131				
Protein (g/day)	Non-pregnant	2957	69.4				
	Pregnant	133	78.6				
Dietary Fiber (g/day)	Non-pregnant	2957	14.4	25	5		
	Pregnant	133	17.3	28	8*		
Vitamin A (µg RAE/day)	Non-pregnant	2957	549	500	48	3000	<3
	Pregnant	133	728	550	26*	3000	<3
Folate (µg DFE/day)	Non-pregnant	2957	470	320	15	1000	<3
	Pregnant	133	622	520	29*	1000	<3
Vitamin C (mg/day)	Non-pregnant	2957	76.6	60	45	2000	<3
	Pregnant	133	121.0	70	30	2000	<3
Vitamin D (µg/day)	Non-pregnant	2957	3.9	10	>97	100	<3
	Pregnant	133	5.6	10	90*	100	<3
Vitamin E -ATE (mg/day)	Non-pregnant	2957	6.9	12	95		
	Pregnant	133	7.4	12	94*		
Calcium (mg/day)	Non-pregnant	2957	885	800	43	2500	<3
	Pregnant	133	1123	800	24	2500	<3
Iron (mg/day)	Non-pregnant	2957	13.2	8.1	16	45	<3
	Pregnant	133	16.9	22	96*	45	<3
				AI		UL	
Potassium (mg/day)	Non-pregnant	2957	2277	4700	<3		
	Pregnant	133	2660	4700	<3		
Sodium (mg/day) (overconsumed nutrient)	Non-pregnant	2957	3111	1500	>97	2300	84
	Pregnant	133	3523	1500	>97	2300	>97

The values flagged with an asterisk () may be less reliable; interpret with caution **Non-pregnant includes non-lactating.

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. For more detailed tables and standard errors, see usual intake tables for pregnant women in *Appendix E-2.4*.

Table D1.3. Mean intake of nutrients of public health concern by income as a percentage of the poverty threshold, for all ages 2+ WWEIA NHANES 2009-10

Income as % of poverty level and age		Dietary fiber	Vitamin D	Calcium	Potassium
	n	g	µg	mg	mg
Less than 131% poverty:					
Ages 2-5	431	10.9	6.9	992	2036
Ages 6-11	496	13.9	6.3	1073	2254
Ages 12-19	503	14.1	5.4	1060	2319
Ages 20+	1755	15.5	4.7	942	2564
Ages 2+	3185	14.8	5.2	977	2451
131-185% poverty:					
Ages 2-5	93	12.3	6.8	1090	2160
Ages 6-11	145	12.9	5.8	955	2062
Ages 12-19	162	13.4	3.8	939	2096
Ages 20+	743	15.6	4.7	971	2638
Ages 2+	1143	14.9	4.8	973	2499
Over 185% poverty:					
Ages 2-5	266	12.3	6.8	1057	2070
Ages 6-11	422	14.2	5.9	1052	2134
Ages 12-19	482	14.6	5.8	1126	2417
Ages 20+	2730	17.7	5.3	1053	2866
Ages 2+	3900	16.9	5.5	1061	2735

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors, more nutrients and documentation, see:

<http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.4 Prevalence (%) of serum 25-hydroxyvitamin D (25(OH)D) concentration levels for the U.S. population aged 1 year and older, NHANES 2003 -2006

	Serum 25(OH)D < 30 nmol/L*	Serum 25(OH)D < 40 nmol/L*	Serum 25(OH)D 30 -< 50 nmol/L*	Serum 25(OH)D > 125 nmol/L*
	%(95% conf interval)	%(95% conf interval)	%(95% conf interval)	%(95% conf interval)
Total, 1 year and older	8.1 (6.7 – 9.8)	17.2 (14.7 – 20.0)	23.6 (21.6 – 25.8)	0.9 (0.6 – 1.2)
Sex				
Male	6.3 (5.0 – 7.9)	14.6 (12.3 – 17.4)	23.1 (20.8 – 25.6)	0.4 (0.3 – 0.7)
Female	9.9 (8.1 – 11.9)	19.6 (16.9 – 22.7)	24.1 (22.1 – 26.3)	1.3 (0.9 – 1.9)
Age category (years)				
1 to 5	0.7 (0.4 – 1.3)	2.7 (1.8 – 4.0)	8.9 (7.1 – 11.0)	§
6 to 11	1.8 (1.3 – 2.6)	5.7 (4.2 – 7.7)	14.1 (11.5 – 17.2)	§
12 to 19	8.5 (6.5 – 11.2)	17.1 (13.8 – 21.0)	24.2 (21.3 – 27.3)	1.4 (0.9 – 2.1)
20 -39	9.5 (7.6 – 11.8)	19.7 (16.4 – 23.4)	26.2 (23.6 – 29.0)	1.5 (0.9 – 2.4)
40 -59	9.3 (7.4 – 11.7)	20.0 (16.6 – 23.9)	25.0 (22.2 – 28.0)	0.6‡ (0.3 – 1.2)
60 +	8.8 (7.3 – 10.5)	17.8 (15.5 – 20.4)	25.5 (23.7 – 27.4)	0.3‡ (0.1 – 0.6)
Race/Ethnicity				
Non-Hispanic Whites	3.6 (3.0 – 4.4)	9.4 (7.9 – 11.2)	18.1 (16.2 – 20.2)	1.2 (0.8 – 1.7)
Non-Hispanic Blacks	31.1 (27.4 – 35.1)	51.6 (46.7 – 56.5)	39.5 (37.3 – 41.7)	§
Mexican Americans	11.3 (8.7 – 14.6)	24.4 (20.1 – 29.3)	32.9 (29.6 – 36.4)	§

1 ng/ml = 2.5 nmol/L

* Serum 25(OH)D < 30 nmol/L = risk for deficiency

Serum 25(OH)D < 40 nmol/L = level set by IOM equal to EAR

Serum 25(OH)D between 30 -50 nmol/L = at risk of inadequacy

Serum 25(OH)D > 125 nmol/L = maybe reason for concern about excess

‡ Estimate flagged: $30\% \leq RSE < 40\%$ for the prevalence estimate§ Estimate suppressed: $RSE \geq 40\%$ for the prevalence estimate

Source: Centers for Disease Control and Prevention. Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2012. Available from:

http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.

Table D1.5 Vitamin D: Food sources ranked by amounts of vitamin D and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion ¹	Vitamin D in Standard Portion (µg) ¹	Calories per 100 grams ¹	Vitamin D per 100 grams (µg) ¹
Salmon, sockeye, canned	3 ounces	142	17.9	167	21.0
Trout, rainbow, farmed, cooked	3 ounces	143	16.2	168	19.0
Salmon, chinook, smoked	3 ounces	99	14.5	117	17.1
Swordfish, cooked	3 ounces	146	14.1	172	16.6
Sturgeon, mixed species, smoked	3 ounces	147	13.7	173	16.1
Salmon, pink, canned	3 ounces	117	12.3	138	14.5
Fish oil, cod liver	1 tsp	41	11.3	902	250
Cisco, smoked	3 ounces	150	11.3	177	13.3
Salmon, sockeye, cooked	3 ounces	144	11.1	169	13.1
Salmon, pink, cooked	3 ounces	130	11.1	153	13.0
Sturgeon, mixed species, cooked	3 ounces	115	11.0	135	12.9
Whitefish, mixed species, smoked	3 ounces	92	10.9	108	12.8
Mackerel, Pacific and jack, cooked	3 ounces	171	9.7	201	11.4
Salmon, coho, wild, cooked	3 ounces	118	9.6	139	11.3
Mushrooms, portabella, exposed to UV light, grilled	½ cup	18	7.9	29	13.1
Tuna, light, canned in oil, drained	3 ounces	168	5.7	198	6.7
Halibut, Atlantic and Pacific, cooked	3 ounces	94	4.9	111	5.8
Herring, Atlantic, cooked	3 ounces	173	4.6	203	5.4
Sardine, canned in oil, drained	3 ounces	177	4.1	208	4.8
Rockfish, Pacific, mixed species, cooked	3 ounces	93	3.9	109	4.6
Whole milk ²	1 cup	149	3.2	61	1.3
Whole chocolate milk ²	1 cup	208	3.2	83	1.3
Tilapia, cooked	3 ounces	109	3.1	128	3.7
Flatfish (flounder and sole), cooked	3 ounces	73	3.0	86	3.5
Reduced fat chocolate milk (2%) ²	1 cup	190	3.0	76	1.2
Yogurt (various types and flavors) ²	8 ounces	98-254	2.0-3.0	43-112	0.9-1.3
Milk (non-fat, 1% and 2%) ²	1 cup	83-122	2.9	34-50	1.2
Soy milk ²	1 cup	109	2.9	45	1.2
Low-fat chocolate milk (1%) ²	1 cup	178	2.8	71	1.1
Fortified ready-to-eat cereals (various) ²	1/3 - 1 ¼ cup	74-247	0.2-2.5	248-443	0.8-8.6
Orange juice, fortified ²	1 cup	117	2.5	47	1.0
Almond milk (all flavors) ²	1 cup	91-120	2.4	38-50	1.0
Rice drink ²	1 cup	113	2.4	47	1.0
Pork, cooked (various cuts)	3 ounces	122-390	0.2-2.2	143-459	0.2-2.6
Mushrooms, morel, raw	½ cup	10	1.7	31	5.1
Margarine (various) ²	1 Tbsp	75-100	1.5	533-717	10.7
Mushrooms, Chanterelle, raw	½ cup	10	1.4	38	5.3
Egg, hard-boiled	1 large	78	1.1	155	2.2

¹Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: <http://www.ars.usda.gov/nutrientdata>.

²Vitamin D fortified

Table D1.6. Calcium: Food sources ranked by amounts of calcium and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion ¹	Calcium in Standard Portion (mg) ¹	Calories per 100 grams ¹	Calcium per 100 grams (mg) ¹
Fortified ready-to-eat cereals (various) ²	¾ - 1 ¼ cup	70-197	137-1000	234-394	455-3333
Pasteurized process American cheese	2 ounces	210	593	371	1045
Parmesan cheese, hard	1.5 ounces	167	503	392	1184
Plain yogurt, nonfat	8 ounces	127	452	56	199
Romano cheese	1.5 ounces	165	452	387	1064
Almond milk (all flavors) ²	1 cup	91-120	451	38-50	188
Pasteurized process Swiss cheese	2 ounces	189	438	334	772
Tofu, raw, regular, prepared with calcium sulfate	½ cup	94	434	76	350
Gruyere cheese	1.5 ounces	176	430	413	1011
Vanilla yogurt, low-fat	8 ounces	193	388	85	171
Plain yogurt, low-fat	8 ounces	143	415	63	183
Pasteurized process American cheese food	2 ounces	187	387	330	682
Fruit yogurt, low-fat	8 ounces	238	383	105	169
Orange juice, calcium fortified ²	1 cup	117	349	47	140
Soy milk (all flavors) ²	1 cup	109	340	45	140
Ricotta cheese, part skim	½ cup	171	337	138	272
Swiss cheese	1.5 ounces	162	336	380	791
Evaporated milk	½ cup	170	329	135	261
Sardines, canned in oil, drained	3 ounces	177	325	208	382
Provolone cheese	1.5 ounces	149	321	351	756
Monterey cheese	1.5 ounces	159	317	373	746
Mustard spinach (tendergreen), raw	1 cup	33	315	22	210
Muenster cheese	1.5 ounces	156	305	368	717
Low-fat milk (1%)	1 cup	102	305	42	125
Mozzarella cheese, part-skim	1.5 ounces	128	304	301	716
Skim milk (nonfat)	1 cup	83	299	34	122
Reduced fat milk (2%)	1 cup	122	293	50	120
Colby cheese	1.5 ounces	167	291	394	685
Low-fat chocolate milk (1%)	1 cup	178	290	71	116
Cheddar cheese	1.5 ounces	173	287	406	675
Rice drink ²	1 cup	113	283	47	118
Whole buttermilk	1 cup	152	282	62	115
Whole chocolate milk	1 cup	208	280	83	112
Whole milk	1 cup	149	276	61	113
Reduced fat chocolate milk (2%)	1 cup	190	273	76	109
Ricotta cheese, whole milk	½ cup	216	257	174	207

¹Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: <http://www.ars.usda.gov/nutrientdata>.

²Calcium fortified

Table D1.7. Potassium: Food sources ranked by amounts of potassium and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion¹	Potassium in Standard Portion (mg)¹	Calories per 100 grams¹	Potassium per 100 grams (mg)¹
Potato, baked, flesh and skin	1 medium	163	941	94	544
Prune juice, canned	1 cup	182	707	71	276
Carrot juice, canned	1 cup	94	689	40	292
Passion-fruit juice, yellow or purple	1 cup	126-148	687	51-60	278
Tomato paste, canned	¼ cup	54	669	82	1014
Beet greens, cooked from fresh	½ cup	19	654	27	909
Adzuki beans, cooked	½ cup	147	612	128	532
White beans, canned	½ cup	149	595	114	454
Plain yogurt, nonfat	1 cup	127	579	56	255
Tomato puree	½ cup	48	549	38	439
Sweet potato, baked in skin	1 medium	103	542	90	475
Salmon, Atlantic, wild, cooked	3 ounces	155	534	182	628
Clams, canned	3 ounces	121	534	142	628
Pomegranate juice	1 cup	134	533	54	214
Plain yogurt, low-fat	8 ounces	143	531	63	234
Tomato juice, canned	1 cup	41	527	17	217
Orange juice, fresh	1 cup	112	496	45	200
Soybeans, green, cooked	½ cup	127	485	141	539
Chard, swiss, cooked	½ cup	18	481	20	549
Lima beans, cooked	½ cup	108	478	115	508
Mackerel, various types, cooked	3 ounces	114-171	443-474	134-201	521-558
Vegetable juice, canned	1 cup	48	468	19	185
Chili with beans, canned	½ cup	144	467	112	365
Great northern beans, canned	½ cup	150	460	114	351
Yam, cooked	½ cup	79	456	116	670
Halibut, cooked	3 ounces	94	449	111	528
Tuna, yellowfin, cooked	3 ounces	111	448	130	527
Acorn squash, cooked	½ cup	58	448	56	437
Snapper, cooked	3 ounces	109	444	128	522
Soybeans, mature, cooked	½ cup	149	443	173	515
Tangerine juice, fresh	1 cup	106	440	43	178
Pink beans, cooked	½ cup	126	430	149	508
Chocolate milk (1%, 2% and whole)	1 cup	178-208	418-425	71-83	167-170
Amaranth leaves, cooked	½ cup	14	423	21	641
Banana	1 medium	105	422	89	358
Spinach cooked from fresh or canned	½ cup	21-25	370-419	23	346-466
Black turtle beans, cooked	½ cup	121	401	130	433
Peaches, dried, uncooked	¼ cup	96	399	239	996
Prunes, stewed	½ cup	133	398	107	321
Rockfish, Pacific, cooked	3 ounces	93	397	109	467
Rainbow trout, wild or farmed, cooked	3 ounces	128-143	381-383	150-168	448-450
Skim milk (nonfat)	1 cup	83	382	34	156
Refried beans, canned, traditional	½ cup	106	380	89	319

Table D1.7. Potassium, continued

Food	Standard Portion Size	Calories in Standard Portion¹	Potassium in Standard Portion (mg)¹	Calories per 100 grams¹	Potassium per 100 grams (mg)¹
Apricots, dried, uncooked	¼ cup	78	378	241	1162
Pinto beans, cooked	½ cup	123	373	143	436
Lentils, cooked	½ cup	115	365	116	369
Avocado	½ cup	120	364	160	485
Tomato sauce, canned	½ cup	30	364	24	297
Plantains, slices, cooked	½ cup	89	358	116	465
Kidney beans, cooked	½ cup	113	357	127	403
Navy beans, cooked	½ cup	128	354	140	389

¹Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: <http://www.ars.usda.gov/nutrientdata>.

Table D1.8. Dietary fiber: Food sources ranked by amounts of dietary fiber and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion¹	Dietary fiber in Standard Portion (g)¹	Calories per 100 grams¹	Dietary fiber per 100 grams (g)¹
High fiber bran ready-to eat-cereal	1/3 – 3/4 cup	60-81	9.1-14.3	200-260	29.3-47.5
Navy beans, cooked	1/2 cup	127	9.6	140	10.5
Small white beans, cooked	1/2 cup	127	9.3	142	10.4
Yellow beans, cooked	1/2 cup	127	9.2	144	10.4
Shredded wheat ready-to-eat cereal (various)	1-1 1/4 cup	155-220	5.0-9.0	321-373	9.6-15.0
Cranberry (roman) beans, cooked	1/2 cup	120	8.9	136	10.0
Adzuki beans, cooked	1/2 cup	147	8.4	128	7.3
French beans, cooked	1/2 cup	114	8.3	129	9.4
Split peas, cooked	1/2 cup	114	8.1	116	8.3
Chickpeas, canned	1/2 cup	176	8.1	139	6.4
Lentils, cooked	1/2 cup	115	7.8	116	7.9
Pinto beans, cooked	1/2 cup	122	7.7	143	9.0
Black turtle beans, cooked	1/2 cup	120	7.7	130	8.3
Mung beans, cooked	1/2 cup	106	7.7	105	7.6
Black beans, cooked	1/2 cup	114	7.5	132	8.7
Artichoke, globe or French, cooked	1/2 cup	45	7.2	53	8.6
Lima beans, cooked	1/2 cup	108	6.6	115	7.0
Great northern beans, canned	1/2 cup	149	6.4	114	4.9
White beans, canned	1/2 cup	149	6.3	114	4.8
Kidney beans, all types, cooked	1/2 cup	112	5.7	127	6.4
Pigeon peas, cooked	1/2 cup	102	5.6	121	6.7
Cowpeas, cooked	1/2 cup	99	5.6	116	6.5
Wheat bran flakes ready-to-eat cereal (various)	3/4 cup	90-98	4.9-5.5	310-328	16.9-18.3
Pear	1 medium	101	5.5	57	3.1
Pumpkin seeds, whole, roasted	1 ounce	126	5.2	446	18.4
Baked beans, canned, plain	1/2 cup	119	5.2	94	4.1
Soybeans, cooked	1/2 cup	149	5.2	173	6.0
Plain rye wafer crackers	2 wafers	73	5.0	334	22.9
Avocado	1/2 cup	120	5.0	160	6.7
Broadbeans (fava beans), cooked	1/2 cup	94	4.6	110	5.4
Pink beans, cooked	1/2 cup	126	4.5	149	5.3
Apple, with skin	1 medium	95	4.4	52	2.4
Green peas, cooked (frsh, frzn, cnd)	1/2 cup	59-67	3.5-4.4	69-84	4.1-5.5
Refried beans, canned	1/2 cup	107	4.4	90	3.7
Chia seeds, dried	1 Tbsp	58	4.1	486	34.4
Bulgur, cooked	1/2 cup	76	4.1	83	4.5
Mixed vegetables, cooked from frozen	1/2 cup	59	4.0	65	4.4
Raspberries	1/2 cup	32	4.0	52	6.5
Blackberries	1/2 cup	31	3.8	43	5.3
Collards, cooked	1/2 cup	32	3.8	33	4.0

Table D1.8. Dietary fiber, continued

Food	Standard Portion Size	Calories in Standard Portion¹	Dietary fiber in Standard Portion (g)¹	Calories per 100 grams¹	Dietary fiber per 100 grams (g)¹
Soybeans, green, cooked	½ cup	127	3.8	141	4.2
Prunes, stewed	½ cup	133	3.8	107	3.1
Sweet potato, baked in skin	1 medium	103	3.8	90	3.3
Figs, dried	¼ cup	93	3.7	249	9.8
Pumpkin, canned	½ cup	42	3.6	34	2.9
Potato, baked, with skin	1 medium	163	3.6	94	2.1
Popcorn, air-popped	3 cups	93	3.5	387	14.5
Almonds	1 ounce	164	3.5	579	12.5
Pears, dried	¼ cup	118	3.4	262	7.5
Whole wheat spaghetti, cooked	½ cup	87	3.2	124	4.5
Parsnips, cooked	½ cup	55	3.1	71	4.0
Sunflower seed kernels, dry roasted	1 ounce	165	3.1	582	11.1
Orange	1 medium	69	3.1	49	2.2
Banana	1 medium	105	3.1	89	2.6
Guava	1 fruit	37	3.0	68	5.4
Oat bran muffin	1 small	178	3.0	270	4.6
Pearled barley, cooked	½ cup	97	3.0	123	3.8
Winter squash, cooked	½ cup	38	2.9	37	2.8
Dates	¼ cup	104	2.9	282	8.0
Pistachios, dry roasted	1 ounce	161	2.8	567	9.9
Pecans, oil roasted	1 ounce	203	2.7	715	9.5
Hazelnuts or filberts	1 ounce	178	2.7	628	9.7
Peanuts, oil roasted	1 ounce	170	2.7	599	9.4
Whole wheat paratha bread	1 ounce	92	2.7	326	9.6
Quinoa, cooked	½ cup	111	2.6	120	2.8

¹Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: <http://www.ars.usda.gov/nutrientdata> .

Table D1.9. Iron: Food sources ranked by amounts of iron and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion ¹	Iron in Standard Portion (mg) ¹	Calories per 100 grams ¹	Iron per 100 grams (mg) ¹
Organ meats (spleen, liver, giblets, heart, kidney or lung) various, cooked	3 ounces	84-235	4.5-33.5	99-277	5.3-39.4
Fortified ready-to-eat cereals (various)	½ -1 ½ cup	89-230	5.1-19.6	310-443	19.4-67.7
Fortified instant cereals (various), prepared	1 cup	174-241	5.1-14.7	62-96	2.1-6.7
Clams, cooked, breaded and fried	3 ounces	172	11.8	202	13.9
Octopus, cooked, moist heat	3 ounces	139	8.1	164	9.5
Coconut milk, canned	1 cup	445	7.5	197	3.3
Tofu, raw, regular, prep. w/ Ca sulfate	½ cup	94	6.6	76	5.4
Oysters, eastern, wild/farmed, cooked, dry heat	3 ounces	67	6.1-6.6	79	7.2-7.8
Oysters, cooked, breaded and fried	3 ounces	169	5.9	199	7.0
Mussels, blue, cooked, moist heat	3 ounces	146	5.7	172	6.7
Liverwurst spread	¼ cup	168	4.9	305	8.9
Soybeans, mature, cooked	½ cup	149	4.4	173	5.1
Chili with beans, canned	½ cup	128	4.4	112	3.4
Beef, plate steak, boneless, outside skirt, all grades, grilled ²	3 ounces	240-248	4.3-4.4	282-292	5.1-5.2
Mushrooms, morel, raw	½ cup	10	4.0	31	12.2
White beans, canned or cooked	½ cup	125-149	3.3-3.9	114-139	3.0-3.7
Lentils, cooked	½ cup	115	3.3	116	3.3
Spinach, cooked from fresh, frzn or cnd	½ cup	21-32	1.9-3.2	23-34	2.0-3.6
Beef, shoulder pot roast, boneless, 0" fat, all grades, braised ²	3 ounces	167-173	3.1	196-204	3.5-3.6
Beef, loin, tenderloin steak, boneless, 0" fat, all grades, grilled ²	3 ounces	168-179	2.7-3.0	198-211	3.2-3.6
Ground beef (95% lean/5% fat), cooked	3 ounces	164	2.8	193	3.2
Black turtle beans, cooked	½ cup	121	2.7	130	2.9
Kidney beans, cooked	½ cup	113	2.6	127	2.9
Sardines, canned in oil, drained	3 ounces	177	2.5	208	2.9
Bagel, enriched	1 sm (3" dia)	182	2.5	264	3.6
Chickpeas, cooked	½ cup	134	2.4	164	2.9
Pumpkin/squash seed kernels, roasted	1 ounce	163	2.3	574	8.1
Adzuki beans, cooked	½ cup	147	2.3	128	2.0
Hearts of palm, canned	½ cup	21	2.3	28	3.1
Yardlong beans, cooked	½ cup	101	2.3	118	2.6
Lima beans, cooked	½ cup	108	2.3	115	2.4
Tomato puree, canned	½ cup	48	2.3	38	1.8
Navy beans, cooked	½ cup	127	2.2	140	2.4
Cowpeas, cooked	½ cup	100	2.2	116	2.5

¹Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: <http://www.ars.usda.gov/nutrientdata>.

²Lean and fat or lean only

Table D1.10. USDA Food Intake Patterns (Healthy U.S.-Style Patterns) recommended daily intake amounts, weekly amounts for vegetable and protein foods subgroups.

Energy Level of Pattern*	1,000	1,200	1,400	1,600	1,800	2,000	2,200	2,400	2,600	2,800	3,000	3,200
Food Group												
Fruits	1 c	1 c	1½ c	1½ c	1½ c	2 c	2 c	2 c	2 c	2½ c	2½ c	2½ c
Vegetables	1 c	1½ c	1½ c	2 c	2½ c	2½ c	3 c	3 c	3½ c	3½ c	4 c	4 c
Dark green vegetables (c/wk)	½	1	1	1½	1½	1½	2	2	2½	2½	2½	2½
Red/Orange vegetables (c/wk)	2½	3	3	4	5½	5½	6	6	7	7	7½	7½
Dry beans and peas(c/wk)	½	½	½	1	1½	1½	2	2	2½	2½	3	3
Starchy vegetables (c/wk)	2	3½	3½	4	5	5	6	6	7	7	8	8
Other vegetables (c/wk)	1½	2½	2½	3½	4	4	5	5	5½	5½	7	7
Grains	3 oz eq	4 oz eq	5 oz eq	5 oz eq	6 oz eq	6 oz eq	7 oz eq	8 oz eq	9 oz eq	10 oz eq	10 oz eq	10 oz eq
Whole grains	1½ oz eq	2 oz eq	2½ oz eq	3 oz eq	3 oz eq	3 oz eq	3½ oz eq	4 oz eq	4½ oz eq	5 oz eq	5 oz eq	5 oz eq
Other grains	1½ oz eq	2 oz eq	2½ oz eq	2 oz eq	3 oz eq	3 oz eq	3½ oz eq	4 oz eq	4½ oz eq	5 oz eq	5 oz eq	5 oz eq
Protein Foods	2 oz eq	3 oz eq	4 oz eq	5 oz eq	5 oz eq	5½ oz eq	6 oz eq	6½ oz eq	6½ oz eq	7 oz eq	7 oz eq	7 oz eq
Meat, poultry, eggs (oz/wk)	10	14	19	23	23	26	28	31	31	33	33	33
Seafood (oz/wk)	3	4	6	8	8	8	9	10	10	10	10	10
Nuts seeds, soy (oz/wk)	2	2	3	4	4	5	5	5	5	6	6	6
Dairy	2 c	2.5 c	2.5 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c
Oils	15 g	17 g	17 g	22 g	24 g	27 g	29 g	31 g	34 g	36 g	44 g	51g
Limits for:												
Solid fats	10g	7g	7g	8g	11g	18g	18g	23g	25g	26g	31g	40g
Added Sugars	17g	12g	13g	14g	19g	30g	32g	39g	43g	45g	53g	69g

*Food group amounts shown in cup (c) or ounce equivalents (oz eq). Oils, solid fats, and added sugars are shown in grams (g).

Notes continue on next page.

Table D1.10. USDA Food Intake Patterns (Healthy U.S.-Style Patterns), continued

Quantity equivalents for each food group are:

- Grains, 1 ounce equivalent is: ½ cup cooked rice, pasta, or cooked cereal; 1 ounce dry pasta or rice; 1 slice bread; 1 small muffin (1 oz); 1 cup RTE cereal flakes.
- Fruits and vegetables, 1 cup equivalent is: 1 cup raw or cooked fruit or vegetable, 1 cup fruit or vegetable juice, 2 cups leafy salad greens.
- Protein Foods, 1 ounce equivalent is: 1 ounce lean meat, poultry, or fish; 1 egg; ¼ cup cooked dry beans or tofu; 1 Tbsp peanut butter; ½ ounce nuts or seeds.
- Milk, 1 cup equivalent is: 1 cup milk or yogurt, 1½ ounces natural cheese such as Cheddar cheese or 2 ounces of processed cheese.

Source: Center for Nutrition Policy and Promotion, USDA. USDA Food Patterns. For more information see Appendix E-3.1: Adequacy of the USDA Food Patterns

Table D1.11. Energy levels used for assignment of individuals to USDA Food Intake Patterns

Males, age	Sedentary¹ Male^s	Moderately Active² Male	Active³ Male	Females, age	Sedentary¹ Female	Moderately Active² Female	Active³ Female
2	1000	1000	1000	2	1000	1000	1000
3	1000	1400	1400	3	1000	1200	1400
4	1200	1400	1600	4	1200	1400	1400
5	1200	1400	1600	5	1200	1400	1600
6	1400	1600	1800	6	1200	1400	1600
7	1400	1600	1800	7	1200	1600	1800
8	1400	1600	2000	8	1400	1600	1800
9	1600	1800	2000	9	1400	1600	1800
10	1600	1800	2200	10	1400	1800	2000
11	1800	2000	2200	11	1600	1800	2000
12	1800	2200	2400	12	1600	2000	2200
13	2000	2200	2600	13	1600	2000	2200
14	2000	2400	2800	14	1800	2000	2400
15	2200	2600	3000	15	1800	2000	2400
16	2400	2800	3200	16	1800	2000	2400
17	2400	2800	3200	17	1800	2000	2400
18	2400	2800	3200	18	1800	2000	2400
19-20	2600	2800	3000	19-20	2000	2200	2400
21-25	2400	2800	3000	21-25	2000	2200	2400
26-30	2400	2600	3000	26-30	1800	2000	2400
31-35	2400	2600	3000	31-35	1800	2000	2200
36-40	2400	2600	2800	36-40	1800	2000	2200
41-45	2200	2600	2800	41-45	1800	2000	2200
46-50	2200	2400	2800	46-50	1800	2000	2200
51-55	2200	2400	2800	51-55	1600	1800	2200
56-60	2200	2400	2600	56-60	1600	1800	2200
61-65	2000	2400	2600	61-65	1600	1800	2000
66-70	2000	2200	2600	66-70	1600	1800	2000
71-75	2000	2200	2600	71-75	1600	1800	2000
76 and up	2000	2200	2400	76 and up	1600	1800	2000

¹Sedentary means a lifestyle that includes only the physical activity of independent living.

²Moderately Active means a lifestyle that includes physical activity equivalent to walking about 1.5 to 3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living.

³Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living.

Source: Center for Nutrition Policy and Promotion, USDA. USDA Food Patterns. Available at http://www.cnpp.usda.gov/sites/default/files/usda_food_patterns/EstimatedCalorieNeedsPerDayTable.pdf

Table D1.12. Percent of total energy intake from the 32 as-consumed food subcategories,* NHANES 2009-10.

Subcategory	% of total energy	
	consumption	Cumulative %
BURGERS, SANDWICHES, and TACOS	13.8	13.8
DESSERTS and SWEET SNACKS	8.5	22.3
SUGAR-SWEETENED and DIET BEVERAGES	6.5	28.8
RICE, PASTA, GRAIN-BASED MIXED DISHES	5.5	34.3
CHIPS, CRACKERS, and SAVORY SNACKS	4.6	38.9
PIZZA	4.3	43.2
MEAT, POULTRY, SEAFOOD MIXED DISHES	3.9	47.1
VEGETABLES (Incl. Beans and Peas, not Starchy)	3.8	50.9
ALCOHOLIC BEVERAGES	3.8	54.8
STARCHY VEGETABLES	3.8	58.6
YEAST BREADS AND TORTILLAS	3.8	62.4
HIGHER FAT MILK/YOGURT	3.5	65.8
BREAKFAST CEREALS AND BARS	3.5	69.3
POULTRY (Not incl. Deli and Mixed Dishes)	3.3	72.6
CANDY AND SUGARS	3.1	75.6
FRUIT (non-juice)	2.7	78.4
MEATS (Not incl. Deli and Mixed Dishes)	2.1	80.5
LOWFAT MILK/YOGURT	1.9	82.4
QUICK BREADS (Biscuits, Muffins, Pancakes, Waffles)	1.9	84.4
100% FRUIT JUICE	1.8	86.2
NUTS, SEEDS, AND SOY	1.7	87.9
EGGS	1.5	89.4
RICE AND PASTA	1.5	90.8
COFFEE AND TEA	1.4	92.3
SPREADS	1.3	93.6
SOUPS	1.3	95.0
DELI/CURED PRODUCTS (Meat and Poultry)	1.3	96.3
CHEESE	1.3	97.6
SEAFOOD (Not incl. Mixed Dishes)	1.1	98.7
CONDIMENTS AND GRAVIES	0.7	99.4
SALAD DRESSINGS	0.3	99.7
WATERS	0.0	99.7

*Collapsed from the 150 WWEIA Food Categories.

Note: does not total to 100% because baby foods and formulas are not included.

Source: Analysis of What We Eat in America (WWEIA) Food categories for NHANES 2009-10, population ages 2+. (see *Appendix E-2.9*)

Table D1.13. Percent of individuals consuming 1, 2, or 3 meals per day, and number of snacks consumed, by age/sex groups, NHANES 2009-2010

	3 meals total	3 meals + ≤1 snack	3 meals + 2-3 snacks	3 meals + ≥4 snacks	2 meals total	2 meals + ≤1 snack	2 meals + 2-3 snacks	2 meals + ≥4 snacks	1 meal total	1 meal + ≤1 snack	1 meal + 2-3 snacks	1 meal + ≥4 snacks
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	84	9	42	32	16	1	8	7	1	0	0	1
Ages 6-11	73	17	37	19	22	4	10	8	5	1	2	1
Ages 12-19	57	14	27	15	36	8	17	11	8	2	3	2
Ages 20-29	49	10	28	11	39	9	16	14	12	1	6	4
Ages 30-39	59	10	27	22	34	7	17	10	7	2	4	1
Ages 40-49	60	10	32	18	33	4	18	11	6	1	1	4
Ages 50-59	64	11	31	21	31	5	14	13	5	1	3	1
Ages 60-69	72	13	38	21	24	5	13	6	4	0	0	1
Ages 70+	64	18	34	12	32	7	18	7	3	1	1	2
20+	60	12	31	18	33	6	16	11	7	0	1	2
Females:												
Ages 2-5	84	9	38	36	15	1	7	7	1	0	0	0
Ages 6-11	68	15	40	13	30	4	14	12	3	0	1	2
Ages 12-19	49	11	27	10	41	13	19	9	10	1	5	4
Ages 20-29	55	13	23	18	38	8	18	12	7	1	4	3
Ages 30-39	63	9	30	24	34	7	19	7	3	1	1	1
Ages 40-49	64	14	31	20	29	5	12	12	7	1	2	4
Ages 50-59	69	14	28	26	29	4	11	14	3	1	1	1
Ages 60-69	72	8	36	28	26	3	14	9	2	0	1	1
Ages 70+	70	19	32	18	29	7	14	8	1	0	1	0
20+	65	13	30	22	31	6	15	11	4	1	2	2
M/F 2+	63	12	31	20	31	6	15	10	5	1	2	2

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.14. Percent of individuals skipping specific meals, by age/sex groups, NHANES 2009-2010

Age/sex	% skipping breakfast	% skipping lunch	% skipping dinner
Males:			
Ages 2-5	6	7	4
Ages 6-11	13	13	6
Ages 12-19	26	19	7
Ages 20-29	28	23	12
Ages 30-39	19	22	8
Ages 40-49	16	25	6
Ages 50-59	12	23	7
Ages 60-69	9	18	6
Ages 70+	5	28	7
Females:			
Ages 2-5	5	7	5
Ages 6-11	14	16	5
Ages 12-19	25	25	11
Ages 20-29	22	24	7
Ages 30-39	14	17	9
Ages 40-49	13	22	8
Ages 50-59	8	19	8
Ages 60-69	6	18	6
Ages 70+	4	21	6
Males and Females ages 2+	15	20	7

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.15. Meal and snack intake over time—percent reporting consumption of each meal, by age/sex group, NHANES 2005-2006 to 2009-2010

	Breakfast 2005-2006	Breakfast 2007-2008	Breakfast 2009-2010	Lunch 2005- 2006	Lunch 2007- 2008	Lunch 2009- 2010	Dinner 2005- 2006	Dinner 2007- 2008	Dinner 2009- 2010	Snacks 2005- 2006	Snacks 2007- 2008	Snacks 2009- 2010
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	96	94	94	92	91	93	96	96	96	99	98	97
Ages 6-11	91	87	87	88	90	87	97	94	94	98	95	96
Ages 12-19	71	74	74	78	81	81	92	88	93	93	95	92
Ages 20-29	69	72	72	73	82	77	88	91	88	98	94	96
Ages 30-39	82	81	81	85	77	78	90	89	92	95	95	96
Ages 40-49	83	84	84	79	79	75	94	94	94	99	97	97
Ages 50-59	88	88	88	79	80	77	92	91	93	95	98	97
Ages 60-69	91	91	91	74	74	82	95	91	94	94	95	94
Ages 70+	95	95	95	74	70	72	92	94	93	94	93	94
Ages 20+	83	84	84	78	78	77	92	92	92	96	95	96
Females:												
Ages 2-5	97	95	95	91	90	93	95	95	95	96	97	97
Ages 6-11	90	86	86	88	91	84	96	94	95	97	98	98
Ages 12-19	71	75	75	80	82	75	92	89	89	94	95	94
Ages 20-29	74	78	78	79	81	76	89	94	93	94	96	95
Ages 30-39	88	86	86	83	77	83	92	92	91	97	95	97
Ages 40-49	85	87	87	79	82	78	93	94	92	97	98	94
Ages 50-59	92	92	92	81	83	81	94	95	92	98	98	97
Ages 60-69	93	94	94	79	76	82	95	94	94	98	99	97
Ages 70+	96	96	96	79	78	79	93	93	94	93	94	94
Ages 20+	87	88	88	80	80	80	93	94	93	96	97	96
M/F Ages 2+	85	85	85	80	81	80	93	92	93	96	96	96

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2005-06, 2007-08, 2009-2010. For standard errors and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.16. Percent of energy from each meal and snack occasion over time, by age/sex group, NHANES 2005-2006 to 2009-2010

	Breakfast 2005- 2006	Breakfast 2007- 2008	Breakfast 2009- 2010	Lunch 2005- 2006	Lunch 2007- 2008	Lunch 2009- 2010	Dinner 2005- 2006	Dinner 2007- 2008	Dinner 2009- 2010	Snacks 2005- 2006	Snacks 2007- 2008	Snacks 2009- 2010
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	19	20	20	26	24	26	27	27	26	28	28	28
Ages 6-11	17	19	19	26	27	26	30	29	31	26	25	25
Ages 12-19	14	15	15	26	26	25	35	33	33	26	26	26
Ages 20-29	15	15	15	24	26	25	34	34	34	28	26	26
Ages 30-39	15	15	15	29	25	24	32	35	36	24	22	25
Ages 40-49	15	15	15	22	24	22	39	37	37	24	23	25
Ages 50-59	16	16	16	23	25	22	38	36	37	23	23	25
Ages 60-69	19	19	19	21	21	23	39	37	39	21	24	20
Ages 70+	22	22	22	21	19	20	38	38	39	18	20	19
Ages 20+	16	16	16	24	24	23	36	36	36	24	23	24
Females:												
Ages 2-5	20	19	19	24	23	24	26	26	27	30	29	29
Ages 6-11	19	19	19	26	27	24	31	30	33	24	26	24
Ages 12-19	14	16	16	25	27	25	35	30	33	26	28	26
Ages 20-29	15	16	16	26	25	23	33	36	35	26	25	25
Ages 30-39	17	18	18	26	23	25	34	35	33	23	25	24
Ages 40-49	16	17	17	24	24	23	37	36	35	23	25	24
Ages 50-59	18	18	18	25	24	23	37	37	36	21	23	23
Ages 60-69	19	18	18	22	22	22	39	36	37	20	23	23
Ages 70+	22	21	21	22	24	24	36	37	38	20	19	18
Ages 20+	17	18	18	24	24	23	35	36	35	23	24	23
M/F Ages 2+	17	17	17	25	25	24	35	35	35	24	24	24

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2005-06, 2007-08, 2009-2010. For standard errors and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.17. Percent of nutrient intake from snacks by age/sex group, NHANES 2009-2010

Age/sex	Food	Protein	Dietary	Vitamin			Iron	Potas-	Sodium*	Caffeine	Saturated
	energy		fiber	Folate	D	Calcium		sium			Fat*
	%	%	%	%	%	%	%	%	%	%	%
Males:											
Ages 2-5	28	19	25	18	24	27	18	26	18	36	26
Ages 6-11	25	15	22	17	21	23	18	22	16	41	24
Ages 12-19	26	14	23	17	17	23	18	21	16	60	23
Ages 20-29	26	14	22	21	22	28	20	24	15	48	18
Ages 30-39	25	12	19	17	17	24	17	21	13	45	17
Ages 40-49	25	14	21	19	20	25	17	22	14	48	21
Ages 50-59	25	14	21	18	17	24	17	21	13	43	23
Ages 60-69	20	11	16	13	14	22	13	18	11	37	17
Ages 70+	19	10	16	11	9	19	11	17	9	41	18
Females:											
Ages 2-5	29	21	28	17	29	32	19	29	18	44	30
Ages 6-11	24	14	25	17	14	19	19	20	16	39	23
Ages 12-19	26	16	26	20	19	26	21	24	19	47	24
Ages 20-29	25	14	21	16	18	25	17	22	15	39	23
Ages 30-39	24	13	22	14	16	24	15	22	14	42	20
Ages 40-49	24	14	19	18	17	28	18	22	14	40	24
Ages 50-59	23	13	20	17	15	23	17	20	13	42	22
Ages 60-69	23	14	19	14	16	26	15	21	13	42	24
Ages 70+	18	10	15	11	13	20	11	16	10	35	18

*Overconsumed nutrient

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: <http://seprl.ars.usda.gov/Services/docs.htm?docid=18349>

Table D1.18. Vegetable density (cup equivalents per 1000 calorie) for all vegetable subgroups, by point of purchase, NHANES 2003-2004 to 2009-2010

Point of purchase	2003-2004	2005-2006	2007-2008	2009-2010
DARK GREEN VEGETABLES (cup eq/1000 calorie)				
Store	0.04	0.05	0.05	0.06
Restaurant	0.07	0.08	0.09	0.09
Quick serve restaurant	0.02	0.02	0.03	0.03
School/day care	0.01	0.02	0.01	0.02
Other	0.05	0.08	0.07	0.07
RED AND ORANGE VEGETABLES (cup eq/1000 calorie)				
Store	n/a	0.16	0.16	0.16
Restaurant	n/a	0.23	0.23	0.20
Quick serve restaurant	n/a	0.22	0.17	0.17
School/day care	n/a	0.19	0.17	0.14
Other	n/a	0.23	0.22	0.22
STARCHY VEGETABLES (cup eq/1000 calorie)				
Store	0.20	0.18	0.20	0.19
Restaurant	0.23	0.24	0.26	0.24
Quick serve restaurant	0.24	0.22	0.23	0.23
School/day care	0.16	0.17	0.21	0.12
Other	0.22	0.23	0.25	0.25
OTHER VEGETABLES (cup eq/1000 calorie)				
Store	0.20	0.20	0.20	0.22
Restaurant	0.44	0.42	0.42	0.38
Quick serve restaurant	0.26	0.28	0.23	0.25
School/day care	0.16	0.16	0.13	0.12
Other	0.32	0.33	0.27	0.35

Source: Analysis of food group content, expressed as Food Pattern Equivalents, by point of purchase for What We Eat in America, NHANES 2003-2004, 2005-2006, 2007-2008, 2009-2010, population ages 2+ (see *Appendix E-2.15*).

Table D1.19. Body mass index (BMI)*, by sex, age, and race/ethnicity, adults ages 20 years and older, NHANES 2009-2012

	Normal weight % (SE)	Overweight % (SE)	Obese % (SE)
All adults ages 20 y and older	29.6 (0.9)	33.3 (0.8)	35.3 (0.8)
Men	26.5 (1.1)	38.1 (0.9)	34.5 (1.1)
Women	32.6 (1.0)	28.8 (1.1)	36.0 (1.0)
Age group (years)			
20-39	36.8 (1.8)	29.5 (1.2)	31.5 (1.3)
40-59	24.5 (1.0)	35.9 (1.2)	38.0 (1.0)
≥60	25.4 (1.1)	35.7 (1.1)	37.5 (1.3)
Race/ethnicity**			
Non-Hispanic White	31.2 (1.2)	33.5 (1.1)	33.4 (1.1)
Non-Hispanic Black	21.7 (0.9)	27.7 (1.1)	48.7 (1.4)
Hispanic	21.0 (1.0)	37.5 (1.2)	40.8 (1.2)
Race/ethnicity by sex			
Men			
Non-Hispanic White	26.7 (1.5)	38.4 (1.1)	34.3 (1.3)
Non-Hispanic Black	28.5 (1.1)	31.7 (1.5)	37.9 (1.5)
Hispanic	19.4 (1.4)	41.5 (1.5)	38.5 (1.5)
Women			
Non-Hispanic White	35.7 (1.4)	28.8 (1.7)	32.5 (1.5)
Non-Hispanic Black	16.2 (1.2)	24.5 (1.4)	57.5 (1.7)
Hispanic	22.7 (1.1)	33.5 (1.4)	43.0 (1.5)

* Normal weight = $18.5 \leq \text{BMI} < 25 \text{ kg/m}^2$; Overweight = $25 \leq \text{BMI} < 30 \text{ kg/m}^2$; Obese = $\text{BMI} \geq 30 \text{ kg/m}^2$

Estimates are age-adjusted to the year 2000 standard population using three age groups: 20–39 years, 40–59 years, and 60 years and over; estimates are weighted; all pregnant women excluded from analysis. SE = standard error.

**Participants with a race-Hispanic origin categorized as “other” are included in overall estimates but are not separately reported.

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Body Mass Index, Adults 20 y and over, NHANES 2009 -2012.

Table D1.20. Percent of overweight and obesity* by income in relation to poverty level, adults ages 20 years and older

Income as % of poverty level	% Overweight 1988-1994	% Obese 1988-1994	% Overweight 1999-2002	% Obese 1999-2002	% Overweight 2003-2006	% Obese 2003-2006	% Overweight 2007-2010	% Obese 2007-2010
Below 100%	31.5	28.1	30	34.7	30.7	35	32.5	37.2
100%-199%	31.9	26.1	33.2	34.1	30.6	35.9	33.2	37.3
200%-399%	33.3	22.7	36.5	32.1	33.3	35.7	31.8	36.8
400% or more	33.7	18.7	36.7	25.5	35.8	28.9	35.6	31.3

*Overweight = $25 \leq \text{BMI} < 30 \text{ kg/m}^2$; Obese = $\text{BMI} \geq 30 \text{ kg/m}^2$.

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. U.S. Department of Health and Human Services. Table 74. Healthy weight, overweight, and obesity among persons 20 years of age and over, by selected characteristics: United States, selected years 1960–1962 through 2007–2010. Health, United States, 2011. 2011. Available from: <http://www.cdc.gov/nchs/data/hus/2011/074.pdf>.

Table D1.21. Trends in prevalence of abdominal obesity[@] among adults, by age, sex, and race/ethnicity, NHANES*

		1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010	2011-2012
	Overall	46.4	43.4	52.1	51.6	52.7	52.8	54.2
	Men	37.1	39.1	42.5	44.8	43.4	43	43.5
	Women	55.4	57.1	61.3	58.2	61.6	62.3	64.7
Age group (years)**	Men							
	20 - 39	25.3	26.5	28.7	29.9	28.5	NA	NA
	40 - 59	41.8	43.9	49.8	52.7	49.4	NA	NA
	60 +	52.8	55	57.2	60.9	60.4	NA	NA
	Women							
	20 - 39	43.8	45.6	48.5	46.2	51.3	NA	NA
	40 - 59	60.3	59.9	66.7	63.5	65.5	NA	NA
	60 +	69.1	73.5	76.3	72.4	73.8	NA	NA
Race/ethnicity Overall	Non-Hispanic White	45.8	48.4	51.8	51.2	53.3	52.3	53.8
	Non-Hispanic Black	52.4	52.3	57.5	57.1	57.4	60.2	60.9
	Mexican American	48.1	49.9	55	51.4	55.5	58.4	57.4
	Men							
	Non-Hispanic White	38.6	42.4	45.1	46.2	46.6	45.3	44.5
	Non-Hispanic Black	31.5	30.6	35.1	40	38.9	39.5	41.5
	Mexican American	35.8	34.5	38	34.8	41.6	43.4	43.2
	Women							
	Non-Hispanic White	52.9	54.1	57.9	56.3	59.7	59.3	63.3
	Non-Hispanic Black	69.7	70.1	75.7	71	72.3	77.7	75.9
	Mexican American	60.2	66.9	73.8	70.5	71	75.5	71.6

[@]Abdominal obesity, as measured by waist circumference (WC) is defined as WC >102 cm in men and >88 cm in women

*All data from 1999 -2012, except age group –source: Ford ES, Maynard LM, Li C. Trends in mean waist circumference and abdominal obesity among US adults, 1999-2012. JAMA. 2014;312(11):1151-3. PMID: 25226482. <http://www.ncbi.nlm.nih.gov/pubmed/25226482>.

**Age group data only available from 1999 -2008 – source: Ford ES, Li C, Zhao G, Tsai J. Trends in obesity and abdominal obesity among adults in the United States from 1999-2008. Int J Obes (Lond). 2011;35(5):736-43. PMID: 20820173. <http://www.ncbi.nlm.nih.gov/pubmed/20820173>.

Age adjustment was performed using the direct method using the projected year 2000 US population aged 20 years or older.

NA = data not available.

Table D1.22. Body mass index (BMI) * among children and adolescents ages 2 to 19 years, NHANES 2009-2012

	Normal weight % (SE)	Overweight % (SE)	Obese % (SE)
Total	64.8 (0.8)	14.9 (0.6)	16.9 (0.6)
Sex			
Boys	63.7 (1.0)	14.9 (0.8)	17.6 (0.9)
Girls	65.9 (1.3)	14.9 (0.8)	16.1 (0.7)
Age group (years)			
2- 5	72.1 (1.5)	14.5 (1.3)	10.2 (0.9)
6-11	62.7 (1.1)	15.5 (0.8)	17.9 (0.9)
12-19	62.7 (1.2)	14.6 (0.8)	19.4 (1.1)
Race/ethnicity**			
Non-Hispanic White	68.2 (1.2)	14.1 (1.0)	14.0 (1.0)
Non-Hispanic Black	60.0 (1.4)	14.9 (0.7)	22.1 (1.2)
Hispanic	58.4 (0.9)	17.2 (0.7)	21.8 (0.6)
Boys			
Non-Hispanic White	66.8 (1.6)	14.5 (1.5)	14.4 (1.5)
Non-Hispanic Black	61.2 (1.8)	13.6 (1.1)	21.9 (1.4)
Hispanic	57.1 (1.3)	16.4 (0.9)	23.7 (1.0)
Girls			
Non-Hispanic White	69.8 (1.9)	13.7 (1.4)	13.6 (1.2)
Non-Hispanic Black	58.7 (2.0)	16.3 (1.3)	22.3 (2.0)
Hispanic	59.7 (1.2)	18.0 (0.9)	19.8 (1.1)

*5th - 84th percentile = normal weight; 85th - 94th percentile = overweight; ≥95th percentile = obese.

**Race-Hispanic origin classified as “other” not separately reported by included in overall estimates. Analyses based on age at the time of exam and exclude pregnant women.

SE = standard error.

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Body Mass Index Among Children and Adolescents Ages 2 – 19 years, NHANES 2009 -2012.

Table D1.23. Hypertension, lipid profile, and diabetes by body mass index (BMI) and waist circumference, adults ages 20 years and older, NHANES 2009-2012

	Total cholesterol^f % (SE) ≥ 240 mg/dl	HDL-C^g % (SE) < 40 mg/dl	LDL-C^h % (SE) ≥ 160 mg/dl	Triglyceridesⁱ % (SE) ≥ 200 mg/dl	Hypertension^{*@^δ} % (SE)	Diabetes^{**Ω} % (SE)
BMI^e						
Normal weight	12.1 (0.8)	8.5 (0.7)	8 (0.8)	4.8 (0.7)	20.0 (1.1)	5.5 (0.8)
Over weight	15.2 (1)	18.8 (1)	12 (1.2)	12 (0.8)	26.4 (0.8)	9.0 (0.9)
Obese	11.7 (0.6)	30.2 (1.3)	11.2 (0.8)	17.2 (1.6)	39.2 (0.8)	20.3 (1.2)
Waist Circumference (cm)^{&}						
Men ≤102, Women ≤ 88	12.1 (0.8)	13.7 (0.8)	8 (0.9)	7.6 (0.8)	21.2 (0.9)	6.0 (0.9)
Men >102, Women >88	13.4 (0.6)	24.9 (1.1)	12.1 (0.9)	14.8 (1.3)	34.6 (0.6)	16.2 (0.9)
BMI, waist circumference (cm) by sex						
Men						
Normal weight	9.7 (1.1)	14.2 (1)	8.3 (1.3)	7 (1.4)	20.1 (1.2)	8.8 (1.6)
Over weight	13.7 (1)	26.8 (1.7)	11 (1.5)	15.6 (1.4)	28.1 (1.3)	10.0 (1.3)
Obese	10.9 (0.9)	42.2 (1.7)	10.2 (1.1)	20.2 (1.9)	39.1 (1.2)	21.6 (1.6)
≤102 cm	12 (1)	20.4 (1.1)	9.3 (0.9)	10.8 (1.2)	23.3 (1)	8.3 (1.2)
>102 cm	11.3 (1)	40.3 (1.6)	11 (1.3)	20.4 (2)	37.2 (1)	19.6 (1.3)
Women						
Normal weight	13.6 (1.1)	4.3 (0.7)	7.7 (0.9)	3.2 (0.7)	19.9 (1.3)	3.2 (0.7)
Over weight	16.7 (1.4)	8.6 (0.9)	12.8 (1.5)	7 (1.1)	24.3 (1)	7.8 (0.8)
Obese	12.3 (0.8)	18.9 (1.4)	11.9 (1.2)	14.2 (1.9)	39.2 (1)	19.2 (1.1)
≤ 88 cm	12.1 (1.1)	3.6 (0.5)	5.9 (1.2)	2.4 (0.6)	17.8 (1.3)	2.6 (0.6)
> 88 cm	14.9 (0.7)	14.9 (1)	12.8 (0.9)	11.2 (1.2)	32.9 (0.7)	13.9 (0.9)

* Adults ages 18 years and older.

@ Hypertension is defined as having measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication. Estimates are based on the average of up to 3 measurements.

**Total diabetes is the sum of self-reported diabetes and undiagnosed diabetes. Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes only during pregnancy. Undiagnosed diabetes is defined as fasting plasma glucose (FPG) of at least 126 mg/dL or a hemoglobin A1c of at least 6.5% and no reported physician diagnosis. Respondents had fasted for at least 8 hours and less than 24 hours. The definition of undiagnosed diabetes was based on recommendations from the American Diabetes Association. For more information, see Standards of medical care in diabetes – 2010. Diabetes Care 2010; 33 (suppl 1): S11-S61.

Notes continue on next page

Table D1.23, continued

[€]BMI= 18.5-24.9 kg/m² = normal weight; BMI =25-29.9 kg/m²= overweight; BMI = \geq 30 kg/m²= obese.

[&]Abdominal obesity, as measured by waist circumference (WC) is defined as WC >102 cm in men and >88 cm in women

SE = standard error.

Source –

[€]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total cholesterol and high density lipoprotein cholesterol (HDL), adult 20 years and over, NHANES 2009 -2012.

[†]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults 20 years and over, NHANES 2009-2012.

[‡]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high blood pressure, adults 18 years and over, NHANES 2009-2012.

^ΩCenters for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total diabetes, in adults 20 years and over, NHANES 2009 -2012.

Table D1.24. Lipid profile by weight status, among children and adolescents, NHANES 2009-2012

	Total cholesterol*[§]‡ ≥ 200 mg/dL % (SE)	HDL-C**[‡] < 40 mg/dL % (SE)	LDL-C***[#]Ω ≥ 130 mg/dL % (SE)	Triglycerides***[§]Ω ≥ 130 mg/dL % (SE)
Body mass index (BMI)				
Normal weight	6.9 (0.7)	7.7 (0.6)	6.7 (1.4)	6.5 (1.2)
Overweight	7.1 (1.2)	16.4 (2.3)	8.0 (2.1)	11.4 (2.7)
Obese	11.3 (1.5)	30.5 (2.5)	6.8 (1.8)	24.1 (3.4)
Weight Status by Sex				
Boys				
Normal weight	5.1 (0.7)	8.8 (1.1)	6.1 (2.0) [@]	5.8 (1.4)
Overweight	5.3 (1.4)	16.9 (3.2)	7.5 (2.7) [@]	11.6 (2.9)
Obese	13.2 (2.4)	35.1 (2.6)	8.8 (3.0) [@]	38.6 (5.0)
Girls				
Normal weight	8.7 (1.1)	6.5 (0.9)	7.3 (1.8)	7.2 (2.5) [@]
Overweight	9.1 (2.1)	15.8 (2.6)	+	11.2 (4.4) [@]
Obese	9.1 (1.9)	25.5 (3.7)	4.6 (1.8) [@]	7.9 (2.4)

Analyses based on age at exam and exclude pregnant adolescents. Estimates are weighted.

[§]Cut-point criteria based on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents .

*Data for children and adolescents ages 6 to 19 years old.

**Data for children and adolescents ages 12 – 19 years old.

[#]LDL-C calculated using the Friedewald equation (which is valid when triglyceride <400 mg/dL).

Normal weight = 5th-84th percentile; overweight = 85th-94th percentile; obese = ≥95th percentile.

[@]Relative standard error (RSE)≥30 but < 40; + = RSE≥40.

SE = standard error.

Sources:

[‡]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol among children and adolescents ages 6 –19 years, NHANES 2009 -2012.

^ΩCenters for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Low density lipoprotein cholesterol (LDL-C) and triglycerides among adolescents ages 12-19 years, NHANES 2009-2012.

Table D1.25. Prevalence of high and borderline high blood pressure (BP) in children, 2009-2012

	High BP* % (SE)	Borderline high BP* % (SE)
Total	1.7 (0.2)	8.3 (0.7)
Boys	1.7 (0.4)	12.0 (1.3)
Girls	1.6 (0.2)	4.6 (0.8)
Age group (years)		
8 - 12	1.8 (0.4)	3.8 (0.7)
13 -17	1.5 (0.4)	12.4 (1.1)
Race/Ethnicity**		
Non-Hispanic White	1.4 (0.3)	7.2 (0.9)
Non-Hispanic Black	2.3 (0.5)	12.1 (1.3)
Hispanic	1.8 (0.6) [@]	8.5 (1.4)
Body Mass Index (BMI)		
Normal weight	1.4 (0.3)	5.4 (0.8)
Overweight	+	10.9 (1.6)
Obese	1.8 (0.6) [@]	16.2 (1.8)
Race/Ethnicity by Sex		
Boys		
Non-Hispanic White	**	10.8 (1.8)
Non-Hispanic Black	2.5 (0.7)	16.6 (2.0)
Hispanic	+	12.7 (2.3)
Girls		
Non-Hispanic White	1.8 (0.4)	3.8 (1.1)
Non-Hispanic Black	+	7.5 (1.6)
Hispanic	1.5 (0.6) [@]	4.3 (1.0)
BMI by Sex		
Boys		
Normal weight	1.8 (0.5)	8.6 (1.5)
Overweight	+	16.3 (2.8)
Obese	1.8 (0.6) [@]	20.1 (3.0)
Girls		
Normal weight	1.0 (0.3)	2.4 (0.8) [@]
Overweight	+	5.3 (1.2)
Obese	+	12.0 (2.7)

Analyses based on age at exam and exclude pregnant adolescents. Estimates are weighted. SE = standard error.

*Borderline high BP was defined as a systolic or diastolic BP ≥ 90 th percentile but < 95 th percentile or BP levels $\geq 120/80$ mm Hg and high BP was defined as a systolic or diastolic BP ≥ 95 th percentile. Definitions are based on the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescent. Estimates are based on the average of up to 3 measurements.

**Race-Hispanic origin classified as “other” not separately reported but included in overall estimates

Normal weight = 5th - 84th percentile; overweight = 85th - 94th percentile; obese = ≥ 95 th percentile

[@] Relative standard error (RSE) ≥ 30 but < 40 ; + = RSE ≥ 40 .

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high and borderline high blood pressure (BP), children and adolescents, Ages 8-17 years, NHANES 2009-2012.

Table D1.26. Prevalence of overweight and obesity among youth ages 3 to 19* years with type 2 diabetes by race and ethnicity , compared to youth without type 2 diabetes, SEARCH population, 2001-2004

Children ages 3 to 19 years with type 2 diabetes who are:			Children ages 3 to 19 without diabetes** who are:	
	N	% (95% CI)		% (95% CI)
Overweight [€]			Overweight [€]	
All	50	10.4 (6.7,15.9)	All	16.1 (15.0,17.3)
Non-Hispanic White	10	13.9 (6.3,28)	Non-Hispanic White	15.9 (14.3,17.6)
Non-Hispanic Black	15	8 (3.2,18.4)	Non-Hispanic Black	14.8 (13.4,16.3)
Hispanic	11	10.5 (4.2,23.8)	Hispanic	18.8 (16.6,21.1)
Asian Pacific Islander	7	14.9 (4.4,39.9)	Asian Pacific Islander	--
American Indian	7	3.3(0.4,20.7)	American Indian	--
Obese ^{&}			Obese ^{&}	
All	331	79.4 (72.8, 84.8)	All	16.9 (15.8,18.0)
Non-Hispanic White	64	68.8 (53.2,81)	Non-Hispanic White	15.8 (14.3,17.5)
Non-Hispanic Black	111	91.1 (81,96.1)	Non-Hispanic Black	20.2 (18.6,21.9)
Hispanic	63	75 (59.8,85.7)	Hispanic	18.3 (16.2,20.5)
Asian Pacific Islander	34	68.2 (43.4,85.7)	Asian Pacific Islander	--
American Indian	59	88 (67.9, 96.2)	American Indian	--

* 93% of children with type 2 diabetes are 12 -19 years old.

** US population estimates based on non-diabetic youth (NHANES 2001–2004).

-- NHANES does not contain large enough samples of Asian Pacific Islander I and American Indian to provide comparable estimates.

[€]Overweight defined as BMI from the 85th to <95th percentile for age and sex

[&]Obesity defined as BMI \geq 95th percentile.

Source: Liu LL, Lawrence JM, Davis C, Liese AD, Pettitt DJ, Pihoker C, et al. Prevalence of overweight and obesity in youth with diabetes in USA: the SEARCH for Diabetes in Youth study. *Pediatr Diabetes*. 2010;11(1):4-11. PMID: 19473302. <http://www.ncbi.nlm.nih.gov/pubmed/19473302>.

Table D1.27. Prevalence of hypertension and diabetes in US adults, NHANES 2009-2012

	Hypertension* [‡]	Total Diabetes** ^Ω
	% (SE)	% (SE)
Overall	29.1 (0.6)	12.3 (0.8)
Men	29.8 (0.8)	14.0 (1.0)
Women	28.3 (0.6)	10.8 (0.8)
Age group (years)		
18-39 ^{&}	7.1 (0.4)	3.2 (0.5)
40-59	31.7 (1.2)	13.5 (1.3)
≥60	66.3 (1.3)	26 (1.7)
Race/ethnicity[@]		
Non-Hispanic white	27.9 (0.7)	9.8 (0.8)
Non-Hispanic black	41.5 (0.9)	18.4 (1.3)
Hispanic	26.1 (0.9)	19.3 (1.5)
Race/ethnicity by sex		
Men		
Non-Hispanic White	28.9 (1.1)	11.7 (1.3)
Non-Hispanic Black	40.5 (1.1)	18.8 (1.8)
Hispanic	26.2 (1.4)	21 (1.7)
Women		
Non-Hispanic White	26.8 (0.8)	8.0 (0.9)
Non-Hispanic Black	42.1 (1.3)	18.1 (1.5)
Hispanic	25.8 (0.8)	17.6 (1.9)

Estimates are age-adjusted to the year 2000 standard population. Estimates are weighted. All pregnant women excluded from analysis.

SE = standard error.

*Hypertension is reported for adults ages 18 yrs and older and is defined as having measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication. Estimates are based on the average of up to 3 measurements.

**Total diabetes is reported for adults ages 20 years and older and is the sum of self-reported diabetes and undiagnosed diabetes. Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes only during pregnancy. Undiagnosed diabetes is defined as fasting plasma glucose (FPG) of at least 126 mg/dL or a hemoglobin A1c of at least 6.5% and no reported physician diagnosis. Respondents had fasted for at least 8 hours and less than 24 hours.

& Data for diabetes is reported for adults ages 20 to 39 years old.

@Participants with a race-Hispanic origin categorized as “other” are included in overall estimates but are not separately reported.

Sources:

[‡]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high blood pressure, adults 18 years and over, NHANES 2009-2012.

^ΩCenters for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total diabetes, in adults 20 years and over, NHANES 2009 -2012

Table D1.28. Prevalence of type 2 diabetes by sex, age, and race/ethnicity in children and adolescents*

	Cases with type 2 diabetes	Prevalence /1000 youth (95% CI)
Overall (< 20 years old)	819	0.46 (0.43 - 0.49)
Sex		
Boys	314	0.35 (0.31 - 0.39)
Girls	505	0.58 (0.53 - 0.63)
Age group (years)		
10 to 14	198	0.23 (0.2 - 0.26)
15 to 19	621	0.68 (0.63 - 0.74)
Race/ethnicity		
Non-Hispanic White	172	0.17 (0.15 - 0.2)
Non-Hispanic Black	209	1.06 (0.93 - 1.22)
Hispanic	317	0.79 (0.7 - 0.88)
Asian Pacific Islander	46	0.34 (0.26 - 0.46)
American Indian	75	1.2 (0.96 - 1.51)

*2009 SEARCH population

Source: Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. JAMA. 2014;311(17):1778-86. PMID: 24794371.

<http://www.ncbi.nlm.nih.gov/pubmed/24794371>.

Table D1.29. Cancer incidence and death rates per 100,000 persons by age category, sex and race and ethnicity, United States, 2007 -2011*

Rates per 100,000 persons	Incidence Breast	Death Breast	Incidence Prostate	Death Prostate	Incidence Colorectal	Death Colorectal	Incidence Lung & Bronchus	Death Lung & Bronchus
Age (years), men and women								
<20	0	0	0	0	0.1	0	0	0
20-34	1.8	0.9	0	0	1.2	0.6	0.3	0.1
35-44	9.3	5.2	0.6	0.1	4.1	2.5	1.3	1
45-54	22	14.5	9.7	1.6	14.2	9.1	8.6	7.7
55-64	25.5	21.7	32.7	8.5	21.2	17.6	21.4	19.7
65-74	21.3	20.6	36.3	20.1	23.9	21.9	31.7	30.6
75-84	14.4	21	16.8	36.8	23.2	27.3	27.9	29.8
>84	5.7	16.2	3.8	33	12.1	20.9	8.9	11.2
Men								
all race/ethnicities	–	–	147.8	22.3	50.6	19.1	72.2	61.6
Non-Hispanic White	–	–	139.9	20.6	49.6	18.5	72.4	61.4
Non-Hispanic Black	–	–	223.9	48.9	62.3	27.7	93	75.7
Hispanic	–	–	121.8	18.5	44.3	15.8	39.6	30.5
Asian/Pacific Islander	–	–	79.3	10	43.1	13.1	49.4	34.7
American Indian/Alaska Native	–	–	71.5	21.2	45.5	19.2	49.5	50
Women								
all race/ethnicities	124.6	22.2	–	–	38.2	13.5	51.1	38.5
Non-Hispanic White	128	21.7	–	–	37.3	13	53.8	39.8
Non-Hispanic Black	122.8	30.6	–	–	47.5	18.5	51.2	36.5
Hispanic	91.3	14.5	–	–	30.6	9.9	25.5	14
Asian/Pacific Islander	93.6	11.3	–	–	32	9.5	28.1	18.4
American Indian/Alaska Native	79.3	15.2	–	–	35.5	15.6	34.7	32.4

*SEER 18, 2007 -2011; rates (numbers) of new cases and deaths are per 100,000 persons and are age-adjusted to the 2000 U.S. standard population. Data are from selected statewide and metropolitan area cancer registries that meet the data quality criteria for all invasive cancer sites combined. Rates cover approximately 95% of the U.S. population.

Source: Data are from NCI factsheets, and can be found in the SEER Cancer Statistics Review (http://seer.cancer.gov/csr/1975_2011/)

Breast cancer - <http://seer.cancer.gov/statfacts/html/breast.html> , Prostate Cancer - <http://seer.cancer.gov/statfacts/html/prost.html> ,

Colon and Rectum Cancer - <http://seer.cancer.gov/statfacts/html/colorect.html> ,Lung and Bronchus Cancer - <http://seer.cancer.gov/statfacts/html/lungb.html>

Scientific Report of the 2015 Dietary Guidelines Advisory Committee

1 **Table D1.30. Estimates of the prevalence and number of US adults ages 50 years and older with osteoporosis**
 2 **(OP) and low bone mass (LBM) at either the femoral neck or lumbar spine (NHANES 2005-2010)**

	OP Prevalence * % (SE)	OP N (95% CI)**	BM Prevalence * % (SE)	LBM, N (95% CI)**
Both Sexes				
Overall (ages 50 above)	10.3 (0.37)	10.2 (9.4,10.9)	43.9 (0.72)	43.4 (42.0,44.8)
Men				
Overall	4.3 (0.40)	2.0 (1.6,2.3)	35.2 (0.93)	16.1 (15.3,17.0)
Age group (years)				
50-59	3.4 (0.68)	0.7 (0.4,1.0)	30.7 (1.78)	6.3 (5.6,7.0)
60-69	3.3 (0.73)	0.5 (0.3,0.7)	32.9 (1.82)	4.6 (4.1,5.1)
70-79	5.0 (0.78)	0.4 (0.3,0.5)	41.8 (2.51)	3.1 (2.7,3.5)
80+	10.9 (1.7)	0.4 (0.3,0.6)	53.1 (2.82)	2.2 (1.9,2.4)
Race/ethnicity[@]				
Non-Hispanic White	3.9 (0.39)	1.4 (1.1,1.6)	36.0 (1.13)	12.7 (11.9,13.4)
Non-Hispanic Black	1.3* (0.40)	0.1 (0.02,0.1)	21.3 (1.75)	0.9 (0.8,1.1)
Mexican American	5.9 (1.08)	0.1 (0.1,0.2)	38.3 (2.55)	0.9 (0.7,1.0)
Women				
Overall	15.4 (0.63)	8.2 (7.5,8.9)	51.4 (0.93)	27.3 (26.3,28.3)
Age group (years)				
50-59	6.8 (0.83)	1.5 (1.1,1.8)	49.3 (1.69)	10.6 (9.9,11.3)
60-69	12.3 (1.44)	1.9 (1.5,2.3)	53.4 (1.54)	8.2 (7.7,8.6)
70-79	25.7 (1.56)	2.4 (2.1,2.6)	51.8 (1.70)	4.7 (4.4,5.1)
80+	34.9 (2.44)	2.5 (2.2,2.8)	52.7 (3.07)	3.8 (3.3,4.2)
Race/ethnicity ^c				
Non-Hispanic White	15.8 (0.81)	6.3 (5.7,7.0)	52.6 (1.17)	21.1 (20.2,22.0)
Non-Hispanic Black	7.7 (1.10)	0.4 (0.3,0.5)	36.2 (2.03)	2.0 (1.8,2.2)
Mexican American	20.4 (1.70)	0.5 (0.4,0.6)	47.8 (2.33)	1.1 (1.0,1.2)

3 * Prevalence from NHANES 2005-2010 has been adjusted to the age, sex, and race/ethnic distribution of the US
 4 population at the time of the 2010 Census using the direct method.

5 **Count expressed in millions; 95% CI=95% confidence limits

6 [@] Other races not shown separately

7 OP = osteoporosis; LBM= low bone mass; NH= non-Hispanic. SE = standard error.

8 Osteoporosis and low bone mass were defined using the WHO criteria. Specifically, osteoporosis was defined as a
 9 T-score \leq -2.5 at either the femoral neck or the lumbar spine. Among those without osteoporosis, low bone mass
 10 was defined as those with T-scores between -1.0 and -2.5 at either skeletal site. The reference group for calculation
 11 of the scores at the femoral neck for both men and women, consisted of 20-29 non-Hispanic White females from
 12 NHANES III. As there is no internationally recommended reference group for the lumbar spine, the reference group
 13 for calculation of these scores at the lumbar spine consisted of 30-year old White females from the DXA
 14 manufacturer reference database. These reference groups were used to calculate T-scores for all race/ethnic groups
 15 and for both sexes.

16 Source: Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The Recent Prevalence of
 17 Osteoporosis and Low Bone Mass in the United States Based on Bone Mineral Density at the Femoral Neck or
 18 Lumbar Spine. J Bone Miner Res. 2014. PMID: 24771492. <http://www.ncbi.nlm.nih.gov/pubmed/24771492>.

19

20 **Table D1.31 Studies included in the analysis of Dietary Patterns Composition. Abbreviations listed below are**
 21 **used in Figures D1.56 to D1.60**

Abbreviation Used in Figures	Study/Cohort	Citation
<u>Interventions—feeding studies</u>		
DASH	DASH – Dietary Approaches to Stop Hypertension Trial	<u>Karanja</u> et al. 1999 ⁹⁷
OMNI CHO	OmniHeart trial – Carbohydrate-rich pattern	Swain et al. 2008 ¹⁰¹
OMNI PRO	OmniHeart trial – higher-protein pattern	
OMNI UNSAT	OmniHeart trial – higher unsaturated fat pattern	
<u>Interventions—other</u>		
EVOO	PREDIMED (Prevención con Dieta Mediterránea) trial. Extra Virgin Olive Oil group	Estruch et al. 2013 ⁹⁴
NUTS	PREDIMED Mixed nuts group	
<u>Cohorts--Med Diet score</u>		
SUN F (CVD endpoint)	Seguimiento Universidad de Navarra (SUN) project. Female subjects	Martínez-González et al. 2010 ⁹⁸
SUN M (CVD endpoint)	SUN project. Male subjects	
SUN (blood pressure endpoint)	Seguimiento Universidad de Navarra (SUN) project	Núñez-Córdoba et al. 2009 ⁹⁹
NHS (CVD endpoint)	Nurses' Health Study	Fung et al. 2009 ⁹⁵
EPIC PAN F	European Prospective Investigation into Cancer and Nutrition – Physical Activity, Nutrition, Alcohol, Cessation of Smoking, Eating Out of Home and Obesity project (EPIC-PANACEA) Female subjects	Romaguera et al. 2009 ¹⁰⁰
EPIC PAN M		
EPIC SPAIN	EPIC Spanish Cohort	Buckland et al. 2011 ⁹³
WAICAP	Washington Heights-Inwood Columbia Aging Project (WHICAP)	Scarmeas et al. 2006 ¹¹²
NHS (cognitive decline endpoint)	Nurses' Health Study	Samieri et al. 2013 ¹¹¹
<u>Cohorts/Other scores</u>		
WHI	Women's Health Initiative	George et al. 2014 ⁹⁶
HPFS	Health Professionals Follow-up Study	McCullough et al. 2000 ¹¹⁴
EPIC POT F	EPIC Potsdam (Germany) study Female Subjects	von Ruesten et al. 2010 ¹⁰⁹
EPIC POT M		

22

23 **Table D1.31, continued**
 24

Abbreviation Used in Figures	Study/Cohort	Citation
<u>Factor/Cluster Analyses</u>		
NHS (type 2 diabetes endpoint)	Nurses' Health Study	Fung et al. 2004 ¹⁰³
NHS (CHD endpoint)	Nurses' Health Study	Fung et al. 2001 ¹⁰⁴
HPFS	Health Professionals Follow-up Study	Hu et al. 2000 ¹⁰⁵
FOS	Framingham Offspring Study	McKeown et al. 2002 ¹⁰⁷
WHITEHALL	Whitehall II study	Brunner et al. 2008 ¹⁰²
SHANGHAI	Shanghai Women's Health Study	Villegas et al. 2010 ¹⁰⁸
SINGAPORE	Singapore Chinese Health Study	Butler 2010 ¹¹⁰

25
 26

27 **Table D1.32. Composition of three USDA Food Patterns (Healthy U.S.-Style, Healthy Vegetarian, and**
 28 **Healthy Mediterranean-style) at the 2000 calorie level. Daily or weekly amounts from selected food groups,**
 29 **subgroups, and components.**

Food group	Healthy US-style Pattern	Healthy Vegetarian Pattern	Healthy Med-style Pattern
Fruit	2 c per day	2 c per day	2 ½ c per day
Vegetables	2 ½ c per day	2 ½ c per day	2 ½ c per day
-Legumes	1 ½ c per wk	3 c per wk	1 ½ c per wk
Whole Grains	3 oz eq per day	3 oz eq per day	3 oz eq per day
Dairy	3 c per day	3 c per day	2 c per day
Protein Foods	5 ½ oz eq per day	3 ½ oz eq per day	6 ½ oz eq per day
--Meat	12 ½ oz eq/wk	--	12 ½ oz eq/wk
--Poultry	10 ½ oz eq/wk	--	10 ½ oz eq/wk
--Seafood	8 oz eq/wk	--	15 oz eq/wk
--Eggs	3 oz eq/wk	3 oz eq/wk	3 oz eq/wk
--Nuts/seeds	4 oz eq/wk	7 oz eq/wk	4 oz eq/wk
--Processed soy	½ oz eq/wk	8 oz eq/wk	½ oz eq/wk
Oils	27 g per day	27 g per day	27 g per day

30 Source: Food Pattern Modeling report: *Appendix E-3.7 Developing Vegetarian and Mediterranean-style Food*
 31 *Patterns*

32

33 **Table D1.33. Nutrients in the three USDA Food Patterns (Healthy US Style, Healthy Vegetarian, and Healthy**
 34 **Mediterranean-style) at the 2000 calorie level as a percent of the goal or limit for a 19 to 30 year old woman.**

Nutrient	Healthy US-style Pattern % goal/limit	Healthy Vegetarian Pattern % goal/limit	Healthy Med-style Pattern % goal/limit
Protein -%RDA	198	155	194
Protein -%calorie	18	14	18
Fat-%calorie	33	34	32
Saturated fat* - %calorie	8	8	8
CHO-%RDA	197	211	199
CHO-%calorie	51	55	52
Fiber -% goal	109	126	112
Calcium-%RDA	127	133	100
Iron-%RDA	93	96	95
Vitamin D-%RDA	46	37	42
Potassium-%AI	71	70	71
Sodium*-%UL	78	61	73

35 *overconsumed nutrient

36 Source: Food Pattern Modeling report: Developing Vegetarian and Mediterranean-style Food Patterns (see
 37 *Appendix E-3.7*)

38
 39
 40
 41

Part D Chapter 1. Figures

Figure Number	Figure Title, by chapter section
Nutrients of Concern	
Figure D1.1	Percent of population with usual intakes below EAR
Figure D1.2	Percent of population with usual intakes above AI
Figure D1.3	Sodium: Percent of age/sex groups with usual intakes above UL
Figure D1.4	Saturated fat: Percent of age/sex groups with usual intake above 10% of calories
Figure D1.5	Supplement users: Percent with usual intakes from foods, beverages, and supplements greater than the UL
Figure D1.6	Caffeine: mean and percentiles of usual intake by age/sex groups-adults
Figure D1.7	Caffeine: mean and percentiles of usual intake by age/sex groups-children and adolescents
Figure D1.8	USDA Food Patterns: Range of nutrients in patterns as a percent of the target levels for all age/gender groups
Food Groups	
Figure D1.9	Total Fruit: Estimated percent of persons below, at, or above recommendation
Figure D1.10	Whole fruit vs. fruit juice consumption by age/sex groups
Figure D1.11	Total Vegetables: Estimated percent of persons below, at, or above recommendation
Figure D1.12	Dark Green vegetables: Estimated percent of persons below, at, or above recommendation
Figure D1.13	Red and Orange vegetables: Estimated percent of persons below, at, or above recommendation
Figure D1.14	Beans and Peas: Estimated percent of persons below, at, or above recommendation
Figure D1.15	Starchy vegetables: Estimated percent of persons below, at, or above recommendation
Figure D1.16	Other vegetables: Estimated percent of persons below, at, or above recommendation
Figure D1.17	Whole grains: Estimated percent of persons below, at, or above recommendation
Figure D1.18	Refined grains: Estimated percent of persons below, at, or above limits
Figure D1.19	Dairy: Estimated percent of persons below, at, or above recommendation
Figure D1.20	Total Protein foods: Estimated percent of persons below, at, or above recommendation

Figure D1.21	Meat, poultry, eggs: Estimated percent of persons below, at, or above recommendation
Figure D1.22	Seafood: Estimated percent of persons below, at, or above recommendation
Figure D1.23	Nuts, seeds, soy: Estimated percent of persons below, at, or above recommendation
Figure D1.24	Empty calories: Estimated percent of persons below, at, or above limits
Figure D1.25	Fruit: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.26	Vegetables: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.27	Whole grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.28	Refined grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.29	Dairy: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.30	Protein Foods: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group
Figure D1.31	Added sugars intakes in 2001-04 and 2007-10 by age/sex groups in comparison to added sugars limits in the USDA Food Patterns.
	Food categories
Figure D1.32	Percent of Total intake from mixed dishes
Figure D1.33	Percent of Energy Intake from Major food categories
Figure D1.34	Food sources Saturated Fat
Figure D1.35	Food Sources of Sodium
Figure D1.36	Food Sources of Added Sugars
Figure D1.37	Caffeine sources by age group
Figure D1.38	Percent of beverage energy from various beverages, all persons 2+
	Eating Behaviors
Figure D1.39	Number of meals reported per day by age/sex group
Figure D1.40	Percent of total daily intake of nutrients of concern from each eating occasion, for the population 2+
Figure D1.41	Percent of calories by where food was obtained and consumed
Figure D1.42	Fruit group density: cups per 1000 calories by where obtained and eating location, over time (2003-2004 to 2009-2010)
Figure D1.43	Vegetable density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10)
Figure D1.44	Vegetable subgroup density: cups per 1000 calories by where obtained, over time (2003-2004 to 2009-2010)

Figure D1.45	Dairy group density: cups per 1000 calories by where obtained, over time (2003-2004 to 2009-2010)
Figure D1.46	Grain group density (whole and refined) : ounce eqs per 1000 calories by where obtained over time (2003-2004 to 2009-2010)
Figure D1.47	Protein Foods Group density: ounce eqs per 1000 calories by where obtained, over time (2001-2004 vs. 2007-2010)
Figure D1.48	Sodium density: milligrams per 1000 calories by where obtained and eating location, over time (2003-2004 to 2009-2010)
Figure D1.49	Saturated fat density: percent of energy by where obtained, over time (2003-2004 to 2009-2010)
Figure D1.50	Empty calorie density: calories per 1000 calories by where obtained, over time (2003-2004 to 2009-2010)
Figure D1.51	Added sugars density: Added sugars per 1000 calories by where obtained, over time (2003-2004 to 2009-2010)
Figure D1.52	Solid fats density: Solid fats per 1000 calories by where obtained, over time (2003-2004 to 2009-2010)

Health Conditions

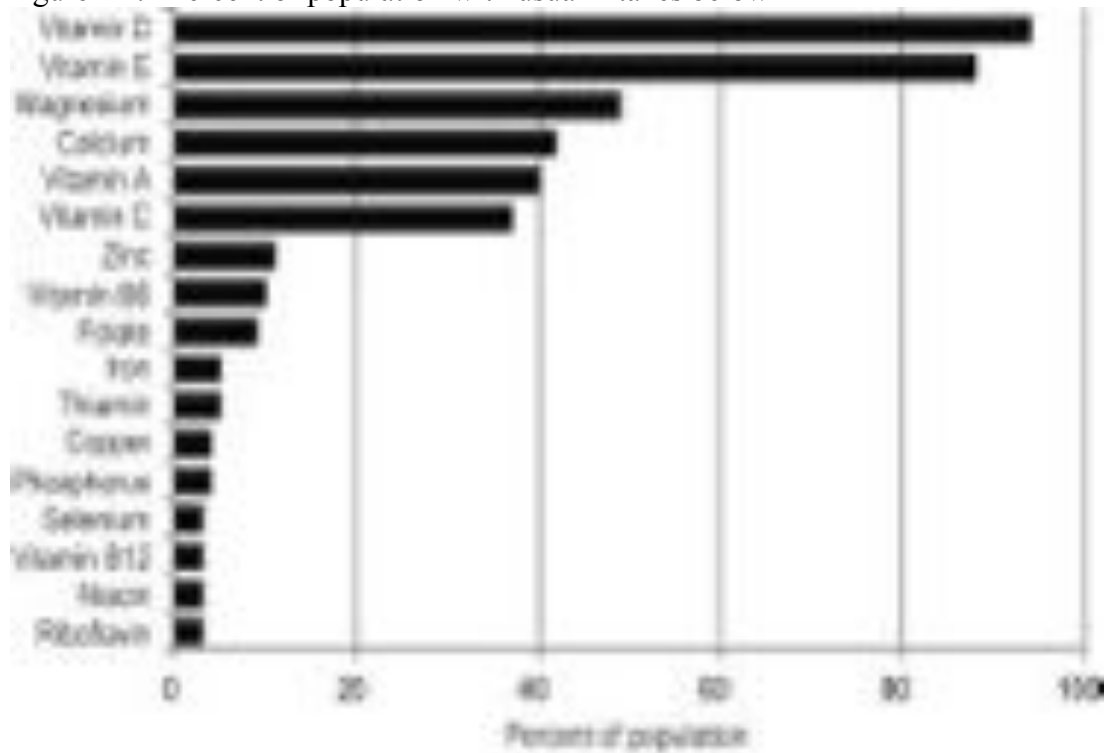
Figure D1.53	Trends in overweight and obesity, Males and Females ages 20+.
Figure D1.54	Trends in overweight and obesity, Boys and Girls ages 2-19.
Figure D1.55	Prevalence and number of CVD risk factors by weight category, among adults 18 years and older, NHANES 2007-10.

Dietary Patterns Composition

Figure D1.56	Vegetable intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual vegetable intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.
Figure D1.57	Fruit intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual fruit intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.
Figure D1.58	Dairy intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual dairy intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.
Figure D1.59	Red and processed meat intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual red and processed meat intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.
Figure D1.60	Seafood intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual seafood intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.
Figure D1.61	Average HEI-2010 scores for Americans by age group, 2009-10

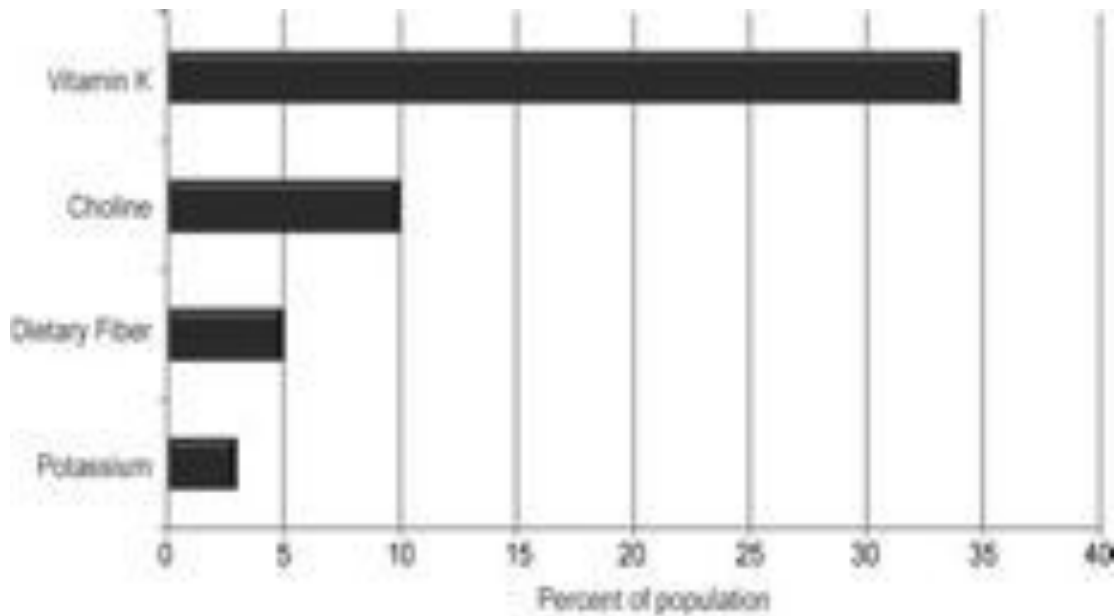
Figure D1.62 Intake from Protein Foods subgroups by self-identified vegetarians in comparison to non-vegetarian and amounts in USDA Food Pattern at 2000 calories.

Figure D1.1 Percent of population with usual intakes below EAR



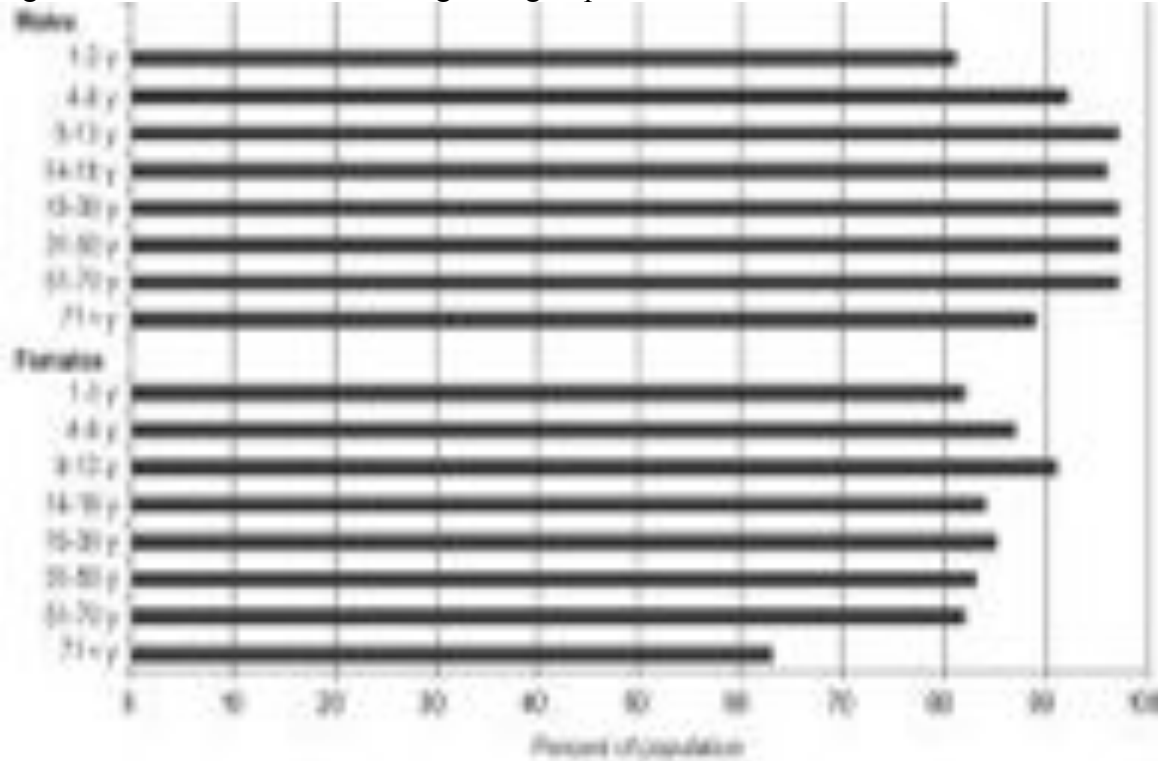
Source: What We Eat in America, NHANES 2007-2010

Figure D1.2 Percent of population with usual intakes above AI



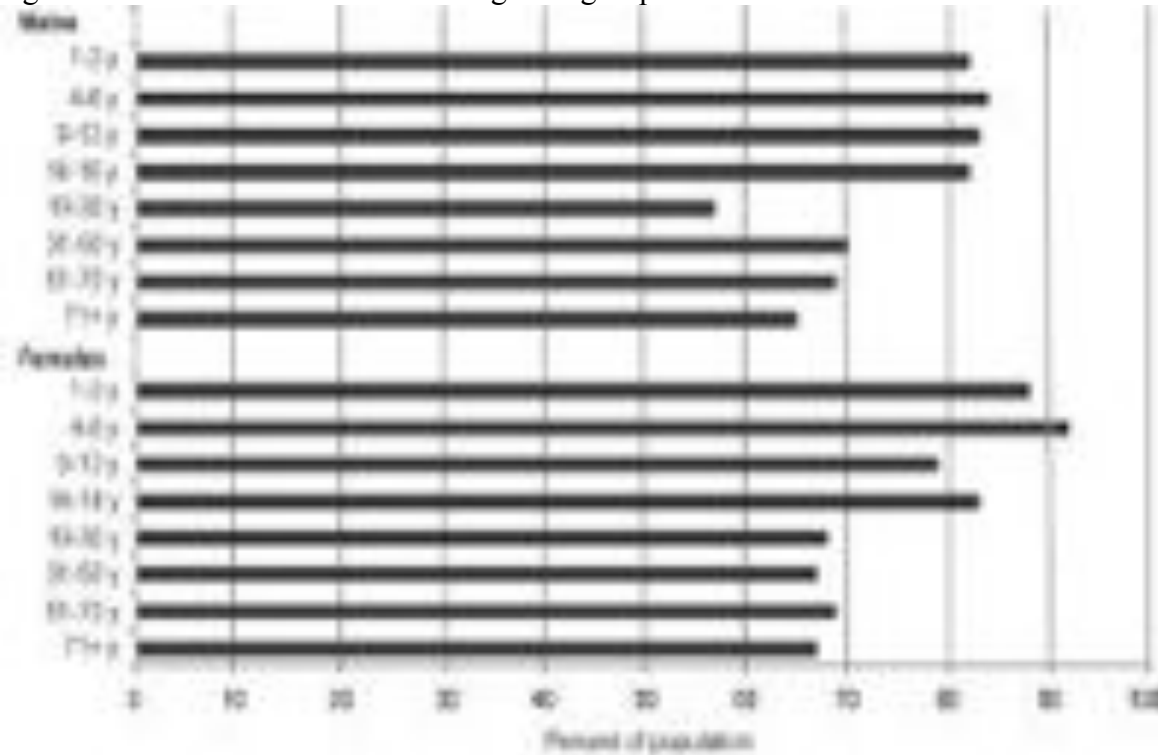
Source: What We Eat in America, NHANES 2007-2010

Figure D1.3 Sodium: Percent of age/sex groups with usual intakes above UL



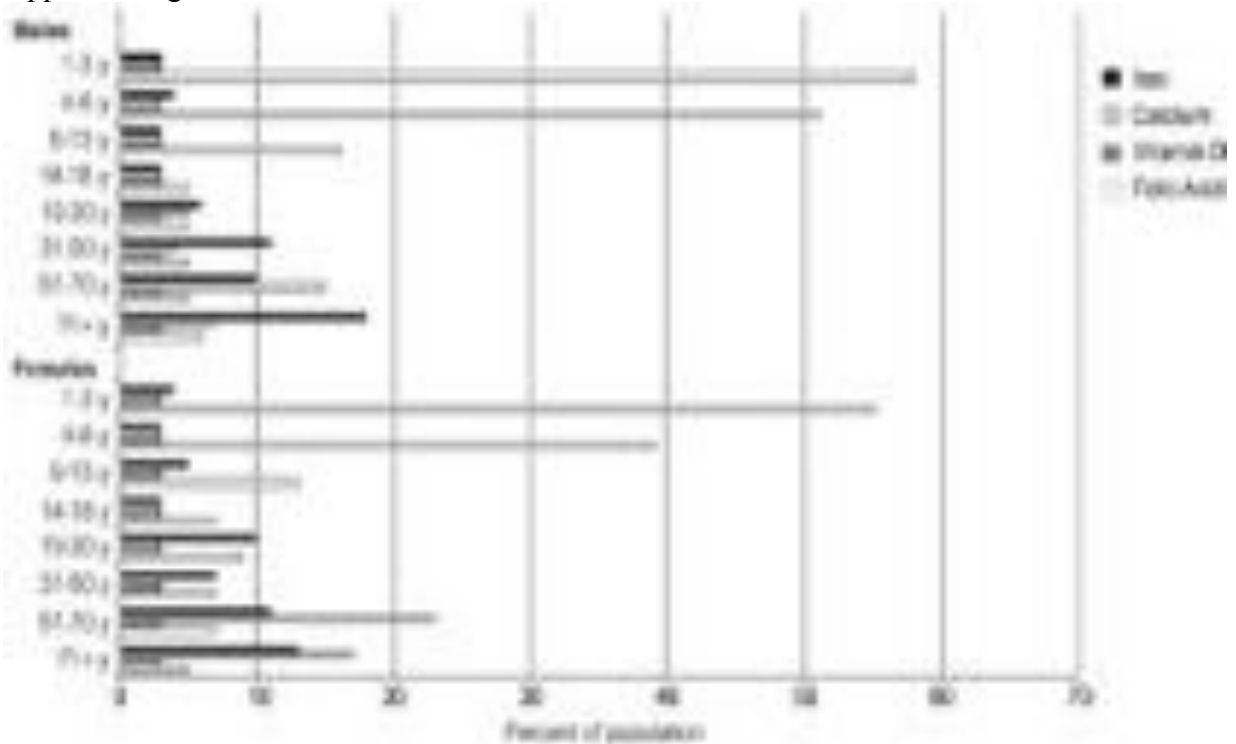
Source: What We Eat in America, NHANES 2007-2010

Figure D1.4 Saturated fat: Percent of age/sex groups with usual intake above 10% of calories



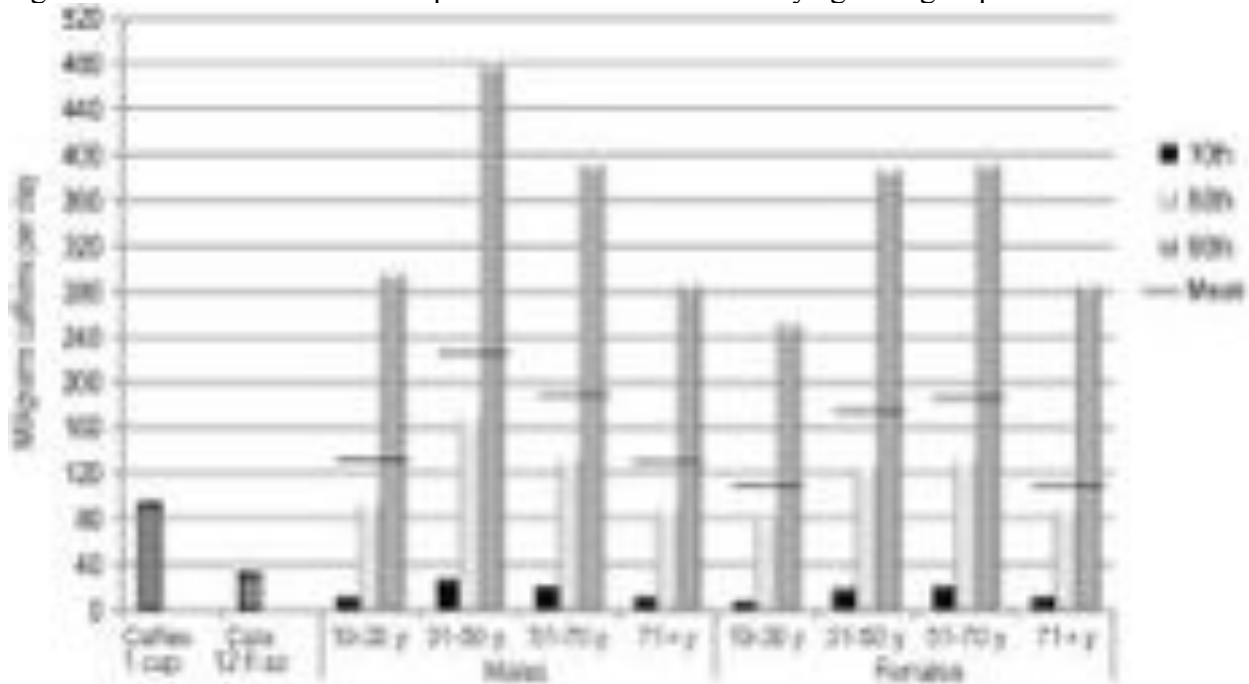
Source: What We Eat in America, NHANES 2007-2010

Figure D1.5 Supplement users: Percent with usual intakes from foods, beverages, and supplements greater than the UL



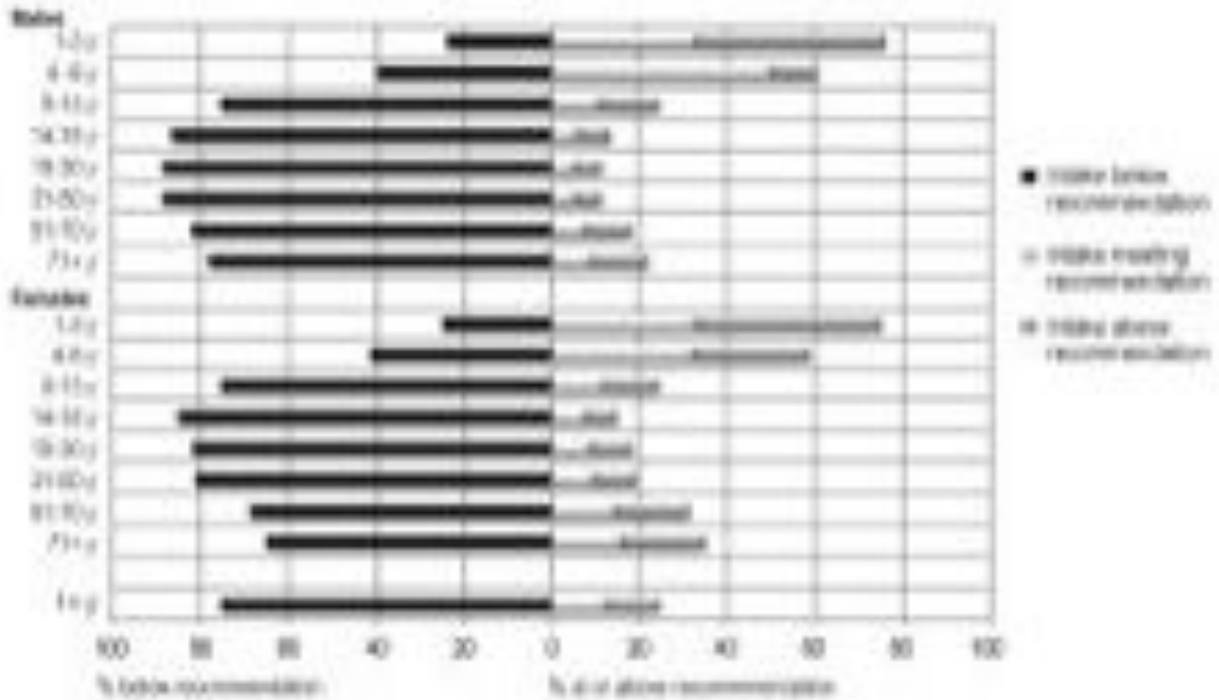
Source: What We Eat in America, NHANES 2007-2010

Figure D1.6 Caffeine: mean and percentiles of usual intake by age/sex groups-adults



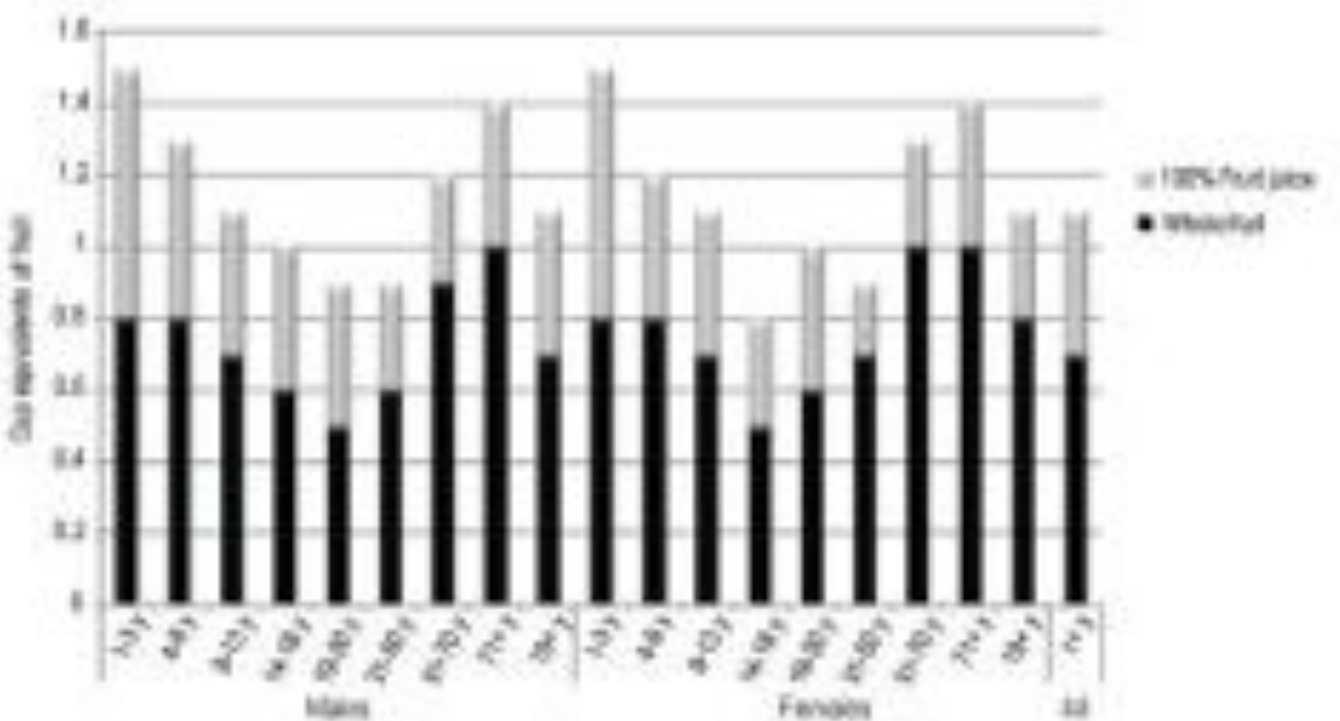
Source: What We Eat in America, NHANES 2007-2010

Figure D1.9 Total Fruit: Estimated percent of persons below, at, or above recommendation



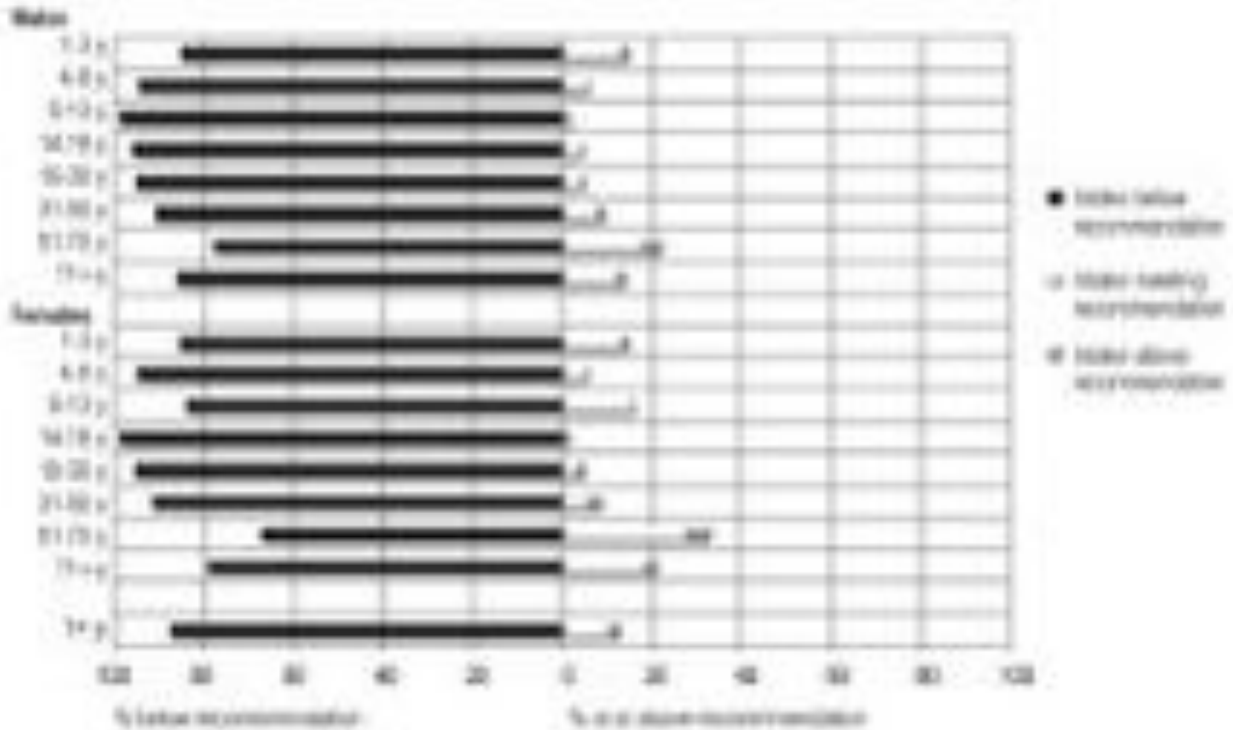
Source: What We Eat in America, NHANES 2007-2010

Figure D1.10 Whole fruit vs. fruit juice consumption by age/sex groups



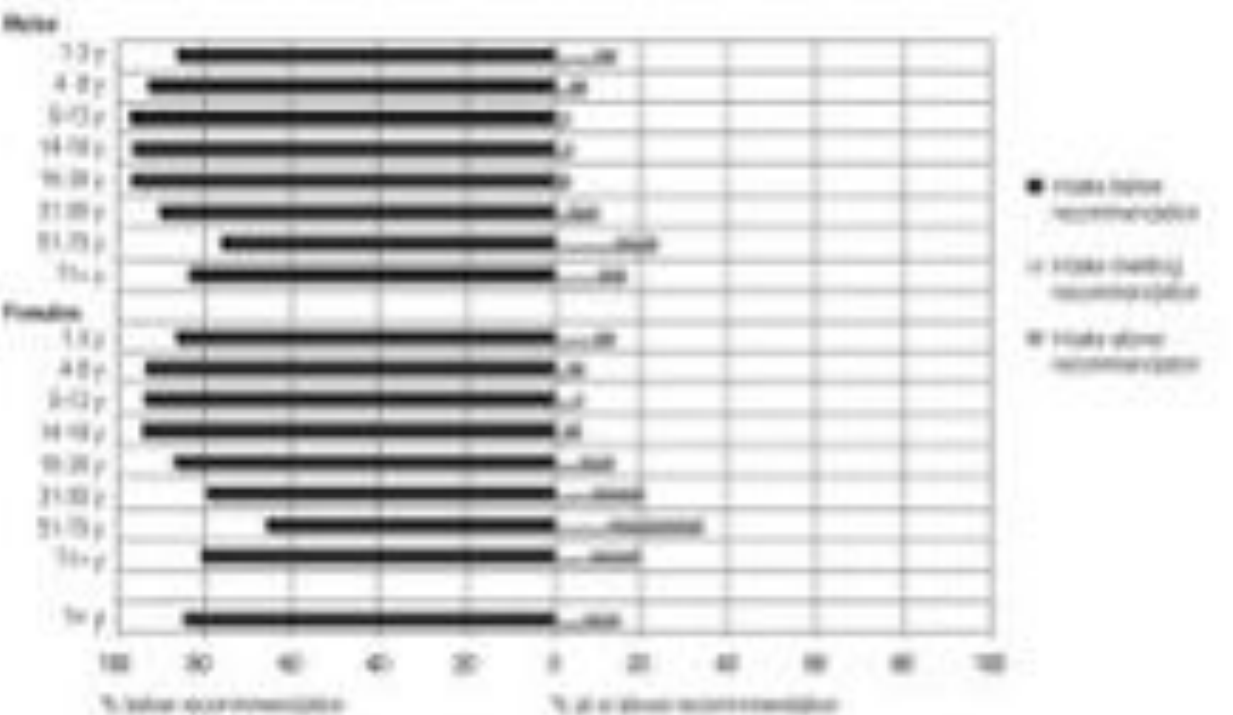
Source: What We Eat in America, NHANES 2007-2010

Figure D1.11 Total Vegetables: Estimated percent of persons below, at, or above recommendation



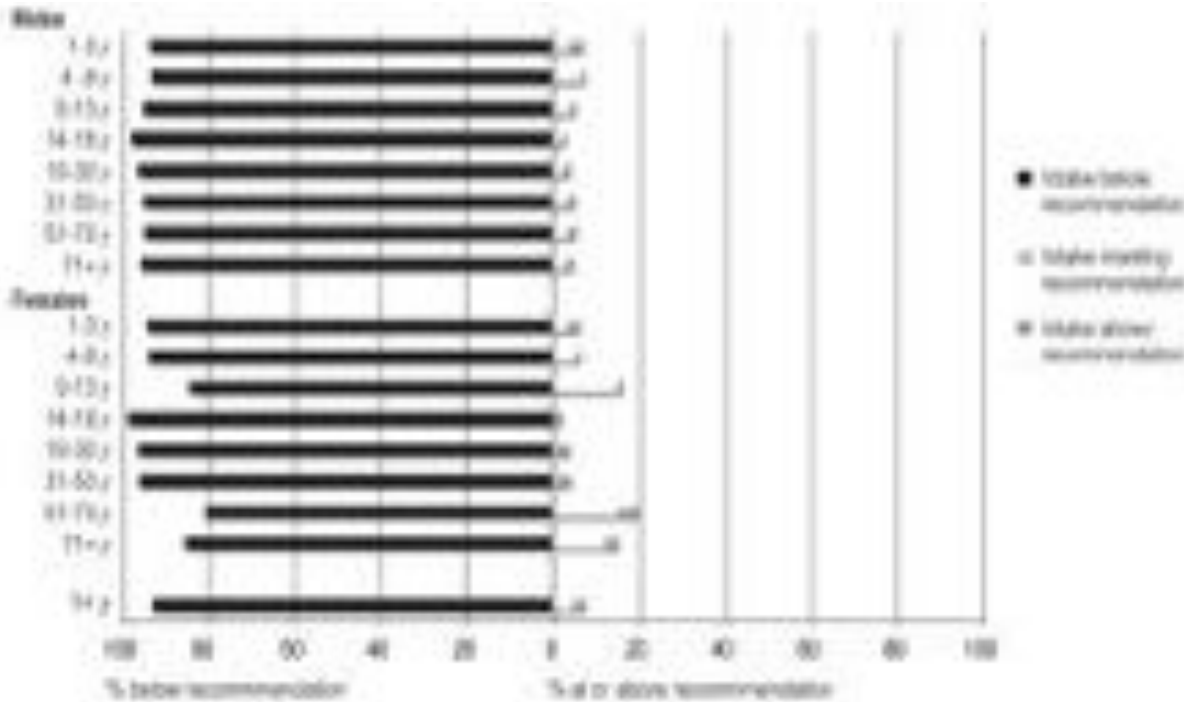
Source: What We Eat in America, NHANES 2007-2010

Figure D1.12 Dark Green vegetables: Estimated percent of persons below, at, or above recommendation



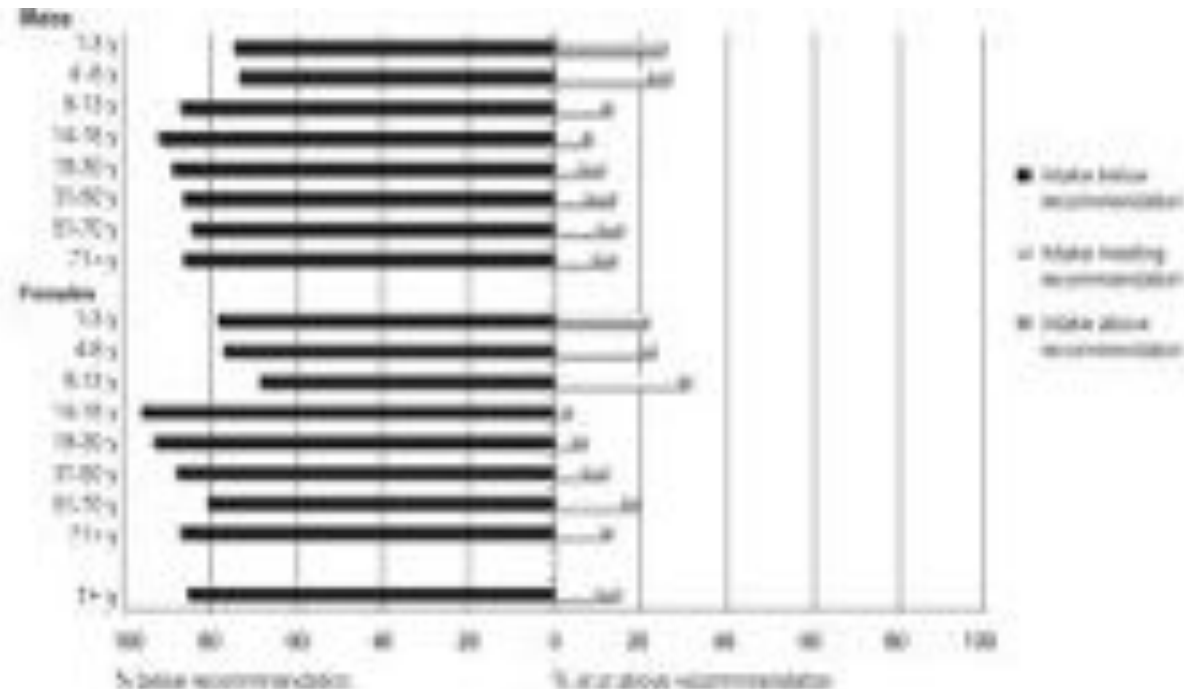
Source: What We Eat in America, NHANES 2007-2010

Figure D1.13 Red and Orange vegetables: Estimated percent of persons below, at, or above recommendation



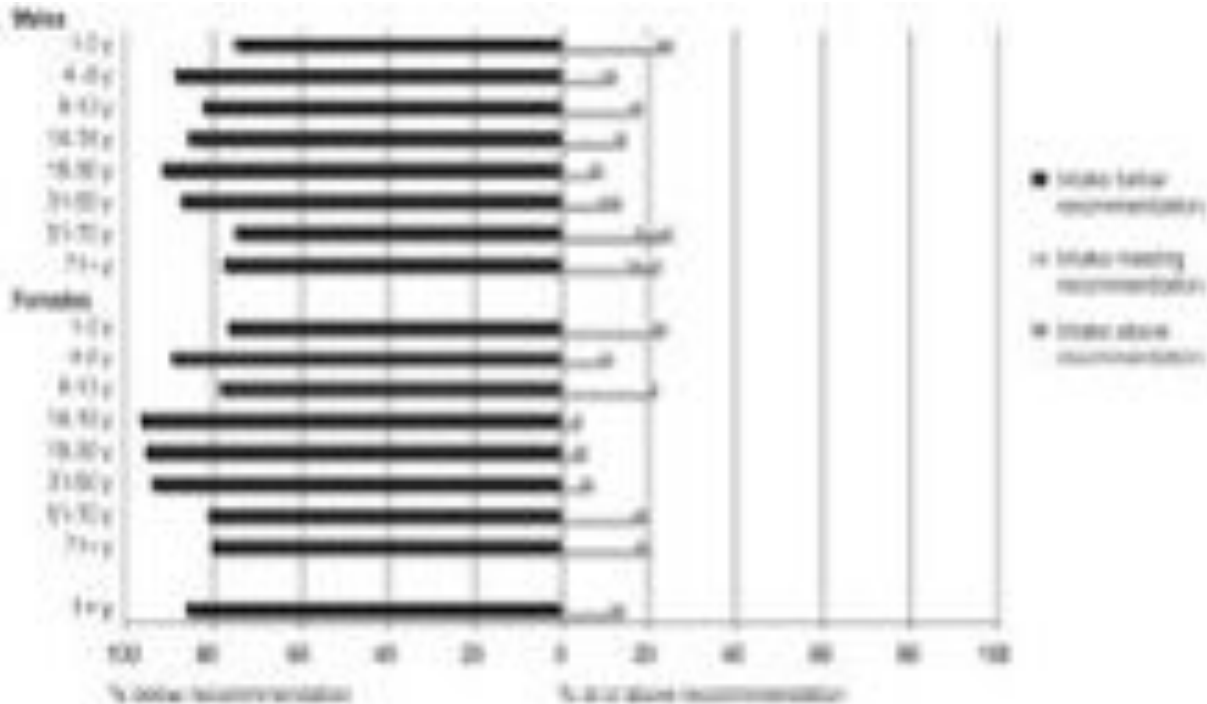
Source: What We Eat in America, NHANES 2007-2010

Figure D1.14 Beans and Peas: Estimated percent of persons below, at, or above recommendation



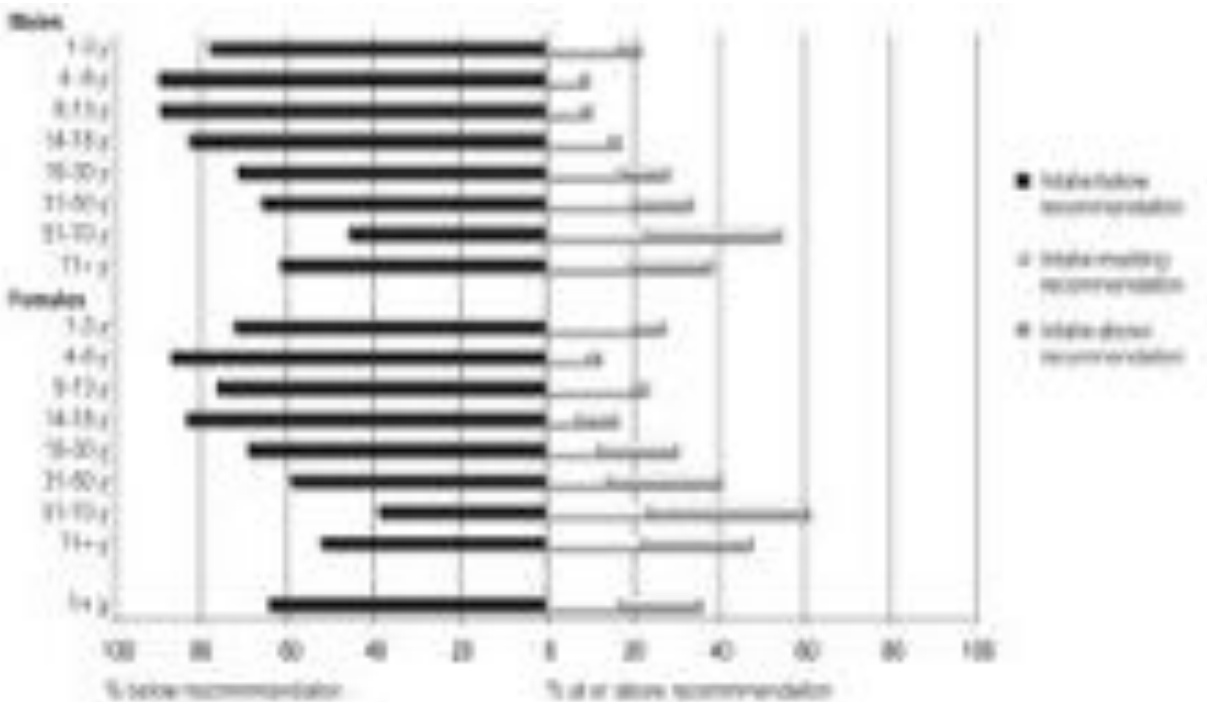
Source: What We Eat in America, NHANES 2007-2010

Figure D1.15 Starchy vegetables: Estimated percent of persons below, at, or above recommendation



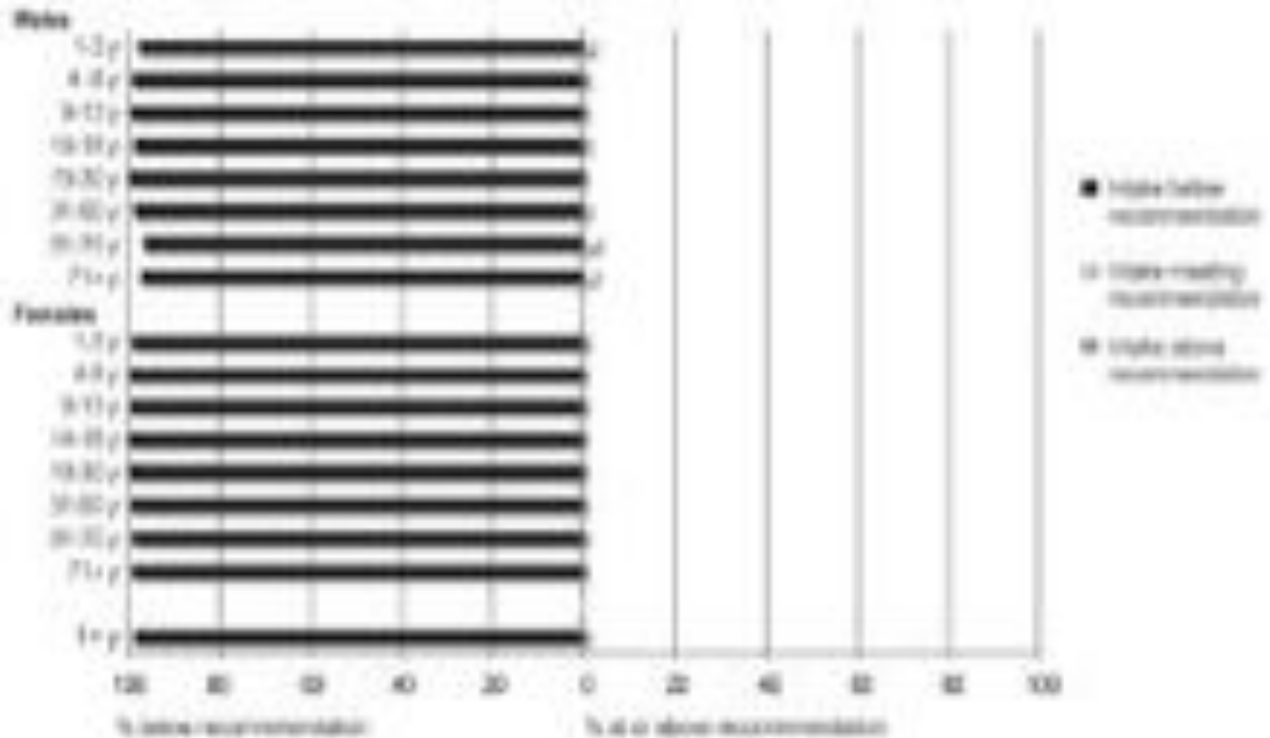
Source: What We Eat in America, NHANES 2007-2010

Figure D1.16 Other vegetables: Estimated percent of persons below, at, or above recommendation



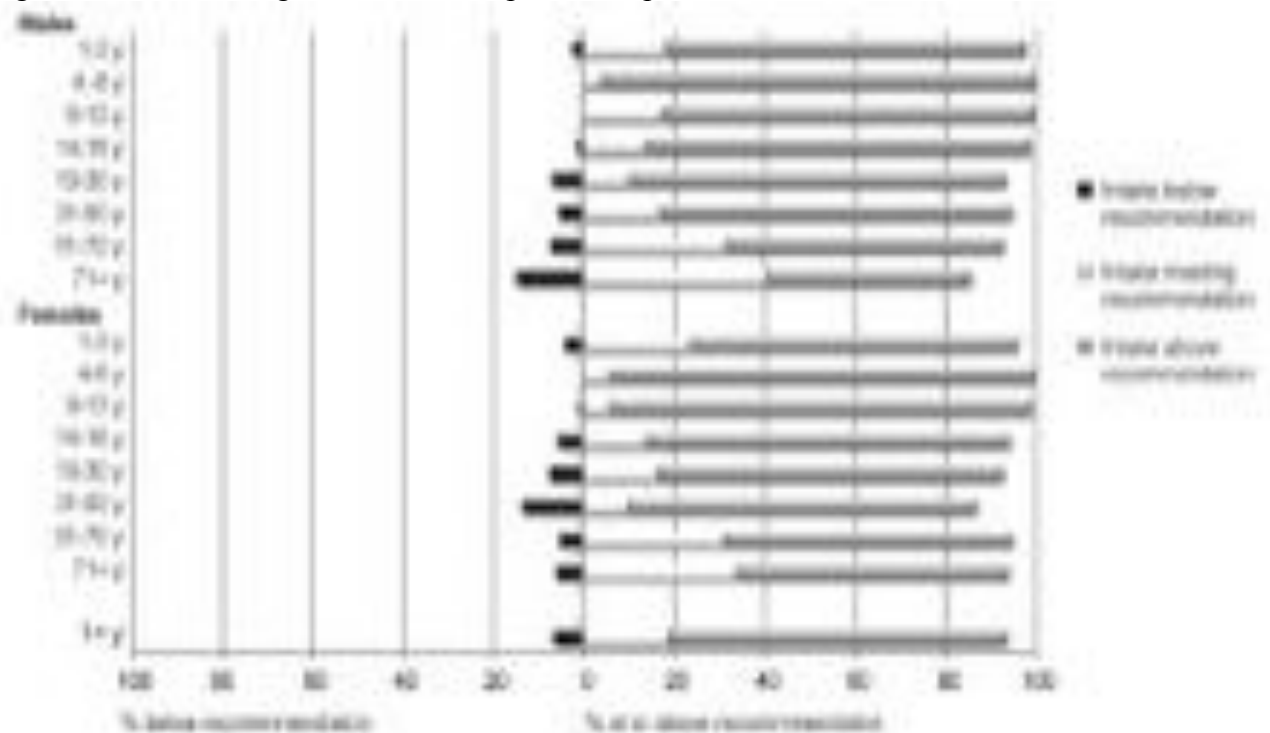
Source: What We Eat in America, NHANES 2007-2010

Figure D1.17 Whole grains: Estimated percent of persons below, at, or above recommendation



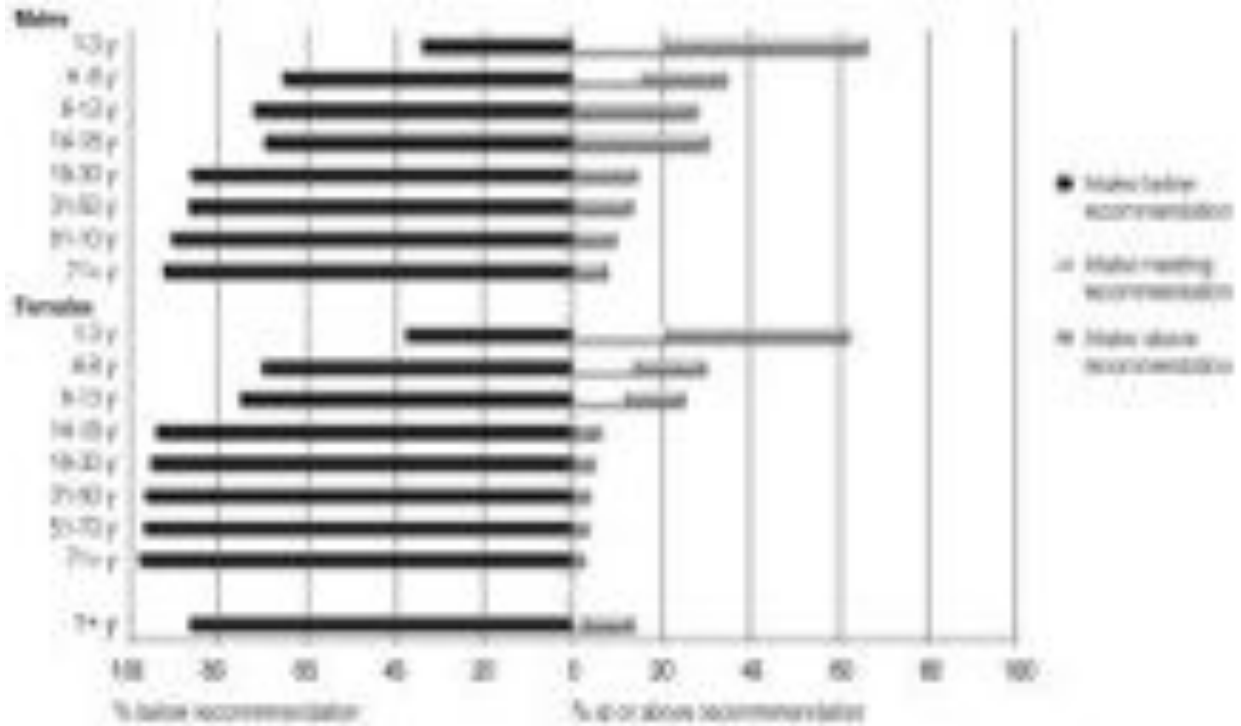
Source: What We Eat in America, NHANES 2007-2010

Figure D1.18 Refined grains: Estimated percent of persons below, at, or above limits



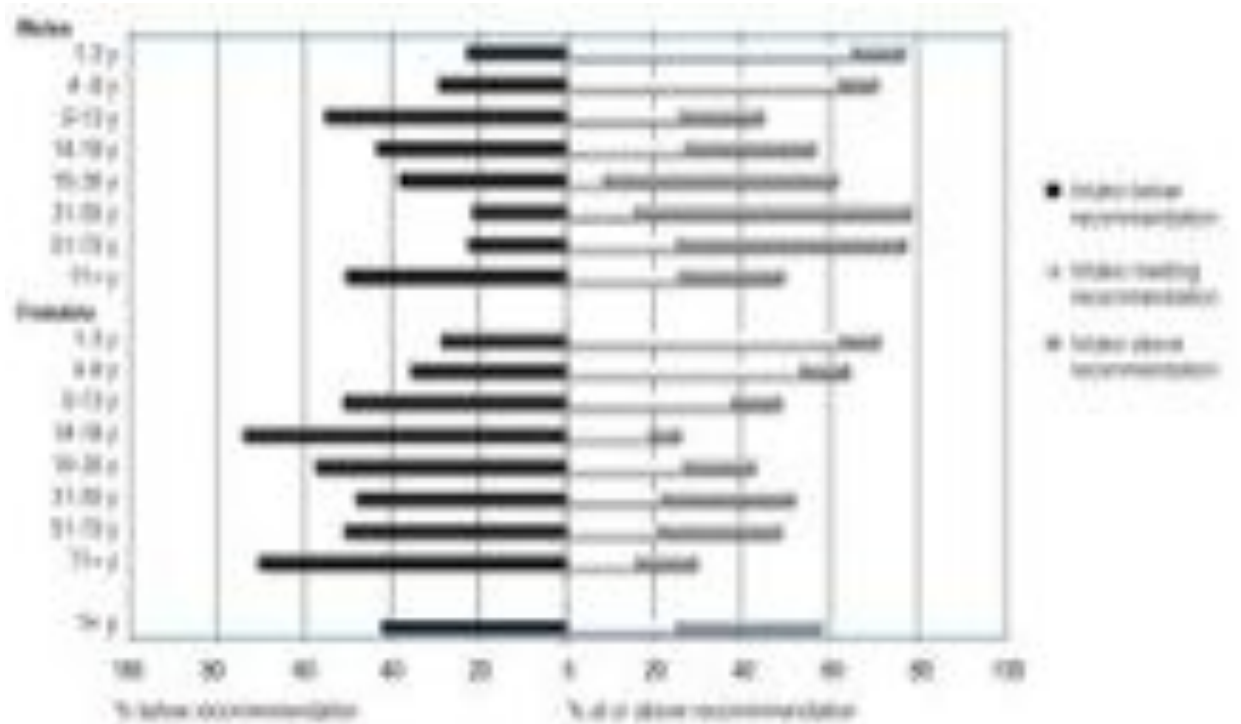
Source: What We Eat in America, NHANES 2007-2010

Figure D1.19 Dairy: Estimated percent of persons below, at, or above recommendation



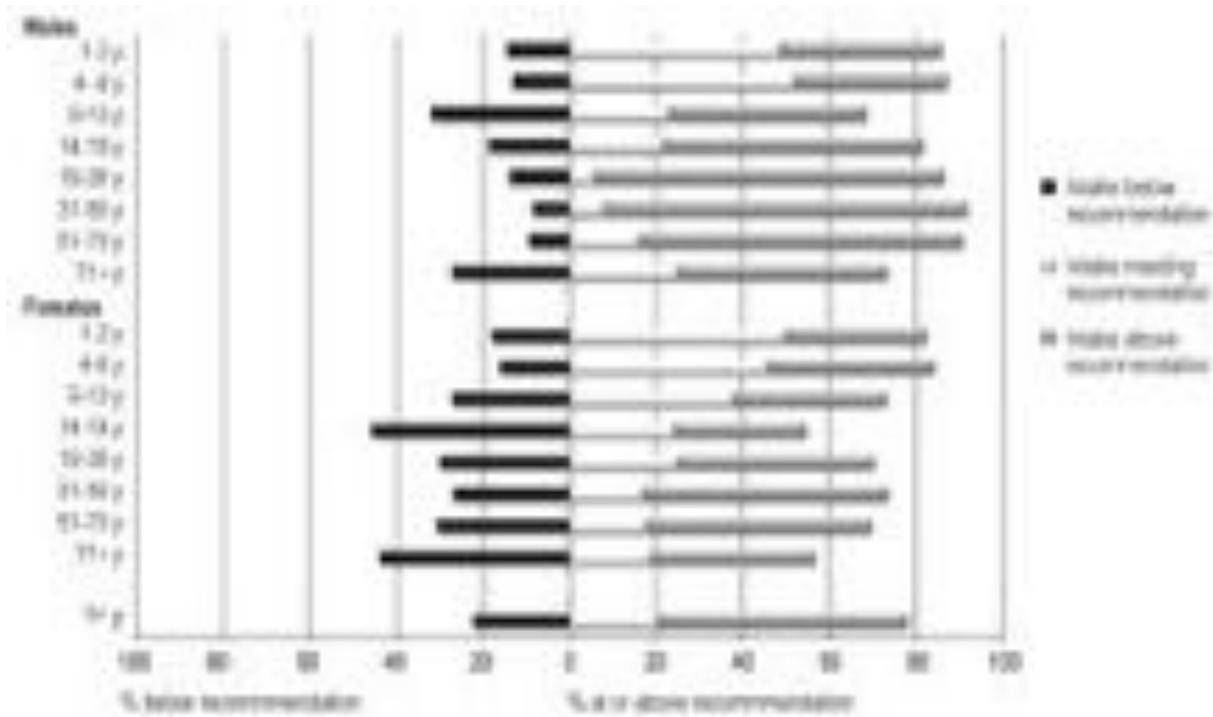
Source: What We Eat in America, NHANES 2007-2010

Figure D1.20 Total Protein foods: Estimated percent of persons below, at, or above recommendation



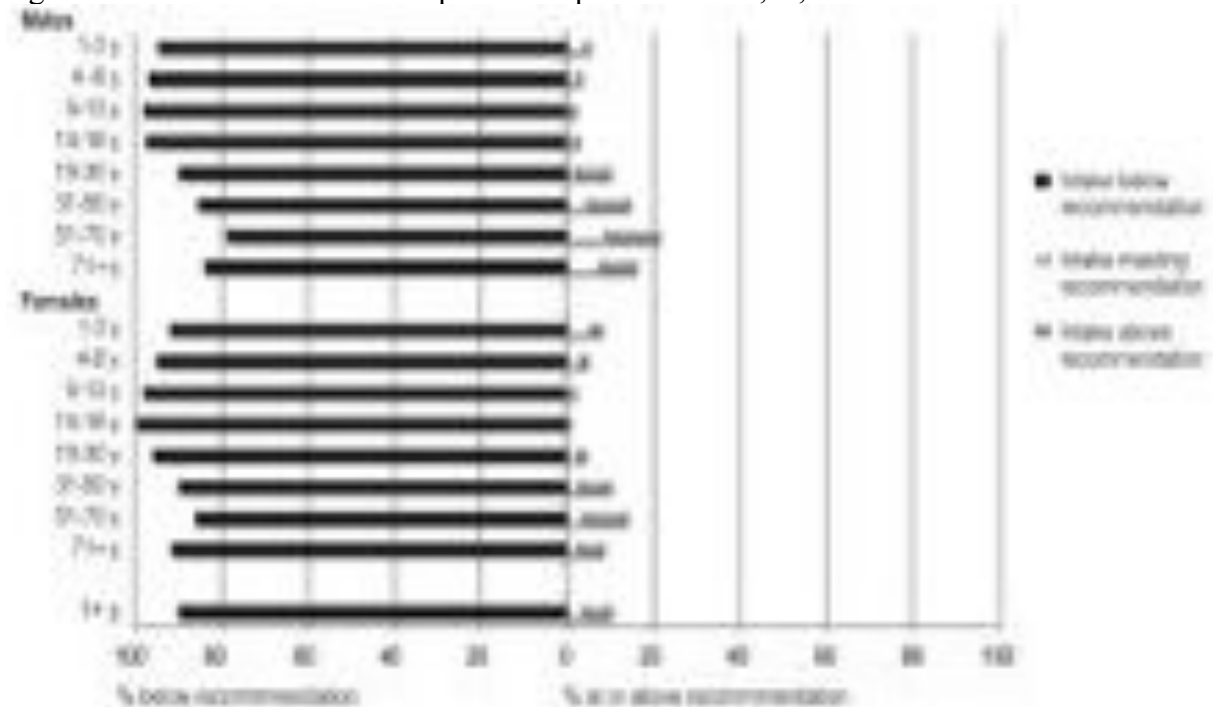
Source: What We Eat in America, NHANES 2007-2010

Figure D1.21 Meat, poultry, eggs: Estimated percent of persons below, at, or above recommendation



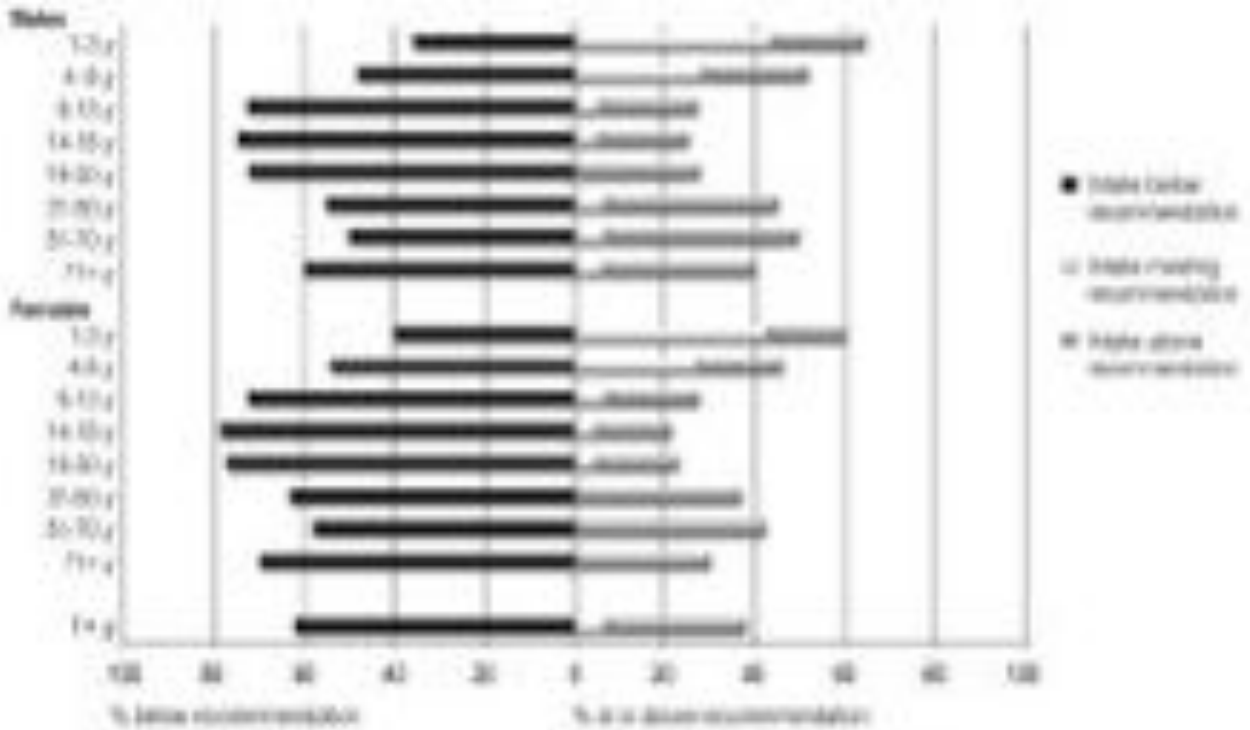
Source: What We Eat in America, NHANES 2007-2010

Figure D1.22 Seafood: Estimated percent of persons below, at, or above recommendation



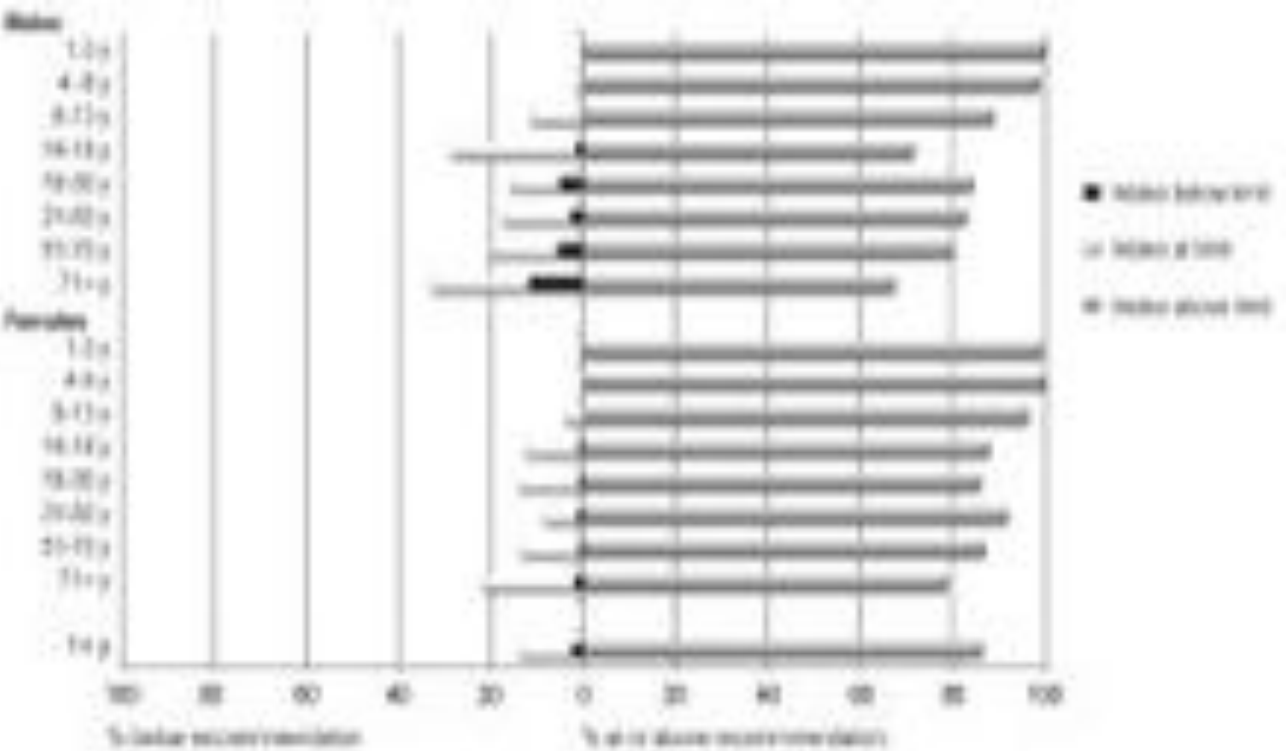
Source: What We Eat in America, NHANES 2007-2010

Figure D1.23 Nuts, seeds, soy: Estimated percent of persons below, at, or above recommendation



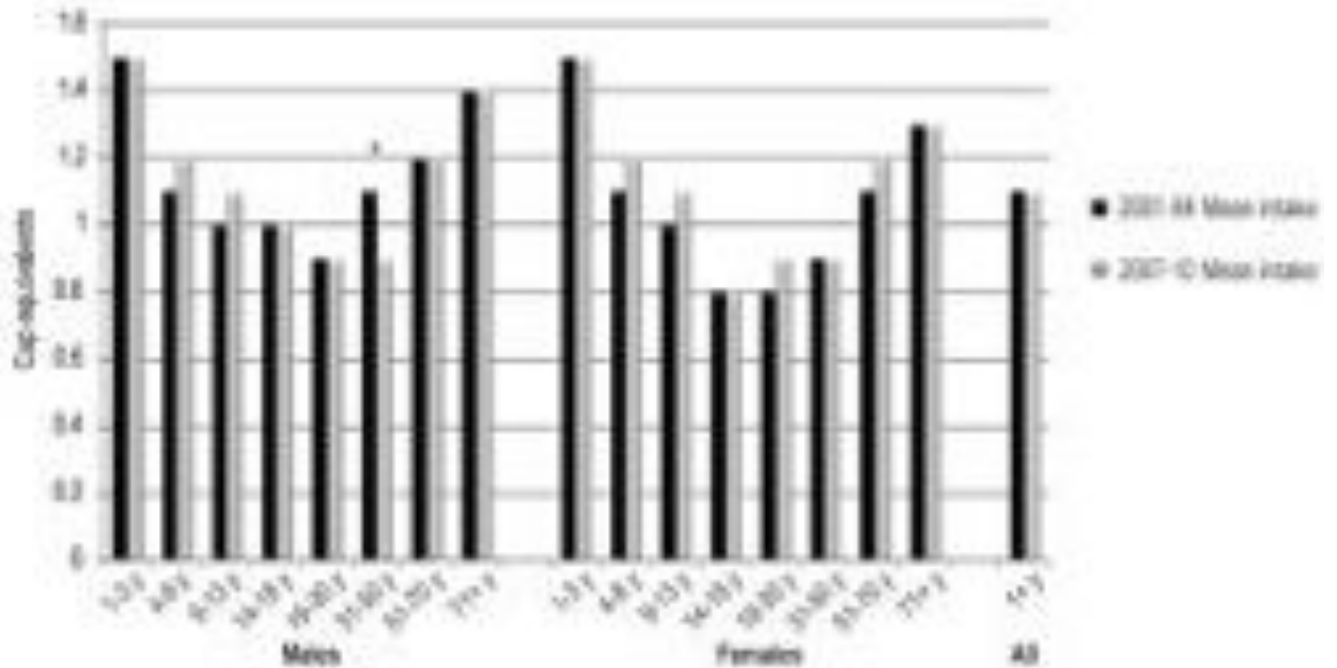
Source: What We Eat in America, NHANES 2007-2010

Figure D1.24 Empty calories: Estimated percent of persons below, at, or above limits



Source: What We Eat in America, NHANES 2007-2010

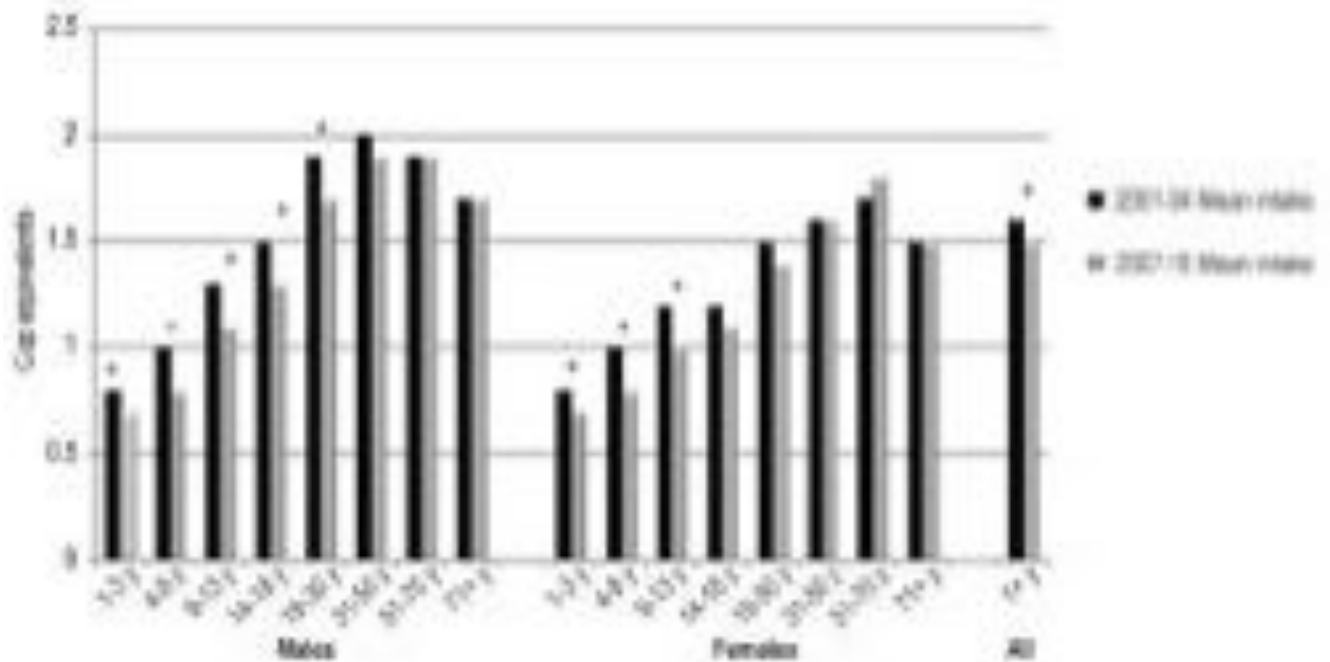
Figure D1.25 Fruit: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

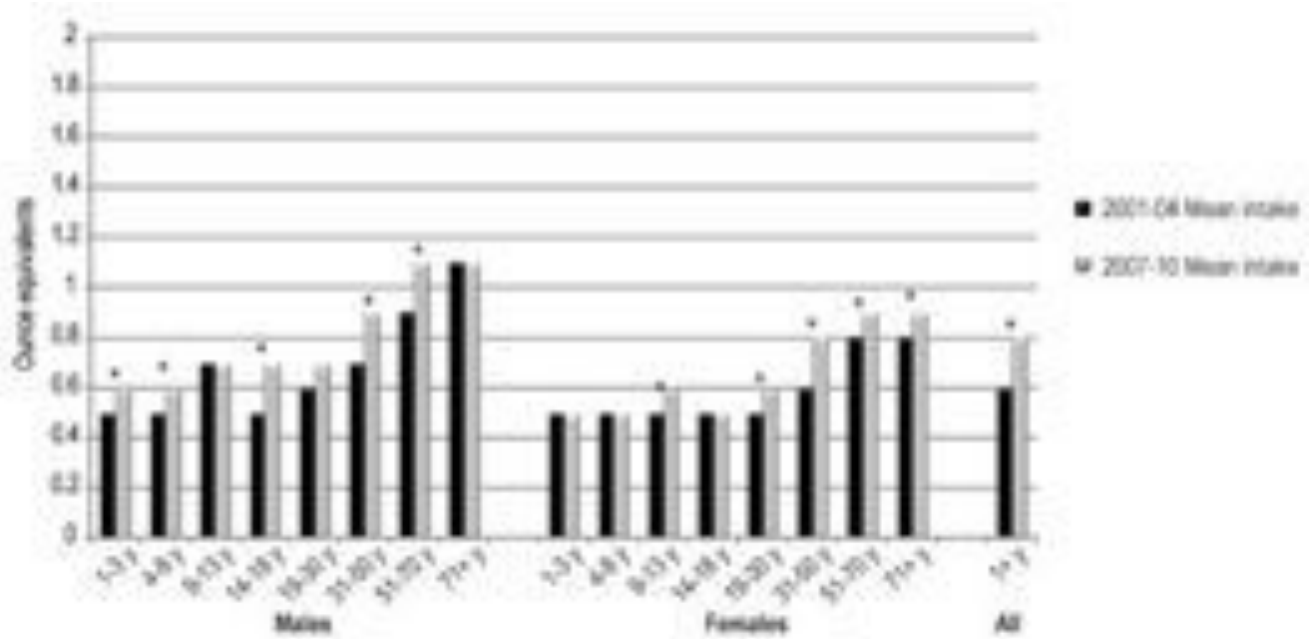
Figure D1.26 Vegetables: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

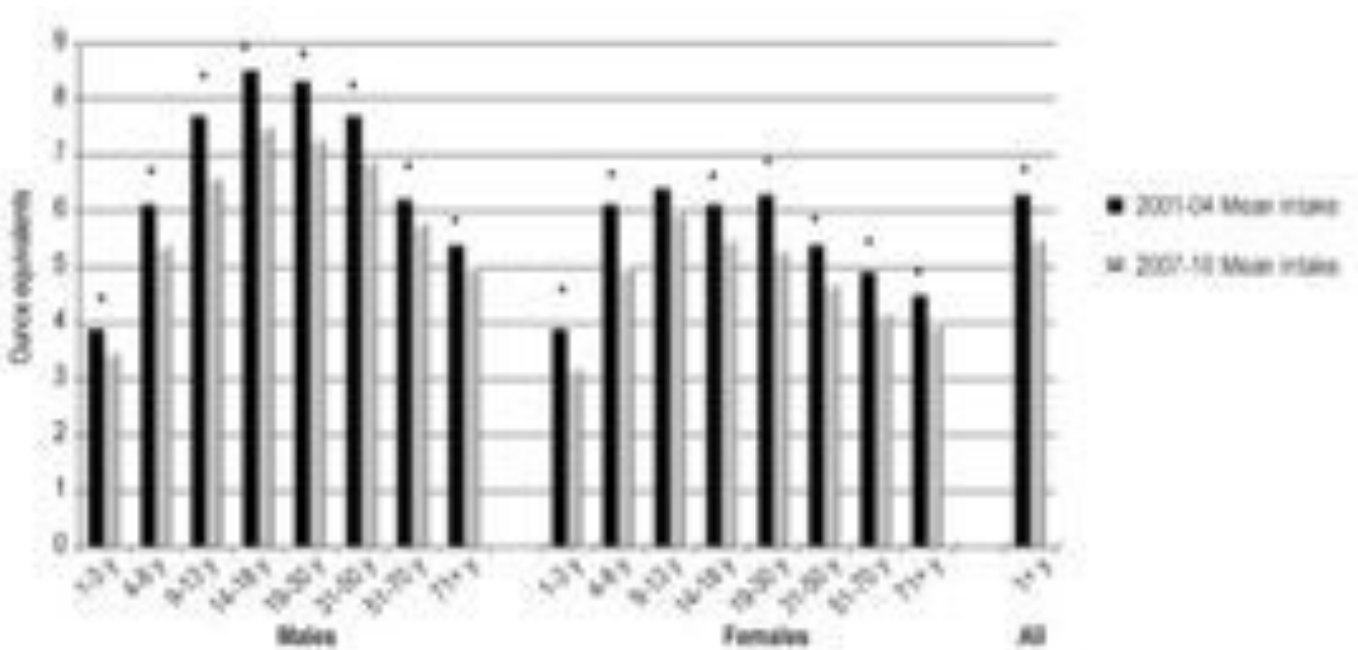
Figure D1.27 Whole grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

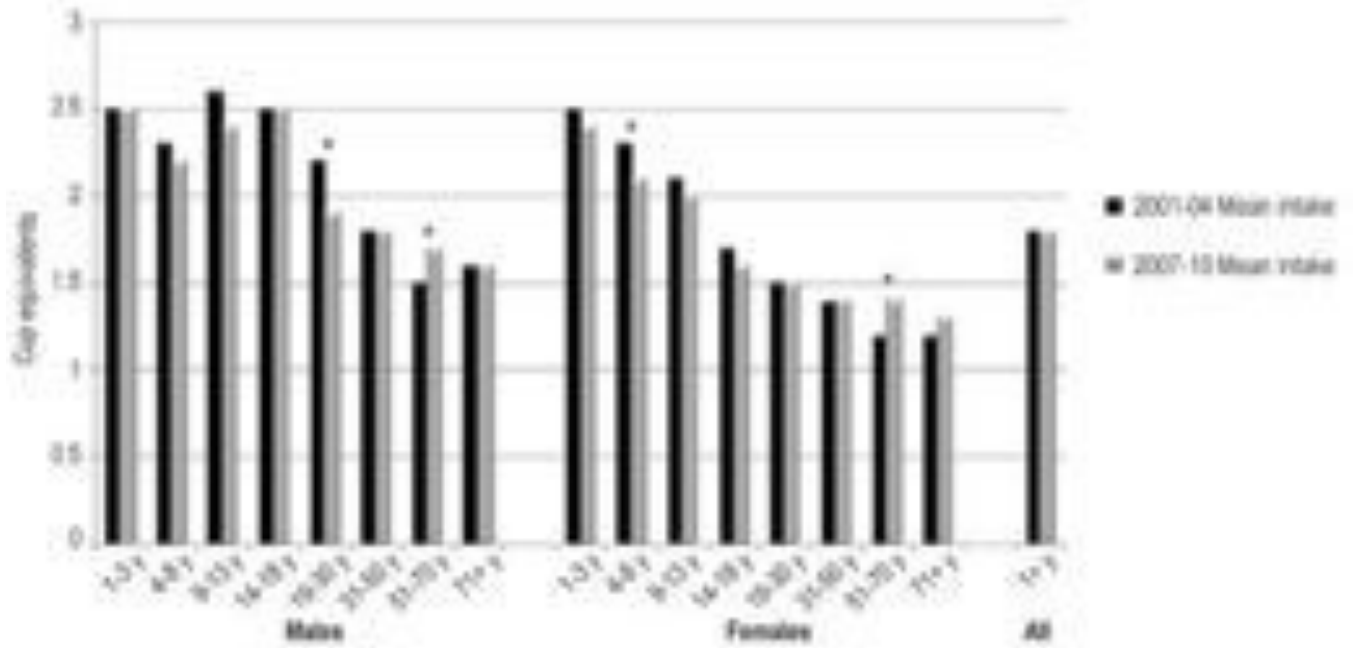
Figure D1.28 Refined grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

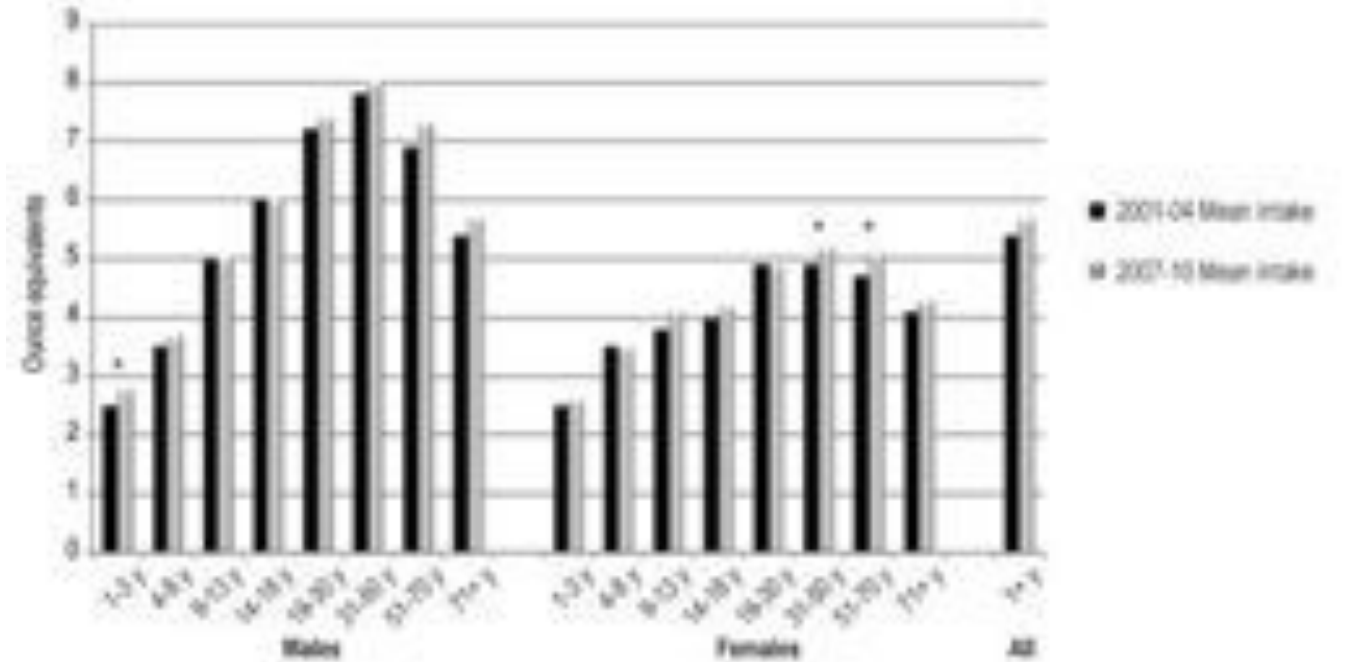
Figure D1.29 Dairy: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

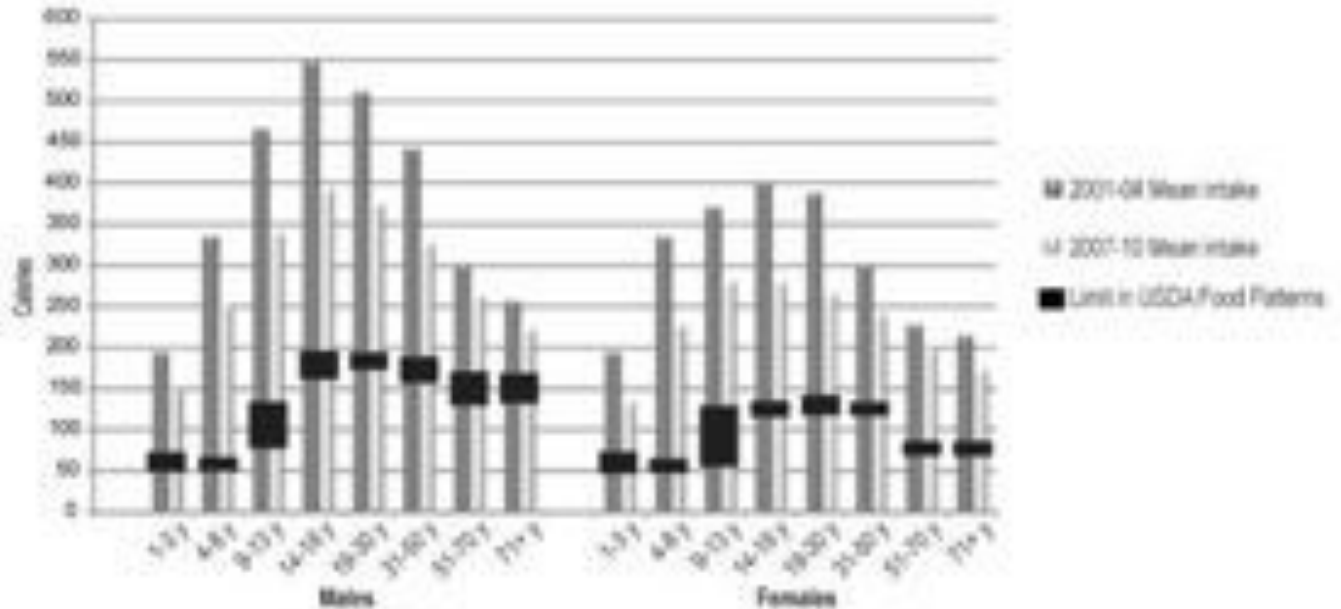
Figure D1.30 Protein Foods: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

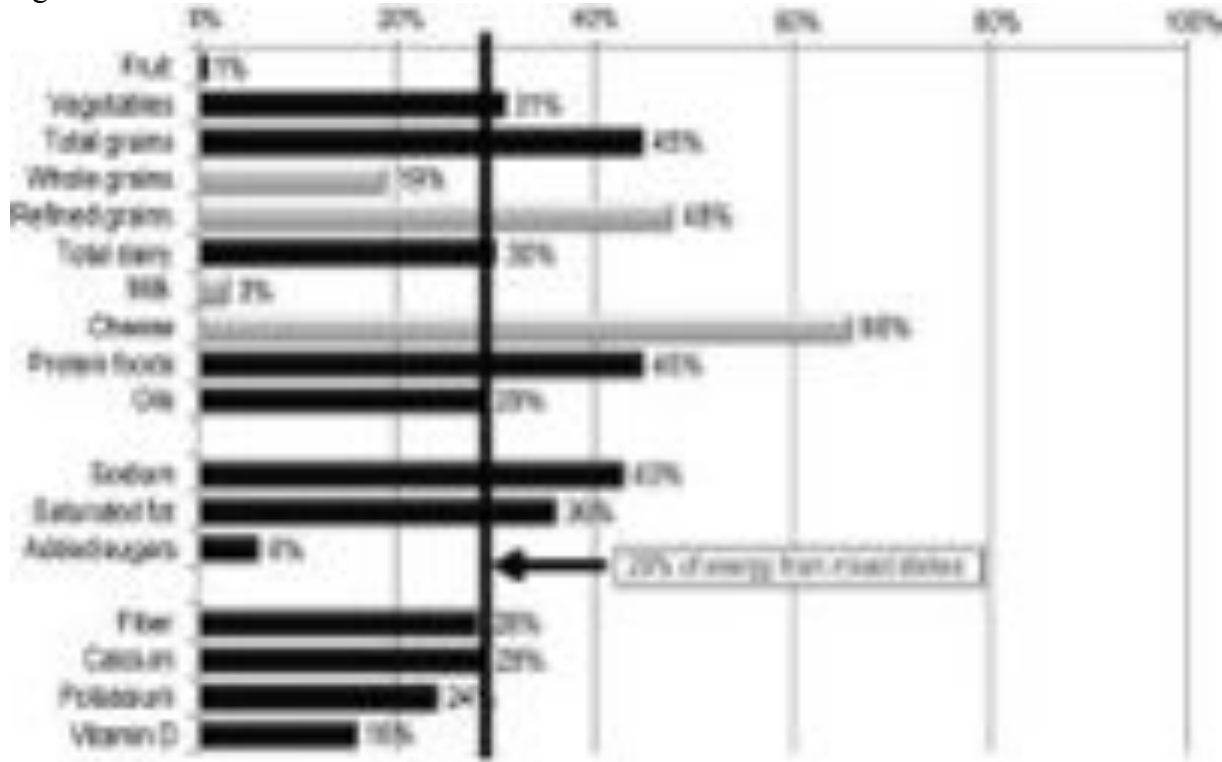
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.31 Added sugars intakes in 2001-04 and 2007-10 by age/sex groups in comparison to added sugars limits in the USDA Food Patterns



Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

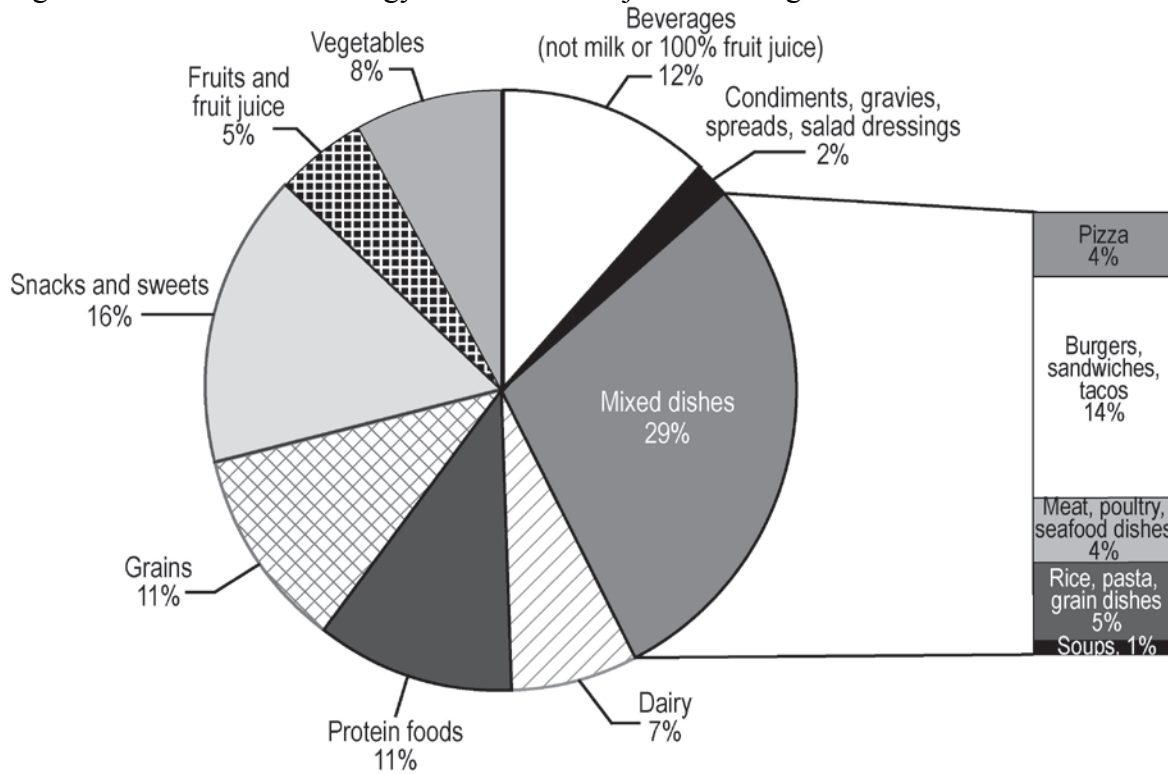
Figure D1.32 Percent of Total intake from mixed dishes



Note: Bars in lighter shades are for subgroups that “break out” the food group above them.

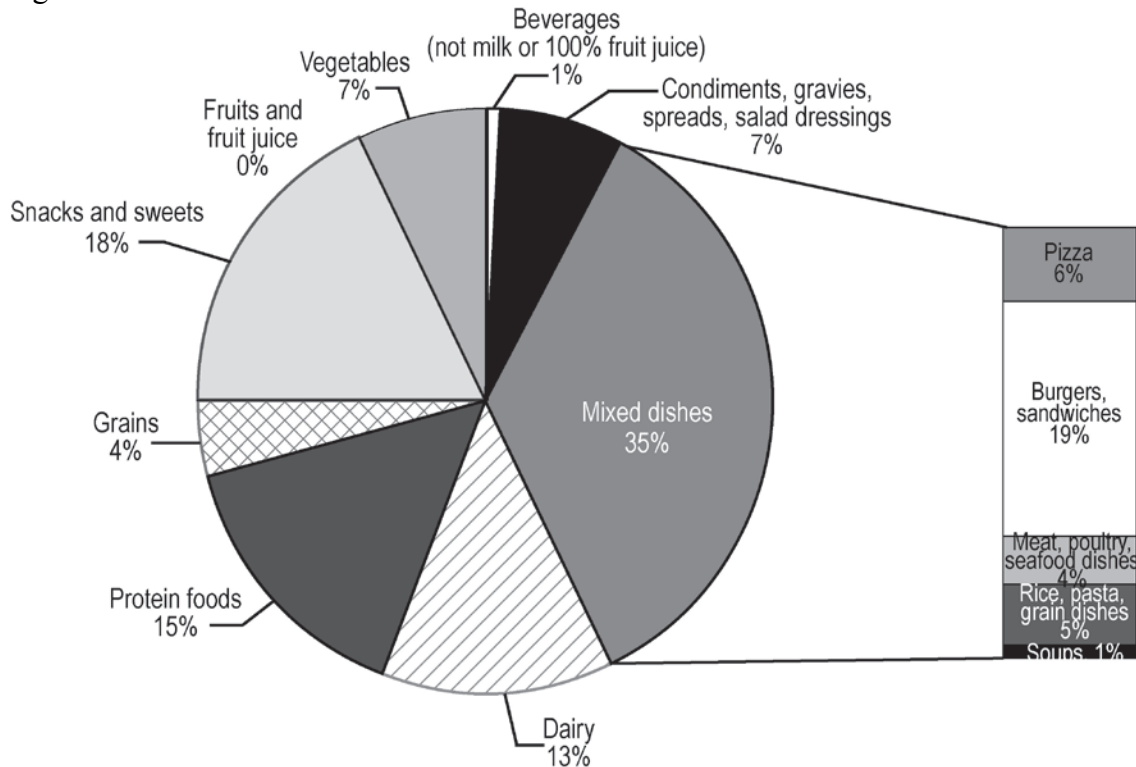
Source: What We Eat in America, NHANES 2009-2010

Figure D1.33 Percent of Energy Intake from Major food categories



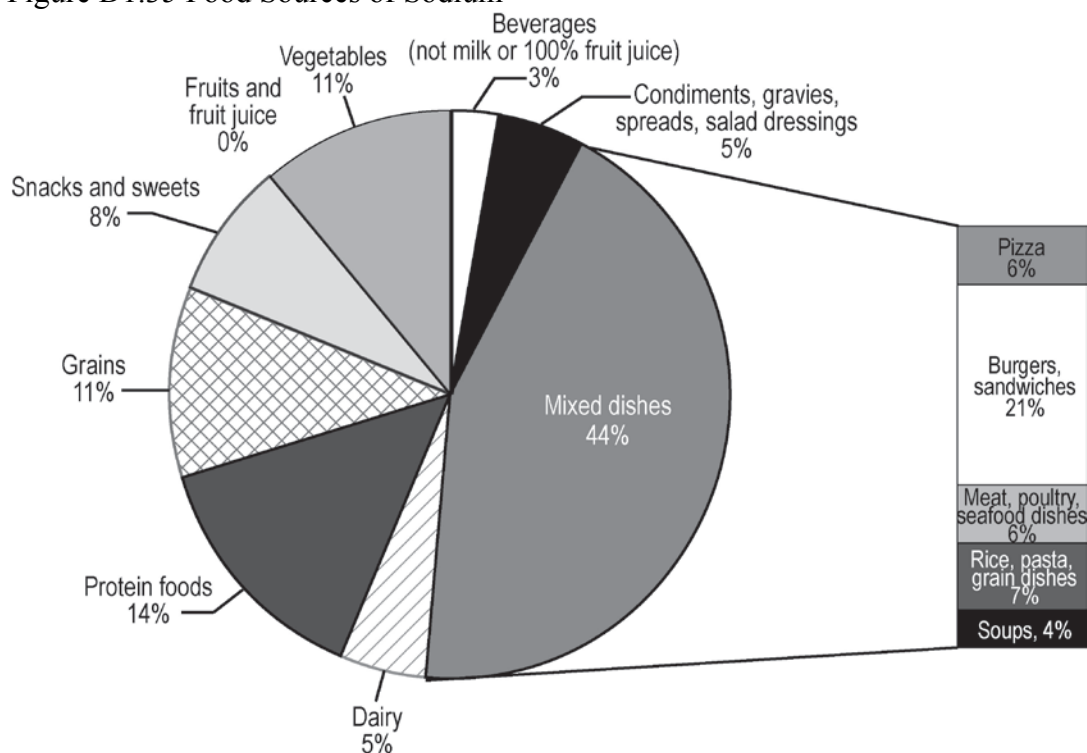
Source: What We Eat in America, NHANES 2009-2010

Figure D1.34 Food sources Saturated Fat



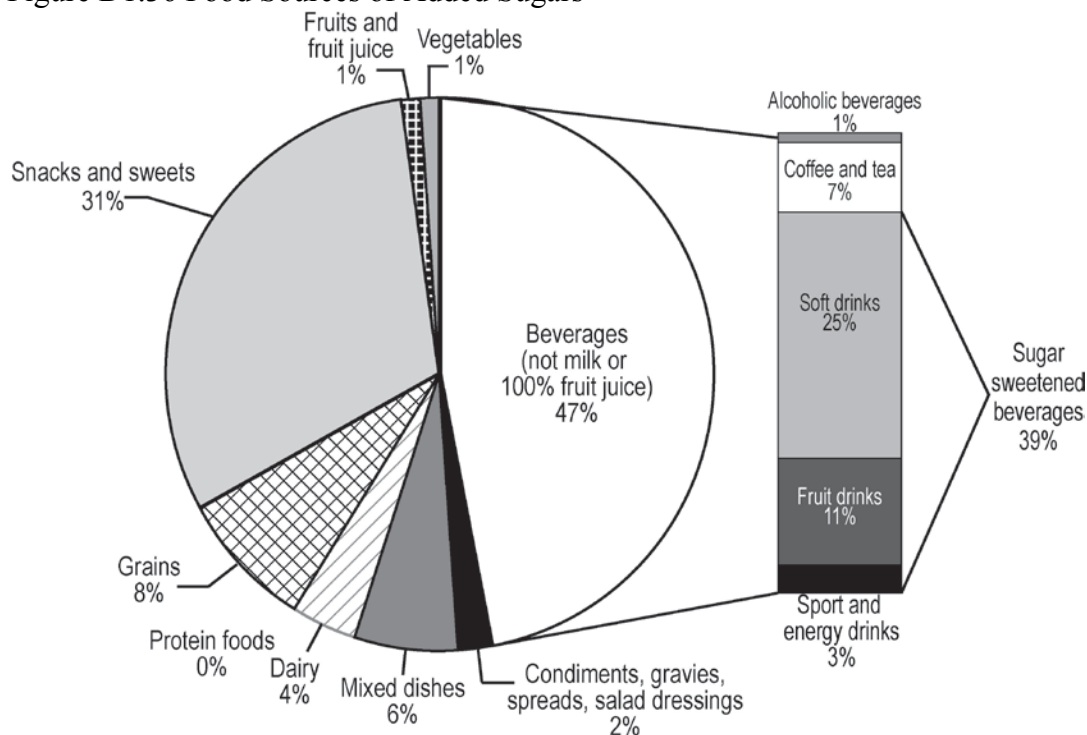
Source: What We Eat in America, NHANES 2009-2010

Figure D1.35 Food Sources of Sodium



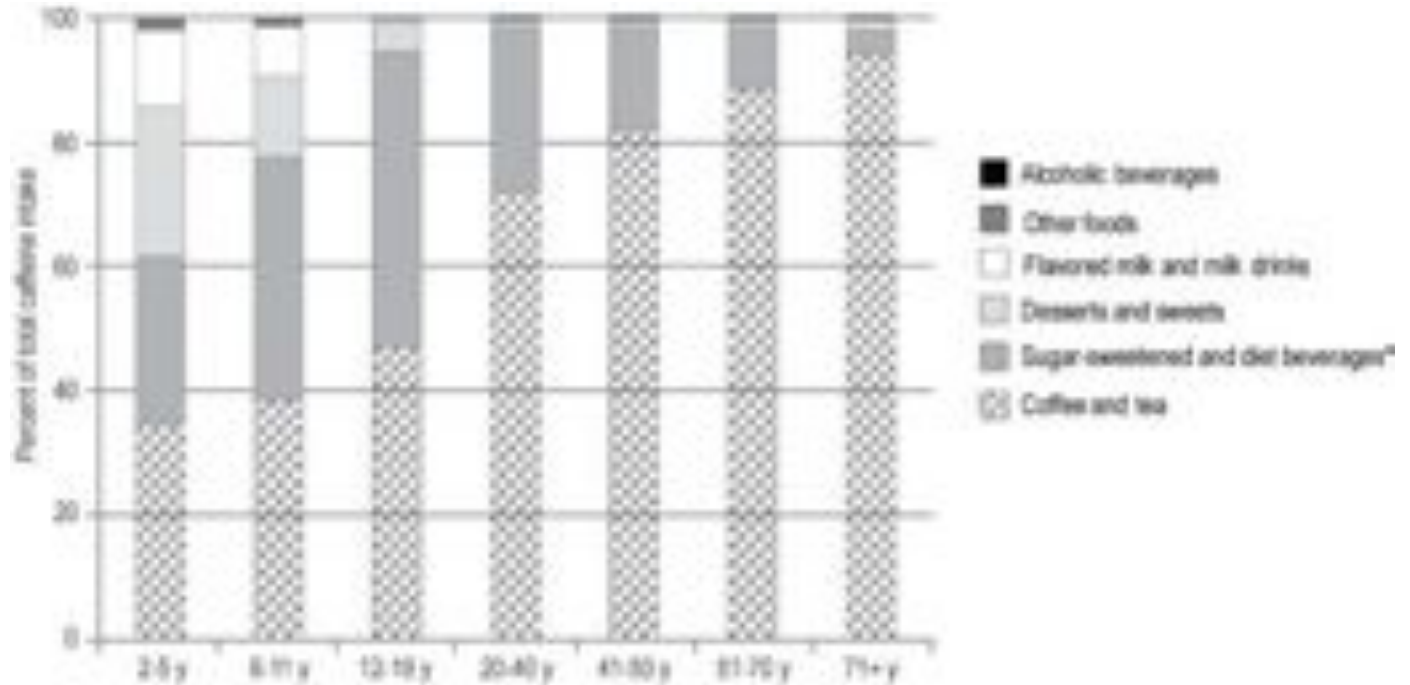
Source: What We Eat in America, NHANES 2009-2010

Figure D1.36 Food Sources of Added Sugars



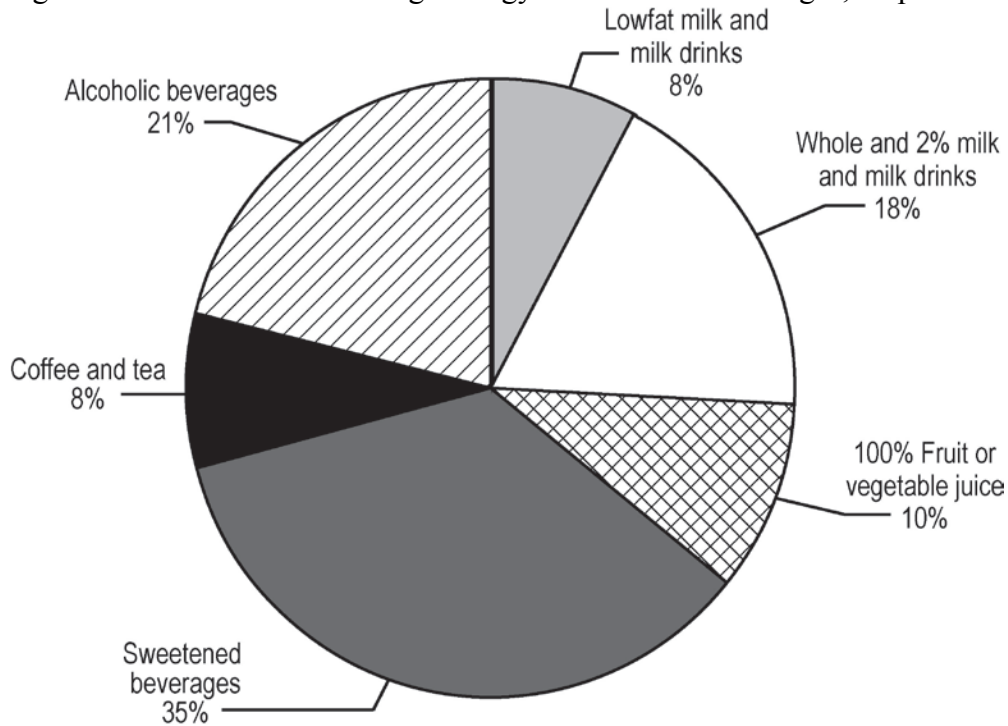
Source: What We Eat in America, NHANES 2009-2010

Figure D1.37 Caffeine sources by age group



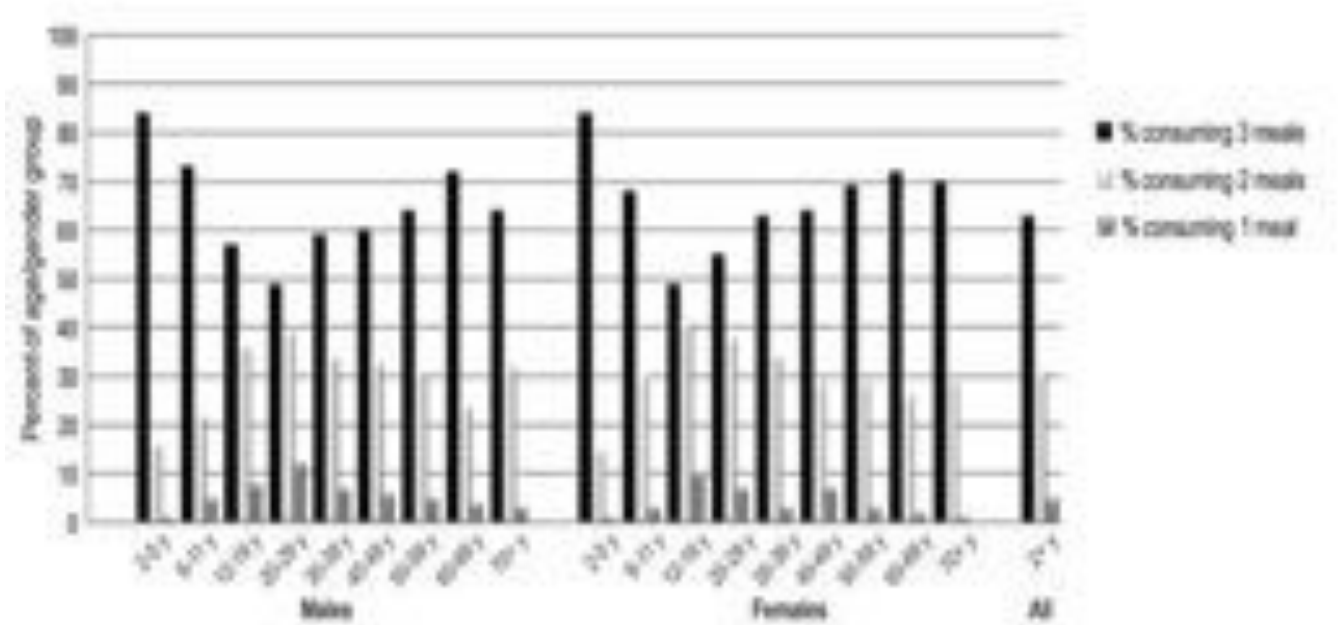
Source: What We Eat in America, NHANES 2009-2010

Figure D1.38 Percent of beverage energy from various beverages, all persons 2+



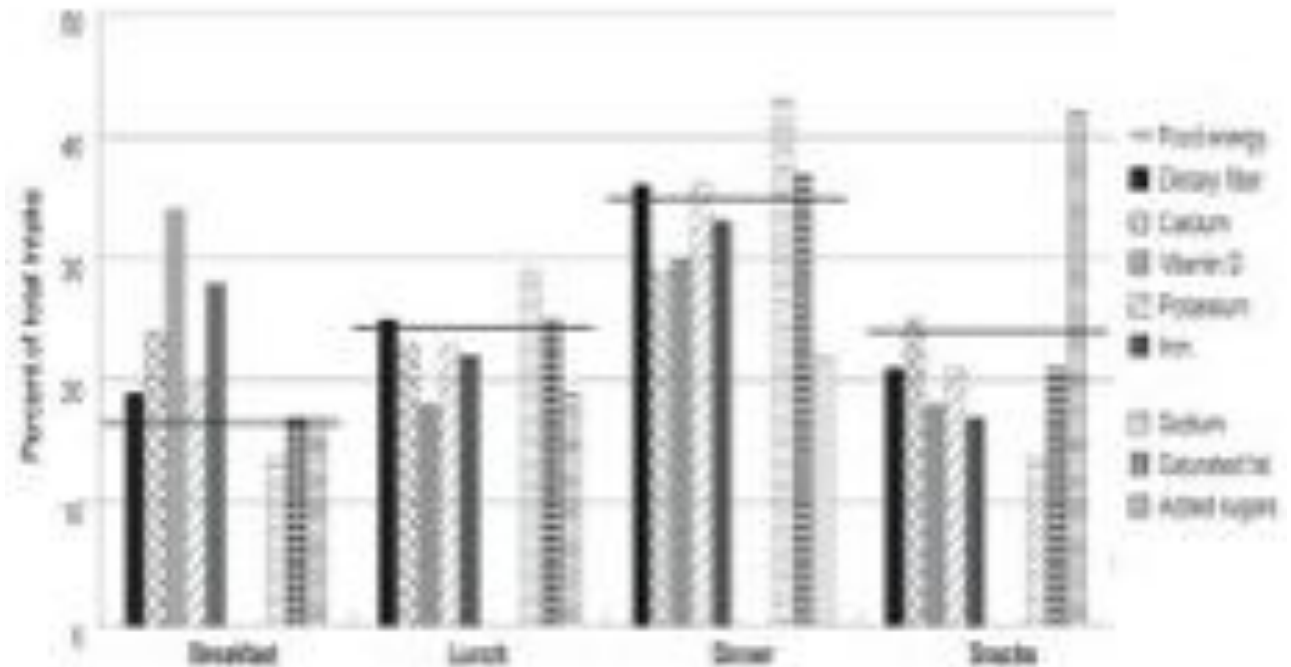
Source: What We Eat in America, NHANES 2009-2010

Figure D1.39 Number of meals reported per day by age/sex group



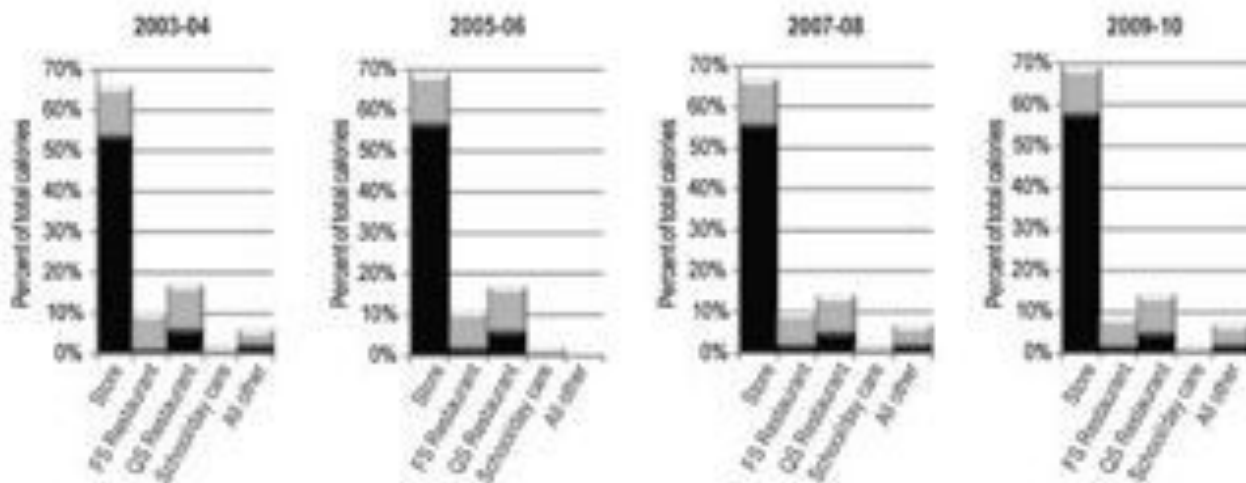
Source: What We Eat in America, NHANES 2009-2010

Figure D1.40 Percent of total daily intake of nutrients of concern from each eating occasion, for the population 2+



Source: What We Eat in America, NHANES 2009-2010

Figure D1.41 Percent of calories by where food was obtained and consumed

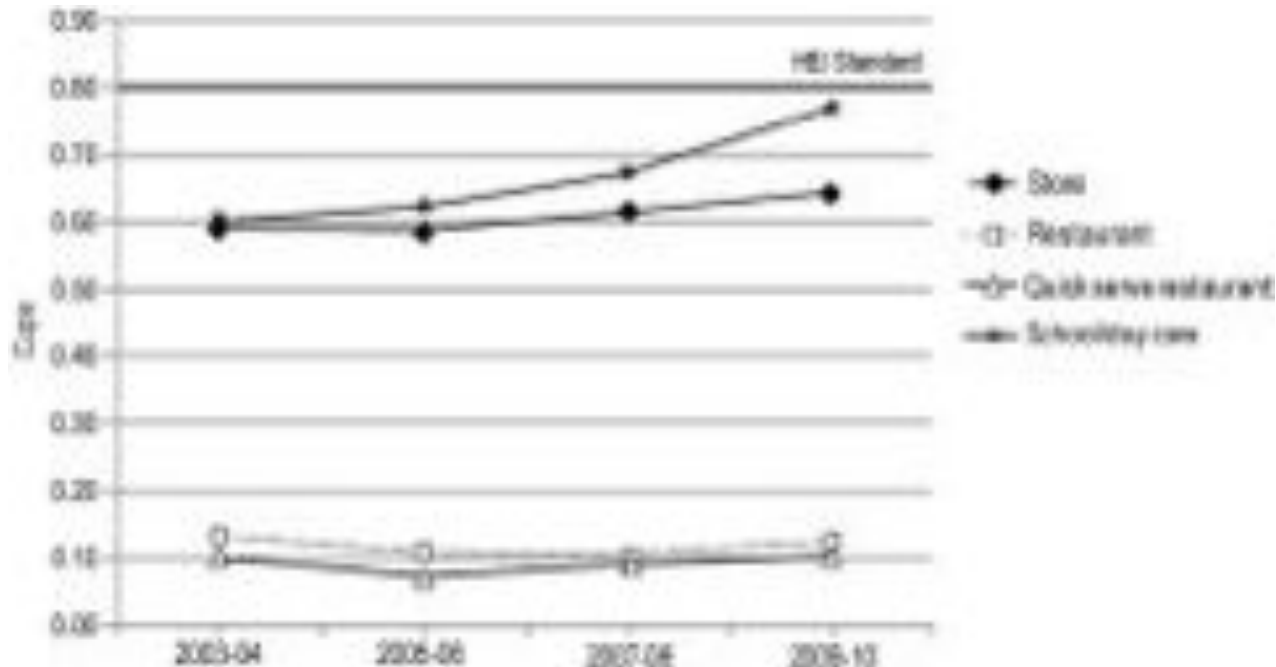


Darker shading indicates food eaten at home; lighter shading indicates food eaten away from home.

FS = Full Service (sit-down service); QS = Quick Service (fast food, food trucks, etc.)

Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

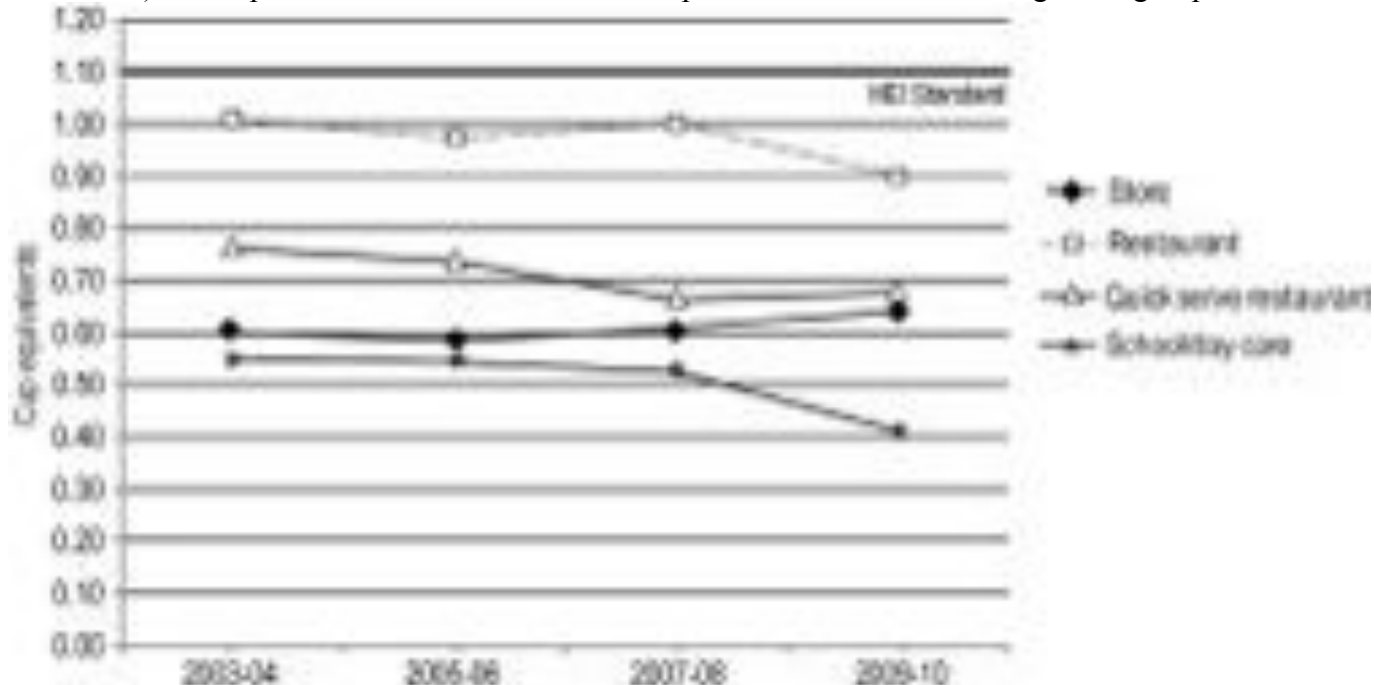
Figure D1.42 Fruit group density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the fruit group.



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)

Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

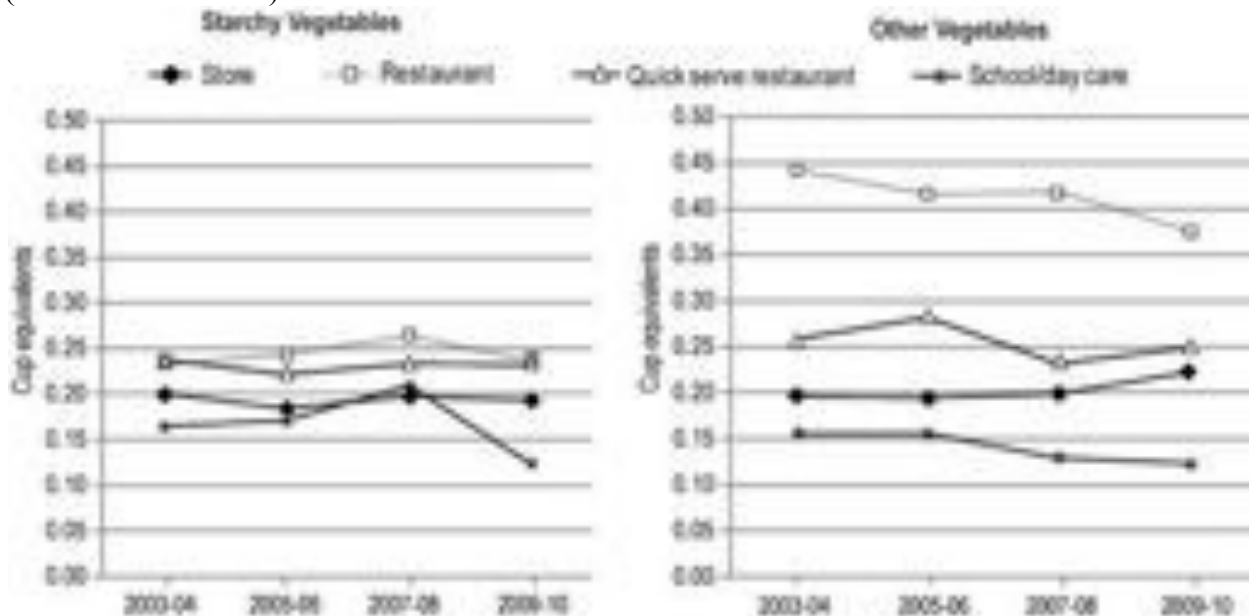
Figure D1.43 Vegetable density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the vegetable group



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)

Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

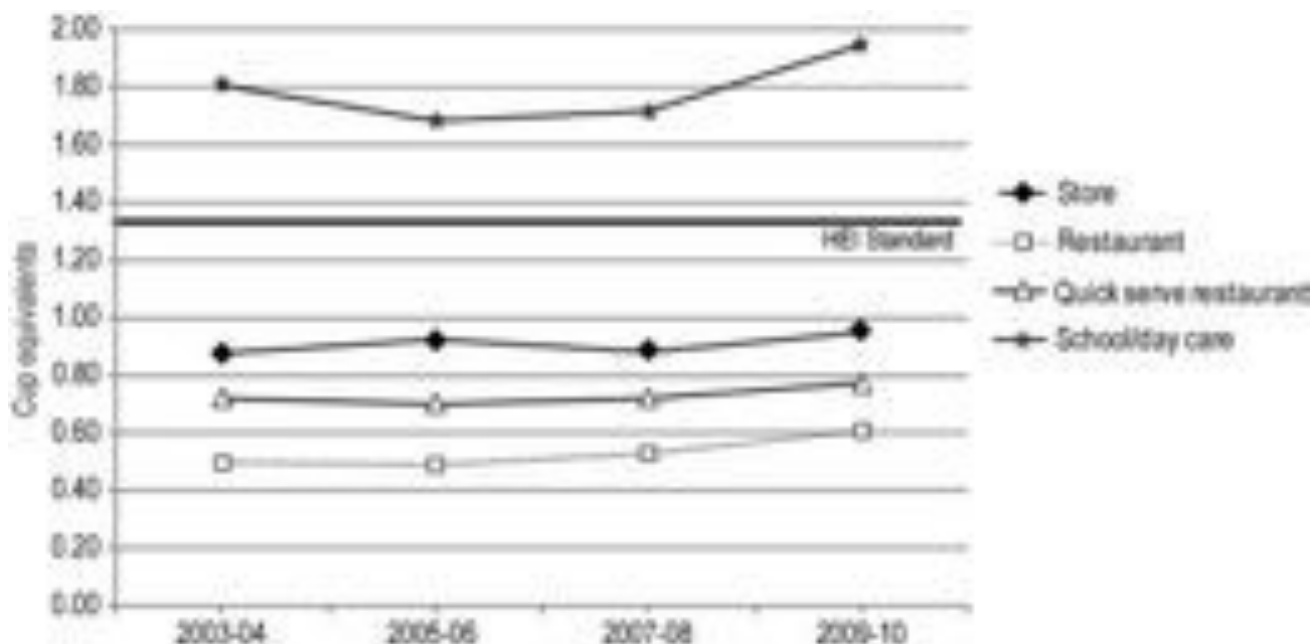
Figure D1.44 Vegetable subgroup density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10)



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)

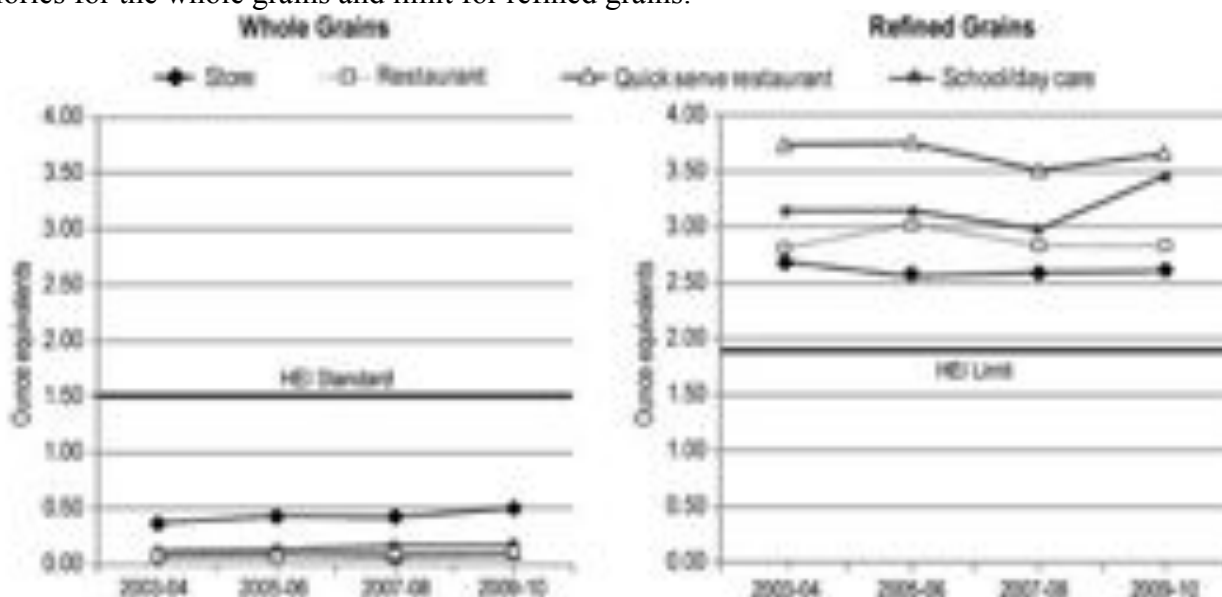
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.45 Dairy group density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the dairy group



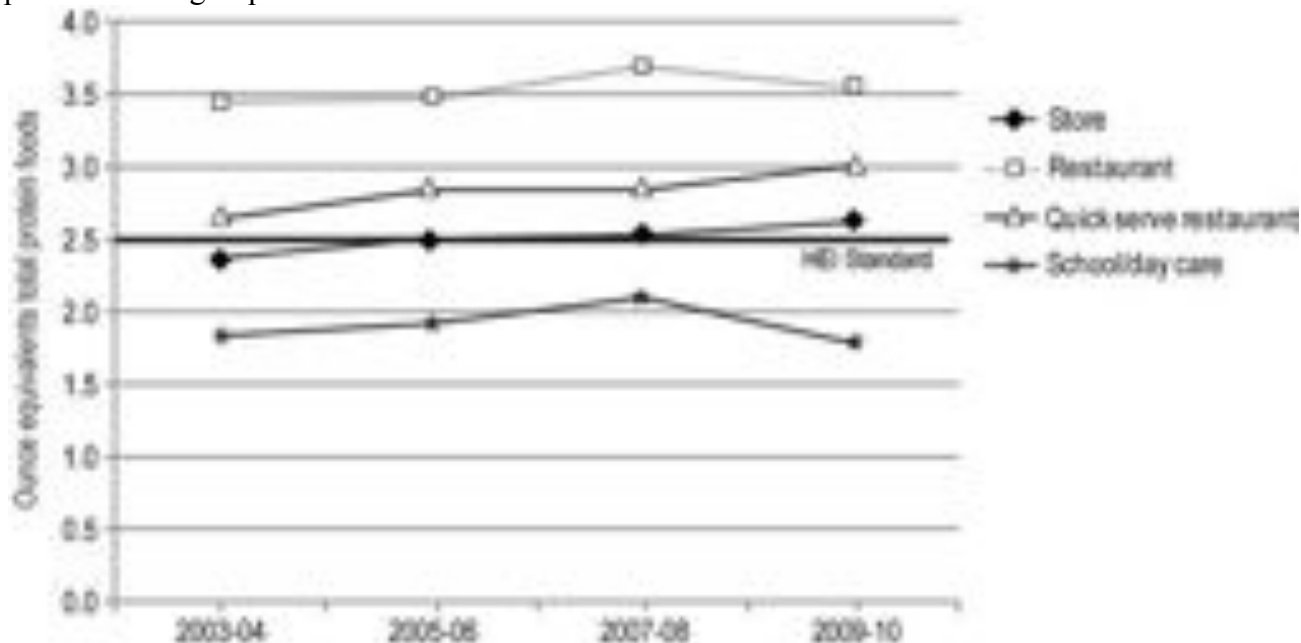
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.46 Grain group density (whole and refined) : ounce eqs per 1000 calories by where obtained over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the whole grains and limit for refined grains.



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

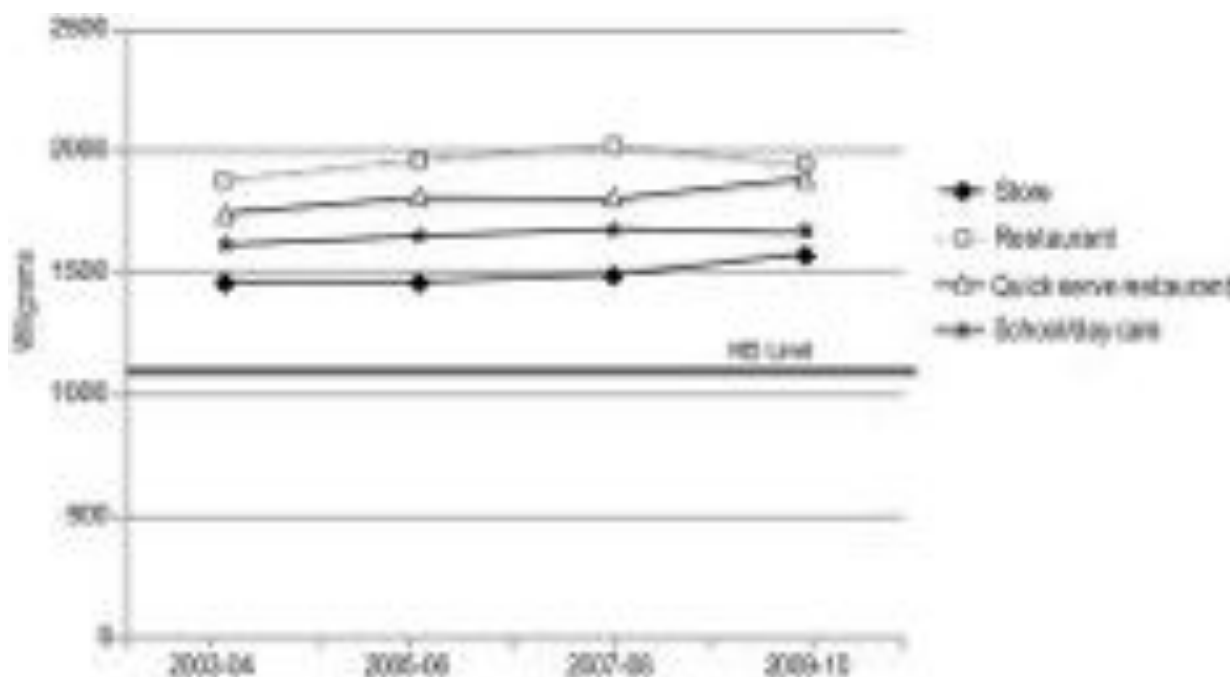
Figure D1.47 Protein Foods Group density: ounce eqs per 1000 calories by where obtained, over time (2001-04 vs. 2007-10) in comparison to the 2010 HEI standard per 1000 calories for the protein foods group.



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)

Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

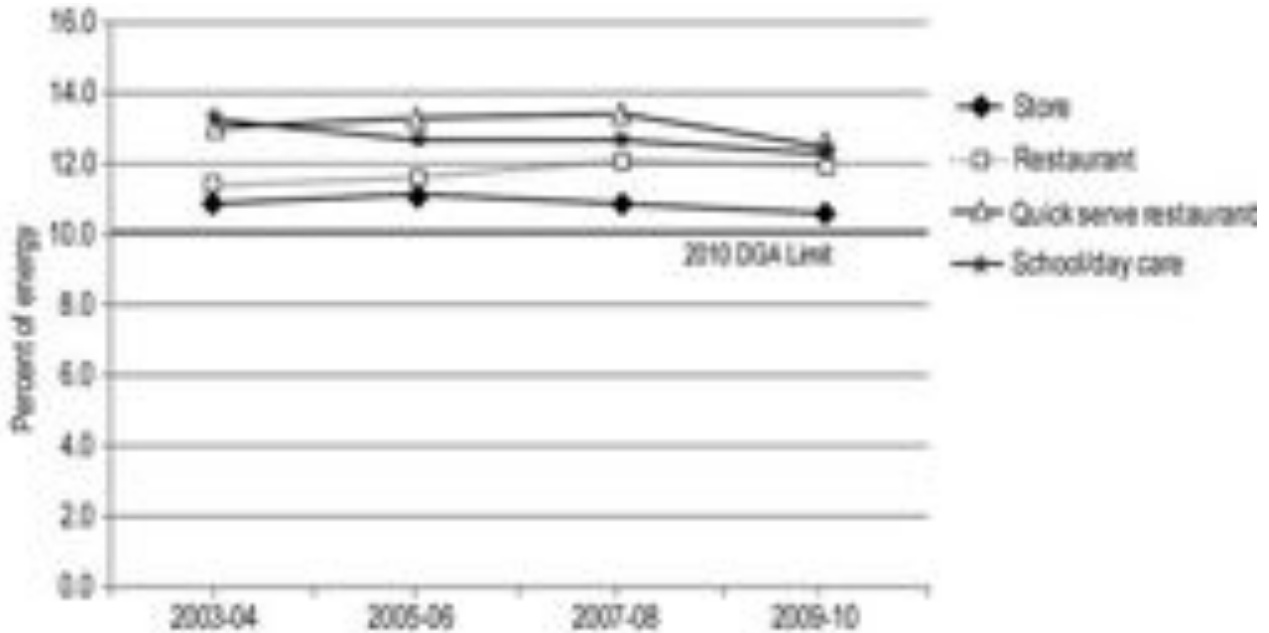
Figure D1.48 Sodium density: milligrams per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI limit per 1000 calories for sodium.



Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)

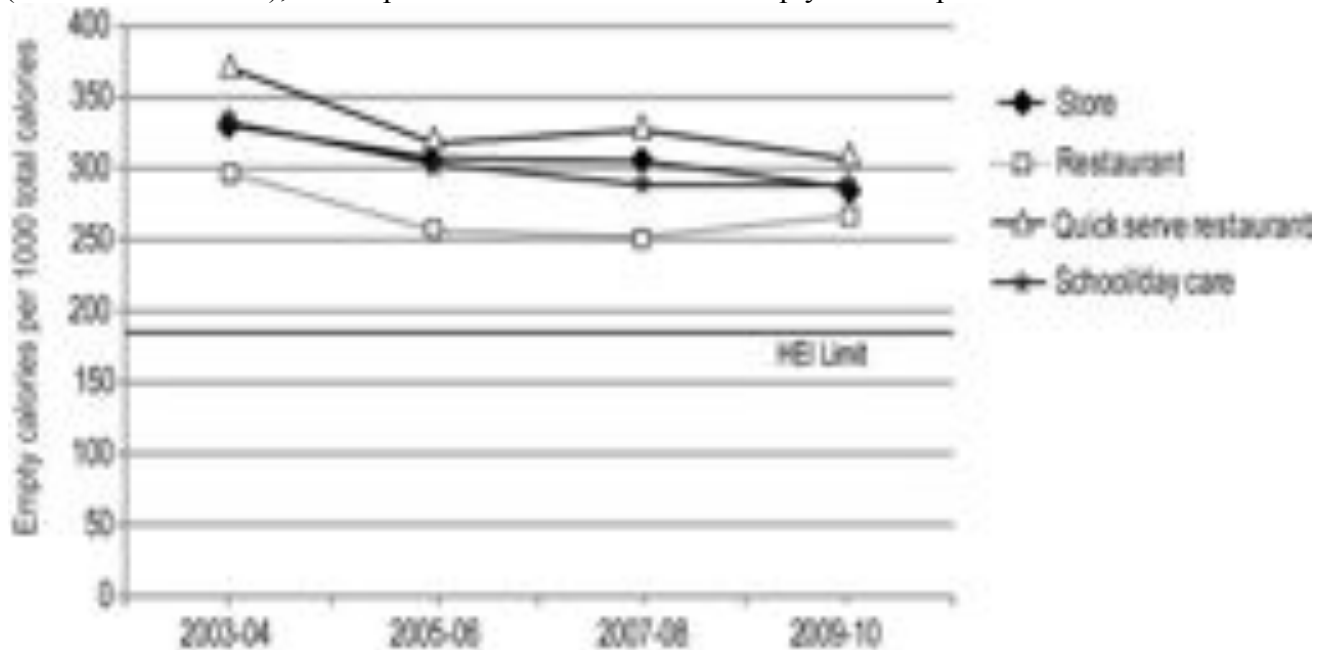
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.49 Saturated fat density: percent of energy by where obtained, over time (2003-04 to 2009-10), in comparison to the 2010 DGA limit for saturated fat as a percent of energy.



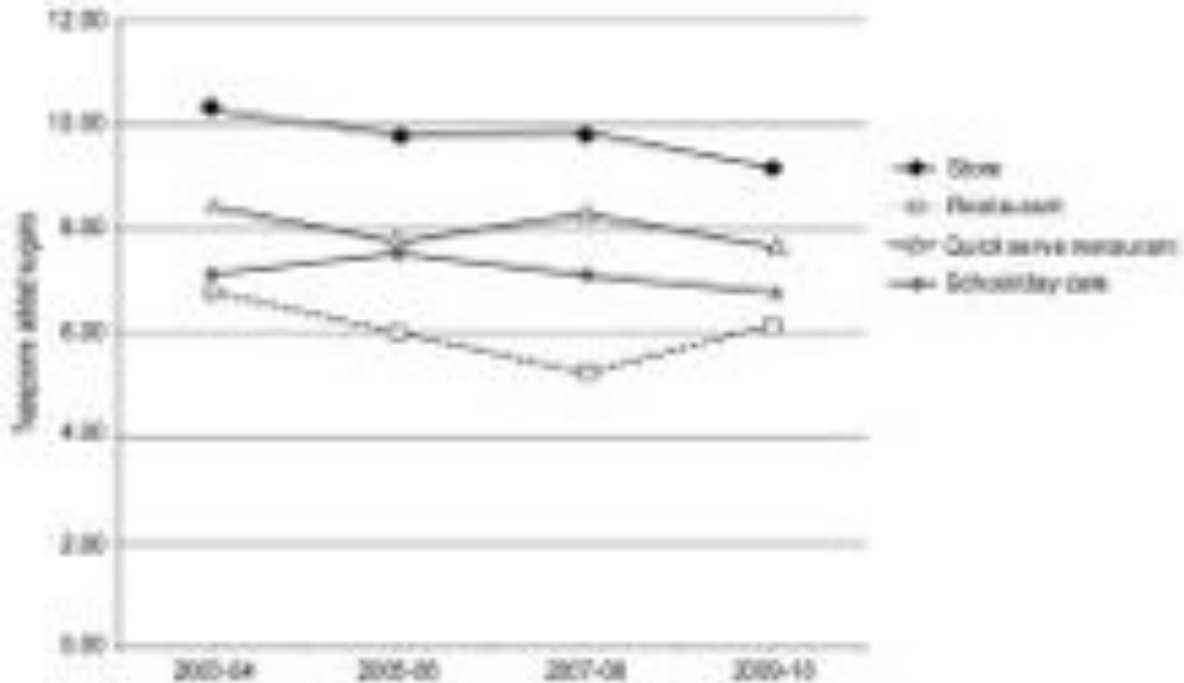
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.50 Empty calorie density: calories per 1000 calories by where obtained, over time (2003-04 to 2009-10), in comparison to the HEI limit for empty calories per 1000 calories.



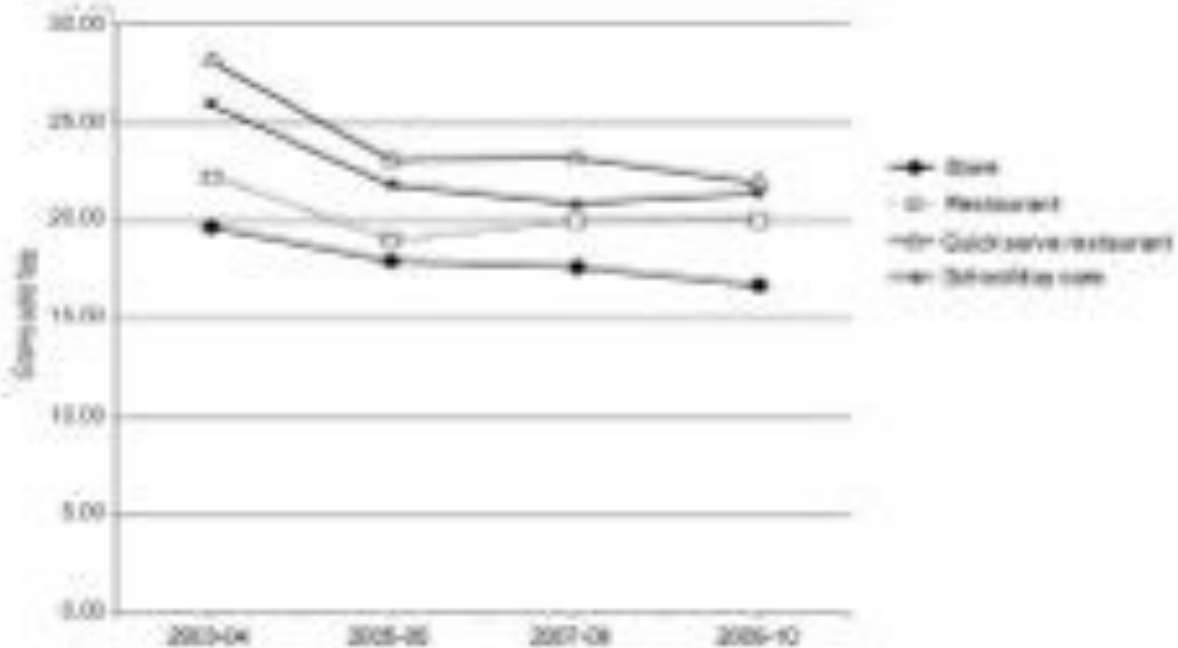
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.51 Added sugars density: Added sugars per 1000 calories by where obtained, over time (2003-04 to 2009-10)



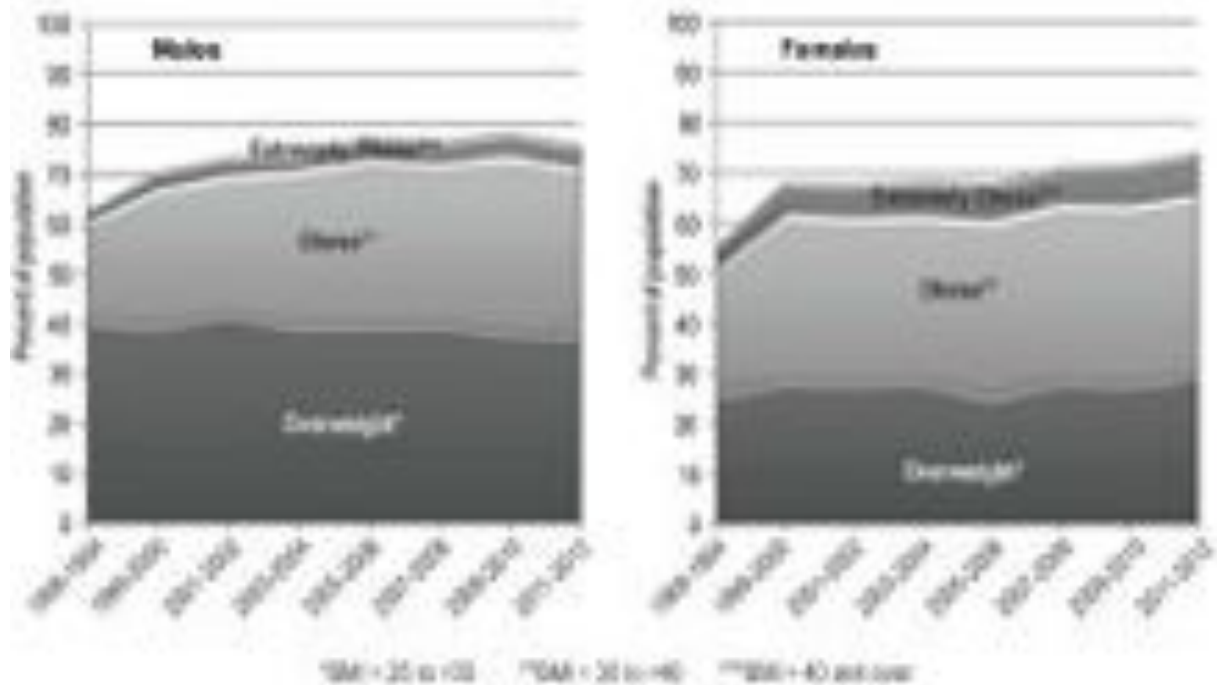
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.52 Solid fats density: Solid fats per 1000 calories by where obtained, over time (2003-04 to 2009-10)



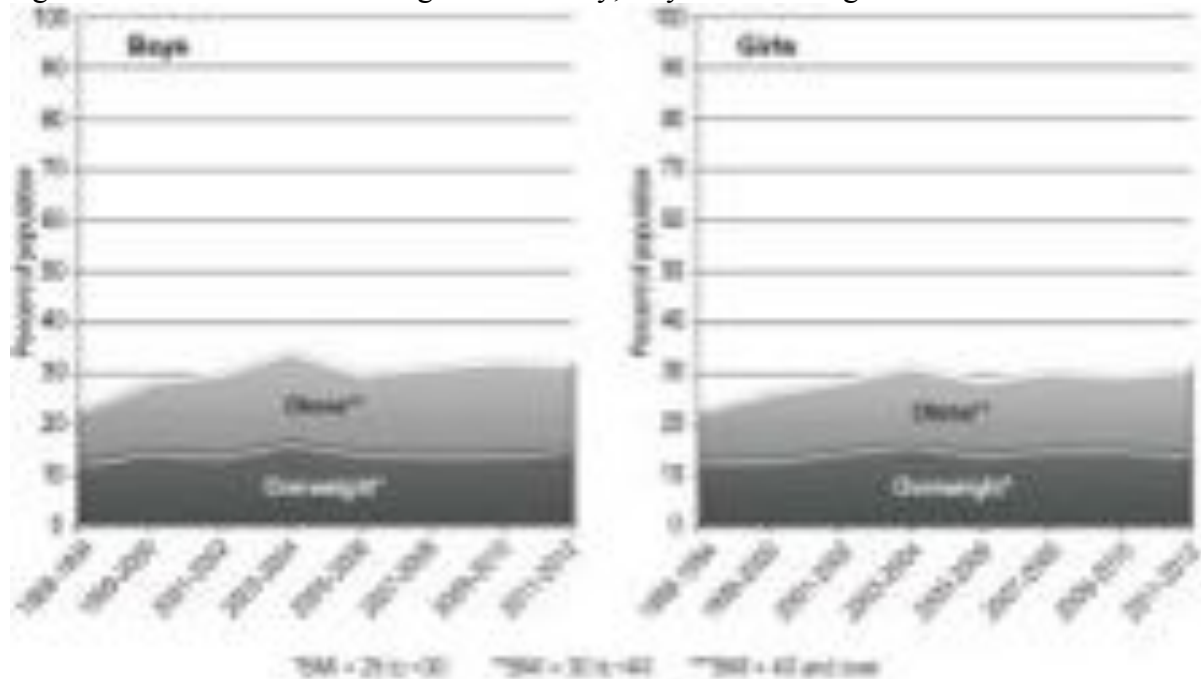
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
 Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.53 Trends in overweight and obesity, Males and Females ages 20+.



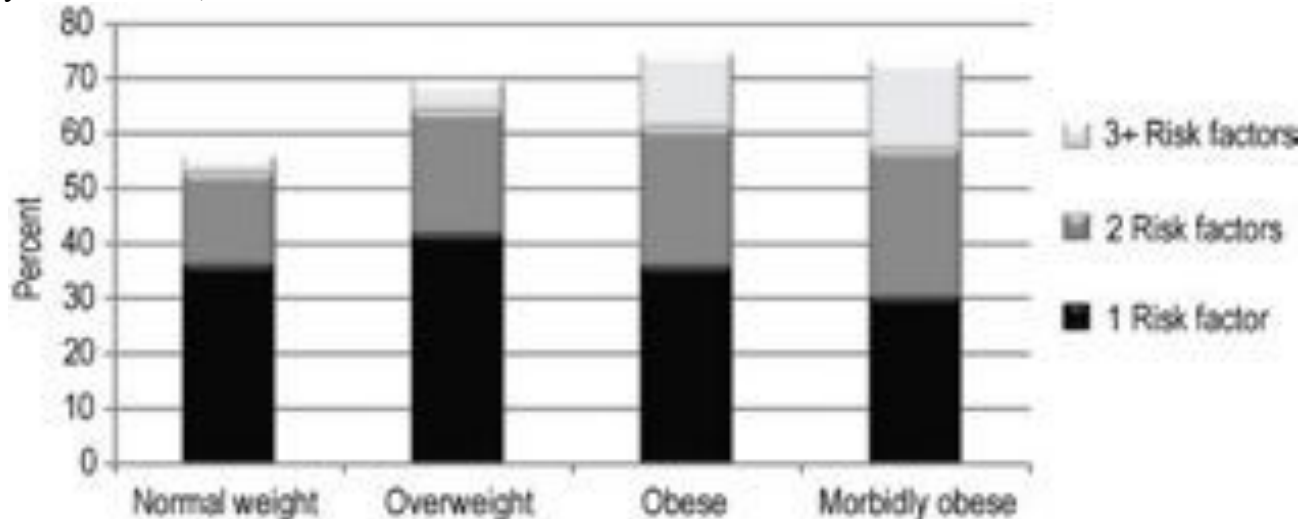
Source: Fryar, CD, Carroll, MD, Ogden, CL. Prevalence of Overweight and Obesity among Adults: United States, 1960–1962 Through 2011–2012. CDC/NCHS, the Health E-Stat, September 2014

Figure D1.54 Trends in overweight and obesity, Boys and Girls ages 2-19.



Source: Fryar, CD, Carroll, MD, Ogden, CL. Prevalence of Overweight and Obesity among Children and Adolescents: United States, 1963–1965 Through 2011–2012. CDC/NCHS, the Health E-Stat, September 2014

Figure D1.55 Prevalence and number of CVD risk factors by weight category, among adults 18 years and older, NHANES 2007-10.



Note: Risk factors included: total diabetes, total hypertension, total dislipidemia, and self reported smoking

Source: Saydah S, Bullard KM, Cheng Y, Ali MK, Gregg EW, Geiss L, et al. Trends in cardiovascular disease risk factors by obesity level in adults in the United States, NHANES 1999-2010. Obesity (Silver Spring). 2014.

Figure D1.56 Vegetable intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual vegetable intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



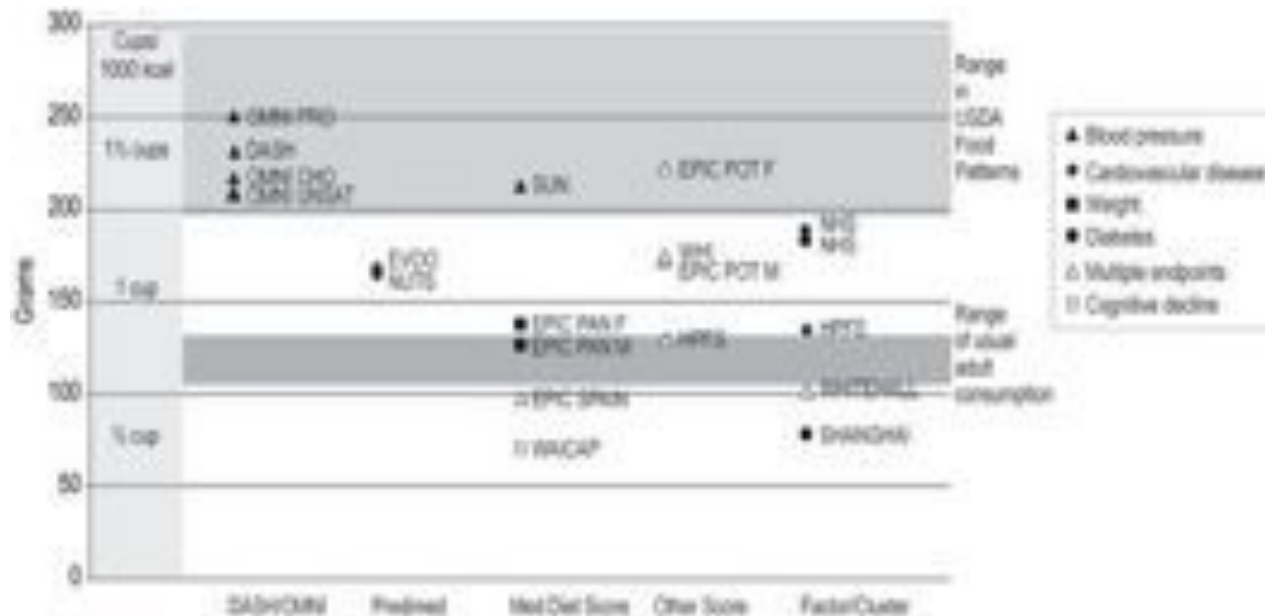
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31.

Figure D1.57 Fruit intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual fruit intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



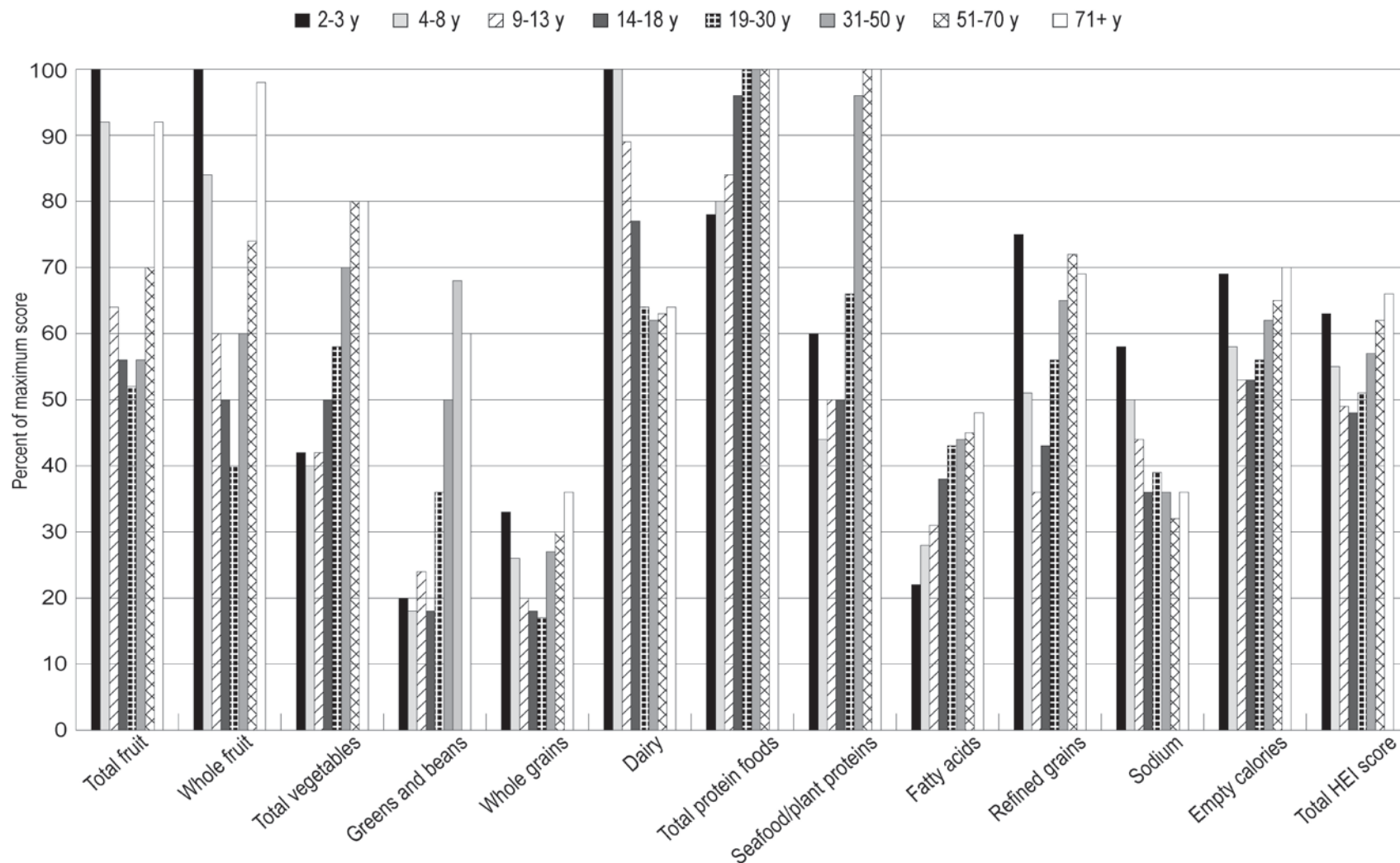
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31.

Figure D1.58 Dairy intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual dairy intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



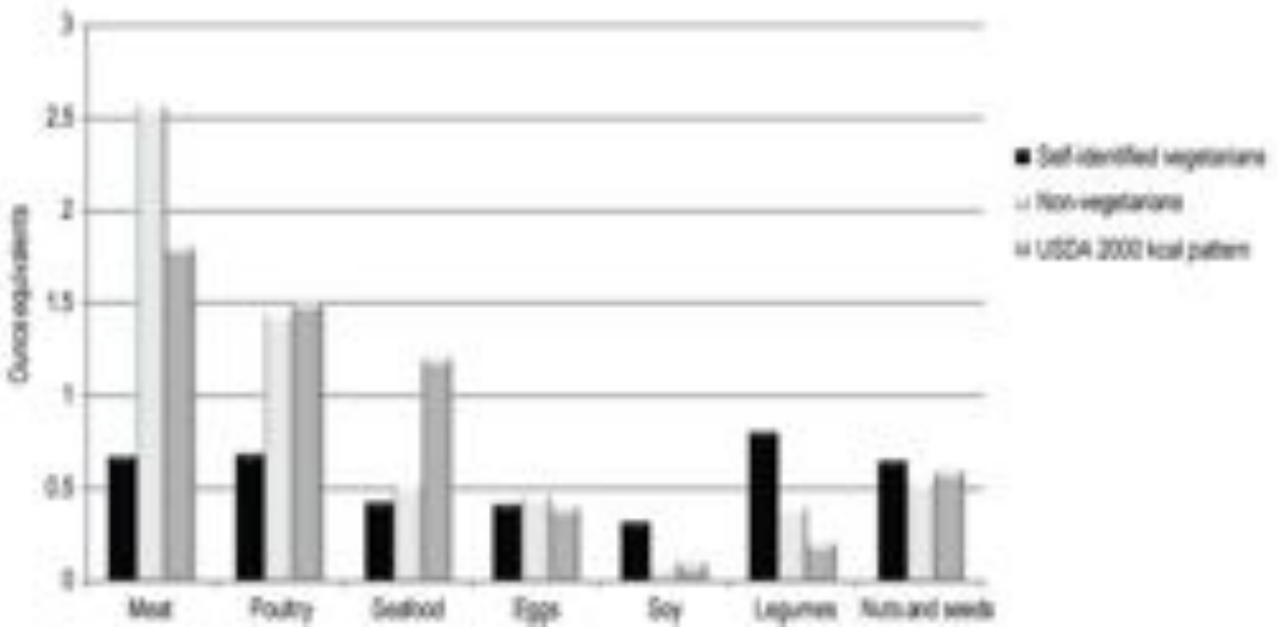
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31

Figure D1.61 Average HEI-2010 component scores for Americans by age group, 2009-10, as a percent of the total possible score for each component.



Source: HEI scores for Americans by age group, What We Eat in America, NHANES 2009-10 Appendix E2.x. Average Healthy Eating Index-2010 Scores for Americans ages 2 years and older

Figure D1.62 Intake from Protein Foods subgroups by self-identified vegetarians in comparison to non-vegetarian and to amounts in USDA Food Pattern at 2000 calories.



Source: Juan, WY, S. Yamini, P. Britten (2014) Food intake patterns of self-identified vegetarians among the U.S. population, 2007-2010. 38th Nutrient Data Bank Conference, May 2014 http://www.nutrientdataconf.org/PastConf/NDBC38/NNDC38_PosterAbstracts.pdf

Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes

INTRODUCTION

A healthy diet is a pillar of well-being throughout the lifespan. It promotes the achievement of healthy pregnancy outcomes; supports normal growth, development and aging; helps maintain healthful body weight; reduces chronic disease risks; and promotes overall health and well-being. Previous Dietary Guidelines Advisory Committees focused on examining specific foods, nutrients, and dietary components and their relationships to health outcomes. In its review, however, the 2010 DGAC noted that it is often not possible to separate the effects of individual nutrients and foods, and that the totality of diet—the combinations and quantities in which foods and nutrients are consumed—may have synergistic and cumulative effects on health and disease.¹ This approach has been adopted by others as well (e.g. American Heart Association, American College of Cardiology and the National Cancer Institute) and is being used by the 2015 DGAC. The 2010 Committee acknowledged the importance of dietary patterns and recommended additional research in this area. After the release of the *2010 Dietary Guidelines for Americans*, the USDA Nutrition Evidence Library (NEL) completed a systematic review project examining the relationships between dietary patterns and several health outcomes, including cardiovascular disease (CVD), body weight and type 2 diabetes.² Their report has been used by the 2015 DGAC.

As also noted in the *2010 Dietary Guidelines for Americans*, individuals can achieve a healthy diet in multiple ways and preferably with a wide variety of foods and beverages. Optimal nutrition can be attained with many dietary patterns and a single dietary pattern approach or prescription is unnecessary. Indeed, for long-term maintenance, a dietary pattern to support optimal nutrition and health should be based on the biological and medical needs as well as preferences of the individual.

Dietary patterns are defined as the quantities, proportions, variety or combinations of different foods and beverages in diets, and the frequency with which they are habitually consumed. Americans consume many habitual dietary patterns, rather than a “typical American pattern,” which reflect their life experiences and wide-ranging personal, socio-cultural and other environmental influences. The nutritional quality of a dietary pattern can be determined by assessing the nutrient content of its constituent foods and beverages and comparing these characteristics to age- and sex-specific nutrient requirements and standards for nutrient adequacy, as shown in *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends* for the USDA Food Patterns, including the “Healthy U.S.-style Pattern,” the “Healthy Mediterranean-style Pattern,” and the “Healthy Vegetarian Pattern.” Understanding the array of dietary patterns in a population and their nutrient quality allows a more complete

39 characterization of individual eating behaviors and enables their examination in relationship with
 40 diverse health outcomes. For these reasons, the DGAC focused on considering the evidence for
 41 overall dietary patterns in addition to key foods and nutrients. A major goal was to describe the
 42 common characteristics of a healthy diet, which informed and is complementary to the
 43 quantitative description of dietary patterns provided in *Part D. Chapter 1: Food and Nutrient*
 44 *Intakes, and Health: Current Status and Trends*.

45
 46 Dietary patterns can be characterized in three main ways, drawing from Dr. Susan Krebs-Smith's
 47 presentation to the DGAC during the second public meeting (available at
 48 www.DietaryGuidelines.gov). The first is by the use of an a priori index that is based on a set of
 49 dietary recommendations for a healthy dietary pattern as a result of scientific consensus or
 50 proposed by investigators using an evidence-based approach. An individual's index/score is
 51 derived by comparing and quantifying their adherence to the criterion food and/or nutrient
 52 component of the index and then summed up over all components. A population's average mean
 53 and individual component scores can be similarly determined. Examples of dietary quality scores
 54 include: the Healthy Eating Index (HEI)-2005 and 2010,³ the Alternate HEI (AHEI) and updated
 55 AHEI-2010,⁴ the Recommended Food Score (RFS),⁵ the Dietary Approaches to Stop
 56 Hypertension (DASH) score,⁶ the Mediterranean Diet Score (MDS),⁷ and the Alternate
 57 Mediterranean Diet Score (aMed).⁸

58
 59 The second method of dietary pattern assessment is through data-driven approaches, such as
 60 cluster analysis (which addresses the question, "Using the self-reported food and beverage intake
 61 data are there groups of people with distinct (non-overlapping) dietary patterns?") and factor
 62 analysis (which addresses the question, "Which components of the diet track together to explain
 63 variations in food or beverage intake across diet patterns?"). These data-driven approaches are
 64 outcome-independent. That is, the relationships between the dietary patterns and intermediate or
 65 longer-term health outcomes are examined once the patterns themselves are defined. Other data-
 66 driven approaches are outcome-dependent, such as reduced rank regression (which addresses the
 67 question, "What combination of foods explains the most variation in one or more intermediate
 68 health markers?").

69
 70 The third method examines individuals' food and beverage intake preferences as they are
 71 commonly defined by foods included or eliminated. In cohort studies, this pattern is usually
 72 based upon qualitative self-reported behaviors rather than detailed questionnaires. Vegetarianism
 73 and its various forms (e.g., ovo-lacto vegetarianism) are examples of this type of dietary pattern.

74
 75 The dietary patterns approach has a number of major strengths. The method captures the
 76 relationship between the overall diet and its constituent foods, beverages and nutrients in
 77 relationship to outcomes of interest and quality, thereby overcoming the collinearity among
 78 single foods and nutrients. In so doing, it considers the inherent interactions between foods and

79 nutrients in promoting health or increasing disease risk. Because foods are consumed in
80 combinations, it is difficult, if not impossible, to determine their separate effects on health.
81 Relationships or effects attributed to a particular food or nutrient may be accurate or reflect those
82 of other dietary components acting in synergy. The dietary pattern approach has advanced
83 nutrition research by capturing overall food consumption behaviors and its quality in relationship
84 to health.

85

86 Despite these considerable strengths, however, the approach has several limitations that are
87 important to consider. First, the dietary assessment instruments used to define the dietary
88 patterns (e.g., food frequency questionnaires [FFQ] and 24-hour or multi-day dietary recalls or
89 records) are based upon self-report and may introduce levels of report bias that can attenuate
90 diet-health relationships. The FFQ has been evaluated as a valid and reliable measure of usual
91 food and nutrient intake. However, the extent to which data from FFQs are valid measures of
92 dietary patterns is not well established. Second, dietary patterns are not uniformly defined by
93 investigators and vary substantially from one study to the next even though studies may use the
94 same nomenclature. This may hamper cross-study comparisons and limits reproducibility. Third,
95 scoring algorithms used to evaluate dietary pattern adherence may differ and affect the results of
96 studies examining specific health outcomes. Fourth, data-driven methods may not derive
97 comparable patterns in different populations because these patterns may be population specific.
98 Lastly, dietary patterns do not assess the frequency of meal and snack consumption, specific
99 combinations of foods consumed together, and aspects of food purchase and preparation, all of
100 which may influence the overall dietary pattern.

101

102 Another challenge to examining dietary patterns is that randomized dietary intervention studies
103 have used different approaches for ensuring that subjects comply with the intervention diet when
104 testing their relationships with health outcomes. For example, randomized controlled trials
105 (RCTs), such as Prevencion con Dieta Mediterranean (PREDIMED), coached participants to
106 follow a dietary pattern and provided them with key foods (e.g., olive oil or nuts) to facilitate
107 adherence. In contrast, feeding studies (another form of intervention study), such as those
108 conducted in the DASH and the Optimal Macronutrient Intake Trial for Heart Health
109 (OmniHeart), provided all food to be consumed to each participant. These study designs across
110 randomized trials and feeding studies provide strong evidence for the benefits and risks of
111 particular dietary patterns because a prescribed intervention allows relatively precise definition
112 of dietary exposures, and randomization helps ensure that any potential confounding variables
113 are randomly distributed between study arms. However, some trials (i.e. DASH, OmniHeart) are
114 necessarily restricted to testing a dietary pattern's effect on an intermediate outcome or a
115 surrogate endpoint, such as blood lipids, because of the complexities involved in maintaining
116 dietary compliance over long study duration. Additionally, the feeding trials fail to represent
117 what happens in real world situations. Thus, well-conducted observational cohort studies provide
118 an important evidentiary complement to RCTs because they enable the study of hard endpoints

119 for disease in addition to intermediate outcomes and often provide a wider range of exposures for
120 study.

121
122 Dietary patterns and their food and nutrient characteristics are at the core of the conceptual
123 model that has guided the DGAC's work (see *Part B. Chapter 2: 2015 DGAC Themes and*
124 *Recommendations: Integrating the Evidence*), and the relationship of dietary patterns to health
125 outcomes is the centerpiece of this chapter. The Committee considered evidence about the
126 relationship of diet with several health outcomes that are listed as major public health outcomes
127 of concern in *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and*
128 *Trends*. Several of these outcomes—CVD, overweight and obesity, type 2 diabetes, congenital
129 anomalies, and bone health—also were addressed by the 2010 DGAC. Others—cancers (lung,
130 colon, prostate and breast) and neurological and psychological illness—while previously
131 addressed, are considered here in more depth and represent an expanded list of health outcomes
132 for which there is growing evidence of a diet-disease relationship. The 2015 Committee was not
133 able to consider the relationship between dietary patterns during the peri- and prenatal period and
134 pregnancy outcomes (e.g., birth weight, preterm birth, pregnancy complications) or other cancer
135 outcomes, such as total cancer mortality or gynecological, pancreatic, and gastric-esophageal
136 cancers due to time limitations and limited work done in these areas involving dietary patterns.
137 However, it is important to note that recently the NIH-AARP Diet and Health Study (n =
138 492,823) conducted in the United States demonstrated that high adherence on several indices (the
139 HEI-2010, the AHEI-2010, the aMED, and DASH) was associated with lower risk of overall
140 CVD and cancer mortality.⁹ The authors concluded that this finding provides further credence
141 for using the dietary pattern approach, indicating that multiple dietary indices reflecting core
142 tenets of a healthy diet may lower the risk of mortality outcomes.⁹

143
144 Over the course of the DGAC's review, when strong or moderate evidence related to dietary
145 patterns and a particular health outcome was available, the Committee focused its discussion on
146 dietary patterns and, as possible, highlighted the most consistent common food and nutrient
147 characteristics identified in the dietary patterns literature. When only limited or insufficient
148 evidence related to dietary patterns and a particular health outcome was available (as in the case
149 of congenital anomalies and neurological and psychological illnesses), the Committee
150 summarized these findings and also provided a brief summary of existing evidence on specific
151 foods and/or nutrients and selected health outcomes.

152
153 In addition to its work on dietary patterns, the DGAC considered conducting an evidence review
154 on the relationship between the role of the microbiome and various health outcomes. This novel
155 area of research has generated considerable interest in the scientific community and the lay
156 public. Investigators are examining the diversity of organisms (i.e., microbes) that inhabit
157 different parts of the body such as the gut, mouth, skin, and vagina, and are attempting to
158 understand how the microbial communities are influenced by diet, environment, host genetics

159 and other microbes, as well as their association with various health outcomes. The DGAC
160 conducted an exploratory search but did not find sufficient evidence to address this question in
161 the 2015 report. However, the Committee considers the microbiome to be an emerging topic of
162 potential importance to future DGACs.

163

164 **LIST OF QUESTIONS**

165 **Dietary Patterns and Cardiovascular Disease**

166 1. What is the relationship between dietary patterns and risk of cardiovascular disease?

167

168 **Dietary Patterns and Body Weight**

169 2. What is the relationship between dietary patterns and measures of body weight or obesity?

170

171 **Dietary Patterns and Type 2 Diabetes**

172 3. What is the relationship between dietary patterns and risk of type 2 diabetes?

173

174 **Dietary Patterns and Cancer**

175 4. What is the relationship between dietary patterns and risk of cancer?

176

177 **Dietary Patterns and Congenital Anomalies**

178 5. What is the relationship between dietary patterns and risk of congenital anomalies?

179

180 **Dietary Patterns and Neurological and Psychological Illnesses**

181 6. What is the relationship between dietary patterns and risk of neurological and psychological
182 illnesses?

183

184 **Dietary Patterns and Bone Health**

185 7. What is the relationship between dietary patterns and bone health?

186

187 **METHODOLOGY**

188 For the first time, the 2015 DGAC included a chapter focusing solely on the relationship between
189 dietary patterns and health outcomes. Although the 2010 DGAC considered some research on
190 certain dietary patterns and specific health outcomes, notably body weight, they did not complete

191 NEL systematic reviews on this research. The 2015 DGAC began by acknowledging a desire to
192 continue and expand on the total diet approach initiated by the 2010 DGAC. They then identified
193 outcomes of public health concern on which to focus their reviews.
194

195 For the purposes of the 2015 DGAC, dietary patterns were defined as the quantities, proportions,
196 variety or combinations of different foods and beverages in diets, and the frequency with which
197 they are habitually consumed. Because the purpose of the Dietary Guidelines is to develop food-
198 based recommendations to promote health and reduce risk of diet-related disease, one of the key
199 aspects of the research that the DGAC considered was a description of the foods and beverages
200 consumed by participants in the studies that the Committee reviewed. This was particularly
201 important for the NEL systematic reviews, for which a description of foods and beverages was a
202 key criterion for inclusion. Data on nutrients were not required for inclusion, but were considered
203 when provided as part of the dietary pattern description.
204

205 Self-reported food and beverage intake was typically assessed using a qualitative or semi-
206 quantitative food intake questionnaire (i.e., FFQ). However, some studies used other methods,
207 such as 24-hour recalls. When reviewing the evidence, the Committee attempted to adhere to the
208 language used by the study authors in describing food groupings. There was variability across
209 the food groupings, and this was particularly apparent in the meat group; for example, “total
210 meat” may have been defined as “meat, sausage, fish, and eggs,” “red meat, processed meat, and
211 poultry,” or various other combinations of meat. Similarly, “vegetables” seemed to most often
212 exclude potatoes, but some studies included potatoes, yet they rarely provided information on
213 how the potatoes were consumed (e.g., fried versus baked). When reported in the studies, the
214 Committee considered these definitions in their review.
215

216 Because of the variability in dietary patterns methodology and food groupings reported, the
217 Committee focused on providing a qualitative description of healthy dietary patterns.
218 Additionally, as most studies reported intake in relative terms (e.g., comparing the first and fifth
219 quintiles or across tertiles), the Committee has presented its conclusions with relative
220 terminology (e.g., “higher” and “lower” in a certain component). Quantitative information on
221 dietary patterns is provided in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health:
222 Current Status and Trends*** as part of the Dietary Patterns Composition section.
223

224 A number of studies in the scientific literature describe diets based on macronutrient proportion
225 or test only a specific food group or nutrient in the diet. For example, a low-carbohydrate diet fits
226 this description and has been of public interest. The DGAC reviewed the body of evidence
227 related to this type of diet as part of Question 2. Additionally, the Committee examined the
228 results of exploratory searches on low-carbohydrate diets (defined as less than 45 percent of
229 calories from carbohydrate) and all of the health outcomes considered in this chapter published
230 since 2000. Overall, it appears that only limited evidence is available to address the relationship

231 between low-carbohydrate diets and health, particularly evidence derived from U.S.-based
 232 populations. The most evidence available focuses on low-carbohydrate diets and body weight.
 233 The 2010 DGAC examined the relationship between macronutrient proportion and various body
 234 weight outcomes, concluding that:

235 *“1) There is strong and consistent evidence that when calorie intake is controlled,*
 236 *macronutrient proportion of the diet is not related to losing weight; 2) A moderate body of*
 237 *evidence provides no data to suggest that any one macronutrient is more effective than any*
 238 *other for avoiding weight re-gain in weight reduced persons; 3) A moderate body of evidence*
 239 *demonstrates that diets with less than 45% of calories as carbohydrates are not more*
 240 *successful for long-term weight loss (12 months). There is also some evidence that they may*
 241 *be less safe. In shorter-term studies, low-calorie, high-protein diets may result in greater*
 242 *weight loss, but these differences are not sustained over time; and 4) A moderate amount of*
 243 *evidence demonstrates that intake of dietary patterns with less than 45% calories from*
 244 *carbohydrate or more than 35% calories from protein are not more effective than other diets*
 245 *for weight loss or weight maintenance, are difficult to maintain over the long term, and may*
 246 *be less safe.”*

247 The published literature since that review does not provide sufficient evidence to change these
 248 conclusions. Thus, in summary, although studies that examine macronutrient proportion or that
 249 test only a specific food group or nutrient are important, they answer different questions related
 250 to diet and health than those proposed by the DGAC. In addition, these studies generally did not
 251 meet the DGAC’s definition of a dietary pattern study unless a full description of the dietary
 252 pattern consumed was provided and appropriate methods were used to adjust for the confounding
 253 of foods and nutrients.

254
 255 Questions 1, 2, and 3 were answered using existing reports, systematic reviews, and meta-
 256 analyses. All three of these questions were addressed in the NEL Dietary Patterns Systematic
 257 Review Project. This project was supported by USDA’s Center for Nutrition Policy and
 258 Promotion and was informed by a Technical Expert Collaborative of experts in dietary patterns
 259 research.² Additionally, the DGAC reviewed reports from systematic reviews recently conducted
 260 by the National Heart, Lung, and Blood Institute (NHLBI) that included dietary patterns
 261 research. For Question 1, the DGAC used the NHLBI *Lifestyle Interventions to Reduce*
 262 *Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group*¹⁰ and the
 263 associated American Heart Association (AHA)/ American College of Cardiology (ACC)
 264 *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*.¹¹ For Question 2, the DGAC
 265 used the NHLBI *Managing Overweight and Obesity in Adults: Systematic Evidence Review from*
 266 *the Obesity Expert Panel*¹² and the associated AHA/ACC/The Obesity Society (TOS) *Guideline*
 267 *for the Management of Overweight and Obesity in Adults*.¹³ For all three questions, in an attempt
 268 to capture new research published since the searches for these systematic reviews were
 269 completed, the Committee considered existing systematic reviews and meta-analyses published
 270 in peer-reviewed journals since 2008. The existing systematic reviews and meta-analyses

271 considered by the DGAC had to meet the general inclusion criteria of the DGAC, and were
272 required to consider dietary patterns and the outcomes of interest. A description of the process
273 the DGAC used to answer existing report questions is provided in *Part C: Methodology*. The
274 DGAC followed this approach, including consideration of reference overlap, for all three
275 questions. For more information on the existing reports, systematic reviews, and meta-analyses
276 considered by the DGAC, the reader is encouraged to review the original sources, which are
277 referenced within each evidence review.

278
279 Questions 4, 5, 6, and 7 were answered using NEL systematic reviews. A description of the NEL
280 process is provided in *Part C: Methodology*. All reviews were conducted in accordance with
281 NEL methodology, and the DGAC made all substantive decisions required throughout the
282 process to ensure that the most complete and relevant body of evidence was identified and
283 evaluated to answer each question. All steps in the process were documented to ensure
284 transparency and reproducibility. Specific information about individual systematic reviews can
285 be found at www.NEL.gov, including the search strategy, inclusion and exclusion criteria, a
286 complete list of included and excluded articles, and a detailed write-up describing the included
287 studies and the body of evidence. A link for each question is provided following each evidence
288 review.

289
290 Introductory sections were written for Questions 4, 5, 6, and 7 because the conclusion statements
291 for these questions were graded limited or insufficient. The purpose of the introduction was to
292 provide a brief description of the current evidence available related to foods and nutrients and the
293 health outcome of interest. However, this evidence was not considered in developing the dietary
294 pattern conclusion statements. During the course of the dietary pattern reviews, the DGAC chose
295 to highlight particular components of the diet, which are discussed further in *Part D. Chapter 6:*
296 *Cross-Cutting Topics of Public Health Importance*.

297
298 **Question 1: What is the relationship between dietary patterns and risk of**
299 **cardiovascular disease?**

300 **Source of evidence:** Existing reports

301

302 **Conclusion**

303 The DGAC concurs with the conclusions of the NEL Dietary Patterns Systematic Review Project
304 and AHA/ACC *Guideline on Lifestyle Management to Reduce Cardiovascular Risk* that strong
305 and consistent evidence demonstrates that dietary patterns associated with decreased risk of CVD
306 are characterized by higher consumption of vegetables, fruits, whole grains, low-fat dairy, and
307 seafood, and lower consumption of red and processed meat, and lower intakes of refined grains,
308 and sugar-sweetened foods and beverages relative to less healthy patterns. Regular consumption
309 of nuts and legumes and moderate consumption of alcohol also are shown to be components of a

310 beneficial dietary pattern in most studies. Randomized dietary intervention studies have
 311 demonstrated that healthy dietary patterns exert clinically meaningful impact on cardiovascular
 312 risk factors, including blood lipids and blood pressure. Additionally, research that includes
 313 specific nutrients in their description of dietary patterns indicate that patterns that are lower in
 314 saturated fat, cholesterol, and sodium and richer in fiber, potassium, and unsaturated fats are
 315 beneficial for reducing cardiovascular disease risk. **DGAC Grade: Strong**

316

317 **Implications**

318 Individuals are encouraged to consume dietary patterns that emphasize vegetables, fruits, whole
 319 grains, legumes, and nuts; include low-fat dairy products and seafood; limit sodium, saturated
 320 fat, refined grains, and sugar-sweetened foods and beverages; and are lower in red and processed
 321 meats. Multiple dietary patterns can achieve these food and nutrient patterns and are beneficial
 322 for cardiovascular health, and they should be tailored to individuals' biological needs and
 323 cultural as well as individual food preferences. The Committee recommends the development
 324 and implementation of programs and services at the individual and population levels that
 325 facilitate the improvement in eating behaviors consistent with the above dietary patterns.

326

327 **Review of the Evidence**

328 The DGAC examined research compiled in the NEL Dietary Patterns Systematic Review
 329 Project, which included 55 articles summarizing evidence from 52 prospective cohort studies and
 330 7 RCTs, and the 2013 AHA/ACC Lifestyle Guideline and associated NHLBI Lifestyle Report,
 331 which included primarily RCTs. The Committee drew additional evidence and effect size
 332 estimates from six published systematic reviews/meta-analyses published since 2008 that
 333 included one or more studies not covered in the NEL or NHLBI Lifestyle reports.¹⁴⁻¹⁹ In total,
 334 142 articles were considered in these reports, of which 35 were included in two or more reviews.
 335 Little evidence on the contribution of dietary patterns to CVD risk factors in the pediatric
 336 populations was available, and that which was published was not systematically reviewed.

337

338 Most evidence examining hard disease endpoints comes from large, prospective cohort studies in
 339 adults using a priori scores to rank individuals with respect to adherence to dietary patterns of
 340 interest. Though the observational design allows the necessary duration of follow-up to observe
 341 CVD endpoints, comparison across studies was difficult because of different methods for
 342 deriving scores and different versions of scores measuring adherence to the same dietary pattern.
 343 In the Mediterranean dietary indices and the AHEI scores, moderate alcohol was included as a
 344 “positive” component (associated with potential benefits). Red and processed meats were
 345 “negative” (potentially detrimental) components in the Mediterranean scores, AHEI scores, and
 346 DASH. Certain scores also included sugars or sugar-sweetened beverages as negative
 347 components. Poultry was considered as a positive component in the original AHEI. Total high-
 348 fat dairy was a negative component in the Mediterranean diet scores, but dairy was a positive

349 component when meeting recommended intakes for the HEI-2005, and low-fat dairy was
350 positive in the DASH scores. As the NEL systematic review points out, several components of
351 scores associated with decreased CVD risk recurred in multiple dietary patterns and were
352 associated as part of scores and as individual components with reduced CVD risk. These
353 included consumption of vegetables, fruits, whole grains, nuts, legumes, unsaturated fats, and
354 fish.

355
356 The NHLBI Lifestyle Report summarized the evidence from two RCTs of the DASH dietary
357 pattern and two trials testing DASH variations with differing levels of sodium or macronutrients.
358 The diet provided to participants in standard DASH intervention trials was high in vegetables,
359 fruits, low-fat dairy products, whole grains, poultry, fish, and nuts. It also was low in sweets,
360 sugar-sweetened beverages, and reduced in (or lower in) red and processed meats. The DASH
361 dietary pattern is high in fiber and potassium and low in sodium, saturated fat, total fat, and
362 cholesterol. It is rich in potassium, magnesium, and calcium, as well as protein and fiber.

363
364 In contrast to the patterns described above, vegetarian diets were defined by what they excluded.
365 Variations included: vegan (no meat, fish, eggs, or dairy); lacto-ovo vegetarian (includes eggs
366 and dairy, but no fish or meat), and pesco-vegetarian (includes fish, but no meat) diets. The
367 content of these diets varied substantially, though they tended to emphasize plant based foods,
368 especially fruits and vegetables, legumes, nuts, and whole grains.

369 ***Dietary Patterns and Blood Pressure (BP)***

371 **DASH or DASH-style Dietary Patterns**

372 The NEL systematic review and AHA/ACC Lifestyle Guideline conclude that strong and
373 consistent evidence from RCTs demonstrates that compared to a dietary pattern that is relatively
374 high in saturated fat and sodium and low in vegetables and fruits, the DASH-style dietary pattern
375 reduced BP by approximately 6/3 mmHg (systolic blood pressure/diastolic blood pressure)
376 across subgroups defined by sex, race, age, and hypertension status. The DASH trial provided all
377 food to participants for 8 weeks. Fat intake was relatively low at 26 percent of energy (7 percent
378 each monounsaturated and saturated, 10 percent polyunsaturated), compared to 36 percent in the
379 control group. Carbohydrates accounted for 57 percent of energy and protein for 18 percent.
380 Sodium was stable at 3,000 mg/day and body weight did not change. Variations of the DASH
381 diet also lowered blood pressure: in the OmniHeart Trial, compared to the standard DASH,
382 replacing 10 percent of calories from carbohydrate with either the same calorie content of protein
383 or with unsaturated fat (8 percent MUFA and 2 percent PUFA) lowered systolic BP by 1 mmHg.
384 Among adults with BP 140–159/90–95 mmHg, these substitutions lowered systolic BP by 3
385 mmHg relative to standard DASH.^{2, 11}

386
387 Observational evidence summarized in the NEL report included one cohort showing that
388 increased DASH score was associated with small, but decreased levels of systolic and diastolic

389 BP over time;²⁰ two others cohorts showed no relationship between DASH scores and risk of
 390 hypertension.^{21, 22}

391

392 **Mediterranean-Style Dietary Patterns**

393 Several RCTs provide limited to moderate evidence on the benefits of a Mediterranean-style diet
 394 for reducing blood pressure. The AHA/ACC Lifestyle Guideline conclude that consuming a
 395 Mediterranean dietary pattern instead of a lower-fat dietary pattern had beneficial effects on
 396 blood pressure. The NHLBI Lifestyle Report reviewed two RCTs of free-living middle-aged or
 397 older adults (with type 2 diabetes or at least three CVD risk factors) in which a Mediterranean
 398 diet intervention reduced BP by 6–7/2–3 mmHg.^{23, 24} The report also reviewed one observational
 399 study of healthy younger adults. Higher adherence to a Mediterranean-style diet, as measured
 400 through a Mediterranean score, was associated with a decrease in BP of 2–3/1–2 mmHg.²⁵

401

402 **Vegetarian Dietary Patterns**

403 Evidence for the blood pressure benefits of vegetarian dietary patterns is more limited, but
 404 moderately consistent trends appear to exist. A recent meta-analysis of seven RCTs found that
 405 consumption of vegetarian diets was associated with a reduction in mean systolic blood pressure
 406 (-4.8 mm Hg; 95% CI = -6.6 to -3.1; p<0.01) and diastolic blood pressure (-2.2 mm Hg; 95% CI
 407 = -3.5 to -1.0) compared with the consumption of omnivorous diets.¹⁹ The AHA/ACC Lifestyle
 408 Guideline did not find sufficient evidence to examine vegetarian dietary patterns, and the NEL
 409 systematic review summarized only three studies comparing blood pressure outcomes in lacto-
 410 ovo vegetarian diets versus non-vegetarian diets in which meat and fish were consumed. Of the
 411 two studies, one was a large prospective cohort that found no association with blood pressure,²⁶
 412 and the other was a RCT among individuals with hypertension that demonstrated a decrease in
 413 systolic blood pressure, but not diastolic blood pressure.²⁷ The more recent EPIC-Oxford cohort
 414 found lower systolic, but not diastolic blood pressure compared to the findings of Crowe, 2013.²⁸

415

416 **Other Dietary Patterns**

417 As summarized in the NEL systematic review, adherence to the *2005 Dietary Guidelines for*
 418 *Americans* was related to lower blood pressure in one study of healthy young adults. Zamora et
 419 al reported 20-year findings from the CARDIA study including 4,381 Black and White young
 420 adults.²⁹ Participants in the highest (vs. lowest) quartile of adherence to the 2005 Dietary
 421 Guidelines had significantly less increase in systolic and diastolic blood pressure over time.

422

423 ***Dietary Patterns and Blood Lipids***

424 **DASH or DASH-style Dietary Patterns**

425 As reviewed in the NHLBI Lifestyle Report, RCTs of the DASH diet show favorable effects on
 426 low-density lipoprotein cholesterol (LDL-C) and total cholesterol: high-density lipoprotein
 427 cholesterol (total-C: HDL-C) ratio, and no effect on triglycerides (TG). Benefits were seen with a

428 variety of different macronutrient compositions, though they were enhanced when some
429 carbohydrates in the standard DASH pattern were replaced with protein or unsaturated fat. In the
430 standard DASH, when food was supplied to adults with a total cholesterol level of less than 260
431 mg/dL and LDL-C less than 160 mg/dL, and body weight was kept stable, the DASH dietary
432 pattern compared to the control diet decreased LDL-C by 11 mg/dL, decreased HDL-C by 4
433 mg/dL, and had no effect on TG. The OmniHeart trial tested the DASH dietary pattern with
434 different macronutrient compositions among adults with average baseline LDL-C 130 mg/dL,
435 HDL-C 50 mg/dL, and TG 100 mg/dL. Modifying the DASH diet by replacing 10 percent of
436 calories from carbohydrate with 10 percent of calories from protein decreased LDL-C by 3
437 mg/dL, decreased HDL-C by 1 mg/dL, and decreased TG by 16 mg/dL compared to the DASH
438 dietary pattern. Replacing 10 percent of calories from carbohydrate with 10 percent of calories
439 from unsaturated fat (8 percent MUFA and 2 percent PUFA) decreased LDL-C similarly,
440 increased HDL-C by 1 mg/dL, and decreased TG by 10 mg/dL compared to the DASH dietary
441 pattern.¹¹

442

443 **Mediterranean-style Dietary Patterns**

444 As with blood pressure, few trials have evaluated the effects of Mediterranean dietary patterns on
445 blood lipids. According to the AHA/ACC Lifestyle Guideline, consuming a Mediterranean-style
446 diet (compared to minimal or no dietary advice) resulted in no consistent effect on plasma LDL-
447 C, HDL-C, and TG. In part, this was due to substantial differences in dietary interventions
448 conducted among free-living middle aged or older adults with or without CVD or at high risk for
449 CVD.¹¹ In the PREDIMED trial (reviewed in both the NHLBI Lifestyle and NEL reports), both
450 treatment groups (Mediterranean diet +olive oil or +nuts) had favorable changes in HDL-C,
451 total-C: HDL-C ratio and TG when compared to the control group, which received minimal
452 advice to follow a lower-fat diet.²³ One of the prospective cohort studies reviewed by the NEL
453 showed each one-point increase in alternate Mediterranean diet score assessed in adolescence
454 and early adulthood was associated with a -6.19 (-10.44, -1.55) mg/dL lower total cholesterol in
455 adulthood but no significant effects on HDL-C.³⁰ Of other observational cohorts reviewed, one
456 reported adherence to a Mediterranean diet was associated with favorable changes in HDL-C and
457 TG,³¹ and another found no associations between adherence to a Mediterranean diet and blood
458 lipids.³²

459

460 **Vegetarian Dietary Patterns**

461 The NEL systematic review included three articles on vegetarian patterns that measured blood
462 pressure or blood lipids.²⁶⁻²⁸ One study reported decreased total-C²⁶ and another reported
463 decreased non-HDL-C in vegetarian versus non-vegetarian participants.²⁸

464

465 **Other Dietary Patterns**

466 Of note, adherence to the *2005 Dietary Guidelines for Americans* also was related to higher
467 HDL-C levels in a cohort of Black and White young adults.²⁹

468

469 *Dietary Patterns and Cardiovascular Disease Outcomes*

470 The NHLBI Lifestyle review did not include any trials examining the evidence of particular
471 dietary patterns with CVD outcomes. Overall, the NEL systematic review found that individuals
472 whose diets mirrored the dietary patterns of interest (typically compared with diets having lower
473 scores) was associated with lower CVD incidence and mortality in 14 out of 17 studies. The
474 studies were predominantly observational, but included some trial evidence, and they typically
475 assessed dietary intakes through self-report. The effect sizes varied substantially, with the
476 decrease in risk of CVD ranging from 22 to 59 percent for increased adherence to various
477 Mediterranean-style dietary patterns and from 20 to 44 percent for increased adherence to a U.S.
478 Dietary Guidelines-related pattern (e.g., HEI or AHEI and updates). The majority of studies that
479 assessed coronary heart disease (CHD) incidence or mortality also reported a favorable
480 association between adherence to a healthy dietary pattern and CHD risk. The lower CHD risk
481 ranged from 29 to 61 percent for greater adherence to Mediterranean-style dietary patterns, from
482 24 to 31 percent for greater adherence to a U.S. Dietary Guidelines-related pattern, and from 14
483 to 27 percent for greater adherence to DASH. Similarly, the majority of studies assessing stroke
484 incidence or mortality reported favorable associations, with the lower stroke risk ranging from 13
485 to 53 percent for greater adherence to a Mediterranean-style dietary pattern and from 14 to 60
486 percent for greater adherence to a U.S. Dietary Guidelines-related pattern.²

487

488 *Mediterranean-style Dietary Patterns*

489 To gather additional information on dietary patterns and CVD outcomes, the DGAC consulted
490 two meta-analyses,^{15, 18} which included many of the same observational prospective cohort
491 studies as one another and as the NEL systematic review. These meta-analyses each reported
492 summary estimates across studies as a 10 percent reduction in risk of CVD (fatal or nonfatal
493 clinical CVD event) per 2-increment increase in adherence to the Mediterranean-style diet. The
494 NEL report also included results from the largest Mediterranean diet trial, PREDIMED, which
495 found that a Mediterranean diet (plus extra virgin olive oil or nuts) had favorable effects in high-
496 risk participants compared to the control group who were advised to reduce dietary fat intake. An
497 approximately 30 percent decrease in risk of major CVD events (a composite endpoint including
498 myocardial infarction, stroke, and deaths) was observed and the trial was stopped early for
499 meeting benefit requirements.^{2, 33} According to food questionnaires measuring adherence to the
500 assigned diet by the end of follow-up, the intervention groups had significantly increased
501 consumption of fish and legumes and non-significant reductions in refined grains and red meat
502 from baseline, in addition to increased intake of supplemental foods (olive oil or nuts depending
503 on the intervention arm), compared to the control group.

504

505 *DASH-style Dietary Patterns*

506 A recent meta-analysis¹⁷ of six prospective cohort studies with CVD endpoints assessed DASH-
507 style diet through the Fung et al. method,⁶ which assigns points based on population-specific

508 quintiles of eight DASH dietary pattern components: fruits, vegetables, nuts and legumes, whole
 509 grains, low-fat dairy, sodium, red and processed meats, and sweetened beverages. This meta-
 510 analysis reported that greater adherence to a DASH-style diet significantly reduced CVD
 511 (Relative Risk [RR]=0.80; 95% CI = 0.74 to 0.86), CHD (RR=0.79; 95% CI = 0.71 to 0.88), and
 512 stroke (RR=0.81; 95% CI = 0.72 to 0.92). All of the studies meta-analyzed also were included
 513 the NEL's evidence base for the DASH-style diet.

514

515 **Vegetarian Dietary Patterns**

516 The NEL systematic review concluded that evidence for the effects of vegetarian dietary patterns
 517 on cardiovascular endpoints is limited. Most of this evidence was from prospective cohort
 518 studies; four out of six studies suggested that a vegetarian dietary pattern was associated with
 519 reduced incidence of ischemic heart disease (IHD) or CVD mortality. A meta-analysis of seven
 520 studies related to CVD mortality and vegetarian diet¹⁴ (including two of the studies from the
 521 NEL systematic review) found that mortality from IHD was significantly lower in vegetarians
 522 than in non-vegetarians (RR=0.71; 95% CI = 0.56 to 0.87). The authors estimated a 16 percent
 523 lower mortality from circulatory diseases (RR=0.84; 95% CI = 0.54 to 1.14) and a 12 percent
 524 lower mortality from cerebrovascular disease (RR=0.88; 95% CI = 0.70 to 1.06) in vegetarians
 525 compared to non-vegetarians.

526

527 *For additional details on this body of evidence, visit:* References 2, 10, 11, 14-19 and *Appendix*
 528 *E-2.26*

529

530

531 **DIETARY PATTERNS AND BODY WEIGHT**

532 **Question 2: What is the relationship between dietary patterns and measures of** 533 **body weight or obesity?**

534 **Source of evidence:** Existing reports

535

536 **Conclusion**

537 The DGAC concurs with the 2013 AHA/ACC/TOS *Guideline for the Management of*
 538 *Overweight and Obesity* that strong evidence demonstrates that, preferably as part of a
 539 comprehensive lifestyle intervention carried out by multidisciplinary teams of professionals or
 540 nutrition professionals, overweight and obese adults can achieve weight loss through a variety of
 541 dietary patterns that achieve an energy deficit. Clinically meaningful weight losses that were
 542 achieved ranged from 4 to 12 kg at 6-month follow-up. Thereafter, slow weight regain is
 543 observed, with total weight loss at 1 year of 4 to 10 kg and at 2 years of 3 to 4 kg. However,
 544 some dietary patterns may be more beneficial in the long-term for cardiometabolic health.

545 **DGAC Grade: Strong**

546

547 The DGAC concurs with the NEL Dietary Patterns Systematic Review Project that moderate
 548 evidence indicates dietary patterns that are higher in vegetables, fruits, and whole grains; include
 549 seafood and legumes; are moderate in dairy products (particularly low and non-fat dairy) and
 550 alcohol; lower in meats (including red and processed meats), and low in sugar-sweetened foods
 551 and beverages, and refined grains are associated with favorable outcomes related to healthy body
 552 weight (including lower BMI, waist circumference, or percent body fat) or risk of obesity.
 553 Components of the dietary patterns associated with these favorable outcomes include higher
 554 intakes of unsaturated fats and lower intakes of saturated fats, cholesterol, and sodium. **DGAC**

555 **Grade: Moderate**

556

557 Evidence for children is limited, but studies in the NEL Dietary Patterns Systematic Review
 558 Project and the systematic review focused on this age group by Ambrosini et al.³⁴ suggest that
 559 dietary patterns in childhood or adolescence that are higher in energy-dense and low-fiber foods,
 560 such as sweets, refined grains, and processed meats, as well as sugar-sweetened beverages,
 561 whole milk, fried potatoes, certain fats and oils, and fast foods increase the risk of obesity later
 562 on in life. **DGAC Grade: Limited**

563

564 **Implications**

565 To achieve and maintain a healthy body weight, individuals are encouraged to consume dietary
 566 patterns that are higher in vegetables, fruits, and whole grains; include seafood and legumes; are
 567 moderate in dairy products (with an emphasis on low- and non-fat dairy), and alcohol; and are
 568 lower in meats (including red and processed meats), sugar-sweetened foods and beverages, and
 569 refined grains. During childhood and adolescence, a time period critical for the prevention of
 570 obesity later in life, a dietary pattern similar to that associated with a healthy weight in adults
 571 should be encouraged.

572

573 Among overweight and obese individuals, an energy deficit is necessary to achieve weight loss.
 574 This can be achieved through a variety of evidence-based dietary patterns and should be
 575 approached with comprehensive lifestyle interventions. While it is possible to lose weight on
 576 his/her own, it is more successful if conducted by trained professionals or by referral to a
 577 nutrition professional for individual or group counseling (for more details refer to
 578 *AHA/ACC/TOS Guideline for the Management of Overweight and Obesity*¹³ algorithm Box
 579 11B). Strategies should be based on the individual's preferences and health status and consider
 580 the socio-cultural influences on lifestyle behaviors that relate to long-term behavior maintenance.
 581 These approaches are best complemented with population-based approaches, as mentioned in
 582 **Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change** and **Part D.**
 583 **Chapter 4: Food Environment and Settings**, which will allow all factors influencing lifestyle
 584 behaviors to be addressed as defined in the socio-ecological model.

585

586 **Review of the Evidence**

587 The DGAC considered evidence from the 2013 AHA/ACC/TOS Obesity Guideline and
 588 associated NHLBI Obesity Report, which included only randomized trials,^{12, 13} the NEL Dietary
 589 Patterns Systematic Review Project,² which included 38 studies predominately of prospective
 590 cohort design and a few randomized trials, and two systematic reviews/meta-analyses published
 591 since 2008.^{34, 35} In total, 81 articles were considered in these reports. The published reviews
 592 provided evidence for the pediatric population (included 7 studies of which 2 overlapped with
 593 those in the NEL review) and further evidence for dietary patterns related to the Mediterranean-
 594 style diet and its effect on obesity and weight loss (all randomized trials of which 1 out of the 16
 595 studies overlapped with the NEL review).

596

597 ***Dietary Patterns and the Management of Overweight and Obesity***

598 In the NHLBI Obesity Report, the 12 randomized studies described in summary Table 3.1 of the
 599 report all confirm that to lose weight, a variety of dietary pattern approaches can be used and a
 600 reduction in caloric intake is required. The energy balance equation requires that for weight loss,
 601 one must consume less energy than one expends or expend more energy than one consumes. The
 602 report states that any one of the following methods can be used to reduce food and calorie intake:
 603 prescription of 1,200 to 1,500 kcal/day for women and 1,500 to 1,800 kcal/day for men (kcal
 604 levels are usually adjusted for the individual's body weight); prescription of a 500 kcal/day or
 605 750 kcal/day energy deficit; or prescription of an evidence-based diet that restricts certain food
 606 types (such as high-carbohydrate foods, low-fiber foods, or high-fat foods) in order to create an
 607 energy deficit by reduced food intake.

608

609 For the different dietary approaches (provided either as part of a comprehensive lifestyle change
 610 intervention carried out by a multi-disciplinary team of trained professionals or within nutrition
 611 interventions conducted by nutrition professionals) that the authors of the report evaluated, it is
 612 evident that all prescribed diets that achieved an energy deficit were associated with weight loss.
 613 There was no apparent superiority of one approach when behavioral components were balanced
 614 in the treatment arms. Results indicated that average weight loss is maximal at 6 months with
 615 smaller losses maintained for up to 2 years, while treatment and follow-up taper. Weight loss
 616 achieved by dietary techniques aimed at reducing daily energy intake ranges from 4 to 12 kg at
 617 6-month follow-up. Thereafter, slow weight regain is observed, with total weight loss at 1 year of
 618 4 to 10 kg and at 2 years of 3 to 4 kg. The following dietary approaches are associated with
 619 weight loss if reduction in dietary energy intake is achieved:

620

- 621 • A diet from the European Association for the Study of Diabetes Guidelines, which
 622 focuses on targeting food groups, rather than formal prescribed energy restriction while
 623 still achieving an energy deficit.

- 624 • Higher protein (25 percent of total calories from protein, 30 percent of total calories from
625 fat, 45 percent of total calories from carbohydrate) with provision of foods that realized
626 energy deficit.
- 627 • Higher protein Zone™-type diet (5 meals/day, each with 40 percent of total calories from
628 carbohydrate, 30 percent of total calories from protein, 30 percent of total calories from
629 fat) without formal prescribed energy restriction but realized energy deficit.
- 630 • Lacto-ovo-vegetarian-style diet with prescribed energy restriction.
- 631 • Low-calorie diet with prescribed energy restriction.
- 632 • Low-carbohydrate (initially less than 20 g/day carbohydrate) diet without formal
633 prescribed energy restriction but realized energy deficit.
- 634 • Low-fat (10 percent to 25 percent of total calories from fat) vegan-style diet without
635 formal prescribed energy restriction but realized energy deficit.
- 636 • Low-fat (20 percent of total calories from fat) diet without formal prescribed energy
637 restriction but realized energy deficit.
- 638 • Low-glycemic load diet, either with formal prescribed energy restriction or without
639 formal prescribed energy restriction but with realized energy deficit.
- 640 • Lower fat (≤ 30 percent fat), high dairy (4 servings/day) diets with or without increased
641 fiber and/or low-glycemic index/load foods (low-glycemic load) with prescribed energy
642 restriction.
- 643 • Macronutrient-targeted diets (15 percent or 25 percent of total calories from protein; 20
644 percent or 40 percent of total calories from fat; 35 percent, 45 percent, 55 percent, or 65
645 percent of total calories from carbohydrate) with prescribed energy restriction.
- 646 • Mediterranean-style diet with prescribed energy restriction.
- 647 • Moderate protein (12 percent of total calories from protein, 58 percent of total calories
648 from carbohydrate, 30 percent of total calories from fat) with provision of foods that
649 realized energy deficit.
- 650 • Provision of high-glycemic load or low-glycemic load meals with prescribed energy
651 restriction.
- 652 • The AHA-style Step 1 diet (with prescribed energy restriction of 1,500 to 1,800 kcal/day,
653 <30 percent of total calories from fat, <10 percent of total calories from saturated fat).

654 Although these dietary patterns with an energy deficit will result in weight loss during a 6-
655 months to 2-year period, long-term health implications with certain patterns may be detrimental
656 to cardiometabolic health. These associations have been discussed in the dietary patterns and
657 cardiovascular health section as well as the saturated fat and cardiovascular health section.

658

659 As presented in Table D2.1 at the end of the chapter, the results of the randomized studies
 660 considered in the AHA/ACC/TOS Guideline provide evidence for what works in terms of the
 661 components of a comprehensive lifestyle intervention or nutrition interventions that are needed
 662 to achieve weight loss with the variety of dietary approaches described above.

663

664 ***Dietary Patterns and their Association with Body Weight***

665 A total of 14 studies met the inclusion criteria for the index/score question of the NEL systematic
 666 review and were categorized based on dietary pattern exposure. Two major categories were
 667 identified: (1) studies that examined exposure based on a Mediterranean-designated dietary
 668 pattern and (2) studies that examined exposure based on expert dietary guidelines
 669 recommendations. Taken together, there were six studies on Mediterranean-designated diet
 670 scores,^{23, 31, 32, 36-38} five studies on dietary guidelines-based indices,³⁹⁻⁴³ two studies on
 671 Mediterranean-designated scores and dietary guidelines indices,^{44, 45} and one study that used a
 672 trial-based customized score.⁴⁶ Two of the studies were RCTs of positive quality^{23, 46} and 12
 673 were prospective cohort studies. The studies were carried out between 2006 and 2012.

674

675 The sample sizes for prospective cohort studies ranged from 732 to 373,803 participants, with
 676 follow-up times from 1.5 to 20 years. Ten out of 12 of the prospective cohort studies were
 677 conducted with generally healthy adults with a mean age of 25 to 63 years. Two studies were
 678 conducted with children and adolescents (one with girls).^{39, 40} The two RCTs were conducted in
 679 adults with elevated chronic disease risk: one study with a Mediterranean-designated diet
 680 intervention on older adults at increased CVD risk with more than 90 percent overweight or
 681 obese²³ and one study using an a priori diet intervention on men with pre-existing metabolic
 682 syndrome.⁴⁶ The sample sizes for the RCTs were from 187 to 769 subjects and duration of
 683 follow-up ranged from 3 to 12 months.

684

685 **Mediterranean-style Dietary Pattern**

686 Four out of the six studies evaluating the Mediterranean style dietary pattern were conducted in
 687 Spain.^{23, 32, 36, 37} Of the other two, one study was the European multicenter study that was part of
 688 the EPIC-Physical Activity, Nutrition, Alcohol Consumption, Cessation of Smoking, Eating out
 689 of Home, and Obesity (EPIC-PANACEA) study,³⁸ and one was conducted in the United States.³¹

690

691 Dietary Patterns and Body Weight and Incidence of Overweight and/or Obesity

692 The Prevencion con Dieta Mediterranean (PREDIMED) study tested the effects of a
 693 Mediterranean diet on the primary prevention of cardiovascular disease in a high-risk group of
 694 men and women. Subjects either had type 2 diabetes or three cardiovascular disease risk factors
 695 (such as hypertension or current smoking) and 90 percent were overweight or obese defined as
 696 BMI ≥ 25 kg/m². The PREDIMED trial randomly assigned participants to three interventions: (1)
 697 Mediterranean diet with extra virgin olive oil, (2) Mediterranean diet with mixed nuts, and (3)

698 low-fat diet. At end of 3 months of a 4-year clinical trial, the authors found that the
 699 Mediterranean diet score increased in the two Mediterranean diet groups of the trial and
 700 remained unchanged in the low-fat group. However, no significant changes in body weight and
 701 adiposity occurred within or between groups from baseline to the 3 months. Beunza et al., 2010
 702 reported on a prospective cohort study in Spain, the Seguimiento Universidad de Navarra (SUN)
 703 study.³⁶ Participants with the highest adherence to a Mediterranean dietary pattern, assessed
 704 using the Trichopoulou Mediterranean Diet Score (MDS) were found to have lower average
 705 yearly weight gain, -0.059 kg/y (95% CI = -0.111 to -0.008 kg/y; p for trend = 0.02), than
 706 participants in the lowest adherence group.⁷ However, the MDS was not associated with
 707 incidence of overweight or obesity in participants who were normal weight at baseline. Mendez
 708 et al., 2006 reported on the EPIC-Spain prospective cohort study.³⁷ Adherence to a
 709 Mediterranean diet was assessed using a slight modification of the Trichopoulou MDS, with
 710 exposure categorized in tertiles of low (0-3), medium (4-5), and high (6-8) adherence.
 711 Participants with highest MDS adherence had reduced incidence of obesity when overweight at
 712 baseline; overweight women and men were 27 percent and 29 percent, respectively, less likely to
 713 become obese. High MDS adherence was not associated with incidence of overweight in subjects
 714 who were normal weight at baseline. The EPIC-PANACEA study examined the association
 715 between adherence to the relative Mediterranean dietary pattern (rMDS), prospective weight
 716 change, and the incidence of overweight or obesity. Participants with high rMED adherence
 717 gained less weight in 5 years than did participants with low rMED adherence (-0.16 kg; 95% CI
 718 = -0.24 to -0.07 kg) and had a 10 percent lower odds of becoming overweight or obese (OR =
 719 0.90; 95% CI = 0.82 to 0.96). The contribution of each rMED scoring component also was
 720 assessed and it was found that the association between rMED and weight change was no longer
 721 significant when meat and meat products were not part of the score. Lastly, a meta-analysis of
 722 the odds ratio scores of all 10 European countries showed that a 2-point increase in rMED score
 723 was associated with 3 percent (95% CI = 1 to 5%) lower odds of becoming overweight or obese
 724 over 5 years.

725

726 Dietary Patterns and Waist Circumference

727 Rumawas et al., 2009 conducted a prospective cohort study using a subset of the Framingham
 728 Offspring and Spouse (FOS) study.³¹ Dietary exposure was assessed in quintiles of low to high
 729 adherence to the Mediterranean style dietary pattern score (MSDPS). Participants with a higher
 730 MSDPS had significantly lower waist circumference (p for trend < 0.001). Tortosa et al., 2007
 731 reported on the association of the Mediterranean dietary pattern and metabolic syndrome in the
 732 SUN study conducted in Spain.³² Participants in the highest tertile of adherence to the MDS had
 733 lower waist circumference, -0.05 cm over 6 years (p for trend = 0.038), compared to the lowest
 734 tertile.

735

736 Although some mixed results from prospective studies may be due to differences in the length of
 737 follow up, definition of the Mediterranean dietary pattern and population included, the results of

738 randomized studies indicate a significant reduction in body weight when calories are restricted.
 739 A high quality meta-analysis (AMSTAR rating of 11) on the association of a Mediterranean-
 740 style diet with body weight conducted by Esposito included 16 randomized studies of which
 741 one³² overlapped with the NEL systematic review was included in the DGAC body of evidence
 742 for this question. The meta-analysis included studies conducted in the United States, Italy, Spain,
 743 France, Israel, Greece, Germany, and the Netherlands that lasted from 4 weeks to 24 months
 744 with a total of 3,436 participants. Using a random effects model, participants in the
 745 Mediterranean diet group had significant weight loss (mean difference between Mediterranean
 746 diet and control diet, -1.75 kg; 95% CI = -2.86 to -0.64) and reduction in BMI (mean difference,
 747 -0.57 kg/m²; 95% CI = 0.93 to 0.21 kg/m²) compared to those in the control arm. The effect of
 748 Mediterranean diet on body weight was greater in association with energy restriction (mean
 749 difference, -3.88 kg; 95% CI = -6.54 to -1.21 kg), increased physical activity (-4.01 kg; 95% CI
 750 = -5.79 to -2.23 kg), and follow up longer than 6 months (-2.69 kg; 95% CI = -3.99 to -1.38 kg).
 751 Across all 16 studies, the Mediterranean style dietary pattern did not cause weight gain.

752

753 **Dietary Guidelines-Based Indices**

754 Of the seven studies conducted on dietary guidelines-based indices, three studies were conducted
 755 in the United States with U.S.-based indices.^{39, 41, 43} One study was conducted in Germany with
 756 an index developed in the United States,⁴⁰ and two studies were conducted in France (one used a
 757 French index,⁴² and the other compared six different dietary scores).⁴⁴

758

759 Dietary Patterns and Body Weight and Incidence of Overweight and/or Obesity

760 Gao et al., 2008 reported on a prospective cohort study of White, African American, Hispanic,
 761 and Chinese men and women in the Multi-Ethnic Study of Atherosclerosis (MESA) in the US.
 762 Two versions of the 2005 HEI were used: the original and a modified version that adjusted the
 763 food group components to incorporate levels of caloric need based on sex, age, and activity
 764 level.⁴¹ For the overall population, there was an inverse association between quintiles of each
 765 HEI score and BMI (p<0.001). The risk of obesity in normal weight participants was inversely
 766 associated with HEI scores only for Whites (p<0.05). A comparison of the HEI-1995 and HEI-
 767 2005 scores indicated that beta-coefficients, as predictors of body weight and BMI, were higher
 768 for the HEI-2005 scores in Whites. Zamora et al., 2010 analyzed data from the prospective
 769 cohort study, Coronary Artery Risk Development in Young Adults (CARDIA), conducted in the
 770 United States, to examine the association between diets consistent with the 2005 Dietary
 771 Guidelines and subsequent weight gain in Black and White young adults.⁴³ The Diet Quality
 772 Index (DQI) included 10 components of the 2005 Dietary Guidelines relating to the
 773 consumption of total fat, saturated fat, cholesterol, added sugars, reduced-fat milk, fruit,
 774 vegetables, whole grains, nutrient-dense foods, and limited sodium and alcohol intake. They
 775 found, a 10-point increase in DQI score was associated with a 10 percent lower risk of gaining
 776 10 kg in normal-weight Whites. However, the same magnitude increase in score was associated
 777 with a 15 percent higher risk in obese Blacks (p<0.001). Kesse-Guyot et al., 2009 conducted a

778 prospective cohort study in France to examine the association between adherence to a dietary
 779 score based on the French 2001 nutritional guidelines (Programme National Nutrition Sante'
 780 guidelines score (PNNS-GS) and changes in body weight, body fat distribution, and obesity
 781 risk.⁴² The PNNS-GS includes 12 nutritional components: fruit and vegetables, starchy foods,
 782 whole grains, dairy products, meat, seafood, added fat, vegetable fat, sweets, water and soda,
 783 alcohol, and salt. The last PNNS-GS component is physical activity. In fully adjusted models, an
 784 increase of one PNNS-GS unit was associated with lower weight gain ($p=0.004$), and lower BMI
 785 gain ($p=0.002$). An increase of 1 PNNS-GS unit was associated with a lower probability of
 786 becoming overweight (including obese) (OR = 0.93; 95% CI = 0.88 to 0.99). Similarly, an
 787 increase of 1 PNNS-GS unit was associated with a lower probability of becoming obese (OR =
 788 0.89; 95% CI = 0.80 to 0.99).

789
 790 Two studies were conducted in children. Cheng et al., 2010 analyzed data from a prospective
 791 cohort study conducted in Germany, the Dortmund Nutritional and Anthropometric
 792 Longitudinally Designed (DONALD) study, to examine whether the diet quality of healthy
 793 children before puberty was associated with body composition at onset of puberty.⁴⁰ Adherence
 794 to a diet pattern was assessed by the Revised Children's Diet Quality Index (RC-DQI) which was
 795 based on the Dietary Guidelines for Americans. In this study, a higher dietary quality was
 796 associated with a higher energy intake, and children with a lower diet quality had lower BMI and
 797 Fat Mass Index (FMI) Z-scores at baseline ($p<0.01$) but not at onset of puberty. Berz et al., 2011
 798 reported on a prospective cohort study to assess the effects of the DASH eating pattern on BMI
 799 in adolescent females over a 10-year period.³⁹ Only seven out of the 10 original components of
 800 the DASH score were used; the three excluded were added sugars, discretionary fats and oils,
 801 and alcohol. Overall, girls in the highest vs. lowest quintile of DASH score had an adjusted mean
 802 BMI of 24.4 vs. 26.3 kg/m² ($p<0.05$).

803

804 Dietary Patterns and Waist Circumference

805 Gao et al, found, for the overall population in the MESA study, an inverse association between
 806 quintiles of each HEI score and waist circumference (WC) ($p<0.001$).⁴¹ The study by Kesse-
 807 Guyot conducted in France showed, in fully adjusted models, an increase of one PNNS-GS unit
 808 was associated with lower waist circumference gain ($p=0.01$) and lower waist-to-hip ratio gain
 809 ($p=0.02$).⁴²

810

811 **Other Indices**

812 Jacobs et al., 2009 conducted an RCT in Norway, the Oslo Diet and Exercise Study, to examine
 813 the effect of changes in diet patterns on body weight and other outcomes among men who met
 814 the criteria for the metabolic syndrome ($n=187$ men).⁴⁶ Study participants were randomly
 815 assigned to: (1) the diet protocol, (2) the exercise protocol, (3) the diet + exercise protocol, or (4)
 816 the control protocol. The trial duration was 12 months. The authors created their own diet score
 817 to assess adherence to the intervention. The score was based on summing the participants

818 ranking of intake (across tertiles) of 35 food groups that, based on the literature, had a beneficial
 819 neutral or detrimental effect on health. A higher score reflected greater adherence to the diet
 820 intervention. Over the course of the intervention, the diet score increased by 2 points (SD \pm 5.5)
 821 in both diet groups, with a decrease of an equivalent amount in the exercise and control groups.
 822 A 10-point change in the diet score during the intervention period was associated with a 3.5 kg
 823 decrease in weight, a 2.8 cm decrease in waist circumference and 1.3 percent decrease in percent
 824 body fat (all significant at $p < 0.0001$).

825

826 **Studies that Compared Various Dietary Indices**

827 In a study by Lassale et al., subjects were participants in the SUPplementation en Vitamines et
 828 Mineraux Antioxydants (SU.VI.MAX) study and diet quality was assessed using a
 829 Mediterranean Score (MDS, rMED, MSDPS), the Diet Quality Index-International (DQI-I), the
 830 2005 Dietary Guidelines for Americans Adherence Index (DGAI), and the French Programme
 831 National Nutrition Sante-Guidelines Score (PNNS-GS).⁴⁴ Overall, better adherence to a
 832 Mediterranean diet (except for the MSDPS) or expert dietary guidelines was associated with
 833 lower weight gain in men who were normal weight at baseline (p for trend = < 0.05). In addition,
 834 among the 1,569 non-obese men at baseline, the odds of becoming obese associated with one
 835 standard deviation increase in dietary score ranged from OR = 0.63 (95% CI = 0.51 to 0.78) for
 836 the DGAI to OR = 0.72 (95% CI = 0.59 to 0.88) for the MDS, only the MSDPS was non-
 837 significant. In women, no association between diet scores and weight gain or incidence of
 838 obesity was found. Woo et al., 2008 reported on a prospective cohort study in Hong Kong to
 839 examine adherence to a diet pattern using the MDS and the Diet Quality Index International
 840 (DQI-I).⁴⁵ They found that increased adherence to either the MDS or DQI-I was not associated
 841 with becoming overweight.

842

843 **Dietary Patterns from Data-Driven Methods**

844 In the NEL review, a total of 11 studies from prospective cohort studies were included that either
 845 used factor or cluster analyses to derive dietary patterns. Eight of the eleven studies were
 846 conducted in the United States, with additional studies from the United Kingdom, Iran, and
 847 Sweden. The sample sizes ranged from 206 to 51,670 participants with follow-up times from 3 to
 848 20 years. The majority of the studies were conducted with generally healthy adult men and
 849 women,⁴⁷⁻⁵² five studies included women only,⁵³⁻⁵⁷ and one was conducted in children to
 850 examine weight gain in adolescence over the period of follow-up.⁵⁶ Outcomes examined
 851 included change in body weight (3 studies), BMI (7 studies), and waist circumference (6
 852 studies); one study examined both percent body fat and incidence of overweight/obesity.

853

854 Most of the studies found at least two generic food patterns: a “healthy/prudent” food pattern and
 855 an “unhealthy/western” pattern. Generally, healthy patterns were associated with more favorable
 856 body weight outcomes, while the opposite was seen for unhealthy patterns. However, not all
 857 studies reported significant associations. There was a potential difference in associations found

858 by sex: of the three studies that analyzed men and women separately, men tended to have null
 859 results. However, data were insufficient to draw conclusions about population subgroups.
 860 Furthermore, because the patterns are data-driven, they represent what was consumed by the
 861 study population, and thus it is difficult to compare across the disparate patterns. The one study
 862 that analyzed the dietary patterns of pre-pubescent children transitioning into adolescence
 863 showed that patterns vary widely at this age and caution should be observed when analyzing
 864 these data because the diet of children changes rapidly, as does their weight.

865
 866 The DGAC considered the systematic review by Ambrosini et al. that included seven articles,
 867 two of which overlapped with the NEL review.³⁴ Results demonstrated a positive association
 868 between a dietary pattern high in energy-dense, high fat, and low fiber foods and later obesity (4
 869 of the 7 studies), while three studies demonstrated null associations. The seven longitudinal
 870 studies of children from the United Kingdom, United States, Australia, Norway, Finland, and
 871 Colombia had follow-up periods ranging from 2 to 21 years and had sample sizes from 427 to
 872 6772 individuals. The studies determined dietary patterns using factor or cluster analysis (5) or
 873 reduced rank regression (2).

874
 875 *For additional details on this body of evidence, visit:* References 2, 13, 34, 35 and *Appendix E-*
 876 *2.27*

877

878 **DIETARY PATTERNS AND TYPE 2 DIABETES**

879 **Question 3: What is the relationship between dietary patterns and risk of type 2**
 880 **diabetes?**

881 **Source of evidence:** Existing reports

882

883 **Conclusion**

884 Moderate evidence indicates that healthy dietary patterns higher in vegetables, fruits, and whole
 885 grains and lower in red and processed meats, high-fat dairy products, refined grains, and
 886 sweets/sugar-sweetened beverages reduce the risk of developing type 2 diabetes. **DGAC Grade:**
 887 **Moderate**

888

889 Evidence is lacking for the pediatric population.

890

891 **Implications**

892 To reduce the risk of developing type 2 diabetes, individuals are encouraged to consume dietary
 893 patterns that are rich in vegetables, fruits, and whole grains and lower in red and processed
 894 meats, high-fat dairy, refined grains, and sweets/sugar-sweetened beverages in addition to

895 maintaining a healthy body weight. Diabetes can be prevented through the consumption of a
 896 variety of healthy dietary patterns that share these components and that are tailored to the
 897 biological needs and socio-cultural preferences of the individual and carried out preferably
 898 through counseling by a nutrition professional.

899

900 **Review of the Evidence**

901 The Committee considered two sources of evidence. The primary source was the NEL Dietary
 902 Patterns Systematic Review Project which included 37 studies predominantly of prospective
 903 cohorts design and some randomized trials (n=8).² This primary source was supplemented by a
 904 published meta-analysis⁵⁸ that included 15 cohort studies of which 13 overlapped with the NEL
 905 review.⁵⁸ The meta-analysis provided an estimate of the effect size of incident type 2 diabetes
 906 associated with a healthy and unhealthy dietary pattern.

907

908 Although the NEL rated the overall body of evidence for type 2 diabetes as limited, this was
 909 primarily a result of examining the different methods for defining dietary patterns (e.g. indices,
 910 data driven, and reduce rank regression) separately. As such, the NEL noted these
 911 methodological inconsistencies across studies but stated general support for the consumption of a
 912 dietary pattern rich in vegetables and fruits and low in high-fat dairy and meats. The DGAC
 913 concurred with this conclusion. However, the DGAC has elevated the grade of the entire body of
 914 evidence to moderate given that the NEL findings were corroborated by the results of a high
 915 quality meta-analysis (AMSTAR rating of 11) and the magnitude of the associations that showed
 916 when the results of 15 cohort studies are pooled, evidence indicated a 21 percent reduction in the
 917 risk of developing type 2 diabetes associated with dietary patterns characterized by high
 918 consumption of whole grains, vegetables, and fruit. Conversely, a 44 percent increased risk of
 919 developing type 2 diabetes was seen with an unhealthy dietary pattern characterized by higher
 920 consumption of red or processed meats, high-fat dairy, refined grains, and sweets.

921

922 ***Dietary Patterns and Incident Type 2 Diabetes***

923 **Dietary Approaches to Stop Hypertension (DASH)**

924 One study used the DASH score in a cohort of 820 U.S. adults ages 40 to 69 years and with
 925 equal sex distribution and racial diversity.⁵⁹ Liese et al. found adherence to the DASH score was
 926 associated with markedly reduced odds of type 2 diabetes in Whites but not in the total
 927 population, or in the Blacks and Hispanics, which comprised the majority of this cohort.

928

929 **Mediterranean-style Dietary Patterns**

930 Three studies assessed Mediterranean-style dietary pattern adherence (Mediterranean Diet Score
 931 [MDS]) with sample sizes ranging from 5,000 to more than 20,000 in both Mediterranean and
 932 U.S. populations. One study conducted in Spain with the SUN cohort (n=13,380) found a
 933 favorable association between the MDS (the original MDS of Trichopoulou) and risk of type 2

934 diabetes. Overall, a 2-point increase in MDS was associated with a 35 percent reduction in risk
 935 of type 2 diabetes.⁶⁰ Another study, conducted in Greece with the EPIC-Greece cohort
 936 (n=22,295), also assessed the relationship between the MDS and type 2 diabetes. In this second
 937 Mediterranean population, adherence to the MDS also was favorably associated with decreased
 938 risk of diabetes.⁶¹ Conversely, a study conducted in the United States, using the authors'
 939 MedDiet Score with the Multi-Ethnic Study of Atherosclerosis (MESA) cohort (n=5,390) found
 940 no association between their MedDiet Score and type 2 diabetes incidence in the total
 941 population, in men or women, or in specific racial/ethnic groups.⁶²

942

943 **Dietary Indices based on the Dietary Guidelines**

944 Four studies used dietary guidelines-based indices such as the AHEI and the Diet Quality Index
 945 (DQI). The sample sizes of the studies ranged from 1,821 to 80,029. A study that assessed
 946 adherence to the AHEI in the United States found a favorable association between AHEI score
 947 and risk of incident type 2 diabetes in women in the Nurses' Health Study (n=80,029).⁶³ In the
 948 CARDIA study (n=4,381), also from the United States, the authors found no association between
 949 DQI-2005 score and type 2 diabetes incidence in the total population or in Blacks or Whites.²⁹
 950 Studies from outside the United States included one conducted in Australia using a Total Diet
 951 score in the Blue Mountains Eye Study (BMES, n=1,821) and one from Germany using a
 952 German Food Pyramid Index with the EPIC-Potsdam cohort (n=23,531). Neither found an
 953 association between these scores and incident type 2 diabetes.^{64, 65} Thus, evidence for an
 954 association only exists with the AHEI, which does contain slightly different components from
 955 the other indices, such as nuts and legumes, trans fat, EPA + DHA (n-3 FAs), PUFAs, alcohol,
 956 red and processed meat.

957

958 **Data-Driven Approaches**

959 Eleven studies used factor analysis and one study used cluster analysis. These analyses were all
 960 conducted using data from prospective cohort studies published between 2004 and 2012 and had
 961 sample sizes ranging from 690 to more than 75,000 individuals. Five studies were conducted in
 962 the United States and the rest from developed countries around the world. Each study identified
 963 one to four dietary patterns, with the most common comparison between "western"/"unhealthy"
 964 and "prudent"/"healthier" patterns; a total of 35 diverse dietary patterns were identified within
 965 the body of evidence. Many studies had null findings, particularly studies with duration of less
 966 than 7 years of follow up.⁶⁶⁻⁶⁹ Patterns associated with lower risk of type 2 diabetes were
 967 characterized by higher intakes of vegetables, fruits, low-fat dairy products, and whole grains,
 968 and those associated with increased risk were characterized by higher intakes of red meat, sugar-
 969 sweetened foods and drinks, French fries, refined grains, and high-fat dairy products. However,
 970 the food groups identified varied substantially, even among patterns with the same name.

971

972 Three prospective cohort studies used reduced rank regression to examine the relationship
 973 between dietary patterns and type 2 diabetes.⁷⁰⁻⁷² Two of the studies were conducted in the

974 United States and one in the United Kingdom. The sample sizes were 880 for Liese (2009),
 975 2,879 for Imamura (2009), and 6,699 for McNaughton (2008). The independent variables in
 976 these studies were dietary pattern scores, and biomarkers were used as response variables in two
 977 of the studies. Dietary patterns that included meat intake and incident type 2 diabetes were
 978 positively associated in the two studies that used biomarkers as response variables, though the
 979 definitions of meat differed.^{70, 71} However, because so few studies were available and the
 980 methodology used and different populations considered varied so much, the information was
 981 insufficient to assess consistency or draw conclusions.

982

983 **Other Dietary Patterns**

984 The body of evidence examined included seven studies conducted between 2004 and 2013,
 985 consisting of six RCTs⁷³⁻⁷⁹ and one prospective cohort study (PCS).⁸⁰ Two studies were
 986 conducted in the United States; one in the United States and Canada; one in Spain (2
 987 PREDIMED articles); and one each in Greece, Italy, and Sweden. The sample sizes of the RCTs
 988 ranged from 82 to 1,224 participants and the PCS had a sample size of 41,387 participants. All
 989 eight studies were conducted in adults. RCT duration ranged from 6 weeks to a median of 4
 990 years and the PCS duration was 2 years. The RCTs were primary prevention studies of at-risk
 991 participants. Baseline health status in the study participants included those with mild
 992 hypercholesterolemia, overweight or obesity, metabolic syndrome, abdominal obesity, and three
 993 or more CVD risk factors, including metabolic syndrome. The PCS participants were individuals
 994 in the Adventist Health Study who did not have type 2 diabetes.⁸⁰ Three studies looked at a
 995 Mediterranean-style diet,^{75, 77-79} one study examined the Nordic diet (defined by the authors of
 996 the study as a diet rich in high-fiber plant foods, fruits, berries, vegetables, whole grains,
 997 rapeseed oil, nuts, fish and low-fat milk products, but low in salt, added sugars, and saturated
 998 fats),⁷³ and three studies looked at either the DASH diet or a variation of the DASH diet,^{74, 76} or a
 999 vegetarian diet.⁸⁰

1000

1001 Two of the seven studies examined the association between adherence to a dietary pattern and
 1002 incidence of type 2 diabetes.^{79, 80} Although the results of both studies showed a favorable
 1003 association between either a Mediterranean-style or a vegetarian dietary pattern and incidence of
 1004 type 2 diabetes the studies differed in design and dietary pattern used to assess diet exposure.
 1005 The other studies examined the intermediate outcomes of impaired glucose tolerance and/or
 1006 insulin resistance and are discussed in the next section.

1007

1008 ***Dietary Patterns and Intermediate Outcomes***

1009 Five studies examined adherence to a dietary pattern and intermediate outcomes related to
 1010 glucose tolerance and/or insulin resistance: two RCTs^{23, 46} and three prospective cohort studies.<sup>29,
 1011 31, 64</sup> It was difficult to assess food components across these studies, as numerous different scores
 1012 were used and no compelling number of studies used any one score or index. Even so, favorable
 1013 associations between dietary patterns and intermediate outcomes were found.

1014
 1015 The two RCTs were conducted in populations in Europe that were at risk of diabetes. An early
 1016 report from the PREDIMED trial showed that a Mediterranean diet decreased fasting blood
 1017 glucose, fasting insulin, and HOMA-IR scores in a Spanish population at risk of CVD.²³ In the
 1018 Oslo Diet and Exercise Study (ODES), increased adherence to the authors' a priori diet score
 1019 resulted in decreased fasting insulin and insulin after a glucose challenge, but not fasting glucose,
 1020 in Norwegian men with metabolic syndrome.⁴⁶ Results from prospective cohort studies were
 1021 consistent in showing a favorable association between diet score and fasting glucose, fasting
 1022 insulin or HOMA-IR,^{29, 31} with the exception of one study that found the association with fasting
 1023 glucose only in men.⁶⁴

1024 1025 **Data-Driven Approaches**

1026 Variations in populations studies, definition of outcomes, dietary assessment methodologies, and
 1027 methods used to derive patterns resulted in a highly variable set of dietary patterns, thus making
 1028 it difficult to draw conclusions from studies using data-driven approaches. For example, one
 1029 study measured fasting blood glucose with a cutoff of 6.1 and greater mmol/L;⁴⁷ another study
 1030 measured plasma glucose with a cutoff of 5.1 and greater mmol/L,⁸¹ while a third study
 1031 measured plasma glucose after an overnight fast and after a standard 75 g oral glucose tolerance
 1032 test.⁸² Three prospective cohort studies assessed the association between dietary patterns and
 1033 plasma glucose levels. Two U.S. studies derived patterns using cluster analysis^{47, 81} and one
 1034 study conducted in Denmark used factor analysis.⁸² Duffey et al. identified two diet clusters:
 1035 “Prudent Diet” and “Western Diet”;⁴⁷ Kimokoti et al. identified five clusters: “Heart Healthier,”
 1036 “Lighter Eating,” “Wine and Moderate Eating,” “Higher Fat,” and “Empty Calories”;⁸¹ and Lau
 1037 et al. derived two factors: “Modern” and “Traditional.”⁸²

1038
 1039 *For additional details on this body of evidence, visit:* References 2, 58, and *Appendix E-2.28*

1040 1041 1042 **DIETARY PATTERNS AND CANCER**

1043 **Existing Evidence around Foods and Nutrients and Cancer**

1044 The role of dietary composition in cancer risk has been postulated since ancient times, yet
 1045 scientific evidence for such relationships was sparse until nearly a century ago. Experimental
 1046 models of cancer based upon chemical carcinogens, radiation, viral-transmission, and inherited
 1047 genetic variations gradually emerged in first half of the 20th century and were soon found to be
 1048 influenced by dietary and nutritional interventions. The establishment of population-based cancer
 1049 registries around the globe in the years following World War II clearly indicated that the
 1050 incidence and mortality of specific cancers and the patterns of cancers varied widely between
 1051 countries. Soon, studies of migrant populations demonstrated that in parallel with acculturation,

1052 cancer risk evolved toward that observed in the adopted country, implicating a strong role for
1053 environmental influences, such as dietary patterns, in cancer risk. When coupled with national
1054 food consumption data, relationships between dietary patterns or components and cancer risk
1055 were hypothesized. The development of dietary assessment tools, such as FFQs, paved the way
1056 for large prospective epidemiologic cohort studies designed to examine more precisely the role
1057 of dietary patterns, foods, and specific nutrients in the risk of various cancers.⁸³ Additional diet
1058 assessment tools, such as food diaries, and single and multi-day 24-hr recalls enhanced the
1059 ability to undertake population studies and mechanism-based RCTs. These studies were made
1060 possible by USDA support of research to advance laboratory methods to define the nutrient
1061 content of foods in the U.S. food supply and establish a database that, when coupled with diet
1062 assessment tools, provides an estimated intake of energy, macronutrients, vitamins, minerals and
1063 other dietary variables. More recently, inclusion into the database of non-nutrient bioactive
1064 components primarily found in vegetables and fruits has enhanced the ability to define human
1065 intake of bioactive components that may affect health and disease.

1066
1067 In 1982, the American Institute for Cancer Research (AICR), a part of the World Cancer
1068 Research Fund (WCRF) global philanthropic network, was established. Together, the mission of
1069 WCRF/AICR is to fund research and disseminate evidence-based cancer prevention guidelines to
1070 the public. In 1997, the AICR/WCRF published the results of a comprehensive multi-year effort
1071 to systematically review the published scientific literature and develop dietary guidelines for
1072 cancer prevention.⁸⁴ With a rapid expansion of available data in the subsequent years, the
1073 process was repeated for the 2007 AICR/WCRF report.⁸⁵ This effort has been enhanced in
1074 subsequent years by the AICR/WCRF Continuous Update Project (CUP), in which data are
1075 reviewed and updated on a continuous, rolling basis for specific cancers, with several reports
1076 completed annually.⁸⁶ This effort is accomplished through a rigorous systematic review process
1077 in which scientific evidence is gathered, reviewed and judged by panels of experts in nutrition
1078 and cancer in order to generate nutrition and cancer prevention goals for policy makers, the
1079 general population, and individuals seeking to reduce cancer risk.⁸⁷ The most recent summary
1080 of the systematic review which documents important information about the relationship between
1081 specific foods, nutrients and other lifestyle behavior and cancer risk is found in Table D2.2.

1082
1083 As previously mentioned, the 2015 DGAC chose to determine whether an examination of dietary
1084 patterns, could inform the understanding of diet and cancer risk. As this scientific literature is
1085 relatively early in its development, we limited our search to the four most common malignancies
1086 affecting the American public—lung, breast, colon/rectal, and prostate—which account for the
1087 majority of the cancer burden in the United States. Although the published literature on dietary
1088 patterns and cancer risk is relatively young, the DGAC felt it was important to examine the
1089 evidence and conclusions, consider the implications for development of dietary guidelines, and
1090 indicate areas for future research.

1091

1092 **Table D2.2. American Institute for Cancer Research / World Cancer Research Fund**
 1093 **(AICR/WCRF) Summary of Strong Evidence on Diet, Nutrition, Physical Activity, and**
 1094 **Cancer Prevention, updated 2014**

	Mouth, Pharynx, Larynx (2007)	Nasopharynx (2007)	Esophagus (2007)	Lung (2007)	Stomach (2007)	Pancreas (2007)	Gall bladder (2007)	Liver (2007)	Colorectum (2011)	Breast Premenopause (2010)	Breast (Postmenopause) (2010)	Ovary (2014)	Endometrium (2013)	Prostate (2014)	Kidney (2007)	Skin (2007)
↓↓ Convincing decreased risk. ↓ Probable decreased risk. ↑↑ Convincing increased risk. ↑ Probable increased risk. • Substantial effect on risk unlikely.																
Foods containing dietary fiber									↓↓							
Aflatoxins	↓		↓		↓			↑↑								
Non-starchy vegetables ¹					↓											
Allium vegetables									↓							
Garlic	↓		↓	↓	↓											
Fruits ²									↑↑							
Red meat									↑↑							
Processed meat																
Cantonese-style salted fish		↑														
Diets high in calcium ³									↓							
Salt, salted and salty foods					↑											
Glycemic load													↑			
Arsenic in drinking water				↑↑												↑
Mate			↑													
Alcoholic drinks ⁴	↑↑		↑↑					↑	↑↑	↑↑	↑↑				•	
Coffee					•								↓		•	
Beta-carotene ⁵				↑↑										•		•
Physical activity ⁶									↓↓		↓		↓			
Body fatness ⁷			↑↑			↑↑	↑		↑↑	↓	↑↑	↑	↑↑	↑	↑↑	
Adult attained height						↑			↑↑	↑	↑↑	↑↑		↑		
Greater birth weight										↑						
Lactation										↓↓	↓↓					

1095

¹ Includes evidence on foods containing carotenoids for mouth, pharynx, larynx; foods containing beta-carotene for esophagus; foods containing vitamin C for esophagus.

² Includes evidence on foods containing carotenoids for mouth, pharynx, larynx, and lung; foods containing beta-carotene for esophagus; food containing vitamin C for esophagus.

³ Evidence is from milk and studies using supplements for colorectum.

⁴ Convincing increased risk for men and probably increased risk for women for colorectum. Evidence applies to adverse effect for kidney.

⁵ Evidence derived from studies using supplements for lung.

⁶ Convincing increased risk for colon not rectum.

⁷ Probable increased risk for advanced not non-advanced prostate cancer.

1096 **AICR/WCRF Evidence Stratification**⁸⁷

1097 **Convincing:** The evidence for a convincing grade is strong enough to support a causal relationship. This relationship is
 1098 robust enough that it is unlikely to be modified of research in the foreseeable future. A grade of “convincing” requires evidence
 1099 from more than one study type, data from at least two cohort studies, no unexplained heterogeneity between study types with
 1100 regard to the presence or absence of an association, good quality studies where random or systematic errors are unlikely, presence
 1101 of a dose-response relationship, and strong and plausible experimental evidence relating typical human exposures to relevant
 1102 cancer outcomes.

1103 **Probable:** The criteria for determining a probable diet and cancer relationship include: evidence from at least two cohort
 1104 studies or at least five case-control studies, no substantial unexplained heterogeneity between or within study types in the
 1105 presence or absence of an association or direction of effect, good quality studies where the likelihood of random or systematic
 1106 error is low, and evidence for biologic plausibility.

1107 **Limited—suggestive:** This grade is assigned when the evidence is too limited to permit a probable or convincing judgment,
 1108 but there is evidence of a direction of effect. The evidence may have methodological flaws, or there may be a limited number of
 1109 studies. A grade of “limited-suggestive” requires the following: evidence from at least two cohort studies or five case-control
 1110 studies, there is some evidence for biologic plausibility, and the direction of the effect is generally consistent, although there may
 1111 be some unexplained heterogeneity.

1112 **Limited—no conclusion:** This grade describes diet and cancer relationships where the evidence was ample for review by
 1113 the panel, but it was too limited to receive one of the other grades. The available studies may be of good quality, but limited in
 1114 number or yielding inconsistent results.

1115 **Substantial effect on risk unlikely:** This grade is assigned when the evidence is strong that a particular nutrient, food,
 1116 dietary pattern, or physical activity is unlikely to have a substantial causal relationship to a cancer outcome. Data must be strong
 1117 enough that modification in the foreseeable future is unlikely.

1118

1119 **Question 4: What is the relationship between dietary patterns and risk of cancer?**1120 **Source of evidence:** NEL systematic review

1121

1122 **Conclusions**

1123 **Colon/Rectal Cancer:** Moderate evidence indicates an inverse association between dietary
 1124 patterns that are higher in vegetables, fruits, legumes, whole grains, lean meats/seafood, and low-
 1125 fat dairy and moderate in alcohol; and low in red and/or processed meats, saturated fat, and
 1126 sodas/sweets relative to other dietary patterns and the risk of colon/rectal cancer. Conversely,
 1127 diets that are higher in red/processed meats, French fries/potatoes, and sources of sugars (i.e.,
 1128 sodas, sweets, and dessert foods) are associated with a greater colon/rectal cancer risk. **DGAC**

1129 **Grade: Moderate**

1130

1131 **Breast Cancer:** Moderate evidence indicates that dietary patterns rich in vegetables, fruit, and
 1132 whole grains, and lower in animal products and refined carbohydrate, are associated with
 1133 reduced risk of post-menopausal breast cancer. The data regarding this dietary pattern and pre-
 1134 menopausal breast cancer risk point in the same direction, but the evidence is limited due to
 1135 fewer studies. **DGAC Grade: Moderate for postmenopausal breast cancer risk; Limited for**
 1136 **premenopausal breast cancer risk**

1137

1138 **Lung Cancer:** Limited evidence from a small number of studies suggests a lower risk of lung
 1139 cancer associated with dietary patterns containing more frequent servings of vegetables, fruits,

1140 seafood, grains/cereals, and legumes, and lean versus higher fat meats and lower fat or non-fat
 1141 dairy products. Despite reported modest significant reductions in risk, definitive conclusions
 1142 cannot be established at this time due to the small number of articles, as well as wide variation in
 1143 study design, dietary assessment, and case ascertainment. **DGAC Grade: Limited**

1144

1145 **Prostate Cancer:** No conclusion can be drawn regarding the relationship between dietary
 1146 patterns and the risk of prostate cancer. This is due to limited evidence from a small number of
 1147 studies with wide variation in study design, dietary assessment methodology and prostate cancer
 1148 outcome ascertainment. **DGAC Grade: Grade not assignable**

1149

1150 **Implications**

1151 The data accumulating regarding the impact of dietary patterns on risk of certain types of cancers
 1152 supports the concept that a healthy dietary pattern may significantly reduce the overall burden of
 1153 cancer in the United States. Emerging studies on dietary patterns support the findings of expert
 1154 reviews regarding individual foods and nutrients. Effective strategies to initiate early in life and
 1155 maintain a healthy dietary pattern and body weight, coupled with regular physical activity, will
 1156 significantly reduce the cancer burden in America.

1157

1158 **Review of the Evidence**

1159 ***Dietary Patterns and Colorectal Cancer***

1160 This systematic review included 21 articles from prospective cohort studies and one article from
 1161 an RCT published since 2000 that examined the relationship between dietary patterns and risk of
 1162 colorectal cancer.⁸⁸⁻¹⁰⁹ The articles used diverse methodology to assess dietary patterns. Nine
 1163 articles used indices/scores to assess dietary patterns, 10 articles used data-driven methods, and
 1164 three used other approaches.

1165

1166 The dietary patterns examined in this systematic review were defined in various ways, making
 1167 comparisons between articles difficult. However, despite general heterogeneity in this body of
 1168 evidence, some protective dietary patterns emerged, particularly in articles where patterns were
 1169 defined by index or score; articles using data-driven methods were less consistent. Patterns
 1170 emphasizing vegetables, fruits, fish/seafood, legumes, low-fat dairy, and whole grains were
 1171 generally associated with reduced risk of colorectal cancer. Patterns higher in red/processed
 1172 meats, potatoes/French fries, and sodas/sweets/added sugars were generally associated with
 1173 increased risk of colorectal cancer.

1174

1175 The relationship between dietary patterns and colorectal cancer risk often varied by sex and
 1176 tumor location. Results based on analysis by sex were mixed, while analysis in tumor subgroups
 1177 seemed to indicate that dietary patterns may be more strongly associated with tumor
 1178 development in distal regions of the colon/rectum. Although most cohort studies make extensive

1179 efforts to include participants across a wide range of race/ethnic groups and across the socio-
 1180 economic continuum, there still may be some groups for which the association between dietary
 1181 patterns and colorectal cancer risk cannot be reliably assessed and therefore conclusions cannot
 1182 be drawn.

1183

1184 ***Dietary Patterns and Breast Cancer***

1185 This systematic review included 25 prospective cohort studies and one RCT published since
 1186 2000 that examined the relationship between dietary patterns and risk of breast cancer.^{94, 101, 104,}
 1187 ¹¹⁰⁻¹³¹ The studies used multiple approaches to assess dietary patterns and cancer risk. Eight
 1188 studies used indices/scores to assess dietary patterns, 13 studies used factor or principal
 1189 components analysis, two used reduced rank regression, two made comparisons on the basis of
 1190 animal product consumption, and one conducted an RCT of a low-fat dietary pattern.

1191

1192 This moderate body of evidence encompassed a large diversity in methods to assess or determine
 1193 dietary patterns, making comparison across studies challenging. Despite this variability, 17 of the
 1194 included studies found statistically significant relationships between dietary patterns and breast
 1195 cancer risk, particularly among certain groups of women. Because a variety of different
 1196 methodologies were employed to derive dietary patterns, and the patterns, while similar in many
 1197 respects, were composed of different combinations of foods and beverages, it was difficult to
 1198 determine which patterns had the greatest impact on breast cancer risk reduction.

1199

1200 The relationship between dietary patterns and breast cancer risk may be more consistent among
 1201 postmenopausal women, but additional research is needed to explore the relationships for both
 1202 pre- and post- menopausal cancer. Certain histopathologic and molecular phenotypes of breast
 1203 cancer may be affected more by certain dietary patterns, but this has not yet been explored
 1204 sufficiently. For example, limited studies to date suggest that estrogen or progesterone receptor
 1205 status of breast cancers may define subgroups with unique dietary risk profiles, but no
 1206 conclusions can be drawn at this time. More research is needed to explore other factors that may
 1207 influence the relationship between dietary patterns during various stages of life and breast cancer
 1208 risk, such as anthropometrics, BMI (including weight change over adulthood), physical activity,
 1209 sedentary behavior, and reproductive history, including ages of menarche, age of menopause,
 1210 parity, and breast feeding.

1211

1212 ***Dietary Patterns and Lung Cancer***

1213 This systematic review included three prospective cohort studies and one nested case-cohort
 1214 study published since 2000 that examined the relationship between dietary patterns and risk of
 1215 lung cancer.^{101, 104, 132, 133} The studies used different methods to assess dietary patterns. Two
 1216 studies used an index/score to measure adherence to a dietary pattern, one study derived dietary
 1217 patterns using principal components analysis, and another based dietary patterns on participant
 1218 reports of animal product intake. With only four relevant studies that used different approaches

1219 for assessing or determining dietary patterns, the evidence available to examine the relationship
1220 between dietary patterns and risk of lung cancer is limited.

1221

1222 ***Dietary Patterns and Prostate Cancer***

1223 This systematic review included seven prospective cohort studies (from six different cohorts)
1224 published since 2000 that examined the relationship between dietary patterns and risk of prostate
1225 cancer.^{101, 134-139} The studies used different methods to assess dietary patterns. Three studies used
1226 index/scores to assess dietary patterns, two studies used factor analysis, one study used principle
1227 components analysis, and one made comparisons on the basis of animal product consumption.

1228

1229 Most of the seven studies included in this systematic review did not detect clear or consistent
1230 relationships between dietary patterns and risk of prostate cancer, though one found that
1231 adherence to the Dietary Guidelines (assessed using the HEI-2005 and AHEI-2010) was
1232 associated with a lower risk of prostate cancer, particularly among men who had a prostate-
1233 specific antigen screening in the past 3 years. Because these studies used a range of different
1234 approaches for assessing dietary patterns in populations with variable cancer screening patterns,
1235 had heterogeneous prostate cancer outcome ascertainment, and were typically limited to dietary
1236 exposure late in life, the results were inconclusive regarding risk for clinically significant
1237 prostate cancer.

1238

1239 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3344>

1240

1241 **DIETARY PATTERNS AND CONGENITAL ANOMALIES**

1242 **Existing Evidence around Foods and Nutrients and Congenital Anomalies**

1243 It is well established that adequate folate status is critical for the prevention of neural tube
1244 defects, specifically anencephaly and spina bifida, as well as other birth defects.¹⁴⁰ Folate is
1245 often described by its source, with “folate” referring to naturally occurring folate from food
1246 sources, and “folic acid” referring to the synthetic form used in dietary supplements and food
1247 fortification. After mandatory fortification of enriched cereal products with folic acid in 1998,
1248 serum folate concentrations in the U.S. population more than doubled, and rates of neural tube
1249 defects decreased by 20 to 30 percent.^{141, 142}

1250

1251 Despite this decrease, nearly one fifth of females ages 14 to 30 years do not meet the estimated
1252 average requirement for folate, the level deemed to be adequate for one half of healthy females
1253 in the age group.¹⁴³ The current U.S. Preventive Services Task Force recommends that women
1254 capable of becoming pregnant should take 400 to 800 micrograms of folic acid daily from
1255 fortified food or supplements in addition to a healthy diet rich in food sources of folate and folic
1256 acid to reduce risk of neural tube and other birth defects.¹⁴⁴ Women with a history of a pregnancy

1257 affected by a neural tube defect or who are at high risk of neural tube defects require 4 mg of
1258 synthetic folic acid supplements daily under the supervision of a physician.¹⁴⁵ Given the
1259 emphasis on a healthy diet, the DGAC was interested in understanding which dietary patterns, if
1260 any, were associated with a decreased risk of congenital anomalies among women of
1261 reproductive age.

1262

1263 **Question 5: What is the relationship between dietary patterns and risk of**
1264 **congenital anomalies?**

1265 **Source of evidence:** NEL systematic review

1266

1267 **Conclusion**

1268 Limited evidence suggests that healthy maternal dietary patterns during the preconception period
1269 that are higher in vegetables, fruits, and grains, and lower in red and processed meats, and low in
1270 sweets were associated with lower risk of developing of neural tube defects, particularly among
1271 women who do not take folic acid supplements. Whereas some dietary patterns were associated
1272 with lower risk of developing anencephaly, others were associated with lower risk of developing
1273 spina bifida.

1274

1275 Evidence is insufficient to determine an association between maternal dietary patterns and
1276 congenital heart defects or cleft lip/palate.

1277

1278 All studies were consistent in demonstrating that folic acid supplementation periconceptionally
1279 was associated with a decreased risk of having a child with a birth defect (e.g. neural tube
1280 defects, congenital heart defects, and cleft lip/palate). **DGAC Grade: Neural Tube Defects –**
1281 **Limited; Congenital Heart Defects – Grade not assignable; Cleft Lip/Palate – Grade not**
1282 **assignable**

1283

1284 **Implications**

1285 Women of reproductive age should consume folic acid in the form of a supplement or through
1286 fortified foods in the range recommended by the U.S. Preventive Services Task Force (400 to
1287 800 micrograms) in addition to consuming a diet rich in vegetables, fruits, and grains; lower in
1288 red and processed meats; and low in sweets.

1289

1290 **Review of the Evidence**

1291 This series of systematic reviews included five case-control studies (using data from three
1292 cohorts) published since 1980 that examined the relationship between maternal dietary patterns
1293 and congenital anomalies in infants.¹⁴⁶⁻¹⁵⁰ Three articles examined neural tube defects,^{146, 147, 149}

1294 two articles examined congenital heart defects,^{147, 150} and two articles examined orofacial
 1295 clefts.^{146, 148}

1296

1297 Although all five case-control studies reported significant associations between dietary patterns
 1298 and risk of congenital anomalies in women not taking folic acid supplementation, the variability
 1299 of dietary patterns methodology used and composition of dietary patterns identified made it
 1300 difficult to draw conclusions. All studies were consistent in finding that folate delivered
 1301 periconceptionally in food or as a supplement as a key nutrient was associated with lower risk of
 1302 developing congenital anomalies. It should be noted that some of the included studies were
 1303 conducted in countries with mandatory folate fortification, while others were from countries that
 1304 prohibit such fortification.

1305

1306 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3356>

1307

1308 **DIETARY PATTERNS AND NEUROLOGICAL AND PSYCHOLOGICAL** 1309 **ILLNESSES**

1310 **Existing Evidence around Foods and Nutrients and Neurological and** 1311 **Psychological Illnesses**

1312 Neuropsychological development and function is increasingly recognized as a high national
 1313 priority for health promotion and chronic disease prevention. Two major components of
 1314 neuropsychological function are *cognition*, the ability to reason, and *mood*, balanced and
 1315 appropriate to enable optimal cognition.

1316

1317 Nutrition for optimal neurodevelopment in very young children has long been a subject of
 1318 research. The 2010 DGAC concluded that moderate evidence supported a positive relationship
 1319 between maternal dietary intakes of n-3 from seafood and improved cognitive ability in
 1320 infants.¹⁵¹ The rising numbers of U.S. older adults and the potential human and financial cost of
 1321 age-related cognitive impairments, such as Alzheimer's disease and other dementias, also have
 1322 helped drive national interest in chronic mental disease.^{152, 153} Separately, depression affected 8
 1323 percent of Americans for at least two weeks annually from 2007-2010, and of these, 80 percent
 1324 report functional impairment.¹⁵⁴ Many preclinical and human studies have established
 1325 relationships between traditional nutrients (e.g., omega-3 fatty acids) and central nervous system
 1326 composition and function. Studies appearing in the last few years reflect the increasing research
 1327 interest in the links between diet and neurological health.

1328

1329 The hypothesis that nutrition can reduce and/or play a role in the treatment of these mental
 1330 diseases and their related burdens has been studied in relation to several nutrients and foods,
 1331 including the B vitamins, vitamin E, and selenium.^{155, 156} The omega-3 fatty acids

1332 eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are among the most studied
 1333 nutrients for neural health, in part because DHA is a major component of the brain, specifically
 1334 gray matter and its synapses, and the specialized light detecting cells of the retina. DHA, in
 1335 particular, supports the amplitude and signaling speed of neural response. EPA has emerged as a
 1336 nutrient with antidepressive properties and continued studies to define its role in prevention and
 1337 therapy are underway. Sufficiently strong medical evidence has been obtained for EPA and DHA
 1338 such that supplements are now considered as complementary therapy for major depressive
 1339 disorder by the American Psychiatric Association¹⁵⁷ and more recent data from a meta-analysis
 1340 has found them effective.¹⁵⁸ Before 2010, the number of published dietary pattern studies was
 1341 small. However, a more substantial literature on dietary patterns and neuropsychological health
 1342 has been published since 2010. The DGAC was therefore able to consider prevention of adult
 1343 neuropsychological ill health for the first time.

1344

1345 **Question 6: What is the relationship between dietary patterns and risk of**
 1346 **neurological and psychological illnesses?**

1347 **Source of evidence:** NEL systematic review

1348

1349 **Conclusion**

1350 Limited evidence suggests that a dietary pattern containing an array of vegetables, fruits, nuts,
 1351 legumes and seafood consumed during adulthood is associated with lower risk of age-related
 1352 cognitive impairment, dementia, and/or Alzheimer’s disease. Although the number of studies
 1353 available on dietary patterns and neurodegenerative disease risk is expanding, this body of
 1354 evidence, which is made up of high-quality observational studies, has appeared only in recent
 1355 years and is rapidly developing. It employs a wide range of methodology in study design,
 1356 definition and measurement ascertainment of cognitive outcomes, and dietary pattern
 1357 assessment. **DGAC Grade: Limited**

1358

1359 Limited evidence suggests that dietary patterns emphasizing seafood, vegetables, fruits, nuts, and
 1360 legumes are associated with lower risk of depression in men and non-perinatal women. However,
 1361 the body of evidence is primarily composed of observational studies and employs a range of
 1362 methodology in study design, definition, and measurement of dietary patterns and ascertainment
 1363 of depression/depressive signs and symptoms. Studies on dietary patterns in other populations,
 1364 such as women in the post-partum period, children and adolescents, as well as those in various
 1365 ethnic and cultural groups, are too limited to draw conclusions. **DGAC Grade: Adults –**
 1366 **Limited; Children, adolescents, and women in the post-partum period – Grade not**
 1367 **assignable**

1368

1369 **Implications**

1370 Dietary patterns emphasizing vegetables, fruits, seafood, legumes and nuts similar to those that
 1371 achieve chronic disease risk reduction are consistent with maintaining neurocognitive health,
 1372 including cognitive ability in healthy aging, and balanced mood.

1373

1374 **Review of the Evidence**1375 ***Dietary Patterns and Cognitive Impairment, Dementia, and Alzheimer's Disease***

1376 This systematic review includes 30 articles (two articles analyzed data taken from RCTs and 28
 1377 articles used data from prospective cohort studies) published since 1980 (with all but two
 1378 published since 2008) that examined the relationship between dietary patterns and age-related
 1379 cognitive impairment, dementia, and/or Alzheimer's disease.¹⁵⁹⁻¹⁸⁸ Twenty of the articles
 1380 included in this review assessed the relationship between dietary patterns and cognitive
 1381 impairment, 10 articles examined cognitive impairment or dementia, and eight articles looked at
 1382 Alzheimer's disease.

1383

1384 The articles used several different methods to assess dietary patterns. Two articles analyzed data
 1385 from RCTs that tested or described dietary patterns, 23 articles used indices/scores to assess
 1386 dietary patterns quality or adherence, three articles used data-driven methods, and three used
 1387 reduced rank regression. Most (18 of 28) articles found an association between dietary patterns
 1388 and age-related cognitive impairment, dementia, and/or Alzheimer's disease. Despite some
 1389 heterogeneity in this body of evidence, some common elements of dietary patterns were
 1390 associated with measures of cognitive impairment, dementia, and/or Alzheimer's disease:

1391

1392 • Patterns higher in vegetables, fruits, nuts, legumes, and seafood were generally associated
 1393 with reduced risk of age-related cognitive impairment, dementia, and/or Alzheimer's
 1394 disease.

1395 • Patterns higher in red and/or processed meats were generally associated with greater age-
 1396 related cognitive impairment. Relatively few studies reported on refined sugar and added
 1397 salt, and patterns including these nutrients tended to report greater cognitive impairment.

1398 Although some studies included participants from a range of race/ethnic and socioeconomic
 1399 groups, the results are most applicable to the general healthy aging population. In addition,
 1400 dietary patterns were derived using dietary intake measured at baseline only, and therefore, may
 1401 not reflect patterns consumed throughout relevant periods of life before enrollment in the study,
 1402 or changes in intake that may have occurred over the duration of the study. Similarly, several
 1403 studies measured cognitive function only at a single time point (follow-up), and therefore, could
 1404 not assess change in cognitive function over time. Finally, though these studies controlled for a
 1405 number of confounders, not all apparently relevant potential confounders were adjusted for (e.g.,
 1406 existing or family history of cognitive decline, dementia, or Alzheimer's disease; baseline health

1407 status; changes in dietary intake over time) and, as with all association studies, residual
 1408 confounding is possible.

1409

1410 ***Dietary Patterns and Depression***

1411 This systematic review includes nineteen articles (17 from prospective cohort studies, and 2
 1412 using data from RCTs) published since 1980 (all of which were published since 2008) that
 1413 assessed the relationship between dietary patterns and depression.^{175, 182, 189-205}

1414

1415 The articles used several different methods to assess dietary patterns. Two studies tested the
 1416 effects of dietary patterns as part of an RCT, six articles used indices/scores to assess dietary
 1417 patterns, 10 articles used data-driven methods, and one used reduced rank regression. Despite
 1418 methodological and outcome heterogeneity in this body of evidence, some protective dietary
 1419 patterns emerged:

1420

1421 • Patterns emphasizing seafood, vegetables, fruits, and nuts, were generally associated with
 1422 reduced risk of depression.

1423 • Patterns emphasizing red and processed meats and refined sugar were generally
 1424 associated with increased risk of depression.

1425 This body of evidence did have several limitations. There was considerable variability in how the
 1426 outcome of depression was assessed, with some studies using various depression scales, some
 1427 using physician diagnosis/hospital discharge records, and others using proxies such as use of
 1428 depression medication. Although most studies make extensive efforts to include participants
 1429 across a wide range of race/ethnic groups and across the socio-economic continuum, there still
 1430 may be some subgroups for which the association between dietary patterns and depression risk
 1431 cannot be reliably assessed and therefore conclusions cannot be drawn for them. Research is
 1432 needed to determine whether dietary patterns are associated with risk of depression in
 1433 particularly vulnerable subgroups, specifically children, adolescents, young adults, and women
 1434 during the post-partum period. Additional limitations within this body of evidence make it
 1435 difficult to draw stronger conclusions, including assessment of dietary patterns and depression
 1436 outcomes at a single point in time, potential for residual confounding despite adjustment for a
 1437 number of factors, and few studies conducted in U.S.-based populations.

1438

1439 ***For additional details on this body of evidence, visit:*** <http://NEL.gov/topic.cfm?cat=3352>

1440

1441

1442 **DIETARY PATTERNS AND BONE HEALTH**

1443 **Existing Evidence around Foods and Nutrients and Bone Health**

1444 Low bone mineral density and osteoporosis are common in the United States, particularly in
 1445 older adults, and its contribution to disability and cost to the health care system continues to rise
 1446 in parallel to longer life expectancy. As described in *Part D. Chapter 1: Food and Nutrient*
 1447 *Intakes, and Health: Current Status and Trends*, more than half of women ages 60 to 69 years
 1448 have low bone mass and approximately 12 percent meet established criteria for osteoporosis. The
 1449 prevalence of osteoporosis increases with age; about one-quarter of women ages 70 to 79 years
 1450 and about one-third of women older than age 80 years have osteoporosis. Low bone mass is less
 1451 common in older men but is increasingly recognized. Among U.S. men ages 60 to 69 years,
 1452 about a third have low bone mass and this increases to about 40 percent and slightly more than
 1453 50 percent for men ages 70 to 79 years and 80 years and older, respectively.

1454
 1455 Poor bone health and osteoporotic fractures are a major cause of morbidity and mortality in the
 1456 elderly and account for significant health care costs. Understanding the extent to which dietary
 1457 factors can help improve bone health and reduce the incidence of fractures across all segments of
 1458 the population, particularly in the elderly, is important for the health and well-being of the
 1459 nation.

1460
 1461 The most critical nutrients for healthy bone are calcium, vitamin D, and phosphorous. As part of
 1462 their 2011 report on Calcium and Vitamin D, the Institute of Medicine extensively reviewed the
 1463 available data and updated the Dietary Reference Intakes (DRIs) for calcium and vitamin D for
 1464 men and women across life stages.²⁰⁶ The new reference values were based upon a strong body
 1465 of evidence regarding bone growth and maintenance. At the time of the report, these bone health
 1466 outcomes (in particular bone mass [bone mineral content]) were the only indicators on which
 1467 there was sufficient scientific evidence to define DRIs; a thorough review of other outcomes
 1468 (bone mineral density, risk of fractures, and osteoporosis) provided mixed and inconclusive
 1469 results, and thus did not inform the DRIs. *Part D. Chapter 1: Food and Nutrient Intakes, and*
 1470 *Health: Current Status and Trends* of this DGAC report concluded that calcium and vitamin D
 1471 were shortfall nutrients of public health concern. The estimated low levels of intake in various
 1472 age and sex groups place many at risk for suboptimal bone health. The DGAC asked additional
 1473 questions regarding bone health that went beyond those relating to the role of specific and well-
 1474 known nutrients on bone remodeling. Specifically, the DGAC considered the influence of dietary
 1475 patterns and their relationship to bone health and specific bone health outcomes across the
 1476 lifespan, including bone density and fractures. This approach enabled the DGAC to consider the
 1477 relationship between the total diet and its component foods and nutrients, acting in combination,
 1478 on bone health outcomes. This section reviews this evidence and forms the basis for the DGAC
 1479 recommendation for action at individual and population level as well as its research
 1480 recommendations.

1481

1482 **Question 7: What is the relationship between dietary patterns and bone health?**1483 **Source of evidence:** NEL systematic review

1484

1485 **Conclusion**

1486 Limited evidence suggests that a dietary pattern higher in vegetables, fruits, grains, nuts, and
 1487 dairy products, and lower in meats and saturated fat, is associated with more favorable bone
 1488 health outcomes in adults, including decreased risk of fracture and osteoporosis, as well as
 1489 improved bone mineral density. Although a growing number of studies are examining the
 1490 relationship between dietary patterns and bone health in adults, the number of high-quality
 1491 studies is modest and those available employ a wide range of methodologies in study design,
 1492 dietary assessment techniques, and varying bone health outcomes.

1493

1494 Definitive conclusions regarding the relationship between dietary patterns and bone health
 1495 outcomes (bone mineral density and bone mineral content) in children and adolescents cannot be
 1496 drawn due to the limited evidence from a small number of studies with wide variation in study
 1497 design, dietary assessment methodology, and bone health outcomes. **DGAC Grade: Adults –**
 1498 **Limited; Children and Adolescents - Grade not assignable**

1499

1500 **Implications**

1501 Only limited evidence is available on the relationships between *dietary patterns* and bone health
 1502 outcomes in adults and other age groups. Although there is strong evidence on the roles of
 1503 vitamin D and calcium in bone health across the age spectrum, further research is needed on
 1504 dietary patterns that are most beneficial.

1505

1506 **Review of the Evidence**

1507 This systematic review included two articles that used data from RCTs and 11 articles from
 1508 prospective cohort studies published since 2000 that examined the relationship between dietary
 1509 patterns and bone health.²⁰⁷⁻²¹⁹

1510

1511 The articles employ diverse methodologies to assess dietary patterns. Four articles used an index
 1512 or score, six articles used factor analysis/principal components analysis, two articles used
 1513 reduced rank regression, and two articles tested dietary patterns in an intervention study where
 1514 bone health or fractures were either secondary or tertiary trial outcomes. Seven studies assessed
 1515 risk of fracture, six studies assessed bone mineral density, bone mineral content, or bone mass,
 1516 and one study examined risk of osteoporosis. The dietary patterns examined in this systematic
 1517 review were defined in various ways, making comparisons between articles difficult. However,
 1518 despite heterogeneity in this body of evidence, some common characteristics of dietary patterns

1519 associated with better or adverse bone health outcomes emerged, particularly in articles where
 1520 patterns were defined by index or score. Articles using data-driven methods were less consistent.
 1521 The following overall conclusions can be drawn:

1522

- 1523 • Patterns emphasizing vegetables, fruits, legumes, nuts, dairy, and
 1524 cereals/grains/pasta/rice, and unsaturated fats were generally associated with more
 1525 favorable bone health outcomes.
- 1526 • Patterns higher in meats and saturated fats were generally associated with increased risk
 1527 of adverse bone health outcomes.
- 1528 • Results were far less consistent for added sugars, alcohol, and sodium in relation to bone
 1529 health.

1530 Although many cohort studies make extensive efforts to include participants across a wide range
 1531 of race/ethnic groups and across the socio-economic continuum, there still may be some groups
 1532 for which the association between dietary patterns and bone health cannot yet be determined
 1533 (i.e., children, adolescents).

1534

1535 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3360>

1536

1537

1538 **CHAPTER SUMMARY**

1539 The dietary patterns approach captures the relationship between the overall diet and its
 1540 constituent foods, beverages, and nutrients in relationship to outcomes of interest. Numerous
 1541 dietary patterns were identified, with the most common ones defined using indices or scores such
 1542 as the HEI-2010, the AHEI-2010, or various Mediterranean-style dietary patterns, the DASH
 1543 pattern, vegetarian patterns, and data-driven approaches.

1544

1545 The Committee’s examination of the association between dietary patterns and various health
 1546 outcomes revealed remarkable consistency in the findings and implications that are noteworthy.
 1547 When looking at the dietary pattern conclusion statements across the various health outcomes,
 1548 certain characteristics of the diet were consistently identified (see Table D2.3). Common
 1549 characteristics of dietary patterns associated with positive health outcomes include higher intake
 1550 of vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and nuts; moderate
 1551 intake of alcohol (among adults); lower consumption of red and processed meat, and low intake
 1552 of sugar-sweetened foods and drinks, and refined grains. Vegetables and fruits are the only
 1553 characteristics of the diet that were consistently identified in every conclusion statement across
 1554 the health outcomes. Whole grains were identified slightly less consistently compared to
 1555 vegetables and fruits, but were identified in every conclusion with moderate to strong evidence.
 1556 For studies with limited evidence, grains were not as consistently defined and/or they were not

1557 identified as a key characteristic. Low- or non-fat dairy, seafood, legumes, nuts, and alcohol
1558 were identified as beneficial characteristics of the diet for some, but not all, outcomes. For
1559 conclusions with moderate to strong evidence, higher intake of red and processed meats was
1560 identified as detrimental compared to lower intake. Higher consumption of sugar-sweetened
1561 foods and beverages as well as refined grains were identified as detrimental in almost all
1562 conclusion statements with moderate to strong evidence.
1563
1564

1565 **Table D2.3. Description of the dietary patterns highlighted in the DGAC’s Conclusion Statements that are associated with benefit related**
 1566 **to the health outcome of interest. (Note: The reader is directed to the full Conclusion Statement above for more information on the relationship between dietary**
 1567 **patterns and the health outcome. In some cases, dietary components were associated with increased health risk and this is noted in the table.)**
 1568

Health Outcome	DGAC Grade ^a	Description of the Dietary Pattern Associated with Beneficial Health Outcomes
Cardiovascular disease	Strong	Dietary patterns characterized by higher consumption of <i>vegetables, fruits, whole grains, low-fat dairy, and seafood</i> , and lower consumption of <i>red and processed meat</i> , and lower intakes of <i>refined grains</i> , and <i>sugar-sweetened foods and beverages</i> relative to less healthy patterns; regular consumption of <i>nuts and legumes</i> ; moderate consumption of <i>alcohol</i> ; lower in <i>saturated fat, cholesterol, and sodium</i> and richer in <i>fiber, potassium, and unsaturated fats</i> .
Measures of body weight or obesity	Moderate	Dietary patterns that are higher in <i>vegetables, fruits, and whole grains</i> ; include <i>seafood and legumes</i> ; are moderate in <i>dairy products (particularly low and non-fat dairy)</i> and <i>alcohol</i> ; lower in <i>meats (including red and processed meats)</i> , and low in <i>sugar-sweetened foods and beverages, and refined grains</i> ; higher intakes of <i>unsaturated fats</i> and lower intakes of <i>saturated fats, cholesterol, and sodium</i> .
	Limited	Dietary patterns in childhood or adolescence that are higher in energy-dense and low-fiber foods, such as <i>sweets, refined grains, and processed meats</i> , as well as <i>sugar-sweetened beverages, whole milk, fried potatoes, certain fats and oils, and fast foods</i> are associated with an increased risk.
Type 2 diabetes	Moderate	Dietary patterns higher in <i>vegetables, fruits, and whole grains</i> and lower in <i>red and processed meats, high-fat dairy products, refined grains, and sweets/sugar-sweetened beverages</i> .
Cancer	Moderate	Colon/Rectal Cancer: Dietary patterns that are higher in <i>vegetables, fruits, legumes, whole grains, lean meats/seafood, and low-fat dairy</i> and moderate in <i>alcohol</i> ; and low in <i>red and/or processed meats, saturated fat, and sodas/sweets</i> . (Conversely, diets that are higher in <i>red/processed meats, French fries/potatoes, and sources of sugars (i.e., sodas, sweets, and dessert foods)</i> are associated with a greater risk.)
	Moderate (post) / Limited (pre)	Breast Cancer: Dietary patterns rich in <i>vegetables, fruit, and whole grains</i> , and lower in <i>animal products and refined carbohydrate</i> .
	Limited	Lung Cancer: Dietary patterns containing more frequent servings of <i>vegetables, fruits, seafood, grains/cereals, and legumes</i> , and <i>lean versus higher fat meats and lower fat or non-fat dairy products</i> .
	Not assignable	Prostate Cancer: N/A
Congenital anomalies	Limited – Neural tube defects	Neural tube defects: Dietary patterns during the preconception period that are higher in <i>vegetables, fruits, and grains</i> , and lower in <i>red and processed meats</i> , and low in <i>sweets</i> .
	Not assignable	Congenital heart defects or cleft lip/palate: N/A
Neurological and psychological illnesses	Limited	Age-related cognitive impairment, dementia, and/or Alzheimer’s disease: Dietary patterns containing an array of <i>vegetables, fruits, nuts, legumes and seafood</i> .
	Limited	Depression: Dietary patterns emphasizing <i>seafood, vegetables, fruits, nuts, and legumes</i> .
Bone health	Limited	Adults: Dietary patterns higher in <i>vegetables, fruits, grains, nuts, and dairy products</i> , and lower in <i>meats and saturated fat</i> .
	Not assignable	Children: N/A

1569 ^a The DGAC Grade presented represents the grade the Committee provided for the conclusion statement with the dietary pattern components described. Some health outcomes had
 1570 more than one graded conclusion. Only the conclusion statements that describe dietary pattern components are presented here. Post = Post-menopausal; Pre = Pre-menopausal

1571 As alcohol is a unique aspect of the diet, the DGAC considered evidence from several sources to
 1572 inform recommendations. As noted above, moderate alcohol intake among adults was identified
 1573 as a component of a healthy dietary pattern associated with some health outcomes, which
 1574 reaffirms conclusions related to moderate alcohol consumption by the 2010 DGAC. The
 1575 Committee also concurs with the conclusions reached by the 2010 DGAC on the relationship
 1576 between alcohol intake and unintentional injury and lactation.¹ However, as noted in Table D2.1,
 1577 evidence also suggests that alcoholic drinks are associated with increased risk for certain cancers,
 1578 including pre- and post-menopausal breast cancer. After consideration of this collective
 1579 evidence, the Committee concurs with the 2010 DGAC that if alcohol is consumed, it should be
 1580 consumed in moderation, and only by adults. However, it is not recommended that anyone begin
 1581 drinking or drink more frequently on the basis of potential health benefits because moderate
 1582 alcohol intake also is associated with increased risk of violence, drowning, and injuries from falls
 1583 and motor vehicle crashes. Women should be aware of a moderately increased risk of breast
 1584 cancer even with moderate alcohol intake. There are many circumstances in which people should
 1585 not drink alcohol:

- 1586 • Individuals who cannot restrict their drinking to moderate levels.
- 1587 • Anyone younger than the legal drinking age.
- 1588 • Women who are pregnant or who may be pregnant.
- 1589 • Individuals taking prescription or over-the-counter medications that can interact with
 1590 alcohol.
- 1591 • Individuals with certain specific medical conditions (e.g., liver disease,
 1592 hypertriglyceridemia, pancreatitis).
- 1593 • Individuals who plan to drive, operate machinery, or take part in other activities that
 1594 require attention, skill, or coordination or in situations where impaired judgment could
 1595 cause injury or death (e.g., swimming).

1596 Finally, because of the substantial evidence clearly demonstrating the health benefits of
 1597 breastfeeding, occasionally consuming an alcoholic drink does not warrant stopping
 1598 breastfeeding. However, women who are breastfeeding should be very cautious about drinking
 1599 alcohol, if they choose to drink at all.^{§§}

1600
 1601 The common characteristics of a healthy dietary pattern found in the conclusion statements
 1602 across the outcomes examined implies that following a dietary pattern associated with reduced
 1603 risk of CVD, overweight, and obesity will have positive health benefits beyond these categories

^{§§} If the infant's breastfeeding behavior is well established, consistent, and predictable (no earlier than at 3 months of age), a mother may consume a single alcoholic drink if she then waits at least 4 hours before breastfeeding. Alternatively, she may express breast milk before consuming the drink and feed the expressed milk to her infant later.

1604 of health outcomes. Thus, the U.S. population should be encouraged and guided to consume
 1605 dietary patterns that are rich in vegetables, fruits, whole grains, seafood, legumes, and nuts;
 1606 moderate in low- and non-fat dairy products and alcohol (among adults); lower in red and
 1607 processed meat; and low in sugar-sweetened foods and beverages and refined grains. These
 1608 dietary patterns can be achieved in many ways and should be tailored to the individual's
 1609 biological and medical needs as well as socio-cultural preferences. As described in the DGAC's
 1610 conceptual model, a multi-level process at individual and population levels is required to help
 1611 achieve a healthy diet and other lifestyle behaviors so as to achieve chronic disease risk
 1612 reduction and overall well-being. The Committee recommends the development and
 1613 implementation of programs and services that facilitate the improvement in eating behaviors
 1614 consistent with healthy dietary patterns in various settings, including preventive services in our
 1615 healthcare and public health systems as well as those that reach populations in other settings of
 1616 influence such as preschool and school settings and workplaces.

1617
 1618 The dietary pattern characteristics being recommended by the 2015 DGAC reaffirms the dietary
 1619 pattern characteristics recommended by the 2010 DGAC, despite the fact that different
 1620 approaches were employed. Additionally, this dietary pattern aligns with recommendations from
 1621 other groups, including AICR and AHA/ACC. The majority of evidence considered focuses on
 1622 dietary patterns consumed in adulthood on health risks, primarily risks of chronic disease
 1623 development and, in the case of pregnancy, birth defects. Very little evidence considered here
 1624 was directed to dietary patterns in children, and risk reduction studies evaluating children's diets
 1625 and risk of overweight and obesity provided limited evidence. No conclusions on chronic disease
 1626 apply directly to evidence developed in children. Recommendations based on adult studies have
 1627 implications for children based on general nutritional principles but caution is warranted,
 1628 considering the fact that children with developing bodies and neurocognitive capabilities present
 1629 unique nutritional issues.

1630

1631

1632 **NEEDS FOR FUTURE RESEARCH**

- 1633 1. Conduct additional dietary patterns research for other health outcomes to strengthen the
 1634 evidence beyond CVD and body weight in populations of various ethnic backgrounds and
 1635 life course stages in order for future DGACs to draw stronger conclusions.

1636

1637 **Rationale:** The NEL systematic reviews demonstrated that considerable CVD research
 1638 related to dietary patterns is available. However, it also is important to note, that unlike CVD,
 1639 some of the other health outcomes are more heterogeneous and thus may require greater
 1640 specificity in the examination of diet and disease risk. There is a clear need for all studies
 1641 examining the relationship between dietary patterns and health outcomes to include the full
 1642 age spectrum and to take a life course perspective (including pregnancy); insufficient
 1643 research is being devoted to children and how diseases may evolve over time. An increased

1644 emphasis should be placed on understanding how the diets of all those in the U.S. population
 1645 from various ethnic backgrounds may be associated with health outcomes, thereby
 1646 broadening knowledge beyond Hispanics and African Americans to include the diversity that
 1647 exists in the United States today. This may require our national nutrition monitoring
 1648 programs to over-sample individuals from other national origins to conduct subgroup
 1649 analysis.

1650

1651 2. Improve the understanding of how to more precisely characterize dietary patterns by their
 1652 food constituents and the implications of the food constituents on nutrient adequacy through
 1653 the use of Food Pattern Modeling. More precise characterization, particularly of protein
 1654 foods, is needed.

1655

1656 **Rationale:** Researchers are characterizing dietary patterns very differently and yet
 1657 sometimes use similar nomenclatures. This makes it difficult to compare results across
 1658 studies and as demonstrated in the NEL systematic reviews, can impair the grading of the
 1659 body of evidence as strong. The reason why researchers are not replicating others findings in
 1660 different populations may be a function of publication bias. It is important for editors of
 1661 scientific journals and peer reviewers to appreciate the replication of findings first and then
 1662 value a research group's methodological nuance that may improve the examination of the
 1663 association between dietary patterns and health outcomes. Perhaps what should be stressed is
 1664 a harmonization of research methods across various cohorts or randomized trials, similar to
 1665 what is being done at the National Cancer Institute's Dietary Patterns Methods Project^{9, 220}
 1666 led by Drs. Krebs-Smith and Reedy. The use of Food Pattern Modeling as demonstrated in
 1667 ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***
 1668 allows questions about the adequacy of the dietary patterns given specific food constituents
 1669 to be addressed and how modifications of the patterns by altering the foods for specific
 1670 population groups or to meet specific nutrient targets can be achieved.

1671

1672 3. Examine the long-term cardio-metabolic effects of the various dietary patterns identified in
 1673 the *AHA/ACC/TOS Guidelines for the Management of Overweight and Obesity in Adults* that
 1674 are capable of resulting in short-term weight loss (see Question 2, above).

1675

1676 **Rationale:** Although the research to date demonstrates that to lose weight, a variety of
 1677 dietary pattern approaches can be used if a reduction in caloric intake is achieved, the long-
 1678 term effects of these diets on cardio-metabolic health are not well known. Emerging research
 1679 is exploring health effects of variations of the low-carbohydrate, higher protein/fat dietary
 1680 pattern. In some approaches (such as Atkins), the dietary pattern which emphasizes animal
 1681 products, may achieve a macronutrient composition that is higher in saturated fat. Others
 1682 may emphasize plant-based proteins and fats and may achieve a lower saturated fat content
 1683 and may be higher in polyunsaturated fats and dietary fiber. Research is needed to determine

1684 the impact of these alternative approaches, and perhaps others, on CVD risk profiles as well
 1685 as other health outcomes. As mentioned in the review of the literature associated with
 1686 saturated fat and cardiovascular disease in *Part D. Chapter 6: Cross-Cutting Topics of*
 1687 *Public Health Importance*, substituting one macronutrient for another may result in
 1688 unintended consequences. Careful consideration to the types of foods that are used in these
 1689 diets and in particular the type of fat and amount of added sugars should be taken into
 1690 account.

1691
 1692

1693 REFERENCES

- 1694 1. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory
 1695 Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of
 1696 Agriculture and the Secretary of Health and Human Services: U.S. Department of
 1697 Agriculture, Agricultural Research Service, Washington D.C.; 2010. Available from:
 1698 <http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm>.
- 1699 2. Nutrition Evidence Library. A series of systematic reviews on the relationship between
 1700 dietary patterns and health outcomes. Alexandria, VA: U.S. Department of Agriculture,
 1701 Center for Nutrition Policy and Promotion; 2014. Available from:
 1702 <http://www.nel.gov/vault/2440/web/files/DietaryPatterns/DPRptFullFinal.pdf>.
- 1703 3. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al.
 1704 Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet.* 2013;113(4):569-80.
 1705 PMID: 23415502. <http://www.ncbi.nlm.nih.gov/pubmed/23415502>.
- 1706 4. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough ML, Wang M, et al. Alternative
 1707 dietary indices both strongly predict risk of chronic disease. *J Nutr.* 2012;142(6):1009-18.
 1708 PMID: 22513989. <http://www.ncbi.nlm.nih.gov/pubmed/22513989>.
- 1709 5. Kant AK, Schatzkin A, Graubard BI, Schairer C. A prospective study of diet quality and
 1710 mortality in women. *JAMA.* 2000;283(16):2109-15. PMID: 10791502.
 1711 <http://www.ncbi.nlm.nih.gov/pubmed/10791502>.
- 1712 6. Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence
 1713 to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern*
 1714 *Med.* 2008;168(7):713-20. PMID: 18413553.
 1715 <http://www.ncbi.nlm.nih.gov/pubmed/18413553>.
- 1716 7. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean
 1717 diet and survival in a Greek population. *N Engl J Med.* 2003;348(26):2599-608. PMID:
 1718 12826634. <http://www.ncbi.nlm.nih.gov/pubmed/12826634>.
- 1719 8. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean
 1720 diet and incidence of and mortality from coronary heart disease and stroke in women.
 1721 *Circulation.* 2009;119(8):1093-100. PMID: 19221219.
 1722 <http://www.ncbi.nlm.nih.gov/pubmed/19221219>.
- 1723 9. Reedy J, Krebs-Smith SM, Miller PE, Liese AD, Kahle LL, Park Y, et al. Higher diet
 1724 quality is associated with decreased risk of all-cause, cardiovascular disease, and cancer
 1725 mortality among older adults. *J Nutr.* 2014;144(6):881-9. PMID: 24572039.
 1726 <http://www.ncbi.nlm.nih.gov/pubmed/24572039>.

- 1727 10. National Heart Lung and Blood Institute. Lifestyle Interventions to Reduce
1728 Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group.
1729 Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of
1730 Health; 2013. Available from: [http://www.nhlbi.nih.gov/health-pro/guidelines/in-](http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm)
1731 [develop/cardiovascular-risk-reduction/lifestyle/index.htm](http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm).
- 1732 11. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013
1733 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of
1734 the American College of Cardiology/American Heart Association Task Force on Practice
1735 Guidelines. *J Am Coll Cardiol*. 2014;63(25 Pt B):2960-84. PMID: 24239922.
1736 <http://www.ncbi.nlm.nih.gov/pubmed/24239922>.
- 1737 12. National Heart Lung and Blood Institute. Managing overweight and obesity in adults:
1738 Systematic evidence review from the Obesity Expert Panel, 2013. Bethesda, MD: U.S.
1739 Department of Health and Human Services, National Institutes of Health; 2013.
1740 Available from: <http://www.nhlbi.nih.gov/guidelines/obesity/ser/index.htm>.
- 1741 13. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013
1742 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a
1743 report of the American College of Cardiology/American Heart Association Task Force on
1744 Practice Guidelines and The Obesity Society. *J Am Coll Cardiol*. 2014;63(25 Pt B):2985-
1745 3023. PMID: 24239920. <http://www.ncbi.nlm.nih.gov/pubmed/24239920>.
- 1746 14. Huang T, Yang B, Zheng J, Li G, Wahlqvist ML, Li D. Cardiovascular disease mortality
1747 and cancer incidence in vegetarians: a meta-analysis and systematic review. *Ann Nutr*
1748 *Metab*. 2012;60(4):233-40. PMID: 22677895.
1749 <http://www.ncbi.nlm.nih.gov/pubmed/22677895>.
- 1750 15. Martinez-Gonzalez MA, Bes-Rastrollo M. Dietary patterns, Mediterranean diet, and
1751 cardiovascular disease. *Curr Opin Lipidol*. 2014;25(1):20-6. PMID: 24370845.
1752 <http://www.ncbi.nlm.nih.gov/pubmed/24370845>.
- 1753 16. Rees K, Hartley L, Flowers N, Clarke A, Hooper L, Thorogood M, et al. 'Mediterranean'
1754 dietary pattern for the primary prevention of cardiovascular disease. *Cochrane Database*
1755 *Syst Rev*. 2013;8:CD009825. PMID: 23939686.
1756 <http://www.ncbi.nlm.nih.gov/pubmed/23939686>.
- 1757 17. Salehi-Abargouei A, Maghsoudi Z, Shirani F, Azadbakht L. Effects of Dietary
1758 Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular
1759 diseases--incidence: a systematic review and meta-analysis on observational prospective
1760 studies. *Nutrition*. 2013;29(4):611-8. PMID: 23466047.
1761 <http://www.ncbi.nlm.nih.gov/pubmed/23466047>.
- 1762 18. Sofi F, Macchi C, Abbate R, Gensini GF, Casini A. Mediterranean diet and health status:
1763 an updated meta-analysis and a proposal for a literature-based adherence score. *Public*
1764 *Health Nutr*. 2013:1-14. PMID: 24476641.
1765 <http://www.ncbi.nlm.nih.gov/pubmed/24476641>.
- 1766 19. Yokoyama Y, Nishimura K, Barnard ND, Takegami M, Watanabe M, Sekikawa A, et al.
1767 Vegetarian diets and blood pressure: a meta-analysis. *JAMA Intern Med*.
1768 2014;174(4):577-87. PMID: 24566947. <http://www.ncbi.nlm.nih.gov/pubmed/24566947>.
- 1769 20. Dauchet L, Kesse-Guyot E, Czernichow S, Bertrais S, Estaquio C, Péneau S, et al.
1770 Dietary patterns and blood pressure change over 5-y follow-up in the SU.VI.MAX
1771 cohort. *Am J Clin Nutr*. 2007;85(6):1650-6. PMID: 17556705.
1772 <http://www.ncbi.nlm.nih.gov/pubmed/17556705>.

- 1773 21. Camões M, Oliveira A, Pereira M, Severo M, Lopes C. Role of physical activity and diet
1774 in incidence of hypertension: a population-based study in Portuguese adults. *Eur J Clin*
1775 *Nutr.* 2010;64(12):1441-9. PMID: 20808327.
1776 <http://www.ncbi.nlm.nih.gov/pubmed/20808327>.
- 1777 22. Folsom AR, Parker ED, Harnack LJ. Degree of concordance with DASH diet guidelines
1778 and incidence of hypertension and fatal cardiovascular disease. *Am J Hypertens.*
1779 2007;20(3):225-32. PMID: 17324731. <http://www.ncbi.nlm.nih.gov/pubmed/17324731>.
- 1780 23. Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, Ruiz-Gutiérrez V, Covas
1781 MI, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a
1782 randomized trial. *Ann Intern Med.* 2006;145(1):1-11. PMID: 16818923.
1783 <http://www.ncbi.nlm.nih.gov/pubmed/16818923>.
- 1784 24. Jula A, Marniemi J, Huupponen R, Virtanen A, Rastas M, Rönnemaa T. Effects of diet
1785 and simvastatin on serum lipids, insulin, and antioxidants in hypercholesterolemic men: a
1786 randomized controlled trial. *JAMA.* 2002;287(5):598-605. PMID: 11829698.
1787 <http://www.ncbi.nlm.nih.gov/pubmed/11829698>.
- 1788 25. Núñez-Córdoba JM, Valencia-Serrano F, Toledo E, Alonso A, Martínez-González MA.
1789 The Mediterranean diet and incidence of hypertension: the Seguimiento Universidad de
1790 Navarra (SUN) Study. *Am J Epidemiol.* 2009;169(3):339-46. PMID: 19037007.
1791 <http://www.ncbi.nlm.nih.gov/pubmed/19037007>.
- 1792 26. Burr ML, Butland BK. Heart disease in British vegetarians. *Am J Clin Nutr.* 1988;48(3
1793 Suppl):830-2. PMID: 3414590. <http://www.ncbi.nlm.nih.gov/pubmed/3414590>.
- 1794 27. Margetts BM, Beilin LJ, Armstrong BK, Vandongen R. A randomized control trial of a
1795 vegetarian diet in the treatment of mild hypertension. *Clin Exp Pharmacol Physiol.*
1796 1985;12(3):263-6. PMID: 3896594. <http://www.ncbi.nlm.nih.gov/pubmed/3896594>.
- 1797 28. Crowe FL, Appleby PN, Travis RC, Key TJ. Risk of hospitalization or death from
1798 ischemic heart disease among British vegetarians and nonvegetarians: results from the
1799 EPIC-Oxford cohort study. *Am J Clin Nutr.* 2013;97(3):597-603. PMID: 23364007.
1800 <http://www.ncbi.nlm.nih.gov/pubmed/23364007>.
- 1801 29. Zamora D, Gordon-Larsen P, He K, Jacobs DR, Shikany JM, Popkin BM. Are the 2005
1802 Dietary Guidelines for Americans Associated With reduced risk of type 2 diabetes and
1803 cardiometabolic risk factors? Twenty-year findings from the CARDIA study. *Diabetes*
1804 *Care.* 2011;34(5):1183-5. PMID: 21478463.
1805 <http://www.ncbi.nlm.nih.gov/pubmed/21478463>.
- 1806 30. van de Laar RJ, Stehouwer CD, van Bussel BC, Prins MH, Twisk JW, Ferreira I.
1807 Adherence to a Mediterranean dietary pattern in early life is associated with lower arterial
1808 stiffness in adulthood: the Amsterdam Growth and Health Longitudinal Study. *J Intern*
1809 *Med.* 2013;273(1):79-93. PMID: 22809371.
1810 <http://www.ncbi.nlm.nih.gov/pubmed/22809371>.
- 1811 31. Rumawas ME, Meigs JB, Dwyer JT, McKeown NM, Jacques PF. Mediterranean-style
1812 dietary pattern, reduced risk of metabolic syndrome traits, and incidence in the
1813 Framingham Offspring Cohort. *Am J Clin Nutr.* 2009;90(6):1608-14. PMID: 19828705.
1814 <http://www.ncbi.nlm.nih.gov/pubmed/19828705>.
- 1815 32. Tortosa A, Bes-Rastrollo M, Sanchez-Villegas A, Basterra-Gortari FJ, Nuñez-Cordoba
1816 JM, Martinez-Gonzalez MA. Mediterranean diet inversely associated with the incidence
1817 of metabolic syndrome: the SUN prospective cohort. *Diabetes Care.* 2007;30(11):2957-9.
1818 PMID: 17712023. <http://www.ncbi.nlm.nih.gov/pubmed/17712023>.

- 1819 33. Estruch R, Ros E, Martínez-González MA. Mediterranean diet for primary prevention of
1820 cardiovascular disease. *N Engl J Med.* 2013;369(7):676-7. PMID: 23944307.
1821 <http://www.ncbi.nlm.nih.gov/pubmed/23944307>.
- 1822 34. Ambrosini GL. Childhood dietary patterns and later obesity: a review of the evidence.
1823 *Proc Nutr Soc.* 2014;73(1):137-46. PMID: 24280165.
1824 <http://www.ncbi.nlm.nih.gov/pubmed/24280165>.
- 1825 35. Esposito K, Kastorini CM, Panagiotakos DB, Giugliano D. Mediterranean diet and
1826 weight loss: meta-analysis of randomized controlled trials. *Metab Syndr Relat Disord.*
1827 2011;9(1):1-12. PMID: 20973675. <http://www.ncbi.nlm.nih.gov/pubmed/20973675>.
- 1828 36. Beunza JJ, Toledo E, Hu FB, Bes-Rastrollo M, Serrano-Martínez M, Sánchez-Villegas A,
1829 et al. Adherence to the Mediterranean diet, long-term weight change, and incident
1830 overweight or obesity: the Seguimiento Universidad de Navarra (SUN) cohort. *Am J Clin*
1831 *Nutr.* 2010;92(6):1484-93. PMID: 20962161.
1832 <http://www.ncbi.nlm.nih.gov/pubmed/20962161>.
- 1833 37. Mendez MA, Popkin BM, Jakszyn P, Berenguer A, Tormo MJ, Sánchez MJ, et al.
1834 Adherence to a Mediterranean diet is associated with reduced 3-year incidence of obesity.
1835 *J Nutr.* 2006;136(11):2934-8. PMID: 17056825.
1836 <http://www.ncbi.nlm.nih.gov/pubmed/17056825>.
- 1837 38. Romaguera D, Norat T, Vergnaud AC, Mouw T, May AM, Agudo A, et al.
1838 Mediterranean dietary patterns and prospective weight change in participants of the
1839 EPIC-PANACEA project. *Am J Clin Nutr.* 2010;92(4):912-21. PMID: 20810975.
1840 <http://www.ncbi.nlm.nih.gov/pubmed/20810975>.
- 1841 39. Berz JP, Singer MR, Guo X, Daniels SR, Moore LL. Use of a DASH food group score to
1842 predict excess weight gain in adolescent girls in the National Growth and Health Study.
1843 *Arch Pediatr Adolesc Med.* 2011;165(6):540-6. PMID: 21646587.
1844 <http://www.ncbi.nlm.nih.gov/pubmed/21646587>.
- 1845 40. Cheng G, Gerlach S, Libuda L, Kranz S, Günther AL, Karaolis-Danckert N, et al. Diet
1846 quality in childhood is prospectively associated with the timing of puberty but not with
1847 body composition at puberty onset. *J Nutr.* 2010;140(1):95-102. PMID: 19923386.
1848 <http://www.ncbi.nlm.nih.gov/pubmed/19923386>.
- 1849 41. Gao SK, Beresford SA, Frank LL, Schreiner PJ, Burke GL, Fitzpatrick AL.
1850 Modifications to the Healthy Eating Index and its ability to predict obesity: the Multi-
1851 Ethnic Study of Atherosclerosis. *Am J Clin Nutr.* 2008;88(1):64-9. PMID: 18614725.
1852 <http://www.ncbi.nlm.nih.gov/pubmed/18614725>.
- 1853 42. Kesse-Guyot E, Castetbon K, Estaquio C, Czernichow S, Galan P, Hercberg S.
1854 Association between the French nutritional guideline-based score and 6-year
1855 anthropometric changes in a French middle-aged adult cohort. *Am J Epidemiol.*
1856 2009;170(6):757-65. PMID: 19656810. <http://www.ncbi.nlm.nih.gov/pubmed/19656810>.
- 1857 43. Zamora D, Gordon-Larsen P, Jacobs DR, Popkin BM. Diet quality and weight gain
1858 among black and white young adults: the Coronary Artery Risk Development in Young
1859 Adults (CARDIA) Study (1985-2005). *Am J Clin Nutr.* 2010;92(4):784-93. PMID:
1860 20685947. <http://www.ncbi.nlm.nih.gov/pubmed/20685947>.
- 1861 44. Lassale C, Fezeu L, Andreeva VA, Hercberg S, Kengne AP, Czernichow S, et al.
1862 Association between dietary scores and 13-year weight change and obesity risk in a
1863 French prospective cohort. *Int J Obes (Lond).* 2012;36(11):1455-62. PMID: 22249228.
1864 <http://dx.doi.org/10.1038/ijo.2011.264>.

- 1865 45. Woo J, Cheung B, Ho S, Sham A, Lam TH. Influence of dietary pattern on the
1866 development of overweight in a Chinese population. *Eur J Clin Nutr.* 2008;62(4):480-7.
1867 PMID: 17327865. <http://www.ncbi.nlm.nih.gov/pubmed/17327865>.
- 1868 46. Jacobs DR, Sluik D, Rokling-Andersen MH, Anderssen SA, Drevon CA. Association of
1869 1-y changes in diet pattern with cardiovascular disease risk factors and adipokines: results
1870 from the 1-y randomized Oslo Diet and Exercise Study. *Am J Clin Nutr.* 2009;89(2):509-
1871 17. PMID: 19116328. <http://www.ncbi.nlm.nih.gov/pubmed/19116328>.
- 1872 47. Duffey KJ, Steffen LM, Van Horn L, Jacobs DR, Popkin BM. Dietary patterns matter:
1873 diet beverages and cardiometabolic risks in the longitudinal Coronary Artery Risk
1874 Development in Young Adults (CARDIA) Study. *Am J Clin Nutr.* 2012;95(4):909-15.
1875 PMID: 22378729. <http://www.ncbi.nlm.nih.gov/pubmed/22378729>.
- 1876 48. Hosseini-Esfahani F, Djazaieri SA, Mirmiran P, Mehrabi Y, Azizi F. Which food patterns
1877 are predictors of obesity in Tehranian adults? *J Nutr Educ Behav.* 2012;44(6):564-73.
1878 PMID: 21652267. <http://www.ncbi.nlm.nih.gov/pubmed/21652267>.
- 1879 49. McNaughton SA, Mishra GD, Stephen AM, Wadsworth ME. Dietary patterns throughout
1880 adult life are associated with body mass index, waist circumference, blood pressure, and
1881 red cell folate. *J Nutr.* 2007;137(1):99-105. PMID: 17182808.
1882 <http://www.ncbi.nlm.nih.gov/pubmed/17182808>.
- 1883 50. Newby PK, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and
1884 changes in body mass index and waist circumference in adults. *Am J Clin Nutr.*
1885 2003;77(6):1417-25. PMID: 12791618. <http://www.ncbi.nlm.nih.gov/pubmed/12791618>.
- 1886 51. Newby PK, Muller D, Hallfrisch J, Andres R, Tucker KL. Food patterns measured by
1887 factor analysis and anthropometric changes in adults. *Am J Clin Nutr.* 2004;80(2):504-13.
1888 PMID: 15277177. <http://www.ncbi.nlm.nih.gov/pubmed/15277177>.
- 1889 52. Togo P, Osler M, Sørensen TI, Heitmann BL. A longitudinal study of food intake
1890 patterns and obesity in adult Danish men and women. *Int J Obes Relat Metab Disord.*
1891 2004;28(4):583-93. PMID: 14770197. <http://www.ncbi.nlm.nih.gov/pubmed/14770197>.
- 1892 53. Boggs DA, Palmer JR, Spiegelman D, Stampfer MJ, Adams-Campbell LL, Rosenberg L.
1893 Dietary patterns and 14-y weight gain in African American women. *Am J Clin Nutr.*
1894 2011;94(1):86-94. PMID: 21593501. <http://www.ncbi.nlm.nih.gov/pubmed/21593501>.
- 1895 54. Newby PK, Weismayer C, Akesson A, Tucker KL, Wolk A. Longitudinal changes in
1896 food patterns predict changes in weight and body mass index and the effects are greatest
1897 in obese women. *J Nutr.* 2006;136(10):2580-7. PMID: 16988130.
1898 <http://www.ncbi.nlm.nih.gov/pubmed/16988130>.
- 1899 55. Quatromoni PA, Copenhafer DL, D'Agostino RB, Millen BE. Dietary patterns predict the
1900 development of overweight in women: The Framingham Nutrition Studies. *J Am Diet*
1901 *Assoc.* 2002;102(9):1239-46. PMID: 12792620.
1902 <http://www.ncbi.nlm.nih.gov/pubmed/12792620>.
- 1903 56. Ritchie LD, Spector P, Stevens MJ, Schmidt MM, Schreiber GB, Striegel-Moore RH, et
1904 al. Dietary patterns in adolescence are related to adiposity in young adulthood in black
1905 and white females. *J Nutr.* 2007;137(2):399-406. PMID: 17237318.
1906 <http://www.ncbi.nlm.nih.gov/pubmed/17237318>.
- 1907 57. Schulze MB, Fung TT, Manson JE, Willett WC, Hu FB. Dietary patterns and changes in
1908 body weight in women. *Obesity (Silver Spring).* 2006;14(8):1444-53. PMID: 16988088.
1909 <http://www.ncbi.nlm.nih.gov/pubmed/16988088>.

- 1910 58. Alhazmi A, Stojanovski E, McEvoy M, Garg ML. The association between dietary
1911 patterns and type 2 diabetes: a systematic review and meta-analysis of cohort studies. *J*
1912 *Hum Nutr Diet.* 2014;27(3):251-60. PMID: 24102939.
1913 <http://www.ncbi.nlm.nih.gov/pubmed/24102939>.
- 1914 59. Liese AD, Nichols M, Sun X, D'Agostino RB, Haffner SM. Adherence to the DASH Diet
1915 is inversely associated with incidence of type 2 diabetes: the insulin resistance
1916 atherosclerosis study. *Diabetes Care.* 2009;32(8):1434-6. PMID: 19487638.
1917 <http://www.ncbi.nlm.nih.gov/pubmed/19487638>.
- 1918 60. Martínez-González MA, de la Fuente-Arrillaga C, Nunez-Cordoba JM, Basterra-Gortari
1919 FJ, Beunza JJ, Vazquez Z, et al. Adherence to Mediterranean diet and risk of developing
1920 diabetes: prospective cohort study. *BMJ.* 2008;336(7657):1348-51. PMID: 18511765.
1921 <http://www.ncbi.nlm.nih.gov/pubmed/18511765>.
- 1922 61. Rossi M, Turati F, Lagiou P, Trichopoulos D, Augustin LS, La Vecchia C, et al.
1923 Mediterranean diet and glycaemic load in relation to incidence of type 2 diabetes: results
1924 from the Greek cohort of the population-based European Prospective Investigation into
1925 Cancer and Nutrition (EPIC). *Diabetologia.* 2013;56(11):2405-13. PMID: 23975324.
1926 <http://www.ncbi.nlm.nih.gov/pubmed/23975324>.
- 1927 62. Abiemo EE, Alonso A, Nettleton JA, Steffen LM, Bertoni AG, Jain A, et al.
1928 Relationships of the Mediterranean dietary pattern with insulin resistance and diabetes
1929 incidence in the Multi-Ethnic Study of Atherosclerosis (MESA). *Br J Nutr.*
1930 2013;109(8):1490-7. PMID: 22932232. <http://www.ncbi.nlm.nih.gov/pubmed/22932232>.
- 1931 63. Fung TT, McCullough M, van Dam RM, Hu FB. A prospective study of overall diet
1932 quality and risk of type 2 diabetes in women. *Diabetes Care.* 2007;30(7):1753-7. PMID:
1933 17429059. <http://www.ncbi.nlm.nih.gov/pubmed/17429059>.
- 1934 64. Gopinath B, Rochtchina E, Flood VM, Mitchell P. Diet quality is prospectively
1935 associated with incident impaired fasting glucose in older adults. *Diabet Med.*
1936 2013;30(5):557-62. PMID: 23301551. <http://www.ncbi.nlm.nih.gov/pubmed/23301551>.
- 1937 65. von Ruesten A, Illner AK, Buijsse B, Heidemann C, Boeing H. Adherence to
1938 recommendations of the German food pyramid and risk of chronic diseases: results from
1939 the EPIC-Potsdam study. *Eur J Clin Nutr.* 2010;64(11):1251-9. PMID: 20717136.
1940 <http://www.ncbi.nlm.nih.gov/pubmed/20717136>.
- 1941 66. Malik VS, Fung TT, van Dam RM, Rimm EB, Rosner B, Hu FB. Dietary patterns during
1942 adolescence and risk of type 2 diabetes in middle-aged women. *Diabetes Care.*
1943 2012;35(1):12-8. PMID: 22074723. <http://www.ncbi.nlm.nih.gov/pubmed/22074723>.
- 1944 67. Hodge AM, English DR, O'Dea K, Giles GG. Dietary patterns and diabetes incidence in
1945 the Melbourne Collaborative Cohort Study. *Am J Epidemiol.* 2007;165(6):603-10.
1946 PMID: 17220476. <http://www.ncbi.nlm.nih.gov/pubmed/17220476>.
- 1947 68. Nanri A, Shimazu T, Takachi R, Ishihara J, Mizoue T, Noda M, et al. Dietary patterns
1948 and type 2 diabetes in Japanese men and women: the Japan Public Health Center-based
1949 Prospective Study. *Eur J Clin Nutr.* 2013;67(1):18-24. PMID: 23093343.
1950 <http://www.ncbi.nlm.nih.gov/pubmed/23093343>.
- 1951 69. Nettleton JA, Steffen LM, Ni H, Liu K, Jacobs DR. Dietary patterns and risk of incident
1952 type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes Care.*
1953 2008;31(9):1777-82. PMID: 18544792. <http://www.ncbi.nlm.nih.gov/pubmed/18544792>.

- 1954 70. Liese AD, Weis KE, Schulz M, Toozee JA. Food intake patterns associated with incident
1955 type 2 diabetes: the Insulin Resistance Atherosclerosis Study. *Diabetes Care*.
1956 2009;32(2):263-8. PMID: 19033409. <http://www.ncbi.nlm.nih.gov/pubmed/19033409>.
- 1957 71. McNaughton SA, Mishra GD, Brunner EJ. Dietary patterns, insulin resistance, and
1958 incidence of type 2 diabetes in the Whitehall II Study. *Diabetes Care*. 2008;31(7):1343-8.
1959 PMID: 18390803. <http://www.ncbi.nlm.nih.gov/pubmed/18390803>.
- 1960 72. Imamura F, Lichtenstein AH, Dallal GE, Meigs JB, Jacques PF. Generalizability of
1961 dietary patterns associated with incidence of type 2 diabetes mellitus. *Am J Clin Nutr*.
1962 2009;90(4):1075-83. PMID: 19710193. <http://www.ncbi.nlm.nih.gov/pubmed/19710193>.
- 1963 73. Adamsson V, Reumark A, Fredriksson IB, Hammarström E, Vessby B, Johansson G, et
1964 al. Effects of a healthy Nordic diet on cardiovascular risk factors in
1965 hypercholesterolaemic subjects: a randomized controlled trial (NORDIET). *J Intern Med*.
1966 2011;269(2):150-9. PMID: 20964740. <http://www.ncbi.nlm.nih.gov/pubmed/20964740>.
- 1967 74. Blumenthal JA, Babyak MA, Sherwood A, Craighead L, Lin PH, Johnson J, et al. Effects
1968 of the dietary approaches to stop hypertension diet alone and in combination with
1969 exercise and caloric restriction on insulin sensitivity and lipids. *Hypertension*.
1970 2010;55(5):1199-205. PMID: 20212264.
1971 <http://www.ncbi.nlm.nih.gov/pubmed/20212264>.
- 1972 75. Esposito K, Marfella R, Ciotola M, Di Palo C, Giugliano F, Giugliano G, et al. Effect of a
1973 mediterranean-style diet on endothelial dysfunction and markers of vascular
1974 inflammation in the metabolic syndrome: a randomized trial. *JAMA*. 2004;292(12):1440-
1975 6. PMID: 15383514. <http://www.ncbi.nlm.nih.gov/pubmed/15383514>.
- 1976 76. Gadgil MD, Appel LJ, Yeung E, Anderson CA, Sacks FM, Miller ER. The effects of
1977 carbohydrate, unsaturated fat, and protein intake on measures of insulin sensitivity:
1978 results from the OmniHeart trial. *Diabetes Care*. 2013;36(5):1132-7. PMID: 23223345.
1979 <http://www.ncbi.nlm.nih.gov/pubmed/23223345>.
- 1980 77. Rallidis LS, Lekakis J, Kolomvotsou A, Zampelas A, Vamvakou G, Efstathiou S, et al.
1981 Close adherence to a Mediterranean diet improves endothelial function in subjects with
1982 abdominal obesity. *Am J Clin Nutr*. 2009;90(2):263-8. PMID: 19515732.
1983 <http://www.ncbi.nlm.nih.gov/pubmed/19515732>.
- 1984 78. Salas-Salvadó J, Fernández-Ballart J, Ros E, Martínez-González MA, Fitó M, Estruch R,
1985 et al. Effect of a Mediterranean diet supplemented with nuts on metabolic syndrome
1986 status: one-year results of the PREDIMED randomized trial. *Arch Intern Med*.
1987 2008;168(22):2449-58. PMID: 19064829.
1988 <http://www.ncbi.nlm.nih.gov/pubmed/19064829>.
- 1989 79. Salas-Salvadó J, Bulló M, Babio N, Martínez-González M, Ibarrola-Jurado N, Basora J,
1990 et al. Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of
1991 the PREDIMED-Reus nutrition intervention randomized trial. *Diabetes Care*.
1992 2011;34(1):14-9. PMID: 20929998. <http://www.ncbi.nlm.nih.gov/pubmed/20929998>.
- 1993 80. Tonstad S, Stewart K, Oda K, Batech M, Herring RP, Fraser GE. Vegetarian diets and
1994 incidence of diabetes in the Adventist Health Study-2. *Nutr Metab Cardiovasc Dis*.
1995 2013;23(4):292-9. PMID: 21983060. <http://www.ncbi.nlm.nih.gov/pubmed/21983060>.
- 1996 81. Kimokoti RW, Gona P, Zhu L, Newby PK, Millen BE, Brown LS, et al. Dietary patterns
1997 of women are associated with incident abdominal obesity but not metabolic syndrome. *J*
1998 *Nutr*. 2012;142(9):1720-7. PMID: 22833658.
1999 <http://www.ncbi.nlm.nih.gov/pubmed/22833658>.

- 2000 82. Lau C, Toft U, Tetens I, Carstensen B, Jørgensen T, Pedersen O, et al. Dietary patterns
2001 predict changes in two-hour post-oral glucose tolerance test plasma glucose
2002 concentrations in middle-aged adults. *J Nutr.* 2009;139(3):588-93. PMID: 19158222.
2003 <http://www.ncbi.nlm.nih.gov/pubmed/19158222>.
- 2004 83. Willett W. *Nutritional Epidemiology*. New York: Oxford University Press; 1988.
- 2005 84. World Cancer Research Fund/American Institute for Cancer Research. *Food, Nutrition,
2006 and the Prevention of Cancer: a Global Perspective*. Washington D.C.: AICR; 1997.
- 2007 85. World Cancer Research Fund/American Institute for Cancer Research. *Food, Nutrition,
2008 Physical Activity, and the Prevention of Cancer: a Global Perspective*. Washington D.C.:
2009 AICR; 2007.
- 2010 86. World Cancer Research Fund/American Institute for Cancer Research. Continuous
2011 Update Project (CUP) 2014 [cited 2014 November 6]. Available from:
2012 <http://www.dietandcancerreport.org/cup/>.
- 2013 87. World Cancer Research Fund/American Institute for Cancer Research. Chapter 3:
2014 Judging the Evidence. *Food Nutrition, Physical Activity, and the Prevention of Cancer: a
2015 Global Perspective*. Washington D.C.: AICR; 2007. p. 48-62.
- 2016 88. Agnoli C, Grioni S, Sieri S, Palli D, Masala G, Sacerdote C, et al. Italian Mediterranean
2017 Index and risk of colorectal cancer in the Italian section of the EPIC cohort. *Int J Cancer.*
2018 2013;132(6):1404-11. PMID: 22821300.
2019 <http://www.ncbi.nlm.nih.gov/pubmed/22821300>.
- 2020 89. Bamia C, Lagiou P, Buckland G, Grioni S, Agnoli C, Taylor AJ, et al. Mediterranean diet
2021 and colorectal cancer risk: results from a European cohort. *Eur J Epidemiol.*
2022 2013;28(4):317-28. PMID: 23579425. <http://www.ncbi.nlm.nih.gov/pubmed/23579425>.
- 2023 90. Beresford SA, Johnson KC, Ritenbaugh C, Lasser NL, Snetselaar LG, Black HR, et al.
2024 Low-fat dietary pattern and risk of colorectal cancer: the Women's Health Initiative
2025 Randomized Controlled Dietary Modification Trial. *JAMA.* 2006;295(6):643-54. PMID:
2026 16467233. <http://www.ncbi.nlm.nih.gov/pubmed/16467233>.
- 2027 91. Butler LM, Wang R, Koh WP, Yu MC. Prospective study of dietary patterns and
2028 colorectal cancer among Singapore Chinese. *Br J Cancer.* 2008;99(9):1511-6. PMID:
2029 18813309. <http://www.ncbi.nlm.nih.gov/pubmed/18813309>.
- 2030 92. Dixon LB, Balder HF, Virtanen MJ, Rashidkhani B, Mannisto S, Krogh V, et al. Dietary
2031 patterns associated with colon and rectal cancer: results from the Dietary Patterns and
2032 Cancer (DIETSCAN) Project. *Am J Clin Nutr.* 2004;80(4):1003-11. PMID: 15447912.
2033 <http://www.ncbi.nlm.nih.gov/pubmed/15447912>.
- 2034 93. Doubeni CA, Major JM, Laiyemo AO, Schootman M, Zauber AG, Hollenbeck AR, et al.
2035 Contribution of behavioral risk factors and obesity to socioeconomic differences in
2036 colorectal cancer incidence. *J Natl Cancer Inst.* 2012;104(18):1353-62. PMID: 22952311.
2037 <http://www.ncbi.nlm.nih.gov/pubmed/22952311>.
- 2038 94. Engeset D, Dyachenko A, Ciampi A, Lund E. Dietary patterns and risk of cancer of
2039 various sites in the Norwegian European Prospective Investigation into Cancer and
2040 Nutrition cohort: the Norwegian Women and Cancer study. *Eur J Cancer Prev.*
2041 2009;18(1):69-75. PMID: 19077568. <http://www.ncbi.nlm.nih.gov/pubmed/19077568>.
- 2042 95. Flood A, Rastogi T, Wirfalt E, Mitrou PN, Reedy J, Subar AF, et al. Dietary patterns as
2043 identified by factor analysis and colorectal cancer among middle-aged Americans. *Am J
2044 Clin Nutr.* 2008;88(1):176-84. PMID: 18614739.
2045 <http://www.ncbi.nlm.nih.gov/pubmed/18614739>.

- 2046 96. Fung T, Hu FB, Fuchs C, Giovannucci E, Hunter DJ, Stampfer MJ, et al. Major dietary
2047 patterns and the risk of colorectal cancer in women. *Arch Intern Med.* 2003;163(3):309-
2048 14. PMID: 12578511. <http://www.ncbi.nlm.nih.gov/pubmed/12578511>.
- 2049 97. Fung TT, Hu FB, Wu K, Chiuve SE, Fuchs CS, Giovannucci E. The Mediterranean and
2050 Dietary Approaches to Stop Hypertension (DASH) diets and colorectal cancer. *Am J Clin*
2051 *Nutr.* 2010;92(6):1429-35. PMID: 21097651.
2052 <http://www.ncbi.nlm.nih.gov/pubmed/21097651>.
- 2053 98. Fung TT, Hu FB, Schulze M, Pollak M, Wu T, Fuchs CS, et al. A dietary pattern that is
2054 associated with C-peptide and risk of colorectal cancer in women. *Cancer Causes*
2055 *Control.* 2012;23(6):959-65. PMID: 22535146.
2056 <http://www.ncbi.nlm.nih.gov/pubmed/22535146>.
- 2057 99. Jarvandi S, Davidson NO, Schootman M. Increased risk of colorectal cancer in type 2
2058 diabetes is independent of diet quality. *PLoS One.* 2013;8(9):e74616. PMID: 24069323.
2059 <http://www.ncbi.nlm.nih.gov/pubmed/24069323>.
- 2060 100. Kesse E, Clavel-Chapelon F, Boutron-Ruault MC. Dietary patterns and risk of colorectal
2061 tumors: a cohort of French women of the National Education System (E3N). *Am J*
2062 *Epidemiol.* 2006;164(11):1085-93. PMID: 16990408.
2063 <http://www.ncbi.nlm.nih.gov/pubmed/16990408>.
- 2064 101. Key TJ, Appleby PN, Spencer EA, Travis RC, Roddam AW, Allen NE. Cancer incidence
2065 in vegetarians: results from the European Prospective Investigation into Cancer and
2066 Nutrition (EPIC-Oxford). *Am J Clin Nutr.* 2009;89(5):1620S-6S. PMID: 19279082.
2067 <http://www.ncbi.nlm.nih.gov/pubmed/19279082>.
- 2068 102. Kim MK, Sasaki S, Otani T, Tsugane S, Japan Public Health Center-based Prospective
2069 Study G. Dietary patterns and subsequent colorectal cancer risk by subsite: a prospective
2070 cohort study. *Int J Cancer.* 2005;115(5):790-8. PMID: 15704172.
2071 <http://www.ncbi.nlm.nih.gov/pubmed/15704172>.
- 2072 103. Kyro C, Skeie G, Loft S, Overvad K, Christensen J, Tjonneland A, et al. Adherence to a
2073 healthy Nordic food index is associated with a lower incidence of colorectal cancer in
2074 women: the Diet, Cancer and Health cohort study. *Br J Nutr.* 2013;109(5):920-7. PMID:
2075 22874538. <http://www.ncbi.nlm.nih.gov/pubmed/22874538>.
- 2076 104. Mai V, Kant AK, Flood A, Lacey JV, Jr., Schairer C, Schatzkin A. Diet quality and
2077 subsequent cancer incidence and mortality in a prospective cohort of women. *Int J*
2078 *Epidemiol.* 2005;34(1):54-60. PMID: 15649959.
2079 <http://www.ncbi.nlm.nih.gov/pubmed/15649959>.
- 2080 105. Miller PE, Cross AJ, Subar AF, Krebs-Smith SM, Park Y, Powell-Wiley T, et al.
2081 Comparison of 4 established DASH diet indexes: examining associations of index scores
2082 and colorectal cancer. *Am J Clin Nutr.* 2013;98(3):794-803. PMID: 23864539.
2083 <http://www.ncbi.nlm.nih.gov/pubmed/23864539>.
- 2084 106. Reedy J, Mitrou PN, Krebs-Smith SM, Wirfalt E, Flood A, Kipnis V, et al. Index-based
2085 dietary patterns and risk of colorectal cancer: the NIH-AARP Diet and Health Study. *Am*
2086 *J Epidemiol.* 2008;168(1):38-48. PMID: 18525082.
2087 <http://www.ncbi.nlm.nih.gov/pubmed/18525082>.
- 2088 107. Wirfalt E, Midthune D, Reedy J, Mitrou P, Flood A, Subar AF, et al. Associations
2089 between food patterns defined by cluster analysis and colorectal cancer incidence in the
2090 NIH-AARP diet and health study. *Eur J Clin Nutr.* 2009;63(6):707-17. PMID: 18685556.
2091 <http://www.ncbi.nlm.nih.gov/pubmed/18685556>.

- 2092 108. Wu K, Hu FB, Fuchs C, Rimm EB, Willett WC, Giovannucci E. Dietary patterns and risk
2093 of colon cancer and adenoma in a cohort of men (United States). *Cancer Causes Control*.
2094 2004;15(9):853-62. PMID: 15577287. <http://www.ncbi.nlm.nih.gov/pubmed/15577287>.
- 2095 109. Terry P, Hu FB, Hansen H, Wolk A. Prospective study of major dietary patterns and
2096 colorectal cancer risk in women. *Am J Epidemiol*. 2001;154(12):1143-9. PMID:
2097 11744520. <http://www.ncbi.nlm.nih.gov/pubmed/11744520>.
- 2098 110. Adebamowo CA, Hu FB, Cho E, Spiegelman D, Holmes MD, Willett WC. Dietary
2099 patterns and the risk of breast cancer. *Ann Epidemiol*. 2005;15(10):789-95. PMID:
2100 16257363. <http://www.ncbi.nlm.nih.gov/pubmed/16257363>.
- 2101 111. Agurs-Collins T, Rosenberg L, Makambi K, Palmer JR, Adams-Campbell L. Dietary
2102 patterns and breast cancer risk in women participating in the Black Women's Health
2103 Study. *Am J Clin Nutr*. 2009;90(3):621-8. PMID: 19587089.
2104 <http://www.ncbi.nlm.nih.gov/pubmed/19587089>.
- 2105 112. Baglietto L, Krishnan K, Severi G, Hodge A, Brinkman M, English DR, et al. Dietary
2106 patterns and risk of breast cancer. *Br J Cancer*. 2011;104(3):524-31. PMID: 21157446.
2107 <http://www.ncbi.nlm.nih.gov/pubmed/21157446>.
- 2108 113. Buckland G, Travier N, Cottet V, Gonzalez CA, Lujan-Barroso L, Agudo A, et al.
2109 Adherence to the mediterranean diet and risk of breast cancer in the European
2110 prospective investigation into cancer and nutrition cohort study. *Int J Cancer*.
2111 2013;132(12):2918-27. PMID: 23180513.
2112 <http://www.ncbi.nlm.nih.gov/pubmed/23180513>.
- 2113 114. Butler LM, Wu AH, Wang R, Koh WP, Yuan JM, Yu MC. A vegetable-fruit-soy dietary
2114 pattern protects against breast cancer among postmenopausal Singapore Chinese women.
2115 *Am J Clin Nutr*. 2010;91(4):1013-9. PMID: 20181808.
2116 <http://www.ncbi.nlm.nih.gov/pubmed/20181808>.
- 2117 115. Cade JE, Taylor EF, Burley VJ, Greenwood DC. Common dietary patterns and risk of
2118 breast cancer: analysis from the United Kingdom Women's Cohort Study. *Nutr Cancer*.
2119 2010;62(3):300-6. PMID: 20358467. <http://www.ncbi.nlm.nih.gov/pubmed/20358467>.
- 2120 116. Cade JE, Taylor EF, Burley VJ, Greenwood DC. Does the Mediterranean dietary pattern
2121 or the Healthy Diet Index influence the risk of breast cancer in a large British cohort of
2122 women? *Eur J Clin Nutr*. 2011;65(8):920-8. PMID: 21587285.
2123 <http://www.ncbi.nlm.nih.gov/pubmed/21587285>.
- 2124 117. Cottet V, Touvier M, Fournier A, Touillaud MS, Lafay L, Clavel-Chapelon F, et al.
2125 Postmenopausal breast cancer risk and dietary patterns in the E3N-EPIC prospective
2126 cohort study. *Am J Epidemiol*. 2009;170(10):1257-67. PMID: 19828509.
2127 <http://www.ncbi.nlm.nih.gov/pubmed/19828509>.
- 2128 118. Couto E, Sandin S, Lof M, Ursin G, Adami HO, Weiderpass E. Mediterranean dietary
2129 pattern and risk of breast cancer. *PLoS One*. 2013;8(2):e55374. PMID: 23390532.
2130 <http://www.ncbi.nlm.nih.gov/pubmed/23390532>.
- 2131 119. Fung TT, Hu FB, Holmes MD, Rosner BA, Hunter DJ, Colditz GA, et al. Dietary
2132 patterns and the risk of postmenopausal breast cancer. *Int J Cancer*. 2005;116(1):116-21.
2133 PMID: 15756679. <http://www.ncbi.nlm.nih.gov/pubmed/15756679>.
- 2134 120. Fung TT, Hu FB, McCullough ML, Newby PK, Willett WC, Holmes MD. Diet quality is
2135 associated with the risk of estrogen receptor-negative breast cancer in postmenopausal
2136 women. *J Nutr*. 2006;136(2):466-72. PMID: 16424129.
2137 <http://www.ncbi.nlm.nih.gov/pubmed/16424129>.

- 2138 121. Fung TT, Hu FB, Hankinson SE, Willett WC, Holmes MD. Low-carbohydrate diets,
2139 dietary approaches to stop hypertension-style diets, and the risk of postmenopausal breast
2140 cancer. *Am J Epidemiol*. 2011;174(6):652-60. PMID: 21832271.
2141 <http://www.ncbi.nlm.nih.gov/pubmed/21832271>.
- 2142 122. Fung TT, Schulze MB, Hu FB, Hankinson SE, Holmes MD. A dietary pattern derived to
2143 correlate with estrogens and risk of postmenopausal breast cancer. *Breast Cancer Res*
2144 *Treat*. 2012;132(3):1157-62. PMID: 22218885.
2145 <http://www.ncbi.nlm.nih.gov/pubmed/22218885>.
- 2146 123. Link LB, Canchola AJ, Bernstein L, Clarke CA, Stram DO, Ursin G, et al. Dietary
2147 patterns and breast cancer risk in the California Teachers Study cohort. *Am J Clin Nutr*.
2148 2013;98(6):1524-32. PMID: 24108781. <http://www.ncbi.nlm.nih.gov/pubmed/24108781>.
- 2149 124. Mannisto S, Dixon LB, Balder HF, Virtanen MJ, Krogh V, Khani BR, et al. Dietary
2150 patterns and breast cancer risk: results from three cohort studies in the DIETSCAN
2151 project. *Cancer Causes Control*. 2005;16(6):725-33. PMID: 16049811.
2152 <http://www.ncbi.nlm.nih.gov/pubmed/16049811>.
- 2153 125. Prentice RL, Caan B, Chlebowski RT, Patterson R, Kuller LH, Ockene JK, et al. Low-fat
2154 dietary pattern and risk of invasive breast cancer: the Women's Health Initiative
2155 Randomized Controlled Dietary Modification Trial. *JAMA*. 2006;295(6):629-42. PMID:
2156 16467232. <http://www.ncbi.nlm.nih.gov/pubmed/16467232>.
- 2157 126. Sant M, Allemani C, Sieri S, Krogh V, Menard S, Tagliabue E, et al. Salad vegetables
2158 dietary pattern protects against HER-2-positive breast cancer: a prospective Italian study.
2159 *Int J Cancer*. 2007;121(4):911-4. PMID: 17455245.
2160 <http://www.ncbi.nlm.nih.gov/pubmed/17455245>.
- 2161 127. Schulz M, Hoffmann K, Weikert C, Nothlings U, Schulze MB, Boeing H. Identification
2162 of a dietary pattern characterized by high-fat food choices associated with increased risk
2163 of breast cancer: the European Prospective Investigation into Cancer and Nutrition
2164 (EPIC)-Potsdam Study. *Br J Nutr*. 2008;100(5):942-6. PMID: 18377685.
2165 <http://www.ncbi.nlm.nih.gov/pubmed/18377685>.
- 2166 128. Sieri S, Krogh V, Pala V, Muti P, Micheli A, Evangelista A, et al. Dietary patterns and
2167 risk of breast cancer in the ORDET cohort. *Cancer Epidemiol Biomarkers Prev*.
2168 2004;13(4):567-72. PMID: 15066921. <http://www.ncbi.nlm.nih.gov/pubmed/15066921>.
- 2169 129. Terry P, Suzuki R, Hu FB, Wolk A. A prospective study of major dietary patterns and the
2170 risk of breast cancer. *Cancer Epidemiol Biomarkers Prev*. 2001;10(12):1281-5. PMID:
2171 11751446. <http://www.ncbi.nlm.nih.gov/pubmed/11751446>.
- 2172 130. Trichopoulou A, Bamia C, Lagiou P, Trichopoulos D. Conformity to traditional
2173 Mediterranean diet and breast cancer risk in the Greek EPIC (European Prospective
2174 Investigation into Cancer and Nutrition) cohort. *Am J Clin Nutr*. 2010;92(3):620-5.
2175 PMID: 20631204. <http://www.ncbi.nlm.nih.gov/pubmed/20631204>.
- 2176 131. Velie EM, Schairer C, Flood A, He JP, Khattree R, Schatzkin A. Empirically derived
2177 dietary patterns and risk of postmenopausal breast cancer in a large prospective cohort
2178 study. *Am J Clin Nutr*. 2005;82(6):1308-19. PMID: 16332665.
2179 <http://www.ncbi.nlm.nih.gov/pubmed/16332665>.
- 2180 132. Balder HF, Goldbohm RA, van den Brandt PA. Dietary patterns associated with male
2181 lung cancer risk in the Netherlands Cohort Study. *Cancer Epidemiol Biomarkers Prev*.
2182 2005;14(2):483-90. PMID: 15734976. <http://www.ncbi.nlm.nih.gov/pubmed/15734976>.

- 2183 133. Gnagnarella P, Maisonneuve P, Bellomi M, Rampinelli C, Bertolotti R, Spaggiari L, et al.
 2184 Red meat, Mediterranean diet and lung cancer risk among heavy smokers in the
 2185 COSMOS screening study. *Ann Oncol.* 2013;24(10):2606-11. PMID: 23956193.
 2186 <http://www.ncbi.nlm.nih.gov/pubmed/23956193>.
- 2187 134. Ax E, Garmo H, Grundmark B, Bill-Axelsson A, Holmberg L, Becker W, et al. Dietary
 2188 patterns and prostate cancer risk: report from the population based ULSAM cohort study
 2189 of swedish men. *Nutr Cancer.* 2014;66(1):77-87. PMID: 24325263.
 2190 <http://www.ncbi.nlm.nih.gov/pubmed/24325263>.
- 2191 135. Bosire C, Stampfer MJ, Subar AF, Park Y, Kirkpatrick SI, Chiuve SE, et al. Index-based
 2192 dietary patterns and the risk of prostate cancer in the NIH-AARP diet and health study.
 2193 *Am J Epidemiol.* 2013;177(6):504-13. PMID: 23408548.
 2194 <http://www.ncbi.nlm.nih.gov/pubmed/23408548>.
- 2195 136. Kenfield SA, DuPre N, Richman EL, Stampfer MJ, Chan JM, Giovannucci EL.
 2196 Mediterranean diet and prostate cancer risk and mortality in the health professionals
 2197 follow-up study. *Eur Urol.* 2014;65(5):887-94. PMID: 23962747.
 2198 <http://www.ncbi.nlm.nih.gov/pubmed/23962747>.
- 2199 137. Muller DC, Severi G, Baglietto L, Krishnan K, English DR, Hopper JL, et al. Dietary
 2200 patterns and prostate cancer risk. *Cancer Epidemiol Biomarkers Prev.* 2009;18(11):3126-
 2201 9. PMID: 19861522. <http://www.ncbi.nlm.nih.gov/pubmed/19861522>.
- 2202 138. Tseng M, Breslow RA, DeVellis RF, Ziegler RG. Dietary patterns and prostate cancer
 2203 risk in the National Health and Nutrition Examination Survey Epidemiological Follow-up
 2204 Study cohort. *Cancer Epidemiol Biomarkers Prev.* 2004;13(1):71-7. PMID: 14744736.
 2205 <http://www.ncbi.nlm.nih.gov/pubmed/14744736>.
- 2206 139. Wu K, Hu FB, Willett WC, Giovannucci E. Dietary patterns and risk of prostate cancer in
 2207 U.S. men. *Cancer Epidemiol Biomarkers Prev.* 2006;15(1):167-71. PMID: 16434606.
 2208 <http://www.ncbi.nlm.nih.gov/pubmed/16434606>.
- 2209 140. Wolff T, Witkop CT, Miller T, Syed SB. Folic Acid Supplementation for the Prevention
 2210 of Neural Tube Defects: An Update of the Evidence for the U.S. Preventive Services
 2211 Task Force. Evidence Synthesis No. 70. AHRQ Publication No. 09-051132-EF-1.
 2212 Rockville, Maryland: Agency for Healthcare Research and Quality. 2009 May.
- 2213 141. Pitkin RM. Folate and neural tube defects. *Am J Clin Nutr.* 2007;85(1):285S-8S. PMID:
 2214 17209211. <http://www.ncbi.nlm.nih.gov/pubmed/17209211>.
- 2215 142. Yang Q, Cogswell ME, Hamner HC, Carriquiry A, Bailey LB, Pfeiffer CM, et al. Folic
 2216 acid source, usual intake, and folate and vitamin B-12 status in US adults: National
 2217 Health and Nutrition Examination Survey (NHANES) 2003-2006. *Am J Clin Nutr.*
 2218 2010;91(1):64-72. PMID: 19828716. <http://www.ncbi.nlm.nih.gov/pubmed/19828716>.
- 2219 143. Bailey RL, Dodd KW, Gahche JJ, Dwyer JT, McDowell MA, Yetley EA, et al. Total
 2220 folate and folic acid intake from foods and dietary supplements in the United States:
 2221 2003-2006. *Am J Clin Nutr.* 2010;91(1):231-7. PMID: 19923379.
 2222 <http://www.ncbi.nlm.nih.gov/pubmed/19923379>.
- 2223 144. U. S. Preventive Services Task Force. Folic Acid to Prevent Neural Tube Defects:
 2224 Preventive Medication. Recommendation Summary May 2009 [cited 2014 November].
 2225 Available from: <http://www.uspreventiveservicestaskforce.org/uspstf/uspnsrnfol.htm>.
- 2226 145. Food and Nutrition Board - Institute of Medicine. Dietary Reference Intakes: The
 2227 Essential Guide to Nutrient Requirements. Washington, DC: The National Academies
 2228 Press; 2006.

- 2229 146. Carmichael SL, Yang W, Feldkamp ML, Munger RG, Siega-Riz AM, Botto LD, et al.
 2230 Reduced risks of neural tube defects and orofacial clefts with higher diet quality. *Arch*
 2231 *Pediatr Adolesc Med.* 2012;166(2):121-6. PMID: 21969361.
 2232 <http://www.ncbi.nlm.nih.gov/pubmed/21969361>.
- 2233 147. Sotres-Alvarez D, Siega-Riz AM, Herring AH, Carmichael SL, Feldkamp ML, Hobbs
 2234 CA, et al. Maternal dietary patterns are associated with risk of neural tube and congenital
 2235 heart defects. *Am J Epidemiol.* 2013;177(11):1279-88. PMID: 23639938.
 2236 <http://www.ncbi.nlm.nih.gov/pubmed/23639938>.
- 2237 148. Vujkovic M, Ocke MC, van der Spek PJ, Yazdanpanah N, Steegers EA, Steegers-
 2238 Theunissen RP. Maternal Western dietary patterns and the risk of developing a cleft lip
 2239 with or without a cleft palate. *Obstet Gynecol.* 2007;110(2 Pt 1):378-84. PMID:
 2240 17666614. <http://www.ncbi.nlm.nih.gov/pubmed/17666614>.
- 2241 149. Vujkovic M, Steegers EA, Looman CW, Ocke MC, van der Spek PJ, Steegers-
 2242 Theunissen RP. The maternal Mediterranean dietary pattern is associated with a reduced
 2243 risk of spina bifida in the offspring. *BJOG.* 2009;116(3):408-15. PMID: 19187373.
 2244 <http://www.ncbi.nlm.nih.gov/pubmed/19187373>.
- 2245 150. Obermann-Borst SA, Vujkovic M, de Vries JH, Wildhagen MF, Looman CW, de Jonge
 2246 R, et al. A maternal dietary pattern characterised by fish and seafood in association with
 2247 the risk of congenital heart defects in the offspring. *BJOG.* 2011;118(10):1205-15.
 2248 PMID: 21585642. <http://www.ncbi.nlm.nih.gov/pubmed/21585642>.
- 2249 151. Nutrition Evidence Library; U.S. Department of Agriculture. What are the effects of
 2250 maternal dietary intake of omega-3 fatty acids on breast milk composition and infant
 2251 health outcomes? [cited 2014 November]. Available from:
 2252 http://nel.gov/evidence.cfm?evidence_summary_id=250144.
- 2253 152. Hebert LE, Weuve J, Scherr PA, Evans DA. Alzheimer disease in the United States
 2254 (2010-2050) estimated using the 2010 census. *Neurology.* 2013;80(19):1778-83. PMID:
 2255 23390181. <http://www.ncbi.nlm.nih.gov/pubmed/23390181>.
- 2256 153. Dorsey ER, George BP, Leff B, Willis AW. The coming crisis: obtaining care for the
 2257 growing burden of neurodegenerative conditions. *Neurology.* 2013;80(21):1989-96.
 2258 PMID: 23616157. <http://www.ncbi.nlm.nih.gov/pubmed/23616157>.
- 2259 154. Centers for Disease Control and Prevention. QuickStats: Prevalence of Current
 2260 Depression* Among Persons Aged ≥ 12 Years, by Age Group and Sex — United States,
 2261 National Health and Nutrition Examination Survey, 2007–2010. *Morbidity and Mortality*
 2262 *Weekly Report (MMWR)* [Internet]. 2012; 60(51):[1747 p.]. Available from:
 2263 http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.
- 2264 155. Barnes JL, Tian M, Edens NK, Morris MC. Consideration of nutrient levels in studies of
 2265 cognitive decline. *Nutr Rev.* 2014;72(11):707-19. PMID: 25323849.
 2266 <http://www.ncbi.nlm.nih.gov/pubmed/25323849>.
- 2267 156. Rita Cardoso B, Silva Bandeira V, Jacob-Filho W, Franciscato Cozzolino SM. Selenium
 2268 status in elderly: Relation to cognitive decline. *J Trace Elem Med Biol.* 2014. PMID:
 2269 25220532. <http://www.ncbi.nlm.nih.gov/pubmed/25220532>.
- 2270 157. Gelenberg AJ, Freeman MP, Markowitz JC, Rosenbaum JF, Thase ME, Trivedi MH, et
 2271 al. Practice Guideline for the Treatment of Patients With Major Depressive Disorder,
 2272 Third Edition. page 51 and page 92. 2010. Available from:
 2273 http://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/mdd.pdf
 2274 .

- 2275 158. Grosso G, Pajak A, Marventano S, Castellano S, Galvano F, Bucolo C, et al. Role of
2276 omega-3 fatty acids in the treatment of depressive disorders: a comprehensive meta-
2277 analysis of randomized clinical trials. *PLoS One*. 2014;9(5):e96905. PMID: 24805797.
2278 <http://www.ncbi.nlm.nih.gov/pubmed/24805797>.
- 2279 159. Cherbuin N, Anstey KJ. The Mediterranean diet is not related to cognitive change in a
2280 large prospective investigation: the PATH Through Life study. *Am J Geriatr Psychiatry*.
2281 2012;20(7):635-9. PMID: 21937919. <http://www.ncbi.nlm.nih.gov/pubmed/21937919>.
- 2282 160. Eskelinen MH, Ngandu T, Tuomilehto J, Soininen H, Kivipelto M. Midlife healthy-diet
2283 index and late-life dementia and Alzheimer's disease. *Dement Geriatr Cogn Dis Extra*.
2284 2011;1(1):103-12. PMID: 22163237. <http://www.ncbi.nlm.nih.gov/pubmed/22163237>.
- 2285 161. Féart C, Samieri C, Rondeau V, Amieva H, Portet F, Dartigues JF, et al. Adherence to a
2286 Mediterranean diet, cognitive decline, and risk of dementia. *JAMA*. 2009;302(6):638-48.
2287 PMID: 19671905. <http://www.ncbi.nlm.nih.gov/pubmed/19671905>.
- 2288 162. Gardener SL, Rainey-Smith SR, Barnes MB, Sohrabi HR, Weinborn M, Lim YY, et al.
2289 Dietary patterns and cognitive decline in an Australian study of ageing. *Mol Psychiatry*.
2290 2014. PMID: 25070537. <http://www.ncbi.nlm.nih.gov/pubmed/25070537>.
- 2291 163. Gu Y, Luchsinger JA, Stern Y, Scarmeas N. Mediterranean diet, inflammatory and
2292 metabolic biomarkers, and risk of Alzheimer's disease. *J Alzheimers Dis*.
2293 2010;22(2):483-92. PMID: 20847399. <http://www.ncbi.nlm.nih.gov/pubmed/20847399>.
- 2294 164. Gu Y, Nieves JW, Stern Y, Luchsinger JA, Scarmeas N. Food combination and
2295 Alzheimer disease risk: a protective diet. *Arch Neurol*. 2010;67(6):699-706. PMID:
2296 20385883. <http://www.ncbi.nlm.nih.gov/pubmed/20385883>.
- 2297 165. Kesse-Guyot E, Amieva H, Castetbon K, Henegar A, Ferry M, Jeandel C, et al.
2298 Adherence to nutritional recommendations and subsequent cognitive performance:
2299 findings from the prospective Supplementation with Antioxidant Vitamins and Minerals
2300 2 (SU.VI.MAX 2) study. *Am J Clin Nutr*. 2011;93(1):200-10. PMID: 21106918.
2301 <http://www.ncbi.nlm.nih.gov/pubmed/21106918>.
- 2302 166. Kesse-Guyot E, Andreeva VA, Jeandel C, Ferry M, Hercberg S, Galan P. A healthy
2303 dietary pattern at midlife is associated with subsequent cognitive performance. *J Nutr*.
2304 2012;142(5):909-15. PMID: 22457391. <http://www.ncbi.nlm.nih.gov/pubmed/22457391>.
- 2305 167. Kesse-Guyot E, Andreeva VA, Lassale C, Ferry M, Jeandel C, Hercberg S, et al.
2306 Mediterranean diet and cognitive function: a French study. *Am J Clin Nutr*.
2307 2013;97(2):369-76. PMID: 23283500. <http://www.ncbi.nlm.nih.gov/pubmed/23283500>.
- 2308 168. Kesse-Guyot E, Andreeva VA, Ducros V, Jeandel C, Julia C, Hercberg S, et al.
2309 Carotenoid-rich dietary patterns during midlife and subsequent cognitive function. *Br J*
2310 *Nutr*. 2014;111(5):915-23. PMID: 24073964.
2311 <http://www.ncbi.nlm.nih.gov/pubmed/24073964>.
- 2312 169. Koyama A, Houston DK, Simonsick EM, Lee JS, Ayonayon HN, Shahar DR, et al.
2313 Association Between the Mediterranean Diet and Cognitive Decline in a Biracial
2314 Population. *J Gerontol A Biol Sci Med Sci*. 2014. PMID: 24994847.
2315 <http://www.ncbi.nlm.nih.gov/pubmed/24994847>.
- 2316 170. Martínez-Lapiscina EH, Clavero P, Toledo E, Estruch R, Salas-Salvadó J, San Julián B,
2317 et al. Mediterranean diet improves cognition: the PREDIMED-NAVARRA randomised
2318 trial. *J Neurol Neurosurg Psychiatry*. 2013;84(12):1318-25. PMID: 23670794.
2319 <http://www.ncbi.nlm.nih.gov/pubmed/23670794>.

- 2320 171. Nicolas A, Faisant C, Nourhashemi F, Lanzmann-Petithory D, Vellas B. Associations
2321 between nutritional intake and cognitive function in a healthy ageing sample: A 4-year
2322 reassessment. . *European Journal of Geriatrics*. 2000;2(3):114-9.
- 2323 172. Olsson E, Karlström B, Kilander L, Byberg L, Cederholm T, Sjögren P. Dietary Patterns
2324 and Cognitive Dysfunction in a 12-Year Follow-up Study of 70 Year Old Men. *J*
2325 *Alzheimers Dis*. 2014. PMID: 25062901.
2326 <http://www.ncbi.nlm.nih.gov/pubmed/25062901>.
- 2327 173. Ozawa M, Ninomiya T, Ohara T, Doi Y, Uchida K, Shirota T, et al. Dietary patterns and
2328 risk of dementia in an elderly Japanese population: the Hisayama Study. *Am J Clin Nutr*.
2329 2013;97(5):1076-82. PMID: 23553168. <http://www.ncbi.nlm.nih.gov/pubmed/23553168>.
- 2330 174. Parrott MD, Shatenstein B, Ferland G, Payette H, Morais JA, Belleville S, et al.
2331 Relationship between diet quality and cognition depends on socioeconomic position in
2332 healthy older adults. *J Nutr*. 2013;143(11):1767-73. PMID: 23986363.
2333 <http://www.ncbi.nlm.nih.gov/pubmed/23986363>.
- 2334 175. Psaltopoulou T, Kyrozis A, Stathopoulos P, Trichopoulos D, Vassilopoulos D,
2335 Trichopoulou A. Diet, physical activity and cognitive impairment among elders: the
2336 EPIC-Greece cohort (European Prospective Investigation into Cancer and Nutrition).
2337 *Public Health Nutr*. 2008;11(10):1054-62. PMID: 18205988.
2338 <http://www.ncbi.nlm.nih.gov/pubmed/18205988>.
- 2339 176. Roberts RO, Geda YE, Cerhan JR, Knopman DS, Cha RH, Christianson TJ, et al.
2340 Vegetables, unsaturated fats, moderate alcohol intake, and mild cognitive impairment.
2341 *Dement Geriatr Cogn Disord*. 2010;29(5):413-23. PMID: 20502015.
2342 <http://www.ncbi.nlm.nih.gov/pubmed/20502015>.
- 2343 177. Samieri C, Grodstein F, Rosner BA, Kang JH, Cook NR, Manson JE, et al.
2344 Mediterranean diet and cognitive function in older age. *Epidemiology*. 2013;24(4):490-9.
2345 PMID: 23676264. <http://www.ncbi.nlm.nih.gov/pubmed/23676264>.
- 2346 178. Samieri C, Okereke OI, E Devore E, Grodstein F. Long-term adherence to the
2347 Mediterranean diet is associated with overall cognitive status, but not cognitive decline,
2348 in women. *J Nutr*. 2013;143(4):493-9. PMID: 23365105.
2349 <http://www.ncbi.nlm.nih.gov/pubmed/23365105>.
- 2350 179. Samieri C, Sun Q, Townsend MK, Chiuve SE, Okereke OI, Willett WC, et al. The
2351 association between dietary patterns at midlife and health in aging: an observational
2352 study. *Ann Intern Med*. 2013;159(9):584-91. PMID: 24189593.
2353 <http://www.ncbi.nlm.nih.gov/pubmed/24189593>.
- 2354 180. Scarmeas N, Stern Y, Mayeux R, Manly JJ, Schupf N, Luchsinger JA. Mediterranean diet
2355 and mild cognitive impairment. *Arch Neurol*. 2009;66(2):216-25. PMID: 19204158.
2356 <http://www.ncbi.nlm.nih.gov/pubmed/19204158>.
- 2357 181. Scarmeas N, Stern Y, Tang MX, Mayeux R, Luchsinger JA. Mediterranean diet and risk
2358 for Alzheimer's disease. *Ann Neurol*. 2006;59(6):912-21. PMID: 16622828.
2359 <http://www.ncbi.nlm.nih.gov/pubmed/16622828>.
- 2360 182. Shatenstein B, Ferland G, Belleville S, Gray-Donald K, Kergoat MJ, Morais J, et al. Diet
2361 quality and cognition among older adults from the NuAge study. *Exp Gerontol*.
2362 2012;47(5):353-60. PMID: 22386581. <http://www.ncbi.nlm.nih.gov/pubmed/22386581>.
- 2363 183. Smith PJ, Blumenthal JA, Babyak MA, Craighead L, Welsh-Bohmer KA, Browndyke
2364 JN, et al. Effects of the dietary approaches to stop hypertension diet, exercise, and caloric
2365 restriction on neurocognition in overweight adults with high blood pressure.

- 2366 Hypertension. 2010;55(6):1331-8. PMID: 20305128.
 2367 <http://www.ncbi.nlm.nih.gov/pubmed/20305128>.
- 2368 184. Tangney CC, Kwasny MJ, Li H, Wilson RS, Evans DA, Morris MC. Adherence to a
 2369 Mediterranean-type dietary pattern and cognitive decline in a community population. *Am*
 2370 *J Clin Nutr*. 2011;93(3):601-7. PMID: 21177796.
 2371 <http://www.ncbi.nlm.nih.gov/pubmed/21177796>.
- 2372 185. Titova OE, Ax E, Brooks SJ, Sjögren P, Cederholm T, Kilander L, et al. Mediterranean
 2373 diet habits in older individuals: associations with cognitive functioning and brain
 2374 volumes. *Exp Gerontol*. 2013;48(12):1443-8. PMID: 24126083.
 2375 <http://www.ncbi.nlm.nih.gov/pubmed/24126083>.
- 2376 186. Tsivgoulis G, Judd S, Letter AJ, Alexandrov AV, Howard G, Nahab F, et al. Adherence
 2377 to a Mediterranean diet and risk of incident cognitive impairment. *Neurology*.
 2378 2013;80(18):1684-92. PMID: 23628929.
 2379 <http://www.ncbi.nlm.nih.gov/pubmed/23628929>.
- 2380 187. Wengreen H, Munger RG, Cutler A, Quach A, Bowles A, Corcoran C, et al. Prospective
 2381 study of Dietary Approaches to Stop Hypertension- and Mediterranean-style dietary
 2382 patterns and age-related cognitive change: the Cache County Study on Memory, Health
 2383 and Aging. *Am J Clin Nutr*. 2013;98(5):1263-71. PMID: 24047922.
 2384 <http://www.ncbi.nlm.nih.gov/pubmed/24047922>.
- 2385 188. Wengreen HJ, Neilson C, Munger R, Corcoran C. Diet quality is associated with better
 2386 cognitive test performance among aging men and women. *J Nutr*. 2009;139(10):1944-9.
 2387 PMID: 19675102. <http://www.ncbi.nlm.nih.gov/pubmed/19675102>.
- 2388 189. Akbaraly TN, Brunner EJ, Ferrie JE, Marmot MG, Kivimaki M, Singh-Manoux A.
 2389 Dietary pattern and depressive symptoms in middle age. *Br J Psychiatry*.
 2390 2009;195(5):408-13. PMID: 19880930. <http://www.ncbi.nlm.nih.gov/pubmed/19880930>.
- 2391 190. Akbaraly TN, Sabia S, Shipley MJ, Batty GD, Kivimaki M. Adherence to healthy dietary
 2392 guidelines and future depressive symptoms: evidence for sex differentials in the
 2393 Whitehall II study. *Am J Clin Nutr*. 2013;97(2):419-27. PMID: 23283506.
 2394 <http://www.ncbi.nlm.nih.gov/pubmed/23283506>.
- 2395 191. Chan R, Chan D, Woo J. A Prospective Cohort Study to Examine the Association
 2396 between Dietary Patterns and Depressive Symptoms in Older Chinese People in Hong
 2397 Kong. *PLoS One*. 2014;9(8):e105760. PMID: 25148515.
 2398 <http://www.ncbi.nlm.nih.gov/pubmed/25148515>.
- 2399 192. Chatzi L, Melaki V, Sarri K, Apostolaki I, Roumeliotaki T, Georgiou V, et al. Dietary
 2400 patterns during pregnancy and the risk of postpartum depression: the mother-child 'Rhea'
 2401 cohort in Crete, Greece. *Public Health Nutr*. 2011;14(9):1663-70. PMID: 21477412.
 2402 <http://www.ncbi.nlm.nih.gov/pubmed/21477412>.
- 2403 193. Chocano-Bedoya PO, O'Reilly EJ, Lucas M, Mirzaei F, Okereke OI, Fung TT, et al.
 2404 Prospective study on long-term dietary patterns and incident depression in middle-aged
 2405 and older women. *Am J Clin Nutr*. 2013;98(3):813-20. PMID: 23885043.
 2406 <http://www.ncbi.nlm.nih.gov/pubmed/23885043>.
- 2407 194. Jacka FN, Cherbuin N, Anstey KJ, Butterworth P. Dietary patterns and depressive
 2408 symptoms over time: examining the relationships with socioeconomic position, health
 2409 behaviours and cardiovascular risk. *PLoS One*. 2014;9(1):e87657. PMID: 24489946.
 2410 <http://www.ncbi.nlm.nih.gov/pubmed/24489946>.

- 2411 195. Le Port A, Gueguen A, Kesse-Guyot E, Melchior M, Lemogne C, Nabi H, et al.
 2412 Association between dietary patterns and depressive symptoms over time: a 10-year
 2413 follow-up study of the GAZEL cohort. *PLoS One*. 2012;7(12):e51593. PMID: 23251585.
 2414 <http://www.ncbi.nlm.nih.gov/pubmed/23251585>.
- 2415 196. Lucas M, Chocano-Bedoya P, Shulze MB, Mirzaei F, O'Reilly É, Okereke OI, et al.
 2416 Inflammatory dietary pattern and risk of depression among women. *Brain Behav Immun*.
 2417 2014;36:46-53. PMID: 24095894. <http://www.ncbi.nlm.nih.gov/pubmed/24095894>.
- 2418 197. McMartin SE, Kuhle S, Colman I, Kirk SF, Veugelers PJ. Diet quality and mental health
 2419 in subsequent years among Canadian youth. *Public Health Nutr*. 2012;15(12):2253-8.
 2420 PMID: 22414240. <http://www.ncbi.nlm.nih.gov/pubmed/22414240>.
- 2421 198. Okubo H, Miyake Y, Sasaki S, Tanaka K, Murakami K, Hirota Y, et al. Dietary patterns
 2422 during pregnancy and the risk of postpartum depression in Japan: the Osaka Maternal and
 2423 Child Health Study. *Br J Nutr*. 2011;105(8):1251-7. PMID: 21144112.
 2424 <http://www.ncbi.nlm.nih.gov/pubmed/21144112>.
- 2425 199. Rienks J, Dobson AJ, Mishra GD. Mediterranean dietary pattern and prevalence and
 2426 incidence of depressive symptoms in mid-aged women: results from a large community-
 2427 based prospective study. *Eur J Clin Nutr*. 2013;67(1):75-82. PMID: 23212131.
 2428 <http://www.ncbi.nlm.nih.gov/pubmed/23212131>.
- 2429 200. Ruusunen A, Lehto SM, Mursu J, Tolmunen T, Tuomainen TP, Kauhanen J, et al.
 2430 Dietary patterns are associated with the prevalence of elevated depressive symptoms and
 2431 the risk of getting a hospital discharge diagnosis of depression in middle-aged or older
 2432 Finnish men. *J Affect Disord*. 2014;159:1-6. PMID: 24679382.
 2433 <http://www.ncbi.nlm.nih.gov/pubmed/24679382>.
- 2434 201. Sánchez-Villegas A, Delgado-Rodríguez M, Alonso A, Schlatter J, Lahortiga F, Serra
 2435 Majem L, et al. Association of the Mediterranean dietary pattern with the incidence of
 2436 depression: the Seguimiento Universidad de Navarra/University of Navarra follow-up
 2437 (SUN) cohort. *Arch Gen Psychiatry*. 2009;66(10):1090-8. PMID: 19805699.
 2438 <http://www.ncbi.nlm.nih.gov/pubmed/19805699>.
- 2439 202. Sánchez-Villegas A, Martínez-González MA, Estruch R, Salas-Salvadó J, Corella D,
 2440 Covas MI, et al. Mediterranean dietary pattern and depression: the PREDIMED
 2441 randomized trial. *BMC Med*. 2013;11:208. PMID: 24229349.
 2442 <http://www.ncbi.nlm.nih.gov/pubmed/24229349>.
- 2443 203. Skarupski KA, Tangney CC, Li H, Evans DA, Morris MC. Mediterranean diet and
 2444 depressive symptoms among older adults over time. *J Nutr Health Aging*.
 2445 2013;17(5):441-5. PMID: 23636545. <http://www.ncbi.nlm.nih.gov/pubmed/23636545>.
- 2446 204. Torres SJ, Nowson CA. A moderate-sodium DASH-type diet improves mood in
 2447 postmenopausal women. *Nutrition*. 2012;28(9):896-900. PMID: 22480799.
 2448 <http://www.ncbi.nlm.nih.gov/pubmed/22480799>.
- 2449 205. Vilela AA, Farias DR, Eshriqui I, Vaz JoS, Franco-Sena AB, Castro MB, et al.
 2450 Prepregnancy Healthy Dietary Pattern Is Inversely Associated with Depressive
 2451 Symptoms among Pregnant Brazilian Women. *J Nutr*. 2014;144(10):1612-8. PMID:
 2452 25143375. <http://www.ncbi.nlm.nih.gov/pubmed/25143375>.
- 2453 206. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin
 2454 D and Calcium; Edited by A Catharine Ross CLT, Ann L Yaktine, and Heather B Del
 2455 Valle. *Dietary Reference Intakes for Calcium and Vitamin D*. Washington (DC): National
 2456 Academies Press (US); 2011.

- 2457 207. Benetou V, Orfanos P, Pettersson-Kymmer U, Bergstrom U, Svensson O, Johansson I, et
 2458 al. Mediterranean diet and incidence of hip fractures in a European cohort. *Osteoporos*
 2459 *Int.* 2013;24(5):1587-98. PMID: 23085859.
 2460 <http://www.ncbi.nlm.nih.gov/pubmed/23085859>.
- 2461 208. Bullo M, Amigo-Correig P, Marquez-Sandoval F, Babio N, Martinez-Gonzalez MA,
 2462 Estruch R, et al. Mediterranean diet and high dietary acid load associated with mixed
 2463 nuts: effect on bone metabolism in elderly subjects. *J Am Geriatr Soc.* 2009;57(10):1789-
 2464 98. PMID: 19807791. <http://www.ncbi.nlm.nih.gov/pubmed/19807791>.
- 2465 209. Dai Z, Butler LM, van Dam RM, Ang LW, Yuan JM, Koh WP. Adherence to a
 2466 vegetable-fruit-soy dietary pattern or the Alternative Healthy Eating Index is associated
 2467 with lower hip fracture risk among Singapore Chinese. *J Nutr.* 2014;144(4):511-8. PMID:
 2468 24572035. <http://www.ncbi.nlm.nih.gov/pubmed/24572035>.
- 2469 210. Feart C, Lorrain S, Ginder Coupez V, Samieri C, Letenneur L, Paineau D, et al.
 2470 Adherence to a Mediterranean diet and risk of fractures in French older persons.
 2471 *Osteoporos Int.* 2013;24(12):3031-41. PMID: 23783645.
 2472 <http://www.ncbi.nlm.nih.gov/pubmed/23783645>.
- 2473 211. Langsetmo L, Hanley DA, Prior JC, Barr SI, Anastassiades T, Towheed T, et al. Dietary
 2474 patterns and incident low-trauma fractures in postmenopausal women and men aged \geq 50
 2475 y: a population-based cohort study. *Am J Clin Nutr.* 2011;93(1):192-9. PMID: 21068350.
 2476 <http://www.ncbi.nlm.nih.gov/pubmed/21068350>.
- 2477 212. Langsetmo L, Poliquin S, Hanley DA, Prior JC, Barr S, Anastassiades T, et al. Dietary
 2478 patterns in Canadian men and women ages 25 and older: relationship to demographics,
 2479 body mass index, and bone mineral density. *BMC Musculoskelet Disord.* 2010;11:20.
 2480 PMID: 20109205. <http://www.ncbi.nlm.nih.gov/pubmed/20109205>.
- 2481 213. McTiernan A, Wactawski-Wende J, Wu L, Rodabough RJ, Watts NB, Tylavsky F, et al.
 2482 Low-fat, increased fruit, vegetable, and grain dietary pattern, fractures, and bone mineral
 2483 density: the Women's Health Initiative Dietary Modification Trial. *Am J Clin Nutr.*
 2484 2009;89(6):1864-76. PMID: 19403636. <http://www.ncbi.nlm.nih.gov/pubmed/19403636>.
- 2485 214. Monjardino T, Lucas R, Ramos E, Barros H. Associations between a priori-defined
 2486 dietary patterns and longitudinal changes in bone mineral density in adolescents. *Public*
 2487 *Health Nutr.* 2014;17(1):195-205. PMID: 23149164.
 2488 <http://www.ncbi.nlm.nih.gov/pubmed/23149164>.
- 2489 215. Monma Y, Niu K, Iwasaki K, Tomita N, Nakaya N, Hozawa A, et al. Dietary patterns
 2490 associated with fall-related fracture in elderly Japanese: a population based prospective
 2491 study. *BMC Geriatr.* 2010;10:31. PMID: 20513246.
 2492 <http://www.ncbi.nlm.nih.gov/pubmed/20513246>.
- 2493 216. Noh HY, Song YJ, Lee JE, Joung H, Park MK, Li SJ, et al. Dietary patterns are
 2494 associated with physical growth among school girls aged 9-11 years. *Nutr Res Pract.*
 2495 2011;5(6):569-77. PMID: 22259683. <http://www.ncbi.nlm.nih.gov/pubmed/22259683>.
- 2496 217. Park SJ, Joo SE, Min H, Park JK, Kim Y, Kim SS, et al. Dietary patterns and
 2497 osteoporosis risk in postmenopausal Korean women. *Osong Public Health Res Perspect.*
 2498 2012;3(4):199-205. PMID: 24159515. <http://www.ncbi.nlm.nih.gov/pubmed/24159515>.
- 2499 218. Samieri C, Ginder Coupez V, Lorrain S, Letenneur L, Alles B, Feart C, et al. Nutrient
 2500 patterns and risk of fracture in older subjects: results from the Three-City Study.
 2501 *Osteoporos Int.* 2013;24(4):1295-305. PMID: 22976577.
 2502 <http://www.ncbi.nlm.nih.gov/pubmed/22976577>.

- 2503 219. Wosje KS, Khoury PR, Claytor RP, Copeland KA, Hornung RW, Daniels SR, et al.
2504 Dietary patterns associated with fat and bone mass in young children. *Am J Clin Nutr.*
2505 2010;92(2):294-303. PMID: 20519562. <http://www.ncbi.nlm.nih.gov/pubmed/20519562>.
2506 220. George SM, Ballard-Barbash R, Manson JE, Reedy J, Shikany JM, Subar AF, et al.
2507 Comparing indices of diet quality with chronic disease mortality risk in postmenopausal
2508 women in the Women's Health Initiative Observational Study: evidence to inform
2509 national dietary guidance. *Am J Epidemiol.* 2014;180(6):616-25. PMID: 25035143.
2510 <http://www.ncbi.nlm.nih.gov/pubmed/25035143>.
2511
2512
2513

2514 **Table D2.1.** *AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults, 2013*

<p>Critical Question 4a. Among overweight and obese adults, what is the efficacy/effectiveness of a comprehensive lifestyle intervention program (i.e., comprised of diet, physical activity, and behavior therapy) in facilitating weight loss or maintenance of lost weight?</p> <p>Critical Question 4b. What characteristics of delivering comprehensive lifestyle interventions (e.g., frequency and duration of treatment, individual versus group sessions, onsite versus telephone/email contact) are associated with greater weight loss or weight loss maintenance?</p>		
Intervention/Question	Included Studies	Evidence Statement (Strength of Evidence)
3.4.1. Description of the Diet, Physical Activity, and Behavior Therapy Components in High-Intensity, Onsite Lifestyle Interventions	12 RCTs	ES1. The principal components of an effective high-intensity, on-site comprehensive-lifestyle intervention include: 1) prescription of a moderately-reduced calorie diet; 2) a program of increased physical activity; and 3) the use of behavioral strategies to facilitate adherence to diet and activity recommendations. (<i>High</i>)
3.4.2. Comprehensive Interventions Compared with Usual Care, Minimal Care, or No-Treatment Control	15 RCTs	ES 2a (Short-Term Weight Loss). In overweight and obese individuals in whom weight loss is indicated and who wish to lose weight, comprehensive lifestyle interventions consisting of diet, physical activity, and behavior therapy (all 3 components) produce average weight losses of up to 8 kg in 6 months of frequent (i.e., initially weekly), onsite treatment provided by a trained interventionist* in group or individual sessions. Such losses (which can approximate reductions of 5% to 10% of initial weight) are greater than those produced by usual care (i.e., characterized by the limited provision of advice or educational materials). Comparable 6-month weight losses have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. (<i>High</i>)
		ES 2b (Intermediate-Term Weight Loss). Longer-term comprehensive lifestyle interventions, which additionally provide weekly to monthly on-site treatment for another 6 months, produce average weight losses of up to 8 kg at 1 year, losses which are greater than those resulting from usual care. Comparable 1-year weight losses have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. (<i>Moderate</i>)
		ES 2c (Long-Term Weight Loss). Comprehensive lifestyle interventions which, after the first year, continue to provide bimonthly or more frequent intervention contacts, are associated with gradual weight regain of 1 to 2 kg/year (on average), from the weight loss achieved at 6 to 12 months. Long-term (>1 year) weight losses, however, remain larger than those associated with usual care. Comparable findings have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. (<i>High</i>)
3.4.3. Efficacy/Effectiveness of Electronically Delivered, Comprehensive Interventions in Achieving Weight Loss Evidence Statement	13 RCTs	ES 3. Electronically delivered, comprehensive weight loss interventions developed in academic settings, which include frequent self-monitoring of weight, food intake, and physical activity—as well as personalized feedback from a trained interventionist*—can produce weight loss of up to 5 kg at 6 to 12 months, a loss which is greater than that resulting from no or minimal intervention (i.e., primarily knowledge based) offered on the internet or in print. (<i>Moderate</i>)
3.4.4. Efficacy/Effectiveness of Comprehensive, Telephone-Delivered Lifestyle Interventions in Achieving Weight Loss	3 RCTs	ES 4. In comprehensive lifestyle interventions that are delivered by telephone or face-to-face counseling, and which also include the use of either commercially-prepared prepackaged meals or an interactive web based program, the telephone delivered and face-to-face delivered interventions produced similar mean net weight losses of approximately 5 kg at 6 months and 24 months, compared with a usual care control group.

		(<i>Low</i>)
3.4.5. Efficacy/Effectiveness of Comprehensive Weight Loss Programs in Patients Within a Primary Care Practice Setting Compared With Usual Care	4 RCTs	ES 5. In studies to date, low to moderate-intensity lifestyle interventions for weight loss provided to overweight or obese adults by primary care practices alone, have not been shown to be effective. (<i>Low</i>)
3.4.6. Efficacy/Effectiveness of Commercial-Based, Comprehensive Lifestyle Interventions in Achieving Weight Loss	4 RCTs	ES 6. Commercial-based, comprehensive weight loss interventions that are delivered in person have been shown to induce an average weight loss of 4.8 kg to 6.6 kg at 6 months in 2 trials when conventional foods are consumed and 6.6 kg to 10.1 kg at 12 months in 2 trials with provision of prepared food, losses that are greater than those produced by minimal-treatment control interventions. (<i>Low</i>)
3.4.7. Efficacy/Effectiveness of Very Low-Calorie Diets, as Used as Part of a Comprehensive Lifestyle Intervention, in Achieving Weight Loss	4 RCTs	ES 7a. Comprehensive, high intensity on-site lifestyle interventions that include a medically supervised very low-calorie diet (often defined as <800 kcal/day), as provided by complete meal replacement products, produce total weight loss of approximately 14.2 kg to 21 kg over 11 to 14 weeks, which is larger than that produced by no intervention or a usual care control group (i.e., advice and education only). (<i>High</i>)
		ES 7b. Following the cessation of a high intensity lifestyle intervention with a medically supervised very-low calorie diet of 11 to 14 weeks, weight regain of 3.1 kg to 3.7 kg has been observed during the ensuing 21 to 38 weeks of non-intervention follow-up. (<i>High</i>)
		ES 7c. The prescription of various types (resistance or aerobic training) and doses of moderate intensity exercise training (e.g., brisk walking 135 to 250 minutes/week), delivered in conjunction with weight loss maintenance therapy does not reduce the amount of weight regained after the cessation of the very-low calorie diet, as compared with weight loss maintenance therapy alone. (<i>Low</i>)
3.4.8. Efficacy/Effectiveness of Comprehensive Lifestyle Interventions in Maintaining Lost Weight	14 RCTs	ES 8a. After initial weight loss, some weight regain can be expected, on average, with greater regain observed over longer periods of time. Continued provision of a comprehensive weight loss maintenance program (onsite or by telephone), for periods of up to 2.5 years following initial weight loss, reduces weight regain, as compared to the provision of minimal intervention (e.g., usual care). The optimal duration of weight loss maintenance programs has not been determined. (<i>Moderate</i>)
		ES 8b. 35% to 60% of overweight/obese adults who participate in a high intensity long-term comprehensive lifestyle intervention maintain a loss of $\geq 5\%$ of initial body weight at ≥ 2 year's follow-up (post-randomization). (<i>Moderate</i>)
3.4.9. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss: Intervention	10 RCTs	ES 9a (Moderate-Intensity Interventions). Moderate intensity, on-site comprehensive lifestyle interventions, which provide an average of 1 to 2 treatment sessions per month typically produce mean weight losses of 2 kg to 4 kg in 6 to 12 months, losses which generally are greater than those produced by usual care (i.e., minimal intervention control group). (<i>High</i>)
		ES 9b (Low-intensity Interventions). Low intensity, on-site comprehensive lifestyle interventions, which provide fewer than monthly treatment sessions do not consistently produce weight loss when compared to usual care. (<i>Moderate</i>)
		ES 9c (Effect of intervention intensity). When weight loss with each intervention intensity (i.e., low, moderate, and high) is compared to usual care, high-intensity lifestyle interventions (≥ 14 sessions in 6 months) typically produce greater net-of-control weight losses than low-to-moderate intensity interventions. (<i>Moderate</i>)

3.4.10. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss or Weight	15 RCTs	ES 10. There do not appear to be substantial differences in the size of the weight losses produced by individual- and group-based sessions in high-intensity, comprehensive lifestyle intervention delivered on site by a trained interventionist*. (<i>Low</i>)
3.4.11. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss or Weight Loss Maintenance: Onsite Versus Electronically Delivered Interventions		ES 11. Weight losses observed in comprehensive lifestyle interventions, which are delivered onsite by a trained interventionist* in initially weekly and then biweekly group or individual sessions, are generally greater than weight losses observed in comprehensive interventions that are delivered by Internet or email and which include feedback from a trained interventionist. (<i>Low</i>)

2515

2516

Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change

INTRODUCTION

Individual behavior change lies at the inner core of the social-ecological model that forms the basis of the 2015 Dietary Guidelines for American Advisory Committee (DGAC) conceptual model (see *Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence*). For this reason, it is crucial to identify the behavioral strategies that individuals living in the United States can follow to improve their healthy lifestyle behaviors as well as the key contextual factors that facilitate the ability of individuals to consume healthy diets.

In the past, American families seldom consumed food prepared outside their homes and, for the most part, consumed their meals as a family unit. However, these behaviors have changed dramatically in recent years. Today, 33 percent of calories are consumed outside the home and it is becoming more common for individuals to eat alone and to bring meals prepared outside into their homes (see *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*). Eating away from home is associated with increased caloric intake and poorer dietary quality compared to eating at home.¹ As recognized by the 2010 DGAC these major changes in eating behaviors can be expected to have a negative impact on the quality of the diets consumed and the risk of obesity among the U.S. population.²

Other individual lifestyle behaviors related to dietary intakes and obesity risk also have changed in recent decades. The U.S. population has become increasingly sedentary,³ with daily hours of screen time exposure becoming a serious public health concern due to its potential negative influence on dietary and weight outcomes. For example, it has been hypothesized that TV viewing time has a negative influence on dietary habits of individuals because of unhealthy snacking while watching TV and through exposure to advertisements of unhealthy food products.⁴ In turn, excess caloric intake coupled with sedentary time directly resulting from excessive TV may increase the risk of obesity. Suboptimal sleep patterns associated with today's busy lives also have been identified as a potential risk factor for poor dietary behaviors and body weight outcomes.⁵

In response to these trends, interest has grown in the potential of behavioral strategies that individuals can use to improve their dietary behaviors. Specifically, self-monitoring of diet, physical activity, and body weight has been identified as a potential key component of successful healthy lifestyle interventions.⁶ Diet self-monitoring may, in turn, be facilitated by the availability and use of menus displaying calorie labels and the Nutrition Facts label on packaged foods.

38 Recognizing the importance of these dietary and lifestyle behaviors to the health and well-being
39 of the U.S. population, the DGAC reviewed recent evidence to address questions on the
40 relationship between eating out, family shared meals, sedentary behavior, and diet and weight
41 outcomes. The DGAC also sought to examine associations between sleep patterns, dietary
42 intakes, and obesity risk. However, after conducting preliminary literature searches, the
43 Committee determined sleep patterns was an emerging area with an insufficient body of
44 evidence and did not include specific questions on this topic.

45

46 The DGAC also focused on identifying evidence that could provide individuals with tools to
47 improve their dietary choices and body weight status. Specifically, the Committee reviewed
48 recent evidence on the impact of diet and weight self-monitoring, and on use of food and menu
49 labels on dietary intake and weight outcomes. The DGAC was interested in reviewing the
50 evidence on the use of mobile health (m-health) technologies to improve dietary and weight
51 outcomes, and after a preliminary review was conducted, determined that this, too, was an
52 emerging area and that a full evidence review was premature. However, key m-health studies
53 focused on self-monitoring were identified, and thus were reviewed as part of the body of
54 evidence on self-monitoring. This chapter addresses sedentary behaviors, but not physical
55 activity behaviors in general because these are addressed in *Part D. Chapter 7: Physical*
56 *Activity*.

57

58 Consistent with the DGAC conceptual model presented in *Part B. Chapter 1: Introduction*, this
59 chapter also addresses major contextual factors that influence the ability of individuals to
60 implement healthy dietary and other lifestyles, including the prevention of sedentary behaviors.
61 The Committee focused on the association between diet, body weight, and chronic disease
62 outcomes and two contextual factors that are highly relevant in the United States—household
63 food insecurity and acculturation.

64

65 Household food insecurity is defined as “access to enough food for an active, healthy life. It
66 includes at a minimum (a) the ready availability of nutritionally adequate and safe foods, and (b)
67 an assured ability to acquire acceptable foods in socially acceptable ways (e.g., without resorting
68 to emergency food supplies, scavenging, stealing, or other coping strategies)”.⁷ Thus, household
69 food insecurity is a condition that exists whenever the availability of nutritionally adequate and
70 safe foods, or the ability to acquire acceptable foods in socially acceptable ways, is limited or
71 uncertain.⁷ In 2013, 49.1 million people in the United States lived in food insecure households,
72 and of these, 8.6 million are children.¹ Household food insecurity is suggested to be an
73 independent risk factor for poor physical and mental health outcomes across the lifespan.^{8,9}

74

75 The second contextual factor the DGAC addressed—acculturation—reflects that the United
76 States continues to be a nation of immigrants.^{10,11} Acculturation has been defined both as the
77 “process by which immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a

78 new culture”,¹² and as the “gradual exchange between immigrants’ original attitudes and
 79 behavior and those of the host culture”.¹³ Acculturation is relevant for individual dietary
 80 behaviors because evidence suggests that the healthy lifestyles with which immigrants arrive
 81 deteriorate as they integrate or assimilate into mainstream American culture.¹⁴ Moreover,
 82 evidence suggests that to be effective in helping immigrants retain their healthy lifestyles,
 83 nutrition education programs, including those that are a part of food assistance programs, must
 84 be tailored to their different levels of acculturation.¹⁴

85
 86 Given the strong relevance of household food insecurity and acculturation as contextual factors
 87 influencing healthy lifestyles, the DGAC examined associations between them and diet, obesity
 88 risk, and whenever possible, corresponding chronic disease risk factors.

89

90 **LIST OF QUESTIONS**

91 **Eating Out**

- 92 1. What is the relationship between eating out and/or take away meals and body weight in
 93 children and adults?

94

95 **Family Shared Meals**

- 96 2. What is the relationship between frequency and regularity of family shared meals and
 97 measures of dietary intake in U.S. population groups?
 98 3. What is the relationship between frequency and regularity of family shared meals and
 99 measures of body weight and obesity in U.S. population groups?

100

101 **Sedentary Behavior, Including Screen Time**

- 102 4. What is the relationship between sedentary behavior and measures of dietary intake and body
 103 weight in adults?
 104 5. How effective are behavioral interventions in youth that focus on reducing recreational
 105 sedentary screen time and improving physical activity and/or diet?

106

107 **Self-Monitoring**

- 108 6. What is the relationship between use of diet and body weight self-monitoring strategies and
 109 body weight outcomes in adults and youth?

110

111 **Food and Menu Labeling**

- 112 7. What is the relationship between knowledge and use of food and menu labels and measures
113 of dietary intake in U.S. population groups?

114

115 **Household Food Insecurity (HFI)**

- 116 8. What is the relationship between household food insecurity (HFI) and measures of dietary
117 intake and body weight?

118

119 **Acculturation**

- 120 9. What is the relationship between acculturation and measures of dietary intake?
121 10. What is the relationship between acculturation and body weight?
122 11. What is the relationship between acculturation and risk of cardiovascular disease (CVD)?
123 12. What is the relationship between acculturation and risk of type 2 diabetes?

124

125 **METHODOLOGY**

126 All of the questions covered in this chapter— eating out, family shared meals, sedentary
127 behavior, self-monitoring, food and menu labeling, household food insecurity, and
128 acculturation—were answered using Nutrition Evidence Library (NEL) systematic reviews. A
129 description of the NEL process is provided in *Part C: Methodology*. All reviews were conducted
130 in accordance with NEL methodology, and the DGAC made all substantive decisions required
131 throughout the process to ensure that the most complete and relevant body of evidence was
132 identified and evaluated to answer each question. All steps in the process were documented to
133 ensure transparency and reproducibility. Specific information about individual systematic
134 reviews can be found at www.NEL.gov, including the search strategy, inclusion and exclusion
135 criteria, a complete list of included and excluded articles, and detailed documentation describing
136 the included studies and the body of evidence. A link to this website is provided following each
137 evidence review.

138

139 **EATING OUT**

140 The majority of Americans consume meals outside of the home one or more times per week (see
141 *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*). The
142 2010 DGAC concluded that “strong and consistent evidence indicates that children and adults
143 who eat fast food are at increased risk of weight gain, overweight, and obesity”.² With this
144 relationship as a foundation, the 2015 DGAC updated and expanded the review of the “eating
145 out” topic. Specifically, the “fast food” category was broadened to capture other types of eating
146 out venues (e.g., quick serve, casual, formal restaurants, and grocery store take-out).

147 Terminology used to define the exposure was modified from “eating out,” to the broader term
 148 “eating out and/or take away meals” to reflect the inclusion of meals eaten out at a broader array
 149 of restaurant venues as well as takeout or ready-to-eat foods or meals purchased and consumed
 150 either away from or in the home. The population of interest remained healthy individuals ages 2
 151 years and older.

152

153 **Question 1: What is the relationship between eating out and/or take away meals**
 154 **and body weight in children and adults?**

155 **Source of evidence:** Update to 2010 DGAC’s NEL systematic review

156 **Conclusion**

157 Among adults, moderate evidence from prospective cohort studies in populations ages 40 years or
 158 younger at baseline indicates higher frequency of fast food consumption is associated with higher
 159 body weight, body mass index (BMI), and risk for obesity. **DGAC Grade: Moderate**

160

161 Among children, limited evidence from prospective cohort studies in populations ages 8 to 16
 162 years at baseline suggests that higher frequency of fast food consumption is associated with
 163 increased adiposity, BMI z-score, or risk of obesity during childhood, adolescence, and during the
 164 transition from adolescence into adulthood. **DGAC Grade: Limited**

165

166 Insufficient evidence is available to assess the relationship between frequency of other types of
 167 restaurant and takeout meals and body weight outcomes in children and adults. **DGAC Grade:**
 168 **Grade Not assignable**

169

170 **Implications**

171 Given that one-third of calories are consumed outside of the home (see *Part D. Chapter 1: Food*
 172 *and Nutrient Intakes, and Health: Current Status and Trends*), individuals should limit the
 173 frequency of eating at fast-food establishments. When eating out, one should choose healthy foods
 174 and beverages within their calorie needs to avoid increases in body weight.

175

176 **Review of the Evidence**

177 Fifteen prospective studies examined the relationship between eating out and/or take away meals
 178 and measures of body weight in adults and children.¹⁵⁻²⁹ Eleven studies in the United States^{16-18,}
 179 ^{20-23, 25-28} and four international studies (one each from Canada, the United Kingdom, Australia,
 180 and Spain)^{15, 19, 24, 29} were reviewed. Men and women and boys and girls were well represented
 181 and the majority of studies within the United States included diverse populations.

182

183 In children, seven prospective cohort studies^{19, 21, 22, 24, 27-29} examined the relationship between
184 frequency of fast-food meals, or consumption of other types of meals and anthropometric
185 outcomes and, overall, found mixed results. Six studies examined fast-food meals^{19, 21, 22, 24, 28, 29}.
186 three studies^{19, 28, 29} indicated increased fast food intake, particularly more than twice per week,
187 was associated with increased risk of obesity, BMI/BMI z-score or body fat, two^{22, 24} found no
188 association, and one²¹ found no association in boys and a negative association in girls. Two
189 studies looked at a variety of non-fast-food meals away from home, using varying definitions of
190 food establishments and meal types and reported mixed findings for a relationship with weight-
191 related outcomes.^{27, 28}

192
193 In adolescents transitioning to adulthood, one study found high baseline frequency of fast food
194 intake was associated with increased BMI z-scores at 5-year follow-up.²⁵ In adults, evidence
195 consistently demonstrated a relationship between higher frequency of fast-food meal
196 consumption and body weight outcomes. Five prospective cohort studies (three cohorts) reported
197 a higher frequency of intake of meals from fast food locations, or intake exceeding once per
198 week, was associated with higher weight gain, BMI, and risk of obesity.^{17, 18, 20, 23, 26} A
199 “moderate” grade was assigned (as opposed to the “strong” grade assigned by the 2010 DGAC)
200 because the evidence based was small (five studies focused on fast food, three from the same
201 cohort), all of which were prospective cohort studies; few studies controlled for energy intake
202 and no study reported actual food consumed; and the method of measurement of “eating out”
203 varied among studies. Evidence related to the association between frequency of meals from other
204 types of restaurants and intake of all takeout meals and weight is limited, but indicates traditional
205 restaurant meal frequency may not be associated with weight outcomes.^{17, 18} Two studies^{15, 16}
206 examined total meals away from home or meal types eaten away from home, which came from
207 both fast food and restaurant locations, and reported frequency was associated with increased
208 body weight outcomes for most meal types. Two studies from the same cohort found no
209 significant relationship between frequency of meals from restaurants (non-fast-food
210 establishments), and weight-related outcomes.

211
212 *For additional details on this body of evidence, visit: <http://NEL.gov/topic.cfm?cat=3371>*

213 214 **FAMILY SHARED MEALS**

215 Data from cross-sectional studies suggest that when families share meals, they achieve better diet
216 quality and improved nutrient intake, and to some extent, are better able to maintain appropriate
217 body weight.³⁰⁻³⁶ The definition of family shared meals in the literature varies, with some
218 defining it as the number of a specific meal eaten together (e.g., dinner), or any meal, prepared at
219 home or outside of home, that is shared among individuals living in the same household.³⁷
220 Family mealtime may act as a protective factor for many nutritional health-related problems. For
221 example, they provide an opportunity for parents to model good eating behaviors and create a

222 positive atmosphere by providing time for social interaction and thus a sense of social support
 223 for all members.^{38, 39} Shared meals may be important in every stage of the lifecycle to support
 224 healthy growth, development, and weight, though the evidence for adults is mixed. The
 225 importance of the family in supporting positive behaviors is clearly part of the life course
 226 approach embodied in the DGAC’s conceptual model (see *Part B. Chapter 2: 2015 DGAC*
 227 *Themes and Recommendations: Integrating the Evidence*). As a result, the Committee decided
 228 to explore the relationship between family shared meals and dietary intake as well as weight
 229 outcomes from high-quality epidemiological studies to determine if there is a cause and effect
 230 association.

231

232 **Question 2: What is the relationship between frequency/regularity of family**
 233 **shared meals and measures of dietary intake in U.S. population groups?**

234 **Source of evidence:** NEL systematic review

235 **Conclusion**

236 Insufficient evidence on the association between frequency of family shared meals and measures of
 237 dietary intake is available to draw a conclusion. **DGAC Grade: Grade not assignable**

238

239 **Implications**

240 The DGAC determined that a grade was not assignable due to the insufficient evidence for this
 241 question. Therefore, no implications were developed.

242

243 **Review of the Evidence**

244 Two studies in the United States with the duration of 5 to 10 years from one prospective cohort
 245 examined the relationship between frequency/regularity of family meals and measures of dietary
 246 intake in U.S. population groups.^{40, 41} The studies included adolescents transitioning from early
 247 to middle adolescence (middle school to high school)⁴⁰ and adolescents transitioning to early
 248 adulthood.⁴¹ These studies found more frequent consumption of family meals was associated
 249 with improved dietary intake, specifically an increase in fruits and/or vegetables, and calcium-
 250 rich or milk-based foods.^{40, 41} Given that the evidence is limited to these two studies using data
 251 from the same cohort at two time points, the Committee could not assign a grade.

252

253 *For additional details on this body of evidence, visit:*

254 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250455

255

256 **Question 3: What is the relationship between frequency/regularity of family**
 257 **shared meals and measures of body weight in U.S. population groups?**

258 **Source of evidence:** NEL systematic review

259 **Conclusion**

260 Limited evidence from prospective studies shows inconsistent relationships between the number of
 261 family shared meals and body weight of children and adolescents. **DGAC Grade: Limited**

262

263 **Implications**

264 The very limited evidence available on the relationship between family shared meals and measures
 265 of body weight precludes developing implications for this question. Shared meals may be
 266 important in every stage of the lifecycle to support healthy growth, development, and weight;
 267 however, more studies are warranted to determine if there is a direct effect. In the absence of such
 268 studies, meal times may still be an optimal time for parents to provide role modeling behaviors in
 269 terms of what foods to eat and, for the elderly encouragement to eat given the social support of
 270 other individuals.

271

272 **Review of the Evidence**

273 Six studies, which included one randomized control trial (RCT)⁴² and five prospective cohort
 274 studies (4 cohorts)⁴³⁻⁴⁷ examined the relationship between frequency/regularity of family meals
 275 and measures of body weight in U.S. populations. The study duration for the RCT was 6
 276 months⁴² and the prospective cohort studies⁴³⁻⁴⁷ ranged in duration from 1 to 5 years. The study
 277 population was children and adolescents ages 4 to 15 years.

278

279 Three out of four prospective cohort studies found no significant association between the
 280 frequency of family shared meals, BMI, or overweight status. Evidence from one prospective
 281 study (two articles) showed that an increase in the frequency of family shared meals lowered the
 282 likelihood of becoming overweight or the persistence of overweight. One study found that
 283 among overweight children, eating more family breakfast and dinner meals was associated with
 284 lower likelihood of becoming overweight or remaining overweight over a 4-year period. Another
 285 article reported children who typically ate more breakfast meals with their families had a lower
 286 rate of increase in BMI over 5 years. The number of dinner meals eaten with the family was not
 287 associated with a change in BMI.

288

289 One RCT included an intervention that simultaneously focused on four household routines,
 290 including family shared meals.⁴² Although a reduction in body weight occurred, family meal
 291 frequency did not change.⁴²

292 This body of evidence had several limitations, including that studies did not use a standard
293 definition for family shared meals, two studies assessed only family dinners, two studies
294 assessed breakfast and dinner meals, and two studies assessed all meals. No study assessed the
295 quality or source of meals consumed.

296
297 *For additional details on this body of evidence, visit:*

298 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250460

299

300 **SEDENTARY BEHAVIOR, INCLUDING SCREEN TIME**

301 The *Physical Activity Guidelines for Americans* recommend that adults engage in at least 150
302 minutes (2.5 hours) of moderate- to vigorous-intensity physical activity each week and two days
303 a week of strength training.⁴⁸ Youth ages 6 to 17 years should engage in 60 minutes or more of
304 daily physical activity.⁴⁸ Unfortunately, the vast majority of Americans do not get the physical
305 activity they need; only 20 percent of adults meet both the aerobic and strength training
306 recommendations and less than 20 percent of adolescents meet the youth guideline.^{49, 50} In
307 addition, one-third of adults engage in no leisure-time physical activity.⁵¹ Regular physical
308 activity is associated with myriad health benefits, including reduced risk of chronic disease, and
309 physical, mental, and cognitive benefits, irrespective of body weight.⁴⁸ Physical inactivity is
310 associated with increased risk of overweight and obesity, CVD, type 2 diabetes, breast and colon
311 cancer, and overall all-cause mortality.⁵²

312

313 Sedentary behavior, which refers to any waking activity predominantly done while in a sitting or
314 reclining posture, is gaining considerable public health interest as a chronic disease risk factor
315 and therefore a potential area for interventions to target, with reducing screen time often a focus.
316 The American Academy of Pediatrics (AAP) recommends no more than 2 hours a day of screen
317 time (including television and other types of media) for children ages 2 years and older and none
318 for children younger than age 2 years.⁵³ However, children ages 8 to 18 years spend an average
319 of 7 hours on screen time each day.⁵⁴ The U.S. Report Card on Physical Activity for Youth gave
320 the sedentary behavior indicator a grade of “D” for youth meeting the AAP’s screen time
321 recommendation.⁵⁵ Rates of screen time are similar among males and females, yet
322 disproportionately higher for African American youth compared to Caucasian youth (63.3
323 percent not meeting AAP recommendation vs. 44.6 percent).⁵⁶ For this topic, two questions were
324 addressed by the DGAC, the first with a NEL systematic review focused on the transition from
325 childhood to adulthood and sedentary behavior in adults. The second question used the 2014
326 Community Preventive Services Task Force Obesity Prevention and Control (Community Guide)
327 systematic review to examine the effectiveness of interventions among youth to reduce sedentary
328 screen time and increase physical activity.

329 **Question 4: What is the relationship between sedentary behavior and dietary**
 330 **intake and body weight in adults?**

331 **Source of evidence:** NEL systematic review

332 **Conclusion**

333 Moderate and consistent evidence from prospective studies that followed cohorts of youth into
 334 adulthood supports that adults have a higher body weight and incidence of overweight and obesity
 335 when the amount of TV viewing is higher in childhood and adolescence. **DGAC Grade:**

336 **Moderate**

337
 338 Moderate evidence from prospective studies suggests no association between sedentary behavior in
 339 adulthood and change in body weight, body composition, or incidence of overweight or obesity in
 340 adulthood. **DGAC Grade: Moderate**

341
 342 Insufficient evidence exists to address the association between sedentary behavior and dietary
 343 intake in adults. **DGAC Grade: Grade Not Assignable**

344
 345 **Implications**

346 Sedentary behavior, including TV watching and screen time, should be limited during childhood to
 347 lower the likelihood of excess body weight or overweight and obesity in adulthood. Federal, state,
 348 and local policies and programs to support school and community-based programs to identify and
 349 reduce sedentary behavior among children and adolescents are needed to help them achieve and
 350 maintain healthy weight status as they transition into adulthood. Although an apparent lack of
 351 association exists between sedentary behavior and change in body weight status in adulthood,
 352 adults are encouraged to adopt and sustain levels of physical activity consistent with the *Physical*
 353 *Activity Guidelines for Americans* to promote health and to achieve and sustain a healthy weight
 354 status.

355
 356 **Review of the Evidence**

357 This evidence review included 23 studies from 18 prospective cohorts that examined the
 358 relationship between sedentary behavior and body weight status in adults.⁵⁷⁻⁷⁹ Study locations
 359 included six studies from Australia,^{59, 60, 65, 74, 75, 77} six studies from the United Kingdom,^{61, 69, 70,}
 360 ^{73, 76, 78} seven studies from the United States,^{57, 58, 62, 66, 67, 71, 79} two studies from New Zealand,^{63, 64}
 361 and one study each from Canada⁷² and Spain.⁶⁸ The mean age of participants ranged from 23
 362 years to 60 years. Longitudinal studies followed participants from childhood (5 to 16 years) to
 363 adulthood (21 to 45 years). Three studies (two cohorts)^{57, 59, 75} had an all-female sample and the
 364 remainder of the studies included both males and females.

365 Increasing levels of TV viewing during childhood and adolescence predicted higher BMI^{64, 65, 69,}
366 ⁷⁶ and increased incidence of overweight and obesity in adulthood.^{58, 64, 65, 76} The lack of
367 association between adult sedentary behavior (TV viewing, commute time or composite
368 measures of sedentary behavior) and body weight change or body weight status are mostly
369 consistent, despite methodological differences in measurement of sedentary behavior. Among
370 two studies that assessed the relationship between sedentary behavior in adulthood and dietary
371 intake, one study found an association between TV viewing and lower compliance with
372 recommended dietary guidance.⁶⁶ The other study found that more TV viewing was associated
373 with greater intake of calories from fat, but not total calories or calories from sweets.⁷¹
374

375 Methodological approaches differed with regard to population and cohort size, types of sedentary
376 behavior considered, and timeframes studied. Only one study directly measured sedentary
377 behavior⁶¹ and few studies adjusted analysis for energy intake and other potential mediators,
378 such as dietary intake. The majority of studies were conducted in Caucasian populations;
379 therefore diverse ethnic and racial groups were underrepresented.
380

381 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3343>
382

383 **Question 5: How effective are behavioral interventions in youth that focus on**
384 **reducing recreational sedentary screen time and improving physical activity**
385 **and/or diet?**

386 **Source of evidence:** *Community Preventive Services Task Force Obesity Prevention and*
387 *Control: Behavioral Interventions that Aim to Reduce Recreational Sedentary Screen Time*
388 *(Community Guide)*⁸⁰ Available at:
389 <http://www.thecommunityguide.org/obesity/RRbehavioral.html>

390 **Conclusion**

391 The DGAC concurs with the Community Guide,⁸⁰ which found strong evidence that behavioral
392 interventions are effective in reducing recreational sedentary screen time among children ages 13
393 years and younger. Limited evidence was available to assess the effectiveness of these
394 interventions among adults and no evidence was available for adolescents ages 14 years and older.

395 **DGAC Grade: Strong**

396

397 **Implications**

398 The Community Guide identified effective behavioral interventions to reduce recreational screen
399 time and recommended that they be implemented in a variety of settings. The DGAC concurs with
400 this recommendation because of the potential for these interventions to have beneficial effects on
401 children's diet and weight status. Multifaceted interventions to reduce recreational sedentary screen

402 time may include home, school, neighborhood, and pediatric primary care settings, and emphasize
403 parental, family, and peer-based social support, coaching or counseling sessions, and electronic
404 tracking and monitoring of the use of screen-based technologies.
405

406 **Review of the Evidence**

407 The Community Guide review classified behavioral screen time interventions as: 1) screen-time-
408 only interventions that focus only on reducing recreational sedentary screen time, and 2) screen-
409 time-plus interventions, which focus on reducing recreational sedentary screen time and
410 increasing physical activity and/or improving diet. These interventions are used to teach
411 behavioral self-management skills through one or more of the following components: classroom-
412 based education, tracking and monitoring, coaching or counseling sessions, and family-based or
413 peer social support. The Community Guide review focused on both high- and low-intensity
414 interventions to reduce sedentary behavior in youth. High-intensity interventions included the
415 use of an electronic monitoring device to limit screen time or at least three personal or computer-
416 tailored interactions. Low-intensity interventions included two or fewer personal or computer-
417 tailored interactions. This review included 49 studies with 61 arms. Studies were included that
418 had an intervention component with one or more outcomes of interest. Study duration was 1.5
419 months to 2 years.

420
421 The study populations were mostly children younger than age 13 years and collectively were
422 racially and ethnically diverse. All studies were conducted in the United States within a variety
423 of settings, including schools (20 studies), homes (8 studies), communities (6 studies), primary
424 care clinics (4 studies), research institutes (5 studies), and in multiple settings (4 studies).
425 Settings were a mix of urban and suburban areas.

426
427 Evidence indicated that behavioral screen time interventions are effective in reducing
428 recreational sedentary screen time (47 study arms), improving physical activity (42 study arms),
429 improving diet (37 study arms), and improving or maintaining weight status (38 study arms).
430 Studies were found to be effective among children ages 13 years and younger. The evidence
431 showed that both screen-time-only and screen-time-plus interventions are both effective at
432 reducing recreational sedentary screen time. However, screen-time-only interventions showed
433 greater reductions in TV viewing and composite screen time compared to screen-time-plus
434 interventions. All studies demonstrated effectiveness among both males and females. Forty-five
435 studies that reported racial distribution showed intervention effectiveness in all groups: white (20
436 studies), black (14 studies), Hispanic (11 studies), Asian/Pacific Islander (10 studies), American
437 Indian or Alaska Native (3 studies), and other (7 studies).

438
439 ***For additional details on this body of evidence, visit:***

440 <http://www.thecommunityguide.org/obesity/RRbehavioral.html>

441 **SELF-MONITORING**

442 In the context of comprehensive behavioral lifestyle interventions for weight management, self-
 443 monitoring refers to the process by which an individual observes and records specific
 444 information reflecting his or her dietary intake, physical activity, and/or body weight. As a
 445 component of behavioral weight-management programs, self-monitoring is typically coupled
 446 with goal setting and performance feedback. Goal setting involves specifying a target or
 447 recommended level for dietary intake, physical activity, and/or body weight. Self-monitoring
 448 provides information that allows the individual to judge whether targets have been met, and if
 449 not, to use the feedback from self-monitoring to adjust future actions so as to meet the target. A
 450 high frequency of self-monitoring is commonly associated with greater adherence to other
 451 weight management strategies and with greater success in lifestyle programs for weight
 452 management.⁸¹

453
 454 The goal of this systematic review was to determine whether self-monitoring of diet and/or
 455 weight is associated with body weight outcomes. This review included studies examining the
 456 effect of self-weighing or self-monitoring of diet, such as counting calories and/or monitoring
 457 foods consumed. Although paper diaries are the traditional method for self-monitoring new
 458 technological approaches are emerging, such as the use of websites, smart phone “apps,” and
 459 interactive voice response phone calls. Because self-monitoring is often a component of weight
 460 loss and weight maintenances interventions, it is important to understand its effect on body
 461 weight outcomes.

462

463 **Question 6: What is the relationship between use of diet and weight self-** 464 **monitoring strategies and body weight outcomes in adults and youth?**

465 **Source of evidence:** NEL systematic review

466 **Conclusion**

467 Moderate evidence, primarily in overweight adult women living in the United States, indicates that
 468 self-monitoring of diet, weight, or both, in the context of a behavioral weight management
 469 intervention, incorporating goal setting and performance feedback, improves weight-loss
 470 outcomes. **DGAC Grade: Moderate**

471

472 Limited but consistent evidence suggests that higher frequency or greater adherence to self-
 473 monitoring of diet, weight, or both, in the context of a behavioral weight management program, is
 474 associated with better weight-loss outcomes. **DGAC Grade: Limited**

475

476 **Implications**

477 Self-monitoring coupled with goal setting and performance feedback can be used to enhance
478 outcomes in weight management programs and should be incorporated into these programs for
479 weight management.

480

481 **Review of the Evidence**

482 Twenty studies (4 RCTs,⁸²⁻⁸⁵ 15 prospective cohort studies,⁸⁶⁻¹⁰⁰ and 1 retrospective cohort
483 study¹⁰¹) examined the relationship between diet and weight self-monitoring strategies and body
484 weight outcomes in adults and youth. The study durations ranged from 3 months to 3.25 years.
485 The study samples predominantly included women. Five studies were exclusively in women, one
486 study was in pregnant women,⁸⁸ and one study was in children.⁸³ Sixteen studies were conducted
487 in the United States^{84-87, 89-100} and four were international (one each from the United Kingdom,
488 Australia, Netherlands, and Japan).^{82, 83, 88, 101}

489

490 Three RCTs showed that weight management interventions, delivered through mail or email
491 which included self-monitoring of diet, weight, or both, coupled with behavioral change
492 strategies, such as goal setting, personalized feedback, shaping, stimulus control, and problem
493 solving, resulted in significantly greater weight losses than did interventions that did not
494 emphasize self-monitoring.^{82, 84, 85} One weight loss maintenance study in children found no effect
495 for self-monitoring through Short Message Service on BMI.⁸³

496

497 Sixteen cohort studies in adults found higher frequency or greater adherence to diet and weight
498 self-monitoring was associated with favorable body weight outcomes.⁸⁶⁻¹⁰¹ One study with
499 overweight pregnant women provided a four-session behavior change program with a gestational
500 weight gain chart and a recommendation for regular self-weighing.⁸⁸ The women in the
501 intervention arm lost more weight 6 weeks after delivery compared to a control group that
502 received one brief education session. Four studies assessed different methods of self-monitoring,
503 including paper diaries, Internet-based or mobile applications, and found that no specific method
504 was superior to others.^{87, 93, 94, 98}

505

506 The limitations of the evidence were that study participants were predominately overweight or
507 obese, educated, Caucasian, females between the ages of 30 to 60 years, thus limiting
508 generalizability to broader population groups.

509

510 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3374>

511

512 FOOD AND MENU LABELING

513 Food and menu labels can provide information that improves an individual’s food selection and
514 potentially improves body weight outcomes. Research focusing upon the impact of food labeling
515 on body weight and other health outcomes is beginning to emerge. The U.S. Food and Drug
516 Administration (FDA) recently finalized regulations requiring calorie information to be listed on
517 menus and menu boards in chain restaurants, similar retail establishments, and vending machines
518 with 20 or more locations. Studying the effects of this regulation on dietary choices, weight and
519 chronic disease outcomes will provide an opportunity to understand how policy works in real-
520 world conditions.

521
522 Some studies, including existing reviews, have examined the impact of restaurant calorie
523 labeling on free-living consumer food selection and have had mixed results. Few studies have
524 actually measured calories consumed as a result of menu labeling. A recent systematic review
525 including 17 studies with experimental or quasi-experimental designs evaluated whether menu-
526 based nutrition information affects the selection and consumption of calories in restaurants and
527 other foodservice establishments.¹⁰² Five of these studies measured the association between the
528 introduction of menu labeling and average calories purchased per transaction in fast-food
529 restaurants before and after implementation of policies that required restaurants to add calorie
530 values to menus. Data collection varied in terms of duration (2 weeks to 6 months) and time from
531 menu changes (from 4 weeks to one year after menu calorie labeling took place). Only one of the
532 five reported a statistically significant association between the introduction of menu labeling and
533 the selection of fewer calories.

534
535 Overall, however, the review concluded that menu labeling of calories alone did not decrease
536 calories selected or consumed but that the addition of contextual or interpretive information on
537 menus, such as daily caloric recommendations or physical activity equivalents, assisted
538 consumers to select and consume fewer calories.¹⁰² Additionally, there appeared to be a
539 difference in sex response such that women tended to use the information to select and consumer
540 fewer calories than men.

541
542 The intent of this NEL systematic review was to focus on controlled trials that isolated the
543 impact of menu labeling on food selection and consumption at the individual level. The
544 Committee was also interested in the effects of menu labeling on body weight outcomes;
545 however there was insufficient evidence from RCTs examining the association between food and
546 menu labels and body weight to complete a systematic review with body weight as the outcome.
547

548 **Question 7: What is the effect of use of food and menu labels on measures of**
 549 **food selection and dietary intake in U.S. population groups?**

550 **Source of evidence:** NEL systematic review

551 **Conclusion**

552 Limited and inconsistent evidence exists to support an association between menu calorie labels
 553 and food selection or consumption. **DGAC Grade: Limited**

554

555 **Implications**

556 The impact of food and menu labeling on food selection and health outcomes is limited by the
 557 heterogeneous approaches and the modest number of high quality studies, particularly RCTs. Thus,
 558 no implication could be drawn from the RCTs although policy level studies suggest that menu
 559 labeling of calories alone will not decrease calories selected or consumed but that addition of
 560 contextual or interpretive information on menus, such as daily caloric recommendations or
 561 physical activity equivalents, can assist consumers to select and consume fewer calories.¹⁰² The
 562 new menu labeling regulations recently finalized by the FDA will provide an opportunity for
 563 further food and nutrition policy research in real-world settings.

564

565 **Review of the Evidence**

566 Ten RCTs¹⁰³⁻¹¹² were included in this body of evidence that compared menu calorie labeling on
 567 food selection. Three of the ten studies also measured calorie intake of a test meal.¹⁰⁷⁻¹⁰⁹
 568 Results were mixed regarding the influence of menu calorie labeling on food selection. Five
 569 studies found no effect of calorie information alone on food selection.^{104, 105, 107, 108, 110} Three
 570 studies found calorie labeling led to selection of fewer calories.^{103, 109, 112} Two studies showed
 571 mixed results. One¹⁰⁶ found an impact of calorie labeling with women, but not men, and
 572 another¹¹¹ found that parents ordered fewer calories for their children, but not for themselves
 573 when calorie information was included on a test menu.

574

575 Two studies found that providing calorie labels with either recommended daily caloric intake
 576 information¹⁰⁹ or physical activity equivalents¹⁰⁸ resulted in the consumption of fewer calories at
 577 a test meal. One study did not find an effect of calorie labeling on calorie consumption.¹⁰⁷ Two
 578 studies examining physical activity equivalents as a component of the calorie labeling found a
 579 decrease in the calorie content of selected food items.^{104, 108} One study that examined the effect
 580 of calorie labeling and value pricing (structuring product prices such that the per unit cost
 581 decreases as portion size increases) also showed no association between calorie labeling and
 582 food selection or consumption.

583

584 This body of evidence has many limitations: two of the ten studies were conducted in actual
 585 restaurant settings, limiting the external validity of the findings; three studies measured food
 586 intake; some studies included pricing as a confounder, while others did not; and all studies were
 587 conducted in one session. The methodological complexities of laboratory studies limit
 588 generalizability to free living populations.

589

590 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3379>

591

592 **HOUSEHOLD FOOD INSECURITY**

593 Food insecurity is a leading nutrition-related public health issue that is associated with reduced
 594 food intake or hunger because the household lacks money and other resources for food. Food
 595 insecurity can compromise nutritional intake, potentially leading to increased risk of chronic
 596 diseases.⁹ In addition, food insecurity may promote anxiety and psychological distress, further
 597 affecting the health and well-being of an individual or family.^{113, 114} Food insecurity is typically
 598 measured by survey questionnaires, such as the U.S. Household Food Security Survey Module,
 599 an 18-item questionnaire that assesses characteristics at the household level and severity of food
 600 insecurity (e.g., moderate or severe) over the past 12 months. The standard method of scoring
 601 consists of households being considered food secure if respondents affirm less than 3 scale items,
 602 food insecure if 3 to 7 items are affirmed, and severely food insecure if 8 or more items are
 603 affirmed.⁹ Surveys in the United States indicate that 14.3 percent or more of households
 604 experienced food insecurity at least once during 2013.¹ Rates of food insecurity are substantially
 605 higher than the national average for those households with incomes near or below the Federal
 606 poverty line (38.4 percent vs. 14.3 percent), those households with children and a single parent,
 607 and for African American- and Hispanic-headed households.¹ Rates of food insecurity are more
 608 common in rural areas and large cities compared to suburban and exurban areas surrounding
 609 cities.¹ Among food-insecure households, 62 percent are participating in one or more of the
 610 three largest Federal food and nutrition assistance programs (Supplemental Nutrition Assistance
 611 Program [SNAP], Special Supplementation Program for Women, Infants, and Children [WIC],
 612 and the National School Breakfast and Lunch Programs).¹ The causes of food insecurity are
 613 multifactorial and the types of nutrition-related problems resulting from food insecurity are
 614 diverse, differing across the life cycle. Among food insecure households, the cycle of having
 615 enough food followed by inadequate amounts has been associated with stress in pregnant
 616 women,¹¹³ poor diet quality among adults,^{115, 116} poor glycemic control among diabetics,¹¹⁷ and
 617 high visceral body fat and body weight gain in some but not all cross-sectional studies of
 618 children and adults.¹¹⁸⁻¹²⁰ Each of these conditions has a well-documented impact in the
 619 development of chronic diseases.^{121, 122} Thus, the 2015 DGAC chose to examine the relationship
 620 between food insecurity and diet quality as well as the causal nature of this public health issue on
 621 body weight with a systematic review of prospective cohorts.

622

623 *For additional details on this body of evidence, visit: <http://NEL.gov/topic.cfm?cat=3372>*

624

625 **Question 8: What is the relationship between household food insecurity (HFI) and**
626 **measures of diet quality and body weight?**

627 **Source of evidence:** NEL systematic review

628 **Conclusion**

629 Limited and inconsistent evidence from studies conducted in adults and children ages 3 to 6 years
630 suggests that a positive association may exist between persistent and/or progressing household
631 food insecurity and higher body weight in older adults, pregnant women, and young children. No
632 studies reported a relationship with lower body weight. **DGAC Grade: Limited**

633

634 Insufficient evidence was available from prospective studies to assess the relationship between
635 household food insecurity and dietary intake. **DGAC Grade: Grade Not assignable**

636

637 **Implications**

638 Federal food assistance programs, which play an important role in providing relief to families in
639 economic distress, should carefully document and monitor food insecurity and nutritional risk in
640 program participants. Participants should receive tailored counseling to choose foods with their
641 limited budgets that meet the *Dietary Guidelines for Americans* and to achieve or maintain a
642 healthy body weight. Federal food assistance programs should also regularly assess, evaluate, and
643 update the methods they use to help recipients select healthier foods, consistent with best practices.

644

645 **Review of the Evidence**

646 This systematic review included nine prospective cohort studies examining the relationship
647 between household food insecurity and body weight status.^{118, 123-130} In adults, four prospective
648 cohort studies assessed the relationship between household food insecurity and measures of body
649 weight, with one study focusing on elderly men and women¹²⁶ and three studies focusing only on
650 women.^{118, 128, 130} The study of older adults derived data from two large cohorts including the
651 Health and Retirement Survey and the Asset and Health Dynamics among the Oldest Old.¹²⁶ The
652 studies on women ranged in size from 303 to 1,707, with the data derived from relatively small
653 cohort study populations, including the Bassett Mothers Health Project cohort study,¹²⁸ the
654 Pregnancy, Infection, and Nutrition cohort,¹¹⁸ and the Fragile Families and Child Wellbeing
655 Study.¹³⁰ The study of older adults focused on a relatively homogenous population who were
656 mostly Caucasian.¹²⁶ Of the studies of women, two assessed diverse populations,^{118, 130} while one
657 had a study population almost entirely composed of Caucasian women.¹²⁸

658

659 In children, a total of five prospective cohort studies (three cohorts)^{123-125, 127, 129} assessed the
 660 relationship between household food insecurity and measures of body weight, with one of the
 661 five studies assessing household food insufficiency, a similar measure considered more severe
 662 than the concept of food security, although not as severe as hunger.¹²⁴ Four of the studies were
 663 conducted on populations in the United States^{123, 125, 127, 129} and one study in a Canadian
 664 population.¹²⁴ The studies ranged in size from 1,514 to 28,353 subjects. The data were derived
 665 from nationally representative cohorts, including three studies using data from the Early Child
 666 Longitudinal Study-Kindergarten Cohort,^{123, 125, 129} one study using data from the Longitudinal
 667 Study of Child Development in Quebec,¹²⁴ and one study deriving data from a large cohort
 668 participating in the Massachusetts WIC Program.¹²⁷

670 Based on this evidence, the impact of food insecurity on body weight is not clear. Among older
 671 adults, becoming food insecure during follow-up was positively associated with BMI in one
 672 large cohort but there was no association in a different cohort from the same study.¹²⁶ Among
 673 pregnant women, findings were inconsistent, with 1 of 2 studies suggesting no association
 674 between food insecurity and pregnancy weight gain outcomes.¹²⁸ One study found null findings
 675 among the marginally food secure, but greater weight gain (absolute and relative to the 2009
 676 IOM Guidelines),¹³¹ and severe pre-gravid obesity among food insecure women.¹¹⁸ Among
 677 children, findings were inconsistent. Two studies found no association between food insecurity
 678 and body weight outcomes.^{123, 129} Dubois et al. found that food insufficiency was associated
 679 greater likelihood of overweight and obesity in preschool-aged children.¹²⁴ One study found that
 680 persistent food insecurity without hunger was associated with child obesity but non-persistent
 681 food insecurity with hunger was not associated with obesity risk.¹²⁷ Jyoti et al. reported that there
 682 was an association between food insecurity and weight gain for girls but not boys.¹²⁵ However,
 683 the data provided some suggestion of an association between food insecurity and higher body
 684 weight among girls and those who are of low birth weight.

685
 686 *For additional details on this body of evidence, visit: <http://NEL.gov/topic.cfm?cat=3372>*
 687

688 **ACCULTURATION**

689 Immigrants continue to represent a significant proportion of the United States population and
 690 evidence indicates that immigrants adopt the dietary habits and disease patterns of host
 691 cultures.¹⁴ Federal food assistance and nutrition education programs are aware of the need to
 692 tailor services and messaging according to the level of acculturation of immigrant communities.
 693 It is essential for this acculturation-sensitive tailoring to take into account the level of dietary
 694 acculturation and the socio-economic characteristics such as health literacy, language, and other
 695 cultural preferences of immigrant communities. Thus, understanding how dietary habits, body
 696 weight, and chronic disease outcomes are influenced by the process of acculturation is an
 697 important public health issue for the United States. However, because immigrants can take

698 different paths during the process of acculturation, this construct has proven to be difficult to
 699 conceptualize and measure. The four paths of acculturation (assimilation, integration,
 700 segregation, and marginalization) refer to the degree in which immigrants retain their host
 701 culture and adopt the culture of their new country.¹⁴ This explains, at least in part, why the
 702 evidence from prospective studies continues to be limited in nature, as shown in this chapter.

703

704 **Question 9: What is the relationship between acculturation and measures of**
 705 **dietary intake?**

706 **Source of evidence:** NEL systematic review

707 **Conclusion**

708 Limited evidence from cross-sectional studies suggests that in adults of Latino/Hispanic national
 709 origin, particularly among women and persons of Mexican origin, higher acculturation to the
 710 United States is associated with lower fruit and vegetable intake, as well as higher intake of fast
 711 food. Insufficient evidence is available for children, Asians and African Americans in general, and
 712 among populations of diverse Latino/Hispanic national origin to draw a conclusion regarding the
 713 association between measures of acculturation and dietary intake. **DGAC Grade: Limited**

714

715 **Implications**

716 Federal food assistance and nutrition education programs need to support immigrants in
 717 maintaining the healthy dietary habits they had when they arrived and in not acquiring unhealthy
 718 dietary patterns as they acculturate to mainstream America. This can be achieved by, among other
 719 things, effectively reaching out to immigrant families to facilitate their enrollment in programs
 720 such as SNAP and WIC and ensuring access to fresh vegetables and fruits. These community
 721 outreach programs are needed because in addition to their risk of adopting unhealthy dietary
 722 behaviors, immigrants may also have language limitations and/or a lack of understanding of the
 723 program enrollment procedures.

724

725 **Review of the Evidence**

726 This systematic review included 17 studies, 15 cross-sectional studies,¹³²⁻¹⁴⁶ and two longitudinal
 727 studies^{147, 148} that examined the relationship between multidimensional or multiple proxy
 728 measures of acculturation and dietary intake. Study populations included ten Latino/Hispanic
 729 populations^{132-136, 138-140, 144, 145} (five in Mexican Americans) and^{132, 133, 135, 136, 140} six Asian
 730 populations;^{137, 141-143, 146, 147} one study included both Asian and Latino/Hispanic populations.¹⁴⁸
 731 Two studies included children^{135, 148} and three studies included only women.^{134, 138, 140} Study
 732 locations included one national¹⁴⁰ and one U.S.-Mexican border state study,¹³⁶ ten studies from
 733 California,^{132, 133, 135, 137-139, 143, 145, 146, 148} and one study each from Massachusetts, Hawaii,¹⁴⁷ New
 734 York,¹⁴¹ and a Midwestern city.

735
 736 In adults of Latino/Hispanic national origin, evidence from nine cross-sectional analyses
 737 suggests that higher acculturation to the United States is associated with lower adherence to
 738 recommended dietary patterns. Among adults of Latino/Hispanic national origin, primarily
 739 women and those of Mexican origin, higher acculturation is consistently associated with lower
 740 fruit and vegetable intake, as well as higher intake of fast food. In children and youth of
 741 Latino/Hispanic national origin, emerging evidence was identified from two cross-sectional
 742 studies suggesting a negative association between acculturation and dietary behaviors. In a study
 743 of children ages 3 to 5 years who were proxied by caregiver acculturation, acculturation was
 744 associated with higher intake of sweets. In a study among adolescents, acculturation was
 745 associated with higher intake of fast foods.

746
 747 Among Asian populations, emerging evidence from five cross-sectional and two longitudinal
 748 studies suggests that higher acculturation is associated with lower adherence to recommended
 749 dietary patterns. In adults, six studies among Asian populations (mainly Korean, Chinese and
 750 Filipino) suggest higher acculturation is associated with higher fast food and alcohol
 751 consumption.^{137, 141-143, 146, 147} One study suggests higher acculturation is associated with
 752 increased fast food consumption among Asian adolescents.¹⁴⁸

753
 754 Insufficient evidence is available among children, those of Latino/Hispanic national origin
 755 (other than Mexican-Americans), and among immigrant populations from Asia, Africa, Europe,
 756 and the Middle East regarding the association between measures of acculturation and dietary
 757 intake.

758
 759 *For additional details on this body of evidence, visit:*
 760 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250436

761
 762 **Question 10: What is the relationship between acculturation and body weight?**

763 **Source of evidence:** NEL systematic review

764 **Conclusion**

765 Limited evidence suggests a relationship between higher acculturation to the United States and
 766 increased body weight. This relationship varies by national origin and gender. Specifically,
 767 findings were mixed in both Asian and Latino/Hispanic populations. In Asians, the association was
 768 stronger in women than men and in Latino/Hispanic populations; associations were stronger in
 769 Mexican-born women. **DGAC Grade: Limited**

770

771 **Implications**

772 Federal food assistance and nutrition education programs need to support immigrants against the
 773 risk of becoming overweight or obese as they acculturate to mainstream America. This can be
 774 achieved by among other things, effectively reaching out to immigrant families to facilitate their
 775 enrollment in programs such as SNAP and WIC and ensuring access to low-energy and high-
 776 nutrient dense dietary patterns rich in vegetables and fruits and whole grain foods. These
 777 community outreach programs are needed because in addition to their risk of adopting unhealthy
 778 dietary behaviors, immigrants may also have language limitations and/or a lack of understanding
 779 of the program enrollment procedures.

780

781 **Review of the Evidence**

782 This systematic review includes 13 studies:^{133, 137, 141, 143, 144, 146, 147, 149-154} 12 cross-sectional
 783 studies,^{133, 137, 141, 143, 144, 146, 149-154} and one longitudinal study.¹⁴⁷ The populations included seven
 784 Asian,^{137, 141, 143, 146, 147, 150, 151} five Latino/Hispanic (four Mexican-American and one Puerto
 785 Rican),^{133, 144, 149, 152, 153} and included adults ranging in age from 35 to 75 years. Five studies were
 786 analyzed by gender.^{141, 143, 146, 153, 154} Three of the studies included national samples,^{149, 152, 154} five
 787 studies were from California,^{133, 137, 143, 146, 153} and one study each was from Hawaii,¹⁴⁷
 788 Louisiana,¹⁵¹ Maryland,¹⁵⁰ Massachusetts,¹⁴⁴ New York.¹⁴¹ Two studies included samples from
 789 the country of origin (Vietnam and Korea).^{143, 151}

790

791 Among Asian populations, the majority of the data suggest a positive relationship between
 792 acculturation and increased body weight, but results are not consistent. Among Latinos/Hispanic
 793 populations, the association has been documented mostly among women of Mexican origin.

794

795 *For additional details on this body of evidence, visit:*

796 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250437

797

798 **Question 11: What is the relationship between acculturation and risk of** 799 **cardiovascular disease (CVD)?**

800 **Source of evidence:** NEL systematic review

801 **Conclusion**

802 No conclusion can be drawn regarding the relationship between acculturation to the United States
 803 and the risk of CVD. This is due to the small number of studies, wide variation in methodology
 804 used to assess acculturation, and limited representation of ethnic groups in the body of evidence.
 805 Very limited evidence from a small number of cross-sectional studies conducted in
 806 Latino/Hispanic populations suggest a positive relationship between language acculturation and
 807 elevation in LDL cholesterol and no relationship between acculturation and blood pressure.

808 Insufficient evidence is available for other race/ethnic populations and among children for these
 809 outcomes and other CVD outcomes. **DGAC Grade: Grade not assignable**

810

811 **Implications**

812 The DGAC determined that a grade was not assignable due to the insufficient evidence for this
 813 question. Therefore, no implications were developed.

814

815 **Review of the Evidence**

816 This systematic review includes six cross-sectional studies in adult men and women between the
 817 ages of 40 to 60 years.^{144, 154-158} The study populations included five Latino/Hispanic^{144, 155-158}
 818 and one multicultural population¹⁵⁴ and the data were predominately derived from large, multi-
 819 state or national data sets.

820

821 Three studies found a positive relationship between language acculturation and elevated blood
 822 lipid levels,^{154, 156, 157} but results varied by acculturation indicator. Two studies assessed the
 823 association between acculturation and blood pressure in Latino/Hispanic populations and no
 824 association was found.^{156, 157} Two studies assessed the relationship between acculturation and
 825 hypertension in Latino/Hispanic and a multicultural population and found no association.^{144, 154}

826 Two studies suggest a positive association between language acculturation and CVD risk
 827 factors,^{155, 158} but results varied as a function of language acculturation indicator used.

828 The studies used different methods to assess acculturation, including three studies that used
 829 multidimensional scales^{144, 155, 157} and three studies that relied on the assessment of acculturation
 830 proxies.^{154, 156, 158}

831

832 The preponderance of evidence was in predominately Mexican American populations, but other
 833 Hispanic/Latino national origin groups were represented.

834

835 *For additional details on this body of evidence, visit:*

836 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250438

837

838 **Question 12: What is the relationship between acculturation and risk of type 2**
 839 **diabetes?**

840 **Source of evidence:** NEL systematic review

841 **Conclusion**

842 Conclusions regarding the relationship between acculturation and type 2 diabetes cannot be drawn
 843 due to limited evidence from a very small number of cross-sectional studies and study populations,
 844 limitations in acculturation assessment methodology that did not take into account potential

845 confounders and effect modifiers, and lack of standardized assessment of outcomes. **DGAC**

846 **Grade: Grade not assignable**

847

848 **Implications**

849 The DGAC determined that a grade was not assignable due to the insufficient evidence for this
850 question. Therefore, no implications were developed.

851

852 **Review of the Evidence**

853 This systematic review included four cross-sectional studies.^{144, 152, 159, 160} Two of the studies
854 used National Health and Nutrition Examination Survey (NHANES) data on Hispanic/Latino
855 participants,^{152, 160} one study used the Multi-Ethnic Study of Atherosclerosis (MESA) cohort,¹⁵⁹
856 which included Mexican, other Hispanic, and Chinese populations, and one study used the
857 Boston Puerto Rican Health Study cohort.¹⁴⁴

858

859 The studies used different methods to assess acculturation. Four different multidimensional
860 scales were used^{144, 159, 160} and one study relied on the assessment of two acculturation proxies.¹⁵²

861 All measures took into consideration language usage with some only using this proxy and others
862 including additional proxies for acculturation.

863

864 ***For additional details on this body of evidence, visit:***

865 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250439

866

867 **CHAPTER SUMMARY**

868 The individual is at the innermost core of the social-ecological model. In order for policy
869 recommendations such as the *Dietary Guidelines for Americans* to be fully implemented,
870 motivating and facilitating behavioral change at the individual level is required. The collective
871 work presented in this chapter suggests a number of promising behavior change strategies that
872 can be used to favorably impact a range of health related outcomes and to enhance the
873 effectiveness of interventions. These include reducing screen time, reducing the frequency of
874 eating out at fast- food restaurants, increasing frequency of family shared meals, and self-
875 monitoring of diet and body weight as well as effective food labeling to target healthier food
876 choices. These strategies complement comprehensive lifestyle interventions and nutrition
877 counseling by qualified nutrition professionals. Timely feedback from registered
878 dietitians/nutritionists and other qualified health professionals and engagement of the individual
879 as appropriate in individual and group counseling will enhance outcomes. For this approach to
880 work, it will be essential for the food environments where low-income individuals live to
881 facilitate access to the selection of healthy food choices that respect their cultural preferences.
882 Likewise, food and calorie label education should be designed to be understood for low literacy

883 audiences some of which may have additional English language fluency limitations. While
 884 viable approaches are available now, additional research is necessary to improve the scientific
 885 foundation for more effective guidelines on individual level behavior change for all individuals
 886 living in the United States, taking into account the social, economic and cultural environments in
 887 which they live.

888
 889 The evidence reviewed in this chapter indicates that the social, economic, and cultural context in
 890 which individuals live may facilitate or hinder their ability to choose and consume dietary
 891 patterns that are consistent with the Dietary Guidelines. Specifically household food insecurity
 892 hinders the access to healthy diets for millions of Americans. Also, immigrants are at high risk of
 893 losing the healthier dietary patterns characteristic of their cultural background as they acculturate
 894 into mainstream America. Furthermore, preventive nutrition services that take into account the
 895 social determinants of health are largely unavailable in our health system to systematically
 896 address the nutrition-related health problems of Americans including overweight and obesity,
 897 CVD, type 2 diabetes, and other chronic diseases. In summary, this chapter calls for: a)
 898 continuous support of Federal programs to help alleviate the consequences of household food
 899 insecurity, b) food and nutrition assistance programs to take into account the risk that immigrants
 900 have of giving up their healthier dietary habits soon after arriving in the United States, and c)
 901 efforts to provide all individuals living in the United States with the environments, knowledge,
 902 and tools needed to implement effective individual- or family-level behavioral change strategies
 903 to improve the quality of their diets and reduce sedentary behaviors. As indicated in *Part D*
 904 *Chapter 4: Food Environment and Settings* and *Part D Chapter 5: Food Sustainability and*
 905 *Safety*, achieving these goals will require changes at all levels of the social-ecological model
 906 through coordinated efforts among health care and social and food systems from the national to
 907 the local level.

908

909 **NEEDS FOR FUTURE RESEARCH**

910 **Eating Out**

911 1. Develop a standard methodology to collect and characterize various types of eating venues.

912 **Rationale:** This recommendation is fundamental to conducting rigorous research, evaluating
 913 findings from multiple studies, and developing policies to promote healthy eating among
 914 people who frequent eating out venues and/or consume take away meals.

915

916 2. Conduct rigorously designed research to examine the longitudinal impact of obtaining or
 917 consuming meals away from home from various types of commonly frequented venues on
 918 changes in food and beverage intakes (frequency, quantity, and composition), body weight,
 919 adiposity, and health profiles from childhood to adulthood in diverse (racial/ethnic,
 920 socioeconomic, cultural, and geographic) groups of males and females.

921 **Rationale:** Most groups in the U.S. population regularly consume meals that are prepared
 922 away from home and the landscape of fast food and other types of food procurement and
 923 consumption venues is increasingly complex. The potential for eating out and/or take away
 924 meals to influence diet quality, energy balance, body mass and composition, and the risks of
 925 health-related morbidities across the lifespan among our diverse population underscores the
 926 importance of understanding this issue.

927

928 **Family Shared Meals**

929 3. Conduct studies in diverse populations that assess not only frequency of family shared meals,
 930 but also quality of family shared meals.

931 **Rationale:** Our understanding of the importance of family shared meals in terms of how they
 932 contribute in a positive way to body weight and overall health and well-being requires a
 933 rigorous examination of the dietary quality of these meals compared to other meals consumed
 934 by family members.

935

936 4. Conduct RCTs to isolate the effect of interventions that increase the frequency of family
 937 meals from other health and parenting behaviors that may be associated with dietary intake
 938 and weight status.

939 **Rationale:** Family shared meals are commonly implemented as one component of lifestyle
 940 interventions that include an array of other behavioral and parenting strategies for weight
 941 management. To improve our understanding of the causal pathway of how family shared meals
 942 contributes to maintaining or achieving a health weight, the specific contribution of family
 943 shared meals to weight outcomes independent of other behavioral strategies needs to be
 944 ascertained.

945

946 **Sedentary Behavior**

947 5. Develop improved and better standardized and validated tools to assess sedentary behaviors
 948 and activities that children, adolescents, and adults regularly engage in.

949 **Rationale:** Our understanding of the impact of sedentary behaviors on diet, energy balance,
 950 body mass, adiposity, and health is currently compromised by reliance on subjective
 951 assessments, including self-reports of daily activity patterns, and by inadequate techniques to
 952 document and quantify the array of sedentary activities people engage in (beyond TV viewing
 953 and (or) computer screen time). It also would be beneficial for researchers to document the
 954 potential benefits and implications of reducing one type of sedentary behavior (e.g. screen
 955 time) on other sedentary behaviors (e.g., reading for leisure, arts and crafts, listening to music)
 956 and indices of health (e.g. sleep quality and duration).

957

958 6. Conduct prospective research to examine the effects and mechanisms of the quantity,
 959 patterns, and changes of sedentary behaviors on diet quality, energy balance, body weight,
 960 adiposity, and health across the life span in groups within the U.S. population with diverse
 961 personal, cultural, economic, and geographic characteristics.

962 **Rationale:** Emerging, but limited, evidence implicates sedentary behaviors with adverse
 963 health-related outcomes, especially in children and adolescents as they transition into
 964 adulthood. However, an improved understanding of why these relationships exist will help in
 965 developing appropriate and effective approaches and policies to reduce the amount of time
 966 people spend engaging in sedentary behaviors.

967

968 **Self-Monitoring**

969 7. Evaluate the impact of different types, modalities, and frequencies of self-monitoring on
 970 body weight outcomes during both the weight loss intervention and maintenance periods.

971 **Rationale:** Self-monitoring is associated with improved weight management. However, the
 972 current practice of recommending daily self-monitoring may represent a barrier to its
 973 implementation and/or continued use. Hence, it is important to determine whether lower
 974 frequencies of self-monitoring can produce beneficial effects on weight outcomes.

975

976 8. Evaluate the comparative effectiveness of performance feedback from self-monitoring
 977 delivered through automated systems versus personal interactions with a counselor.

978 **Rationale:** Automated feedback derived from self-monitoring data and delivered
 979 electronically can produce beneficial changes on weight outcomes. However, the comparative
 980 effectiveness and cost efficiency of feedback delivered through non-personal modalities versus
 981 personal interactions has yet to be determined.

982

983 9. Test the effectiveness of self-monitoring on weight outcomes in understudied groups,
 984 including ethnic/racial minorities, low education, low literacy, and low numeracy
 985 populations, males, and subjects younger than age 30 years and older than age 60 years.

986 **Rationale:** Evidence regarding the effectiveness of self-monitoring has been derived largely
 987 from research conducted on well educated, middle-class, white women. Hence, it is important
 988 to determine whether the beneficial effects of self-monitoring on weight outcomes are
 989 generalizable to understudied groups.

990

991 10. Conduct RCTs based on sound behavioral change theories that incorporate self-monitoring,
 992 employ heterogeneous populations, and are powered for small effect sizes and high attrition
 993 rates, to test the short- (e.g., 3 months) and long-term (e.g., 12 months) effects of mobile health
 994 technologies on dietary and weight outcomes.

995 **Rationale:** Mobile health technologies have the potential to reach larger portions of the
 996 populations than face-to-face interventions, but the effect sizes of mobile technologies may be
 997 small and the attrition rates may be large. Larger, more representative study populations and
 998 longer study periods will permit an assessment of the generalizability and sustainability of
 999 mobile health technologies.

1000

1001 **Food and Menu Labeling**

1002 11. Develop novel labeling approaches to provide informative strategies to convey caloric intake
 1003 values on food items consumed at home and in restaurant settings.

1004 **Rationale:** Menu labels can include different types of information in addition to calories.
 1005 These include physical activity equivalents, and daily caloric needs. Very few studies have
 1006 been designed to examine the optimal combination of menu label information to prevent
 1007 excessive caloric intake. This will be very valuable evidence to inform the calorie label policy
 1008 that has just been enacted by the FDA.

1009

1010 12. Compare labeling strategies across various settings, such as restaurants, stores, and the home
 1011 to determine their efficacy in altering food selection and health outcomes, including weight.

1012 **Rationale:** The great majority of menu labeling RCT's have been conducted under laboratory
 1013 conditions. Given the recent FDA regulations, future studies will be able to impact the
 1014 effectiveness of these polices across settings as accessed by diverse free living populations.

1015

1016 13. Evaluate the process and impact of recent FDA menu labeling regulation.

1017 **Rationale:** The new FDA regulation provides a unique opportunity to understand the impact of
 1018 menu labeling on consumers dietary behaviors in "real world" settings.

1019

1020 **Household Food Insecurity**

1021 14. Conduct prospective cohort studies that cover a wide age range and include children,
 1022 families, older adults, and ethnically/racially diverse populations and describe potential effect
 1023 modifiers such as gender, ethnic and cultural factors, family structure, area of residence (i.e.,
 1024 urban vs. rural), employment, and use of social support systems while examining the
 1025 relationship between household food insecurity, dietary intake, and body weight.

1026 **Rationale:** Understanding the temporal process of when and how long food insecurity occurs
 1027 within a family/individual's lifetime and their response to this economic stressor is critical to
 1028 conducting rigorous research and comparing finding across studies in order to develop and
 1029 implement intervention studies and policies to alleviate this public health problem.

1030

1031 15. Standardize research methodology, including developing a consistent approach to measuring
 1032 food insecurity and use of measured height and weight to reduce the likelihood of responder
 1033 bias.

1034 **Rationale:** The measurement error issues related to the use of self-reported weight have been
 1035 well documented in the literature. In order to conduct rigorous studies in this area that can be
 1036 compared and evaluated as to the causal nature of the role of food insecurity on body weight,
 1037 standard methodology is warranted both in the measurement of the exposure as well as the
 1038 outcome.

1039

1040 **Acculturation**

1041 16. Conduct prospective longitudinal studies including those that start in early childhood to track
 1042 dietary intake, sedentary behaviors, body weight, and chronic disease outcomes across the
 1043 lifespan. Include the diversity of ethnic/racial groups in the United States, including
 1044 individuals and families of diverse national origins. Include comparison groups in countries
 1045 of origin to rule out, among other things, the potential confounding by internal migration
 1046 from rural to urban area within the country of origin.

1047 **Rationale:** Acculturation is a time-dependent life course process that requires longitudinal
 1048 studies to be properly understood. Because the impact of acculturation on dietary, weight and
 1049 health outcomes can be expected to be modified by the life course stage of life when
 1050 individuals migrate to the United States, prospective acculturation studies need to start
 1051 following individuals from very early childhood.

1052

1053 17. Develop a standard tool to measure acculturation or validation of multidimensional
 1054 acculturation scales in different immigrant groups and in different languages.

1055 **Rationale:** Acculturation is a complex construct that is seldom measured with
 1056 multidimensional scales that can capture the different paths that migrant scan take with regards
 1057 to the acculturation process, including assimilation, integration, segregation, and
 1058 marginalization. Although research in acculturation measurement has been conducted among
 1059 Hispanic/Latinos, it has been predominantly based on Mexican American populations and little
 1060 acculturation measurement research has been conducted among other groups, including
 1061 individuals from Asia, Africa, Europe, and the Middle East.

1062

1063 **Sleep Patterns**

1064 18. Conduct prospective studies that start in childhood (including transition to adulthood), to
 1065 investigate the longitudinal effect of sleep patterns on diet and body weight outcomes while
 1066 accounting for confounders, mediators, and moderators including: physical activity,
 1067 socioeconomic variables (such as education, employment, household income), sex, alcohol

1068 intake, smoking status (including new smoker, new non-smoker), media use/screen time, and
1069 depression.

1070 **Rationale:** While research associates short sleep duration and disordered sleep patterns with
1071 adverse differences and changes in food and beverage consumption, body weight, and indices
1072 of metabolic and cardiovascular health, less is known about the impact of potential modifying
1073 lifestyle factors. This research will help delineate the role of sleep patterns, duration and
1074 quality, i.e., mediator or moderator, on diet and weigh-related outcomes. Research in children
1075 shows that sleep deprivation and weight are related but this relationship is not apparent in adult
1076 studies. This may be due to the fact that energy intake increases during transition to short sleep
1077 duration, but levels off when short sleep duration becomes consistent.

1078

1079 19. Conduct studies to assess the effects of diet on sleep quality to examine the mechanism by
1080 which dietary intake, energy intake, and energy expenditure may impact sleep.

1081 **Rationale:** Most research has focused on sleep quality and duration as modifying factors on
1082 diet, body weight, and health. A paucity of research exists on the potential impact of diet on
1083 sleep-related outcomes. This line of research would use diet as the means to improve indices of
1084 sleep, which in turn may subsequently improve health-related outcomes.

1085

1086 REFERENCES

- 1087 1. Coleman-Jensen A, Gregory C, Singh A. Household food security in the United States in
1088 2013. 2014. <http://www.ers.usda.gov/media/1565415/err173.pdf>.
- 1089 2. United States. Department of Agriculture. Human Nutrition Information Service. Dietary
1090 Guidelines Advisory Committee., United States. Agricultural Research Service. Report of
1091 the Dietary Guidelines Advisory Committee on the dietary guidelines for Americans,
1092 2010 : to the Secretary of Agriculture and the Secretary of Health and Human Services.
1093 2010:vi, 445 p. PMID: 16634830.
1094 http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/2010DG
1095 [ACReport-camera-ready-Jan11-11.pdf](http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/2010DG).
- 1096 3. Owen N, Healy GN, Matthews CE, Dunstan DW. Too much sitting: the population health
1097 science of sedentary behavior. *Exerc Sport Sci Rev.* 2010;38(3):105-13. PMID:
1098 20577058. <http://www.ncbi.nlm.nih.gov/pubmed/20577058>.
- 1099 4. McGinnis JM, Gootman JA, Kraak VI. Food marketing to children and youth: threat or
1100 opportunity? 2006.
1101 [http://static.publico.pt/docs/pesoemedida/Iom_Food_Marketing_To_Children_Exec_Sum](http://static.publico.pt/docs/pesoemedida/Iom_Food_Marketing_To_Children_Exec_Summary_2006.pdf)
1102 [mary_2006.pdf](http://static.publico.pt/docs/pesoemedida/Iom_Food_Marketing_To_Children_Exec_Summary_2006.pdf).
- 1103 5. Golem DL, Martin-Biggers JT, Koenings MM, Davis KF, Byrd-Bredbenner C. An
1104 integrative review of sleep for nutrition professionals. *Adv Nutr.* 2014;5(6):742-59.
1105 PMID: 25398735. <http://www.ncbi.nlm.nih.gov/pubmed/25398735>.
- 1106 6. Butryn ML, Webb V, Wadden TA. Behavioral treatment of obesity. *Psychiatr Clin North*
1107 *Am.* 2011;34(4):841-59. PMID: 22098808.
1108 <http://www.ncbi.nlm.nih.gov/pubmed/22098808>.

- 1109 7. Core indicators of nutritional state for difficult-to-sample populations. *J Nutr.* 1990;120
 1110 Suppl 11:1559-600. PMID: 2243305. <http://www.ncbi.nlm.nih.gov/pubmed/2243305>.
- 1111 8. Perez-Escamilla R. Food insecurity in children: impact on physical, psychoemotional and
 1112 social development. In: Ross CA, Caballero RJ, Cousins RJ, Tucker KL, Ziegler TR,
 1113 editors. *Modern Nutrition in Health and Disease*. Baltimore, MD: Lippincott Williams &
 1114 Wilkins; 2013. p. 1006-15.
- 1115 9. Lee JS, Gundersen C, Cook J, Laraia B, Johnson MA. Food insecurity and health across
 1116 the lifespan. *Adv Nutr.* 2012;3(5):744-5. PMID: 22983862.
 1117 <http://www.ncbi.nlm.nih.gov/pubmed/22983862>.
- 1118 10. Statistical portrait of the foreign-born population in the United States, 2010. Pew
 1119 Research Hispanic Trends Project. 2012.
 1120 [http://www.pewhispanic.org/2014/04/29/statistical-portrait-of-the-foreign-born-](http://www.pewhispanic.org/2014/04/29/statistical-portrait-of-the-foreign-born-population-in-the-united-states-2012/)
 1121 [population-in-the-united-states-2012/](http://www.pewhispanic.org/2014/04/29/statistical-portrait-of-the-foreign-born-population-in-the-united-states-2012/).
- 1122 11. Yearbook of immigration statistics: 2013. Table 1 Persons Obtaining Lawful Permanent
 1123 Resident Status: Fiscal Years 1820 to 2013. [http://www.dhs.gov/yearbook-immigration-](http://www.dhs.gov/yearbook-immigration-statistics-2013-lawful-permanent-residents)
 1124 [statistics-2013-lawful-permanent-residents](http://www.dhs.gov/yearbook-immigration-statistics-2013-lawful-permanent-residents).
- 1125 12. Abraido-Lanza AF, White K, Vasques E. Immigrant populations and health. In:
 1126 Anderson N, editor. *Encyclopedia of Health and Behavior*. Newbury Park, CA: Sage;
 1127 2004. p. 533-7.
- 1128 13. Redfield R, Linton R, Herskovits MJ. Memorandum for the study of acculturation.
 1129 *American Anthropologist.* 1936;38(1):149-52.
- 1130 14. Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and
 1131 incidence of type 2 diabetes among Latinos. *J Nutr.* 2007;137(4):860-70. PMID:
 1132 17374645. <http://www.ncbi.nlm.nih.gov/pubmed/17374645>.
- 1133 15. Bes-Rastrollo M, Basterra-Gortari FJ, Sanchez-Villegas A, Marti A, Martinez JA,
 1134 Martinez-Gonzalez MA. A prospective study of eating away-from-home meals and
 1135 weight gain in a Mediterranean population: the SUN (Seguimiento Universidad de
 1136 Navarra) cohort. *Public Health Nutr.* 2010;13(9):1356-63. PMID: 19954575.
 1137 <http://www.ncbi.nlm.nih.gov/pubmed/19954575>.
- 1138 16. Boggs DA, Rosenberg L, Coogan PF, Makambi KH, Adams-Campbell LL, Palmer JR.
 1139 Restaurant foods, sugar-sweetened soft drinks, and obesity risk among young African
 1140 American women. *Ethn Dis.* 2013;23(4):445-51. PMID: 24392607.
 1141 <http://www.ncbi.nlm.nih.gov/pubmed/24392607>.
- 1142 17. Duffey KJ, Gordon-Larsen P, Jacobs DR, Jr., Williams OD, Popkin BM. Differential
 1143 associations of fast food and restaurant food consumption with 3-y change in body mass
 1144 index: the Coronary Artery Risk Development in Young Adults Study. *Am J Clin Nutr.*
 1145 2007;85(1):201-8. PMID: 17209197. <http://www.ncbi.nlm.nih.gov/pubmed/17209197>.
- 1146 18. Duffey KJ, Gordon-Larsen P, Steffen LM, Jacobs DR, Jr., Popkin BM. Regular
 1147 consumption from fast food establishments relative to other restaurants is differentially
 1148 associated with metabolic outcomes in young adults. *J Nutr.* 2009;139(11):2113-8.
 1149 PMID: 19776183. <http://www.ncbi.nlm.nih.gov/pubmed/19776183>.
- 1150 19. Fraser LK, Clarke GP, Cade JE, Edwards KL. Fast food and obesity: a spatial analysis in
 1151 a large United Kingdom population of children aged 13-15. *Am J Prev Med.*
 1152 2012;42(5):e77-85. PMID: 22516506. <http://www.ncbi.nlm.nih.gov/pubmed/22516506>.
- 1153 20. French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound
 1154 of Prevention study: dietary, behavioral and demographic correlates. *Int J Obes Relat*

- 1155 Metab Disord. 2000;24(10):1353-9. PMID: 11093299.
 1156 <http://www.ncbi.nlm.nih.gov/pubmed/11093299>.
- 1157 21. Haines J, Neumark-Sztainer D, Wall M, Story M. Personal, behavioral, and
 1158 environmental risk and protective factors for adolescent overweight. *Obesity* (Silver
 1159 Spring). 2007;15(11):2748-60. PMID: 18070766.
 1160 <http://www.ncbi.nlm.nih.gov/pubmed/18070766>.
- 1161 22. Laska MN, Murray DM, Lytle LA, Harnack LJ. Longitudinal associations between key
 1162 dietary behaviors and weight gain over time: transitions through the adolescent years.
 1163 *Obesity* (Silver Spring). 2012;20(1):118-25. PMID: 21701567.
 1164 <http://www.ncbi.nlm.nih.gov/pubmed/21701567>.
- 1165 23. Li F, Harmer P, Cardinal BJ, Bosworth M, Johnson-Shelton D, Moore JM, et al. Built
 1166 environment and 1-year change in weight and waist circumference in middle-aged and
 1167 older adults: Portland Neighborhood Environment and Health Study. *Am J Epidemiol*.
 1168 2009;169(4):401-8. PMID: 19153214. <http://www.ncbi.nlm.nih.gov/pubmed/19153214>.
- 1169 24. MacFarlane A, Cleland V, Crawford D, Campbell K, Timperio A. Longitudinal
 1170 examination of the family food environment and weight status among children. *Int J*
 1171 *Pediatr Obes*. 2009;4(4):343-52. PMID: 19922051.
 1172 <http://www.ncbi.nlm.nih.gov/pubmed/19922051>.
- 1173 25. Niemeier HM, Raynor HA, Lloyd-Richardson EE, Rogers ML, Wing RR. Fast food
 1174 consumption and breakfast skipping: predictors of weight gain from adolescence to
 1175 adulthood in a nationally representative sample. *J Adolesc Health*. 2006;39(6):842-9.
 1176 PMID: 17116514. <http://www.ncbi.nlm.nih.gov/pubmed/17116514>.
- 1177 26. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR, Jr., et al.
 1178 Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year
 1179 prospective analysis. *Lancet*. 2005;365(9453):36-42. PMID: 15639678.
 1180 <http://www.ncbi.nlm.nih.gov/pubmed/15639678>.
- 1181 27. Taveras EM, Berkey CS, Rifas-Shiman SL, Ludwig DS, Rockett HR, Field AE, et al.
 1182 Association of consumption of fried food away from home with body mass index and diet
 1183 quality in older children and adolescents. *Pediatrics*. 2005;116(4):e518-24. PMID:
 1184 16199680. <http://www.ncbi.nlm.nih.gov/pubmed/16199680>.
- 1185 28. Thompson OM, Ballew C, Resnicow K, Must A, Bandini LG, Cyr H, et al. Food
 1186 purchased away from home as a predictor of change in BMI z-score among girls. *Int J*
 1187 *Obes Relat Metab Disord*. 2004;28(2):282-9. PMID: 14647177.
 1188 <http://www.ncbi.nlm.nih.gov/pubmed/14647177>.
- 1189 29. Bisset S, Gauvin L, Potvin L, Paradis G. Association of body mass index and dietary
 1190 restraint with changes in eating behaviour throughout late childhood and early
 1191 adolescence: a 5-year study. *Public Health Nutr*. 2007;10(8):780-9. PMID: 17381909.
 1192 <http://www.ncbi.nlm.nih.gov/pubmed/17381909>.
- 1193 30. Anderson SE, Whitaker RC. Household routines and obesity in US preschool-aged
 1194 children. *Pediatrics*. 2010;125(3):420-8. PMID: 20142280.
 1195 <http://www.ncbi.nlm.nih.gov/pubmed/20142280>.
- 1196 31. Ayala GX, Baquero B, Arredondo EM, Campbell N, Larios S, Elder JP. Association
 1197 between family variables and Mexican American children's dietary behaviors. *J Nutr*
 1198 *Educ Behav*. 2007;39(2):62-9. PMID: 17346653.
 1199 <http://www.ncbi.nlm.nih.gov/pubmed/17346653>.

- 1200 32. Gillman MW, Rifas-Shiman SL, Frazier AL, Rockett HR, Camargo CA, Jr., Field AE, et
1201 al. Family dinner and diet quality among older children and adolescents. *Arch Fam Med.*
1202 2000;9(3):235-40. PMID: 10728109. <http://www.ncbi.nlm.nih.gov/pubmed/10728109>.
- 1203 33. Videon TM, Manning CK. Influences on adolescent eating patterns: the importance of
1204 family meals. *J Adolesc Health.* 2003;32(5):365-73. PMID: 12729986.
1205 <http://www.ncbi.nlm.nih.gov/pubmed/12729986>.
- 1206 34. Hammons AJ, Fiese BH. Is frequency of shared family meals related to the nutritional
1207 health of children and adolescents? *Pediatrics.* 2011;127(6):e1565-74. PMID: 21536618.
1208 <http://www.ncbi.nlm.nih.gov/pubmed/21536618>.
- 1209 35. Sobal J, Hanson K. Family meals and body weight in US adults. *Public Health Nutr.*
1210 2011;14(9):1555-62. PMID: 21356147. <http://www.ncbi.nlm.nih.gov/pubmed/21356147>.
- 1211 36. Chan JC, Sobal J. Family meals and body weight. Analysis of multiple family members
1212 in family units. *Appetite.* 2011;57(2):517-24. PMID: 21763740.
1213 <http://www.ncbi.nlm.nih.gov/pubmed/21763740>.
- 1214 37. Fulkerson JA, Larson N, Horning M, Neumark-Sztainer D. A review of associations
1215 between family or shared meal frequency and dietary and weight status outcomes across
1216 the lifespan. *J Nutr Educ Behav.* 2014;46(1):2-19. PMID: 24054888.
1217 <http://www.ncbi.nlm.nih.gov/pubmed/24054888>.
- 1218 38. Savage JS, Fisher JO, Birch LL. Parental influence on eating behavior: conception to
1219 adolescence. *J Law Med Ethics.* 2007;35(1):22-34. PMID: 17341215.
1220 <http://www.ncbi.nlm.nih.gov/pubmed/17341215>.
- 1221 39. Ventura AK, Birch LL. Does parenting affect children's eating and weight status? *Int J*
1222 *Behav Nutr Phys Act.* 2008;5:15. PMID: 18346282.
1223 <http://www.ncbi.nlm.nih.gov/pubmed/18346282>.
- 1224 40. Burgess-Champoux TL, Larson N, Neumark-Sztainer D, Hannan PJ, Story M. Are family
1225 meal patterns associated with overall diet quality during the transition from early to
1226 middle adolescence? *J Nutr Educ Behav.* 2009;41(2):79-86. PMID: 19304252.
1227 <http://www.ncbi.nlm.nih.gov/pubmed/19304252>.
- 1228 41. Larson N, Fulkerson J, Story M, Neumark-Sztainer D. Shared meals among young adults
1229 are associated with better diet quality and predicted by family meal patterns during
1230 adolescence. *Public Health Nutr.* 2013;16(5):883-93. PMID: 22857517.
1231 <http://www.ncbi.nlm.nih.gov/pubmed/22857517>.
- 1232 42. Haines J, McDonald J, O'Brien A, Sherry B, Bottino CJ, Schmidt ME, et al. Healthy
1233 Habits, Happy Homes: randomized trial to improve household routines for obesity
1234 prevention among preschool-aged children. *JAMA Pediatr.* 2013;167(11):1072-9. PMID:
1235 24019074. <http://www.ncbi.nlm.nih.gov/pubmed/24019074>.
- 1236 43. Fulkerson JA, Neumark-Sztainer D, Hannan PJ, Story M. Family meal frequency and
1237 weight status among adolescents: cross-sectional and 5-year longitudinal associations.
1238 *Obesity (Silver Spring).* 2008;16(11):2529-34. PMID: 18719674.
1239 <http://www.ncbi.nlm.nih.gov/pubmed/18719674>.
- 1240 44. Gable S, Chang Y, Krull JL. Television watching and frequency of family meals are
1241 predictive of overweight onset and persistence in a national sample of school-aged
1242 children. *J Am Diet Assoc.* 2007;107(1):53-61. PMID: 17197271.
1243 <http://www.ncbi.nlm.nih.gov/pubmed/17197271>.

- 1244 45. Miller DP. Associations between the home and school environments and child body mass
1245 index. *Soc Sci Med*. 2011;72(5):677-84. PMID: 21227558.
1246 <http://www.ncbi.nlm.nih.gov/pubmed/21227558>.
- 1247 46. Sen B. Frequency of family dinner and adolescent body weight status: evidence from the
1248 national longitudinal survey of youth, 1997. *Obesity (Silver Spring)*. 2006;14(12):2266-
1249 76. PMID: 17189555. <http://www.ncbi.nlm.nih.gov/pubmed/17189555>.
- 1250 47. Taveras EM, Rifas-Shiman SL, Berkey CS, Rockett HR, Field AE, Frazier AL, et al.
1251 Family dinner and adolescent overweight. *Obes Res*. 2005;13(5):900-6. PMID:
1252 15919844. <http://www.ncbi.nlm.nih.gov/pubmed/15919844>.
- 1253 48. United States. Department of Health and Human Services. 2008 physical activity
1254 guidelines for Americans : be active, healthy, and happy! ODPHP publication. 2008(no
1255 U0036):ix, 61 p. PMID: 15630651. <http://www.health.gov/paguidelines/pdf/paguide.pdf>.
- 1256 49. Healthy People 2020. PA-2. Increase the proportion of adults who meet current federal
1257 physical activity guidelines for aerobic physical activity and for muscle-strengthening
1258 activity. Washington, DC: U.S. Department of Health and Human Services. Office of
1259 Disease Prevention and Health Promotion; [cited 2014]. Available from:
1260 <https://www.healthypeople.gov/2020/topics-objectives/topic/physical-activity/objectives>.
- 1261 50. Healthy People 2020. PA-3. Increase the proportion of adolescents who meet current
1262 federal physical activity guidelines for aerobic physical activity and for muscle-
1263 strengthening activity. Washington, DC: U.S. Department of Health and Human Services.
1264 Office of Disease Prevention and Health Promotion; [cited 2014]. Available from:
1265 <https://www.healthypeople.gov/2020/topics-objectives/topic/physical-activity/objectives>.
- 1266 51. Healthy People 2020. PA-1. Reduce the proportion of adults who engage in no leisure-
1267 time physical activity. Washington, DC: U.S. Department of Health and Human Services.
1268 Office of Disease Prevention and Health Promotion; [cited 2014]. Available from:
1269 https://www.healthypeople.gov/node/5052/data_details.
- 1270 52. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Effect of physical
1271 inactivity on major non-communicable diseases worldwide: an analysis of burden of
1272 disease and life expectancy. *Lancet*. 2012;380(9838):219-29. PMID: 22818936.
1273 <http://www.ncbi.nlm.nih.gov/pubmed/22818936>.
- 1274 53. Policy Statement: children, adolescents, and the media. *Pediatrics*. 2013;132(5):958-61.
1275 <http://pediatrics.aappublications.org/content/132/5/958.full.html>.
- 1276 54. Rideout V. Zero to eight: children's media use in America 2013. 2013.
1277 <https://www.common sense media.org/sites/default/files/research/zero-to-eight-2013.pdf>.
- 1278 55. 2014 United States Report Card on Physical Activity for Children and Youth. 2014.
1279 http://www.physicalactivityplan.org/reportcard/NationalReportCard_longform_final%20for%20web.pdf.
- 1280
- 1281 56. Fakhouri TH, Hughes JP, Brody DJ, Kit BK, Ogden CL. Physical activity and screen-
1282 time viewing among elementary school-aged children in the United States from 2009 to
1283 2010. *JAMA Pediatr*. 2013;167(3):223-9. PMID: 23303439.
1284 <http://www.ncbi.nlm.nih.gov/pubmed/23303439>.
- 1285 57. Blanck HM, McCullough ML, Patel AV, Gillespie C, Calle EE, Cokkinides VE, et al.
1286 Sedentary behavior, recreational physical activity, and 7-year weight gain among
1287 postmenopausal U.S. women. *Obesity (Silver Spring)*. 2007;15(6):1578-88. PMID:
1288 17557996. <http://www.ncbi.nlm.nih.gov/pubmed/17557996>.

- 1289 58. Boone JE, Gordon-Larsen P, Adair LS, Popkin BM. Screen time and physical activity
1290 during adolescence: longitudinal effects on obesity in young adulthood. *Int J Behav Nutr*
1291 *Phys Act.* 2007;4:26. PMID: 17559668. <http://www.ncbi.nlm.nih.gov/pubmed/17559668>.
- 1292 59. De Cocker KA, van Uffelen JG, Brown WJ. Associations between sitting time and
1293 weight in young adult Australian women. *Prev Med.* 2010;51(5):361-7. PMID:
1294 20638408. <http://www.ncbi.nlm.nih.gov/pubmed/20638408>.
- 1295 60. Ding D, Sugiyama T, Owen N. Habitual active transport, TV viewing and weight gain: a
1296 four year follow-up study. *Prev Med.* 2012;54(3-4):201-4. PMID: 22342646.
1297 <http://www.ncbi.nlm.nih.gov/pubmed/22342646>.
- 1298 61. Ekelund U, Brage S, Besson H, Sharp S, Wareham NJ. Time spent being sedentary and
1299 weight gain in healthy adults: reverse or bidirectional causality? *Am J Clin Nutr.*
1300 2008;88(3):612-7. PMID: 18779275. <http://www.ncbi.nlm.nih.gov/pubmed/18779275>.
- 1301 62. French SA, Mitchell NR, Hannan PJ. Decrease in television viewing predicts lower body
1302 mass index at 1-year follow-up in adolescents, but not adults. *J Nutr Educ Behav.*
1303 2012;44(5):415-22. PMID: 22591582. <http://www.ncbi.nlm.nih.gov/pubmed/22591582>.
- 1304 63. Hancox RJ, Milne BJ, Poulton R. Association between child and adolescent television
1305 viewing and adult health: a longitudinal birth cohort study. *Lancet.* 2004;364(9430):257-
1306 62. PMID: 15262103. <http://www.ncbi.nlm.nih.gov/pubmed/15262103>.
- 1307 64. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and long-term risk
1308 for obesity: a 32-year prospective birth cohort study. *Pediatrics.* 2008;122(5):955-60.
1309 PMID: 18977973. <http://www.ncbi.nlm.nih.gov/pubmed/18977973>.
- 1310 65. Mamun AA, O'Callaghan MJ, Williams G, Najman JM. Television watching from
1311 adolescence to adulthood and its association with BMI, waist circumference, waist-to-hip
1312 ratio and obesity: a longitudinal study. *Public Health Nutr.* 2013;16(1):54-64. PMID:
1313 22687709. <http://www.ncbi.nlm.nih.gov/pubmed/22687709>.
- 1314 66. Meyer AM, Evenson KR, Couper DJ, Stevens J, Pereria MA, Heiss G. Television,
1315 physical activity, diet, and body weight status: the ARIC cohort. *Int J Behav Nutr Phys*
1316 *Act.* 2008;5:68. PMID: 19091124. <http://www.ncbi.nlm.nih.gov/pubmed/19091124>.
- 1317 67. Mortensen LH, Siegler IC, Barefoot JC, Gronbaek M, Sorensen TI. Prospective
1318 associations between sedentary lifestyle and BMI in midlife. *Obesity (Silver Spring).*
1319 2006;14(8):1462-71. PMID: 16988090. <http://www.ncbi.nlm.nih.gov/pubmed/16988090>.
- 1320 68. Nunez-Cordoba JM, Bes-Rastrollo M, Pollack KM, Segui-Gomez M, Beunza JJ, Sayon-
1321 Orea C, et al. Annual motor vehicle travel distance and incident obesity: a prospective
1322 cohort study. *Am J Prev Med.* 2013;44(3):254-9. PMID: 23415122.
1323 <http://www.ncbi.nlm.nih.gov/pubmed/23415122>.
- 1324 69. Parsons TJ, Manor O, Power C. Television viewing and obesity: a prospective study in
1325 the 1958 British birth cohort. *Eur J Clin Nutr.* 2008;62(12):1355-63. PMID: 17717536.
1326 <http://www.ncbi.nlm.nih.gov/pubmed/17717536>.
- 1327 70. Pulsford RM, Stamatakis E, Britton AR, Brunner EJ, Hillsdon MM. Sitting behavior and
1328 obesity: evidence from the Whitehall II study. *Am J Prev Med.* 2013;44(2):132-8. PMID:
1329 23332328. <http://www.ncbi.nlm.nih.gov/pubmed/23332328>.
- 1330 71. Raynor DA, Phelan S, Hill JO, Wing RR. Television viewing and long-term weight
1331 maintenance: results from the National Weight Control Registry. *Obesity (Silver Spring).*
1332 2006;14(10):1816-24. PMID: 17062812.
1333 <http://www.ncbi.nlm.nih.gov/pubmed/17062812>.

- 1334 72. Saunders TJ, Tremblay MS, Despres JP, Bouchard C, Tremblay A, Chaput JP. Sedentary
1335 behaviour, visceral fat accumulation and cardiometabolic risk in adults: a 6-year
1336 longitudinal study from the Quebec Family Study. *PLoS One*. 2013;8(1):e54225. PMID:
1337 23326600. <http://www.ncbi.nlm.nih.gov/pubmed/23326600>.
- 1338 73. Stamatakis E, Hamer M, Mishra GD. Early adulthood television viewing and
1339 cardiometabolic risk profiles in early middle age: results from a population, prospective
1340 cohort study. *Diabetologia*. 2012;55(2):311-20. PMID: 22057195.
1341 <http://www.ncbi.nlm.nih.gov/pubmed/22057195>.
- 1342 74. Sugiyama T, Ding D, Owen N. Commuting by car: weight gain among physically active
1343 adults. *Am J Prev Med*. 2013;44(2):169-73. PMID: 23332335.
1344 <http://www.ncbi.nlm.nih.gov/pubmed/23332335>.
- 1345 75. van Uffelen JG, Watson MJ, Dobson AJ, Brown WJ. Sitting time is associated with
1346 weight, but not with weight gain in mid-aged Australian women. *Obesity (Silver Spring)*.
1347 2010;18(9):1788-94. PMID: 20111018. <http://www.ncbi.nlm.nih.gov/pubmed/20111018>.
- 1348 76. Viner RM, Cole TJ. Television viewing in early childhood predicts adult body mass
1349 index. *J Pediatr*. 2005;147(4):429-35. PMID: 16227025.
1350 <http://www.ncbi.nlm.nih.gov/pubmed/16227025>.
- 1351 77. Wijndaele K, Healy GN, Dunstan DW, Barnett AG, Salmon J, Shaw JE, et al. Increased
1352 cardiometabolic risk is associated with increased TV viewing time. *Med Sci Sports
1353 Exerc*. 2010;42(8):1511-8. PMID: 20139784.
1354 <http://www.ncbi.nlm.nih.gov/pubmed/20139784>.
- 1355 78. Pinto Pereira SM, Power C. Sedentary behaviours in mid-adulthood and subsequent body
1356 mass index. *PLoS One*. 2013;8(6):e65791. PMID: 23762427.
1357 <http://www.ncbi.nlm.nih.gov/pubmed/23762427>.
- 1358 79. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and
1359 long-term weight gain in women and men. *N Engl J Med*. 2011;364(25):2392-404.
1360 PMID: 21696306. <http://www.ncbi.nlm.nih.gov/pubmed/21696306>.
- 1361 80. Increasing physical activity: behavioral and social approaches: Guide to Community
1362 Preventive Services; [cited 2014 September 10]. Available from:
1363 <http://www.thecommunityguide.org/pa/behavioral-social/index.html>.
- 1364 81. Burke LE, Wang J, Sevick MA. Self-monitoring in weight loss: a systematic review of
1365 the literature. *J Am Diet Assoc*. 2011;111(1):92-102. PMID: 21185970.
1366 <http://www.ncbi.nlm.nih.gov/pubmed/21185970>.
- 1367 82. Adachi Y, Sato C, Yamatsu K, Ito S, Adachi K, Yamagami T. A randomized controlled
1368 trial on the long-term effects of a 1-month behavioral weight control program assisted by
1369 computer tailored advice. *Behav Res Ther*. 2007;45(3):459-70. PMID: 16713991.
1370 <http://www.ncbi.nlm.nih.gov/pubmed/16713991>.
- 1371 83. de Niet J, Timman R, Bauer S, van den Akker E, de Klerk C, Kordy H, et al. Short
1372 message service reduces dropout in childhood obesity treatment: a randomized controlled
1373 trial. *Health Psychol*. 2012;31(6):797-805. PMID: 22468714.
1374 <http://www.ncbi.nlm.nih.gov/pubmed/22468714>.
- 1375 84. Steinberg DM, Tate DF, Bennett GG, Ennett S, Samuel-Hodge C, Ward DS. The efficacy
1376 of a daily self-weighing weight loss intervention using smart scales and e-mail. *Obesity
1377 (Silver Spring)*. 2013;21(9):1789-97. PMID: 23512320.
1378 <http://www.ncbi.nlm.nih.gov/pubmed/23512320>.

- 1379 85. Wing RR, Crane MM, Thomas JG, Kumar R, Weinberg B. Improving weight loss
 1380 outcomes of community interventions by incorporating behavioral strategies. *Am J*
 1381 *Public Health*. 2010;100(12):2513-9. PMID: 20966375.
 1382 <http://www.ncbi.nlm.nih.gov/pubmed/20966375>.
- 1383 86. Bartfield JK, Stevens VJ, Jerome GJ, Batch BC, Kennedy BM, Vollmer WM, et al.
 1384 Behavioral transitions and weight change patterns within the PREMIER trial. *Obesity*
 1385 (Silver Spring). 2011;19(8):1609-15. PMID: 21455122.
 1386 <http://www.ncbi.nlm.nih.gov/pubmed/21455122>.
- 1387 87. Burke LE, Styn MA, Sereika SM, Conroy MB, Ye L, Glanz K, et al. Using mHealth
 1388 technology to enhance self-monitoring for weight loss: a randomized trial. *Am J Prev*
 1389 *Med*. 2012;43(1):20-6. PMID: 22704741.
 1390 <http://www.ncbi.nlm.nih.gov/pubmed/22704741>.
- 1391 88. Harrison CL, Teede HJ, Lombard CB. How effective is self-weighing in the setting of a
 1392 lifestyle intervention to reduce gestational weight gain and postpartum weight retention?
 1393 *Aust N Z J Obstet Gynaecol*. 2014;54(4):382-5. PMID: 24738837.
 1394 <http://www.ncbi.nlm.nih.gov/pubmed/24738837>.
- 1395 89. Kong A, Beresford SA, Alfano CM, Foster-Schubert KE, Neuhauser ML, Johnson DB, et
 1396 al. Self-monitoring and eating-related behaviors are associated with 12-month weight loss
 1397 in postmenopausal overweight-to-obese women. *J Acad Nutr Diet*. 2012;112(9):1428-35.
 1398 PMID: 22795495. <http://www.ncbi.nlm.nih.gov/pubmed/22795495>.
- 1399 90. Linde JA, Jeffery RW, French SA, Pronk NP, Boyle RG. Self-weighing in weight gain
 1400 prevention and weight loss trials. *Ann Behav Med*. 2005;30(3):210-6. PMID: 16336072.
 1401 <http://www.ncbi.nlm.nih.gov/pubmed/16336072>.
- 1402 91. Peterson ND, Middleton KR, Nackers LM, Medina KE, Milsom VA, Perri MG. Dietary
 1403 self-monitoring and long-term success with weight management. *Obesity (Silver Spring)*.
 1404 2014;22(9):1962-7. PMID: 24931055. <http://www.ncbi.nlm.nih.gov/pubmed/24931055>.
- 1405 92. Steinberg DM, Levine EL, Lane I, Askew S, Foley PB, Puleo E, et al. Adherence to self-
 1406 monitoring via interactive voice response technology in an eHealth intervention targeting
 1407 weight gain prevention among Black women: randomized controlled trial. *J Med Internet*
 1408 *Res*. 2014;16(4):e114. PMID: 24780934.
 1409 <http://www.ncbi.nlm.nih.gov/pubmed/24780934>.
- 1410 93. Turk MW, Elci OU, Wang J, Sereika SM, Ewing LJ, Acharya SD, et al. Self-monitoring
 1411 as a mediator of weight loss in the SMART randomized clinical trial. *Int J Behav Med*.
 1412 2013;20(4):556-61. PMID: 22936524. <http://www.ncbi.nlm.nih.gov/pubmed/22936524>.
- 1413 94. Turner-McGrievy GM, Beets MW, Moore JB, Kaczynski AT, Barr-Anderson DJ, Tate
 1414 DF. Comparison of traditional versus mobile app self-monitoring of physical activity and
 1415 dietary intake among overweight adults participating in an mHealth weight loss program.
 1416 *J Am Med Inform Assoc*. 2013;20(3):513-8. PMID: 23429637.
 1417 <http://www.ncbi.nlm.nih.gov/pubmed/23429637>.
- 1418 95. VanWormer JJ, Linde JA, Harnack LJ, Stovitz SD, Jeffery RW. Self-weighing frequency
 1419 is associated with weight gain prevention over 2 years among working adults. *Int J Behav*
 1420 *Med*. 2012;19(3):351-8. PMID: 21732212.
 1421 <http://www.ncbi.nlm.nih.gov/pubmed/21732212>.
- 1422 96. VanWormer JJ, Martinez AM, Martinson BC, Crain AL, Benson GA, Cosentino DL, et
 1423 al. Self-weighing promotes weight loss for obese adults. *Am J Prev Med*. 2009;36(1):70-
 1424 3. PMID: 18976879. <http://www.ncbi.nlm.nih.gov/pubmed/18976879>.

- 1425 97. Wing RR, Hamman RF, Bray GA, Delahanty L, Edelstein SL, Hill JO, et al. Achieving
 1426 weight and activity goals among diabetes prevention program lifestyle participants. *Obes*
 1427 *Res.* 2004;12(9):1426-34. PMID: 15483207.
 1428 <http://www.ncbi.nlm.nih.gov/pubmed/15483207>.
- 1429 98. Wing RR, Papandonatos G, Fava JL, Gorin AA, Phelan S, McCaffery J, et al.
 1430 Maintaining large weight losses: the role of behavioral and psychological factors. *J*
 1431 *Consult Clin Psychol.* 2008;76(6):1015-21. PMID: 19045969.
 1432 <http://www.ncbi.nlm.nih.gov/pubmed/19045969>.
- 1433 99. Ma J, Xiao L, Blonstein AC. Measurement of self-monitoring web technology acceptance
 1434 and use in an e-health weight-loss trial. *Telemed J E Health.* 2013;19(10):739-45. PMID:
 1435 23952787. <http://www.ncbi.nlm.nih.gov/pubmed/23952787>.
- 1436 100. Krukowski RA, Harvey-Berino J, Bursac Z, Ashikaga T, West DS. Patterns of success:
 1437 online self-monitoring in a web-based behavioral weight control program. *Health*
 1438 *Psychol.* 2013;32(2):164-70. PMID: 22545978.
 1439 <http://www.ncbi.nlm.nih.gov/pubmed/22545978>.
- 1440 101. Johnson F, Wardle J. The association between weight loss and engagement with a web-
 1441 based food and exercise diary in a commercial weight loss programme: a retrospective
 1442 analysis. *Int J Behav Nutr Phys Act.* 2011;8:83. PMID: 21810222.
 1443 <http://www.ncbi.nlm.nih.gov/pubmed/21810222>.
- 1444 102. Sinclair SE, Cooper M, Mansfield ED. The influence of menu labeling on calories
 1445 selected or consumed: a systematic review and meta-analysis. *J Acad Nutr Diet.*
 1446 2014;114(9):1375-88 e15. PMID: 25037558.
 1447 <http://www.ncbi.nlm.nih.gov/pubmed/25037558>.
- 1448 103. Burton S, Creyer EH, Kees J, Huggins K. Attacking the obesity epidemic: the potential
 1449 health benefits of providing nutrition information in restaurants. *Am J Public Health.*
 1450 2006;96(9):1669-75. PMID: 16873758. <http://www.ncbi.nlm.nih.gov/pubmed/16873758>.
- 1451 104. Dowray S, Swartz JJ, Braxton D, Viera AJ. Potential effect of physical activity based
 1452 menu labels on the calorie content of selected fast food meals. *Appetite.* 2013;62:173-81.
 1453 PMID: 23220355. <http://www.ncbi.nlm.nih.gov/pubmed/23220355>.
- 1454 105. Ellison B, Lusk JL, Davis D. Looking at the label and beyond: the effects of calorie
 1455 labels, health consciousness, and demographics on caloric intake in restaurants. *Int J*
 1456 *Behav Nutr Phys Act.* 2013;10:21. PMID: 23394433.
 1457 <http://www.ncbi.nlm.nih.gov/pubmed/23394433>.
- 1458 106. Gerend MA. Does calorie information promote lower calorie fast food choices among
 1459 college students? *J Adolesc Health.* 2009;44(1):84-6. PMID: 19101463.
 1460 <http://www.ncbi.nlm.nih.gov/pubmed/19101463>.
- 1461 107. Harnack LJ, French SA, Oakes JM, Story MT, Jeffery RW, Rydell SA. Effects of calorie
 1462 labeling and value size pricing on fast food meal choices: results from an experimental
 1463 trial. *Int J Behav Nutr Phys Act.* 2008;5:63. PMID: 19061510.
 1464 <http://www.ncbi.nlm.nih.gov/pubmed/19061510>.
- 1465 108. James A, Adams-Huet B, Shah M. Menu Labels Displaying the Kilocalorie Content or
 1466 the Exercise Equivalent: Effects on Energy Ordered and Consumed in Young Adults. *Am*
 1467 *J Health Promot.* 2014. PMID: 24575727.
 1468 <http://www.ncbi.nlm.nih.gov/pubmed/24575727>.

- 1469 109. Roberto CA, Larsen PD, Agnew H, Baik J, Brownell KD. Evaluating the impact of menu
1470 labeling on food choices and intake. *Am J Public Health*. 2010;100(2):312-8. PMID:
1471 20019307. <http://www.ncbi.nlm.nih.gov/pubmed/20019307>.
- 1472 110. Roseman MG, Mathe-Souleik K, Higgins JA. Relationships among grocery nutrition label
1473 users and consumers' attitudes and behavior toward restaurant menu labeling. *Appetite*.
1474 2013;71:274-8. PMID: 24012965. <http://www.ncbi.nlm.nih.gov/pubmed/24012965>.
- 1475 111. Tandon PS, Wright J, Zhou C, Rogers CB, Christakis DA. Nutrition menu labeling may
1476 lead to lower-calorie restaurant meal choices for children. *Pediatrics*. 2010;125(2):244-8.
1477 PMID: 20100765. <http://www.ncbi.nlm.nih.gov/pubmed/20100765>.
- 1478 112. Wisdom J, Downs JS, Loewenstein G. Promoting health choices: information versus
1479 convenience. *Am Econ J Appl Econ*. 2010;2(2):164-78.
- 1480 113. Laraia BA, Siega-Riz AM, Gundersen C, Dole N. Psychosocial factors and
1481 socioeconomic indicators are associated with household food insecurity among pregnant
1482 women. *J Nutr*. 2006;136(1):177-82. PMID: 16365079.
1483 <http://www.ncbi.nlm.nih.gov/pubmed/16365079>.
- 1484 114. Perez-Escamilla R, Pinheiro de Toledo Vianna R. Food insecurity and the behavioral and
1485 intellectual development of children: a review of the evidence. *J Appl Res Child*.
1486 2012;3(1). <http://digitalcommons.library.tmc.edu/childrenatrisk/vol3/iss1/9/>.
- 1487 115. Leung CW, Epel ES, Ritchie LD, Crawford PB, Laraia BA. Food Insecurity Is Inversely
1488 Associated with Diet Quality of Lower-Income Adults. *J Acad Nutr Diet*.
1489 2014;114(12):1943-53 e2. PMID: 25091796.
1490 <http://www.ncbi.nlm.nih.gov/pubmed/25091796>.
- 1491 116. Hartline-Grafton H. Food Insecurity and Obesity: understanding the connections. 2011.
1492 http://frac.org/pdf/frac_brief_understanding_the_connections.pdf.
- 1493 117. Seligman HK, Jacobs EA, Lopez A, Tschann J, Fernandez A. Food insecurity and
1494 glycemic control among low-income patients with type 2 diabetes. *Diabetes Care*.
1495 2012;35(2):233-8. PMID: 22210570. <http://www.ncbi.nlm.nih.gov/pubmed/22210570>.
- 1496 118. Laraia BA, Siega-Riz AM, Gundersen C. Household food insecurity is associated with
1497 self-reported pregravid weight status, gestational weight gain, and pregnancy
1498 complications. *J Am Diet Assoc*. 2010;110(5):692-701. PMID: 20430130.
1499 <http://www.ncbi.nlm.nih.gov/pubmed/20430130>.
- 1500 119. Eisenmann JC, Gundersen C, Lohman BJ, Garasky S, Stewart SD. Is food insecurity
1501 related to overweight and obesity in children and adolescents? A summary of studies,
1502 1995-2009. *Obes Rev*. 2011;12(5):e73-83. PMID: 21382151.
1503 <http://www.ncbi.nlm.nih.gov/pubmed/21382151>.
- 1504 120. Hunger and obesity: understanding a food insecurity paradigm: workshop summary.
1505 2011. http://www.nap.edu/openbook.php?record_id=13102.
- 1506 121. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav*.
1507 2007;91(4):449-58. PMID: 17543357. <http://www.ncbi.nlm.nih.gov/pubmed/17543357>.
- 1508 122. Laraia BA. Food insecurity and chronic disease. *Adv Nutr*. 2013;4(2):203-12. PMID:
1509 23493536. <http://www.ncbi.nlm.nih.gov/pubmed/23493536>.
- 1510 123. Bhargava A, Jolliffe D, Howard LL. Socio-economic, behavioural and environmental
1511 factors predicted body weights and household food insecurity scores in the Early
1512 Childhood Longitudinal Study-Kindergarten. *Br J Nutr*. 2008;100(2):438-44. PMID:
1513 18275621. <http://www.ncbi.nlm.nih.gov/pubmed/18275621>.

- 1514 124. Dubois L, Farmer A, Girard M, Porcherie M. Family food insufficiency is related to
 1515 overweight among preschoolers. *Soc Sci Med*. 2006;63(6):1503-16. PMID: 16777308.
 1516 <http://www.ncbi.nlm.nih.gov/pubmed/16777308>.
- 1517 125. Jyoti DF, Frongillo EA, Jones SJ. Food insecurity affects school children's academic
 1518 performance, weight gain, and social skills. *J Nutr*. 2005;135(12):2831-9. PMID:
 1519 16317128. <http://www.ncbi.nlm.nih.gov/pubmed/16317128>.
- 1520 126. Kim K, Frongillo EA. Participation in food assistance programs modifies the relation of
 1521 food insecurity with weight and depression in elders. *J Nutr*. 2007;137(4):1005-10.
 1522 PMID: 17374668. <http://www.ncbi.nlm.nih.gov/pubmed/17374668>.
- 1523 127. Metallinos-Katsaras E, Must A, Gorman K. A longitudinal study of food insecurity on
 1524 obesity in preschool children. *J Acad Nutr Diet*. 2012;112(12):1949-58. PMID:
 1525 23174682. <http://www.ncbi.nlm.nih.gov/pubmed/23174682>.
- 1526 128. Olson CM, Strawderman MS. The relationship between food insecurity and obesity in
 1527 rural childbearing women. *J Rural Health*. 2008;24(1):60-6. PMID: 18257872.
 1528 <http://www.ncbi.nlm.nih.gov/pubmed/18257872>.
- 1529 129. Rose D, Bodor JN. Household food insecurity and overweight status in young school
 1530 children: results from the Early Childhood Longitudinal Study. *Pediatrics*.
 1531 2006;117(2):464-73. PMID: 16452367. <http://www.ncbi.nlm.nih.gov/pubmed/16452367>.
- 1532 130. Whitaker RC, Sarin A. Change in food security status and change in weight are not
 1533 associated in urban women with preschool children. *J Nutr*. 2007;137(9):2134-9. PMID:
 1534 17709454. <http://www.ncbi.nlm.nih.gov/pubmed/17709454>.
- 1535 131. Weight gain during pregnancy. 2009.
 1536 http://books.nap.edu/openbook.php?record_id=12584&page=R1.
- 1537 132. Corral I, Landrine H. Acculturation and ethnic-minority health behavior: a test of the
 1538 operant model. *Health Psychol*. 2008;27(6):737-45. PMID: 19025269.
 1539 <http://www.ncbi.nlm.nih.gov/pubmed/19025269>.
- 1540 133. Creighton MJ, Goldman N, Pebley AR, Chung CY. Durational and generational
 1541 differences in Mexican immigrant obesity: is acculturation the explanation? *Soc Sci Med*.
 1542 2012;75(2):300-10. PMID: 22575698. <http://www.ncbi.nlm.nih.gov/pubmed/22575698>.
- 1543 134. Dubowitz T, Subramanian SV, Acevedo-Garcia D, Osypuk TL, Peterson KE. Individual
 1544 and neighborhood differences in diet among low-income foreign and U.S.-born women.
 1545 *Womens Health Issues*. 2008;18(3):181-90. PMID: 18222706.
 1546 <http://www.ncbi.nlm.nih.gov/pubmed/18222706>.
- 1547 135. Erinoshio TO, Berrigan D, Thompson FE, Moser RP, Nebeling LC, Yaroch AL. Dietary
 1548 intakes of preschool-aged children in relation to caregivers' race/ethnicity, acculturation,
 1549 and demographic characteristics: results from the 2007 California Health Interview
 1550 Survey. *Matern Child Health J*. 2012;16(9):1844-53. PMID: 22160613.
 1551 <http://www.ncbi.nlm.nih.gov/pubmed/22160613>.
- 1552 136. Ghaddar S, Brown CJ, Pagan JA, Diaz V. Acculturation and healthy lifestyle habits
 1553 among Hispanics in United States-Mexico border communities. *Rev Panam Salud
 1554 Publica*. 2010;28(3):190-7. PMID: 20963266.
 1555 <http://www.ncbi.nlm.nih.gov/pubmed/20963266>.
- 1556 137. Gomez SL, Kelsey JL, Glaser SL, Lee MM, Sidney S. Immigration and acculturation in
 1557 relation to health and health-related risk factors among specific Asian subgroups in a
 1558 health maintenance organization. *Am J Public Health*. 2004;94(11):1977-84. PMID:
 1559 15514240. <http://www.ncbi.nlm.nih.gov/pubmed/15514240>.

- 1560 138. Kasirye OC, Walsh JA, Romano PS, Beckett LA, Garcia JA, Elvine-Kreis B, et al.
 1561 Acculturation and its association with health-risk behaviors in a rural Latina population.
 1562 *Ethn Dis.* 2005;15(4):733-9. PMID: 16259501.
 1563 <http://www.ncbi.nlm.nih.gov/pubmed/16259501>.
- 1564 139. Matias SL, Stoecklin-Marois MT, Tancredi DJ, Schenker MB. Adherence to dietary
 1565 recommendations is associated with acculturation among Latino farm workers. *J Nutr.*
 1566 2013;143(9):1451-8. PMID: 23864507. <http://www.ncbi.nlm.nih.gov/pubmed/23864507>.
- 1567 140. Montez JK, Eschbach K. Country of birth and language are uniquely associated with
 1568 intakes of fat, fiber, and fruits and vegetables among Mexican-American women in the
 1569 United States. *J Am Diet Assoc.* 2008;108(3):473-80. PMID: 18313430.
 1570 <http://www.ncbi.nlm.nih.gov/pubmed/18313430>.
- 1571 141. Parikh NS, Fahs MC, Shelley D, Yerneni R. Health behaviors of older Chinese adults
 1572 living in New York City. *J Community Health.* 2009;34(1):6-15. PMID: 18830807.
 1573 <http://www.ncbi.nlm.nih.gov/pubmed/18830807>.
- 1574 142. Shin CN, Lach H. Nutritional issues of Korean Americans. *Clin Nurs Res.*
 1575 2011;20(2):162-80. PMID: 21160079. <http://www.ncbi.nlm.nih.gov/pubmed/21160079>.
- 1576 143. Song YJ, Hofstetter CR, Hovell MF, Paik HY, Park HR, Lee J, et al. Acculturation and
 1577 health risk behaviors among Californians of Korean descent. *Prev Med.* 2004;39(1):147-
 1578 56. PMID: 15207996. <http://www.ncbi.nlm.nih.gov/pubmed/15207996>.
- 1579 144. van Rompay MI, McKeown NM, Castaneda-Sceppa C, Falcon LM, Ordovas JM, Tucker
 1580 KL. Acculturation and sociocultural influences on dietary intake and health status among
 1581 Puerto Rican adults in Massachusetts. *J Acad Nutr Diet.* 2012;112(1):64-74. PMID:
 1582 22389874. <http://www.ncbi.nlm.nih.gov/pubmed/22389874>.
- 1583 145. Van Wieren AJ, Roberts MB, Arellano N, Feller ER, Diaz JA. Acculturation and
 1584 cardiovascular behaviors among Latinos in California by country/region of origin. *J*
 1585 *Immigr Minor Health.* 2011;13(6):975-81. PMID: 21626297.
 1586 <http://www.ncbi.nlm.nih.gov/pubmed/21626297>.
- 1587 146. Wang S, Quan J, Kanaya AM, Fernandez A. Asian Americans and obesity in California:
 1588 a protective effect of biculturalism. *J Immigr Minor Health.* 2011;13(2):276-83. PMID:
 1589 21153765. <http://www.ncbi.nlm.nih.gov/pubmed/21153765>.
- 1590 147. Novotny R, Chen C, Williams AE, Albright CL, Nigg CR, Oshiro CE, et al. US
 1591 acculturation is associated with health behaviors and obesity, but not their change, with a
 1592 hotel-based intervention among Asian-Pacific Islanders. *J Acad Nutr Diet.*
 1593 2012;112(5):649-56. PMID: 22709769. <http://www.ncbi.nlm.nih.gov/pubmed/22709769>.
- 1594 148. Unger JB, Reynolds K, Shakib S, Spruijt-Metz D, Sun P, Johnson CA. Acculturation,
 1595 physical activity, and fast-food consumption among Asian-American and Hispanic
 1596 adolescents. *J Community Health.* 2004;29(6):467-81. PMID: 15587346.
 1597 <http://www.ncbi.nlm.nih.gov/pubmed/15587346>.
- 1598 149. Ahluwalia IB, Ford ES, Link M, Bolen JC. Acculturation, weight, and weight-related
 1599 behaviors among Mexican Americans in the United States. *Ethn Dis.* 2007;17(4):643-9.
 1600 PMID: 18072373. <http://www.ncbi.nlm.nih.gov/pubmed/18072373>.
- 1601 150. Chen L, Juon HS, Lee S. Acculturation and BMI among Chinese, Korean and
 1602 Vietnamese adults. *J Community Health.* 2012;37(3):539-46. PMID: 21922164.
 1603 <http://www.ncbi.nlm.nih.gov/pubmed/21922164>.
- 1604 151. Fu H, VanLandingham MJ. Disentangling the effects of migration, selection and
 1605 acculturation on weight and body fat distribution: results from a natural experiment

- 1606 involving Vietnamese Americans, returnees, and never-leavers. *J Immigr Minor Health*.
 1607 2012;14(5):786-96. PMID: 22427069. <http://www.ncbi.nlm.nih.gov/pubmed/22427069>.
- 1608 152. Garcia L, Gold EB, Wang L, Yang X, Mao M, Schwartz AV. The relation of
 1609 acculturation to overweight, obesity, pre-diabetes and diabetes among U.S. Mexican-
 1610 American women and men. *Ethn Dis*. 2012;22(1):58-64. PMID: 22774310.
 1611 <http://www.ncbi.nlm.nih.gov/pubmed/22774310>.
- 1612 153. Hubert HB, Snider J, Winkleby MA. Health status, health behaviors, and acculturation
 1613 factors associated with overweight and obesity in Latinos from a community and
 1614 agricultural labor camp survey. *Prev Med*. 2005;40(6):642-51. PMID: 15850860.
 1615 <http://www.ncbi.nlm.nih.gov/pubmed/15850860>.
- 1616 154. Koya DL, Egede LE. Association between length of residence and cardiovascular disease
 1617 risk factors among an ethnically diverse group of United States immigrants. *J Gen Intern
 1618 Med*. 2007;22(6):841-6. PMID: 17503110.
 1619 <http://www.ncbi.nlm.nih.gov/pubmed/17503110>.
- 1620 155. Daviglius M, Talavera G, Avilés-Santa M, Allison M, Cai J, Criqui M, et al. Prevalence of
 1621 major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino
 1622 individuals of diverse backgrounds in the United States. *JAMA*. 2012;308(17):1775-84.
 1623 PMID: WOS:000310726500024. <http://www.ncbi.nlm.nih.gov/pubmed/23117778>.
- 1624 156. Eamranond PP, Legedza AT, Diez-Roux AV, Kandula NR, Palmas W, Siscovick DS, et
 1625 al. Association between language and risk factor levels among Hispanic adults with
 1626 hypertension, hypercholesterolemia, or diabetes. *Am Heart J*. 2009;157(1):53-9. PMID:
 1627 19081396. <http://www.ncbi.nlm.nih.gov/pubmed/19081396>.
- 1628 157. Eamranond PP, Wee CC, Legedza AT, Marcantonio ER, Leveille SG. Acculturation and
 1629 cardiovascular risk factor control among Hispanic adults in the United States. *Public
 1630 Health Rep*. 2009;124(6):818-24. PMID: 19894424.
 1631 <http://www.ncbi.nlm.nih.gov/pubmed/19894424>.
- 1632 158. Kershaw KN, Greenlund KJ, Stamler J, Shay CM, Daviglius ML. Understanding ethnic
 1633 and nativity-related differences in low cardiovascular risk status among Mexican-
 1634 Americans and non-Hispanic Whites. *Prev Med*. 2012;55(6):597-602. PMID: 23036519.
 1635 <http://www.ncbi.nlm.nih.gov/pubmed/23036519>.
- 1636 159. Kandula NR, Diez-Roux AV, Chan C, Daviglius ML, Jackson SA, Ni H, et al.
 1637 Association of acculturation levels and prevalence of diabetes in the multi-ethnic study of
 1638 atherosclerosis (MESA). *Diabetes Care*. 2008;31(8):1621-8. PMID: 18458142.
 1639 <http://www.ncbi.nlm.nih.gov/pubmed/18458142>.
- 1640 160. Mainous AG, 3rd, Majeed A, Koopman RJ, Baker R, Everett CJ, Tilley BC, et al.
 1641 Acculturation and diabetes among Hispanics: evidence from the 1999-2002 National
 1642 Health and Nutrition Examination Survey. *Public Health Rep*. 2006;121(1):60-6. PMID:
 1643 16416699. <http://www.ncbi.nlm.nih.gov/pubmed/16416699>.

1644

Part D. Chapter 4: Food Environment and Settings

INTRODUCTION

Few American children, adolescents or adults have dietary patterns that are consistent with the Dietary Guidelines for Americans. The reasons for this are numerous, as what people eat is influenced by many complex factors, as discussed in *Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence*. These factors span from individual levels of influence to dimensions of our environment. Improving dietary and lifestyle patterns and reducing diet-related chronic diseases, including obesity, will require actions at the individual behavioral and population and environmental levels. Behavioral strategies are needed to motivate and enhance the capacity of the individual to adopt and improve their lifestyle behaviors.

Specific behavioral efforts related to eating and food[♦] and beverage choices include improving knowledge, attitudes, motivations, and food and cooking skills. Environmental change also is important because the environmental context and conditions affect what and how much people eat and what food choices are available. In addition, actions are needed to address the disparity gaps that currently exist in availability and access to healthy foods in low-income and rural communities.

Health and optimal nutrition and weight management cannot be achieved without a focus on the synergistic linkages and interactions between individuals and their environments, and understanding the different domains of food-related environmental influences. The social environment includes social networks and support systems, such as those provided by family, friends, and community cohesion. The physical environment includes the multiple settings where people obtain and consume food, such as their homes, work places, schools, restaurants, and grocery stores. The macro-environment operates within the broader society and includes food marketing, economic and price structures, food production and distribution systems, transportation, and agricultural practices and policies. Collectively, these environments influence what food choices we make, and where and how much we eat. Although personal responsibility is important, food choices are intertwined with and dependent on the community and environment context.

Interest is growing in the role of the environment in promoting or hindering healthy eating. Although it is up to individuals to decide what and how much they eat and drink, individual behavior to make healthy choices is enhanced when there is a supportive environment with accessible and affordable healthy choices. Thus, individual change is more likely to be facilitated and sustained if the environments within which food choices are made supports healthful options. As with other major public health issues, such as smoking reduction, injury prevention,

[♦] Note: Throughout this chapter, references to “foods” should be taken to mean “foods and beverages.”

37 and infectious disease prevention, greater success at the individual and population levels for
38 reducing obesity and diet-related chronic diseases are not as likely to occur unless environmental
39 influences are identified and modified.

40

41 Meaningful solutions to improve diet and health cannot only be focused just on individuals, or
42 families but must take into account the need for environmental and policy change.

43 Environmental and policy changes can have a sustaining effect on individual behavior change
44 because they can become incorporated into organizational structures and systems, and lead to
45 alterations in sociocultural and societal norms. Both policy and environmental changes also can
46 help reduce disparities by improving access to and availability of healthy food in underserved
47 neighborhoods and communities. Federal nutrition assistance programs, in particular, play a vital
48 role in achieving this objective through access to affordable foods that help millions of
49 Americans meet Dietary Guidelines recommendations.

50

51 The Nation's ultimate goal should be neighborhoods and communities where healthy, affordable
52 food and beverages are available to everyone in the United States in multiple settings, where
53 healthy foods rather than unhealthy foods are the likely choice (optimal default), where social
54 norms embrace and support healthy eating, and where children grow up enjoying the taste of
55 vegetables, fruits, whole grains, and nonfat or low-fat dairy products and water instead of
56 energy-dense foods with low nutrient density and that are high in refined grains, saturated fats,
57 sodium, and added sugars. So too, it is important that these behaviors can be sustained
58 throughout the lifespan and in settings where adults and older adult populations work or are
59 served and reside.

60

61 The questions asked and reviewed in this chapter address place-based environments that
62 influence the foods that individuals, families and households obtain and consume, and on the
63 community settings in which they spend much of their time. The DGAC considered several
64 settings but prioritized four key settings to examine for this report: neighborhood and community
65 food access; child care (early care and education); schools; and worksites. The Committee
66 examined the relationship of these settings to diet quality and weight status. Because of the need
67 to identify effective population-level strategies, the Committee focused specifically on reviewing
68 the scientific literature to determine the impact of place-based obesity prevention and dietary
69 interventions. Because of time demands, the Committee could not address other important
70 settings, such as after-school settings, recreational settings, and faith-based institutions, as well
71 as more macro-environmental influences such as food marketing and economic impacts. Despite
72 the lack of time to examine these settings, the DGAC considers them to be very important
73 environmental influencers on dietary intake.

74

75

76 **LIST OF QUESTIONS**

77 **Food Access**

- 78 1. What is the relationship between neighborhood and community access to food retail settings
79 and individuals' dietary intake and quality?
80 2. What is the relationship between neighborhood and community access to food retail settings
81 and weight status?

82

83 **Early Care and Education**

- 84 3. What is the impact of obesity prevention approaches in early care and education programs on
85 the weight status of children ages 2 to 5 years?

86

87 **Schools**

- 88 4. What is the impact of school-based approaches on the dietary intake, quality, behaviors,
89 and/or preference of school-aged children?
90 5. What is the impact of school-based policies on the dietary intake, quality, behaviors, and/or
91 preferences of school-aged children?
92 6. What is the impact of school-based approaches on the weight status of school-aged children?
93 7. What is the impact of school-based policies on the weight status of school-aged children?

94

95 **Worksite**

- 96 8. What is the impact of worksite-based approaches on the dietary intake, quality, behaviors
97 and/or preferences of employees?
98 9. What is the impact of worksite policies on the dietary intake, quality, behaviors and/or
99 preferences of employees?
100 10. What is the impact of worksite-based approaches on the weight status of employees?
101 11. What is the impact of worksite policies on weight status of employees?

102

103 **METHODOLOGY**

104 Questions related to food access were answered using Nutrition Evidence Library (NEL)
 105 systematic reviews, while questions related to schools and worksites were answered using
 106 existing systematic reviews. The early care and education question was answered using an
 107 existing systematic review with a NEL systematic review update. Descriptions of the NEL
 108 process and the use of existing systematic reviews are provided in *Part C: Methodology*. All
 109 NEL reviews were conducted in accordance with NEL methodology, and the DGAC made all
 110 substantive decisions required throughout the process to ensure that the most complete and
 111 relevant body of evidence was identified and evaluated to answer each question. All steps in the
 112 process were documented to ensure transparency and reproducibility. Specific information about
 113 individual systematic reviews can be found at www.NEL.gov, including the search strategy,
 114 inclusion and exclusion criteria, a complete list of included and excluded articles, and a detailed
 115 write-up describing the included studies and the body of evidence. Specific information about the
 116 use of existing systematic reviews, including the search strategy, inclusion and exclusion criteria,
 117 and a detailed write-up describing the included studies and the body of evidence can be found at
 118 www.DietaryGuidelines.gov. A link for each question is provided following each evidence
 119 review.

121 **FOOD ACCESS**

122 Understanding how access to nutritious and affordable food at various retail establishments--
 123 from convenience stores, to farmers markets, to large box stores--support individuals in their
 124 consumption of a high quality diet and ability to achieve a healthy body weight was the focus of
 125 the food access questions. Because the two food access questions are complementary, the DGAC
 126 choose to develop only one implication statement for both questions.

128 **Question 1: What is the relationship between neighborhood and community 129 access to food retail settings and individuals' dietary intake and quality?**

130 **Source of evidence:** NEL systematic review

131 **Conclusion**

132 Emerging evidence suggests that the relationship between access to farmers' markets/produce
 133 stands and dietary intake and quality is favorable. The body of evidence regarding access to other
 134 food outlets, such as supermarkets, grocery stores, and convenience/corner stores, and dietary
 135 intake and quality is limited and inconsistent. **DGAC Grade: Grade not assignable**

136

137 **Review of the Evidence**

138 This systematic review included 18 studies published between 2007 and 2013, including 15
 139 cross-sectional studies,¹⁻¹⁵ by independent investigators with sufficient sample sizes, 1
 140 longitudinal study¹⁶ and 2 controlled trials^{17, 18} (one RCT and one non-randomized) examining
 141 the relationship between food access and dietary intake and/or quality.

142

143 The studies used multiple approaches to assess food access and dietary intake, quality, and
 144 variety. The majority of studies measured food access by the density of food outlets within a
 145 specified distance from a participant's residence and/or proximity to various food outlets. The
 146 majority of studies assessed dietary intake by focusing on vegetable and fruit consumption; diet
 147 quality and variety were predominantly determined by various validated diet indices including,
 148 but not limited to, the Healthy Eating Index (HEI).

149

150 Although food access was assessed across wide-ranging geographic, ethnic, racial, and income
 151 groups, due to the wide variation in methods used to determine food access, making comparisons
 152 across studies was challenging. Despite this variability, a consistent relationship was identified
 153 between farmers' markets/produce stands and dietary intake.^{6, 15} Two cross-sectional studies
 154 found statistically significant, favorable associations between access to farmers' markets/produce
 155 stands and dietary intake (assessed by individual vegetable and fruit consumption) and diet
 156 variety and quality (both assessed by the HEI). Due to the variability of studies and paucity of
 157 data, no consistent associations regarding dietary outcomes and access to other food outlets were
 158 evident.

159

160 *For additional details on this body of evidence, visit:*
 161 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250425

162

163 **Question 2: What is the relationship between neighborhood and community** 164 **access to food retail settings and weight status?**

165 **Source of evidence: NEL systematic review**

166 **Conclusion**

167 Limited but consistent evidence suggests that the relationship between access to convenience
 168 stores and weight status is unfavorable, with closer proximity and greater access being associated
 169 with significantly higher body mass index (BMI) and/or increased odds of overweight or obesity.

170 **DGAC Grade: Limited**

171

172 The body of evidence on access to other food outlets, such as supermarkets, grocery stores, and
 173 farmers' markets/produce stands, and weight status is limited and inconsistent. **DGAC Grade:**
 174 **Grade not assignable**

175

176 **Review of the Evidence**

177 This systematic review included 26 studies published between 2005 and 2013, including 19
 178 cross-sectional studies^{1, 6, 8, 14, 19-33} and 7 longitudinal studies³⁴⁻⁴⁰ examining the relationship
 179 between food access and weight status.

180

181 The studies used multiple approaches to assess food access and measures of weight status. The
 182 majority of studies measured food access by the density of food outlets within a specified
 183 distance from a participant's residence and/or proximity to various food outlets. The primary
 184 weight status outcome was BMI, which was derived from height and weight.

185

186 Due to the wide variation in methods used to determine food access, making comparison across
 187 studies was challenging. Despite this variability, the relationship between convenience stores and
 188 weight status was consistent across the evidence. Seven studies^{19, 23, 24, 26-28, 37} (six cross-sectional
 189 and one longitudinal) found statistically significant associations between access to convenience
 190 stores and BMI and/or increased odds of overweight or obesity. Five of these studies were
 191 completed in an adult sample; two assessed this relationship among children. Due to the
 192 variability of studies and paucity of data, no consistent associations regarding weight status and
 193 access to other food outlets were evident.

194

195 The evidence base included several studies of weaker design, mostly cross-sectional, by
 196 independent investigators with sufficient sample sizes. The findings across studies were
 197 inconsistent for all food outlet types, except for convenience stores, which were evaluated in
 198 only seven studies. Although food access was assessed across geographic, ethnic, racial and
 199 income groups, the variability in methodology made it difficult to compare studies.

200

201 *For additional details on this body of evidence, visit:*
 202 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250459

203

204 **Implications for the Food Access Topic Area**

205 For people to improve their diets and health, they need to have access to high quality and
 206 affordable healthy foods in environments where they live, work, learn, and/or play across the
 207 lifespan. Limited access to affordable and healthy food is a challenge, particularly for families
 208 living in rural areas and low-income communities. Innovative approaches to bring healthy food
 209 retail options into communities have proliferated, especially in underserved areas. These include
 210 creating financing programs to incentivize grocery store development; improving availability of

211 healthy food at corner stores and bodegas, farmers markets and mobile markets, shelters, food
 212 banks, community gardens/cooperatives, and youth-focused gardens; and creating new forms of
 213 wholesale distribution through food hubs. However, most of these approaches lack adequate
 214 evaluation. These and other promising equity-oriented efforts need to continue and be evaluated
 215 and then successfully scaled up to other communities.

216

217 To ensure healthy food access to everyone in the United States, action is needed across all
 218 levels—Federal, state, and local—to create private-public partnerships and business models, with
 219 the highest priority on those places with greatest need. Similar efforts are needed to reduce
 220 access to, and consumption of, calorie-dense, nutrient-poor foods and sugar-sweetened beverages
 221 in community settings. These efforts need to be seamlessly integrated with food assistance
 222 programs, such as food banks, soup kitchens, and Federal nutrition assistance programs, such as
 223 the Special Supplemental Program for Women, Infants and Children (WIC) and the
 224 Supplemental Nutrition Assistance Program (SNAP) and elder nutrition.

225

226

227 **EARLY CARE AND EDUCATION**

228 About one in five preschool children are overweight or obese,⁴¹ and growing evidence indicates
 229 that preschoolers who are overweight or obese experience negative physical consequences,
 230 including cardio-metabolic abnormalities,⁴² making evident the need for effective efforts to
 231 prevent excessive weight gain for this age group.

232

233 **Question 3: What is the impact of obesity prevention approaches in early care** 234 **and education programs on the weight status of children ages 2 to 5 years?**

235 **Source of evidence:** Existing systematic review with a NEL systematic review update

236

237 **Conclusion**

238 Moderate evidence suggests that multi-component obesity prevention approaches implemented
 239 in child care settings improve weight-related outcomes in preschoolers. A combination of dietary
 240 and physical activity interventions is effective for preventing or slowing excess weight gain and
 241 reducing the proportion of young children ages 2 to 5 years who become overweight or obese.

242 **DGAC Grade: Moderate**

243

244 **Implications**

245 Existing evidence indicates that multi-component interventions that incorporate both nutrition
 246 and physical activity are effective in reducing excessive weight gain in preschool children.
 247 Successful strategies include: curricular enhancements of classroom education for children on

248 both nutrition education and physical activity, outreach engagement to parents about making
249 positive changes in the home, improvements in the nutrition quality of meals and snacks served
250 in the child care program, modifying food service practices, improving the mealtime
251 environment, increasing physical activity play, reducing sedentary behaviors, and improving
252 outdoor playground environments. Evidenced-based healthy eating and physical activity
253 practices should be implemented in child care settings with training and technical assistance for
254 staff. At the Federal, state, and local levels, policies are needed that create strong nutrition and
255 physical activity standards and guidelines in child care settings. There is a need to strengthen
256 policies at the Federal, state, and local levels for strong nutrition and physical activity standards
257 and guidelines in child care settings.

258
259 It is important that child care facilities provide meals and snacks that are consistent with the meal
260 patterns in the Federal Child and Adult Care Food Program (CACFP)⁴³ to ensure that young
261 children have access to healthy meals and snacks and age-appropriate portions. Drinking water
262 also needs to be readily available and accessible to children. Government agencies should ensure
263 access to affordable, nutritious foods through CACFP and maximize participation in the
264 program.

265 266 **Review of the Evidence**

267 This evidence portfolio included one existing systematic review from Zhou et al.⁴⁴ and a de novo
268 NEL systematic review updating the evidence base. The Zhou et al. review included 15
269 controlled trials published between 2000 and 2012; the NEL review included seven studies⁴⁵⁻⁵²
270 (eight publications) published between 2012 and 2014. Both reviews examined the impact of
271 obesity prevention approaches on the weight status of children ages 2 to 5 years.

272
273 The studies used a variety of intervention strategies targeting behaviors that affect body weight.
274 Most approaches were multi-component, with a combination of interventions targeting children,
275 their parents, and/or staff of early care and education programs. The primary weight status
276 outcomes of interest were BMI and BMI z-score.

277
278 The body of available evidence describes a large variation in excessive weight gain prevention
279 approaches, making comparison across studies challenging. Despite this variability, multi-
280 component interventions were effective in reducing BMI and preventing excess weight gain.
281 Seven of 10 multi-component studies included in the Zhou et al. review demonstrated
282 improvements in weight-related outcomes. Six of the seven interventions included in the NEL
283 review demonstrated that multi-component interventions effectively reduce BMI or prevent
284 excess weight gain in children ages 2 to 5 years.

285
286 The evidence base included several studies of strong design by independent investigators,
287 specifically controlled trials, with sufficient sample sizes. Some inconsistency was evident across

288 studies and may be explained by differences in the populations sampled, outcome measures,
 289 duration or exposure of intervention, and follow-up periods. Although the majority of the studies
 290 included in the evidence portfolio effectively reduced BMI or prevented excess weight gain, the
 291 magnitude of the effect as well as the clinical and public health significance was difficult to
 292 assess because of the differences in measures and methodology.

293

294 *For additional details on this body of evidence, visit:* <http://NEL.gov/topic.cfm?cat=3355>

295

296 **SCHOOLS**

297 There are 49.6 million children aged 6-17 years in the United States, and the vast majority are
 298 educated in public or private school settings. School-based programs and policies at the local,
 299 state, and federal levels are cornerstones of food accessibility, availability, and consumption at
 300 schools, which underscore why this setting is a major determinant of nutritional intake and
 301 growth, development, and health of school-aged children. Because the schools questions are
 302 complementary, the DGAC choose to develop only one implication statement for the four
 303 questions.

304

305 **Question 4: What is the impact of school-based approaches on the dietary intake,**
 306 **quality, behaviors, and/or preferences of school-aged children?**

307 **Source of evidence:** Existing systematic reviews

308 **Conclusion**

309 Moderate evidence indicates that multi-component school-based approaches can increase daily
 310 vegetable and fruit consumption in children in grades kindergarten through 8th. Sufficient school-
 311 based studies have not been conducted with youth in grades 9 to 12. Vegetable and fruit
 312 consumption individually, as well as in combination, can be targeted with specific school-based
 313 approaches. **DGAC Grade: Moderate**

314

315 **Review of the Evidence**

316 This evidence portfolio included three systematic reviews,⁵³⁻⁵⁵ two of which included meta-
 317 analyses,^{53, 55} which collectively evaluated 75 studies published between 1985 and 2011. Forty-
 318 nine studies were conducted in the United States and the remaining studies were completed in
 319 other highly developed countries. The systematic reviews examined the impact of school-based
 320 approaches targeting the dietary intake, quality, behaviors and/or preferences of school-aged
 321 children.

322

323 The studies used a variety of intervention strategies. Some approaches were multi-component,
 324 with a combination of interventions targeting children, their parents, and/or the school
 325 environment. The primary dietary outcome of interest was vegetable and fruit intake.
 326

327 In the body of available evidence, the school-based approaches were diverse, making comparison
 328 across studies challenging. Despite this variability, multi-component interventions, and in
 329 particular those that engaged both children and their families, were more effective than single-
 330 component interventions for eliciting significant dietary improvements. Broadly, school-based
 331 intervention programs moderately increased total daily vegetable and fruit intakes and fruit (with
 332 and without fruit juice) intake alone. Furthermore, results showed that school-based economic
 333 incentive programs can effectively increase vegetable and fruit consumption and reduce
 334 consumption of low-nutrient-dense foods while children are at school. Nutrition education
 335 programs that include gardening effectively increased the consumption of vegetables in school-
 336 aged children, along with small, but significant increases in fruit intake.
 337

338 The evidence base included three reviews evaluating several studies by independent investigators
 339 with sufficient sample sizes. Some inconsistency was evident across studies and may be
 340 explained by differences in the populations sampled, outcome measures, duration or exposure of
 341 intervention and follow-up periods. Although findings indicated that school-based approaches
 342 effectively increased the combined intake of vegetable and fruit, the magnitude of the effect as
 343 well as the public health significance was difficult to assess because of differences in measures
 344 and methodology.
 345

346 *For additional details on this body of evidence, visit: Appendix E-2.29a and Appendix E-2.29b*
 347

348 **Question 5: What is the impact of school-based policies on the dietary intake,
 349 quality, behaviors, and/or preferences of school-aged children?**

350 **Source of evidence:** Existing systematic reviews

351 **Conclusion**

352 Strong evidence demonstrates that implementing school policies for nutrition standards to
 353 improve the availability, accessibility, and consumption of healthy foods and beverages sold
 354 outside the school meal programs (competitive foods and beverages) and (or) reducing or
 355 eliminating unhealthy foods and beverages are associated with improved purchasing behavior
 356 and result in higher quality dietary intake by children while at school. **DGAC Grade: Strong**
 357

358 Review of the Evidence

359 This evidence portfolio includes two systematic reviews,^{54, 56} which collectively evaluated 52
360 studies published between 1990 and 2013. Forty-one studies were conducted in the United States
361 and the remaining studies were conducted in other highly-developed countries. The systematic
362 reviews examined the impact of school policies, at the state and district levels, on dietary intake
363 and behaviors.

364

365 The studies included a variety of policies, including economic incentives and both state and
366 school-district policies, targeting behaviors related to dietary intake. The primary outcomes of
367 interest were vegetable and fruit intakes and availability, purchasing, and consumption of
368 competitive foods and beverages (CF&B).

369

370 In the body of available evidence, school policies were diverse, making comparison across
371 studies challenging. Despite this variability, school-based policies targeting the availability of
372 foods and beverages can positively influence the behaviors related to nutrition among children
373 while they are at school. School-based economic incentive programs can effectively increase
374 vegetable and fruit consumption and reduce consumption of low-nutrient-dense foods while
375 children are at school. The implementation of school policies to change the availability and
376 accessibility of healthier foods and beverages versus unhealthy CF&B is associated with the
377 expected changes in consumption within the school setting. In addition, strong and consistent
378 enforcement of more comprehensive policies to change the availability of healthier foods and
379 beverages versus unhealthy CF&B at schools is associated with desired changes in consumption
380 and purchasing within the school setting. Also, policies restricting the use of food as a reward for
381 academic performance or as part of a fundraiser were associated with a reduction in using foods
382 and beverages for these purposes.

383

384 The evidence base included two reviews evaluating several studies by independent investigators
385 with sufficient sample sizes. Although findings indicated that school policies can effectively
386 increase the combined intake of vegetables and fruits and/or decrease the availability,
387 purchasing, and consumption of unhealthy CF&B, the magnitude of the effect as well as the
388 public health significance is difficult to ascertain.

389

390 *For additional details on this body of evidence, visit: Appendix E-2.30 and Appendix E-2.29b*

391

392 **Question 6: What is the impact of school-based approaches on the weight status**
393 **of school-aged children?**

394 **Source of evidence:** Existing systematic reviews

395 **Conclusion**

396 Moderate and generally consistent evidence indicates that multi-component school-based
 397 approaches have beneficial effects on weight status (BMI or BMI-z reduced on average by 0.15
 398 kg/m²), especially for children ages 6 to 12 years. **DGAC Grade: Moderate**

399
 400 The body of evidence regarding the impact of school-based approaches on weight status among
 401 adolescents is limited due to an insufficient number of studies. **DGAC Grade: Not Assignable**
 402

403 **Review of the Evidence**

404 This evidence portfolio included two systematic reviews;^{57, 58} one of which included a meta-
 405 analysis.⁵⁷ Collectively, 108 studies targeting children in school published before August 2012
 406 were evaluated. Forty-nine studies were conducted in the United States and the remaining studies
 407 were completed in other highly developed countries. The systematic reviews examined the
 408 impact of school-based approaches targeting obesity prevention among school-aged children.
 409

410 The studies used a variety of intervention strategies targeting behaviors related to dietary intake
 411 and/or physical activity. Some approaches were multi-component, with a combination of
 412 interventions targeting children, their parents, and/or the school environment. The primary
 413 outcomes of interest were BMI, changes in BMI, rate of weight gain, body fat percentage, waist
 414 circumference, skin fold thickness, and prevalence of overweight and obesity.
 415

416 In the body of available evidence, the school-based approaches were diverse, making comparison
 417 across studies challenging. Despite this variability, school-based interventions significantly
 418 improved weight-related outcomes. Multi-component interventions, and in particular those
 419 implemented longer term (more than 6 months), were more effective than single-component and
 420 short-term (3 to 6 months) interventions. Evidence supporting the effectiveness of school-based
 421 interventions among children ages 6 to 12 years was robust, while findings among adolescents
 422 ages 13 to 18 years were weaker, but trended toward effectiveness.
 423

424 The evidence base included two reviews evaluating several studies by independent investigators
 425 with sufficient sample sizes. Although findings indicated that school-based approaches
 426 effectively improve weight-related outcomes, in particular among children between the ages of 6
 427 and 12 years, a high degree of heterogeneity means these findings should be interpreted
 428 cautiously. Although the magnitude of the effect was clinically meaningful, the public health
 429 significance was difficult to ascertain.
 430

431 *For additional details on this body of evidence, visit: Appendix E-2.31 and Appendix 2.29b*
 432

433

434 **Question 7: What is the impact of school-based policies on the weight status of**
 435 **school-aged children?**

436 **Source of evidence:** Existing systematic reviews

437 **Conclusion**

438 Although moderate evidence indicates that school policies improve dietary intake, limited
 439 evidence suggests that school policies targeting nutrition, alone and in combination with physical
 440 activity, may beneficially affect weight-related outcomes. **DGAC Grade: Limited**

441

442 **Review of the Evidence**

443 This evidence portfolio included two systematic reviews,^{56, 59} which collectively evaluated 45
 444 studies published between 2003 and 2013. Forty studies were conducted in the United States and
 445 the remaining studies were conducted in other highly developed countries. The systematic
 446 reviews examined the impact of school policies, at the state and district levels, on weight-related
 447 outcomes.

448

449 The studies included a variety of policies at the school, school-district, or state level, targeting
 450 behaviors related to dietary intake, alone and in combination with physical activity. The primary
 451 outcome of interest was BMI.

452

453 Limited research exists to systematically review and quantitatively evaluate the effect of school-
 454 based nutrition policies on the weight status of children. In addition, high heterogeneity among
 455 studies warrants caution when drawing conclusions from the results. In the body of available
 456 evidence, the findings related to the impact of school policies targeting nutrition and physical
 457 activity on weight outcomes were mixed. Even so, dietary policies related to the School
 458 Breakfast Program were associated with a lower BMI among students who participated in the
 459 program in comparison to students who did not participate. Overall, school-based, multi-
 460 component interventions including policy elements and policies and laws regarding the
 461 availability and accessibility of CF&B in schools warrant further research as ways to target
 462 childhood obesity.

463

464 The evidence base included two reviews evaluating several studies by independent investigators
 465 with sufficient sample sizes. However, most studies were of weaker design (i.e., cross-sectional)
 466 and findings were inconsistent.

467

468 *For additional details on this body of evidence, visit: Appendix E-2.32 and Appendix E-2.29b*

469

470 **Implications for the Schools Topic Area**

471 Existing evidence indicates that school-based programs designed to improve the food
 472 environment and support healthy behaviors may effectively promote improved dietary intake and
 473 weight status of school-aged children. Programs that emphasize multi-component, multi-
 474 dimensional approaches (including increased physical activity) are important to changing
 475 behavior and need to be reinforced within the home environment, as well as the community,
 476 including neighborhood food retail outlets that surround schools. Policies should strive to
 477 support effective programs that increase availability, accessibility, and consumption of healthy
 478 foods, while reducing less healthy CF&B. The combination of economic incentives along with
 479 specific policies can increase the likelihood that specific approaches will be effective.

480

481 The recently updated USDA nutrition standards for school meals, snacks, and beverages sold in
 482 schools will ensure that students throughout the United States will have healthier school meals
 483 and snack and beverage options, but schools need support and active engagement from students,
 484 parents, teachers, administrators, community members, and their districts and states to
 485 successfully implement and sustain them.

486

487

488 **WORKSITES**

489 Many workplaces are located in areas where food options are limited, which makes the
 490 workplace an important setting for approaches focused on dietary intake and environmental
 491 modifications. Because the worksite questions are complementary, the DGAC choose to develop
 492 only one implication statement for the four questions.

493

494 **Question 8: What is the impact of worksite-based approaches on the dietary**
 495 **intake, quality, behaviors and/or preferences of employees?**

496 **Source of evidence:** Existing systematic reviews

497 **Conclusion**

498 Moderate evidence indicates that multi-component worksite approaches can increase vegetable
 499 and fruit consumption of employees. **DGAC Grade: Moderate**

500

501 **Review of the Evidence**

502

503 This evidence portfolio includes two systematic reviews,^{60, 61} which collectively evaluated 35
 504 studies by independent investigators with sufficient sample sizes published before November
 505 2012. The systematic reviews examined the impact of worksite-based approaches targeting the
 506 dietary intake, quality, behaviors, and/or preferences of employees.

507
 508 The studies used a variety of intervention approaches targeting behaviors related to dietary
 509 intake; some were delivered in-person and others were delivered through the Internet. Some
 510 inconsistencies are evident across studies and may be explained by differences in the populations
 511 sampled and methodologies used, including the types and durations of intervention and follow-
 512 up periods. Some approaches were multi-component, with a combination of interventions
 513 targeting employees and/or the food environment at the worksite. The primary dietary outcome
 514 of interest was vegetable and fruit intake.

515
 516 Among the body of evidence available, multi-component interventions, and in particular those
 517 that incorporated face-to-face contact and nutrition education, were more effective than single-
 518 component interventions for eliciting significant dietary improvements. Overall, worksite-based
 519 intervention programs moderately increase vegetable and fruit intakes, although the magnitude of
 520 the effect is difficult to assess. Nutrition education and internet-based programs appear to be
 521 promising approaches for eliciting desired dietary modifications when incorporated into multi-
 522 component interventions.

523
 524 *For additional details on this body of evidence, visit: Appendix E-2.33a and Appendix E-2.33b*

525
 526 **Question 9: What is the impact of worksite-based policies on the dietary intake,
 527 quality, behaviors and/or preferences of employees?**

528 **Source of evidence:** Existing systematic reviews

529 **Conclusion**

530 Moderate and consistent evidence indicates that worksite nutrition policies, alone and in
 531 combination with environmental changes and/or individual-level nutrition and health
 532 improvement strategies, can improve the dietary intake of employees. Multi-component
 533 interventions appear to be more effective than single-component interventions. **DGAC Grade:**
 534 **Moderate**

535 536 **Review of the Evidence**

537
 538 This evidence portfolio includes one systematic review,⁶² which evaluated 27 studies by
 539 independent investigators with sufficient sample sizes published between 1985 and 2010. The
 540 review examined the evidence for the effectiveness of a variety of worksite health promotion
 541 programs using environmental and/or policy changes either alone or in combination with health
 542 behavior change strategies focused on individual employees.

543

544 Some interventions were multi-component, with a combination of strategies targeting employees
 545 and/or the food environment at the worksite. Strategies included point-of-purchase labeling,
 546 increased availability of healthy food items, and/or educational programs and materials. The
 547 primary dietary outcome of interest was vegetable and fruit intake.

548
 549 In the body of evidence available, the worksite-based policies were diverse, thus it was
 550 challenging to identify the most effective strategies. Despite this variability, multi-component
 551 interventions, and in particular those that targeted individual employees in addition to the
 552 environment, were more effective than single-component interventions for eliciting significant
 553 dietary improvements. Overall, worksite interventions moderately increased vegetable and fruit
 554 intakes.

555
 556 Some inconsistency was evident across studies assessed for the systematic review in regards to
 557 scientific rigor and impact. The inconsistencies may be explained by differences in the
 558 populations sampled and methodologies used, including duration, exposure of the intervention,
 559 and follow-up periods. Although findings indicate that worksite policies increase consumption of
 560 vegetables and fruit, the magnitude of the effect was difficult to assess.

561
 562 *For additional details on this body of evidence, visit: Appendix E-2.34 and Appendix E-2.33b*

563

564 **Question 10: What is the impact of worksite-based approaches on the weight**
 565 **status of employees?**

566 **Source of evidence:** Existing systematic reviews

567 **Conclusion**

568 Moderate and consistent evidence indicates that multi-component worksite approaches targeting
 569 physical activity and dietary behaviors favorably affect weight-related outcomes. **DGAC Grade:**
 570 **Moderate**

571

572 **Review of the Evidence**

573

574 This evidence portfolio includes two systematic reviews,^{61, 63} one of which included meta-
 575 analyses.⁶³ The systematic reviews examined the impact of worksite-based approaches on the
 576 weight status of employees. Collectively, 70 studies published before November 2012 were
 577 evaluated.

578

579 The studies used a variety of intervention strategies targeting behaviors related to weight status;
 580 some were delivered in-person and others were delivered through the Internet. The primary
 581 outcomes of interest were body weight, BMI, and body fat percentage.

582

583 In the body of evidence available, multi-component interventions, and in particular those that
 584 incorporated face-to-face contact and targeted behaviors related to diet and physical activity,
 585 were more effective than single-component interventions for eliciting significant improvements
 586 in weight-related outcomes. Overall, worksite-based intervention programs significantly
 587 decreased body weight, BMI, and body fat percentage. Internet-based programs appeared to be
 588 promising approaches for eliciting behavior changes and improving related health outcomes.
 589

590 The evidence base included two reviews evaluating several studies by independent investigators
 591 with sufficient sample sizes. Some inconsistencies were evident across studies and may be
 592 explained by differences in the populations sampled and methodologies, including duration or
 593 exposure of intervention and follow-up periods. Although findings indicated that worksite-based
 594 approaches effectively improve the weight status of employees, the magnitude of the effect was
 595 difficult to assess.

596

597 *For additional details on this body of evidence, visit: Appendix E-2.35 and Appendix E-2.33b*

598

599 **Question 11: What is the impact of worksite-based policies on the weight status**
 600 **of employees?**

601 **Source of evidence:** Existing systematic reviews

602 **Conclusion**

603 The body of evidence assessing the impact of worksite policies on the weight status of
 604 employees is very limited. **DGAC Grade: Not Assignable**

605

606 **Review of the Evidence**

607

608 This evidence portfolio included one systematic review,⁶² which evaluated 27 studies published
 609 between 1985 and 2010. The review examined the evidence for the effectiveness of worksite
 610 health promotion programs using environmental and/or policy changes either alone or in
 611 combination with individually-focused health behavior change strategies.

612

613 The studies used a variety of policies targeting behaviors that can influence weight status. Some
 614 studies assessed the impact of policies (e.g., catering policies and company policies rewarding
 615 employees for healthy behaviors) combined with individual-level strategies. Some interventions
 616 were multi-component, with a combination of strategies targeting employees (e.g., point-of-
 617 choice messaging including nutrition information in cafeterias and reminders to use stairs) and/or
 618 the food environment at the worksite (e.g., increased availability of healthy food options). The
 619 health outcomes of interest included BMI, blood pressure, and cholesterol.

620

621 In the body of evidence available, worksite policies either alone or in combination with
 622 individually-focused health behavior change strategies did not affect the weight status of
 623 employees. However, interventions incorporating both environmental and individual strategies
 624 can lead to significant improvement in behaviors related to weight status (e.g., dietary intake).
 625 The lack of impact may be due to length of exposure or the duration of the follow-up period.

626

627 The evidence base included one review evaluating several studies by independent investigators
 628 with sufficient sample sizes. The studies were inconsistent in their scientific rigor. Due to the
 629 variability of studies and paucity of data, no consistent associations regarding worksite policies
 630 and the weight status of employees were evident.

631

632 *For additional details on this body of evidence, visit: Appendix E-2.36 and Appendix E-2.33b*

633

634 **Implications for the Worksite Topic Area**

635 Existing evidence indicates that worksite approaches focused on dietary intake can increase fruit
 636 and vegetable intakes of employees. Multi-component programs targeting nutrition education in
 637 combination with dietary modification interventions are found to be effective. Additionally,
 638 environmental modifications in conjunction with a variety of worksite policies targeting dietary
 639 modification, including point-of-purchase information, catering policies, and menu labeling are
 640 effective. Thus, these evidence-based strategies should be implemented in worksites through a
 641 variety of means, such as corporate wellness programs, food service policies, and health benefits
 642 programs. Programs should emphasize multi-component approaches targeting diet and physical
 643 activity while policies should support behavior changes associated with improving health
 644 outcomes such as increasing the availability of healthy foods within the workplace and
 645 encouraging more physical activity throughout the workday. Given that approximately 64
 646 percent of adults are employed and spend an average of 34 hours per week at work, the
 647 workplace remains an important setting for environmental and behavioral interventions for
 648 health promotion and disease prevention.

649

650

651

652 **CHAPTER SUMMARY**

653 Environmental and policy approaches are needed to complement individual-based efforts to
 654 improve diet quality and reduce obesity and other diet-related chronic diseases. These
 655 approaches have the potential for broad and sustained impact at the population level. The DGAC
 656 focused on physical environments (settings) in which foods are available. Our aim was to better
 657 understand the impact of the food environment to promote or hinder diet quality healthy eating in
 658 these settings and to identify the most effective evidence-based diet-related approaches and
 659 policies to improve diet quality and weight status. The DGAC systematically reviewed and

660 graded the scientific evidence in these four settings, community food access, child care, schools
661 and worksites, and their relationships to dietary quality and weight status.

662

663 The DGAC found moderate and promising evidence that multi-component obesity prevention
664 approaches implemented in child care settings, schools, and worksites improve weight-related
665 outcomes; strong to moderate evidence that school and worksite policies are associated with
666 improved dietary intake; and moderate evidence that multi-component school-based and
667 worksite approaches increase vegetable and fruit consumption. For the community food access
668 questions addressing the relationship between food retail settings and dietary intake/quality and
669 weight status the evidence was too limited or insufficient to assign grades. To reduce the
670 disparity gaps that currently exist in low resource and underserved communities, more solution-
671 oriented strategies need to be implemented and evaluated on ways to increase access to and
672 procurement of healthy affordable foods, and also to reduce access to energy-dense, nutrient-
673 poor foods.^{64, 65} Although several innovative approaches are taking place now throughout the
674 country, they generally lack adequate evaluation efforts.

675

676 One striking aspect of the Committee's findings was the power of multi-component interventions
677 over single component interventions. For obesity prevention, effective multi-component
678 interventions incorporated both nutrition and physical activity using a variety of strategies such
679 as environmental policies to improve the availability and provision of healthy foods; increasing
680 opportunities for physical activity, increased parent engagement; and educational approaches,
681 such as a school nutrition curriculum. For multi-component dietary interventions (e.g., to
682 increase consumption of vegetables and fruits) the most effective strategies included nutrition
683 education, parent engagement, and environmental modifications (e.g., policies for nutrition
684 standards, food service changes, point of purchase information).

685

686 The evidence reviewed in this chapter will inform and guide new multi-component individual
687 and environmental and policy approaches in settings where people eat and procure their food to
688 successfully target improvements in dietary intake and weight status. Collaborative partnerships
689 and strategic efforts are needed to translate this evidence to action. Further work on restructuring
690 the environment to facilitate healthy eating and physical activity, especially in high risk
691 populations, is needed to advance evidence-based solutions that can be scaled up.

692

693 **NEEDS FOR FUTURE RESEARCH**

- 694 1. Develop more valid and reliable methods for measuring all aspects of the food environment,
695 including the total food environment of communities. These methods can then be used to
696 assess the impact of the food environment on community health as well as on economic
697 development and growth.

698 **Rationale:** The food environment has become more complex, with more and more retail
 699 outlets selling food and beverages. Having valid and reliable methodologies for a variety of
 700 food environments and settings (tools and new analytical approaches) will allow more
 701 meaningful inquiry into the contributions of various settings in supporting or hindering
 702 nutritional health.

703

704 2. Identify, implement, evaluate, and scale up best practices (including private-public
 705 partnerships) for affordable and sustainable solutions to improving the food environment and
 706 increasing food access, especially in those environments of greatest need.

707 **Rationale:** The environments in which people live, work, learn, and play greatly influence
 708 their food intake. To best guide efforts to improve the food environment, research is needed
 709 to identify and evaluate best practices to direct available resources to new programs and scale
 710 up.

711

712 3. Identify, implement, accelerate, evaluate, and scale up programs that improve access to
 713 healthy food and that can be integrated seamlessly with Federal nutrition assistance
 714 programs, such as SNAP, WIC and elder nutrition.

715 **Rationale:** Federal nutrition assistance programs reach individuals and populations with the
 716 greatest health disparities. Identifying and evaluating initiatives that integrate improvements
 717 in the food environment with Federal programs will help ensure that Federal nutrition
 718 assistance programs have as great an impact as possible.

719

720 4. Conduct additional obesity prevention intervention research in child care settings (e.g., child-
 721 care centers, family child-care homes) to: 1) Identify the most potent components of the
 722 interventions and the optimal combinations for improving diet quality, physical activity, and
 723 weight outcomes; 2) Assess implementation and translation costs and benefits of the
 724 intervention, including impact, cost-effectiveness, generalizability and reach, sustainability
 725 and feasibility; 3) Develop and evaluate culturally appropriate and tailored interventions for
 726 preschool children in low-income and racial/ethnic communities, given the disproportionate
 727 impact of obesity in these groups; 4) Explore intervention strategies on how to use child care
 728 settings as access points to create linkages to parents, caretakers, and health care providers as
 729 partners in health promotion; 5) Evaluate the impact of Federal, state, and local policies,
 730 regulations, and support (e.g., provider training and technical assistance) for child care
 731 programs on the eating and physical activity practices and behaviors, and weight status of
 732 young children.

733 **Rationale:** Early care and education settings are an important venue for interventions
 734 targeting young children. A strong evidence base is essential to identify and support
 735 evidence-based practices and policies that can be implemented at Federal, state, and local
 736 levels and to mobilize efforts to improve healthy eating and physical activity, leading to

737 healthy weight development in these settings. Interventions found to effectively reduce risk
 738 of obesity in one setting need to be appropriately adapted for diverse groups and different
 739 settings.

740

741 5. Improve intervention research methods by the use of stronger study designs and the
 742 development of standardized assessments of body composition, weight status. Develop
 743 enhanced validated measures of diet quality, feeding and physical activity practices, and
 744 physical activity and eating behaviors and policies. Create standardized measures to assess
 745 the nutrition quality of meals and snacks in child care settings, as well as the food and
 746 physical activity environments. Create standardized methods for assessing the relationship of
 747 child care food, nutrition and physical activity-related measures to similar measures
 748 representing non-child care time are needed to provide greater consistency in determining the
 749 contributors to the development and progression of childhood overweight and obesity.

750 **Rationale:** Although many of the studies included in these evidence reviews were
 751 methodologically strong and were controlled studies, some were limited by small sample
 752 size, lack of adequate control for confounding factors, and different outcome measures and
 753 different tools used to measure the outcome variables.

754

755 6. Examine the effect of the recommended Child and Adult Care Food Program (CACFP)
 756 through ongoing periodic evaluations and fill gaps in the knowledge regarding participation,
 757 demand, food procurement and practices, nutrient intake, and food security.

758 **Rationale:** Improvements in school meals and the school food environment have been
 759 fostered by national data from periodic studies such as the USDA/FNS School Nutrition
 760 Dietary Assessment Studies (SNDA), the HHS/CDC School Health Policies and Practices
 761 Studies (SHPPS) and the HHS/NIH C.L.A.S.S. In contrast, considerably fewer periodic
 762 national studies are conducted of meals and dietary intake in child care settings and their
 763 relation to the child care food and physical activity environment.

764

765 7. Conduct new research to document the types and quantities of foods and beverages students
 766 consume both at school and daily outside of school, before, during, and after school-based
 767 healthy eating approaches and policies are implemented.

768 **Rationale:** Effective school-based approaches and policies to improve the availability,
 769 accessibility, and consumption of healthy foods and beverages, and reduce competition from
 770 unhealthy offerings, are central to improving the weight status and health of children and
 771 adolescents. Accurate quantification of the types and quantities of foods and beverages the
 772 students consume before, during, and after approaches and policies are implemented is
 773 fundamental to assessing effectiveness. However, many of the studies included in the
 774 systematic reviews and meta-analyses used by the DGAC to address this issue did not
 775 comprehensively measure or report dietary information. Although the USDA/FNS-sponsored

776 School Nutrition Dietary Assessment (SNDA) series collects student dietary intake data
 777 every 10 years, the DGAC recommends more frequent and consistent data collection,
 778 especially before and periodically after implementation of school-based nutrition and
 779 physical activity policy and program changes.

780

781 8. Improve the quality of research studies designed to assess the effects of school-based
 782 approaches and policies on dietary behaviors and body weight control to reduce the risk of
 783 bias, with an emphasis on randomized controlled trials.

784 **Rationale:** Although the methodological quality of the systematic reviews and meta-analyses
 785 used by the DGAC to evaluate school-based approaches and policies on dietary intake and
 786 body weight outcomes was high, the authors of these reviews commented that the scientific
 787 quality of individual studies was generally poor and the risk of bias high. Many of the studies
 788 were done using quasi-experimental (with or without control), pre-post intervention, or cross-
 789 sectional designs. Future research should prioritize using prospective, repeated measures,
 790 randomized controlled trial experimental designs, with randomization at the individual,
 791 classroom, school, or school district level. Pilot feasibility studies also may be helpful to
 792 quickly identify promising novel approaches to improve dietary intake and weight control
 793 outcomes.

794

795 9. Conduct post-program follow-up assessments lasting longer than 1 year to determine the
 796 long-term retention of the changed nutrition behaviors as well as the usefulness of continuing
 797 to offer the programs while children advance in school grade. Also, conduct research is
 798 needed in adolescents (grades 9-12).

799 **Rationale:** Literature supports that eating and physical activity behaviors and body weight
 800 status of children predict changes over time as they progress into adolescence and adulthood.
 801 Ideally, improvements in dietary intake and weight status achieved due to a given school-
 802 based approach or policy would be sustained over time and progressive improvements would
 803 occur long-term. The vast majority of published research focuses on children in grades K-8,
 804 or ages 4-12 years, and new and improved data are needed on adolescents and the transition
 805 from childhood to adolescence.

806

807 10. Encourage a wider variety of school-based approaches and policies to develop and evaluate
 808 innovative approaches focused on increasing vegetable intakes.

809 **Rationale:** Consumption of non-potato vegetables is below 2010 Dietary Guidelines for
 810 Americans recommendations in both children and adolescents. Published research indicates
 811 that school-based approaches and policies designed to increase vegetable and fruit intakes are
 812 generally more effective at increasing fruit intake, except for –school gardens and economic
 813 incentives, which increase vegetable intake among school-aged children. Some past public
 814 policies (e.g. the Basic 4) treated fruit and vegetables and as a single food group, which props

815 the need for new research that uses prospective, repeated measures, and randomized
 816 controlled trial experimental designs to specifically target increased consumption of healthy
 817 vegetables.

818

819 11. Conduct assessments of the effectiveness of worksite interventions that emphasize obesity
 820 prevention and weight control among workers across racially/ethnically diverse populations,
 821 blue and white collar employees, and at-risk populations. Scientifically rigorous studies
 822 (especially randomized controlled trials) addressing the long-term health impact of worksite-
 823 based approaches and policies that improve employee diet, physical activity, and body
 824 weight control would have public health relevance.

825 **Rationale:** In light of the high rates of obesity and overweight, worksite interventions
 826 targeting obesity prevention and weight control through enhanced dietary behaviors and
 827 increased physical activity among workers is important. The majority of the studies to date
 828 have been conducted for relatively short periods of time, and the long-term impact of these
 829 approaches and policies may prove beneficial.

830

831 REFERENCES

- 832 1. Carroll-Scott A, Gilstad-Hayden K, Rosenthal L, Peters SM, McCaslin C, Joyce R, et al.
 833 Disentangling neighborhood contextual associations with child body mass index, diet,
 834 and physical activity: the role of built, socioeconomic, and social environments. *Soc Sci*
 835 *Med.* 2013;95:106-14. PMID: 23642646.
 836 <http://www.ncbi.nlm.nih.gov/pubmed/23642646>.
- 837 2. Caspi CE, Kawachi I, Subramanian SV, Adamkiewicz G, Sorensen G. The relationship
 838 between diet and perceived and objective access to supermarkets among low-income
 839 housing residents. *Soc Sci Med.* 2012;75(7):1254-62. PMID: 22727742.
 840 <http://www.ncbi.nlm.nih.gov/pubmed/22727742>.
- 841 3. Dean WR, Sharkey JR. Rural and urban differences in the associations between
 842 characteristics of the community food environment and fruit and vegetable intake. *J Nutr*
 843 *Educ Behav.* 2011;43(6):426-33. PMID: 21616721.
 844 <http://www.ncbi.nlm.nih.gov/pubmed/21616721>.
- 845 4. Ding D, Sallis JF, Norman GJ, Saelens BE, Harris SK, Kerr J, et al. Community food
 846 environment, home food environment, and fruit and vegetable intake of children and
 847 adolescents. *J Nutr Educ Behav.* 2012;44(6):634-8. PMID: 21531177.
 848 <http://www.ncbi.nlm.nih.gov/pubmed/21531177>.
- 849 5. Jago R, Baranowski T, Baranowski JC, Cullen KW, Thompson D. Distance to food stores
 850 & adolescent male fruit and vegetable consumption: mediation effects. *Int J Behav Nutr*
 851 *Phys Act.* 2007;4:35. PMID: 17850673. <http://www.ncbi.nlm.nih.gov/pubmed/17850673>.

- 852 6. Jilcott Pitts SB, Wu Q, McGuirt JT, Crawford TW, Keyserling TC, Ammerman AS.
853 Associations between access to farmers' markets and supermarkets, shopping patterns,
854 fruit and vegetable consumption and health indicators among women of reproductive age
855 in eastern North Carolina, U.S.A. *Public Health Nutr.* 2013;16(11):1944-52. PMID:
856 23701901. <http://www.ncbi.nlm.nih.gov/pubmed/23701901>.
- 857 7. Sharkey JR, Johnson CM, Dean WR. Food access and perceptions of the community and
858 household food environment as correlates of fruit and vegetable intake among rural
859 seniors. *BMC Geriatr.* 2010;10:32. PMID: 20525208.
860 <http://www.ncbi.nlm.nih.gov/pubmed/20525208>.
- 861 8. An R, Sturm R. School and residential neighborhood food environment and diet among
862 California youth. *Am J Prev Med.* 2012;42(2):129-35. PMID: 22261208.
863 <http://www.ncbi.nlm.nih.gov/pubmed/22261208>.
- 864 9. Jack D, Neckerman K, Schwartz-Soicher O, Lovasi GS, Quinn J, Richards C, et al.
865 Socio-economic status, neighbourhood food environments and consumption of fruits and
866 vegetables in New York City. *Public Health Nutr.* 2013;16:1197-205. PMID: 23388104.
867 <http://www.ncbi.nlm.nih.gov/pubmed/23388104>.
- 868 10. Moore LV, Diez Roux AV, Nettleton JA, Jacobs DR, Jr. Associations of the local food
869 environment with diet quality--a comparison of assessments based on surveys and
870 geographic information systems: the multi-ethnic study of atherosclerosis. *Am J*
871 *Epidemiol.* 2008;167(8):917-24. PMID: 18304960.
872 <http://www.ncbi.nlm.nih.gov/pubmed/18304960>.
- 873 11. Ollberding NJ, Nigg CR, Geller KS, Horwath CC, Motl RW, Dishman RK. Food outlet
874 accessibility and fruit and vegetable consumption. *Am J Health Promot.* 2012;26(6):366-
875 70. PMID: 22747319. <http://www.ncbi.nlm.nih.gov/pubmed/22747319>.
- 876 12. Powell LM, Zhao Z, Wang Y. Food prices and fruit and vegetable consumption among
877 young American adults. *Health Place.* 2009;15(4):1064-70. PMID: 19523869.
878 <http://www.ncbi.nlm.nih.gov/pubmed/19523869>.
- 879 13. Zenk SN, Lachance LL, Schulz AJ, Mentz G, Kannan S, Ridella W. Neighborhood retail
880 food environment and fruit and vegetable intake in a multiethnic urban population. *Am J*
881 *Health Promot.* 2009;23(4):255-64. PMID: 19288847.
882 <http://www.ncbi.nlm.nih.gov/pubmed/19288847>.
- 883 14. Laska MN, Hearst MO, Forsyth A, Pasch KE, Lytle L. Neighbourhood food
884 environments: are they associated with adolescent dietary intake, food purchases and
885 weight status? *Public Health Nutr.* 2010;13(11):1757-63. PMID: 20529405.
886 <http://www.ncbi.nlm.nih.gov/pubmed/20529405>.
- 887 15. Gustafson A, Lewis S, Perkins S, Wilson C, Buckner E, Vail A. Neighbourhood and
888 consumer food environment is associated with dietary intake among Supplemental
889 Nutrition Assistance Program (SNAP) participants in Fayette County, Kentucky. *Public*
890 *Health Nutr.* 2013;16(7):1229-37. PMID: 23462377.
891 <http://www.ncbi.nlm.nih.gov/pubmed/23462377>.

- 892 16. Boone-Heinonen J, Gordon-Larsen P, Kiefe CI, Shikany JM, Lewis CE, Popkin BM. Fast
893 food restaurants and food stores: longitudinal associations with diet in young to middle-
894 aged adults: the CARDIA study. *Arch Intern Med.* 2011;171(13):1162-70. PMID:
895 21747011. <http://www.ncbi.nlm.nih.gov/pubmed/21747011>.
- 896 17. Gittelsohn J, Vijayadeva V, Davison N, Ramirez V, Cheung LW, Murphy S, et al. A food
897 store intervention trial improves caregiver psychosocial factors and children's dietary
898 intake in Hawaii. *Obesity (Silver Spring).* 2010;18 Suppl 1:S84-90. PMID: 20107467.
899 <http://www.ncbi.nlm.nih.gov/pubmed/20107467>.
- 900 18. Gustafson AA, Sharkey J, Samuel-Hodge CD, Jones-Smith JC, Cai J, Ammerman AS.
901 Food Store Environment Modifies Intervention Effect on Fruit and Vegetable Intake
902 among Low-Income Women in North Carolina. *J Nutr Metab.* 2012;2012:932653. PMID:
903 22315676. <http://www.ncbi.nlm.nih.gov/pubmed/22315676>.
- 904 19. Morland K, Diez Roux AV, Wing S. Supermarkets, other food stores, and obesity: the
905 atherosclerosis risk in communities study. *Am J Prev Med.* 2006;30(4):333-9. PMID:
906 16530621. <http://www.ncbi.nlm.nih.gov/pubmed/16530621>.
- 907 20. Drewnowski A, Aggarwal A, Hurvitz PM, Monsivais P, Moudon AV. Obesity and
908 supermarket access: proximity or price? *Am J Public Health.* 2012;102(8):e74-80. PMID:
909 22698052. <http://www.ncbi.nlm.nih.gov/pubmed/22698052>.
- 910 21. Fiechtner L, Block J, Duncan DT, Gillman MW, Gortmaker SL, Melly SJ, et al.
911 Proximity to supermarkets associated with higher body mass index among overweight
912 and obese preschool-age children. *Prev Med.* 2013;56(3-4):218-21. PMID: 23219681.
913 <http://www.ncbi.nlm.nih.gov/pubmed/23219681>.
- 914 22. Liu GC, Wilson JS, Qi R, Ying J. Green neighborhoods, food retail and childhood
915 overweight: differences by population density. *Am J Health Promot.* 2007;21(4
916 Suppl):317-25. PMID: 17465177. <http://www.ncbi.nlm.nih.gov/pubmed/17465177>.
- 917 23. Ahern M, Brown C, Dukas S. A national study of the association between food
918 environments and county-level health outcomes. *J Rural Health.* 2011;27(4):367-79.
919 PMID: 21967380. <http://www.ncbi.nlm.nih.gov/pubmed/21967380>.
- 920 24. Bodor JN, Rice JC, Farley TA, Swalm CM, Rose D. The association between obesity and
921 urban food environments. *J Urban Health.* 2010;87(5):771-81. PMID: 20458548.
922 <http://www.ncbi.nlm.nih.gov/pubmed/20458548>.
- 923 25. Brown AF, Vargas RB, Ang A, Pebley AR. The neighborhood food resource
924 environment and the health of residents with chronic conditions: the food resource
925 environment and the health of residents. *J Gen Intern Med.* 2008;23(8):1137-44. PMID:
926 18483833. <http://www.ncbi.nlm.nih.gov/pubmed/18483833>
- 927 26. Ford PB, Dziewaltowski DA. Limited supermarket availability is not associated with
928 obesity risk among participants in the Kansas WIC Program. *Obesity (Silver Spring).*
929 2010;18(10):1944-51. PMID: 20057363.
930 <http://www.ncbi.nlm.nih.gov/pubmed/20057363>.

- 931 27. Galvez MP, Hong L, Choi E, Liao L, Godbold J, Brenner B. Childhood obesity and
 932 neighborhood food-store availability in an inner-city community. *Acad Pediatr*.
 933 2009;9:339-43. PMID: 19560992. <http://www.ncbi.nlm.nih.gov/pubmed/19560992>.
- 934 28. Morland KB, Evenson KR. Obesity prevalence and the local food environment. *Health*
 935 *Place*. 2009;15(2):491-5. PMID: 19022700.
 936 <http://www.ncbi.nlm.nih.gov/pubmed/19022700>.
- 937 29. Rundle A, Neckerman KM, Freeman L, Lovasi GS, Purciel M, Quinn J, et al.
 938 Neighborhood food environment and walkability predict obesity in New York City.
 939 *Environ Health Perspect*. 2009;117(3):442-7. PMID: 19337520.
 940 <http://www.ncbi.nlm.nih.gov/pubmed/19337520>.
- 941 30. Salois MJ. Obesity and diabetes, the built environment, and the 'local' food economy in
 942 the United States, 2007. *Econ Hum Biol*. 2012;10(1):35-42. PMID: 21561816.
 943 <http://www.ncbi.nlm.nih.gov/pubmed/21561816>.
- 944 31. Stark JH, Neckerman K, Lovasi GS, Konty K, Quinn J, Arno P, et al. Neighbourhood
 945 food environments and body mass index among New York City adults. *J Epidemiol*
 946 *Community Health*. 2013;67(9):736-42. PMID: 23851151.
 947 <http://www.ncbi.nlm.nih.gov/pubmed/23851151>.
- 948 32. Viola D, Arno PS, Maroko AR, Schechter CB, Sohler N, Rundle A, et al. Overweight and
 949 obesity: can we reconcile evidence about supermarkets and fast food retailers for public
 950 health policy? *J Public Health Policy*. 2013;34(3):424-38. PMID: 23719294.
 951 <http://www.ncbi.nlm.nih.gov/pubmed/23719294>.
- 952 33. Wang MC, Kim S, Gonzalez AA, MacLeod KE, Winkleby MA. Socioeconomic and
 953 food-related physical characteristics of the neighbourhood environment are associated
 954 with body mass index. *J Epidemiol Community Health*. 2007;61:491-8. PMID:
 955 17496257. <http://www.ncbi.nlm.nih.gov/pubmed/17496257>.
- 956 34. Block JP, Christakis NA, O'Malley AJ, Subramanian SV. Proximity to food
 957 establishments and body mass index in the Framingham Heart Study offspring cohort
 958 over 30 years. *Am J Epidemiol*. 2011;174(10):1108-14. PMID: 21965186.
 959 <http://www.ncbi.nlm.nih.gov/pubmed/21965186>.
- 960 35. Gibson DM. The neighborhood food environment and adult weight status: estimates from
 961 longitudinal data. *Am J Public Health*. 2011;101(1):71-8. PMID: 21088263.
 962 <http://www.ncbi.nlm.nih.gov/pubmed/21088263>.
- 963 36. Lee H. The role of local food availability in explaining obesity risk among young school-
 964 aged children. *Soc Sci Med*. 2012;74(8):1193-203. PMID: 22381683.
 965 <http://www.ncbi.nlm.nih.gov/pubmed/22381683>.
- 966 37. Leung CW, Laraia BA, Kelly M, Nickleach D, Adler NE, Kushi LH, et al. The influence
 967 of neighborhood food stores on change in young girls' body mass index. *Am J Prev Med*.
 968 2011;41(1):43-51. PMID: 21665062. <http://www.ncbi.nlm.nih.gov/pubmed/21665062>.

- 969 38. Rossen LM, Curriero FC, Cooley-Strickland M, Pollack KM. Food availability en route
970 to school and anthropometric change in urban children. *J Urban Health*. 2013;90(4):653-
971 66. PMID: 23392564. <http://www.ncbi.nlm.nih.gov/pubmed/23392564>.
- 972 39. Shier V, An R, Sturm R. Is there a robust relationship between neighbourhood food
973 environment and childhood obesity in the USA? *Public Health*. 2012;126(9):723-30.
974 PMID: 22898435. <http://www.ncbi.nlm.nih.gov/pubmed/22898435>.
- 975 40. Sturm R, Datar A. Body mass index in elementary school children, metropolitan area
976 food prices and food outlet density. *Public Health*. 2005;119(12):1059-68. PMID:
977 16140349. <http://www.ncbi.nlm.nih.gov/pubmed/16140349>.
- 978 41. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in
979 the United States, 2011-2012. *JAMA*. 2014;311(8):806-14. PMID: 24570244.
980 <http://www.ncbi.nlm.nih.gov/pubmed/24570244>.
- 981 42. Shashaj B, Bedogni G, Graziani MP, Tozzi AE, DiCorpo ML, Morano D, et al. Origin of
982 cardiovascular risk in overweight preschool children: a cohort study of cardiometabolic
983 risk factors at the onset of obesity. *JAMA Pediatr*. 2014;168(10):917-24. PMID:
984 25111132. <http://www.ncbi.nlm.nih.gov/pubmed/25111132>.
- 985 43. Institute of Medicine. *Child and Adult Care Food Program: Aligning Dietary Guidance
986 for All*. 2011. Washington, DC: National Academies Press. Available from:
987 http://books.nap.edu/openbook.php?record_id=12959&page=R2.
- 988 44. Zhou YE, Emerson JS, Levine RS, Kihlberg CJ, Hull PC. Childhood obesity prevention
989 interventions in childcare settings: systematic review of randomized and nonrandomized
990 controlled trials. *Am J Health Promot*. 2014;28(4):e92-103. PMID: 24200332.
991 <http://www.ncbi.nlm.nih.gov/pubmed/24200332>.
- 992 45. Alkon A, Crowley AA, Neelon SE, Hill S, Pan Y, Nguyen V, et al. Nutrition and
993 physical activity randomized control trial in child care centers improves knowledge,
994 policies, and children's body mass index. *BMC Public Health*. 2014;14(1):215. PMID:
995 24580983. <http://www.ncbi.nlm.nih.gov/pubmed/24580983>.
- 996 46. Cespedes J, Briceno G, Farkouh ME, Vedanthan R, Baxter J, Leal M, et al. Promotion of
997 cardiovascular health in preschool children: 36-month cohort follow-up. *Am J Med*.
998 2013;126(12):1122-6. PMID: 24262725.
999 <http://www.ncbi.nlm.nih.gov/pubmed/24262725>.
- 1000 47. Cespedes J, Briceno G, Farkouh ME, Vedanthan R, Baxter J, Leal M, et al. Targeting
1001 preschool children to promote cardiovascular health: cluster randomized trial. *Am J Med*.
1002 2013;126(1):27-35.e3. PMID: 23062403.
1003 <http://www.ncbi.nlm.nih.gov/pubmed/23062403>.
- 1004 48. De Bock F, Breitenstein L, Fischer JE. Positive impact of a pre-school-based nutritional
1005 intervention on children's fruit and vegetable intake: results of a cluster-randomized trial.
1006 *Public Health Nutr*. 2012;15(3):466-75. PMID: 21859516.
1007 <http://www.ncbi.nlm.nih.gov/pubmed/21859516>.

- 1008 49. De Coen V, De Bourdeaudhuij I, Vereecken C, Verbestel V, Haerens L, Huybrechts I, et
1009 al. Effects of a 2-year healthy eating and physical activity intervention for 3-6-year-olds
1010 in communities of high and low socio-economic status: the POP (Prevention of
1011 Overweight among Pre-school and school children) project. *Public Health Nutr.*
1012 2012;15(9):1737-45. PMID: 22397833. <http://www.ncbi.nlm.nih.gov/pubmed/22397833>.
- 1013 50. Herman A, Nelson BB, Teutsch C, Chung PJ. "Eat Healthy, Stay Active!": a coordinated
1014 intervention to improve nutrition and physical activity among Head Start parents, staff,
1015 and children. *Am J Health Promot.* 2012;27(1):e27-36. PMID: 22950932.
1016 <http://www.ncbi.nlm.nih.gov/pubmed/22950932>.
- 1017 51. Natale RA, Lopez-Mitnik G, Uhlhorn SB, Asfour L, Messiah SE. Effect of a Child Care
1018 Center-Based Obesity Prevention Program on Body Mass Index and Nutrition Practices
1019 Among Preschool-Aged Children. *Health Promot Pract.* 2014. PMID: 24662896.
1020 <http://www.ncbi.nlm.nih.gov/pubmed/24662896>.
- 1021 52. Yin Z, Parra-Medina D, Cordova A, He M, Trummer V, Sosa E, et al. Miranos! Look at
1022 us, we are healthy! An environmental approach to early childhood obesity prevention.
1023 *Child Obes.* 2012;8(5):429-39. PMID: 23061498.
1024 <http://www.ncbi.nlm.nih.gov/pubmed/23061498>[http://online.liebertpub.com/doi/pdfplus/
1025 10.1089/chi.2011.0125](http://online.liebertpub.com/doi/pdfplus/10.1089/chi.2011.0125).
- 1026 53. Evans CE, Christian MS, Cleghorn CL, Greenwood DC, Cade JE. Systematic review and
1027 meta-analysis of school-based interventions to improve daily fruit and vegetable intake in
1028 children aged 5 to 12 y. *Am J Clin Nutr.* 2012;96(4):889-901. PMID: 22952187.
1029 <http://www.ncbi.nlm.nih.gov/pubmed/22952187>.
- 1030 54. Jensen JD, Hartmann H, de Mul A, Schuit A, Brug J, Consortium E. Economic incentives
1031 and nutritional behavior of children in the school setting: a systematic review. *Nutr Rev.*
1032 2011;69(11):660-74. PMID: 22029832. <http://www.ncbi.nlm.nih.gov/pubmed/22029832>.
- 1033 55. Langelotto G GA. Gardening Increases Vegetable Consumption in School-aged
1034 Children: A Meta-analytical Synthesis. *HortTechnology.* 2012;22(4):430-45.
1035 <http://horttech.ashspublications.org/content/22/4/430.abstract>.
- 1036 56. Chriqui JF, Pickel M, Story M. Influence of school competitive food and beverage
1037 policies on obesity, consumption, and availability: a systematic review. *JAMA Pediatr.*
1038 2014;168(3):279-86. PMID: 24473632. <http://www.ncbi.nlm.nih.gov/pubmed/24473632>.
- 1039 57. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y, et al.
1040 Interventions for preventing obesity in children. *Cochrane Database Syst Rev.*
1041 2011(12):CD001871. PMID: 22161367. <http://www.ncbi.nlm.nih.gov/pubmed/22161367>.
- 1042 58. Wang Y WY, Wilson RF, Bleich S, Cheskin L, Weston C, Showell N, Fawole O, Lau B,
1043 Segal J, . Childhood obesity prevention programs: comparative effectiveness review and
1044 meta-analysis. Comparative effectiveness review No. 115. AHRQ Publication No. 13-
1045 EHC081-EF [Internet]. June 2013. Available from:
1046 <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0057623/pdf/TOC.pdf>.

- 1047 59. Williams AJ, Henley WE, Williams CA, Hurst AJ, Logan S, Wyatt KM. Systematic
 1048 review and meta-analysis of the association between childhood overweight and obesity
 1049 and primary school diet and physical activity policies. *Int J Behav Nutr Phys Act.*
 1050 2013;10:101. PMID: 23965018. <http://www.ncbi.nlm.nih.gov/pubmed/23965018>.
- 1051 60. Geaney F, Kelly C, Greiner BA, Harrington JM, Perry IJ, Beirne P. The effectiveness of
 1052 workplace dietary modification interventions: a systematic review. *Prev Med.*
 1053 2013;57(5):438-47. PMID: 23850518. <http://www.ncbi.nlm.nih.gov/pubmed/23850518>.
- 1054 61. Aneni EC, Roberson LL, Maziak W, Agatston AS, Feldman T, Rouseff M, et al. A
 1055 systematic review of internet-based worksite wellness approaches for cardiovascular
 1056 disease risk management: outcomes, challenges & opportunities. *PLoS One.*
 1057 2014;9(1):e83594. PMID: 24421894. <http://www.ncbi.nlm.nih.gov/pubmed/24421894>.
- 1058 62. Kahn-Marshall JL, Gallant MP. Making healthy behaviors the easy choice for employees:
 1059 a review of the literature on environmental and policy changes in worksite health
 1060 promotion. *Health Educ Behav.* 2012;39(6):752-76. PMID: 22872583.
 1061 <http://www.ncbi.nlm.nih.gov/pubmed/22872583>.
- 1062 63. Verweij LM, Coffeng J, van Mechelen W, Proper KI. Meta-analyses of workplace
 1063 physical activity and dietary behaviour interventions on weight outcomes. *Obes Rev.*
 1064 2011;12(6):406-29. PMID: 20546142. <http://www.ncbi.nlm.nih.gov/pubmed/20546142>.
- 1065 64. Bell J, Mora G, Hagan E, Rubin V, Karpyn A. Access to Healthy Food and Why It
 1066 Matters: a Review of the Research. Oakland CA: PolicyLink. The Food Trust; 2013.
 1067 Available from: [http://thefoodtrust.org/uploads/media_items/access-to-healthy-](http://thefoodtrust.org/uploads/media_items/access-to-healthy-food.original.pdf)
 1068 [food.original.pdf](http://thefoodtrust.org/uploads/media_items/access-to-healthy-food.original.pdf).
- 1069 65. Treuhaft S, Karpyn A. The Grocery Gap: Who Has Access to Healthy Food and Why It
 1070 Matters. Oakland, CA: PolicyLink. The Food Trust,; 2010. Available from:
 1071 http://thefoodtrust.org/uploads/media_items/grocerygap.original.pdf.
 1072
 1073

Part D. Chapter 5: Food Sustainability and Safety

INTRODUCTION

In this chapter, the DGAC addresses food and nutrition issues that will inform public health action and policies to promote the health of the population through sustainable diets and food safety. An important reason for addressing sustainable diets, a new area for the DGAC, is to have alignment and consistency in dietary guidance that promotes both health and sustainability. This also recognizes the significant impact of food and beverages on environmental outcomes, from farm to plate to waste disposal, and, therefore, the need for dietary guidance to include the wider issue of sustainability. Addressing this complex challenge is essential to ensure a healthy food supply will be available for future generations. The availability and acceptability of healthy and sustainable food choices will be necessary to attain food security for the U.S. population over time. Integral to this issue is how dietary guidance and individual food choices influence the nation's capacity to meet the nutritional needs of the U.S. population. Food sustainability and food safety are also interrelated in generating a secure food supply. This chapter focuses on both sustainable diets and food safety.

Food Sustainability

Two definitions are relevant to the material presented in this chapter. These terms were slightly modified from the Food and Agriculture Organization (FAO) definitions to operationalize them for the Committee's work.^{1,2}

Sustainable diets: Sustainable diets are a pattern of eating that promotes health and well-being and provides food security for the present population while sustaining human and natural resources for future generations.

Food security: Food security exists when all people now, and in the future, have access to sufficient, safe, and nutritious food to maintain a healthy and active life.

The topic of *current* food security was addressed in Chapter 3 and to some extent in Chapter 4, where federal food programs were discussed. The topic of *long-term* food security was addressed within this chapter through examination of the evidence on sustainable diets.

The environmental impact of food production is considerable and if natural resources such as land, water and energy are not conserved and managed optimally, they will be strained and potentially lost. The global production of food is responsible for 80 percent of deforestation, more than 70 percent of fresh water use, and up to 30 percent of human-generated greenhouse gas (GHG) emissions.³ It also is the largest cause of species biodiversity loss.³ The capacity to

39 produce adequate food in the future is constrained by land use, declining soil fertility,
40 unsustainable water use, and over-fishing of the marine environment.⁴ Climate change, shifts in
41 population dietary patterns and demand for food products, energy costs, and population growth
42 will continue to put additional pressures on available natural resources. Meeting current and
43 future food needs will depend on two concurrent approaches: altering individual and population
44 dietary choices and patterns and developing agricultural and production practices that reduce
45 environmental impacts and conserve resources, while still meeting food and nutrition needs. In
46 this chapter, the Committee focuses primarily on the former, examining the effect of population-
47 level dietary choices on sustainability.

48
49 Foods vary widely in the type and amount of resources required for production, so as population-
50 level consumer demand impacts food production (and imports) it will also indirectly influence
51 how and to what extent resources are used.³ As the focus of the dietary guidelines is to shift
52 consumer eating habits toward healthier alternatives, it is imperative that, in this context, the
53 shift also involve movement toward less resource-intensive diets. Individual and population-level
54 adoption of more sustainable diets can change consumer demand away from more resource-
55 intensive foods to foods that have a lower environmental impact.³

56
57 In this chapter, the DGAC has used an evidence-based approach to evaluate the foods and food
58 components that improve the sustainability of dietary patterns as a step toward this desirable
59 goal. The approach used was to determine dietary patterns that are nutritionally adequate and
60 promote health, while at the same time are more protective of natural resources. This type of
61 comprehensive strategy also has been used by intergovernmental organizations. For example, the
62 FAO has identified the Mediterranean diet as an example of a sustainable diet due to its emphasis
63 on biodiversity and smaller meat portions,⁵ and the European Commission has developed a
64 “2020 Live Well Diet” to reduce GHG emissions through diet change.⁶

65
66 It should be noted that research in the area of dietary patterns and sustainability is rapidly
67 evolving and the methodologies for determining dietary patterns in populations and Life Cycle
68 Analysis of foods/food components and environmental outcomes have made significant advances
69 in recent years.^{7, 8} This is exemplified by the size of evidence base for this question and the fact
70 that several relevant articles have been published even since the close of the 2015 DGAC
71 Nutrition Evidence Library (NEL) scientific review period for this topic.⁹⁻¹¹

72
73 Figure D5.1 outlines the interconnected elements that the DGAC believes are necessary based on
74 current evidence to develop sustainable diets. Sustainable diets are realized by developing a food
75 system that embraces a core set of values illustrated in the figure. These values need to be
76 implemented through robust private and public sector partnerships, practices and policies across
77 the supply chain, extending from farms to distribution and consumption. New well-coordinated
78 policies that include, but are not limited to, agriculture, economics, transportation, energy, water

79 use, and dietary guidance need to be developed. Behaviors of all participants in the food system
 80 are central to creating and supporting sustainable diets.

81
 82
 83

Figure D5.1: Elements needed for sustainable diets



84
 85
 86
 87
 88
 89
 90
 91
 92
 93
 94
 95
 96
 97
 98
 99
 100
 101

Although the addition of sustainability topics in the *Scientific Report of the 2015 Dietary Guidelines Advisory Committee* is new in 2015 it was acknowledged as a topic of strong relevance but not addressed by the 2010 DGAC. It has been a widely discussed aspect of nutrition policy for the past decade in countries such as Germany, Sweden and other Nordic countries, the Netherlands, Australia, and Brazil. For example, in the Netherlands, the Advisory report, *Guidelines for a Healthy Diet: The Ecological Perspective* focused on guidelines that inform both health and ecological benefits using an evidence-based strategy.¹² Nordic countries, such as Sweden, have been researching sustainability and dietary choice since the late 1990s with the most recent edition of the Nordic Nutrition Recommendations (NNR) including an emphasis on the environmental impact of dietary recommendations.¹³ The German Dietary Guidelines developed a “sustainable shopping basket,” which is a consumer guide for shopping in a more sustainable way.¹⁴ Overall, the environmentally sustainable dietary guidance from these countries includes elements identified in this DGAC report as consistent with the extant data: a focus on decreasing meat consumption, choosing seafood from non-threatened stocks,

102 eating more plants and plant-based products, reducing energy intake, and reducing waste. Non-
103 governmental and international organizations, such as the United Nations, the FAO, the
104 Sustainable Development Commission in the United Kingdom (UK), the Institute of Medicine
105 (IOM), the Academy of Nutrition and Dietetics, and the National Research Council have all
106 convened working groups and commissioned reports on sustainable diets.^{2, 15-19} Overall, it is
107 clear that environmental sustainability adds further dimensions to dietary guidance; not just what
108 we eat but where and how food production, processing, and transportation are managed, and
109 waste is decreased.

110
111 The DGAC focused on two main topic areas related to sustainability: dietary patterns and
112 seafood. The identification of dietary patterns that are sustainable is a first step toward driving
113 consumer behavior change and demand and supply-chain changes. Furthermore, dietary patterns
114 were an overall focus area of the 2015 DGAC and allow for a more comprehensive approach to
115 total diet and health. This approach is particularly well suited for assessing overall environmental
116 impacts of food consumption, as all food components of a dietary pattern are identified, and
117 keeping within the context of health outcomes that have been documented for different dietary
118 patterns. The topic area of seafood was chosen because consumption has well-established health
119 benefits and the 2010 DGAC report highlighted the concern for seafood sustainability and called
120 for a better understanding of the environmental impact of aquaculture on seafood contaminants.
121 Meeting these recommendations, however, increases demand for seafood production and this, in
122 turn, poses challenges, as certain seafood species are depleted and marine waters are over fished,
123 while most other species are at the limits of sustainable harvesting. To meet these challenges, as
124 world capture fisheries production has leveled off, aquaculture production has increased to meet
125 demand.²⁰ Therefore, building upon the 2010 DGAC report, the 2015 DGAC addressed the
126 health benefits (nutrients) versus the risks (contaminants) of farm-raised (aquaculture) compared
127 to wild-caught seafood and reviewed the evidence on the worldwide capacity to produce enough
128 seafood to meet dietary guidelines. Overall, promoting sustainable fishing and aquaculture can
129 provide an example for broader ecosystem stewardship.²⁰

130 131 **Food Safety**

132 Food safety was first introduced in the *2000 Dietary Guidelines for Americans*, and the
133 recognition of the importance of food safety continued through the 2010 report. This chapter
134 updates the 2010 DGAC report related to food safety behaviors in the home environment and
135 evaluates new topics of food safety concern with very current and/or updated evidence. The
136 current/updated topics include the safety of beverages, specifically coffee and caffeine, and food
137 additives, specifically aspartame, in the U.S. food supply.

138
139 In 2015, the DGAC addressed new topics of concern. For the first time, the DGAC addressed the
140 safety of coffee/caffeine consumption, as well as the safety of consuming higher doses of
141 caffeine in products such as some energy drinks. The food additive, aspartame, has been the only

142 non-nutritive sweetener to be completely re-evaluated in recent years and the results of this
 143 reevaluation were deemed important because it includes the most recent science on aspartame
 144 and health. These topic areas were chosen for consideration because they are of high public
 145 health concern and very recent evidence has been published that significantly updates the
 146 knowledge base on health aspects related to caffeine and aspartame in the diet.

147
 148 For 2015, the DGAC brought forward the updated food safety principles to reduce risk of
 149 foodborne illnesses. These principles—Clean, Separate, Cook and Chill—are cornerstones of the
 150 Fight BAC! (www.fightbac.org) educational messages developed by the Partnership for Food
 151 Safety Education, a collaboration with the Federal government. These messages are reinforced
 152 by other USDA educational materials, including the *Be Food Safe* (www.befoodsafe.gov)
 153 efforts; *Is it Done Yet?* (www.isitdoneyet.gov); and *Thermy* (www.fsis.usda.gov/thermy), which
 154 outline key elements in thermometer use and placement to ensure proper cooking of meat,
 155 poultry, seafood, and egg products. Additional consumer-friendly information on food safety is
 156 available at www.foodsafety.gov. The DGAC brought forward the guidance for consumers that
 157 has been updated since 2010 on recommended procedures for hand sanitation, washing fresh
 158 produce, preventing cross-contamination, and safe meat, poultry, seafood and egg cooking
 159 temperatures and thermometer use from the FDA, the Center for Disease Control (CDC) and the
 160 Food Safety and Inspection Service (FSIS). The updated food safety tables are located at the end
 161 of this chapter.

162
 163

164 LIST OF QUESTIONS

165 Sustainable Diets

166 *Dietary Patterns*

167 1. What is the relationship between population-level dietary patterns and long-term food
 168 sustainability?

169

170 *Seafood*

171 2. What are the comparative nutrient profiles of current farm-raised versus wild caught
 172 seafood?

173 3. What are the comparative contaminant levels of current farm-raised versus wild caught
 174 seafood?

175 4. What is the worldwide capacity to produce farm-raised versus wild-caught seafood that is
 176 nutritious and safe for Americans?

177

178 Food Safety

179 5. What is the relationship between usual coffee/caffeine consumption and health?

- 180 6. What is the relationship between high-dose caffeine consumption and health?
181 7. What is the relationship between aspartame consumption and health?
182 8. What consumer behaviors prevent food safety problems? (Topic update from 2010 DGAC)

183

184 **METHODOLOGY**

185 **Sustainable Diets**

186 The topic of Question 1 is new for a DGAC review and involves an emerging area of scientific
187 investigation that is not readily addressed by traditional study designs such as randomized
188 controlled trials and prospective cohort studies. The literature related to sustainable diets and
189 dietary patterns involves a combination of food pattern modeling, Life Cycle Assessment (LCA)
190 methodology (examines all processes in the life cycle of each food component - from farm to
191 plate to waste), and determination of the environmental outcomes of the full LCA inventory.
192 Because of the unique nature of these studies, a modified NEL systematic review was conducted
193 for Question 1 on dietary patterns and sustainability. Databases included PubMed, Cochrane,
194 Navigator, and Embase and the search covered from January 2000 to March 2014. For this topic
195 and question, it was necessary to use different methods from those described in an original NEL
196 protocol because not all methods in the protocol could be applied. This is sometimes necessary,
197 according to the Cochrane Collaboration, but requires that methods from the original protocol
198 that could not be implemented in the current review be summarized.²¹ Due to the nature of the
199 evidence, the NEL 6-step process was tailored for the purposes of this systematic review, with
200 modifications to step 3 – extract data and assess the risk of bias. A description of the NEL
201 systematic review process is provided in *Part C: Methodology*. A new data extraction grid was
202 developed with emphasis on modeling studies, LCA methodology, and environmental outcomes.
203 The LCA is a standardized methodological framework for assessing the environmental impact
204 (or load) attributable to the life cycle of a food product. The customized grid was then used by
205 NEL abstractors to extract data from the included articles and this informed the evidence
206 synthesis (see *Appendix E-2.37 Evidence Portfolio*). In addition, NEL abstractors used a
207 different tool to assess individual study quality, not the NEL Bias Assessment Tool (BAT). This
208 alternative tool, the Critical Appraisal Checklist used by the *British Medical Journal*, was
209 appropriate for studies that used a modeling design. This checklist assesses studies that use
210 modeling to extrapolate progression of clinical outcomes, transform final outcomes from
211 intermediate measures, examine relations between inputs and outputs to apportion resource use,
212 and extrapolate findings from one clinical setting or population to another. To attain a high score,
213 studies must report the variables that have been modeled rather than directly observed; what
214 additional variables have been included or excluded; what statistical relations have been
215 assumed; and what evidence supports these assumptions.²²⁻²⁴ The checklist included key
216 components of the *British Medical Journal* checklist for economic evaluations, together with the

217 Eddy checklist on mathematical models. This Critical Appraisal Checklist was reviewed and
218 tested for applicability by two sustainability experts who served as consultants to the DGAC.

219
220 Question 2 on nutrient profiles in farm-raised versus wild-caught seafood was addressed using
221 data analysis from the USDA-Agricultural Research Service (ARS) National Nutrient Database
222 for Standard Reference, Release 27 (<http://www.ars.usda.gov/ba/bhnrc/ndl>).²⁵ The section on
223 finfish and shellfish products included nutrient profiles for both farm-raised and wild-caught
224 seafood for some species. These data were augmented using a USDA-funded report on fatty-acid
225 profiles of commercially available fish* in the United States that assessed additional farmed
226 species and compared results with the USDA-ARS NND.²⁶ Because this question was answered
227 using data analysis, it was not graded (as described in **Part C: Methodology**). For Question 3 on
228 contaminants in farm-raised versus wild-caught seafood, the DGAC used an expert report, the
229 *Report of the Joint Food and Agriculture Organization of the United Nations (FAO) and the*
230 *World Health Organization (WHO) Expert Consultation on the Risks and Benefits of Fish*
231 *Consumption, 2011.*²⁷ This report was chosen as the most updated and comprehensive source of
232 scientific information on the net health assessment of seafood consumption, including a
233 comparison between wild-caught and farm-raised seafood related to contaminants. Data on levels
234 of chemical contaminants (methyl mercury and dioxins) in a large number of seafood species
235 were reviewed, as well as recent scientific literature covering the risks and benefits of seafood
236 consumption. The sections of the report that were used to address the question were “Data on the
237 composition of fish” and “Risk-benefit comparisons.” Lastly, to address Question 4 on the
238 worldwide capacity to produce enough nutritious seafood, the Committee used the FAO’s report
239 on the *State of World Fisheries and Aquaculture, 2012.*²⁰ This was considered the most current
240 and comprehensive source on this topic, specifically the sections on “Selected Issues in Fisheries
241 and Aquaculture” and the “Organization for Economic Cooperation and Development (OECD)-
242 FAO Agricultural Outlook: chapter on fish.” The DGAC focused on matters that directly address
243 world production as it affects the supply of seafood for the U.S. population, particularly as the
244 U.S. relies on significant amounts of imported seafood (~90 percent).

245

246

247 **Food Safety**

248 For Question 5, the DGAC used an overview of systematic reviews (SRs)/meta-analyses (MA) to
249 address the relationship between usual caffeine/coffee consumption and health. This approach
250 allowed the DGAC to address the broad scope of the evidence on usual caffeine and health,
251 which heretofore had not been addressed by a DGAC. The DGAC used a modification of the
252 method described by the Cochrane Collaboration to conduct the review.²⁸ The steps included
253 development of analytical framework, determination of inclusion/exclusion criteria, description
254 of search strategy and databases used, determination of methodological quality using the

* The term “fish” in this chapter refers to finfish, which includes aquatic species such as salmon, tuna, and trout.

255 Assessment of Multiple Systematic Reviews (AMSTAR) tool, data extraction, summary of
256 results and key findings, and development of conclusion and grade for each outcome, as well as
257 implications of the evidence and research recommendations. Overlap of studies included across
258 the SRs/MA for the same health outcome was determined and recorded; however, SRs/MA were
259 not excluded for overlap. This approach allowed the Committee to assess and consider whether
260 SRs/MA on the same topic *independently* assessed similar results and arrived at generally similar
261 conclusions. The focus of this review was to summarize the existing SRs/MA on this question,
262 *not* to re-synthesize the evidence or to conduct a new meta-analysis or meta-synthesis.

263
264 For the overview on usual caffeine/coffee consumption and health, the target population was
265 healthy adults and adults at risk of chronic disease, as well as youth ages 2 years and older. The
266 intervention or exposure was caffeine/coffee consumption. The outcomes were clinical
267 endpoints: 1) chronic diseases, including cardiovascular, type 2 diabetes, and cancer, and total
268 mortality, 2) neurologic and cognitive diseases, including Alzheimer’s and Parkinson’s disease,
269 and 3) pregnancy outcomes, including miscarriage and low birth weight. The included studies
270 were SRs/MA and qualitative SRs; the date range was from 2000 to 2014. Data were extracted
271 for all SRs/MA with emphasis on MA results, including categorical and dose-response MA,
272 fixed or random effects models, heterogeneity and sources of heterogeneity, sub-group analysis,
273 and publication bias (see *Appendix E-2.39b Systematic Review/Meta-Analysis Data Table*). The
274 methodological quality of the included SRs/MA was determined using AMSTAR. Overlap of
275 studies included across the SRs/MA for the same health outcomes was determined and recorded;
276 however, SRs/MA were not excluded for overlap. Rather, the emphasis was to determine
277 consistency across studies.

278
279 For Question 6 on high-dose caffeine and health, a duplication assessment found two SRs and
280 these were used in lieu of conducting a full NEL SR. The details of duplication assessment are
281 provided in *Part C: Methodology*, and the Review of the Evidence for this question provide
282 further detail.

283
284 For Question 7 on aspartame and health, the European Food Safety Authority (EFSA) *Scientific*
285 *Opinion on the Re-evaluation of Aspartame as a Food Additive* was used. This was conducted by
286 the EFSA Panel of Food Additives and Nutrient Sources Added to Food (ANS).²⁹ The Panel
287 based its evaluation on original study reports and information submitted following public calls
288 for data as well as previous evaluations and additional literature that was available up to
289 February 2013. The 2015 DGAC considered only the human studies and related conclusions
290 from the EFSA report; animal studies and *in vitro* studies were not considered.

291 Lastly, this chapter provides a topic update from the 2010 DGAC on consumer behaviors and
292 food safety. Tables on this topic were updated to include the most recent recommendations.
293 Federal sources that were used for the update include: 1) Centers for Disease Control and
294 Prevention (CDC) - Hand washing: Clean Hands Save Lives;³⁰ 2) Food and Drug Administration

295 (FDA) - Food Facts, Raw Produce: Selecting It and Serving It Safely, 2012; Food Safety for
 296 Moms-to-Be: Safe Eats - Meat, Poultry & Seafood;³¹ and 3) USDA/Food Safety and Inspection
 297 Service (FSIS) – Food Safety Fact Sheets.³²

298

299 **SUSTAINABLE DIETS**

300 Evaluating the link between sustainability and dietary guidance will inform policies and practice
 301 to ensure food security for present and future generations. The DGAC concentrated its review on
 302 the inter-relatedness between human health and food sustainability, with a focus on dietary
 303 patterns, a theme of the 2015 DGAC.

304

305 **Dietary Patterns and Sustainability**

306 **Question 1: What is the relationship between population-level dietary patterns**
 307 **and long-term food sustainability?**

308 **Source of Evidence:** Modified NEL systematic review

309 **Conclusion**

310 Consistent evidence indicates that, in general, a dietary pattern that is higher in plant-based
 311 foods, such as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in animal-
 312 based foods is more health promoting and is associated with lesser environmental impact (GHG
 313 emissions and energy, land, and water use) than is the current average U.S. diet. A diet that is
 314 more environmentally sustainable than the average U.S. diet can be achieved without excluding
 315 any food groups. The evidence consists primarily of Life Cycle Assessment (LCA) modeling
 316 studies or land-use studies from highly developed countries, including the United States.

317 **DGAC Grade: Moderate**

318

319 **Implications**

320 A moderate to strong evidence base supports recommendations that the U.S. population move
 321 toward dietary patterns that generally increase consumption of vegetables, fruits, whole grains,
 322 legumes, nuts and seeds, while decreasing total calories and some animal-based foods. This can
 323 be achieved through a variety of dietary patterns, including the Healthy USDA-style Pattern, the
 324 Healthy Vegetarian Pattern, and the Healthy Mediterranean-style Pattern (for more details on the
 325 patterns, see *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and*
 326 *Trends*). Each of these patterns provides more plant-based foods and lower amounts of meat than
 327 are currently consumed by the U.S. population.

328

329 Sustainability considerations provide an additional rationale for following the Dietary Guidelines
330 for Americans and should be incorporated into federal and local nutrition feeding programs when
331 possible. Using sustainability messaging in communication strategies should be encouraged. The
332 application of environmental and sustainability factors to dietary guidelines can be accomplished
333 because of the compatibility and degree of overlap between favorable health and environmental
334 outcomes.

335

336 Much has been done by the private and public sectors to improve environmental policies and
337 practices around production, processing, and distribution *within* individual food categories. It
338 will be important that *both* a greater shift toward healthful dietary patterns and an improved
339 environmental profile across food categories are achieved to maximize environmental
340 sustainability now and to ensure greater progress in this direction over time.

341

342 Consumer friendly information that facilitates understanding the environmental impact of
343 different foods should be considered for inclusion in food and menu labeling initiatives.

344

345 Careful consideration will need to be made to ensure that sustainable diets are affordable for the
346 entire U.S. population.

347

348 Promoting healthy diets that also are more environmentally sustainable now will conserve
349 resources for present and future generations, ensuring that the U.S. population has access to a
350 diet that is healthy as well as sustainable and secure in the future.

351

352

353 **Review of the Evidence**

354 A total of 15 studies met the inclusion criteria for this systematic review.³³⁻⁴⁸ The body of
355 evidence consisted primarily of dietary pattern modeling studies that assessed related
356 environmental outcomes. These studies were conducted between the years 2003 and 2014 in the
357 U.S., the UK, Germany, the Netherlands, France, Spain, Italy, Australia, Brazil, and New
358 Zealand. Dietary patterns that were examined included vegetarian, lacto-ovo vegetarian, and
359 vegan dietary patterns; the average and dietary guidelines-related dietary patterns of respective
360 countries examined; Mediterranean-style dietary patterns; and sustainable diets. The most
361 frequent comparison diet was the average dietary pattern of the country, although numerous
362 studies made additional comparisons across many of the above dietary patterns. Another
363 approach was to examine diet “scenarios” that modeled different percentage replacements of
364 meat and dairy foods with plant-based foods. The modeling studies used cross-sectional
365 assessment of dietary intake from national nutrition surveys of representative adult populations;
366 for example, the British National Diet and Nutrition Survey (NDNS) from studies in the UK,^{34, 39}
367 the National Nutrition Surveys (NNS) in Germany,⁴⁰ or the Australian National Nutrition
368 Survey³⁸ were used to determine the observed average dietary patterns. The average dietary

369 patterns were then compared with other modeled dietary patterns, such as vegetarian or
 370 Mediterranean- style patterns, as described in detail below. All of the countries were highly
 371 developed countries with dietary guidelines and, therefore, generalizable to the U.S. population.
 372 The study quality for the body of evidence ranged from scores of 7/12 to 12/12 (indicating the
 373 evidence was of high quality) using a modified Critical Appraisal Checklist (see *Appendix E-*
 374 *2.37 Evidence Portfolio*).

375
 376 Health outcomes associated with the dietary patterns were most often documented based on
 377 adherence to dietary guidelines-related patterns, variations on vegetarian dietary patterns, or
 378 Mediterranean-style dietary patterns. Diet quality was assessed in some studies using an a priori
 379 index, such as the Healthy Eating Index (HEI) or the WHO Index. In some studies, health
 380 outcomes also were modeled. For example Scarborough et al. used the DIETRON model to
 381 estimate deaths delayed or averted for each diet pattern.⁴⁶ One study assessed the synergy
 382 between health and sustainability scores using the WHO Index and the LCA sustainability score
 383 to assess combined nutritional and ecological value.⁴⁶

384
 385 The environmental impacts that were most commonly modeled were GHG emissions and use of
 386 resources such as agricultural land, energy, and water. In many studies, the environmental impact
 387 for each food/food category was obtained using the LCA method. The LCA is a standardized
 388 methodological framework for assessing the environmental impact (or load) attributable to the
 389 life cycle of a food product. The life cycle for a food typically includes agricultural production,
 390 processing and packaging, transportation, retail, use, and waste disposal.^{33, 49-51} An inventory of
 391 all stages of the life cycle is determined for each food product and a “weight” or number of
 392 points is then attributed to each food or food category, based on environmental impacts such as
 393 resource extraction, land use, and relevant emissions. These environmental impact results can be
 394 translated into measures of damage done to human health, ecosystem quality, and energy
 395 resources using programs such as Eco-Indicator.⁵² In addition to the health assessment
 396 approaches listed above, some studies used LCA analysis with a standardized approach to
 397 determine damages from GHG emissions and use of resources; these damage outcome included
 398 human health as an environmental damage component, such as the number and duration of
 399 diseases and life years lost due to premature death from environmental causes.

400
 401 Few studies assessed food security. These studies assessed food security in terms of the cost
 402 difference between an average dietary pattern for the country studied and a sustainable dietary
 403 pattern for that population.^{36, 39, 48} The basic food basket concept was used in some studies,
 404 representing household costs for a two-adult/two-child household.

405

406 ***Identified Dietary Patterns and Health and Sustainability Outcomes***

407 **Vegetarian and Meat-based Diets**

408 Several studies examined variations on vegetarian diets, or a spectrum from vegan to omnivorous
 409 dietary patterns, and associated environmental outcomes.^{34, 35, 37, 41} Peters et al. examined 42
 410 different dietary patterns and land use in New York, with patterns ranging from low-fat, lacto-
 411 ovo vegetarian diets to high fat, meat-rich omnivorous diets; across this range, the diets met U.S.
 412 dietary guidelines when possible.⁴¹ They found that, overall, increasing meat in the diet increased
 413 per capita land requirements; however, increasing total dietary fat content of low-meat diets (i.e.
 414 vegetarian alternatives) increased the land requirements compared to high-meat diets. In other
 415 words, although meat increased land requirements, diets including meat could feed more people
 416 than some higher fat vegetarian-style diets. Aston et al. assessed a pattern that was modeled on a
 417 feasible UK population in which the proportion of vegetarians in the survey was doubled, and the
 418 remainder adopted a diet pattern consistent with the lowest category of red and processed meat
 419 (RPM) consumers. They found the combination of low RPM + vegetarian diet had health
 420 benefits of lowering the risk of diabetes and colorectal cancer, determined from risk relationships
 421 for RPM and CHD, diabetes, and colorectal cancer from published meta-analyses.⁵³⁻⁵⁵
 422 Furthermore, the expected reduction in GHG for this diet was ~3 percent of current total carbon
 423 dioxide (CO₂) emissions for agriculture. De Carvalho et al. also examined a high RPM dietary
 424 pattern with diet quality assessed using the Brazilian Healthy Eating Index.³⁷ They found that
 425 excessive meat intake was associated not only with poorer diet quality but also with increased
 426 projected GHG emissions (~ 4 percent total CO₂ emitted by agriculture). Taken together, the
 427 results on RPM intake indicate that reduced consumption is expected to improve some health
 428 outcomes and decrease GHG emissions, as well as land use compared to current RPM
 429 consumption. Baroni et al. examined vegan, vegetarian, and omnivorous diets, both organically
 430 and conventionally grown, and found that the organically grown vegan diet had the most
 431 potential health benefits; whereas, the conventionally grown average Italian diet had the least.³⁷
 432 The organically grown vegan diet also had the lowest estimated impact on resources and
 433 ecosystem quality, and the average Italian diet had the greatest projected impact. Beef was the
 434 single food with the greatest projected impact on the environment; other foods estimated to have
 435 high impact included cheese, milk, and seafood.

436
 437 Vegetarian diets, dietary guidelines-related diets, and Mediterranean-style diets were variously
 438 compared with the average dietary patterns in selected countries.^{38, 40, 42, 46} Overall, the estimated
 439 greater environmental benefits, including reduced projected GHG emissions and land use,
 440 resulted from vegan, lacto-ovo vegetarian, and pesco-vegetarian diets, as well as dietary
 441 guidelines-related and Mediterranean-style dietary patterns. These diets had higher overall
 442 predicted health scores than the average diet patterns. Moreover, for the most part, the high
 443 health scores of these dietary patterns were paralleled by high combined estimated sustainability
 444 scores. According to van Doreen et al., the synergy measured across vegetarian, Mediterranean-
 445 style, and dietary guidelines-related scores could be explained by a reduction in consumption of

446 meat, dairy, extras (i.e., snacks and sweets), and beverages, as well as a reduction in overall food
 447 consumption.⁴²

448

449 **Mediterranean-Style Dietary Patterns**

450 The Mediterranean-style dietary pattern was examined in both Mediterranean and non-
 451 Mediterranean countries.^{44,46} In all cases, adherence to a Mediterranean-style dietary pattern—
 452 compared to usual intake—reduced the environmental footprint, including improved GHG
 453 emissions, agricultural land use, and energy and water consumption. Both studies limited either
 454 red and processed meat⁴⁰ or meat and poultry⁴² to less than 1 serving per week, and increased
 455 seafood intake. The authors concluded that adherence to a Mediterranean-style dietary pattern
 456 would make a significant contribution to increasing food sustainability, as well as increasing the
 457 health benefits that are well-documented for this type of diet (see *Part D. Chapter 2: Dietary*
 458 *Patterns, Foods and Nutrients, and Health Outcomes*).

459

460 **Diet Scenarios**

461 Other studies examined different diet “scenarios” that generally replaced animal foods in various
 462 ways with plant foods.^{43,45,47} Scarborough et al. found that a diet with 50 percent reduced total
 463 meat and dairy replaced by fruit, vegetables, and cereals contributed the most to estimated
 464 reduced risk of total mortality and also had the largest potential positive environmental impact.¹³
 465 This diet scenario increased fruit and vegetable consumption by 63 percent and decreased
 466 saturated fat and salt consumption; micronutrient intake was generally similar with the exception
 467 of a drop in vitamin B₁₂.

468

469 Pradhan et al. examined 16 global dietary patterns that differed by food and energy content,
 470 grouped into four categories with per capita intake of low, moderate, high, and very high kcal
 471 diets. They assessed the relationship of these patterns to GHG emissions.⁴³ Low-energy diets had
 472 less than 2,100 kcal/cap/day and were composed of more than 50 percent cereals or more than 70
 473 percent starchy roots, cereals, and pulses. Animal products were minor in this group (<10
 474 percent). Moderate, high, and very high energy diets had 2,100-2,400, 2,400-2,800, and greater
 475 than 2,800 kcal/cap/day, respectively. Very high calorie diets had high amounts of meat and
 476 alcoholic beverages. Overall, very high calorie diets, common in the developed world, exhibited
 477 high total per capita CO_{2eq} emissions due to high carbon intensity and high intake of animal
 478 products; the low-energy diets, on the other hand, had the lowest total per capita CO_{2eq} emissions.

479

480 Lastly, Vieux et al. examined dietary patterns with different indicators of nutritional quality and
 481 found that despite containing large amounts of plant foods, not all diets of the highest nutritional
 482 quality were those with the lowest GHG emissions.⁴⁷ For this study, the diet pattern was assessed
 483 by using nutrient-based indicators; high quality diets had energy density below the median, mean
 484 adequacy ratio above the median, and a mean excess ratio (percentage of maximum
 485 recommended for nutrients that should be limited – saturated fat, sodium, and free sugars) below

486 the median. Four diet patterns were identified based on compliance with these properties to
 487 generate one high quality diet, two intermediate quality diets, and one low quality diet. In this
 488 study, the high quality diets had higher GHG emissions than did the low quality diets. Regarding
 489 the food groups, a higher consumption of starches, sweets and salted snacks, and fats was
 490 associated with lower diet-related GHG emissions and an increased intake of fruit and
 491 vegetables, was associated with increased diet-related GHG emissions. However, the strongest
 492 positive association with GHG emissions was still for the ruminant meat group. Overall, this
 493 study used a different approach from the other studies in this review, as nutritional quality
 494 determined the formation of dietary pattern categories.

495

496 **Sustainable Diets and Costs**

497 Three studies examined sustainable diets and related costs.^{36, 39, 48} Barosh et al. examined food
 498 availability and cost of a health and sustainability (H&S) food basket, developed according to the
 499 principles of the Australian dietary guidelines as well as environmental impact.³⁶ The food
 500 basket approach is a commonly used method for assessing and monitoring food availability and
 501 cost. The typical food basket was based on average weekly food purchases of a reference
 502 household made up of two adults and two children. For the H&S basket, food choices were based
 503 on health principles and environmental impact. The H&S basket was compared to the typical
 504 Australian basket and it was determined that the cost of the H&S basket was more than the
 505 typical basket in five socioeconomic areas; the most disadvantaged spent 30 percent more for the
 506 H&S basket. The authors concluded that the most disadvantaged groups at both neighborhood
 507 and household levels experienced the greatest inequality in accessing an affordable H&S basket.
 508 Macdiarmid et al. examined a sustainable diet (met all energy and nutrient needs and maximally
 509 decreased GHG emissions), a “sustainable with acceptability constraints” diet (added foods
 510 commonly consumed in the UK; met energy, nutrient, and seafood recommendations as well as
 511 recommended minimum intakes for fruits and vegetables and did not exceed the maximum
 512 recommended for red and processed meat), and the average UK diet.⁷ They found that the
 513 sustainable diet that was generated would decrease GHG emissions from primary production (up
 514 to distribution) by 90 percent, but consisted of only seven foods. The acceptability constraints
 515 diet included 52 foods and was projected to reduce GHG emissions by 36 percent. This diet
 516 included meat and dairy but less than the average UK diet. The cost of the sustainable +
 517 acceptability diet was comparable to that of the average UK diet. These results showed that a
 518 sustainable diet that meets dietary requirements and has lower GHG can be achieved without
 519 eliminating meat or dairy products completely, or increasing the cost to the consumer. Lastly,
 520 Wilson et al. examined 16 dietary patterns modeled to determine which patterns would minimize
 521 estimated risk of chronic disease, cost, and GHG emissions.⁴⁸ These patterns included low-cost
 522 and low-cost + low GHG diet patterns, as well as healthy patterns with high vegetable intakes
 523 including Mediterranean or Asian patterns, as well as the average New Zealand pattern. The
 524 authors found that diets that aimed to minimize cost and estimated GHG emissions also had
 525 health advantages, such as the simplified low-cost Mediterranean-style and simplified Asian-

526 style diets, both of which would lower cardiovascular disease and cancer risk, compared to the
527 average New Zealand diet. However, dietary variety was limited and further optimization to
528 lower GHG emissions increased cost.

529
530 Overall, the studies were consistent in showing that higher consumption of animal-based foods
531 was associated with higher estimated environmental impact, whereas consumption of more plant-
532 based foods as part of a lower meat-based or vegetarian-style dietary pattern was associated with
533 estimated lower environmental impact compared to higher meat or non-plant-based dietary
534 patterns. Related to this, the total energy content of the diet was also associated with estimated
535 environmental impact and higher energy diets had a larger estimated impact. For example, for
536 fossil fuel alone, one calorie from beef or milk requires 40 or 14 calories of fuel, respectively,
537 whereas one calorie from grains can be obtained from 2.2 calories of fuel.⁴² Additionally, the
538 evidence showed that dietary patterns that promote health also promote sustainability; dietary
539 patterns that adhered to dietary guidelines were more environmentally sustainable than the
540 population's current average level of intake or pattern. Taken together, the studies agreed on the
541 environmental impact of different dietary patterns, despite varied methods of assessing
542 environmental impact and differences in components of environmental impact assessed (e.g.
543 GHG emissions or land use). The evidence on whether sustainable diets were more or less
544 expensive than typically consumed diets in some locations was limited and inconsistent.

545
546 Three additional reports on the relationship between dietary patterns and sustainability were
547 published after this systematic review was completed. Two of these reports were consistent with,
548 and provided more evidence to support the Committee's findings that dietary guidelines-related
549 diets, Mediterranean-style diets, and vegetarian (and variations) diets are associated with
550 improved environmental outcomes. Tilman and Clark showed that following a Mediterranean,
551 vegetarian (lacto-ovo), or pesco-vegetarian dietary pattern would decrease both current and
552 projected GHG emissions and land use.¹¹ Eshel et al. reported on the five main animal-based
553 categories in the U.S. diet – dairy, beef, poultry, pork, and eggs – and their required feeds
554 including crops, byproducts, and pasture. They found that beef production required more land
555 and irrigation water and produced more GHG emissions than dairy, poultry, pork, or eggs.⁹ In
556 addition, as a standard comparator, staple plant foods had lower land use and GHG emissions
557 than did dairy, poultry, pork, or eggs. In contrast, a report from Heller and Keoleian suggests that
558 an isocaloric shift from the average U.S. diet (at current U.S. per capita intake of 2,534 kcals/day
559 from Loss-Adjusted Food Availability (LAFA) data) to a pattern that adheres to the *2010 Dietary*
560 *Guidelines for Americans* would result in a 12 percent increase in diet-related GHG emissions.¹⁰
561 This result was modified, however, by their finding that if Americans consumed the
562 recommended pattern within the recommended calorie intake level of 2,000 kcal/day, there
563 would be a 1 percent *decrease* in GHG emissions. This finding reinforces the overriding 2010
564 DGA recommendation that all of the guidelines need to be followed, including appropriate
565 calorie intake levels for age, gender, and activity level. Furthermore, in contrast to the findings of

566 Eshel et al. regarding dairy, Heller and Keoleian suggest that increases in dairy to follow 2010
567 DGA recommendations contribute significantly to increased GHG emissions and counters the
568 modeled benefits of decreased meat consumption.¹⁰

569

570 *For additional details on this body of evidence, visit: Appendix E-2.37*

571

572 **Seafood Sustainability**

573 ***Background***

574 Seafood is recognized as an important source of key macro- and micronutrients. The health
575 benefits of seafood, including support of optimal neurodevelopment and prevention of
576 cardiovascular disease, are likely due in large part to long-chain n-3 polyunsaturated fatty acids
577 (PUFA), docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), although seafood also
578 are good sources of other nutrients including protein, selenium, iodine, vitamin D, and choline.²⁷
579 Currently, seafood production is in the midst of rapid expansion to meet growing worldwide
580 demand, but the collapse of some fisheries due to overfishing in past decades raises concerns
581 about the ability to produce safe and affordable seafood to supply the U.S. population and meet
582 current dietary intake recommendations of at least 8 ounces per week.^{20, 56} Capture fisheries
583 (wild caught) production has leveled-off as a proportion of fully exploited stocks, and this is due
584 in part to national and international efforts on seafood sustainably (e.g., the U.S. Magnuson-
585 Stevens Fishery Conservation and Management Act (2006) mandating annual catch limits,
586 managed by the U.S. National Oceanographic and Atmospheric Administration). In contrast, the
587 increased productivity of worldwide aquaculture (farm-raised) is expected to continue and will
588 play a major role in expanding the supply of seafood.²⁰ Expanding farm-raised seafood has the
589 potential to ensure sufficient amounts of seafood to allow the U.S. population to consume levels
590 recommended by dietary guidelines.⁵⁷ Productivity gains should be implemented in a sustainable
591 manner with greater attention to maintaining or enhancing the high nutrient density characteristic
592 of captured seafood. Consistent with overall sustainability goals, farm-raised finfish (e.g.,
593 salmon and trout) is more sustainable than terrestrial animal production (e.g., beef and pork) in
594 terms of GHG emissions and land/water use.^{58, 59} Currently, the United States imports the
595 majority of its seafood (~90 percent), and approximately half of that is farmed.⁶⁰ The major
596 groups commonly referred to as finfish, shellfish, and crustaceans include more than 500 species,
597 and thus, generalizations to all seafood must be made with caution.

598

599 **Question 2: What are the comparative nutrient profiles of current farm-raised**
 600 **versus wild caught seafood?**

601 **Source of evidence:** USDA Agriculture Research Service (ARS) National Nutrient Database
 602 (NND)²⁵ updated with USDA-funded survey of most commonly consumed species in the United
 603 States.²⁶

604

605 **Conclusion**

606 For commonly consumed fish species in the United States, such as bass, cod, trout, and
 607 salmon, farmed-raised seafood has as much or more of the omega-3 fatty acids EPA and DHA
 608 as the same species captured in the wild. In contrast, farmed low-trophic species, such as
 609 catfish and crawfish, have less than half the EPA and DHA per serving than wild caught, and
 610 these species have lower EPA and DHA regardless of source than do salmon. Farm-raised
 611 seafood has higher total fat than wild caught. Recommended amounts of EPA and DHA can
 612 be obtained by consuming a variety of farm-raised seafood, especially high-trophic species,
 613 such as salmon and trout.

614

615 **Implications**

616 The U.S. population should be encouraged to eat a wide variety of seafood that can be wild
 617 caught or farmed, as they are nutrient-dense foods that are uniquely rich sources of healthy fatty
 618 acids. It should be noted that low-trophic farm-raised seafood, such as catfish and crayfish, have
 619 lower EPA and DHA levels than do wild-caught. Nutrient profiles in popular low-trophic farmed
 620 species should be improved through feeding and processing systems that produce and preserve
 621 nutrients similar to those of wild-caught seafood of the same species.

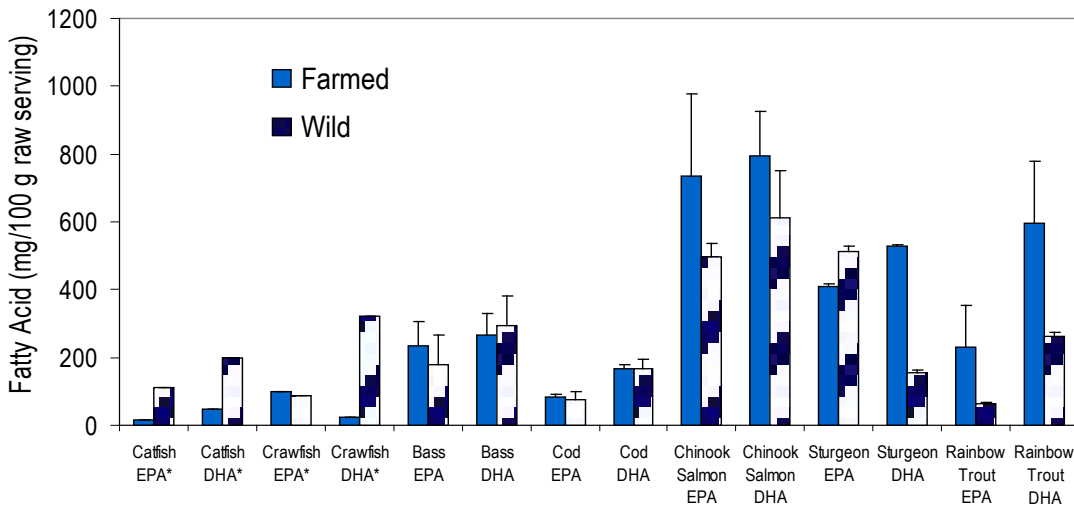
622

623 **Review of the Evidence**

624 The USDA-Agricultural Research Service (ARS) National Nutrient Database (NND) for
 625 Standard Reference, Release 27 was used to address this question
 626 (<http://www.ars.usda.gov/ba/bhnrc/ndl>).²⁵ The section on finfish and shellfish products included
 627 nutrient profiles for both farm-raised and wild-caught seafood for some species. These data were
 628 augmented using a USDA-funded report on fatty-acid profiles of commercially available fish in
 629 the United States that assessed additional farmed species and compared results²⁶ with the USDA-
 630 ARS NND.²⁵ The samples collected were from different regions of the United States during
 631 different seasons. For wild-caught species, the nutrient profile is determined by changes in
 632 environmental conditions, whereas, for farmed species, the nutrient profile is dependent on the
 633 amount, timing, and composition of the feed.²⁶ Because aquaculture diets can be continually
 634 modified, updates are important to monitor EPA and DHA in commercial seafood species, to

635 provide consumers with the most accurate information. The NND provided nutrient profiles for
 636 six seafood species with data on both wild-caught and farm-raised versions: four fish (rainbow
 637 trout, Atlantic and Coho salmon, and catfish), eastern oysters, and mixed species crayfish. The
 638 key nutrients EPA and DHA were on average comparable or greater for farmed trout, salmon,
 639 and oysters compared to wild capture, reflecting the higher total fat content of these farmed
 640 species. On the other hand, low-trophic species, such as catfish and crayfish, when farmed, were
 641 lower in EPA and DHA compared to wild capture. Cladis et al. determined EPA and DHA levels
 642 for five farmed and wild fish species (rainbow trout, white sturgeon, Chinook salmon, Atlantic
 643 cod, striped bass), providing an update and comparison for some of these species (Figure
 644 D5.2)²⁶. Farmed Atlantic salmon was similar between the NND and the update and most other
 645 species compared well; however, Chinook salmon and sturgeon showed differences in EPA and
 646 DHA content (although farmed and wild were not distinguished in the NND). Overall, these data
 647 showed that existing DGAC recommendations to consume a variety of seafood can be met by
 648 consuming a diverse range of species, including farmed species.
 649

650 **Figure D5.2. Comparison of EPA and DHA drawn from data in USDA National Nutrient**
 651 **Database²⁵ and update from Cladis et al.²⁶**
 652



653
 654
 655 *For additional details on this body of evidence, visit: Appendix E-2.38 Evidence Portfolio and*
 656 <http://www.ars.usda.gov/ba/bhnrc/ndl>
 657
 658

659 **Question 3. What are the comparative contaminant levels of current farm-raised**
 660 **versus wild caught seafood?**

661 **Source of evidence:** Report of the Joint United Nations Food and Agriculture
 662 Organization/World Health Organization Expert Consultation on the Risks and Benefits of Fish
 663 Consumption. Rome, 25–29 January 2010. FAO Fisheries and Aquaculture Report No. 978.²⁷

664
 665 **Conclusion**

666 The DGAC concurs with the Consultancy that, for the majority of commercial wild and farmed
 667 species, neither the risks of mercury nor organic pollutants outweigh the health benefits of
 668 seafood consumption, such as decreased cardiovascular disease risk and improved infant
 669 neurodevelopment. However, any assessment evaluates evidence within a time frame and
 670 contaminant composition can change rapidly based on the contamination conditions at the
 671 location of wild catch and altered production practices for farmed seafood. **DGAC Grade:**
 672 **Moderate**

673
 674 **Implications**

675 Based on risk/benefit comparisons, either farmed or wild-caught seafood are appropriate choices
 676 to consume to meet current Dietary Guidelines for Americans for increased seafood
 677 consumption. The DGAC supports the current FDA and EPA recommendations that women who
 678 are pregnant (or those who may become pregnant) and breastfeeding should not eat certain types
 679 of seafood—tilefish, shark, swordfish, and king mackerel—because of their high methyl mercury
 680 contents. Attention should be paid to local seafood advisories when eating seafood caught from
 681 local rivers, streams, and lakes.

682
 683 Based on the most current evidence on mercury levels in albacore tuna provided in the Report of
 684 the Joint United Nations Food and Agriculture Organization/World Health Organization Expert
 685 Consultation on the Risks and Benefits of Fish Consumption, 2010,²⁷ the DGAC recommends
 686 that the EPA and FDA re-evaluate their current recommendations⁶¹ for women who are pregnant
 687 (or for women who may become pregnant) or breastfeeding to limit white albacore tuna to not
 688 more than 6 ounces a week.

689
 690 **Review of the Evidence**

691 The Report of the FAO/WHO Expert Consultation on the Risks and Benefits of Fish
 692 Consumption²⁷ was used to address this question. This report was chosen as the most current and
 693 comprehensive source on contaminants in wild-caught and farm-raised seafood, and the DGAC
 694 focused on data that addressed the specific comparison between the two. The sections of the
 695 report that were used to address the question were “Data on the composition of fish” and “Risk-
 696 benefit comparisons.” The consultancy took a net effects approach, balancing benefits of

697 seafood, especially benefits associated with EPA and DHA, against the adverse effects of
698 mercury and persistent organic pollutants (POPs), including polychlorinated biphenyls,
699 polychlorinated dibenzo-*p*-dioxins, and polychlorinated dibenzofurans, collectively referred to as
700 dioxins. The Expert Consultancy compiled EPA and DHA, mercury, and dioxins compositional
701 data from national databases of the United States, France, Norway, and Japan, as well as an
702 international database. Together, these provided information on total fat, EPA and DHA, total
703 mercury, and dioxins for a large number of seafood species, including three farmed and wild
704 species (salmon, rainbow trout, and halibut). Two specific outcomes were considered for
705 risk/benefit: 1) prenatal exposure and offspring neurodevelopment, and 2) mortality from
706 cardiovascular diseases and cancer.

707
708 Overall, for the species examined, levels of mercury and dioxins were in the same range for
709 farmed and wild seafood. Related to risk/benefit, at the same level of mercury content (lowest [\leq
710 $0.1 \mu\text{g/g}$] and 2nd lowest [$0.1 - 0.5 \mu\text{g/g}$] levels), farmed seafood had the same or higher levels of
711 EPA and DHA as wild-caught. At the same level of dioxin content (2nd lowest [$0.5 - 4 \text{ pg toxic}$
712 $\text{equivalents (TEQ)/g}$] level), farmed seafood had the same or higher levels of EPA and DHA as
713 wild-caught. Only wild-caught Pacific salmon had the lowest level of dioxins ($<0.5 \text{ pg TEQ/g}$).
714 Overall, the quantitative risk/benefit analysis was not different for farmed compared to wild-
715 caught seafood. For both, using the central estimate for benefits of DHA and for harm from
716 mercury, the neurodevelopmental risks of not eating seafood exceeded the risks of eating
717 seafood. Similarly, for coronary heart disease (CHD) in adults, there were CHD mortality
718 benefits from eating seafood and CHD risks from not eating seafood, except for seafood in the
719 highest dioxin category and lowest EPA and DHA category, which did not include any of the
720 farm-raised species considered.

721
722 Albacore tuna, produced only from wild marine fisheries, is a special case of a popular fish
723 highlighted by the 2004 FDA and EPA advisory.^{61, 62} For all levels of intake including more
724 than double the 12 ounces per week recommendation, all evidence was in favor of net benefits
725 for infant development and CHD risk reduction.

726
727 Limitations in the evidence included the small number of farmed and wild seafood species
728 comparisons considered by the Expert Consultancy, and the possibility of rapid change that may
729 occur in the concentration of contaminants locally. In addition, seafood contaminants are closely
730 linked to levels of contaminants in feed.

731
732 ***For additional details on this body of evidence, visit: Report of the Joint Food and Agriculture***
733 ***Organization of the United Nations (FAO) and the World Health Organization (WHO) Expert***
734 ***Consultation on the Risks and Benefits of Fish Consumption, 2011. Available at***
735 **<http://www.fao.org/docrep/014/ba0136e/ba0136e00.pdf>**

736

737 **Question 4: What is the worldwide capacity to produce farm-raised versus wild-**
 738 **caught seafood that is nutritious and safe for the U.S. population?**

739 **Source of evidence:** United National (UN) Food and Agriculture Organization (FAO) report
 740 on *The State of World Fisheries and Agriculture*.²⁰

741
 742 **Conclusions**

743 The DGAC concurs with the FAO report that consistent evidence demonstrates that capture
 744 fisheries increasingly managed in a sustainable way have remained stable over several decades.
 745 However, on average, capture fisheries are fully exploited and their continuing productivity
 746 relies on careful management to avoid over-exploitation and long-term collapse. **DGAC Grade:**
 747 **Strong**

748
 749 The DGAC endorses the FAO report that capture fisheries production plateaued around 1990
 750 while aquaculture has increased since that time to meet increasing demand. Evidence suggests
 751 that expanded seafood production will rely on the continuation of a rapid increase in aquaculture
 752 output worldwide, projected at 33 percent increase by 2021, which will add 15 percent to the
 753 total supply of seafood.²⁰ Distributed evenly to the world's population, this capacity could in
 754 principle meet Dietary Guidelines recommendations for consumption of at least 8 ounces of
 755 seafood per week. Concern exists that the expanded capacity may be for low-trophic level
 756 seafood that has relatively low levels of EPA and DHA compared to other species. Under the
 757 current production, Americans who seek to meet U.S. Dietary Guidelines recommendations must
 758 rely on significant amounts of imported seafood (~90 percent). **DGAC Grade: Moderate**

759
 760 **Implications**

761
 762 Both wild and farmed seafood are major food sources available to support DGAC
 763 recommendations to regularly consume a variety of seafood. Responsible stewardship over
 764 environmental impact will be important as farmed seafood production expands. Availability of
 765 these important foods is critical for future generations of Americans to meet their needs for a
 766 healthy diet. Therefore, strong policy, research, and stewardship support are needed to
 767 increasingly improve the environmental sustainability of farmed seafood systems. From the
 768 standpoint of the dietary guidelines this expanded production needs to be largely in EPA and
 769 DHA rich species and supporting production of low-trophic level species of similar nutrient
 770 density as wild-caught.

771
 772 **Review of the Evidence**

773 The UN FAO report on *The State of World Fisheries and Agriculture* issued in 2012 formed the
 774 basis of the DGAC's evidence review on this topic.²⁰ The FAO report addresses a wide variety
 775 of issues affecting capture fisheries and aquaculture, including economics, infrastructure, and

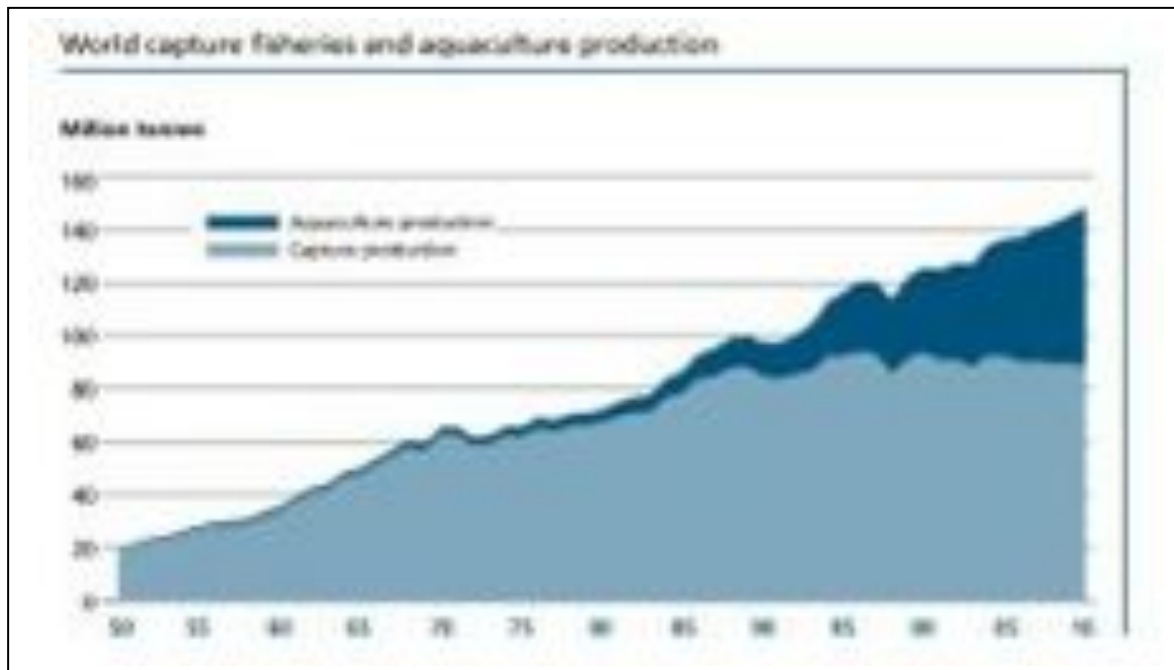
776 labor and government policies. The DGAC focused on matters that directly address the world
 777 production of one important food—seafood—as a first attempt by a DGAC committee to
 778 consider the implications of dietary guidelines for production of a related group of foods.
 779

780 The production of capture fisheries has remained stable at about 90 million tons from 1990-2011
 781 (Figure D5.3).²⁰ At the same time, aquaculture production is rising and will continue to increase.
 782 FAO model projections indicate that in response to the higher demand for seafood, world
 783 fisheries and aquaculture production is projected to grow by 15 percent between 2011 and 2021.
 784 This increase will be mainly due to increased aquaculture output, which is projected to increase
 785 33 percent by 2021, compared with only 3 percent growth in wild capture fisheries over the same
 786 period. It is predicted that aquaculture will remain one of the fastest growing animal food-
 787 producing sectors and will exceed that of beef, pork, or poultry. Aquaculture production is
 788 expected to expand on all continents with variations across countries and regions in terms of the
 789 seafood species produced. Currently, the United States is the leading importer of seafood
 790 products world-wide, with imports making up about 90 percent of seafood consumption.
 791 Continuing to meet Americans needs for seafood will require stable importation or substantial
 792 expansion of domestic aquaculture.

793

794 **Figure D5.3. Comparison of fishery production and aquaculture, 1950-2010**

795



812

813 **For additional details on this body of evidence, visit:** UN FAO report on *The State of World*
 814 *Fisheries and Agriculture, 2012*. Available at <http://www.fao.org/fishery/sofia/en>

815

816 **FOOD SAFETY**

817 The DGAC reviewed evidence of food safety topics was limited to usual coffee/caffeine
 818 consumption, high dose caffeine consumption, and aspartame. Coffee is one of the most widely
 819 consumed beverages in the U.S. and represents a major source of caffeine.⁶³ The effects of
 820 coffee/caffeine consumption have not been evaluated by any prior DGAC. The Committee
 821 reviewed the evidence on normal and excessive coffee/caffeine intake and health outcomes. In
 822 addition, the DGAC reviewed evidence on health outcomes and aspartame; the most widely used
 823 nonnutritive sweetener.

824
 825 Given the importance of food-borne illness prevention, the Committee reviewed the 2010 DGAC
 826 report content related to consumer behaviors and updated the key food safety behavior
 827 principles.

828

829 **Question 5: What is the relationship between usual coffee/caffeine consumption**
 830 **and health?**

831 **Source of Evidence:** Overview of systematic reviews and meta-analyses

832

833 **Coffee/Caffeine and Chronic Disease**

834 **Conclusion**

835 Strong and consistent evidence shows that consumption of coffee within the moderate range (3 to
 836 5 cups/d or up to 400 mg/d caffeine) is not associated with increased risk of major chronic
 837 diseases, such as cardiovascular disease (CVD) and cancer and premature death in healthy
 838 adults. **DGAC Grade: Strong**

839

840 Consistent observational evidence indicates that moderate coffee consumption is associated with
 841 reduced risk of type 2 diabetes and cardiovascular disease in healthy adults. In addition,
 842 consistent observational evidence indicates that regular consumption of coffee is associated with
 843 reduced risk of cancer of the liver and endometrium, and slightly inverse or null associations are
 844 observed for other cancer sites. **DGAC Grade: Moderate**

845

846 **Implications**

847 Moderate coffee consumption can be incorporated into a healthy lifestyle, along with other
 848 behaviors, such as refraining from smoking, consuming a nutritionally balanced diet, maintaining
 849 a healthy body weight, and being physically active. However, it should be noted that coffee, as it
 850 is normally consumed, frequently contains added calories from cream, milk, and added sugars.
 851 Care should be taken to minimize these caloric additions. Furthermore, individuals who do not
 852 consume caffeinated coffee should not start to consume it for health benefits alone.

853 **Review of the Evidence**

854 ***Total Mortality***

855 Evidence suggests a significant inverse relationship between coffee consumption of 1 to 4
 856 cups/day with total mortality, especially CVD mortality. This evidence is based on three meta-
 857 analyses of more than 20 prospective cohort studies.⁶⁴⁻⁶⁶ In general, results were similar for men
 858 and women. The risk reduction associated with each cup of coffee per day was between 3 to 4
 859 percent. In addition, Je and Giovannucci found a significant inverse association between coffee
 860 consumption and CVD mortality.⁶⁵ This association was stronger in women (16 percent lower
 861 risk) than in men (8 percent lower risk). However, no association was found for cancer mortality.
 862 Crippa et al. found that the lowest risk was observed for 4 cups/day for all-cause mortality (16%,
 863 95% CI: 13, 18) and 3 cups/day for CVD mortality (21%, 95% CI: 16, 26).⁶⁴

864

865 ***Cardiovascular Disease***

866 A large and current body of evidence directly addressed the relationship between normal coffee
 867 consumption and risk of CVD. The evidence included 12 systematic reviews with meta-analyses,
 868 all of which had high quality ratings (AMSTAR scores 8/11 – 11/11). CVD incidence and
 869 mortality, as well as CHD, stroke, heart failure, and hypertension were assessed by meta-
 870 analyses that consisted primarily of prospective cohort studies. Intermediate outcomes such as
 871 blood pressure, blood lipids, and blood glucose were assessed by meta-analyses of randomized
 872 controlled trials.

873

874 CVD risk was assessed by a current meta-analysis of 36 prospective cohort studies on long-term
 875 coffee consumption.⁶⁷ This analysis showed a non-linear association, such that the lowest risk of
 876 CVD was seen with moderate coffee consumption (3 to 5 cups/day), but higher intakes (>5
 877 cups/day) were neither protective nor harmful. Overall, moderate consumption of caffeinated,
 878 but not decaffeinated, coffee was associated with a 12 percent lower risk of CVD.

879

880 Results from the assessment of CHD risk in three meta-analyses were not entirely consistent.⁶⁷⁻⁶⁹
 881 Ding et al. found 10 percent lower CHD risk with moderate coffee consumption (3 to 5
 882 cups/day) in a meta-analysis of 30 prospective cohort studies, whereas Wu et al. and Sofi et al. in
 883 meta-analyses of 21 and 10 prospective cohort studies, respectively, found no association
 884 between coffee consumption and CHD risk.⁶⁷⁻⁶⁹ However, in sub-group analysis, Wu et al.
 885 found that habitual moderate coffee consumption (1 to 4 cups/day) was associated with an 18
 886 percent lower risk of CHD among women.⁶⁹ Overall, the meta-analyses of Sofi et al. and Wu et
 887 al. were conducted with smaller bodies of evidence and Ding et al. assessed several more recent
 888 studies.⁶⁷⁻⁶⁹ Of note, coffee brewing methods have changed over time and the filter method has
 889 become more widely used, replacing unfiltered forms of coffee such as boiled coffee that were
 890 more widely reported by participants in earlier studies. Thus, the findings by Ding et al. are
 891 more up to date, reflecting health effects of coffee consumed in recent cohorts.

892 Risk of stroke was assessed in two systematic reviews with meta-analyses of prospective cohort
 893 studies with consistent findings.^{70, 71} Kim et al. found that coffee intake of 4 or more cups/day
 894 had a protective association on risk of stroke.⁷⁰ Larsson et al. documented a non-linear
 895 association such that coffee consumption ranging from 1 to 6 cups/day was associated with an 8
 896 percent to 13 percent lower risk of stroke, and higher intakes were not associated with decreased
 897 or increased risk.⁷¹ The inverse associations were limited to ischemic stroke and no association
 898 was seen with hemorrhagic stroke.

899
 900 Regarding blood pressure, three meta-analyses evaluated the effect of coffee and caffeine on
 901 systolic and diastolic blood pressure using controlled trials.⁷²⁻⁷⁴ The most recent meta-analysis of
 902 10 randomized controlled trials by Steffen et al. showed no effect of coffee on either systolic or
 903 diastolic blood pressure. Similarly, in another meta-analysis of 11 coffee trials and 5 caffeine
 904 trials, caffeine doses of <410 mg/day had no effect on systolic and diastolic blood pressure,
 905 while doses of 410 or more mg/day resulted in a net increase.⁷³ A third meta-analysis showed
 906 that among individuals with hypertension, 200 to 300 mg of caffeine (equivalent to ~2 to 3 cups
 907 filtered coffee) resulted in an acute increase of systolic and diastolic blood pressure.⁷²
 908 Additionally, two meta-analyses quantified the effect of coffee on incidence of hypertension^{74, 75}
 909 and found no association between habitual coffee consumption and risk of hypertension.
 910 However, Zhang et al. documented a slightly elevated risk for light to moderate consumption (1
 911 to 3 cups/day) of coffee compared to less than 1 cup/day.⁷⁵

912
 913 Regarding blood lipids, meta-analyses of short-term randomized controlled trials revealed that
 914 coffee consumption contributed significantly to an increase in total cholesterol and LDL-
 915 cholesterol, but cholesterol-raising effects were primarily limited to unfiltered coffee and filtered
 916 coffee appeared to have minimal effects on serum cholesterol levels.^{76, 77}

917
 918 In a meta-analysis of observational study data, including prospective, retrospective, and case-
 919 control studies, higher amounts of coffee or caffeine had no association with risk of atrial
 920 fibrillation, but low doses of caffeine (<350 mg/day) appeared to have a protective association.⁷⁸
 921 In addition, coffee consumption of 1 to 5 cups/day was found to be inversely associated with risk
 922 of heart failure in a meta-analysis of five prospective studies.⁷⁹ A non-linear association was
 923 documented and the lowest risk was observed for 4 cups/day.⁷⁹

924 925 ***Type 2 Diabetes***

926 Coffee consumption has consistently been associated with a reduced risk of type 2 diabetes. In
 927 four meta-analyses of prospective cohort studies⁸⁰⁻⁸³ and cross-sectional studies,⁸³ coffee
 928 consumption was inversely associated with risk of type 2 diabetes in a dose-response manner.
 929 Compared to non-drinkers, risk for type 2 diabetes was 33 percent lower for those consuming 6
 930 cups/day in the analysis by Ding et al. while the risk was 37 percent lower for those consuming
 931 10 cups/day in the analysis by Jiang et al.^{67, 82} Using a sub-set of the prospective cohorts in the

932 Ding et al. and Jiang et al. meta-analyses, Huxley et al. documented that each cup of coffee was
 933 associated with a 7 percent lower risk of type 2 diabetes.⁸¹ Similarly, van Dam and Hu noted that
 934 consumption of ≥ 6 or ≥ 7 cups/day was associated with a 35 percent lower risk of type 2
 935 diabetes.⁸³ Three meta-analyses⁸⁰⁻⁸² also found protective associations for decaffeinated coffee.
 936 Moderate decaffeinated coffee consumption (3 to 4 cups/day) was associated with a 36 percent
 937 lower risk of type 2 diabetes.⁸¹ Each cup of decaffeinated coffee was associated with a 6 percent
 938 lower risk⁸⁰ while every 2 cups were associated with a 11 percent lower risk.⁸² Both reports also
 939 documented a dose-response association between caffeine and type 2 diabetes risk such that
 940 every 140 mg/day was associated with an 8 percent lower risk in the Ding et al. meta-analysis,
 941 while every 200 mg/day was associated with a 14 percent lower risk in the analysis by Jiang et
 942 al.^{80, 82} However, it remains unclear if this inverse association is independent of coffee
 943 consumption, as Ding et al. indicated that none of the studies included in the caffeine dose-
 944 response analysis adjusted for total coffee.

945
 946 Only one systematic review of nine randomized controlled trials examined the effects of caffeine
 947 on blood glucose and insulin concentrations among those with type 2 diabetes.⁸⁴ Ingestion of 200
 948 to 500 mg of caffeine acutely increased blood glucose concentrations by 16 to 28 percent of the
 949 area under the curve and insulin secretions by 19 to 48 percent of the area under the curve when
 950 taken before a glucose load. At the same time, these trials also noted a decrease in insulin
 951 sensitivity by 14 to 37 percent. Although no study has examined whether the effects of caffeine
 952 on blood glucose and insulin persist in the long term, evidence from prospective cohorts
 953 indicates that the acute effects of caffeine do not translate into long-term risk of type 2 diabetes.
 954 Furthermore, the inverse association between decaffeinated coffee and diabetes risk suggests that
 955 the observed benefit is likely to be due to other constituents in coffee rather than caffeine.

956 957 **Cancer**

958 Several systematic reviews and meta-analyses examined the association between coffee
 959 consumption and risk of cancer. Types of cancer examined by the DGAC included total cancer,
 960 cancers of the lung, liver, breast, prostate, ovaries, endometrium, bladder, pancreas, upper
 961 digestive and respiratory tract, esophagus, stomach, colon, and rectum.

962
 963 In a quantitative summary of 40 prospective cohort studies with an average follow-up of 14.3
 964 years, Yu et al. found a 13 percent lower risk of total cancer among coffee drinkers compared to
 965 non-drinkers or those with lowest intakes.⁸⁵ Risk estimates were similar for men and women. In
 966 sub-group analyses, the authors noted that coffee drinking was associated with a reduced risk of
 967 bladder, breast, buccal and pharyngeal, colorectal, endometrial, esophageal, hepatocellular,
 968 leukemic, pancreatic, and prostate cancers.

969
 970 Tang et al. evaluated five prospective cohorts and eight case-control studies and found that,
 971 overall, those with the highest levels of coffee consumption had a 27 percent higher risk for lung

972 cancer compared to never drinkers or those with least consumption.⁸⁶ An increase in coffee
973 consumption of 2 cups/day was associated with a 14 percent higher risk of developing lung
974 cancer. However, because smoking is an important confounder, when analyses were stratified by
975 smoking status, coffee consumption was marginally protective in non-smokers and was not
976 associated with lung cancer among smokers. When estimates from two studies that examined
977 decaffeinated coffee were summarized, a protective association with lung cancer was seen. No
978 association was seen with lung cancer when only case-control studies were considered.

979
980 Results from two meta-analyses indicate that coffee consumption is associated with a 40 to 50
981 percent lower risk of liver cancer,^{87 88} when considering both cohort and case-control studies. In
982 one meta-analysis, the associations were significant in men but not in women.⁸⁷

983
984 Three meta-analyses of observational studies found no association between coffee
985 consumption,⁸⁹⁻⁹¹ caffeine consumption, or decaffeinated coffee consumption and risk of breast
986 cancer. In all three reports, each 2 cup/day of coffee was marginally associated with a 2 percent
987 lower risk of breast cancer. However, in sub-group analyses, coffee consumption was protective
988 against breast cancer risk in postmenopausal women,⁸⁹ BRCA1 mutation carriers,⁸⁹ and women
989 with estrogen receptor negative breast tumors.⁹⁰

990
991 The association between coffee consumption and risk of prostate cancer was mixed. Cao et al.
992 and Zhong et al. found that regular or high coffee consumption, compared to non- or lowest
993 levels of consumption, was associated with a 12 percent to 17 percent lower risk of prostate
994 cancer in prospective cohort studies.^{92, 93} Further, each 2 cups of coffee per day was associated
995 with a 7 percent lower risk of prostate cancer. However, no associations were seen with case-
996 control data alone or when these studies were examined together with prospective cohort studies.
997 Using a combination of both prospective cohort and case-control data, Discacciati et al. found
998 that each 3 cups/day of coffee was associated with a 3 percent lower risk of localized prostate
999 cancer and an 11 percent lower risk of mortality from prostate cancer.⁹⁴ On the other hand, after
1000 summarizing data from 12 prospective cohort and case-control studies, Park et al. found a 16
1001 percent higher risk of prostate cancer.⁹⁵ However, in sub-group analyses by study design, the
1002 higher risk was observed in case-control but not in cohort studies.

1003
1004 Consumption of coffee was not associated with risk of ovarian cancer in a meta-analysis of seven
1005 prospective cohort studies with more than 640,000 participants.⁹⁶ Two meta-analyses confirmed
1006 an inverse association between coffee consumption and risk of endometrial cancer.^{97, 98} In the
1007 most recent and updated meta-analysis of prospective cohort and case-control studies, compared
1008 to those in the lowest category of coffee consumption, those with the highest intakes of coffee
1009 had a 29 percent lower risk of endometrial cancer.⁹⁸ Each cup of coffee per day was associated
1010 with an 8 percent lower risk of endometrial cancer. Similar results were found in the meta-
1011 analysis by Bravi et al. that included a sub-set of the studies in Je et al. and documented a 20

1012 percent lower risk of endometrial cancer overall, and a 7 percent decrease for each cup of coffee
1013 per day.^{97, 98} However, the association was significant only in case-control studies but not in
1014 cohort studies, most likely due to lower statistical power.

1015
1016 A recent meta-analysis of 23 case-control studies by Zhou et al. found coffee was a risk factor
1017 for bladder cancer. There was a smoking-adjusted increased risk of bladder cancer for those in
1018 the highest (45 percent), second highest, (21 percent), and third highest (8 percent) groups of
1019 coffee consumption, compared to those in the lowest intake group.⁹⁹ No association was,
1020 however, seen in cohort studies.

1021
1022 Two meta-analyses of coffee consumption and pancreatic cancer risk provided mixed results.^{85,}
1023 ¹⁰⁰ Using both prospective cohort and case-control studies, Turati et al. found that coffee
1024 consumption was not associated with risk of pancreatic cancer.¹⁰⁰ However, an increased risk
1025 was seen in case-control studies that did not adjust for smoking. Using a sub-set of prospective
1026 cohorts included in the Turati et al. meta-analysis, Dong et al. found that coffee drinking was
1027 inversely associated with pancreatic cancer risk but did not separate studies based on their
1028 adjustment for smoking status.¹⁰¹ Sub-group analyses revealed a protective association in men,
1029 but not in women.

1030
1031 Turati et al. quantified the association between coffee consumption and various upper digestive
1032 and respiratory tract cancers using data from observational studies.¹⁰² Coffee consumption was
1033 associated with a 36 percent lower risk of oral and pharyngeal cancer but not with risk of
1034 laryngeal cancer, esophageal squamous cell carcinoma, or esophageal adenocarcinoma. In a
1035 meta-analysis of prospective cohort and case-control studies, Zheng et al. noted that coffee was
1036 inversely, but non-significantly, associated with risk of esophageal cancer.¹⁰³ Regarding gastric
1037 cancer, no association between coffee consumption and risk was seen in a meta-analysis of
1038 observational studies by Botelho et al.¹⁰⁴

1039
1040 Three meta-analyses on the association between coffee consumption and colorectal cancer risk
1041 have yielded mixed findings.¹⁰⁵⁻¹⁰⁷ Results from case-control studies suggested coffee
1042 consumption was associated with lower risk of colorectal (15 percent lower) and colon cancer
1043 (21 percent lower), especially in women. However, this inverse association was non-significant
1044 for cohort studies. Using all but one of the case-control studies, Galeone et al. arrived at similar
1045 conclusions as a Li et al. analysis, although associations were in general stronger.^{105, 107} Galeone
1046 et al. also provided suggestive evidence for a dose-response relationship between coffee and
1047 colorectal cancer such that each cup of coffee was associated with a 6 percent lower risk of
1048 colorectal cancer, 5 percent lower risk of colon cancer, and 3 percent lower risk of rectal
1049 cancer.¹⁰⁵ Using several prospective cohort studies, as in the Li et al. meta-analysis, Je et al.
1050 found no significant association of coffee consumption with risk of colorectal cancer.^{106, 107}
1051 Interestingly, no differences were seen by sex but the suggestive inverse associations were

1052 slightly stronger in studies that adjusted for smoking and alcohol.

1053

1054 *For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio,*

1055 *Appendix E-2.39b Systematic Review/Meta-Analysis Data Table,* and References 64-107

1056

1057 **Caffeine and Neurodegenerative Disease**

1058 **Conclusion**

1059 Consistent evidence indicates an inverse association between caffeine intake and risk of

1060 Parkinson's disease. **DGAC Grade: Moderate**

1061

1062 Limited evidence indicates that caffeine consumption is associated with a modestly lower risk of

1063 cognitive decline or impairment and lower risk of Alzheimer's disease. **DGAC Grade: Limited**

1064

1065 **Implications**

1066 Moderate coffee consumption can be incorporated into a healthy lifestyle, along with other

1067 behaviors, such as refraining from smoking, consuming a nutritionally balanced diet, maintaining

1068 a healthy body weight, and being physically active. However, it should be noted that coffee as it

1069 is normally consumed can contain added calories from cream, milk, and added sugars. Care

1070 should be taken to minimize these caloric additions. Furthermore, individuals who do not

1071 consume caffeinated coffee should not start to consume it for health benefits alone.

1072

1073 **Review of the Evidence**

1074 ***Parkinson's Disease***

1075 Evidence from two systematic reviews^{108, 109} and one quantitative meta-analysis¹¹⁰ confirmed an

1076 inverse association between coffee, caffeine, and risk of Parkinson's disease. Qi et al. evaluated

1077 six case-control studies and seven prospective articles and documented a non-linear relationship

1078 between coffee and risk of Parkinson's disease, overall.¹¹⁰ The lowest risk was observed at about

1079 3 cups/day (smoking-adjusted risk reduction was 28 percent). For caffeine, a linear dose-

1080 response was found and every 200 mg/day increment in caffeine intake was associated with a 17

1081 percent lower risk of Parkinson's disease. Using a combination of cohort, case-control, and

1082 cross-sectional data, Costa et al. summarized that the risk of Parkinson's disease was 25 percent

1083 lower among those consuming the highest versus lowest amounts of caffeine.¹⁰⁸ Like Qi et al.,

1084 Costa et al. documented a linear dose-response with caffeine intake such that every 300 mg/day

1085 was associated with a 24 percent lower risk of Parkinson's disease. In both reports, associations

1086 were weaker among women than in men.

1087

1088 **Cognition**

1089 Two systematic reviews^{111, 112} and one meta-analysis¹¹² examined the effects of caffeine from
1090 various sources, including coffee, tea, and chocolate, on cognitive outcomes. Arab et al.
1091 systematically reviewed six longitudinal cohort studies evaluating the effect of caffeine or
1092 caffeine-rich beverages on cognitive decline.¹¹¹ Most studies in this review used the Mini Mental
1093 State Examination Score as a global measure of cognitive decline. The review concluded that
1094 estimates of cognitive decline were lower among caffeine consumers, although there was no
1095 clear dose-response relationship. Studies also showed stronger associations among women than
1096 men. In a meta-analysis of nine cohort and two case-control studies, caffeine intake from various
1097 sources was associated with a 16 percent lower risk of various measures of cognitive
1098 impairment/decline. Specifically, data from four studies indicate that caffeine is associated with a
1099 38 percent lower risk of Alzheimer’s disease.

1100

1101 *For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio,*
1102 *Appendix E-2.39b Systematic Review/Meta-Analysis Data Table, and References 108-112*

1103

1104 **Caffeine and Pregnancy Outcomes**

1105 **Conclusion**

1106 Consistent evidence from observational studies indicates that moderate caffeine intake in
1107 pregnant women is not associated with risk of preterm delivery. **DGAC Grade: Moderate**

1108

1109 Higher caffeine intake is associated with a small increased risk of miscarriage, stillbirth, low
1110 birth weight, and small for gestational age (SGA) births. However, these data should be
1111 interpreted cautiously due to potential recall bias in the case-control studies and confounding by
1112 smoking and pregnancy signal symptoms. The DGAC recognizes that there is limited data to
1113 identify a level of caffeine intake beyond which risk increases. Based on the existing data, the
1114 risk of miscarriage, stillbirth, low birth weight, and SGA births is minimal given the average
1115 caffeine intake of pregnant women in the United States. **DGAC Grade: Limited**

1116

1117 **Implications**

1118 Overall, the evidence supports current recommendations to limit caffeine intake during
1119 pregnancy as a precaution. Based on existing evidence, women who are pregnant or planning to
1120 become pregnant should be cautious and adhere to current recommendations of the American
1121 Congress of Obstetricians and Gynecologists regarding caffeine consumption, and not consume
1122 more than 200 mg caffeine per day (approximately two cups of coffee per day).

1123

1124 **Review of the Evidence**

1125 Two SRs/MA assessed observational studies on the association of caffeine intake with adverse
1126 pregnancy outcomes.^{113, 114} The pregnancy outcomes included miscarriage, pre-term birth,
1127 stillbirth, SGA, and low birth weight. The most recent SR/MA by Greenwood et al. quantified
1128 the association between caffeine intake and adverse pregnancy outcomes from 60 publications
1129 from 53 separate cohort (26) and case-control (27) studies.¹¹³ The evidence covered a variety of
1130 countries with caffeine intake categories that ranged from non-consumers to those consuming
1131 more than 1,000 mg/day. They found that an increment of 100 mg caffeine was associated with a
1132 14 percent increased risk of miscarriage, 19 percent increased risk of stillbirth, 10 percent
1133 increased risk of SGA, and 7 percent increased risk of low birth weight. The risk of pre-term
1134 delivery was not increased significantly. The magnitude of these associations was relatively
1135 small within the range of caffeine intakes of the majority women in the study populations, and
1136 the associations became more pronounced at higher range (≥ 300 mg/day). The authors also note
1137 the substantial heterogeneity observed in the meta-analyses shows that interpretation of the
1138 results should be cautious. In addition, the results from prospective cohort studies and case-
1139 control studies were mixed together. Because coffee consumption is positively correlated with
1140 smoking, residual confounding by smoking may have biased the results toward a positive
1141 direction.

1142
1143 The other SR/MA assessed pre-term birth and the results were in agreement with Greenwood et
1144 al.¹¹³ Maslova et al. reviewed 22 studies (15 cohort and 7 case-control studies) and found no
1145 significant association between caffeine intake and risk of pre-term birth in either case-control or
1146 cohort studies.¹¹⁴ For all of the observational studies assessed across the SRs/MA, most studies
1147 did not adequately adjust for the pregnancy signal phenomenon, i.e. that nausea, vomiting, and
1148 other adverse symptoms are associated with a healthy pregnancy that results in a live birth,
1149 whereas pregnancy signal symptoms occur less frequently when the result is miscarriage. Coffee
1150 consumption decreases with increasing pregnancy signal symptoms, typically during the early
1151 weeks of pregnancy, and this severely confounds the association.¹¹⁵ Greenwood et al. state that
1152 this potential bias is the most prominent argument against a causal role for caffeine in adverse
1153 pregnancy outcomes.¹¹³ Only one randomized controlled trial of caffeine/coffee reduction during
1154 pregnancy has been conducted to date.¹¹⁶ The study found that in pregnant women who
1155 consumed at least three cups of coffee a day and were less than 20 weeks pregnant, a reduction
1156 of 200 mg of caffeine intake (~ 2 cups) per day did not significantly influence birth weight or
1157 length of gestation, compared to those with no decrease in caffeine consumption. The trial did
1158 not examine other outcomes.

1159
1160 ***For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio,***
1161 ***Appendix E-2.39b Systematic Review/Meta-Analysis Data Table,*** and References 113, 114

1162
1163

1164 **Question 6: What is the relationship between high-dose coffee/caffeine**
1165 **consumption and health?**

1166 **Source of Evidence:** Systematic reviews^{117, 118}

1167

1168 **Conclusion**

1169 Evidence on the effects of excessive caffeine intake on the health of adults or children (>400
1170 mg/day for adults; undetermined for children and adolescents) is limited. Some evidence links
1171 high caffeine intake in the form of energy drinks to certain adverse outcomes, such as caffeine
1172 toxicity and cardiovascular events. Randomized controlled trials (RCTs) on the relationship
1173 between high-caffeine energy drinks and cardiovascular risk factors and other health outcomes
1174 report mixed results. Evidence also is limited on the health effects of mixing alcohol with energy
1175 drinks, but some evidence suggests that energy drinks may mask the effects of alcohol
1176 intoxication, so an individual may drink more and increase their risk of alcohol-related adverse
1177 events. **DGAC Grade: Limited**

1178

1179 **Implications**

1180 Early safety signals consisting of case reports of adverse events associated with high-caffeine
1181 drink consumption, including increased emergency room visits, indicate a potential public health
1182 problem. The DGAC agrees with the American Academy of Pediatrics and the American
1183 Medical Association that until safety has been demonstrated, limited or no consumption of high-
1184 caffeine drinks, or other products with high amounts of caffeine, is advised for vulnerable
1185 populations, including children and adolescents. High-caffeine energy drinks and alcoholic
1186 beverages should not be consumed together, either mixed together or consumed at the same
1187 sitting. This is especially true for children and adolescents.

1188 **Background**

1189 According to the FDA, the upper limit of moderate caffeine intake in healthy adult populations
1190 (barring pregnant women) is 400 mg/day, with intakes higher than this being considered
1191 excessive caffeine consumption. The FDA has not defined moderate and excessive intake levels
1192 for children and adolescents. However, according to Health Canada, children should not
1193 consume more than 2.5 mg of caffeine per kg bodyweight per day.¹¹⁹ Although this guideline
1194 pertains only to children up to the age of 12 years, in the literature it is usually applied to
1195 children and adolescents of all ages. A caffeine threshold of 2.5 mg/kg/day would translate into
1196 around 37.5 mg/day for children ages 2 to 5 years with an average weight of 15 kg, 75 mg/day
1197 for youth ages 6 to 12 years with an average weight of 30 kg, and 137.5 mg/day for youth ages
1198 13 to 17 years with an average weight of 55 kg.

1199

1200 The main sources of caffeine among both adults and children are coffee, tea, and carbonated soft
1201 drinks. Another product, which has received a lot of attention recently as a potential source of
1202 excessive caffeine intake, especially among younger populations, is energy drinks.¹²⁰ An energy
1203 drink is a beverage that contains caffeine as its active ingredient, along with other ingredients
1204 such as taurine, herbal supplements, vitamins, and sugar. It is usually marketed as a product that
1205 can improve energy, stamina, athletic performance, or concentration.¹²¹ Energy drinks are
1206 relatively new to the market and have evaded oversight and regulation by the FDA due to their
1207 classification as dietary supplements, or because their components are generally recognized as
1208 safe.¹²¹ Overall, these drinks are highly variable in caffeine content and some products have
1209 excessively high caffeine content (from 50 to 505 mg per can/bottle, with caffeine concentrations
1210 anywhere between 2.5 to 171 mg per fluid ounce).¹²²

1211
1212 Health organizations including the American Academy of Pediatrics, the International Society of
1213 Sports Nutrition, and the American Medical Association have issued position statements on
1214 energy drinks, advising limited or no consumption among children and adolescents. Given the
1215 increasing evidence pointing toward harmful effects of excessive caffeine consumption,¹⁰⁵⁻¹⁰⁷
1216 the FDA requested the IOM to convene a workshop examining the science behind safe levels of
1217 caffeine intake. A report summarizing this workshop was recently published.¹²³ Its main
1218 conclusions were: 1) Children and adolescents are a potential vulnerable group, in whom
1219 caffeine intake could have detrimental health consequences. This is particularly important given
1220 insufficient data on caffeine consumption in this demographic, which is increasingly getting
1221 exposed to new modes of caffeine intake such as energy drinks, 2) not enough is understood
1222 about potential interactions between caffeine and other ingredients commonly found in caffeine-
1223 containing foods and beverages, and 3) more research is needed to identify individual differences
1224 in reactions to caffeine, especially in vulnerable populations, including children with underlying
1225 heart conditions and individuals with genetic predispositions to heart conditions.

1226
1227 The Center for Disease Control (CDC) recently reported on trends in caffeine intake over the
1228 past decade (1999-2010) among U.S. children, adolescents, and young adults.¹²⁴ The CDC found
1229 that although energy drinks were not widely available before 1999, energy drinks made up nearly
1230 6 percent of caffeine intake in 2009-2010, indicating fast growth in U.S. consumption over a
1231 short period of time. When energy drink consumption was assessed in a nationally representative
1232 sample of U.S. secondary school students,¹²⁵ 35 percent of 8th graders, 30 percent of 10th graders,
1233 and 31 percent of 12th graders consumed energy drinks or shots, and consumption was higher for
1234 adolescent boys than girls. Furthermore, energy drink use was associated with higher prevalence
1235 of substance use, as assessed for all grades of U.S. secondary students.

1236
1237 Furthermore, a serious issue of public health concern has been the popular trend of combining
1238 energy drinks with alcoholic beverages. In 2010, the FDA determined that caffeine added to
1239 alcoholic beverages was not generally recognized as safe (GRAS), leading to withdrawal of

1240 premixed, caffeinated alcoholic beverages from the market.¹²⁶ Currently, Health Canada caps
1241 caffeine levels for energy drinks at 100 mg/250 ml (~1 cup) and has determined that an energy
1242 drink container that cannot be resealed be treated as a single-serving container, because the total
1243 volume is usually consumed. They also have mandated that manufacturers add a warning to
1244 labels that energy drinks should not be combined with alcohol. Recently, the CDC has made
1245 public statements on the dangers of mixing alcohol and energy drinks. They indicate that high
1246 amounts of caffeine in energy drinks can mask the intoxicating effects of alcohol, while at the
1247 same time having no effect on the metabolism of alcohol by the liver. Therefore, high amounts of
1248 caffeine in energy drinks may result in an “awake” state of intoxication, thus increasing the risk
1249 of alcohol-related harm and injury (<http://www.cdc.gov/alcohol/fact-sheets/cab.htm>, March
1250 2014).¹²⁷

1251

1252 **Review of the Evidence**

1253 Several case reports of adverse events related to energy drink use have been published. A recent
1254 systematic review of case reports of adverse cardiovascular events related to consumption of
1255 energy drinks documented 17 such published case reports.¹¹⁸ The cardiovascular events
1256 documented included atrial fibrillation, ventricular fibrillation, supraventricular tachycardia,
1257 prolonged QT, and ST elevation. In 41 percent of the cases, the person had consumed large
1258 amounts of energy drinks, and 29 percent of the cases were associated with consumption of
1259 energy drinks together with alcohol or other drugs. In 88 percent of the cases, no underlying
1260 cardiac condition was found that could potentially explain the cardiovascular event, although
1261 other cardiovascular risk factors co-occurred with energy drink consumption before the onset of
1262 the event in most cases. Of the cases that presented with serious adverse events, including
1263 cardiac arrest, the majority occurred with either acute heavy consumption of energy drinks or
1264 consumption in combination with alcohol or other drugs. Overall, the authors concluded that
1265 causality cannot be inferred from this case series, but physicians should routinely inquire about
1266 energy drink consumption in relevant cases and vulnerable consumers should be cautioned
1267 against heavy consumption of energy drinks or concomitant alcohol (or drug) ingestion. This
1268 systematic review is consistent with a recent report from the Drug Abuse Warning Network
1269 (DAWN) on energy drink-related emergency room visits that showed U.S. emergency room
1270 visits temporally related to energy drink consumption doubled between 2007 and 2011.¹²⁸ These
1271 visits were attributed mainly to adverse reactions to energy drinks, but also to combinations with
1272 alcohol or drugs. It is generally agreed that adverse events associated with energy drink
1273 consumption are underreported.

1274

1275 Several short-term RCTs have examined the health effects of energy drink consumption. All of
1276 these have been carried out in adult populations, probably due to ethical constraints in providing
1277 energy drinks to children. Burrows et al. recently published a systematic review of RCTs
1278 examining this question.¹¹⁷ They found 15 such RCTS, examining the effect of variable doses of

1279 energy drinks (mean dose: one and a half 250 ml cans per study session) with differing
 1280 ingredient combinations and concentrations on a number of different health outcomes. The high
 1281 variability in exposure and outcome definitions made a meta-analysis infeasible. Overall, they
 1282 found no consistent effects of energy drinks on cardiorespiratory outcomes (heart rate,
 1283 arrhythmias, blood pressure), pathological outcomes (blood glucose, blood lactate, free fatty
 1284 acids, clinical safety markers), and body composition, with some studies showing positive, some
 1285 inverse, and some no associations. For many of these outcomes, consistent results could not be
 1286 stated due to only one study reporting on them. There was a slight indication of a potential
 1287 positive effect of energy drinks on physiological outcomes (run time to exhaustion, peak oxygen
 1288 uptake, resting energy expenditure). However, the authors concluded that more studies were
 1289 needed before arriving at a definitive conclusion. Two of the studies assessed the simultaneous
 1290 ingestion of alcohol and energy drinks.^{129, 130} One found that when compared with the ingestion
 1291 of alcohol alone, the addition of an energy drink reduced individuals' perception of impairment
 1292 from alcohol, while at the same time, objective measures indicated ongoing deficits in motor
 1293 coordination and visual acuity.¹²⁹ Nor did energy drinks reduce breath alcohol concentration,
 1294 indicating no change or increase in alcohol metabolism by the liver. Another study on energy
 1295 drinks in combination with alcohol and exercise showed that during post-exercise recovery there
 1296 was no effect on arrhythmias within 6 hours of energy drink ingestion in healthy young adults.¹³⁰

1297
 1298 Many of these studies have methodological limitations, such as lack of a true control group
 1299 (water or no drink), a very short follow-up duration of only a few hours, and small sample sizes,
 1300 which could explain the inconsistent findings. In addition, many of these studies did not report
 1301 whether they were commercially funded. Several of those that did report funding sources had
 1302 financial conflicts of interest. Lastly, the doses of energy drinks used in these studies were not
 1303 too high, resulting in caffeine intake levels that fell within the normal range. It is possible that
 1304 excessive caffeine intake due to heavy energy drink consumption adversely affects several health
 1305 outcomes, but this hypothesis was not clearly addressed by these studies. Hence it is difficult to
 1306 ascertain the impact of excessive caffeine intake on health outcomes on the basis of these RCTs.
 1307 In addition, very little data are available on the health effects of excessive caffeine consumption
 1308 in pediatric populations.

1309
 1310 *For additional details on this body of evidence, visit: Appendix E-2.40 Evidence Portfolio and*
 1311 *References 117, 118*

1312
 1313

1314 **Question 7: What is the relationship between consumption of aspartame and**
 1315 **health?**

1316 **Source of Evidence:** *Scientific Opinion on the re-evaluation of aspartame (E 951) as a food*
 1317 *additive (2013), European Food Safety Authority (EFSA) Panel on Food Additives and Nutrient*
 1318 *Sources added to Food*²⁹

1319 **Conclusion**

1320 The DGAC generally concurs with the European Food Safety Authority (EFSA) Panel on Food
1321 Additives that aspartame in amounts commonly consumed is safe and poses minimal health risk
1322 for healthy individuals without phenylketonuria (PKU). **DGAC Grade: Moderate**

1323
1324 Limited and inconsistent evidence suggests a possible association between aspartame and risk of
1325 some hematopoietic cancers (non-Hodgkin lymphoma and multiple myeloma) in men, indicating
1326 the need for more long-term human studies. In addition, limited and inconsistent evidence
1327 indicates a potential for risk of preterm delivery. Due to very limited evidence it is not possible
1328 to draw any conclusions on the relationship between aspartame consumption and headaches.

1329 **DGAC Grade: Limited**

1330

1331 **Implications**

1332 If individuals choose to drink beverages that are sweetened with aspartame, they should stay
1333 below the aspartame Acceptable Daily Intake (ADI) of no more than 50 mg/kg/day (a 12-ounce
1334 diet beverage contains approximately 180 mg of aspartame).¹³¹ To be cautious, adults and
1335 children should be aware of the amount of aspartame they are consuming, given the need for
1336 more long-term human studies. Currently, most Americans are well below the ADI.¹³²

1337

1338 **Background**

1339 Aspartame is the most common low-calorie sweetener used in the United States. It is found in
1340 numerous dietary sources. Although most commonly associated with low-calorie/low-sugar
1341 versions of carbonated and non-carbonated beverages, it also is found in low-calorie/low-sugar
1342 versions of canned fruits and juices; instant cereals; baked goods; ice cream and frozen ices;
1343 candy and chocolate products; jams, jellies, syrups, and condiments; yogurt; and beer. Non-
1344 nutritive sweeteners are regulated by the FDA. The FDA has concluded that aspartame is safe as
1345 a general purpose sweetener in food.¹³³ Given the high interest of the public in the safety of
1346 aspartame, the DGAC reviewed the EFSA report on the sweetener and health outcomes.

1347

1348 **Review of the Evidence**

1349 The most recent European Food Safety Authority report on the re-evaluation of aspartame as a
1350 food additive was used to address this question.²⁹ The EFSA report based its evaluation on
1351 original study reports and information submitted following public calls for data, previous
1352 evaluations, and additional literature that became available up until the end of public consultation
1353 on November 15, 2013. The DGAC focused on results from human studies, not animal studies or
1354 studies conducted *in vitro*. The Mode of Action (MoA) analysis on reproductive and

1355 developmental toxicity of aspartame also was included. Although the EFSA report considered
1356 both published and unpublished studies, the DGAC considered only published studies.

1357

1358 ***Cancer***

1359 A relatively limited body of evidence on human studies has directly addressed the relationship
1360 between aspartame consumption and cancer risk. The most consistent finding in six U.S. and
1361 European case-control studies¹³⁴⁻¹³⁹ was the absence of an adverse relationship between
1362 consumption of low-calorie sweeteners, including aspartame, and risk of some cancers. An
1363 exception was one study in Argentina that found a positive association between long-term use
1364 (≥ 10 y) of artificial sweeteners and risk of urinary tract tumors (UTT), compared to non-users;
1365 although for short-term users, no association was observed.¹³⁴

1366

1367 The findings of two prospective cohort studies^{140, 141} were not consistent. Lim et al. examined a
1368 large cohort of men and women from the NIH-AARP Diet and Health study and found no
1369 association between consumption of aspartame-containing beverages and risk of overall
1370 hematopoietic cancers, brain cancers, or their subtypes.¹⁴⁰ A second large prospective cohort
1371 study by Shernhammer et al. involved the Nurses' Health Study (NHS) and Health Professionals
1372 Follow-up Study (HPFS) cohorts followed over 22 years with dietary intake measured every 4
1373 years.¹⁴¹ In this study, the highest category of aspartame intake (≥ 143 mg/day from diet soda and
1374 aspartame packets) was associated with significantly elevated risk of non-Hodgkin lymphoma
1375 (NHL) and of multiple myeloma in men, but not in women. Both of the prospective cohort
1376 studies that addressed cancer risk had limitations regarding generalizability. The NIH-AARP
1377 cohort had an age range of 50 to 71 years and was, therefore, not generalizable to the overall
1378 adult population. Additionally, the Panel considered the positive findings in Shernhammer et al.
1379 to be preliminary and require replication in other populations because the positive association
1380 between aspartame consumption and NHL was limited to men and lacked a clear dose-response
1381 relationship.²⁹

1382

1383 Further investigation should be considered to ensure that no association exists between
1384 aspartame consumption and specific cancer risk.

1385

1386 ***Preterm Delivery***

1387 Two European cohort studies were used in this evaluation. A large prospective cohort study by
1388 Halldorsson et al.¹⁴² from the Danish National Birth Cohort investigated associations between
1389 consumption of artificially sweetened and sugar-sweetened soft drinks during pregnancy and
1390 subsequent pre-term delivery. Also, a large prospective cohort study of Norwegian women by
1391 Englund-Ögge et al.¹⁴³ investigated the relationship between consumption of artificially
1392 sweetened and sugar-sweetened soft drinks during the first 4 to 5 months of pregnancy and
1393 subsequent pre-term delivery. In addition, La Vecchia combined these two studies in a meta-
1394 analysis that the Panel considered.¹⁴⁴

1395 Regarding the Halldorsson study, significant trends in risk of pre-term delivery with increasing
 1396 consumption of artificially sweetened drinks (carbonated and non-carbonated) were found, but
 1397 not for sugar-sweetened drinks.¹⁴² In the highest exposure groups (≥ 4 servings/d) the odds ratios
 1398 relative to non-consumption were 1.78 (95% CI: 1.19-2.66) and 1.29 (95% CI: 1.05-1.59),
 1399 respectively, for carbonated and non-carbonated artificially sweetened drinks. Associations with
 1400 consumption of artificially sweetened carbonated drinks did not differ according to whether
 1401 delivery was very early (less than 32 weeks) or only moderately or late pre-term.¹⁴² The EFSA
 1402 Panel noted that the prospective design and large size of the study sample were major strengths,
 1403 and that the methods used had no important flaws.²⁹ The Panel agreed with the authors who
 1404 concluded that replication of their findings in another setting was warranted.

1405
 1406 Regarding the Englund-Ögge study, no significant trends were found in risk of pre-term delivery
 1407 with increasing consumption of artificially sweetened drinks or sugar-sweetened drinks.¹⁴³ Small
 1408 elevations of risk were observed with higher consumption of artificially sweetened soft drinks,
 1409 but after adjustment for covariates, these reached significance only when categories of
 1410 consumption were aggregated to four levels, and then the odds ratio for the highest category (≥ 1
 1411 serving/day) was 1.11 (95% CI: 1.00-1.24) compared with non-consumption. This was driven by
 1412 an increase in spontaneous but not medically induced pre-term delivery. Associations with sugar-
 1413 sweetened soft drinks tended to be stronger, with an adjusted odds ratio of 1.25 (95% CI: 1.08-
 1414 1.45) for consumption of at least 1 serving per day. The Panel noted that effects may have been
 1415 underestimated because of inaccuracies in the assessment of dietary exposures, but the method
 1416 was similar to that used by Halldorsson et al., and the same for sugar-sweetened as for artificially
 1417 sweetened soft drinks.²⁹

1418 ***Behavior and Cognition***

1420 **Children**

1421 Two RCTs^{145, 146} and two non-randomized controlled trials^{147, 148} conducted in the United States
 1422 were included in the evidence on effects of aspartame on behavior and cognition in children.
 1423 Wolraich et al. compared diets high in sucrose to diets high in aspartame in 25 preschool and 23
 1424 primary school-age children and found that even when intake exceeded typical dietary levels,
 1425 neither dietary sucrose nor aspartame affected children's behavior or cognitive function.¹⁴⁶
 1426 Shaywitz et al. examined the effect of large doses of aspartame (10 times usual consumption) on
 1427 behavioral/cognitive function in children with attention deficit disorder (ages 5 to 13 years) and
 1428 found no effect of aspartame on cognitive, attentive, or behavioral testing.¹⁴⁶ Roshon and Hagan
 1429 examined 12 preschool children on alternate experimental days with a challenge of sucrose- or
 1430 aspartame-containing drinks and found no significant differences in locomotion, task orientation,
 1431 or learning.¹⁴⁸ Lastly, Kruesi et al. investigated the effect of sugar, aspartame, saccharin, and
 1432 glucose on disruptive behavior in 30 preschool boys on four separate experimental days.¹⁴⁷ There
 1433 was no significant difference in scores of aggression or observer's ratings of behavior in
 1434 response to any of the treatments. The limitations of this evidence were that all of the trials were

1435 approximately 20 to 30 years old, all had small sample sizes, and all were conducted over the
 1436 short-term (1 day to 3 weeks). Overall, the Panel noted that no effects of aspartame on behavior
 1437 and cognition were observed in children in these studies.²⁹

1438

1439 **Adults**

1440 Seven studies on the effect of aspartame on adult behavior and cognition were included in this
 1441 body of evidence. Five RCTs, one non-randomized controlled trial, and one case-control study
 1442 were conducted in the United States. Two of these trials examined a single experimental dose of
 1443 aspartame on one day.^{149, 150} Lapierre et al. examined 15 mg aspartame/kg body weight in 10
 1444 healthy adults and found no significant differences between aspartame and placebo in cognition
 1445 or memory during the study.¹⁴⁹ Ryan-Harshman et al. tested 13 healthy adult men and found no
 1446 change in any behavioral effects measured.¹⁵⁰ A third randomized crossover trial examined 48
 1447 adults over 20 days; half of the participants were given high dose aspartame (45 mg/kg/d) and
 1448 half were given low dose aspartame (15 mg/kg/d).¹⁵¹ This study found no neuropsychologic,
 1449 neurophysiologic, or behavioral effects linked to aspartame consumption. Two trials were
 1450 conducted with pilots or college students to test cognitive abilities related to aviation tasks.^{152, 153}
 1451 In the first study, 12 pilots were given aspartame (50 mg/kg) or placebo and tested for aviation-
 1452 related information processing after a single treatment on one day. The authors detected no
 1453 performance decrements associated with exposure to aspartame. In the follow-up study, college
 1454 students were given repeated dosing of aspartame (50 mg/kg for 9 days) and tested for aviation-
 1455 related cognitive tasks. No impaired performance was observed. One non-randomized crossover
 1456 trial examined the effects of aspartame on mood and well-being in 120 young college women
 1457 and found no difference in changes in mood after consuming a 12-ounce water or aspartame-
 1458 sweetened beverage on a single day.¹⁵⁴ Lastly, a case-control study was conducted with 40 adults
 1459 with unipolar depression and a similar number of subjects without a psychiatric history.¹⁵⁵
 1460 Participants were given aspartame (30 mg/kg) or placebo for 7 days and individuals with
 1461 depression reported an increase in severity of self-scored symptoms between aspartame and
 1462 placebo; whereas the non-depressed matched subjects reported no difference. This suggested that
 1463 individuals with mood disorders may be sensitive to aspartame. Overall, the Panel noted the
 1464 limited number of participants, the short duration of the studies, and the inconsistency of the
 1465 reporting of the results in all adult studies. However, despite these limitations, the Panel
 1466 concluded that there was no evidence that aspartame affects behavior or cognitive function in
 1467 adults.²⁹

1468

1469 ***Other (Headaches, Seizures)***

1470 Several studies examined headaches and seizures. A number of RCTs were conducted to assess
 1471 the incidence of headache after consumption of aspartame. One RCT tested the effects of
 1472 aspartame within 24 hours of consumption (30 mg/kg) on 40 subjects with a history of headache
 1473 and found no difference in the incidence rate of headaches.¹⁵⁶ Another RCT looked at the effect
 1474 of aspartame on frequency and intensity of migraine headaches in 10 subjects with medical

1475 diagnosis of migraine headaches over 4 weeks.¹⁵⁷ The authors found an increase in the frequency
1476 of migraine headaches with the aspartame treatment. In an RCT of 18 subjects with self-
1477 described sensitivity to aspartame, the participants reported headaches on 33 percent of the days,
1478 compared with 24 percent with placebo.¹⁵⁸ The authors concluded that a subset of the population
1479 may be susceptible to headaches induced by aspartame. Lastly, in a survey study of 171 patients
1480 at a headache unit, 8 percent reported that aspartame was a trigger of headaches compared to 2.3
1481 percent for carbohydrates and 50 percent for alcohol.¹⁵⁹ Overall, the Panel concluded the
1482 possible effect of aspartame on headaches had been investigated in various studies which
1483 reported conflicting results, ranging from no effect to the suggestion that a small subset of the
1484 population may be susceptible to aspartame-induced headaches.²⁹ The number of existing studies
1485 was small and not recent and several studies had high dropout rates. The Panel noted that
1486 because of the limitations of the studies, it was not possible to draw a conclusion on the
1487 relationship between aspartame consumption and headaches.

1488
1489 Several small studies assessed seizures. One RCT in children investigated whether aspartame
1490 would induce the occurrence of petit mal seizures.¹⁶⁰ Ten children were given one treatment of
1491 aspartame at the ADI of 40 mg/kg and that treatment exacerbated the number of
1492 electroencephalogram spike waves per hour for these children without a history of seizures. In a
1493 second RCT, aspartame (34 mg/kg) was administered to 10 epileptic children over 2 weeks to
1494 examine the induction of seizures.¹⁴⁵ No difference was found in the occurrence of seizures
1495 between aspartame and placebo exposure. Another RCT studied 18 subjects who claimed to have
1496 experienced epileptic seizures due to aspartame.¹⁶¹ One treatment (50 mg/kg) was administered
1497 on a single day and the authors reported no seizures or other adverse effect from aspartame
1498 treatment in this group. Overall, the Panel concluded that the available data do not provide
1499 evidence for a relationship between aspartame consumption and seizures.²⁹

1500

1501 ***Pregnancy Outcomes: Mode of Action (MoA) analysis***

1502 The EFSA Panel considered that adverse effects on reproduction and development reported for
1503 aspartame in animal studies could be attributed to the metabolite phenylalanine.²⁹ They
1504 undertook a formal Mode of Action (MoA) analysis of the putative role of phenylalanine in
1505 developmental toxicity (as seen in animal studies).

1506

1507 Risk characterization was based on comparison of plasma phenylalanine levels following
1508 aspartame administration with plasma phenylalanine levels associated with developmental
1509 effects in children born from mothers with PKU. Current clinical practice guidelines recommend
1510 PKU patients restrict dietary intake of phenylalanine to keep plasma levels below 360 μ M. The
1511 EFSA Panel noted that intakes of aspartame as a food additive could occur at the same time as
1512 other dietary phenylalanine sources. Therefore, they considered the threshold used for
1513 comparisons should be lowered to allow for simultaneous intake of aspartame with meals. So
1514 plasma phenylalanine from the diet (120 μ M) was subtracted from 360 μ M to determine the

1515 maximum safe plasma concentration of phenylalanine that can be derived from aspartame
1516 (240µM).

1517
1518 The Panel considered that given these conservative assumptions, realistic dietary intake of
1519 aspartame and the confidence intervals provided by the modeling, the peak plasma phenylalanine
1520 levels would not exceed the clinical target threshold of 240µM when a normal individual
1521 consumed aspartame at or below the current ADI of 40 mg/kg body weight/day. Therefore, the
1522 Panel concluded there would not be a risk of adverse effects on pregnancy in the general
1523 population at the current ADI.²⁹

1524
1525 *For additional details on this body of evidence, visit: Appendix E-2.41 Evidence Portfolio and*
1526 *Scientific Opinion on the re-evaluation of aspartame (E 951) as a food additive (2013),*
1527 *European Food Safety Authority (EFSA) Panel on Food Additives and Nutrient Sources added to*
1528 *Food. Available at www.efsa.europa.eu/efsajournal*

1529
1530 **Question 8: What Consumer Behaviors Prevent Food Safety Problems? (Topic**
1531 **update from 2010)**

1532 **Introduction and Methods**

1533 Food safety continues to be an issue of public health importance. Foodborne illness is a
1534 preventable, yet common issue affecting the U.S. population. Each year, approximately 1 in 6
1535 people in the U.S. population become ill, 128,000 are hospitalized, and 3,000 die of foodborne
1536 illness.¹⁶² It is critical to educate consumers and food producers on good techniques and
1537 behaviors for preventing food borne illness.

1538
1539 The 2010 DGAC conducted NEL systematic reviews for the Food Safety and Technology
1540 chapter and provided in-depth guidance on foodborne illness prevention. The 2015 DGAC
1541 reviewed the content related to consumer behavior and the prevention of food safety problems.
1542 The Committee determined that the majority of the 2010 food safety guidance was current and
1543 that only minor updates were necessary. For more information on the evidence review on food
1544 safety, refer to the DGAC 2010 report, Food Safety and Technology Section:
1545 ([http://origin.www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/DGAC/Report/D-8-](http://origin.www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/DGAC/Report/D-8-FoodSafety.pdf)
1546 [FoodSafety.pdf](http://origin.www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/DGAC/Report/D-8-FoodSafety.pdf)).

1547
1548 The four food safety principles—Clean, Separate, Cook, and Chill are the foundation of the Fight
1549 BAC![®] campaign (www.fightbac.org) and are reemphasized in this report. Data from the Centers
1550 for Disease Control and Prevention,³⁰ Food and Drug Administration,³¹ and the Food Safety and
1551 Inspection Service³² were used to update the 2010 DGAC tables on the following topics related
1552 to consumer behavior and food safety:

1553

1554 CLEAN and SEPARATE (Tables D5.1, D5.2, D5.3)

- 1555 • Techniques for hand sanitation, washing fresh produce, and preventing cross-contamination.

1556

1557 COOK and CHILL (Table D5.4)

- 1558 • Temperature control during food preparation and storage.

1559

1560 Table D5.3 includes updated guidance on preventing cross-contamination from shopping to
1561 serving foods. Table D5.4 lists recommended internal temperatures for meat, seafood, eggs, and
1562 leftovers. Additionally, Tables D5.5 and D5.6 provide recommended techniques for using food
1563 and refrigerator/freezer thermometers. Specific changes made to the 2010 tables are detailed in
1564 the footnotes of the tables.

1565
1566
1567
1568

Food Safety—Tables

Table D5.1. Recommended procedures for hand sanitation

When washing hands with soap and water:
<ul style="list-style-type: none"> • Wet your hands with clean, running water (warm or cold), turn off the tap, and apply soap.¹
<ul style="list-style-type: none"> • Lather your hands by rubbing them together with the soap. Be sure to lather the backs of your hands, between your fingers, and under your nails.²
<ul style="list-style-type: none"> • Scrub your hands for at least 20 seconds. Need a timer? Hum the “Happy Birthday” song from beginning to end twice.³
<ul style="list-style-type: none"> • Rinse your hands well under clean, running water.
<ul style="list-style-type: none"> • Dry your hands using a clean towel or air dry them.⁴
<p>If soap and clean, running water are not available, use an alcohol-based hand sanitizer that contains at least 60% alcohol⁵. Hand sanitizers are not as effective when hands are visibly dirty or greasy.⁶ How do you use hand sanitizer:⁷</p>
<ul style="list-style-type: none"> • Apply the product to the palm of one hand (read the label to learn the correct amount).
<ul style="list-style-type: none"> • Rub your hands together.
<ul style="list-style-type: none"> • Rub the product over all surfaces of your hands and fingers until your hands are dry.

1569
1570
1571
1572
1573
1574
1575
1576
1577
1578
1579
1580
1581
1582
1583
1584
1585
1586

Updates to the 2010 DGAC table

¹ Water temperature “warm or cold” and a conservation recommendation of ‘turn off the tap’ were added.

² The soap is to be help while lathering one’s hands, then rub all together. “Scrub all surfaces” was clarified to “the backs of hands, between fingers, and under nails.”

³ “At least” was added to the 20 seconds time frame. To give a time reference, the suggestion to “ hum the Happy Birthday song...” was added.

⁴ The word ‘paper’ was removed as a modifier for towel, and instead it was specified to be a ‘clean’ towel. The option to ‘air dry them’ was added and the option of using an air dryer was removed from the phrase. Also removed was the direction to use your paper towel to turn off the faucet.

⁵ The words ‘clean’ and ‘running’ were inserted in the directions for when water is not available. ‘Hand sanitizer that contains at least 60% alcohol’ replaces ‘gel’.

⁶ This guidance was added.

⁷ The following step was added, “Read the label to learn the correct amount.”

Source: Adapted from <http://www.cdc.gov/handwashing/when-how-handwashing.html>. Accessed June 2, 2014.³⁰

1587
1588

Table D5.2. Recommended techniques for washing produce

When preparing any fresh produce, begin with clean hands. Wash your hands for at least 20 seconds with soap and warm water before and after preparation.
Cut away any damaged or bruised areas on fresh fruits and vegetables before preparing and/or eating. Produce that looks rotten should be discarded.
Wash all produce thoroughly under running water before eating, cutting or cooking . This includes produce grown conventionally or organically at home, or purchased from a grocery store or farmer's market. Washing fruits and vegetables with soap or detergent or using commercial produce washes is not recommended.
Even if you plan to peel the produce before eating, it is still important to wash it first so dirt and bacteria are not transferred from the peel via the knife to the fruit or vegetable ¹ .
Scrub firm produce , such as melons and cucumbers, with a clean produce brush.
Dry produce with a clean cloth towel or paper towel to further reduce bacteria that may be present.
Many pre-cut, bagged, or packaged produce items like lettuce are pre-washed and ready-to-eat. If so, it will be stated on the package and you can use the product without further washing.
If you do choose to wash a product marked "pre-washed" and "ready-to-eat," be sure to use safe handling practices to avoid any cross-contamination (see Table D5.3).

1589
1590
1591

Updates to the 2010 DGAC table

¹ The following explanation was provided: ". . . so dirt and bacteria aren't transferred from the knife onto fruit or vegetable."

1592
1593

Source: Adapted from <http://www.fda.gov/downloads/Food/ResourcesForYou/Consumers/UCM174142.pdf>. Accessed June 2, 2014³¹

1594
1595
1596
1597
1598
1599
1600
1601
1602
1603
1604
1605
1606
1607
1608
1609

1610
1611
1612

Table D5.3. Recommended techniques for preventing cross-contamination

When Shopping:
Separate raw meat, poultry, and seafood from other foods in your grocery-shopping cart. Place these foods in plastic bags to prevent their juices from dripping onto other foods. It is also best to separate these foods from other foods at check out and in your grocery bags.
When Refrigerating Food¹:
Place raw meat, poultry, and seafood in containers or sealed plastic bags to prevent their juices from dripping onto other foods. Raw juices often contain harmful bacteria.
Store eggs in their original carton and refrigerate as soon as possible.
When Preparing Food:
Washing raw poultry, beef, pork, lamb, or veal before cooking it is not recommended. Bacteria in raw meat and poultry juices can be spread to other foods, utensils, and surfaces.
Wash hands and surfaces often. Harmful bacteria can spread throughout the kitchen and get onto cutting boards, utensils, and countertops. To prevent this:
<ul style="list-style-type: none"> • Wash hands with soap and warm water for 20 seconds before and after handling food, and after using the bathroom, changing diapers; or handling pets. • Use hot, soapy water and paper towels or clean cloths to wipe up kitchen surfaces or spills. Wash cloths often in the hot cycle of your washing machine. • Wash cutting boards, dishes, and counter tops with hot, soapy water after preparing each food item and before you go on to the next item. • A solution of 1 tablespoon of unscented, liquid chlorine bleach per gallon of water may be used to sanitize surfaces and utensils.
Cutting Boards:
Always use a clean cutting board.
If possible, use one cutting board for fresh produce and a separate one for raw meat, poultry, and seafood.
Once cutting boards become excessively worn or develop hard-to-clean grooves, they should be replaced.
Marinating Food:
Always marinate food in the refrigerator, not on the counter.
Sauce that is used to marinate raw meat, poultry, or seafood should not be used on cooked foods, unless it is boiled just before using.
When Serving Food:
Always use a clean plate.
Never place cooked food back on the same plate or cutting board that previously held raw food.

1613
1614
1615
1616
1617
1618
1619

Updates to the 2010 DGAC table

¹This sentence was deleted, ““When not possible, store raw animal foods below ready-to-eat foods and separate different types of raw animal foods, such as meat, poultry, and seafood from each other so that they do not cross-contaminate each other.”

Source: Adapted from <http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/washing-food-does-it-promote-food-safety/washing-food> and http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/be-smart-keep-foods-apart/ct_index Accessed June 3, 2014.³²

1620
 1621
 1622
 1623
 1624
 1625

Table D5.4. Recommended safe minimum internal temperatures

Cook to the minimum internal temperatures below, as measured with a clean food thermometer before removing meat from the heat source. For safety and quality, allow meat to rest for at least three minutes before carving or consuming. For reasons of personal preference, consumers may choose to cook meat to higher temperatures.^{1 c}

Food	Degrees Fahrenheit
Ground Meat and Meat Mixtures^a	
Beef, Pork, Veal, Lamb	160
Turkey, Chicken	165
Fresh Beef, Pork, Veal, Lamb^{a,2}	
Steaks, roasts, chops ^a	145
Poultry^a	
Chicken and Turkey, whole	165
Poultry breasts, roasts	165
Poultry thighs, wings	165
Duck and Goose	165
Stuffing (cooked alone or in bird)	165
Fresh Pork^a	160
Ham^a	
Fresh (raw) ³	145
Pre-cooked (to reheat)	140
Eggs and Egg Dishes^a	
Eggs	Cook until yolk and white are firm.
Egg dishes	160
Fresh Seafood^b	
Finfish	145
	Cook fish until it is opaque (milky white) and flakes with a fork.
Shellfish	Cook shrimp, lobster, and scallops until they reach their appropriate color. The flesh of shrimp and lobster should be an opaque (milky white) color. Scallops should be opaque (milky white) and firm.
	Cook clams, mussels, and oysters until their shells open. This means that they are done. Throw away the ones that didn't open.
	Shucked clams and shucked oysters are fully cooked when they are opaque (milky white) and firm ⁴ .
Leftovers and Casseroles^a	165

1626

1627
1628
1629
1630
1631
1632
1633
1634
1635
1636
1637
1638
1639
1640

Updates to the 2010 DGAC table

- 1 An introductory paragraph was added on the topic of allowing for a three-minute rest period after cooking meat.
- 2 Pork was added to the list of fresh meats.
- 3 Fresh (raw) ham was added to the table.
- 4 Information on cooking status of shucked clams and oysters was added.

Sources:

- ^a http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/kitchen-companion-your-safe-food-handbook/ct_index. Accessed June 3, 2014.³²
- ^b <http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm>. Accessed June 3, 2014.³¹
- ^c http://www.fsis.usda.gov/wps/wcm/connect/8e9f95a6-fd35-42d3-b6cb-b07a4b853992/Leftovers_and_Food_Safety.pdf?MOD=AJPERES. Accessed June 3, 2014.³²

Table D5.5. Recommended techniques for food thermometers

To be safe, meat, poultry, and egg ^a and seafood ^b products must be cooked to a safe minimum internal temperature to destroy any harmful microorganisms that may be in the food.
A food thermometer should also be used to ensure that cooked food is held at safe temperatures until served. Cold foods should be held at 40°F or below. Hot foods should be kept hot at 140°F or above. ^a
Most available food thermometers will give an accurate reading within 2 to 4°F. The reading will only be correct, however, if the thermometer is placed in the proper location in the food. ^a
In general, the food thermometer should be placed in the thickest part of the food, away from bone, fat, or gristle. ^a
When the food being cooked is irregularly shaped, such as with a beef roast, check the temperature in several places. Egg dishes and dishes containing ground meat and poultry should be checked in several places. ^a
When measuring the temperature of a thin food, such as a hamburger patty, pork chop, or chicken breast, a thermistor or thermocouple food thermometer should be used, if possible. ^a
However, if using an "instant-read" dial bimetallic-coil food thermometer, the probe must be inserted in the side of the food so the entire sensing area (usually 2 to 3 inches) is positioned through the center of the food. ^a
To avoid burning fingers, it may be helpful to remove the food from the heat source (if cooking on a grill or in a frying pan) and insert the food thermometer sideways after placing the item on a clean spatula or plate. ^a
Food thermometers should be washed with hot soapy water. Most thermometers should not be immersed in water. ^a

1641
1642
1643
1644
1645
1646

- Sources:** ^a http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/appliances-and-thermometers/kitchen-thermometers/ct_index, Accessed June 3, 2014.³²
- ^b <http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm>, Accessed June 3, 2014.³¹

1647
1648

Table D5.6. Recommended techniques for using refrigerator/freezer thermometers

For safety, it is important to verify the temperature of refrigerators and freezers.
Refrigerators should maintain a temperature no higher than 40°F.
Frozen food will hold its top quality for the longest possible time when the freezer maintains 0°F or below.
To measure the temperature in the refrigerator:
Put the thermometer in a glass of water and place in the middle of the refrigerator. Wait 5 to 8 hours. If the temperature is not 38 to 40°F, adjust the refrigerator temperature control. Check again after 5 to 8 hours.
To measure the temperature in the freezer:
Place the thermometer between frozen food packages. Wait 5 to 8 hours. If the temperature is not 0 to 2°F, adjust the freezer temperature control. Check again after 5 to 8 hours. An appliance thermometer can be kept in the refrigerator and freezer to monitor the temperature at all times. This can be critical in the event of a power outage. When the power goes back on, if the refrigerator is still 40°F and the freezer is 0°F or below, the food is safe ¹ .

1649
1650
1651
1652
1653
1654
1655
1656
1657
1658
1659
1660
1661
1662
1663
1664
1665
1666
1667
1668
1669
1670
1671
1672
1673
1674

Updates to the 2010 DGAC table

¹ When referring to the correct freezer temperature, ‘or below’ was added after ‘zero degrees Fahrenheit.’

Source: http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/appliances-and-thermometers/appliance-thermometers/appliance-thermometers_, Accessed June 3, 2014.³²

1675 **CHAPTER SUMMARY**

1676 Access to sufficient, nutritious, and safe food is an essential element of food security for the U.S.
 1677 population. A sustainable diet is one that assures this access for both the current population and
 1678 future generations. This chapter focused on evaluating the evidence around sustainable diets and
 1679 several topic areas of food safety.

1680
 1681 The major findings regarding sustainable diets were that a diet higher in plant-based foods, such
 1682 as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in calories and animal-
 1683 based foods is more health promoting (as discussed in *Part B. Chapter 2: 2015 DGAC Themes*
 1684 *and Recommendations: Integrating the Evidence*) and is associated with less environmental
 1685 impact than is the current U.S. diet. This pattern of eating can be achieved through a variety of
 1686 dietary patterns, including the “Healthy U.S.-style Pattern,” the “Healthy Mediterranean-style
 1687 Pattern,” and the “Healthy Vegetarian Pattern” (see *Part D. Chapter 1: Food and Nutrient*
 1688 *Intakes, and Health: Current Status and Trends* for a description of these patterns). All of these
 1689 dietary patterns are aligned with lower predicted environmental impacts and provide food
 1690 options that can be adopted by the U.S. population. Current evidence shows that the average U.S.
 1691 diet has a potentially larger environmental impact in terms of increased GHG emissions, land
 1692 use, water use, and energy use, compared to the above dietary patterns. This is because the
 1693 current U.S. population intake of animal-based foods is higher and the plant-based foods are
 1694 lower, than proposed in these three dietary patterns. Of note is that no food groups need to be
 1695 eliminated completely to improve food sustainability outcomes.

1696
 1697 A moderate amount of seafood is an important component of two of three of these dietary
 1698 patterns, and has demonstrated health benefits. The seafood industry is in the midst of rapid
 1699 expansion to meet worldwide demand, although capture fishery production has leveled off while
 1700 aquaculture is expanding. The collapse of some fisheries due to overfishing in the past decades
 1701 has raised concern about the ability to produce a safe and affordable supply. In addition, concern
 1702 has been raised about the safety and nutrient content of farm-raised versus wild-caught seafood.
 1703 To supply enough seafood to support meeting dietary recommendations, both farm-raised and
 1704 wild caught seafood will be needed. The review of the evidence demonstrated, in the species
 1705 evaluated, that farm-raised seafood has as much or more EPA and DHA per serving than wild
 1706 caught. Low-trophic seafood, such as catfish and crawfish, regardless of whether wild caught or
 1707 farm-raised seafood, have less than half the EPA and DHA per serving than high-trophic
 1708 seafood, such as salmon and trout.

1709
 1710 Regarding contaminants, for the majority of wild caught and farmed species, neither the risks of
 1711 mercury nor organic pollutants outweigh the health benefits of seafood consumption. Consistent
 1712 evidence demonstrated that wild caught fisheries that have been managed sustainably have
 1713 remained stable over the past several decades; however, wild caught fisheries are fully exploited
 1714 and their continuing productivity will require careful management nationally and internationally

1715 to avoid long-term collapse. Expanded supply of seafood nationally and internationally will be
1716 dependent upon the increase of farm-raised seafood worldwide.

1717
1718 The impact of food production, processing, and consumption on environmental sustainability is
1719 an area of research that is rapidly evolving. As further research is conducted and best practices
1720 evaluated, additional evidence will inform both supply-side participants and consumers on how
1721 best to shift behaviors locally, nationally, and globally to support sustainable diets. Linking
1722 health, dietary guidance and the environment will promote human health and the sustainability of
1723 natural resources and ensure current and long-term food security.

1724
1725 In regards to food safety, updated and previously unexamined areas of food safety were studied.
1726 No previous DGACs have reported on coffee/caffeine consumption and health. Currently, strong
1727 evidence shows that consumption of coffee within the moderate range (3 to 5 cups per day or up
1728 to 400 mg/d caffeine) is not associated with increased long-term health risks among healthy
1729 individuals. In fact, consistent evidence indicates that coffee consumption is associated with
1730 reduced risk of type 2 diabetes and cardiovascular disease in healthy adults. Moreover, moderate
1731 evidence shows a protective association between coffee/caffeine intake and risk of Parkinson's
1732 disease. Therefore, moderate coffee consumption can be incorporated into a healthy dietary
1733 pattern, along with other healthful behaviors. To meet the growing demand of coffee, there is a
1734 need to consider sustainability issues of coffee production in economic and environmental terms.
1735 However, it should be noted that coffee as it is normally consumed can contain added calories
1736 from cream, milk, and added sugars. Care should be taken to minimize the amount of calories
1737 from added sugars and high-fat dairy or dairy substitutes added to coffee.

1738
1739 The marketing and availability of high-caffeine beverages and products is on the rise.
1740 Unfortunately, only limited evidence is currently available to ascertain the safety of high caffeine
1741 intake (greater than 400 mg/day for adults and undetermined for children and adolescents), that
1742 may occur with rapid consumption of large-sized energy drinks. The limited data suggest adverse
1743 health outcomes, such as caffeine toxicity and cardiovascular events. Concern is heightened
1744 when caffeine is combined with alcoholic beverages. Limited or no consumption of high caffeine
1745 drinks, or other products with high amounts of caffeine, is advised for children and adolescents.
1746 Energy drinks with high levels of caffeine and alcoholic beverages should not be consumed
1747 together, either mixed together or consumed at the same sitting.

1748
1749 The DGAC also examined the food additive aspartame. At the level that the U.S. population
1750 consumes aspartame, it appears to be safe. However, some uncertainty continues about increased
1751 risk of hematopoietic cancers in men, indicating a need for more research.

1752

1753 Individual behaviors along with sound government policies and responsible private sector
 1754 practices are all needed to reduce foodborne illnesses. To that end, the DGAC updated the
 1755 established recommendations for handling foods at home.

1756

1757

1758 **NEEDS FOR FUTURE RESEARCH**

1759 **Dietary Patterns and Sustainability**

1760 1. Conduct research to determine whether sustainable diets are affordable and accessible to all
 1761 sectors of the population and how this can be improved, including how policy strategies
 1762 could influence the supply chain (all steps from farm to plate) to affect this improvement.

1763 **Rationale:** Ensuring that sustainable diets are accessible and affordable to all sectors of the
 1764 population is important to promote food security.

1765

1766 2. Develop, conduct, and evaluate in-depth analyses of U.S. domestic dietary patterns and
 1767 determine the degree to which sustainability practices, domestically and internationally, are
 1768 important to food choice and how to increase public awareness of the impact of food choices
 1769 on environmental outcomes.

1770 **Rationale:** Understanding consumer choice across demographic groups and the degree to
 1771 which either health and/or sustainability is a significant decisional criterion as well as the
 1772 degree to which choice theory can be used to improve choices will be important to helping
 1773 drive change.

1774

1775 3. Develop a robust understanding of how production practices, supply chain decisions,
 1776 consumer behaviors, and waste disposal affect the environmental sustainability of various
 1777 practices across the USDA food components of MyPlate.

1778 **Rationale:** Developing sustainable production and supply chain practices for all parts of
 1779 MyPlate, especially meat and dairy products will be important to reduce their environmental
 1780 impact.

1781

1782 4. Determine the potential economic benefits and challenges to supply chain stakeholders in
 1783 relationship to findings in Research Recommendation 3.

1784 **Rationale:** Experience demonstrates that many practices over the past few decades that
 1785 improve the environmental footprint of, for example, production practices, also have led to
 1786 improved profit (e.g., Integrated Pest Management to reduce pesticide use in many fruit and
 1787 vegetables). It is important to know how changes will affect profit to help enable future
 1788 policy in both the private and public spheres.

1789

1790 **Seafood Sustainability**

- 1791 5. Conduct research on methods to ensure the maintenance of nutrient profiles of high-trophic
 1792 level farmed seafood and improve nutrient profiles of low-trophic farmed seafood
 1793 concurrently with research to improve production efficacy.

1794 **Rationale:** The evidence supporting healthfulness of seafood consumption is based on
 1795 consumption of predominantly wild caught species. Many popular low-trophic level farmed
 1796 seafood have nutrient profiles that depend on feeds. Efficient production of seafood with
 1797 nutrient profiles that are known to be healthful should be emphasized.

1798

- 1799 6. Conduct research to develop methods to ensure contaminant levels in all seafood remain at
 1800 levels similar to or lower than at present. Maintain monitoring of contaminant levels for
 1801 capture fisheries to ensure that levels caused by pollution do not rise appreciably. This
 1802 research should include developing effective rapid response approaches if the quality of
 1803 seafood supply is acutely affected.

1804 **Rationale:** Current research findings support the contention that contaminant levels are
 1805 generally well below those that significantly alter the healthfulness of seafood. As industry
 1806 naturally improves efficiency, feeds and environmental conditions should be monitored to
 1807 maintain or reduce priority contaminants and insure significant new contaminants do not
 1808 enter the seafood supply.

1809

1810

1811 **Usual Caffeine/Coffee Intake**

- 1812 7. Evaluate the effects of coffee on health outcomes in vulnerable populations, such as women
 1813 who are pregnant (premature birth, low birth weight, spontaneous abortion).

1814 **Rationale:** Given the limited evidence of the effects of coffee/caffeine consumption on
 1815 pregnancy outcomes, future studies need to establish safe levels of coffee/caffeine
 1816 consumption during pregnancy.

1817

- 1818 8. Examine the effects of coffee on sleep patterns, quality of life, and dependency and
 1819 addiction.

1820 **Rationale:** Because coffee is a known stimulant, future research should examine the effect of
 1821 coffee/caffeine on sleep quality, dependency, addiction, and overall quality of life measures.

1822

- 1823 9. Evaluate the prospective association between coffee/caffeine consumption and cancer at
 1824 different sites.

1825 **Rationale:** Large well-conducted prospective cohort studies that adequately control for
 1826 smoking (status and dosage) and other potential confounders are needed to understand the

1827 association of coffee (caffeinated and decaffeinated) with cancer at different sites.

1828

1829 10. Examine prospectively the effects of coffee/caffeine on cognitive decline, neurodegenerative
1830 diseases, and depression.

1831 **Rationale:** Neurodegenerative diseases affect millions of people worldwide and more than
1832 five million Americans are living with Alzheimer’s disease. Given the limited evidence of
1833 coffee/caffeine on neurodegenerative diseases, well-designed prospective studies should
1834 examine the association of coffee/caffeine consumption on cognitive decline, depression, and
1835 Alzheimer’s disease.

1836

1837 11. Understand the mechanisms underlying the protective effects of coffee on diabetes and CVD.

1838 **Rationale:** Evidence for a biological plausibility for coffee on risk of type 2 diabetes and
1839 CVD stems primarily from animal studies. Randomized controlled trials in humans should
1840 evaluate the effect of coffee/caffeine on measures of glycemia, insulin sensitivity, endothelial
1841 dysfunction, and inflammation.

1842

1843 12. Understand the association between coffee and health outcomes in individuals with existing
1844 CVD, diabetes, cancer, neurodegenerative diseases, or depressive symptoms.

1845 **Rationale:** Strong evidence supports a protective effect of moderate coffee consumption on
1846 chronic disease risk in healthy adults, but its association among those with existing diseases
1847 has been less studied. Given that a substantial number of people suffer from these chronic
1848 diseases, the role of coffee in preventing other health outcomes in such groups remains
1849 understudied.

1850

1851 **High-dose Caffeine Intake**

1852 13. Define excessive caffeine intake and safe levels of consumption for children, adolescents,
1853 and young adults.

1854 **Rationale:** Current research on caffeine and health outcomes has focused primarily on
1855 adults. Given the increasing prevalence of energy drink consumption among children,
1856 adolescents, and young adults, research is needed to identify safe levels of consumption in
1857 these groups.

1858

1859 14. Determine the prevalence of excessive caffeine intake in children and adults beyond intake of
1860 energy drinks.

1861 **Rationale:** Data on the sources (other than energy drinks) and doses of caffeine intake in
1862 children and adults are limited. Identifying the sources and safe levels of consumption will
1863 help in formulating policy and framing recommendations.

1864

1865 15. Examine the effect of excessive consumption of caffeine and energy drinks on health
1866 outcomes in both children and adults.

1867 **Rationale:** Prospective studies of associations of excessive caffeine and energy drink intake
1868 with health outcomes in children and adults are necessary, as randomized controlled trials are
1869 not be feasible given ethical constraints.

1870

1871 16. Conduct observational studies to examine the health effects of alcohol mixed with energy
1872 drinks.

1873 **Rationale:** In recent years, consumption of alcohol energy drinks by adolescents has resulted
1874 in emergency room admissions and deaths. No data exist on the prospective association
1875 between consumption of alcohol energy drinks and health outcomes in both adolescents and
1876 adults.

1877

1878 **Aspartame**

1879 17. Examine the risks of aspartame related to some cancers, especially hematopoietic ones, and
1880 pregnancy outcomes.

1881 **Rationale:** Limited and inconsistent evidence suggests a possible association between
1882 aspartame and risk of hematopoietic cancers (non-Hodgkin lymphoma and multiple
1883 myeloma) in men, indicating the need for long-term human studies. Additionally, limited and
1884 inconsistent evidence indicates a potential for risk of preterm delivery, which warrants
1885 further research.

1886

1887

1888 **REFERENCES**

1889

1890 1. Food and Agriculture Organization of the United Nations. Rome Declaration on World
1891 Food Security and World Food Summit Plan of Action. Rome, Italy,1996. Available
1892 from: <http://www.fao.org/docrep/003/w3613e/w3613e00.htm>.

1893 2. Food And Agriculture Organization Of The United Nations. International Scientific
1894 Symposium Biodiversity And Sustainable Diets United Against Hunger. Rome,2010.
1895 Available from: <http://www.fao.org/ag/humannutrition/28506-0efe4aed57af34e2dbb8dc578d465df8b.pdf>.

1897 3. United Nations Environment Programme. UNEP Year Book: Emerging Issues In Our
1898 Global Environment 2012. Available from: www.unep.org/yearbook/2012/.

1899 4. Nellemann C, MacDevette, M., Manders, T., Eickhout, B., , Svihus B, Prins, A. G.,
1900 Kaltenborn, B. P. (Eds). The environmental food crisis – The environment’s role in
1901 averting future food crises. A UNEP rapid response assessment United Nations
1902 Environment Programme, GRID-Arendal; 2009. Available from: www.grida.no.

- 1903 5. Burlingame B, Dernini S. Sustainable diets: the Mediterranean diet as an example. *Public*
 1904 *Health Nutr.* 2011;14(12A):2285-7. PMID: 22166185.
 1905 <http://www.ncbi.nlm.nih.gov/pubmed/22166185>.
- 1906 6. World Wildlife Federation. Livewell: a balance of healthy and sustainable food choices
 1907 2011. Available from: http://assets.wwf.org.uk/downloads/livewell_report_jan11.pdf.
- 1908 7. Heller MC, Keoleian GA, Willett WC. Toward a life cycle-based, diet-level framework
 1909 for food environmental impact and nutritional quality assessment: a critical review.
 1910 *Environ Sci Technol.* 2013;47(22):12632-47. PMID: 24152032.
 1911 <http://www.ncbi.nlm.nih.gov/pubmed/24152032>.
- 1912 8. United States Department of Agriculture. A Series of Systematic Reviews on the
 1913 Relationship Between Dietary Patterns and Health Outcomes 2014. Available from:
 1914 <http://www.nel.gov/vault/2440/web/files/DietaryPatterns/DPRptFullFinal.pdf>.
- 1915 9. Eshel G, Shepon A, Makov T, Milo R. Land, irrigation water, greenhouse gas, and
 1916 reactive nitrogen burdens of meat, eggs, and dairy production in the United States. *Proc*
 1917 *Natl Acad Sci U S A.* 2014;111(33):11996-2001. PMID: 25049416.
 1918 <http://www.ncbi.nlm.nih.gov/pubmed/25049416>.
- 1919 10. Heller MCaK, G. A. . Greenhouse Gas Emission Estimates of U.S. Dietary Choices and
 1920 Food Loss. *Journal of Industrial Ecology.* 2014.
- 1921 11. Tilman D, Clark M. Global diets link environmental sustainability and human health.
 1922 *Nature.* 2014. PMID: 25383533. <http://www.ncbi.nlm.nih.gov/pubmed/25383533>.
- 1923 12. Health Council of the Netherlands. Guidelines for a healthy diet: the ecological
 1924 perspective. 2011. publication no. 2011/08E. ISBN 978-90-5549-845-1:[Available from:
 1925 <http://www.gezondheidsraad.nl/sites/default/files/201108E.pdf>.
- 1926 13. Nordic Council of Ministers. Nordic Council of Ministers Secretariat. Nordic Nutrition
 1927 Recommendations 2012: Integrating nutrition and physical activity. Available from:
 1928 <http://norden.diva-portal.org/smash/get/diva2:704251/FULLTEXT01.pdf>.
- 1929 14. German Council for Sustainable Development. The Sustainable Shopping Basket: A
 1930 guide to better shopping. 2013. Available from:
 1931 http://www.nachhaltigkeitsrat.de/uploads/media/Brochure_Sustainable_Shopping_Basket_01.pdf.
- 1932 15. Institute of Medicine. Sustainable Diets: Food for Healthy People and a Healthy Planet:
 1933 Workshop Summary. Washington, DC: The National Academies Press; 2014. Available
 1934 from: http://www.nap.edu/openbook.php?record_id=18578&page=R2.
- 1935 16. National Research Council. Toward sustainable agricultural systems in the 21st century
 1936 Washington, DC: The National Academies Press; 2010. Available from:
 1937 http://www.nap.edu/openbook.php?record_id=12832.
- 1938 17. Nordin SM, Boyle M, Kemmer TM, Academy of N, Dietetics. Position of the academy of
 1939 nutrition and dietetics: nutrition security in developing nations: sustainable food, water,
 1940 and health. *J Acad Nutr Diet.* 2013;113(4):581-95. PMID: 23522578.
 1941 <http://www.ncbi.nlm.nih.gov/pubmed/23522578>.
- 1942 18. Sustainable Development Commission, editor. Looking back, Looking Forward:
 1943 Sustainability and UK food policy 2000 – 20112011.
- 1944 19. United Nations DfSD. Open Working Group proposal for Sustainable Development
 1945 Goals 2014. Available from:
 1946 <http://sustainabledevelopment.un.org/content/documents/1579SDGs%20Proposal.pdf>.
- 1947

- 1948 20. FAO Fisheries and Aquaculture Department Food and Agriculture Organization of the
 1949 United Nations. The State of World Fisheries and Aquaculture Rome,2012. Available
 1950 from: <http://www.fao.org/docrep/016/i2727e/i2727e.pdf>.
- 1951 21. Higgins JPT GSe. Cochrane Handbook for Systematic Reviews of Interventions Version
 1952 5.1.0: The Cochrane Collaboration; 2011. Available from: www.cochrane-handbook.org.
- 1953 22. Drummond MF, Jefferson TO. Guidelines for authors and peer reviewers of economic
 1954 submissions to the BMJ. The BMJ Economic Evaluation Working Party. BMJ.
 1955 1996;313(7052):275-83. PMID: 8704542.
 1956 <http://www.ncbi.nlm.nih.gov/pubmed/8704542>.
- 1957 23. Eddy D. The role of mathematical modelling in Assessing medical technology,.
 1958 Technology Assessment. 1985:144-54.
- 1959 24. Stevenson M, Lloyd-Jones M, Morgan MY, Wong R. Non-invasive diagnostic
 1960 assessment tools for the detection of liver fibrosis in patients with suspected alcohol-
 1961 related liver disease: a systematic review and economic evaluation. Health Technol
 1962 Assess. 2012;16(4):1-174. PMID: 22333291.
 1963 <http://www.ncbi.nlm.nih.gov/pubmed/22333291>.
- 1964 25. U.S. Department of Agriculture ARS. USDA National Nutrient Database for Standard
 1965 Reference, Release 27. Nutrient Data Laboratory Home Page2014.
- 1966 26. Cladis DP, Kleiner AC, Freiser HH, Santerre CR. Fatty Acid Profiles of Commercially
 1967 Available Finfish Fillets in the United States. Lipids. 2014. PMID: 25108414.
 1968 <http://www.ncbi.nlm.nih.gov/pubmed/25108414>.
- 1969 27. Food and Agriculture Organization of the United Nations; Geneva WHO. Report of the
 1970 Joint FAO/WHO Expert Consultation on the Risks and Benefits of Fish Consumption.
 1971 Rome. 2011.
- 1972 28. Aromataris E, Pearson A. The systematic review: an overview. Am J Nurs.
 1973 2014;114(3):53-8. PMID: 24572533. <http://www.ncbi.nlm.nih.gov/pubmed/24572533>.
- 1974 29. European Food Safety Authority (EFSA). Food Additives and Nutrient Sources Added
 1975 to food Panel. Scientific Opinion on the re-evaluation of aspartame (E 951) as a food
 1976 additive. Parma, Italy,2013. Available from:
 1977 <http://www.efsa.europa.eu/en/efsajournal/doc/3496.pdf>.
- 1978 30. Centers for Disease Control and Prevention. Handwashing: Clean Hands Save Lives
 1979 2013. Available from: <http://www.cdc.gov/handwashing/>.
- 1980 31. Food and Drug Administration. Food Safety for Moms-to-Be: Safe Eats - Meat, Poultry
 1981 & Seafood 2014 [cited 2014 June 3]. Available from:
 1982 <http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm>.
- 1983 32. United States Department of Agriculture. Food Safety Inspection Service. Food Safety
 1984 Fact Sheets 2013 [cited 2014 June 3]. Available from:
 1985 [http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-](http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets)
 1986 [safety-fact-sheets](http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets).
- 1987 33. International Organisation for Standardisation. Environmental management – Life cycle
 1988 assessment – principles and framework, Geneva, Switzerland 2006a.
- 1989 34. Aston LM, Smith JN, Powles JW. Impact of a reduced red and processed meat dietary
 1990 pattern on disease risks and greenhouse gas emissions in the UK: a modelling study. BMJ
 1991 Open. 2012;2(5). PMID: 22964113. <http://www.ncbi.nlm.nih.gov/pubmed/22964113>.
- 1992 35. Baroni L, Cenci L, Tettamanti M, Berati M. Evaluating the environmental impact of
 1993 various dietary patterns combined with different food production systems. Eur J Clin

- 1994 Nutr. 2007;61(2):279-86. PMID: 17035955.
 1995 <http://www.ncbi.nlm.nih.gov/pubmed/17035955>.
- 1996 36. Barosh L, Friel S, Engelhardt K, Chan L. The cost of a healthy and sustainable diet--who
 1997 can afford it? Aust N Z J Public Health. 2014;38(1):7-12. PMID: 24494938.
 1998 <http://www.ncbi.nlm.nih.gov/pubmed/24494938>.
- 1999 37. de Carvalho AM, Cesar CL, Fisberg RM, Marchioni DM. Excessive meat consumption in
 2000 Brazil: diet quality and environmental impacts. Public Health Nutr. 2013;16(10):1893-9.
 2001 PMID: 22894818. <http://www.ncbi.nlm.nih.gov/pubmed/22894818>.
- 2002 38. Hendrie GA, Ridoutt BG, Wiedmann TO, Noakes M. Greenhouse gas emissions and the
 2003 Australian diet--comparing dietary recommendations with average intakes. Nutrients.
 2004 2014;6(1):289-303. PMID: 24406846. <http://www.ncbi.nlm.nih.gov/pubmed/24406846>.
- 2005 39. Macdiarmid JI, Kyle J, Horgan GW, Loe J, Fyfe C, Johnstone A, et al. Sustainable diets
 2006 for the future: Can we contribute to reducing greenhouse gas emissions by eating a
 2007 healthy diet? Am J Clin Nutr. 2012;96(3):632-9. PMID: 22854399.
 2008 <http://www.ncbi.nlm.nih.gov/pubmed/22854399>.
- 2009 40. Meier T, Christen O. Environmental impacts of dietary recommendations and dietary
 2010 styles: Germany as an example. Environ Sci Technol. 2013;47(2):877-88. PMID:
 2011 23189920. <http://www.ncbi.nlm.nih.gov/pubmed/23189920>.
- 2012 41. Peters CJ, Wilkins JL, Fick GW. Testing a complete-diet model for estimating the land
 2013 resource requirements of food consumption and agricultural carrying capacity: The New
 2014 York State example. Renewable agriculture and food systems. 2007;22(2):145-53.
 2015 <http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=1091328&fileId=S1742170507001767>.
- 2016 42. Pimentel D, Pimentel M. Sustainability of meat-based and plant-based diets and the
 2017 environment. Am J Clin Nutr. 2003;78(3 Suppl):660S-3S. PMID: 12936963.
 2018 <http://www.ncbi.nlm.nih.gov/pubmed/12936963>.
- 2019 43. Pradhan P, Reusser DE, Kropp JP. Embodied greenhouse gas emissions in diets. PLoS
 2020 One. 2013;8(5):e62228. PMID: 23700408.
 2021 <http://www.ncbi.nlm.nih.gov/pubmed/23700408>.
- 2022 44. Saez-Almendros S, Obrador B, Bach-Faig A, Serra-Majem L. Environmental footprints
 2023 of Mediterranean versus Western dietary patterns: beyond the health benefits of the
 2024 Mediterranean diet. Environ Health. 2013;12:118. PMID: 24378069.
 2025 <http://www.ncbi.nlm.nih.gov/pubmed/24378069>.
- 2026 45. Scarborough P, Allender S, Clarke D, Wickramasinghe K, Rayner M. Modelling the
 2027 health impact of environmentally sustainable dietary scenarios in the UK. Eur J Clin
 2028 Nutr. 2012;66(6):710-5. PMID: 22491494.
 2029 <http://www.ncbi.nlm.nih.gov/pubmed/22491494>.
- 2030 46. van Dooren C, Marinussen M, Blonk H, Aiking H, Vellinga P. Exploring dietary
 2031 guidelines based on ecological and nutritional values: A comparison of six dietary
 2032 patterns. Food Policy. 2014;44(0):36-46.
 2033 <http://www.sciencedirect.com/science/article/pii/S0306919213001620>.
- 2034 47. Vieux F, Soler LG, Touazi D, Darmon N. High nutritional quality is not associated with
 2035 low greenhouse gas emissions in self-selected diets of French adults. Am J Clin Nutr.
 2036 2013;97(3):569-83. PMID: 23364012. <http://www.ncbi.nlm.nih.gov/pubmed/23364012>.
- 2037 48. Wilson N, Nghiem N, Ni Mhurchu C, Eyles H, Baker MG, Blakely T. Foods and dietary
 2038 patterns that are healthy, low-cost, and environmentally sustainable: a case study of
 2039

- 2040 optimization modeling for New Zealand. *PLoS One*. 2013;8(3):e59648. PMID:
 2041 23544082. <http://www.ncbi.nlm.nih.gov/pubmed/23544082>.
- 2042 49. Bellows AC, Alcaraz VG, Hallman WK. Gender and food, a study of attitudes in the
 2043 USA towards organic, local, U.S. grown, and GM-free foods. *Appetite*. 2010;55(3):540-
 2044 50. PMID: 20851727. <http://www.ncbi.nlm.nih.gov/pubmed/20851727>.
- 2045 50. Blonk H, Ponsioen, T., Kool, A., Marinussen, M. The Agri-Foorprint Method;
 2046 Methodological LCA Framework, Assumptions and Applied Data, Version 1.0. 2011.
- 2047 51. International Organisation for Standardisation. ISO 14044: Environmental management –
 2048 Life cycle assessment – requirements and guidelines 2006b. Available from:
 2049 http://www.iso.org/iso/catalogue_detail?csnumber=38498.
- 2050 52. Goedkoop M SR. The Eco-Indicator 99, A Damage Oriented Method for Life Cycle
 2051 Assessment Methodology Report 2000. Available from:
 2052 <http://teclim.ufba.br/jsf/indicadores/holan%20ecoindicator%2099.pdf>.
- 2053 53. Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of
 2054 incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and
 2055 meta-analysis. *Circulation*. 2010;121(21):2271-83. PMID: 20479151.
 2056 <http://www.ncbi.nlm.nih.gov/pubmed/20479151>.
- 2057 54. Norat T CD, Lau R, Aune D, Vieira R. WCRF/AICR Systematic Literature Review
 2058 Continuous Update Project Report: The Associations between Food, Nutrition and
 2059 Physical Activity and the Risk of Colorectal Cancer, 2010. Available from:
 2060 [http://www.dietandcancerreport.org/cancer_resource_center/downloads/cu/Colorectal-](http://www.dietandcancerreport.org/cancer_resource_center/downloads/cu/Colorectal-Cancer-SLR-2010.pdf)
 2061 [Cancer-SLR-2010.pdf](http://www.dietandcancerreport.org/cancer_resource_center/downloads/cu/Colorectal-Cancer-SLR-2010.pdf).
- 2062 55. Wiseman M. The second World Cancer Research Fund/American Institute for Cancer
 2063 Research expert report. Food, nutrition, physical activity, and the prevention of cancer: a
 2064 global perspective. *Proc Nutr Soc*. 2008;67(3):253-6. PMID: 18452640.
 2065 <http://www.ncbi.nlm.nih.gov/pubmed/18452640>.
- 2066 56. U.S. Department of Agriculture and U.S. Department of Health and Human Services.
 2067 Dietary Guidelines for Americans, 2010. 7th ed. Washington, DC: U.S. Government
 2068 Printing Office; December 2010.
- 2069 57. National Oceanic and Atmospheric Administration. Fisheries of the United States 2011.
 2070 Available from: http://www.st.nmfs.noaa.gov/st1/fus/fus11/FUS_2011.pdf.
- 2071 58. Bouman AF BA, Overbeek CC, D. P. Bureau, M. Pawlowski,, Gilbert. aPM. Hindcasts
 2072 and future projections of global inland and coastal nitrogen and phosphorus loads due to
 2073 finfish aquaculture. *Reviews in Fisheries Science*. 2013;21(2):112-56.
- 2074 59. Hall S, Delaporte A, Phillips MJ, Beveridge M, O’Keefe, M. Blue frontiers: Managing
 2075 the Environmental Costs of Aquaculture. WorldFish Center, 2011.
- 2076 60. National Oceanic and Atmospheric Administration. Fish Watch: US Seafood Facts [cited
 2077 2014 June 14]. Available from:
 2078 http://www.fishwatch.gov/farmed_seafood/outside_the_us.htm.
- 2079 61. Food and Drug Administration. Fish: What Pregnant Women and Parents Should Know:
 2080 Draft Updated Advice by FDA and EPA 2014. Available from:
 2081 <http://www.fda.gov/Food/FoodborneIllnessContaminants/Metals/ucm393070.htm>.
- 2082 62. United States Environmental Protection Agency. What You Need to Know about
 2083 Mercury in Fish and Shellfish 2004. Available from:
 2084 http://water.epa.gov/scitech/swguidance/fishshellfish/outreach/advice_index.cfm.

- 2085 63. LaComb RP SR, Wilkinson Enns C, Goldman JD. Beverage Choices of U.S. Adults:
2086 What We Eat In America, NHANES 2007-2008. 2011.
- 2087 64. Crippa A, Discacciati A, Larsson SC, Wolk A, Orsini N. Coffee consumption and
2088 mortality from all causes, cardiovascular disease, and cancer: a dose-response meta-
2089 analysis. *Am J Epidemiol*. 2014;180(8):763-75. PMID: 25156996.
2090 <http://www.ncbi.nlm.nih.gov/pubmed/25156996>.
- 2091 65. Je Y, Giovannucci E. Coffee consumption and total mortality: a meta-analysis of twenty
2092 prospective cohort studies. *Br J Nutr*. 2014;111(7):1162-73. PMID: 24279995.
2093 <http://www.ncbi.nlm.nih.gov/pubmed/24279995>.
- 2094 66. Malerba S, Turati F, Galeone C, Pelucchi C, Verga F, La Vecchia C, et al. A meta-
2095 analysis of prospective studies of coffee consumption and mortality for all causes,
2096 cancers and cardiovascular diseases. *Eur J Epidemiol*. 2013;28(7):527-39. PMID:
2097 23934579. <http://www.ncbi.nlm.nih.gov/pubmed/23934579>.
- 2098 67. Ding M, Bhupathiraju SN, Satija A, van Dam RM, Hu FB. Long-term coffee
2099 consumption and risk of cardiovascular disease: a systematic review and a dose-response
2100 meta-analysis of prospective cohort studies. *Circulation*. 2014;129(6):643-59. PMID:
2101 24201300. <http://www.ncbi.nlm.nih.gov/pubmed/24201300>.
- 2102 68. Sofi F, Conti AA, Gori AM, Eliana Luisi ML, Casini A, Abbate R, et al. Coffee
2103 consumption and risk of coronary heart disease: a meta-analysis. *Nutr Metab Cardiovasc*
2104 *Dis*. 2007;17(3):209-23. PMID: 17156982.
2105 <http://www.ncbi.nlm.nih.gov/pubmed/17156982>.
- 2106 69. Wu JN, Ho SC, Zhou C, Ling WH, Chen WQ, Wang CL, et al. Coffee consumption and
2107 risk of coronary heart diseases: a meta-analysis of 21 prospective cohort studies. *Int J*
2108 *Cardiol*. 2009;137(3):216-25. PMID: 18707777.
2109 <http://www.ncbi.nlm.nih.gov/pubmed/18707777>.
- 2110 70. Kim B, Nam Y, Kim J, Choi H, Won C. Coffee Consumption and Stroke Risk: A Meta-
2111 analysis of Epidemiologic Studies. *Korean J Fam Med*. 2012;33(6):356-65. PMID:
2112 23267421. <http://www.ncbi.nlm.nih.gov/pubmed/23267421>.
- 2113 71. Larsson SC, Orsini N. Coffee consumption and risk of stroke: a dose-response meta-
2114 analysis of prospective studies. *Am J Epidemiol*. 2011;174(9):993-1001. PMID:
2115 21920945. <http://www.ncbi.nlm.nih.gov/pubmed/21920945>.
- 2116 72. Mesas AE, Leon-Munoz LM, Rodriguez-Artalejo F, Lopez-Garcia E. The effect of coffee
2117 on blood pressure and cardiovascular disease in hypertensive individuals: a systematic
2118 review and meta-analysis. *Am J Clin Nutr*. 2011;94(4):1113-26. PMID: 21880846.
2119 <http://www.ncbi.nlm.nih.gov/pubmed/21880846>.
- 2120 73. Noordzij M, Uiterwaal CS, Arends LR, Kok FJ, Grobbee DE, Geleijnse JM. Blood
2121 pressure response to chronic intake of coffee and caffeine: a meta-analysis of randomized
2122 controlled trials. *J Hypertens*. 2005;23(5):921-8. PMID: 15834273.
2123 <http://www.ncbi.nlm.nih.gov/pubmed/15834273>.
- 2124 74. Steffen M, Kuhle C, Hensrud D, Erwin PJ, Murad MH. The effect of coffee consumption
2125 on blood pressure and the development of hypertension: a systematic review and meta-
2126 analysis. *J Hypertens*. 2012;30(12):2245-54. PMID: 23032138.
2127 <http://www.ncbi.nlm.nih.gov/pubmed/23032138>.
- 2128 75. Zhang Z, Hu G, Caballero B, Appel L, Chen L. Habitual coffee consumption and risk of
2129 hypertension: a systematic review and meta-analysis of prospective observational studies.

- 2130 Am J Clin Nutr. 2011;93(6):1212-9. PMID: 21450934.
 2131 <http://www.ncbi.nlm.nih.gov/pubmed/21450934>.
- 2132 76. Cai L, Ma D, Zhang Y, Liu Z, Wang P. The effect of coffee consumption on serum
 2133 lipids: a meta-analysis of randomized controlled trials. *Eur J Clin Nutr*. 2012;66(8):872-7.
 2134 PMID: 22713771. <http://www.ncbi.nlm.nih.gov/pubmed/22713771>.
- 2135 77. Jee SH, He J, Appel LJ, Whelton PK, Suh I, Klag MJ. Coffee consumption and serum
 2136 lipids: a meta-analysis of randomized controlled clinical trials. *Am J Epidemiol*.
 2137 2001;153(4):353-62. PMID: 11207153. <http://www.ncbi.nlm.nih.gov/pubmed/11207153>.
- 2138 78. Caldeira D, Martins C, Alves LB, Pereira H, Ferreira JJ, Costa J. Caffeine does not
 2139 increase the risk of atrial fibrillation: a systematic review and meta-analysis of
 2140 observational studies. *Heart*. 2013;99(19):1383-9. PMID: 24009307.
 2141 <http://www.ncbi.nlm.nih.gov/pubmed/24009307>.
- 2142 79. Mostofsky E, Rice MS, Levitan EB, Mittleman MA. Habitual coffee consumption and
 2143 risk of heart failure: a dose-response meta-analysis. *Circ Heart Fail*. 2012;5(4):401-5.
 2144 PMID: 22740040. <http://www.ncbi.nlm.nih.gov/pubmed/22740040>.
- 2145 80. Ding M, Bhupathiraju SN, Chen M, van Dam RM, Hu FB. Caffeinated and decaffeinated
 2146 coffee consumption and risk of type 2 diabetes: a systematic review and a dose-response
 2147 meta-analysis. *Diabetes Care*. 2014;37(2):569-86. PMID: 24459154.
 2148 <http://www.ncbi.nlm.nih.gov/pubmed/24459154>.
- 2149 81. Huxley R, Lee CM, Barzi F, Timmermeister L, Czernichow S, Perkovic V, et al. Coffee,
 2150 decaffeinated coffee, and tea consumption in relation to incident type 2 diabetes mellitus:
 2151 a systematic review with meta-analysis. *Arch Intern Med*. 2009;169(22):2053-63. PMID:
 2152 20008687. <http://www.ncbi.nlm.nih.gov/pubmed/20008687>.
- 2153 82. Jiang X, Zhang D, Jiang W. Coffee and caffeine intake and incidence of type 2 diabetes
 2154 mellitus: a meta-analysis of prospective studies. *Eur J Nutr*. 2014;53(1):25-38. PMID:
 2155 24150256. <http://www.ncbi.nlm.nih.gov/pubmed/24150256>.
- 2156 83. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: a systematic
 2157 review. *Jama*. 2005;294(1):97-104. PMID: 15998896.
 2158 <http://www.ncbi.nlm.nih.gov/pubmed/15998896>.
- 2159 84. Whitehead N, White H. Systematic review of randomised controlled trials of the effects
 2160 of caffeine or caffeinated drinks on blood glucose concentrations and insulin sensitivity
 2161 in people with diabetes mellitus. *J Hum Nutr Diet*. 2013;26(2):111-25. PMID: 23331476.
 2162 <http://www.ncbi.nlm.nih.gov/pubmed/23331476>.
- 2163 85. Yu X, Bao Z, Zou J, Dong J. Coffee consumption and risk of cancers: a meta-analysis of
 2164 cohort studies. *BMC Cancer*. 2011;11:96. PMID: 21406107.
 2165 <http://www.ncbi.nlm.nih.gov/pubmed/21406107>.
- 2166 86. Tang N, Wu Y, Ma J, Wang B, Yu R. Coffee consumption and risk of lung cancer: a
 2167 meta-analysis. *Lung Cancer*. 2010;67(1):17-22. PMID: 19362749.
 2168 <http://www.ncbi.nlm.nih.gov/pubmed/19362749>.
- 2169 87. Sang LX, Chang B, Li XH, Jiang M. Consumption of coffee associated with reduced risk
 2170 of liver cancer: a meta-analysis. *BMC Gastroenterol*. 2013;13:34. PMID: 23433483.
 2171 <http://www.ncbi.nlm.nih.gov/pubmed/23433483>.
- 2172 88. Bravi F, Bosetti C, Tavani A, Gallus S, La Vecchia C. Coffee reduces risk for
 2173 hepatocellular carcinoma: an updated meta-analysis. *Clin Gastroenterol Hepatol*.
 2174 2013;11(11):1413-21.e1. PMID: 23660416.
 2175 <http://www.ncbi.nlm.nih.gov/pubmed/23660416>.

- 2176 89. Jiang W, Wu Y, Jiang X. Coffee and caffeine intake and breast cancer risk: an updated
2177 dose-response meta-analysis of 37 published studies. *Gynecol Oncol.* 2013;129(3):620-9.
2178 PMID: 23535278. <http://www.ncbi.nlm.nih.gov/pubmed/23535278>.
- 2179 90. Li XJ, Ren ZJ, Qin JW, Zhao JH, Tang JH, Ji MH, et al. Coffee consumption and risk of
2180 breast cancer: an up-to-date meta-analysis. *PLoS One.* 2013;8(1):e52681. PMID:
2181 23308117. <http://www.ncbi.nlm.nih.gov/pubmed/23308117>.
- 2182 91. Tang N, Zhou B, Wang B, Yu R. Coffee consumption and risk of breast cancer: a
2183 metaanalysis. *Am J Obstet Gynecol.* 2009;200(3):290.e1-9. PMID: 19114275.
2184 <http://www.ncbi.nlm.nih.gov/pubmed/19114275>.
- 2185 92. Cao S, Liu L, Yin X, Wang Y, Liu J, Lu Z. Coffee consumption and risk of prostate
2186 cancer: a meta-analysis of prospective cohort studies. *Carcinogenesis.* 2014;35(2):256-
2187 61. PMID: 24343360. <http://www.ncbi.nlm.nih.gov/pubmed/24343360>.
- 2188 93. Zhong S, Chen W, Yu X, Chen Z, Hu Q, Zhao J. Coffee consumption and risk of prostate
2189 cancer: an up-to-date meta-analysis. *Eur J Clin Nutr.* 2014;68(3):330-7. PMID:
2190 24300907. <http://www.ncbi.nlm.nih.gov/pubmed/24300907>.
- 2191 94. Discacciati A, Orsini N, Wolk A. Coffee consumption and risk of nonaggressive,
2192 aggressive and fatal prostate cancer--a dose-response meta-analysis. *Ann Oncol.*
2193 2014;25(3):584-91. PMID: 24276028. <http://www.ncbi.nlm.nih.gov/pubmed/24276028>.
- 2194 95. Park CH, Myung SK, Kim TY, Seo HG, Jeon YJ, Kim Y. Coffee consumption and risk
2195 of prostate cancer: a meta-analysis of epidemiological studies. *BJU Int.* 2010;106(6):762-
2196 9. PMID: 20590551. <http://www.ncbi.nlm.nih.gov/pubmed/20590551>.
- 2197 96. Braem MG, Onland-Moret NC, Schouten LJ, Tjønneland A, Hansen L, Dahm CC, et al.
2198 Coffee and tea consumption and the risk of ovarian cancer: a prospective cohort study
2199 and updated meta-analysis. *Am J Clin Nutr.* 2012;95(5):1172-81. PMID: 22440851.
2200 <http://www.ncbi.nlm.nih.gov/pubmed/22440851>.
- 2201 97. Bravi F, Scotti L, Bosetti C, Gallus S, Negri E, La Vecchia C, et al. Coffee drinking and
2202 endometrial cancer risk: a metaanalysis of observational studies. *Am J Obstet Gynecol.*
2203 2009;200(2):130-5. PMID: 19110217. <http://www.ncbi.nlm.nih.gov/pubmed/19110217>.
- 2204 98. Je Y, Giovannucci E. Coffee consumption and risk of endometrial cancer: findings from
2205 a large up-to-date meta-analysis. *Int J Cancer.* 2012;131(7):1700-10. PMID: 22190017.
2206 <http://www.ncbi.nlm.nih.gov/pubmed/22190017>.
- 2207 99. Zhou Y, Tian C, Jia C. A dose-response meta-analysis of coffee consumption and bladder
2208 cancer. *Prev Med.* 2012;55(1):14-22. PMID: 22564775.
2209 <http://www.ncbi.nlm.nih.gov/pubmed/22564775>.
- 2210 100. Turati F, Galeone C, Edefonti V, Ferraroni M, Lagiou P, La Vecchia C, et al. A meta-
2211 analysis of coffee consumption and pancreatic cancer. *Ann Oncol.* 2012;23(2):311-8.
2212 PMID: 21746805. <http://www.ncbi.nlm.nih.gov/pubmed/21746805>.
- 2213 101. Dong J, Zou J, Yu XF. Coffee drinking and pancreatic cancer risk: a meta-analysis of
2214 cohort studies. *World J Gastroenterol.* 2011;17(9):1204-10. PMID: 21448427.
2215 <http://www.ncbi.nlm.nih.gov/pubmed/21448427>.
- 2216 102. Turati F, Galeone C, La Vecchia C, Garavello W, Tavani A. Coffee and cancers of the
2217 upper digestive and respiratory tracts: meta-analyses of observational studies. *Ann Oncol.*
2218 2011;22(3):536-44. PMID: 20943597. <http://www.ncbi.nlm.nih.gov/pubmed/20943597>.
- 2219 103. Zheng JS, Yang J, Fu YQ, Huang T, Huang YJ, Li D. Effects of green tea, black tea, and
2220 coffee consumption on the risk of esophageal cancer: a systematic review and meta-

- 2221 analysis of observational studies. *Nutr Cancer*. 2013;65(1):1-16. PMID: 23368908.
 2222 <http://www.ncbi.nlm.nih.gov/pubmed/23368908>.
- 2223 104. Botelho F, Lunet N, Barros H. Coffee and gastric cancer: systematic review and meta-
 2224 analysis. *Cad Saude Publica*. 2006;22(5):889-900. PMID: 16680342.
 2225 <http://www.ncbi.nlm.nih.gov/pubmed/16680342>.
- 2226 105. Galeone C, Turati F, La Vecchia C, Tavani A. Coffee consumption and risk of colorectal
 2227 cancer: a meta-analysis of case-control studies. *Cancer Causes Control*.
 2228 2010;21(11):1949-59. PMID: 20680435.
 2229 <http://www.ncbi.nlm.nih.gov/pubmed/20680435>.
- 2230 106. Je Y, Liu W, Giovannucci E. Coffee consumption and risk of colorectal cancer: a
 2231 systematic review and meta-analysis of prospective cohort studies. *Int J Cancer*.
 2232 2009;124(7):1662-8. PMID: 19115212. <http://www.ncbi.nlm.nih.gov/pubmed/19115212>.
- 2233 107. Li G, Ma D, Zhang Y, Zheng W, Wang P. Coffee consumption and risk of colorectal
 2234 cancer: a meta-analysis of observational studies. *Public Health Nutr*. 2013;16(2):346-57.
 2235 PMID: 22694939. <http://www.ncbi.nlm.nih.gov/pubmed/22694939>.
- 2236 108. Costa J, Lunet N, Santos C, Santos J, Vaz-Carneiro A. Caffeine exposure and the risk of
 2237 Parkinson's disease: a systematic review and meta-analysis of observational studies. *J*
 2238 *Alzheimers Dis*. 2010;20 Suppl 1:S221-38. PMID: 20182023.
 2239 <http://www.ncbi.nlm.nih.gov/pubmed/20182023>.
- 2240 109. Ishihara L, Brayne C. A systematic review of nutritional risk factors of Parkinson's
 2241 disease. *Nutr Res Rev*. 2005;18(2):259-82. PMID: 19079910.
 2242 <http://www.ncbi.nlm.nih.gov/pubmed/19079910>.
- 2243 110. Qi H, Li S. Dose-response meta-analysis on coffee, tea and caffeine consumption with
 2244 risk of Parkinson's disease. *Geriatr Gerontol Int*. 2014;14(2):430-9. PMID: 23879665.
 2245 <http://www.ncbi.nlm.nih.gov/pubmed/23879665>.
- 2246 111. Arab L, Khan F, Lam H. Epidemiologic evidence of a relationship between tea, coffee, or
 2247 caffeine consumption and cognitive decline. *Adv Nutr*. 2013;4(1):115-22. PMID:
 2248 23319129. <http://www.ncbi.nlm.nih.gov/pubmed/23319129>.
- 2249 112. Santos C, Costa J, Santos J, Vaz-Carneiro A, Lunet N. Caffeine intake and dementia:
 2250 systematic review and meta-analysis. *J Alzheimers Dis*. 2010;20 Suppl 1:S187-204.
 2251 PMID: 20182026. <http://www.ncbi.nlm.nih.gov/pubmed/20182026>.
- 2252 113. Greenwood DC, Thatcher NJ, Ye J, Garrard L, Keogh G, King LG, et al. Caffeine intake
 2253 during pregnancy and adverse birth outcomes: a systematic review and dose-response
 2254 meta-analysis. *Eur J Epidemiol*. 2014;29(10):725-34. PMID: 25179792.
 2255 <http://www.ncbi.nlm.nih.gov/pubmed/25179792>.
- 2256 114. Maslova E, Bhattacharya S, Lin SW, Michels KB. Caffeine consumption during
 2257 pregnancy and risk of preterm birth: a meta-analysis. *Am J Clin Nutr*. 2010;92(5):1120-
 2258 32. PMID: 20844077. <http://www.ncbi.nlm.nih.gov/pubmed/20844077>.
- 2259 115. Peck JD, Leviton A, Cowan LD. A review of the epidemiologic evidence concerning the
 2260 reproductive health effects of caffeine consumption: a 2000-2009 update. *Food Chem*
 2261 *Toxicol*. 2010;48(10):2549-76. PMID: 20558227.
 2262 <http://www.ncbi.nlm.nih.gov/pubmed/20558227>.
- 2263 116. Bech BH, Obel C, Henriksen TB, Olsen J. Effect of reducing caffeine intake on birth
 2264 weight and length of gestation: randomised controlled trial. *BMJ*. 2007;334(7590):409.
 2265 PMID: 17259189. <http://www.ncbi.nlm.nih.gov/pubmed/17259189>.

- 2266 117. Burrows T, Pursey K, Neve M, Stanwell P. What are the health implications associated
2267 with the consumption of energy drinks? A systematic review. *Nutr Rev.* 2013;71(3):135-
2268 48. PMID: 23452281. <http://www.ncbi.nlm.nih.gov/pubmed/23452281>.
- 2269 118. Goldfarb M, Tellier C, Thanassoulis G. Review of published cases of adverse
2270 cardiovascular events after ingestion of energy drinks. *Am J Cardiol.* 2014;113(1):168-
2271 72. PMID: 24176062. <http://www.ncbi.nlm.nih.gov/pubmed/24176062>.
- 2272 119. Government of Canada. Health Canada Health Products. Food Branch. Food Directorate.
2273 Caffeine in Foods 2012 [updated 2012-02-16]. Available from: [http://www.hc-
2274 sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php](http://www.hc-sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php).
- 2275 120. Pomeranz JL, Munsell CR, Harris JL. Energy drinks: an emerging public health hazard
2276 for youth. *J Public Health Policy.* 2013;34(2):254-71. PMID: 23486464.
2277 <http://www.ncbi.nlm.nih.gov/pubmed/23486464>.
- 2278 121. Seifert SM, Schaechter JL, Hershorin ER, Lipshultz SE. Health effects of energy drinks
2279 on children, adolescents, and young adults. *Pediatrics.* 2011;127(3):511-28. PMID:
2280 21321035. <http://www.ncbi.nlm.nih.gov/pubmed/21321035>.
- 2281 122. Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks--a growing problem. *Drug*
2282 *Alcohol Depend.* 2009;99(1-3):1-10. PMID: 18809264.
2283 <http://www.ncbi.nlm.nih.gov/pubmed/18809264>.
- 2284 123. Institute of Medicine. Caffeine in Food and Dietary Supplements: Examining Safety:
2285 Workshop Summary. Washington, DC: The National Academies Press; 2014. Available
2286 from: http://www.nap.edu/openbook.php?record_id=18607.
- 2287 124. Branum AM, Rossen LM, Schoendorf KC. Trends in caffeine intake among U.S. children
2288 and adolescents. *Pediatrics.* 2014;133(3):386-93. PMID: 24515508.
2289 <http://www.ncbi.nlm.nih.gov/pubmed/24515508>.
- 2290 125. Terry-McElrath YM, O'Malley PM, Johnston LD. Energy drinks, soft drinks, and
2291 substance use among United States secondary school students. *J Addict Med.*
2292 2014;8(1):6-13. PMID: 24481080. <http://www.ncbi.nlm.nih.gov/pubmed/24481080>.
- 2293 126. Arria AM, O'Brien MC. The "high" risk of energy drinks. *Jama.* 2011;305(6):600-1.
2294 PMID: 21266673. <http://www.ncbi.nlm.nih.gov/pubmed/21266673>.
- 2295 127. Centers for Disease Control and Prevention. Caffeine and Alcohol. 2014. Available
2296 from: <http://www.cdc.gov/alcohol/fact-sheets/cab.htm>.
- 2297 128. Substance Abuse Mental Health Services Administration. Drug Abuse Warning Network
2298 (DAWN), 2011: National Estimates of Drug-Related Emergency Department
2299 Visits.HHS. Publication No. (SMA) 13-4760, DAWN Series D-39 Rockville, MD2013.
2300 Available from: <http://www.samhsa.gov/data/2k13/DAWN2k11ED/DAWN2k11ED.htm>.
- 2301 129. Ferreira SE, de Mello MT, Pompéia S, de Souza-Formigoni ML. Effects of energy drink
2302 ingestion on alcohol intoxication. *Alcohol Clin Exp Res.* 2006;30(4):598-605. PMID:
2303 16573577. <http://www.ncbi.nlm.nih.gov/pubmed/16573577>.
- 2304 130. Wiklund U, Karlsson M, Ostrom M, Messner T. Influence of energy drinks and alcohol
2305 on post-exercise heart rate recovery and heart rate variability. *Clin Physiol Funct*
2306 *Imaging.* 2009;29(1):74-80. PMID: 19016812.
2307 <http://www.ncbi.nlm.nih.gov/pubmed/19016812>.
- 2308 131. Food and Drug Administration. Food additives permitted for direct addition to food for
2309 human consumption: aspartame. Federal Register1984.
- 2310 132. Magnuson BA, Burdock GA, Doull J, Kroes RM, Marsh GM, Pariza MW, et al.
2311 Aspartame: a safety evaluation based on current use levels, regulations, and toxicological

- 2312 and epidemiological studies. *Crit Rev Toxicol*. 2007;37(8):629-727. PMID: 17828671.
 2313 <http://www.ncbi.nlm.nih.gov/pubmed/17828671>.
- 2314 133. Food And Drug Administration. Food Additives Permitted for Direct Addition to Food
 2315 for Human Consumption 2014. Available from:
 2316 <http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm?fr=172.804>.
- 2317 134. Andreatta MM, Munoz SE, Lantieri MJ, Eynard AR, Navarro A. Artificial sweetener
 2318 consumption and urinary tract tumors in Cordoba, Argentina. *Prev Med*. 2008;47(1):136-
 2319 9. PMID: 18495230. <http://www.ncbi.nlm.nih.gov/pubmed/18495230>.
- 2320 135. Bosetti C, Gallus S, Talamini R, Montella M, Franceschi S, Negri E, et al. Artificial
 2321 sweeteners and the risk of gastric, pancreatic, and endometrial cancers in Italy. *Cancer*
 2322 *Epidemiol Biomarkers Prev*. 2009;18(8):2235-8. PMID: 19661082.
 2323 <http://www.ncbi.nlm.nih.gov/pubmed/19661082>.
- 2324 136. Bunin GR, Kushi LH, Gallagher PR, Rorke-Adams LB, McBride ML, Cnaan A.
 2325 Maternal diet during pregnancy and its association with medulloblastoma in children: a
 2326 children's oncology group study (United States). *Cancer Causes Control*. 2005;16(7):877-
 2327 91. PMID: 16132798. <http://www.ncbi.nlm.nih.gov/pubmed/16132798>.
- 2328 137. Cabaniols C, Giorgi R, Chinot O, Ferahta N, Spinelli V, Alla P, et al. Links between
 2329 private habits, psychological stress and brain cancer: a case-control pilot study in France.
 2330 *J Neurooncol*. 2011;103(2):307-16. PMID: 20835749.
 2331 <http://www.ncbi.nlm.nih.gov/pubmed/20835749>.
- 2332 138. Gallus S, Scotti L, Negri E, Talamini R, Franceschi S, Montella M, et al. Artificial
 2333 sweeteners and cancer risk in a network of case-control studies. *Ann Oncol*.
 2334 2007;18(1):40-4. PMID: 17043096. <http://www.ncbi.nlm.nih.gov/pubmed/17043096>.
- 2335 139. Hardell L, Mild KH, Pahlson A, Hallquist A. Ionizing radiation, cellular telephones and
 2336 the risk for brain tumours. *Eur J Cancer Prev*. 2001;10(6):523-9. PMID: 11916351.
 2337 <http://www.ncbi.nlm.nih.gov/pubmed/11916351>.
- 2338 140. Lim U, Subar AF, Mouw T, Hartge P, Morton LM, Stolzenberg-Solomon R, et al.
 2339 Consumption of aspartame-containing beverages and incidence of hematopoietic and
 2340 brain malignancies. *Cancer Epidemiol Biomarkers Prev*. 2006;15(9):1654-9. PMID:
 2341 16985027. <http://www.ncbi.nlm.nih.gov/pubmed/16985027>.
- 2342 141. Schernhammer ES, Bertrand KA, Birmann BM, Sampson L, Willett WC, Feskanich D.
 2343 Consumption of artificial sweetener- and sugar-containing soda and risk of lymphoma
 2344 and leukemia in men and women. *Am J Clin Nutr*. 2012;96(6):1419-28. PMID:
 2345 23097267. <http://www.ncbi.nlm.nih.gov/pubmed/23097267>.
- 2346 142. Halldorsson TI, Strom M, Petersen SB, Olsen SF. Intake of artificially sweetened soft
 2347 drinks and risk of preterm delivery: a prospective cohort study in 59,334 Danish pregnant
 2348 women. *Am J Clin Nutr*. 2010;92(3):626-33. PMID: 20592133.
 2349 <http://www.ncbi.nlm.nih.gov/pubmed/20592133>.
- 2350 143. Englund-Ogge L, Brantsaeter AL, Haugen M, Sengpiel V, Khatibi A, Myhre R, et al.
 2351 Association between intake of artificially sweetened and sugar-sweetened beverages and
 2352 preterm delivery: a large prospective cohort study. *Am J Clin Nutr*. 2012;96(3):552-9.
 2353 PMID: 22854404. <http://www.ncbi.nlm.nih.gov/pubmed/22854404>.
- 2354 144. La Vecchia C. Low-calorie sweeteners and the risk of preterm delivery: results from two
 2355 studies and a meta-analysis. *J Fam Plann Reprod Health Care*. 2013;39(1):12-3. PMID:
 2356 23296849. <http://www.ncbi.nlm.nih.gov/pubmed/23296849>.

- 2357 145. Shaywitz BA, Anderson GM, Novotny EJ, Ebersole JS, Sullivan CM, Gillespie SM.
 2358 Aspartame has no effect on seizures or epileptiform discharges in epileptic children. *Ann*
 2359 *Neurol.* 1994;35(1):98-103. PMID: 7506878.
 2360 <http://www.ncbi.nlm.nih.gov/pubmed/7506878>.
- 2361 146. Wolraich ML, Lindgren SD, Stumbo PJ, Stegink LD, Appelbaum MI, Kiritsy MC.
 2362 Effects of diets high in sucrose or aspartame on the behavior and cognitive performance
 2363 of children. *N Engl J Med.* 1994;330(5):301-7. PMID: 8277950.
 2364 <http://www.ncbi.nlm.nih.gov/pubmed/8277950>.
- 2365 147. Kruesi MJ, Rapoport JL, Cummings EM, Berg CJ, Ismond DR, Flament M, et al. Effects
 2366 of sugar and aspartame on aggression and activity in children. *Am J Psychiatry.*
 2367 1987;144(11):1487-90. PMID: 3674234. <http://www.ncbi.nlm.nih.gov/pubmed/3674234>.
- 2368 148. Roshon MS, Hagen RL. Sugar consumption, locomotion, task orientation, and learning in
 2369 preschool children. *J Abnorm Child Psychol.* 1989;17(3):349-57. PMID: 2666476.
 2370 <http://www.ncbi.nlm.nih.gov/pubmed/2666476>.
- 2371 149. Lapiere KA, Greenblatt DJ, Goddard JE, Harmatz JS, Shader RI. The neuropsychiatric
 2372 effects of aspartame in normal volunteers. *J Clin Pharmacol.* 1990;30(5):454-60. PMID:
 2373 2347957. <http://www.ncbi.nlm.nih.gov/pubmed/2347957>.
- 2374 150. Ryan-Harshman M, Leiter LA, Anderson GH. Phenylalanine and aspartame fail to alter
 2375 feeding behavior, mood and arousal in men. *Physiol Behav.* 1987;39(2):247-53. PMID:
 2376 3575461. <http://www.ncbi.nlm.nih.gov/pubmed/3575461>.
- 2377 151. Spiers PA, Sabounjian L, Reiner A, Myers DK, Wurtman J, Schomer DL. Aspartame:
 2378 neuropsychologic and neurophysiologic evaluation of acute and chronic effects. *Am J*
 2379 *Clin Nutr.* 1998;68(3):531-7. PMID: 9734727.
 2380 <http://www.ncbi.nlm.nih.gov/pubmed/9734727>.
- 2381 152. Stokes AF, Belger A, Banich MT, Bernadine E. Effects of alcohol and chronic aspartame
 2382 ingestion upon performance in aviation relevant cognitive tasks. *Aviat Space Environ*
 2383 *Med.* 1994;65(1):7-15. PMID: 8117231. <http://www.ncbi.nlm.nih.gov/pubmed/8117231>.
- 2384 153. Stokes AF, Belger A, Banich MT, Taylor H. Effects of acute aspartame and acute alcohol
 2385 ingestion upon the cognitive performance of pilots. *Aviat Space Environ Med.*
 2386 1991;62(7):648-53. PMID: 1898300. <http://www.ncbi.nlm.nih.gov/pubmed/1898300>.
- 2387 154. Pivonka EE, Grunewald KK. Aspartame- or sugar-sweetened beverages: effects on mood
 2388 in young women. *J Am Diet Assoc.* 1990;90(2):250-4. PMID: 2303661.
 2389 <http://www.ncbi.nlm.nih.gov/pubmed/2303661>.
- 2390 155. Walton RG, Hudak R, Green-Waite RJ. Adverse reactions to aspartame: double-blind
 2391 challenge in patients from a vulnerable population. *Biol Psychiatry.* 1993;34(1-2):13-7.
 2392 PMID: 8373935. <http://www.ncbi.nlm.nih.gov/pubmed/8373935>.
- 2393 156. Schiffman SS, Buckley CE, 3rd, Sampson HA, Massey EW, Baraniuk JN, Follett JV, et
 2394 al. Aspartame and susceptibility to headache. *N Engl J Med.* 1987;317(19):1181-5.
 2395 PMID: 3657889. <http://www.ncbi.nlm.nih.gov/pubmed/3657889>.
- 2396 157. Koehler SM, Glaros A. The effect of aspartame on migraine headache. *Headache.*
 2397 1988;28(1):10-4. PMID: 3277925. <http://www.ncbi.nlm.nih.gov/pubmed/3277925>.
- 2398 158. Van den Eeden SK, Koepsell TD, Longstreth WT, Jr., van Belle G, Daling JR, McKnight
 2399 B. Aspartame ingestion and headaches: a randomized crossover trial. *Neurology.*
 2400 1994;44(10):1787-93. PMID: 7936222. <http://www.ncbi.nlm.nih.gov/pubmed/7936222>.

- 2401 159. Lipton RB, Newman LC, Cohen JS, Solomon S. Aspartame as a dietary trigger of
2402 headache. *Headache*. 1989;29(2):90-2. PMID: 2708042.
2403 <http://www.ncbi.nlm.nih.gov/pubmed/2708042>.
- 2404 160. Camfield PR, Camfield CS, Dooley JM, Gordon K, Jollymore S, Weaver DF. Aspartame
2405 exacerbates EEG spike-wave discharge in children with generalized absence epilepsy: a
2406 double-blind controlled study. *Neurology*. 1992;42(5):1000-3. PMID: 1579221.
2407 <http://www.ncbi.nlm.nih.gov/pubmed/1579221>.
- 2408 161. Rowan AJ, Shaywitz BA, Tuchman L, French JA, Luciano D, Sullivan CM. Aspartame
2409 and seizure susceptibility: results of a clinical study in reportedly sensitive individuals.
2410 *Epilepsia*. 1995;36(3):270-5. PMID: 7614911.
2411 <http://www.ncbi.nlm.nih.gov/pubmed/7614911>.
- 2412 162. Centers for Disease Control and Prevention. CDC Estimates of Foodborne Illness in the
2413 United States 2014. Available from: [http://www.cdc.gov/foodborneburden/estimates-](http://www.cdc.gov/foodborneburden/estimates-overview.html)
2414 [overview.html](http://www.cdc.gov/foodborneburden/estimates-overview.html).
2415
2416
2417

Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance

INTRODUCTION

The *Dietary Guidelines for Americans, 2010* included guidance on sodium, saturated fat, and added sugars, and the 2015 DGAC determined that a reexamination of the evidence on these topics was necessary to evaluate whether revisions to the guidance were warranted. These topics were considered to be of public health importance because each has been associated with negative health outcomes when over-consumed. As the Committee considered it essential to address these topics across two or more Subcommittees, Working Groups were formed with representatives from the relevant Subcommittees to ensure that the topics were thoroughly addressed in a coordinated way. Additionally, the Committee acknowledged that a potential unintended consequence of a recommendation on added sugars might be that consumers and manufacturers replace added sugars with low-calorie sweeteners. As a result, the Committee also examined evidence on low-calorie sweeteners to inform statements on this topic. The updated findings in this chapter will help inform recommendations on these topics for the *2015 Dietary Guidelines for Americans*.

Although sodium, saturated fat, and added sugars are receiving particular focus here, it is important to consider these aspects of the diet in the context of a healthy dietary pattern. A healthy dietary pattern has little room for sodium, saturated fat, and added sugars. That said, these components of the diet are modifiable, and strategies at various levels of the socio-ecologic model, ranging from policy to consumer education, can promote shifts in intake to support healthy dietary patterns.

The sodium, saturated fat, and added sugars sections of this chapter provide introductory text related to the topic including the rationale and approach for the Committee’s review. Because the questions within each topic are so complementary, the DGAC choose to develop only one implications section for each topic.

LIST OF QUESTIONS

Sodium

1. What is the relationship between sodium intake and blood pressure in adults?
2. What is the relationship between sodium intake and blood pressure in children?
3. What is the relationship between sodium intake and cardiovascular disease outcomes?

- 36 4. What effect does the interrelationship of sodium and potassium have on blood pressure and
37 cardiovascular disease outcomes?
38

39 **Saturated Fat**

- 40 5. What is the relationship between intake of saturated fat and risk of cardiovascular disease?
41

42 **Added Sugars and Low-Calorie Sweeteners**

- 43 6. What is the relationship between the intake of added sugars and cardiovascular disease, body
44 weight/obesity, type 2 diabetes, and dental caries?
45 7. What is the relationship between the intake of low-calorie sweeteners and body
46 weight/obesity and type 2 diabetes?
47

48 **METHODOLOGY**

49 To answer the questions in this chapter, the Committee relied on existing reports, original
50 Nutrition Evidence Library (NEL) systematic reviews, and NEL updates. The Committee
51 followed the methods described in *Part C. Methodology* without modification to answer these
52 questions. Because the DGAC knew strong existing reports, systematic reviews (SRs), and meta-
53 analyses (MA) were available related to most of the cross-cutting questions, to prevent
54 duplication of efforts, the DGAC relied on these reviews in lieu of conducting original NEL
55 systematic reviews. In some cases, existing reviews, SRs, or MA were not available or required
56 updating. In these cases, NEL systematic reviews or updates were conducted. Complete
57 information on the NEL reviews and updates is provided at www.NEL.gov. The reader also is
58 directed to the original existing reports, which are referenced throughout the chapter, for
59 additional information.
60

61 Four questions addressed dietary sodium intake. For Question 1, the Committee used the 2013
62 National Heart, Lung, and Blood Institute (NHLBI) *Lifestyle Interventions to Reduce*
63 *Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group*¹ and the
64 associated American Heart Association (AHA)/ American College of Cardiology (ACC)
65 *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*.² Although new studies
66 examining the relationship between sodium and blood pressure have been published since the
67 completion of the NHLBI review, including findings from the Prospective Urban Rural
68 Epidemiology (PURE) study,³ the Committee determined the evidence presented in the SR
69 conducted by NHLBI, linking sodium and blood pressure, was strong and that consideration of
70 more recent findings would not change the conclusions. Thus, the Committee did not update the
71 review. For Question 2, the Committee updated the NEL systematic review on sodium and blood
72 pressure in children conducted by the 2010 DGAC. The data reviewed for this question by the
73 2010 DGAC included children, birth to age 18, and the 2015 DGAC updated the sodium review

74 using the same age range. For Question 3, the Committee relied on the NHLBI systematic review
 75 from the Lifestyle Work Group¹ as well as the 2013 Institute of Medicine (IOM) report, *Sodium*
 76 *Intake in Populations*.⁴ Additionally, because the quality and quantity of the evidence on sodium
 77 and cardiovascular disease (CVD) that was used in the two reports is limited, the Committee
 78 updated the sodium and CVD review using a NEL systematic review update from January 2013
 79 to July 2014. The final question in the sodium section, Question 4, also was answered using the
 80 recent NHLBI systematic review from the Lifestyle Work Group.¹ The Committee also used the
 81 2010 IOM Report on *Strategies to Reduce Sodium Intake in the United States* to inform the
 82 implications statements for these questions.⁵

83
 84 Regarding saturated fat, Question 5 was answered using the NHLBI systematic review¹ and
 85 related AHA/ACC *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*,² which
 86 focused on randomized controlled trials (RCTs), as well as existing SRs and MA addressing this
 87 question published in peer-reviewed literature between January 2009 and August 2014.
 88 Particular emphasis was placed on reviews that examined the macronutrient replacement for
 89 saturated fat.

90
 91 The remaining questions in this chapter examined added sugars and low-calorie sweeteners. For
 92 Question 6, the DGAC relied on systematic reviews commissioned by the World Health
 93 Organization (WHO) to address body weight⁶ and dental caries.⁷ Additionally, to capture new
 94 research, the Committee searched for SRs and MA published since January 2012, the completion
 95 of the WHO reviews. Type 2 diabetes was not addressed by the WHO, and therefore, the
 96 Committee relied on existing SRs/MA published since January 2010 to address this health
 97 outcome. No existing SRs/MA examine added sugars and CVD, so the Committee conducted an
 98 original NEL systematic review to address this question (see <http://NEL.gov/topic.cfm?cat=3376>
 99 for complete information on this review). Question 7 on low-calorie sweeteners was answered
 100 using existing SRs/MA published from January 2010 to August 2014. For low-calorie
 101 sweeteners, the Committee was initially interested in the health outcomes of body weight, type 2
 102 diabetes, CVD, and dental caries. However, existing reviews were available only for body
 103 weight and type 2 diabetes. The Committee did not conduct an original NEL systematic review
 104 on CVD or dental caries because of limited time and resources, and because the Committee did
 105 not think sufficient evidence was available to address these health outcomes.

106
 107

108 **SODIUM**

109 **Introduction**

110 From its first edition in 1980, the *Dietary Guidelines for Americans* consistently recommended
 111 the public reduce dietary sodium intakes in order to prevent and treat hypertension, CVD, and
 Scientific Report of the 2015 Dietary Guidelines Advisory Committee

112 stroke. This recommendation is based on evidence supporting a dose-dependent relationship
 113 between sodium intake and blood pressure and observational data identifying associations
 114 between sodium intake and blood pressure and cardiovascular outcomes. However, despite many
 115 years of accumulating evidence and public health guidelines focused on changing individual
 116 behavior to achieve a reduced sodium intake among Americans, consumption continues to far
 117 exceed recommendations. The DGAC has identified dietary sodium as a nutrient of public health
 118 concern because of overconsumption, with usual intakes for those ages 2 years and older at 3,463
 119 mg/day.⁸ Sodium is ubiquitous in the current U.S. food supply and multiple food categories
 120 contribute to excessive sodium intake (see *Part D. Chapter 1: Food and Nutrient Intakes, and*
 121 *Health: Current Status and Trends*, Figure D1.35).

122
 123 Currently, 30 percent of U.S. adults have high blood pressure (see *Part D. Chapter 1: Food and*
 124 *Nutrient Intakes, and Health: Current Status and Trends*). Furthermore, the estimated lifetime
 125 risk of developing hypertension in the U.S. is 90%. The rate of borderline high blood pressure
 126 (defined as a systolic or diastolic blood pressure ≥ 90 th percentile but < 95 th percentile or blood
 127 pressure levels $\geq 120/80$ mm Hg) in youth ages 8 to 17 years is highest in those who are obese
 128 (16.2 percent), slightly lower in those who are overweight (11 percent); and this condition is
 129 present even in those who are normal weight (5 percent). Dietary sodium reduction can
 130 effectively prevent and reduce high blood pressure.⁹⁻¹¹ Given the long-standing awareness of
 131 this health concern and scientific foundation for dietary treatment, the DGAC conducted a
 132 focused review of dietary sodium and its relationship with blood pressure as well as its
 133 relationship with CVD.

134

135 **Question 1: What is the relationship between sodium intake and blood pressure**
 136 **in adults?**

137 **Source of evidence:** Existing reports

138

139 **Conclusions**

140 The DGAC concurs with the three conclusions from the 2013 AHA/ACC Lifestyle Guideline
 141 that apply to adults who would benefit from blood pressure lowering.

142

143 The DGAC concurs that adults who would benefit from blood pressure lowering should “lower
 144 sodium intake.” AHA/ACC Grade: Strong; **DGAC Grade: Strong**

145

146 The DGAC concurs that adults who would benefit from blood pressure lowering should
 147 “Consume no more than 2,400 mg of sodium/day.” The report also indicates that “Further
 148 reduction of sodium intake to 1,500 mg/d can result in even greater reduction in blood pressure”;

149 and concludes that “Even without achieving these goals, reducing sodium intake by at least 1,000
150 mg/d lowers blood pressure.” AHA/ACC Grade: Moderate; **DGAC Grade: Moderate**

151
152 The DGAC concurs that adults who would benefit from blood pressure lowering should
153 “Combine the DASH dietary pattern with lower sodium intake.” AHA/ACC Grade: Strong;
154 **DGAC Grade: Strong**

155 156 **Review of the Evidence**

157 The 2013 AHA/ACC Lifestyle Guideline and associated NHLBI Lifestyle Report summarized
158 strong and consistent evidence that supports dietary sodium reduction as a means to prevent and
159 treat high blood pressure. The studies used to inform the conclusion to lower sodium intake were
160 conducted in older and younger adults, individuals with prehypertension and hypertension, men
161 and women, and African American and non-African American adults. The trials also
162 documented positive effects of sodium reduction that were independent of weight change; and
163 include behavioral interventions where individuals were counseled to reduce sodium, as well as
164 feeding studies.

165
166 The recommendation to combine the DASH dietary pattern with lower sodium is based heavily
167 on the results of the DASH sodium trial, which showed clinically significant lowering of blood
168 pressure with sodium intake of 2,400 mg/day and even lower blood pressure with sodium intake
169 of 1,500 mg/day. The goal of 2,400 or less mg/day was selected because it is the estimated
170 average urinary sodium excretion in the DASH sodium trial.

171
172 The recommendation to reduce sodium intake by 1,000 mg/day even if goals for 2,400 mg/day or
173 1,500 mg/day cannot be reached comes from studies where this level of sodium reduction was
174 beneficial for blood pressure lowering.

175
176 The differences in the evidence grade for the three conclusions related to sodium and blood
177 pressure in adults results from the differences in the number and power of clinical trials
178 supporting each recommendation. For example, a grade of “moderate” was assigned to the
179 second conclusion because fewer clinical trials informed the goals of 2,400 and 1,500 mg/day
180 than for the overall goal of sodium reduction.

181
182 ***For additional details on this body of evidence, visit:*** References 1, 2, 4 and 9 and *Appendix E-*
183 *2.42*

184
185 **Question 2: What is the relationship between sodium intake and blood pressure**
186 **in children?**

187 **Source of evidence:** Existing systematic review with a NEL systematic review update
Scientific Report of the 2015 Dietary Guidelines Advisory Committee

188

189 **Conclusions**

190 The 2015 DGAC concurs with the 2010 DGAC that “a moderate body of evidence has
191 documented that as sodium intake decreases, so does blood pressure in children, birth to age 18
192 years.” **DGAC Grade: Moderate**

193

194 **Review of the Evidence**

195 The 2010 DGAC conducted a systematic review to examine the relationship between sodium
196 intake and blood pressure in children from birth to age 18 years, examining studies published
197 from January 1970 to May 2009. That systematic review included 19 articles from 15
198 intervention studies and four prospective cohort studies.

199

200 The 2015 DGAC updated this systematic review and identified two additional articles published
201 since May 2009, including one RCT and one prospective cohort study.^{12, 13}

202

203 The 2015 DGAC considered the evidence reviewed by the 2010 DGAC related to dietary sodium
204 intake and blood pressure in children, and determined that, based on the two new studies
205 identified in the updated search, changes were not warranted to the conclusion statement or
206 grade. In aggregate, the data reviewed by the 2010 DGAC indicated that sodium reduction
207 modestly lowers BP in infants and children. Neither of the two studies identified in the update
208 found a relationship between dietary sodium intake and blood pressure in healthy, normotensive
209 children.

210

211 *For additional details on this body of evidence, visit:*

212 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250452

213

214 **Question 3: What is the relationship between sodium intake and cardiovascular**
215 **disease outcomes?**

216 **Source of evidence:** Existing report with a NEL systematic review update

217

218 **Conclusions**

219 The DGAC concurs with the IOM Report: *Sodium Intake in Populations*, which concluded that
220 “although the reviewed evidence on associations between sodium intake and direct health
221 outcomes has methodological flaws and limitations, when considered collectively, it indicates a
222 positive relationship between higher levels of sodium intake and risk of CVD. This evidence is
223 consistent with existing evidence on blood pressure as a surrogate indicator of CVD risk.” IOM
224 Grade: Grade not determined, outside the statement of task; **DGAC Grade: Moderate**

225

226 The DGAC concurs with the IOM Report: *Sodium Intake in Populations* that “evidence from
227 studies on direct health outcomes is inconsistent and insufficient to conclude that lowering
228 sodium intakes below 2,300 mg/day either increases or decreases risk of CVD outcomes
229 (including stroke and CVD mortality) or all-cause mortality in the general U.S. population.”
230 IOM Grade: Grade not determined, outside the statement of task; **DGAC Grade: Grade not**
231 **assignable**

232
233 The DGAC concurs with the NHLBI Lifestyle Report, which concluded that “a reduction in
234 sodium intake by approximately 1,000 mg/day reduces CVD events by about 30 percent” and
235 that “higher dietary sodium intake is associated with a greater risk for fatal and nonfatal stroke
236 and CVD.” NHLBI Strength of Evidence: Low; **DGAC Grade: Limited**

237
238 The DGAC concurs with the NHLBI Lifestyle Report that “evidence is not sufficient to
239 determine the association between sodium intake and the development of heart failure.” NHLBI
240 Strength of Evidence: Not assigned due to insufficient evidence; **DGAC Grade: Grade not**
241 **Assignable**

242

243 **Review of the Evidence**

244 The DGAC updated systematic reviews done in 2013 by the IOM⁴ and NHLBI,¹ and identified
245 four additional articles published since 2013, all of which were prospective cohort studies.¹⁴⁻¹⁷

246

247 Of note, the evidence reviewed for the 2013 IOM report was published between 2003 and
248 December 2012. The DGAC concluded that the reviewed evidence on associations between
249 sodium intake and direct health outcomes has methodological flaws and limitations. Specifically,
250 the Committee documented the small number of well-conducted studies evaluating sodium
251 intake and direct health outcomes; the inconsistency in findings across the published literature,
252 possibly due to methodological factors; the lack of comparability in sodium intake levels across
253 studies particularly in international studies; and the absence of strong data related to sodium
254 goals and direct health outcomes, not including hypertension.

255

256 The DGAC considered the conclusions reached by the IOM and NHLBI related to dietary
257 sodium intake and risk of CVD, and determined that the findings from the four new studies
258 identified in the updated search did not warrant changes to the conclusion statements. In
259 aggregate, the data indicate a relationship between higher sodium intake and higher risk of CVD.

260

261 ***For additional details on this body of evidence, visit:***

262 http://NEL.gov/conclusion.cfm?conclusion_statement_id=250457

263

264 **Question 4: What effect does the interrelationship of sodium and potassium have**
 265 **on blood pressure and cardiovascular disease outcomes?**

266 **Source of evidence:** Existing report

267

268 **Conclusions**

269 The DGAC concurs with the NHLBI Lifestyle Report that: “Evidence is not sufficient to
 270 determine whether increasing dietary potassium intake lowers blood pressure.” NHLBI Strength
 271 of Evidence: Not assigned due to insufficient evidence; **DGAC Grade: Not Assignable**

272

273 The DGAC concurs with the NHLBI Lifestyle Report that: “In observational studies with
 274 appropriate adjustments (e.g., blood pressure, sodium intake), higher dietary potassium intake is
 275 associated with lower risk for stroke.” NHLBI Strength of Evidence: Low; **DGAC Grade:**

276 **Limited**

277

278 The DGAC concurs with the NHLBI Lifestyle Report that: “Evidence is not sufficient to
 279 determine an association between dietary potassium intake and coronary heart disease (CHD),
 280 heart failure, and cardiovascular mortality.” NHLBI Strength of Evidence: Not assigned due to
 281 insufficient evidence; **DGAC Grade: Grade not Assignable**

282

283 **Review of the Evidence**

284 The NHLBI Lifestyle Report summarized limited evidence on the relationship between
 285 potassium intake and blood pressure, CHD, heart failure, cardiovascular mortality, or stroke.
 286 Although it is postulated that a high ratio of sodium intake to potassium intake is a stronger risk
 287 factor for hypertension than either factor alone, the evidence base to support this hypothesis is
 288 insufficient for drawing definitive conclusions. Although results of epidemiologic studies
 289 suggest that potassium consumption influences the risk of CVD, the strength of the evidence is
 290 insufficient to draw conclusions about CHD, heart failure, or cardiovascular mortality. The
 291 evidence is limited with regard to stroke, coming from studies with weaker designs in which
 292 investigators were able to make appropriate statistical adjustments for potential confounders of
 293 the relationship.

294

295 *For additional details on this body of evidence, visit:* References 1 and 2

296

297 **Implications**

298 The current average sodium intake in the United States is 3,478 mg/d, far exceeding
 299 recommendations. Given the well-documented relationship between sodium intake and high
 300 blood pressure, sodium intake should be reduced and combined with a healthful dietary pattern

301 (as described in *Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health*
302 *Outcomes*).

303
304 The general population, ages 2 years and older, should rely on the recommendations of the IOM
305 Panel on Dietary Reference Intakes for Electrolytes and Water.⁹ A tolerable upper limit was set
306 by the Panel at 2,300 mg/day based on evidence showing associations between high sodium
307 intake, high blood pressure, and subsequent risk of heart disease, stroke, and mortality. Of note,
308 the AHA/ACC recommendation of less than 2,400 mg/day (see conclusions for sodium question
309 1) is slightly different than the less than 2,300 mg/day recommended by the IOM Panel on
310 Dietary Reference Intakes or the 2010 Dietary Guidelines for Americans; less than 2,400 mg/day
311 was selected because it was the estimated average urinary sodium excretion in the DASH-
312 sodium trial.

313
314 Individuals who would benefit from blood pressure lowering (i.e., those with prehypertension or
315 hypertension), should rely on the recommendations in the 2013 AHA/ACC Lifestyle Guideline.
316 These include: lowering sodium intake in general; or consuming no more than 2,400 mg of
317 sodium/day; or lowering sodium intake to 1,500 mg per day for even greater reduction in blood
318 pressure; or lowering sodium intake by at least 1,000 mg per day even if the goals of 2,400 or
319 1,500 mg per day cannot be met.

320
321 For decades, sodium intake in the United States has exceeded recommendations in spite of
322 numerous national campaigns, through programs such as the NHLBI's National High Blood
323 Pressure Education Program and the CDC's State Heart Disease and Stroke Prevention Program,
324 focused on individual behavior change for sodium reduction. As described in *Part D. Chapter 1:*
325 *Food and Nutrient Intakes, and Health: Current Status and Trends*, sodium is ubiquitous in
326 the U.S. food supply and almost all food categories contribute to intake levels. This unique
327 feature of sodium makes it difficult for individuals to achieve recommended intake. As such, we
328 recommend that a primary emphasis be placed on policies and population-based strategies for
329 sodium reduction while at the same time paying attention to consumer education. Local, state,
330 and Federal agencies should consider a comprehensive and coordinated strategy, that includes
331 partnerships with the food industry, to reduce the sodium content of foods in the United States
332 based on the socio-ecological model highlighted in the 2015 DGAC's conceptual model (see
333 *Part B. Chapter 1: Introduction*).

334
335 These strategies should be consistent with the recommendation described in the 2010 IOM report
336 on *Strategies to Reduce Sodium Intake in the United States*.⁵ The primary strategy that was
337 recommended is that "The FDA should expeditiously initiate a process to set mandatory national
338 standards for the sodium content of foods". This would include: 1) "a modification of the
339 generally recognized as safe (GRAS) status of salt added to processed foods in order to reduce
340 the salt content of the food supply in a stepwise manner"; 2) "FDA should likewise extend its

341 stepwise application of the GRAS modification, adjusted as necessary, to encompass salt added
342 to menu items offered by restaurant/foodservice operations that are sufficiently standardized so
343 as to allow practical implementation”; and 3) “FDA should revisit the GRAS status of other
344 sodium-containing compounds as well as any food additive provisions for such compounds and
345 make adjustments as appropriate, consistent with changes for salt in processed foods and
346 restaurant/foodservice menu items.”

347
348 Population sodium reductions efforts should consider: 1) the varied technical and functional roles
349 that sodium plays in foods and the complexity of reducing sodium in foods; 2) the recent
350 accomplishments and voluntary reduction efforts by the food industry; and 3) consumer demand
351 for lower-sodium products. More information about strategies for reducing sodium intake in the
352 United States can be found in the IOM report, at <http://www.iom.edu/Reports/2010/Strategies-to-Reduce-Sodium-Intake-in-the-United-States.aspx>.

353
354
355 Informative food labels should be used to effectively promote awareness of sodium content in
356 foods. Consumers would benefit from a standardized, easily understood front-of-package (FOP)
357 label on all food and beverage products to give clear guidance about a food’s healthfulness. An
358 example is the FOP label recommended by the IOM,¹⁸ which included calories, and 0 to 3
359 “nutritional” points for added sugars, saturated fat, and sodium. This would be integrated with
360 the Nutrition Facts Panel, allowing consumers to quickly and easily identify nutrients of concern
361 for over-consumption, in order to make healthier choices.

362
363 Public-private-community partnerships should be created to reduce sodium levels in
364 commercially processed and restaurant foods.

365
366 Strategies that complement policies and support consumers to make dietary behavior changes
367 also are needed. These include (but are not limited to): 1) nutrition services and comprehensive
368 lifestyle interventions by multidisciplinary teams;² 2) widely available diet planning tools that
369 include sodium as an area of focus; and 3) educational programs that teach adults simple recipes
370 that emphasize flavoring unsalted foods with spices and herbs.

371
372 Although the evidence on potassium and blood pressure is limited, the DGAC recognizes
373 potassium as a nutrient of concern (see **Part D. Chapter 1: Food and Nutrient Intakes, and**
374 **Health: Current Status and Trends**) and encourages increased potassium intake through
375 potassium-rich foods such as vegetables and fruits (see Table D1.7).

376
377 Interventions, preferably nonpharmacologic, are needed for children because borderline high
378 blood pressure occurs concomitantly with overweight, obesity, and other cardio-metabolic risk
379 factors (see **Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and**
380 **Trends**). Evidence-based strategies in clinical and public health settings need to be implemented

381 and complemented by environmental approaches to reverse these high priority health problems
382 in children.

383
384 For blood pressure lowering and hypertension prevention, action is needed at both the individual
385 and population levels.

386
387 Sodium reduction in youth will require changes in their food environments and school and
388 community-based education on healthful eating.

389
390 School systems should adopt mandatory age-appropriate nutrition and physical activity curricula
391 (K-12) that incorporate the core principles of the future *2015 Dietary Guidelines*.

392
393

394 **SATURATED FAT**

395 **Introduction**

396 The relationship between different types of dietary fats and risk of CVD has been extensively
397 studied in RCTs and epidemiologic studies. It is now well-established that higher intake of *trans*
398 fat from partially hydrogenated vegetable oils is associated with increased risk of CVD and thus,
399 should be minimized in the diet. Numerous RCTs have demonstrated that saturated fat (SFA) as
400 compared to mono- (MUFA) or polyunsaturated fats (PUFA) or carbohydrates increases total
401 and LDL cholesterol. Thus, limiting saturated fat consumption has been a longstanding dietary
402 recommendation to reduce risk of CVD. In particular, previous DGACs have recommended
403 consuming no more than 10 percent of daily calories from saturated fat.

404
405 However, recent meta-analyses of prospective observational studies did not find a significant
406 association between higher saturated fat intake and risk of CVD in large populations. These data
407 have re-ignited the debate regarding the current recommendation to limit saturated fat intake.
408 Therefore, the DGAC chose to conduct a focused review of published systematic reviews and
409 meta-analyses on saturated fat intake and CVD. A central issue in the relationship between
410 saturated fat and CVD is the specific macronutrients that are used to replace it because
411 consuming unsaturated fats versus carbohydrates in place of saturated fat can have different
412 effects on blood lipids and risk of CVD. Thus, the Committee's assessment of the available
413 evidence puts greater emphasis on the replacement macronutrient for saturated fat.

414
415 In the United States, the top sources of foods contributing to saturated fat intake are mixed
416 dishes, particularly burgers and sandwiches, and snacks and sweets (see *Part D. Chapter 1:*
417 *Food and Nutrient Intakes, and Health: Current Status and Trends*). Although saturated fat
418 intake has declined in the past decades, current intake is still high at a median of 11.1 percent of
Scientific Report of the 2015 Dietary Guidelines Advisory Committee

419 daily calories (see *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status*
 420 *and Trends*). Therefore, saturated fat continues to be an area of public health concern and the
 421 DGAC deemed it important to re-evaluate and update the knowledge base on saturated fat intake
 422 and CVD risk.

423

424 **Question 5: What is the relationship between intake of saturated fat and risk of**
 425 **cardiovascular disease?**

426 **Source of evidence:** Existing reports

427

428 **Conclusions**

429 Strong and consistent evidence from RCTs shows that replacing SFA with unsaturated fats,
 430 especially PUFA, significantly reduces total and LDL cholesterol. Replacing SFA with
 431 carbohydrates (sources not defined) also reduces total and LDL cholesterol, but significantly
 432 increases triglycerides and reduces HDL cholesterol.

433

434 Strong and consistent evidence from RCTs and statistical modeling in prospective cohort studies
 435 shows that replacing SFA with PUFA reduces the risk of CVD events and coronary mortality.
 436 For every 1 percent of energy intake from SFA replaced with PUFA, incidence of CHD is
 437 reduced by 2 to 3 percent. However, reducing total fat (replacing total fat with overall
 438 carbohydrates) does not lower CVD risk. Consistent evidence from prospective cohort studies
 439 shows that higher SFA intake as compared to total carbohydrates is not associated with CVD
 440 risk. **DGAC Grade: Strong**

441

442 Evidence is limited regarding whether replacing SFA with MUFA confers overall CVD (or CVD
 443 endpoint) benefits. One reason is that the main sources of MUFA in a typical American diet are
 444 animal fat, and because of the co-occurrence of SFA and MUFA in foods makes it difficult to
 445 tease out the independent association of MUFA with CVD. However, evidence from RCTs and
 446 prospective studies has demonstrated benefits of plant sources of monounsaturated fats, such as
 447 olive oil and nuts on CVD risk. **DGAC Grade: Limited**

448

449 **Implications**

450 Recommendations on saturated fat intake should specify replacement macronutrients and
 451 emphasize replacing saturated fat with unsaturated fats, especially polyunsaturated fats. The
 452 Committee recommends retaining the 10 percent upper limit for saturated fat intake. In practice,
 453 non-hydrogenated vegetable oils that are high in unsaturated fats and relatively low in SFA (e.g.,
 454 soybean, corn, olive, and canola oils) instead of animal fats (e.g., butter, cream, beef tallow, and
 455 lard) or tropical oils (e.g., palm, palm kernel, and coconut oils) should be recommended as the
 456 primary source of dietary fat. Partially hydrogenated oils containing *trans* fat should be avoided.

457

458 In low-fat diets, fats are often replaced with refined carbohydrates and this is of particular
459 concern because such diets are generally associated with dyslipidemia (hypertriglyceridemia and
460 low HDL-C concentrations). Therefore, dietary advice should put the emphasis on optimizing
461 types of dietary fat and not reducing total fat.

462

463 When individuals reduce consumption of refined carbohydrates and added sugars, they should
464 not replace them with foods high in saturated fat. Instead, refined carbohydrates and added
465 sugars should be replaced by healthy sources of carbohydrates (e.g., whole grains, legumes,
466 vegetables, and fruits), and healthy sources of fats (e.g., non-hydrogenated vegetable oils that are
467 high unsaturated fats, and nuts/seeds). The consumption of “low-fat” or “nonfat” products with
468 high amounts of refined grains and added sugars should be discouraged.

469

470 Dietary recommendations on macronutrient composition for reducing CVD risk should be
471 dietary pattern-based emphasizing foods that characterize healthy dietary patterns (see *Part D.*
472 *Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes*). Individuals are
473 encouraged to consume dietary patterns that emphasize vegetables, fruits, whole grains, legumes,
474 and nuts; include low- and non-fat dairy products, poultry, seafood, non-tropical vegetable oils;
475 limit sodium, saturated fat, refined grains, sugar-sweetened foods and beverages, and are lower
476 in red and processed meats. Multiple dietary patterns can achieve these food and nutrient patterns
477 and are beneficial for cardiovascular health, and they should be tailored to individuals’ biological
478 needs and food preferences.

479

480 **Review of the Evidence**

481 The DGAC drew evidence from SRs or MA published between January 2009 and August 2014
482 in English in a peer-reviewed journal, which included RCTs and/or prospective cohort studies.
483 Participants included healthy volunteers as well as individuals at elevated chronic disease risk.
484 The main exposure was SFA, and the main outcomes included LDL-cholesterol (LDL-C), HDL-
485 cholesterol (HDL-C), triglycerides (TG), blood pressure (BP), and incidence of CVD and CHD,
486 CVD- and CHD-related death, myocardial infarction, or stroke. All reviews were high-quality,
487 with ratings ranging from 8 to 11 on AMSTAR. The Committee drew evidence on blood lipids
488 and blood pressure outcomes from the AHA/ACC Lifestyle Guideline and the associated NHLBI
489 Lifestyle Report, which included primarily RCTs on intermediate CVD risk factors. The
490 Committee drew evidence on CVD endpoints and effect size estimates from seven published MA
491 that included one or more studies not covered in these reports.¹⁹⁻²⁵ Little evidence on the
492 contribution of SFA to cardiovascular risk factors in the pediatric populations was available, and
493 that which was published has not been systematically reviewed.

494

495 *Effects of Replacing SFA on LDL-C, HDL-C, and TG*

496 Macronutrients may affect plasma lipids and lipoproteins, which are strong predictors of CVD
497 risk. The NHLBI Lifestyle Report summarized evidence from three feeding trials examining
498 effects on LDL-C of dietary patterns with varying SFA levels: DASH (Dietary Approaches to
499 Stop Hypertension), DASH-Sodium, and DELTA (Dietary Effects on Lipoproteins and
500 Thrombogenic Activity). The results from these trials indicate that reducing total and saturated
501 fat led to a significant reduction in LDL cholesterol in the context of the DASH dietary pattern
502 and the National Cholesterol Education Program (NCEP) Step 1 diet. To estimate the effects of
503 replacing SFA by specific macronutrients such as carbohydrates, MUFA, or PUFA, the NHLBI
504 Lifestyle Report also included two MA from Mensink and Katan (n=1,672), covering the period
505 from 1970 to 1998 (27 controlled trials in the first MA and 60 controlled trials in the second
506 MA) and using the same inclusion/exclusion criteria to estimate changes in plasma lipids when
507 substituting dietary SFA with carbohydrates or other fat types and holding dietary cholesterol
508 constant.^{26, 27} Mensink and Katan found that replacing 1 percent of SFA with an equal amount of
509 carbohydrates, MUFA, or PUFA led to comparable LDL-C reductions: 1.2, 1.3, and 1.8 mg/dL,
510 respectively. Replacing 1 percent of SFA with carbohydrates, MUFA, or PUFA also lowered
511 HDL-C by 0.4, 1.2, and 0.2 mg/dL, respectively. Replacing 1 percent of carbohydrates by an
512 equal amount of MUFA or PUFA raised LDL-C by 0.3 and 0.7 mg/dL, raised HDL-C by 0.3 and
513 0.2 mg/dL, and lowered TG by 1.7 and 2.3 mg/dL, respectively. The 2003 MA by Mensink and
514 Katan²⁷ indicated that the ratio of total to HDL-C, a stronger predictor of CVD risk than total or
515 LDL cholesterol alone, did not change when SFA was replaced by carbohydrates, but the ratio
516 significantly decreased when SFA was replaced by unsaturated fats, especially PUFA.

517
518 In summary, strong and consistent evidence from RCTs shows that replacing SFA with
519 unsaturated fats, especially PUFA, significantly reduces total and LDL cholesterol. Replacing
520 SFA with carbohydrates also reduces total and LDL cholesterol, but significantly increases TG
521 and reduces HDL cholesterol. However, the evidence of beneficial effects on one risk factor does
522 not rule out neutral or opposite effects on unstudied risk factors. To better assess the overall
523 effects of intervention to reduce or modify SFA intake, studies of clinical endpoints are
524 summarized below.

**525
526 *The Relationship between Consumption of Total Fat and SFA and Risk of CVD***

527 A MA by Skeaff et al. in 2009 included 28 U.S. and European cohorts (6,600 CHD deaths
528 among 280,000 participants) and found no clear relationship between total or SFA intake and
529 CHD events or deaths.²⁵ Similarly, Siri-Tarino et al., 2010 found that SFA intake was not
530 associated with risk of CHD, stroke or cardiovascular disease.²⁴ The Siri-Tarino et al., 2010
531 meta-analysis included data from 347,747 participants (11,006 developed CVD) in 21 unique
532 studies, with 16 studies providing risk estimates for CHD and 8 studies providing data for stroke
533 as an endpoint. In the 2012 MA of trials to reduce or modify intake of SFA, Hooper et al. also
534 found no significant associations of total fat reduction with cardiovascular events or mortality.
Scientific Report of the 2015 Dietary Guidelines Advisory Committee

535 Consistent with these prior studies, Chowdhury et al.'s 2014 MA of total SFA also did not
536 specify what macronutrient substituted SFA and again found no association of dietary SFA
537 intake, nor of circulating SFA, with coronary disease.¹⁹ Chowdhury et al. included data from 32
538 observational studies (530,525 participants) of fatty acids from dietary intake, 17 observational
539 studies (25,721 participants) of fatty acid biomarkers, and 27 RCTs (103,052 participants) of
540 fatty acid supplementation.

541
542 The results described above do not explicitly specify the comparison or replacement nutrient, but
543 typically it consists largely of carbohydrates (sources not defined). These results suggest that
544 replacing SFA with carbohydrates is not associated with CVD risk. Taken together, these results
545 suggest that simply reducing SFA or total fat in the diet by replacing it with any type of
546 carbohydrates is not effective in reducing risk of CVD.

547
548 ***Effects of Replacing SFA with Polyunsaturated Fat or Carbohydrates on CVD Events***

549 Hooper et al.'s 2012 Cochrane MA of trials of SFA reduction/modification found that reducing
550 SFA by reducing and/or modifying dietary fat reduced the risk of cardiovascular events by 14
551 percent (pooled RR = 0.86; 95% CI = 0.77 to 0.96, with 24 comparisons and 65,508 participants
552 of whom 7 percent had a cardiovascular event, I² = 50%).²¹ Subgroup analyses revealed this
553 protective effect was driven by dietary fat *modification* rather than reduction and was only
554 apparent in longer trials (2 years or more). Despite the reduction in total cardiovascular events,
555 there was no clear evidence of reductions in any individual outcome (total or non-fatal
556 myocardial infarction, stroke, cancer deaths or diagnoses, diabetes diagnoses), nor was there any
557 evidence that trials of reduced or modified SFA reduced cardiovascular mortality. These results
558 suggest that modifying dietary fat by replacing some saturated (animal) fats with plant oils and
559 unsaturated spreads may reduce risk of heart and vascular disease.

560
561 Emphasizing the benefits of replacement of saturated with polyunsaturated fats, Mozaffarian et
562 al., 2010 found in a MA of 8 trials (13,614 participants with 1,042 CHD events) that modifying
563 fat reduced the risk of myocardial infarction or coronary heart disease death (combined) by 19
564 percent (RR = 0.81; 95% CI = 0.70 to 0.95; p = 0.008), corresponding to 10 percent reduced
565 CHD risk (RR = 0.90; 95% CI = 0.83 to 0.97) for each 5 percent energy of increased PUFA.²³
566 This magnitude of effect is similar to that observed in the Cochrane MA. In secondary analyses
567 restricted to CHD mortality events, the pooled RR was 0.80 (95% CI = 0.65 to 0.98). In
568 subgroup analyses, the RR was greater in magnitude in the four trials in primary prevention
569 populations but non-significant (24 percent reduction in CHD events) compared to a significant
570 reduction of 16 percent in the four trials of secondary prevention populations. Mozaffarian et al.
571 argue that the slightly greater risk reduction in studies of CHD events, compared with predicted
572 effects based on lipid changes alone, is consistent with potential additional benefits of PUFA on
573 other non-lipid pathways of risk, such as insulin resistance. Many of the included trials used

574 vegetable oils containing small amounts of plant-derived n-3 PUFA in addition to omega-6
575 PUFA.

576
577 Consistent with the benefits of replacing SFA with PUFA for prevention of CHD shown in other
578 studies, Farvid et al., 2014 conducted an SR and MA of prospective cohort studies of dietary
579 linoleic acid (LA), which included 13 studies with 310,602 individuals and 12,479 total CHD
580 events (5,882 CHD deaths).²⁰ Farvid et al. found dietary LA intake is inversely associated with
581 CHD risk in a dose-response manner: when comparing the highest to the lowest category of
582 intake, LA was associated with a 15 percent lower risk of CHD events (pooled RR = 0.85; 95%
583 CI = 0.78 to 0.92; I²=35.5%) and a 21% lower risk of CHD deaths (pooled RR = 0.79; 95% CI =
584 0.71 to 0.89; I²=0.0%). A 5 percent of energy increment in LA intake replacing energy from SFA
585 intake was associated with a 9 percent lower risk of CHD events (RR = 0.91; 95% CI = 0.86 to
586 0.96) and a 13 percent lower risk of CHD deaths (RR = 0.87; 95% CI = 0.82 to 0.94). In the
587 meta-analysis conducted by Chowdhury et al., there was no significant association between LA
588 intake and CHD risk, but the analysis was based on a limited number of prospective cohort
589 studies.

590
591 In Jakobsen et al.'s 2009 pooled analysis of 11 cohorts (344,696 persons with 5,249 coronary
592 events and 2,155 coronary deaths), a 5 percent lower energy intake from SFAs and a
593 concomitant higher energy intake from PUFAs reduced risk of coronary events by 13 percent
594 (hazard ratio [HR] = 0.87; 95% CI = 0.77 to 0.97) and coronary deaths by 16 percent (hazard
595 ratio = 0.74; 95% CI = 0.61 to 0.89).²² By contrast, a 5 percent lower energy intake from SFAs
596 and a concomitant higher energy intake from carbohydrates, there was a modest significant direct
597 association between carbohydrates and coronary events (hazard ratio = 1.07; 95% CI = 1.01 to
598 1.14) and no association with coronary deaths (hazard ratio = 0.96; 95% CI = 0.82 to 1.13).
599 Notably, the estimated HRs for carbohydrate intake in this study could reflect high glycemic
600 carbohydrate intake rather than total carbohydrate, as fiber was controlled for in the analyses.
601 MUFA intake was not associated with CHD incidence or death.

602
603 Taken together, strong and consistent evidence from RCTs and statistical modeling in
604 prospective cohort studies shows that replacing SFA with PUFA reduces the risk of CVD events
605 and coronary mortality. For every 1 percent of energy intake from SFA replaced with PUFA,
606 incidence of CHD is reduced by 2 to 3 percent. The evidence is not as clear for replacement by
607 MUFA or replacement with carbohydrate, and likely depends on the type and source.

608 609 ***Methodological Issues***

610 When individuals in natural settings reduce calories from SFA, they typically replaced them with
611 other macronutrients, and the type and source of the macronutrients substituting SFA determine
612 effects on CVD. For this reason, studies specifying the macronutrient type replacing SFA are
613 more informative than those examining only total SFA intake, and the strongest and most

614 consistent evidence for CVD reduction is with replacement of SFA with PUFA in both RCTs
615 and observational studies.

616
617 The differing effects of the type and source of macronutrient substituted may be one reason for
618 the limited evidence regarding whether replacing SFA with MUFA confers CVD benefits and
619 the lack of benefit from carbohydrate substitution. The main sources of MUFA in a typical
620 American diet are animal fats, which could confound potential benefits of SFA-replacement with
621 plant-source MUFA, such as nuts and olive oil, which have demonstrated benefits on CVD risk.
622 To date, evidence testing replacement of SFA by MUFA from different sources is insufficient to
623 reach a firm conclusion. Similarly, most analyses did not distinguish between substitution of
624 saturated fat by different types of carbohydrates (e.g., refined carbohydrate vs. whole grains).

625
626 Of the RCTs included in this evidence summary, the intervention methods used varied from
627 long-term dietary counseling with good generalizability but variable compliance, to providing a
628 whole diet for weeks (e.g., controlled feeding studies) with maximal compliance but limited
629 generalizability. Though the content of the recommended or provided diet is known with greater
630 precision in the RCTs than in observational studies, adherence to the diet is likely variable and
631 could result in lack of compliance and high rates of dropout in long-term trials. Additionally,
632 bias may arise from the lack of blinding in non-supplement dietary intervention trials.

633
634 In prospective observational studies, misclassification of dietary fatty acid intake could bias
635 associations towards the null. In addition, residual confounding by other dietary and lifestyle
636 factors cannot be ruled out through statistical adjustment. Despite these methodological issues,
637 there is high consistency of the evidence from prospective cohort studies and RCTs in supporting
638 the benefits of replacing saturated fat with unsaturated fats especially PUFA in reducing CVD
639 risk.

640
641 *For additional details on this body of evidence, visit:* References 1, 2, 19-25 and *Appendix E-*
642 *2.43*

643

644

645 **ADDED SUGARS AND LOW-CALORIE SWEETENERS**

646 **INTRODUCTION**

647 Added sugars are sugars that are either added during the processing of foods, or are packaged as
648 such, and include sugars (free, mono- and disaccharides), syrups, naturally occurring sugars that
649 are isolated from a whole food and concentrated so that sugar is the primary component (e.g.,
650 fruit juice concentrates), and other caloric sweeteners.²⁸ Added sugars have been discussed in

651 previous iterations of the *Dietary Guidelines*, including a key recommendation in the 2010
652 *Dietary Guidelines* to “Reduce the intake of calories from solid fats and added sugars.” The 2010
653 *Dietary Guidelines* also included guidance stating that, for most people, no more than about 5 to
654 15 percent of calories from solid fats and added sugars (combined) can be reasonably
655 accommodated in a healthy eating pattern. However, as discussed in ***Part D. Chapter 1: Food
656 and Nutrient Intakes, and Health: Current Status and Trends***, the current intake of added
657 sugars still remains high at 268 calories, or 13.4 percent of total calories per day among the total
658 population ages 1 year and older.

659
660 Similar to the healthy eating patterns modeled for the 2010 DGAC, in the three healthy eating
661 patterns modeled for the 2015 DGAC (Healthy U.S.-style Pattern, Healthy Mediterranean-style
662 Pattern, and Healthy Vegetarian Pattern), a limited number of calories are available to be
663 consumed as added sugars (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health:
664 Current Status and Trends***). As shown in Table D.6.1, the full range of these three patterns at all
665 calorie levels allow for 3 to 9 percent of calories from added sugars, after meeting food group
666 and nutrient recommendations. For the patterns appropriate for most people (1600 to 2400
667 calories), the range is 4 to 6 percent of calories from added sugars (or 4.5 to 9.4 teaspoons). The
668 total empty calorie allowance in these patterns is 8 to 19 percent of calories, and based on current
669 consumption patterns, 45 percent of empty calories are allocated to limits for added sugars, with
670 the remainder (55 percent) allocated to solid fats.

671
672

673 **Table D6.1. Added sugars available in the USDA Food Patterns (Healthy U.S.-Style,**
 674 **Healthy Mediterranean-Style, and Healthy Vegetarian Patterns) in calories, teaspoons, and**
 675 **percent of total calories per day***
 676

CALORIE LEVEL	1000	1200	1400	1600	1800	2000	2200	2400	2600	2800	3000	3200
Empty calorie limits available for added sugars (assuming 45% empty calories from added sugars and 55% from solid fat)												
Healthy U.S.-style	68	50	50	54	77	122	126	158	171	180	212	275
Healthy Med-style	63	50	50	81	72	117	126	135	149	158	194	257
Healthy Vegetarian	77	77	81	81	81	131	131	158	158	158	185	234
Average	69	59	60	72	77	123	128	150	159	165	197	255
Average (tsp)	4.3	3.7	3.8	4.5	4.8	7.7	8.0	9.4	9.9	10.3	12.3	15.9
Healthy U.S.-style	7%	4%	4%	3%	4%	6%	6%	7%	7%	6%	7%	9%
Healthy Med-style	6%	4%	4%	5%	4%	6%	6%	6%	6%	6%	6%	8%
Healthy Vegetarian	8%	6%	6%	5%	5%	7%	6%	7%	6%	6%	6%	7%
Average	7%	5%	4%	5%	4%	6%	6%	6%	6%	6%	7%	8%

677 * See *Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends* and Appendix E-3.7
 678 for a full discussion of the food pattern modeling.

679
 680 Although food pattern modeling evaluates the amount of added sugars that can be consumed
 681 while meeting food group and nutrient needs, the DGAC also reviewed scientific literature
 682 examining the relationship between the intake of added sugars and health to inform
 683 recommendations. The Committee focused on the health outcomes most commonly researched
 684 related to added sugars, specifically, body weight and risk of type 2 diabetes, CVD, and dental
 685 caries.

686
 687 As noted above, the Committee acknowledged that a potential unintended consequence of a
 688 recommendation on added sugars might be that consumers and manufacturers replace added
 689 sugars with low-calorie sweeteners. As a result, the Committee also examined evidence on low-
 690 calorie sweeteners to inform statements on this topic. The Committee approached this topic
 691 broadly, including sweeteners labeled as low-calorie sweeteners, non-caloric sweeteners, non-
 692 nutritive sweeteners, artificial sweeteners, and diet beverages. This work is complemented by a
 693 food safety evidence review on aspartame (see *Part D. Chapter 5: Food Sustainability and*
 694 *Safety*). As the evidence on added sugars was considered collectively, the added sugars
 695 conclusions are presented together below, and a similar approach was taken for low-calorie
 696 sweeteners.

697

698 **Question 6: What is the relationship between the intake of added sugars and**
 699 **cardiovascular disease, body weight/obesity, type 2 diabetes, and dental caries?**

700 **Source of evidence:** CVD: NEL systematic review; Body weight/obesity, type 2 diabetes,
 701 and dental caries: Existing reports

702

703 **Conclusions**

704 Strong and consistent evidence shows that intake of added sugars from food and/or sugar-
 705 sweetened beverages are associated with excess body weight in children and adults. The
 706 reduction of added sugars and sugar-sweetened beverages in the diet reduces body mass index
 707 (BMI) in both children and adults. Comparison groups with the highest versus the lowest intakes
 708 of added sugars in cohort studies were compatible with a recommendation to keep added sugars
 709 intake below 10 percent of total energy intake. **DGAC Grade: Strong**

710

711 Strong evidence shows that higher consumption of added sugars, especially sugar-sweetened
 712 beverages, increases the risk of type 2 diabetes among adults and this relationship is not fully
 713 explained by body weight. **DGAC Grade: Strong**

714

715 Moderate evidence from prospective cohort studies indicates that higher intake of added sugars,
 716 especially in the form of sugar-sweetened beverages, is consistently associated with increased
 717 risk of hypertension, stroke, and CHD in adults. Observational and intervention studies indicate a
 718 consistent relationship between higher added sugars intake and higher blood pressure and serum
 719 triglycerides. **DGAC Grade: Moderate**

720

721 The DGAC concurs with the World Health Organization's commissioned systematic review that
 722 moderate consistent evidence supports a relationship between the amount of free sugars
 723 intake and the development of dental caries among children and adults. Moderate evidence also
 724 indicates that caries are lower when free sugars intake is less than 10 percent of energy intake.

725 **DGAC Grade: Moderate**

726

727 **Review of the Evidence**

728 ***Added Sugars and Body Weight/Obesity***

729 These findings come from three recent reports, all using SRs and MA that examined the
 730 relationship between the intake of added sugars and measures of body weight.^{6, 29, 30} Te Morenga
 731 et al.⁶ considered "free sugars,"* while Malik²⁹ and Kaiser et al.³⁰ focused on sugar-sweetened

* Free sugar is defined by WHO as "all monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer, plus sugars naturally present in honey, syrups, and fruit juices." It is used to distinguish between the sugars that are naturally present in fully unrefined carbohydrates such as brown rice, whole wheat pasta, and fruit and those sugars (or carbohydrates) that have been, to some extent, refined
 Scientific Report of the 2015 Dietary Guidelines Advisory Committee

732 beverages. All reviews reported on body weight. The Te Morenga report also reported on body
733 fatness. In the Te Morenga et al. study, 30 trials and 38 cohort studies were included in the
734 analyses. In the Malik et al. study, 10 trials and 22 cohort studies were included in the analyses.
735 Kaiser et al. provided an updated meta-analysis to a previous publication (Mattes³¹) and included
736 a total of 18 trials. In total, 92 articles were considered in these reviews, of which 21 were
737 included in two or more reviews. Children and adults were included in the analyses as were
738 females and males. Diverse demographics (race/ethnicity and geographic location) also were
739 represented by the participants in the respective research studies. All three reviews were high-
740 quality, with ratings of 11 out of 11 using the AMSTAR tool, and they specifically addressed the
741 Committee's question of interest.

742
743 The reviews by Malik et al. and Te Morenga et al. were very consistent. The findings from both
744 reports provide strong evidence that among free-living people consuming ad libitum diets, the
745 intake of added sugars or sugar-sweetened beverages is associated with unfavorable weight
746 status in children and adults. Increased added sugars intake is associated with weight gain;
747 decreased added sugars intake is associated with decreased body weight. Although a dose
748 response cannot be determined at this time, the data analyzed by Te Morenga et al. support
749 limiting added sugars to no more than 10 percent of daily total energy intake based on lowest
750 versus highest intakes from prospective cohort studies. Te Morenga et al. state that, "despite
751 significant heterogeneity in one meta-analysis and potential bias in some trials, sensitivity
752 analyses showed that the trends were consistent and associations remained after these studies
753 were excluded." Despite these limitations the DGAC gave this evidence a grade of **Strong**, as
754 the limitations are those inherent to the primary research on which they are based, notably
755 inadequacy of dietary intake data and variations in the nature and quality of the dietary
756 interventions.

757
758 The Kaiser et al. review concluded that the currently available randomized evidence for the
759 effects of reducing sugar-sweetened beverage intake on obesity is equivocal. However, the
760 DGAC noted methodological issues with this review, particularly the inclusion of both efficacy
761 studies (in more controlled settings) and effectiveness studies (in real world). The outcomes
762 from the effectiveness trials vary substantially, depending how effective the interventions are. As
763 a result, the Committee viewed the reviews by Te Morenga et al. and Malik et al. to be stronger
764 than the Kaiser et al. review.

765 ***Added Sugars and Type 2 Diabetes***

767 Evidence for this question and conclusion came from five SRs and MA published between
768 January 2010 and August 2014.³³⁻³⁷ Four of the reviews focused on sugar-sweetened

(normally by humans but sometimes by animals, such as the free sugars present in honey). They are referred to as "sugars" since they cover multiple chemical forms, including sucrose, glucose, fructose, dextrose, and others.³²

769 beverages^{33-35, 37} and one review examined sugar intake.³⁶ Combined, a total of 17 articles were
770 considered in these reviews, of which nine were included in two or more reviews. Increased
771 consumption of sugar-sweetened beverages was consistently associated with increased risk of
772 type 2 diabetes. Pooled estimated relative risks ranged from 1.20 to 1.28, and included 1.20 (95%
773 CI = 1.12 to 1.29)/330 ml/day of sugar-sweetened soft drinks;³³ 1.26 (95% CI = 1.12 to 1.41) for
774 sugar-sweetened beverages,³⁵ and 1.28 (95% CI = 1.04 to 1.59) for sugar-sweetened fruit
775 juices.³⁷ Comparably, a hazard ratio of 1.29 (1.02, 1.63) was identified for sugar-sweetened
776 beverages.³⁴ These consistently positive associations between sugar-sweetened beverages and
777 type 2 diabetes were attenuated, but still existed, after adjustment for BMI, suggesting that body
778 weight only partly explains the deleterious effects of sugar-sweetened beverages on type 2
779 diabetes. Although the studies were highly heterogeneous, findings from the MA by Malik et al.
780 tentatively showed that consumption of more than one 12-ounce serving per day of sugar-
781 sweetened beverage increased the risk of developing type 2 diabetes by 26 percent, compared to
782 consuming less than one serving per month. Insufficient high-quality data are available to
783 determine a dose-response line or curve between sugar-sweetened beverage consumption and
784 type 2 diabetes risk.

785
786 The issue of generalizability, whether the participants included in this body of evidence are
787 representative of the general U.S. population, was not specifically addressed in the literature
788 reviewed, but the large sample sizes of the pooled data (several hundred thousand subjects from
789 different populations) are noteworthy.

790

791 ***Added Sugars and Cardiovascular Disease***

792 This NEL systematic review included 23 articles published since 2000 that examined the
793 relationship between added sugars and risk of CVD or CVD risk factors such as blood lipids and
794 blood pressure.³⁸⁻⁶⁰ This literature included 11 intervention studies and 12 prospective cohort
795 studies.

796

797 The majority of intervention and observational studies included in this SR provide some
798 evidence among adults in support of an association between higher intake of added sugars,
799 especially in the form of sugar-sweetened beverages, and higher risk of CVD or increased CVD
800 risk factors. More consistent associations were seen between added sugars and elevated serum
801 triglycerides, blood pressure, and increased risk of hypertension, stroke, or CHD. Evidence for
802 associations between added sugars and dyslipidemia (i.e., low HDL, high LDL, and high total
803 cholesterol) was not as consistent, especially among intervention studies.

804

805 The body of evidence examined in this SR had a number of limitations. For example, the
806 intervention studies had extensive heterogeneity in terms of the types and forms of sugars used
807 (i.e., fructose, glucose, sucrose, sugar-sweetened beverages, sweetened milk) and the type of
808 control and/or isocaloric condition used. In addition, most intervention studies had a short

809 duration of the intervention and a small sample size. Most of the observational studies assessed
810 dietary intake only at baseline, and did not take assessments during follow-up. Residual
811 confounding by other dietary and lifestyle factors in observational analyses could not be
812 completely ruled out.

813

814 ***Added Sugars and Dental Caries***

815 These findings were extracted from a World Health Organization (WHO)-commissioned SR by
816 Moynihan et al. published in 2014 examining the association between the amount of sugars
817 intake and dental caries.⁷ The search for SRs/MA published since completion of the WHO
818 review did not yield any additional reviews that met the DGAC's inclusion criteria.

819

820 Moynihan et al. examined total sugars, free sugars, added sugars, sucrose, and non-milk extrinsic
821 (NME) sugars. In the review, eligible studies reported the absolute amount of sugars. Dental
822 caries outcomes included caries prevalence, incidence and/or severity.

823

824 Several databases were searched from 1950 through 2011. From 5,990 papers identified, 55
825 studies (from 65 papers) were eligible, including 3 interventions, 8 cohort studies, 20 population
826 studies, and 24 cross-sectional studies. No RCTs were included. Data variability limited the
827 ability to conduct meta-analysis. Of the 55 studies included in the review, the majority were in
828 children and only four studies were conducted in adults. The terminology used for reporting
829 sugars varied, but most were described as pertaining to free sugars or added sugars.

830

831 The findings indicated consistent evidence of moderate quality supporting a relationship between
832 the amount of sugars consumed and dental caries development across age groups. Of the studies,
833 42 out of 50 studies in children and five out of five in adults reported at least one result for an
834 association between sugars intake with increased caries. Moderate evidence also showed that
835 caries incidence is lower when free sugars intake is less than 10 percent of energy intake. When a
836 less than 5 percent energy intake cutoff was used, a significant relationship between sugars and
837 caries was observed, but the evidence was judged to be of very low quality. Although meta-
838 analysis was limited, analysis of existing data indicated a large effect size (e.g., Standardized
839 Mean Difference for Decayed/Missing/Filled Teeth [DMFT] = 0.82 [CI = 0.67-0.97]) for the
840 relationship of sugars intake and risk of dental caries. A strength of the in-depth SR was the
841 consistency of data, despite methodological weaknesses in many studies, which included unclear
842 definitions of endpoints, questions about outcomes ascertainment, and lack of clarity about the
843 generalizability of individual study results given the study populations used.

844

845 ***For additional details on this body of evidence, visit:*** References 6, 7, 29, 30, 33-37, and 38-60
846 and ***Appendices E-2.44 (body weight), E-2.45 (type 2 diabetes), E-2.46 (dental caries), and***
847 ***<http://NEL.gov/topic.cfm?cat=3376> (CVD)***

848

849 **Question 7: What is the relationship between the intake of low-calorie sweeteners**
 850 **and body weight/obesity and type 2 diabetes?**

851 **Source of evidence:** Existing reports

852

853 **Conclusions**

854 Moderate and generally consistent evidence from short-term RCTs conducted in adults and
 855 children supports that replacing sugar-containing sweeteners with low-calorie sweeteners
 856 reduces calorie intake, body weight, and adiposity. **DGAC Grade: Moderate**

857

858 Long-term observational studies conducted in children and adults provide inconsistent evidence
 859 of an association between low-calorie sweeteners and body weight as compared to sugar-
 860 containing sweeteners. **DGAC Grade: Limited**

861

862 Long-term observational studies conducted in adults provide inconsistent evidence of an
 863 association between low-calorie sweeteners and risk of type 2 diabetes. **DGAC Grade: Limited**

864

865 **Review of the Evidence**

866 ***Low-Calorie Sweeteners and Body Weight/Obesity***

867 The evidence to support these conclusions comes from three SRs/MA published between January
 868 2010 and August 2014.⁶¹⁻⁶³ In total, 39 articles were considered in these reviews, of which six
 869 were included in two or more reviews. Experimentally, the protocols described in the 39 articles
 870 included RCTs and prospective cohort studies. Although results from both experimental designs
 871 were carefully assessed, the DCAC deemed evidence from RCTs to be scientifically stronger and
 872 used it as the foundation for conclusions pertaining to body weight.

873

874 Among prospective cohort studies, low-calorie sweetener intake was not associated with body
 875 weight or fat mass, but was significantly associated with slightly higher BMI (0.03; 95% CI =
 876 0.01 to 0.06).⁶² These findings should be viewed with caution, however, because of the high risk
 877 of reverse causality and the possibility that people with higher body weights would consume
 878 more low-calorie sweetener-containing foods and beverages as a weight-control strategy.

879

880 Evidence from short-term RCTs consistently indicated that low-calorie sweeteners (vs. sugar-
 881 containing foods and beverages) modestly reduce body weight in adults. When evidence from
 882 adults and children were combined, low-calorie sweeteners modestly reduced BMI, fat mass, and
 883 waist circumference. The primary research articles used by Miller and Perez for the MA
 884 contained findings from both adults (n=5 cohorts) and children (n=4 cohorts).⁶² The results of
 885 interventions lasting 3 to 78 weeks indicated that low-calorie sweeteners reduced body weight in
 886 adults (-0.72 kg; 95% CI = -1.15 to -0.30) and children (-1.06 kg; 95% CI = -1.17 to -0.56). Age-

887 specific results were not provided for BMI, fat mass, or waist circumference, but data from both
888 age groups were pooled to show the impact of low-calorie sweeteners vs. sugar-containing
889 foods/beverages on these outcomes.

890

891 In contrast, Brown et al. summarized that very limited evidence from three short-term (12 to 25
892 week) RCTs, which suggested that consumption of low-calorie sweeteners does not influence
893 body weight or BMI in predominantly pre-teenage and teenage youth (ages 10 to 21 years),
894 compared to sugar-sweetened beverage or placebo.⁶¹ The authors cautioned that insufficient data
895 exist to assess causality of low-calorie sweeteners on body weight. The evidence reported in this
896 2010 publication was obtained from very heterogeneous experimental designs and interventions.
897 One study tested the effects of encapsulated aspartame vs. placebo during weight loss; another
898 allowed subjects to exchange sugar-sweetened beverages with either low-calorie sweetener
899 beverages or water (precluding assessment of low-calorie sweetener beverages specifically); and
900 a third was described as a “pilot study.”

901

902 Collectively, evidence is mixed on the impact of low-calorie sweeteners vs. sugar-containing
903 foods/beverages on body weight in children. However, the DGAC deemed evidence presented by
904 Miller and Perez⁶² to be stronger than from Brown et al.⁶¹ because it culminated from a larger,
905 more recent research base and include both systematic review and meta-analysis assessment and
906 evaluation techniques.

907

908 ***Low-Calorie Sweeteners and Type 2 Diabetes***

909 Evidence to address the impact of low-calorie sweeteners (specifically artificially sweetened soft
910 drinks, ASSD) on risk of type 2 diabetes comes from two SRs/MA published between January
911 2010 and August 2014.^{33, 34} The data from one of the reviews also is represented in the second
912 review.

913

914 Greenwood et al. reported that higher consumption of ASSD predicts increased risk of type 2
915 diabetes.³³ The summary RR for ASSD on type 2 diabetes risk was 1.13 (95% CI = 1.02 to 1.25,
916 $p < 0.02$) per 330 ml/day, based on four analyses from three prospective observational studies.
917 Although the finding indicates a positive association between ASSD and type 2 diabetes risk, the
918 trend was not consistent and may indicate an alternative explanation, such as confounding by
919 lifestyle factors or reverse causality (e.g., individuals with higher BMI at baseline may use
920 ASSD as a means to control weight).

921

922 Romaguera et al. also reported that higher consumption of ASSD was associated with increased
923 risk of type 2 diabetes.³⁴ In adjusted models, one 336 g (12 oz) daily increment in ASSD
924 consumption was associated with a hazard ratio for type 2 diabetes of 1.52 (95% CI = 1.26 to
925 1.83). High consumers of ASSD showed almost twice the hazard ratio of developing type 2
926 diabetes compared with low consumers (adjusted HR = 1.93; 95% CI = 1.47 to 2.54; p for trend

927 <0.0001). However, the association was attenuated and became statistically not significant when
928 BMI was included in the model (HR = 1.13, 95% CI = 0.85 to 1.52; p for trend = 0.24). The
929 authors offered these interpretations of the findings: “In light of these findings, we have two
930 possible explanations of the association between artificially sweetened soft drinks and diabetes:
931 (1) the observed association is driven by reverse causality and residual confounding, given that
932 the underlying health of people consuming artificially sweetened soft drinks may be
933 compromised and their risk of type 2 diabetes increased; or (2) the association between
934 artificially sweetened soft drinks and type 2 diabetes is mediated through increased BMI.” The
935 authors argued that explanation 1 is more likely correct based on reverse causality, but new
936 research would be needed to clarify the issue.

937
938 Collectively, both studies report a positive association between ASSD and type 2 diabetes risk
939 that was confounded by baseline BMI. The experimental designs of the studies included in these
940 reviews analyzed associations, but precluded the assessment of cause and effect relationships,
941 and future experimental studies should examine the relationship between ASSD and biomarkers
942 of insulin resistance and other diabetes biomarkers.

943
944 *For additional details on this body of evidence, visit:* References 33, 34, and 61-63 and
945 *Appendices E-2.47* (body weight) and *E-2.48* (type 2 diabetes)

946 947 **Implications**

948 Obesity, type 2 diabetes, CVD, and dental caries are major public health concerns. Added sugars
949 intake negatively impacts all of these conditions, and strong evidence supports reducing added
950 sugars intake to reduce health risks. Added sugars are frequently used in food/beverage
951 processing and provide calories but no other nutrients. Since 39 percent of added sugars are from
952 sugar-sweetened beverages, efforts are needed to reduce these beverages (see Figure D1.36.
953 Food Sources of Added Sugars). Currently, the mean intake of added sugars in the U.S.
954 population is 13%, and from 15% to 17% in children 9 and older, adolescents, and young adults.

955
956 The DGAC recommends limiting added sugars to a maximum of 10% of total daily caloric
957 intake. This recommendation is supported by: 1) the food pattern modeling analysis conducted
958 by the 2015 DGAC and 2) the scientific evidence review on added sugars and chronic disease
959 risk conducted by the Committee. The food pattern analysis, based on the Healthy U.S.-Style
960 Pattern, the Healthy Vegetarian Pattern, and the Healthy Mediterranean-Style Pattern (see *Part*
961 *D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends* and
962 *Appendix E-3.7*), demonstrates that when added sugars in foods and beverages exceeds 3% to
963 9% of total calories, depending on calorie level, a healthful food pattern may be difficult to
964 achieve and nutrient density may be adversely affected (Table D6.1). The scientific evidence on
965 added sugars and chronic disease risk also supports this limit.

966

967 The recommendation to limit added sugars, especially sugar-sweetened beverages, is consistent
968 with recommendations from national and international organizations including the American
969 Academy of Pediatrics, World Health Organization, American Heart Association, Centers for
970 Disease Control and Prevention, and the American Diabetes Association (Table D6.2).

971

972 When low-calorie sweeteners are used to replace sugar, the resulting reduction in calories can
973 help to achieve short-term weight loss. However, there is insufficient evidence (due to a paucity
974 of data) to recommend the use of low-calorie sweeteners as a strategy for long-term weight loss
975 and weight maintenance. Since the long-term effects of low-calorie sweeteners are still uncertain,
976 those sweeteners should not be recommended for use as a primary replacement/substitute for
977 added sugars in foods and beverages.

978

979 Policies and programs at local, state, and national levels in both the private sector and public
980 sector are necessary to support efforts to lower added sugars in beverages and foods and to limit
981 availability of sugar-sweetened beverages and snacks. Suggested specific approaches for
982 reducing added sugars intake include:

983

984 • Water is the preferred beverage choice. Strategies are needed to encourage the US
985 population, especially children and adolescents, to drink water when they are thirsty. Water
986 provides a healthy, low-cost, zero-calorie beverage option. Free, readily accessible, safe
987 water should be available in public settings, as well as child care facilities, schools, worksites
988 and other community places and promoted in all settings where beverages are offered.

989 • The Nutrition Facts Panel (NFP) should include added sugars (in grams and teaspoons) and
990 include a percent daily value, to assist consumers in making informed dietary decisions by
991 identifying the amount of added sugars in foods and beverages.

992 • Consumers would benefit from a standardized, easily understood front-of-package (FOP)
993 label on all food and beverage products to give clear guidance about a food's healthfulness.
994 An example is the FOP label recommended by the IOM,¹⁸ which included calories, and 0 to 3
995 "nutritional" points for added sugars, saturated fat, and sodium. This would be integrated
996 with the NFP, allowing consumers to quickly and easily identify nutrients of concern for
997 over-consumption, in order to make healthier choices.

998 • Economic and pricing approaches, using incentives and disincentives should be explored to
999 promote the purchase of healthier foods and beverages. For example, higher sugar-sweetened
1000 beverage taxes may encourage consumers to reduce sugar-sweetened beverage consumption.
1001 Using the revenues from the higher sugar-sweetened beverage taxes for nutrition health
1002 promotion efforts or to subsidize fruits and vegetables could have public health benefits.

- 1003 • Efforts to reduce added sugars in foods and sugar-sweetened beverages in school meals and
1004 through the new smart snacks in schools should continue and also be expanded to other
1005 settings, including early child care (through the Child and Adult Care Food Program-
1006 CACFP), parks, recreation centers, sports leagues, after school programs, work sites and
1007 other community settings.
- 1008 • Policies that limit exposure and marketing of foods and beverages high in added sugars to
1009 young children, youth and adolescents are needed as dietary preferences are established early
1010 in life.
- 1011 • Young adults (ages 20-29 years) are among the greatest consumers of sugar-sweetened
1012 beverages and are directly targeted in sugar-sweetened beverage marketing campaigns.
1013 Health promotion efforts and policies are needed to reduce sugar-sweetened beverages in
1014 settings, such as postsecondary institutions and worksites.
- 1015 • Policy changes within the federal Supplemental Nutrition Assistance Program (SNAP),
1016 similar to policies in place for the WIC program, should be considered to encourage purchase
1017 of healthier options, including foods and beverages low in added sugars. Pilot studies using
1018 incentives and restrictions should be tested and evaluated.
- 1019 • Public education campaigns are needed to increase the public’s awareness of the health
1020 effects of added sugars and help consumers reduce added sugars intake and reduce intake of
1021 sugar-sweetened beverages through policy, food environment and education initiatives.
- 1022
- 1023
- 1024
- 1025
- 1026
- 1027
- 1028

1029 **Table D6.2. Recommendations or statements related to added sugars or sugar-sweetened**
 1030 **beverages from international and national organizations**

Organization	Recommendation/Statement Related to Added Sugars and/or Sugar-Sweetened Beverages
World Health Organization (WHO) ⁶⁴	<ul style="list-style-type: none"> • WHO recommends reduced intake of free sugars throughout the life-course (<i>strong recommendation</i>). • In both adults and children, WHO recommends that intake of free sugars not to exceed 10% of total energy (<i>strong recommendation</i>). • WHO suggests further reduction to below 5% of total energy (<i>conditional recommendation</i>).
American Heart Association (AHA) ⁶⁵	The AHA recommends reductions in added sugars with an upper limit of half of the discretionary calorie allowance that can be accommodated within the appropriate energy intake level needed for a person to achieve or maintain a healthy weight based on the USDA food intake patterns. Most American women should eat or drink no more than 100 calories per day from added sugars (about 6 teaspoons), and most American men should eat or drink no more than 150 calories per day from added sugars (about 9 teaspoons).
HealthyPeople 2020 ⁶⁶	Objective NWS-17.2: Reduce consumption of calories from added sugars (Target: 10.8%)
American Academy of Pediatrics (AAP) ⁶⁷⁻⁶⁹	<p>Limit consumption of sugar-sweetened beverages (consistent evidence)</p> <p>Pediatricians should work to eliminate sweetened drinks in schools</p> <p><i>Note: Due to limited studies in children, the American Academy of Pediatrics (AAP) has no official recommendations regarding the use of non-caloric sweeteners.</i></p>
American Diabetes Association (ADA) ^{70, 71}	<p><u>Prevention</u></p> <p>Research has shown that drinking sugary drinks is linked to type 2 diabetes, and the American Diabetes Association recommends that people limit their intake of sugar-sweetened beverages to help prevent diabetes.</p> <p><u>Diabetes Management</u></p> <p>People with diabetes should limit or avoid intake of sugar-sweetened beverages (from any caloric sweetener including high fructose corn syrup and sucrose) to reduce risk for weight gain and worsening of cardiometabolic risk profile. (Evidence rating B)</p>
NHLBI Expert Panel Guidelines for Cardiovascular Health and Risk Reduction in Childhood ⁷²	Reduced intake of sugar-sweetened beverages is associated with decreased obesity measures (Grade B).

1031
 1032
 1033

1034 **CHAPTER SUMMARY**

1035 The DGAC encourages the consumption of healthy dietary patterns that are low in saturated fat,
 1036 added sugars, and sodium. The conclusions in this chapter complement the findings from *Part D.*
 1037 *Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends* and *Part D.*
 1038 *Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes*. The goals for the
 1039 general population are: less than 2,300 mg dietary sodium per day (or age-appropriate Dietary
 1040 Reference Intake amount), less than 10 percent of total calories from saturated fat per day, and a
 1041 maximum of 10 percent of total calories from added sugars per day.

1042
 1043 Sodium, saturated fat, and added sugars are not intended to be reduced in isolation, but as a part
 1044 of a healthy dietary pattern. Rather than focusing purely on reduction, emphasis should be placed
 1045 on replacement and shifts in food intake and eating patterns. Sources of saturated fat should be
 1046 replaced with unsaturated fat, particularly polyunsaturated fatty acids. Similarly, added sugars
 1047 should be reduced in the diet and not replaced with low-calorie sweeteners, but rather with
 1048 healthy options, such as water in place of sugar-sweetened beverages. For sodium, emphasis
 1049 should be placed on expanding industry efforts to reduce the sodium content of foods and
 1050 helping consumers understand how to flavor unsalted foods with spices and herbs.

1051
 1052 Achieving reductions in sodium, saturated fat, and added sugars, can all be accomplished and are
 1053 more attainable by eating a healthy dietary pattern. For all three of these components of the diet,
 1054 policies and programs at local, state, and national levels in both the private and public sector are
 1055 necessary to support reduction efforts. Similarly, the Committee supports efforts in labeling and
 1056 other campaigns to increase consumer awareness and understanding of sodium, saturated fats,
 1057 and added sugars in foods and beverages. The Committee encourages the food industry to
 1058 continue reformulating and making changes to certain foods to improve their nutrition profile.
 1059 Examples of such actions include lowering sodium and added sugars content, achieving better
 1060 saturated fat to polyunsaturated fat ratio, and reducing portion sizes in retail settings (restaurants,
 1061 food outlets, and public venues, such as professional sports stadiums and arenas). The
 1062 Committee also encourages the food industry to market these improved products to consumers.

1063
 1064

1065 **NEEDS FOR FUTURE RESEARCH**

- 1066 1. Design and conduct studies with sufficient power to define the impact of improving dietary
 1067 quality, including the lowering of dietary sodium intake, on hypertension and relevant
 1068 disease outcomes, including cardiovascular disease, stroke, peripheral vascular disease,
 1069 kidney disease, and others. The interactions with patterns of therapeutic medication use (e.g.,
 1070 diuretics, antihypertensives, and lipid-lowering) should be considered.

1071

1072 **Rationale:** The current literature is incomplete, limited in power and durations, and often
 1073 compromised by methodological challenges that must be addressed in well-designed studies
 1074 with relevant clinical outcomes.

1075

1076 2. Assess the accuracy of 24-hour urine collections for sodium assessment in populations with
 1077 different health conditions (e.g., diabetes, chronic kidney disease, heart failure,
 1078 cardiovascular disease) and interactions with different patterns of medication use (e.g.,
 1079 diuretics, antihypertensives).

1080

1081 **Rationale:** If there is systematic error in sodium assessment because individuals with various
 1082 co-morbidities who are taking medications systematically do not provide accurate urine
 1083 collections, paradoxical findings between sodium and health outcomes may be observed.

1084

1085 3. Examine the effect of behavioral interventions, with novel approaches (e.g., flavorful recipes,
 1086 cooking techniques) on adherence to dietary sodium recommendations.

1087

1088 **Rationale:** For decades, the population has exceeded dietary sodium intake
 1089 recommendations. A public health approach that results in reformulation of commercially
 1090 processed foods to lower sodium content should be the primary strategy for decreasing
 1091 sodium intake in the U.S. population. However, individual support for public health policies
 1092 will be needed to further document demand for changes in the sodium food environment. To
 1093 this end, interventions that modify individual knowledge, attitudes, and behaviors around
 1094 sodium intake should be evaluated.

1095

1096 4. Examine the effect of low sodium intake on taste preferences for sodium and healthy dietary
 1097 patterns.

1098

1099 **Rationale:** It has been argued that populations desire higher levels of sodium intake and will
 1100 inevitably revert to higher levels of sodium intakes after acute reductions in sodium intake. It
 1101 has also been argued that after six weeks of reduced sodium intake, taste preferences are
 1102 modified such that higher sodium is no longer desirable. Studies are needed to elucidate the
 1103 effects of lowering sodium intake on diet preferences.

1104

1105 5. Document the relationship between portion size and sodium intake.

1106

1107 **Rationale:** These data are needed to inform whether dietary recommendations for sodium
 1108 should be adjusted for caloric intake. It is known that the absolute amount of sodium intake is
 1109 highly correlated with caloric intake. As a result, the absolute recommended amount of
 1110 sodium is harder to achieve for a larger, high energy consuming person than for a smaller,

1111 low energy consuming person. The science to inform whether sodium density confers
 1112 different risk than absolute intake of sodium is limited because of methodologic limitations
 1113 in surveys where both calories and sodium intake can be calculated. Furthermore, the
 1114 existing correlation between sodium and calories may be an artifact of the current food
 1115 supply.

1116

1117 6. Determine the effects of replacement of saturated fat with different types of carbohydrates
 1118 (e.g., refined vs. whole grains) on cardiovascular disease risk.

1119

1120 **Rationale:** Most randomized controlled trials and prospective cohort studies compared
 1121 saturated fat with total carbohydrates. It is important to distinguish different types of
 1122 carbohydrates (e.g. refined vs. whole grains) in future studies.

1123

1124 7. Examine the effects that replacement of saturated fat with polyunsaturated fat vs.
 1125 monounsaturated fat has on cardiovascular disease risk.

1126

1127 **Rationale:** Most existing studies have examined the effects of substituting PUFA for
 1128 saturated fat on cardiovascular disease risk. Future studies should also examine the potential
 1129 benefits of substituting monounsaturated fat from plant sources such as olive oil and
 1130 nuts/seeds for saturated fat on cardiovascular disease risk.

1131

1132 8. Examine lipid and metabolic effects of specific oils modified to have different fatty acid
 1133 profiles (e.g. commodity soy oil [high linoleic acid] vs. high oleic soy oil).

1134

1135 **Rationale:** As more modified vegetable oils become commercially available, it is important
 1136 to assess their long-term health effects. In addition, future studies should examine lipid and
 1137 metabolic effects of plant oils that contain a mix of *n*-9, *n*-6, and *n*-3 fatty acids, as a
 1138 replacement for animal fat, on cardiovascular disease risk factors.

1139

1140 9. Examine the effects of saturated fat from different sources, including animal products (e.g.
 1141 butter, lard), plant (e.g., palm vs. coconut oils), and production systems (e.g. refined
 1142 deodorized bleached vs. virgin coconut oil) on blood lipids and cardiovascular disease risk.

1143

1144 **Rationale:** Different sources of saturated fat contain different fatty acid profiles and thus,
 1145 may result in different lipid and metabolic effects. In addition, virgin and refined coconut oils
 1146 have different effects in animal models, but human data are lacking.

1147

1148 10. Conduct gene-nutrient interaction studies by measuring genetic variations in relevant genes
 1149 that will enable evaluation of effects of specific diets for individualized nutrition
 1150 recommendations.

1151
 1152 **Rationale:** Individuals with different genetic background may respond to the same dietary
 1153 intervention differently in terms of blood lipids and other cardiovascular disease risk factors.
 1154 Future studies should explore the potential role of genetic factors in modulating the effects of
 1155 fat type modification on health outcomes.

1156
 1157 11. Identify sources and names of added sugars and low-calorie sweeteners used in the food
 1158 supply and quantify their consumption levels and trends in the U.S. diet.

1159
 1160 **Rationale:** It is unclear whether all food and nutrient databases capture all added sugars
 1161 because: 1) added sugars have varied and inconsistent nomenclature and may not be
 1162 recognized as added sugars in nutrient analyses; and 2) many foods with added sugars have
 1163 formulations considered proprietary by the manufacturers and for this reason actual added
 1164 sugars content is difficult to obtain. Accurate assessment of added sugars in the U.S. diet is
 1165 needed to quantify the population level exposure and subsequent health risks from added
 1166 sugars. The lack of information on the various added sugars in the food supply hinders efforts
 1167 to make policy about consumption.

1168
 1169 12. Conduct prospective research with strong experimental designs and multiple measurements
 1170 of the consumption of added sugars and low-calorie sweeteners on health outcomes, such as
 1171 body weight, adiposity, and clinical markers of type 2 diabetes and cardiovascular disease.

1172
 1173 **Rationale:** High heterogeneity exists among published research with regard to the types and
 1174 forms of added sugars and low-calorie sweeteners-containing foods/beverages used for
 1175 interventions, which precludes assessing the effects of specific added sugars and low-calorie
 1176 sweeteners on body weight, adiposity, and cardio-metabolic health in adults and children.
 1177 Many studies use single baseline measurements of diet to reflect usual patterns and quantities
 1178 of intake over time. New research should emphasize assessments within the context of usual
 1179 dietary intakes and patterns of food and beverage consumption in free-living populations,
 1180 along with specific added sugars and low-calorie sweeteners, especially those that are
 1181 currently understudied. Large prospective studies with repeated measurements of low-calorie
 1182 sweeteners are needed to monitor their long-term effects on cancer and other health
 1183 outcomes.

1184
 1185 13. Design studies that emphasize assessments of relationships between the intakes of added
 1186 sugars and low-calorie sweeteners and body weight, adiposity, and cardio-metabolic health in
 1187 diverse sub-populations who are at high risk of obesity and related morbidities.

1188
 1189 **Rationale:** Insufficient evidence exists to assess the impact of added sugars and low-calorie
 1190 sweeteners contained in foods and beverages on individuals from diverse populations who

1191 have high risk for adverse health outcomes. These include (but not limited to) different
 1192 race/ethnicity groups; low income groups, especially those with food insecurity; groups who
 1193 live in specific geographic locations with high prevalence of obesity (e.g. inner city, rural,
 1194 and Southern regions of the United States); and age and sex groups (women, children, and
 1195 elderly adults).

1196

1197 14. Assess and improve approaches and policies to reduce the amount of added sugars in the
 1198 food and beverage supply as well as in school and community settings.

1199

1200 **Rationale:** Results from this research would assist policy makers and the private sector in
 1201 establishing sustainable approaches and policies to limit the availability and consumption of
 1202 added sugars. These approaches and policies would also be important for multi-component
 1203 strategies to improve weight control and health among people living in the United States.

1204

1205 15. Conduct consumer research to identify and test elements of a standardized, easily understood
 1206 front-of-package label.

1207

1208 **Rationale:** Research is needed to provide an evidence base to support the need and identify
 1209 critical elements of a front of package label. This is particularly important to support the
 1210 Food and Drug Administration in implementing a front-of-package labeling system.

1211

1212

1213 REFERENCES

- 1214 1. National Heart Lung and Blood Institute. Lifestyle Interventions to Reduce
 1215 Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group.
 1216 Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of
 1217 Health; 2013. Available from: [http://www.nhlbi.nih.gov/health-pro/guidelines/in-](http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm)
 1218 [develop/cardiovascular-risk-reduction/lifestyle/index.htm](http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm).
- 1219 2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013
 1220 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of
 1221 the American College of Cardiology/American Heart Association Task Force on Practice
 1222 Guidelines. *J Am Coll Cardiol*. 2014;63(25 Pt B):2960-84. PMID: 24239922.
 1223 <http://www.ncbi.nlm.nih.gov/pubmed/24239922>.
- 1224 3. Mente A, O'Donnell MJ, Rangarajan S, McQueen MJ, Poirier P, Wielgosz A, et al.
 1225 Association of urinary sodium and potassium excretion with blood pressure. *N Engl J*
 1226 *Med*. 2014;371(7):601-11. PMID: 25119606.
 1227 <http://www.ncbi.nlm.nih.gov/pubmed/25119606>.
- 1228 4. Institute of Medicine. Sodium Intake in Populations: Assessment of Evidence.
 1229 Washington, D.C.: The National Academies Press; 2013. Available from:
 1230 http://www.nap.edu/catalog.php?record_id=18311.

- 1231 5. Institute of Medicine. Strategies to Reduce Sodium Intake in the United States.
1232 Washington D.C.: The National Academies Press; 2010.
- 1233 6. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review
1234 and meta-analyses of randomised controlled trials and cohort studies. *BMJ*.
1235 2013;346:e7492. PMID: 23321486. <http://www.ncbi.nlm.nih.gov/pubmed/23321486>.
- 1236 7. Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to
1237 inform WHO guidelines. *J Dent Res*. 2014;93(1):8-18. PMID: 24323509.
1238 <http://www.ncbi.nlm.nih.gov/pubmed/24323509>.
- 1239 8. U.S. Department of Agriculture. Agricultural Research Services. Nutrient Intakes from
1240 Food: Mean Amounts Consumed per Individual, by Gender and Age, *What We Eat in*
1241 *America*, NHANES 2009-2010. 2012. Available from: www.ars.usda.gov/ba/bhnrc/fsrg.
- 1242 9. Institute of Medicine. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride,
1243 and Sulfate. Washington, D.C.: The National Academies Press; 2005. Available from:
1244 http://books.nap.edu/openbook.php?record_id=10925.
- 1245 10. Graudal NA, Hubeck-Graudal T, Jürgens G. Effects of low-sodium diet vs. high-sodium
1246 diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride
1247 (Cochrane Review). *Am J Hypertens*. 2012;25(1):1-15. PMID: 22068710.
1248 <http://www.ncbi.nlm.nih.gov/pubmed/22068710>.
- 1249 11. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory
1250 Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of
1251 Agriculture and the Secretary of Health and Human Services: U.S. Department of
1252 Agriculture, Agricultural Research Service, Washington D.C.; 2010. Available from:
1253 <http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm>.
- 1254 12. Cotter J, Cotter MJ, Oliveira P, Cunha P, Polonia J. Salt intake in children 10-12 years
1255 old and its modification by active working practices in a school garden. *J Hypertens*.
1256 2013;31(10):1966-71. PMID: 24107730.
1257 <http://www.ncbi.nlm.nih.gov/pubmed/24107730>.
- 1258 13. Shi L, Krupp D, Remer T. Salt, fruit and vegetable consumption and blood pressure
1259 development: a longitudinal investigation in healthy children. *Br J Nutr*.
1260 2014;111(4):662-71. PMID: 24326147. <http://www.ncbi.nlm.nih.gov/pubmed/24326147>.
- 1261 14. Cook NR, Appel LJ, Whelton PK. Lower levels of sodium intake and reduced
1262 cardiovascular risk. *Circulation*. 2014;129(9):981-9. PMID: 24415713.
1263 <http://www.ncbi.nlm.nih.gov/pubmed/24415713>.
- 1264 15. Joosten MM, Gansevoort RT, Mukamal KJ, Lambers Heerspink HJ, Geleijnse JM,
1265 Feskens EJ, et al. Sodium excretion and risk of developing coronary heart disease.
1266 *Circulation*. 2014;129(10):1121-8. PMID: 24425751.
1267 <http://www.ncbi.nlm.nih.gov/pubmed/24425751>.
- 1268 16. O'Donnell M, Mente A, Rangarajan S, McQueen MJ, Wang X, Liu L, et al. Urinary
1269 sodium and potassium excretion, mortality, and cardiovascular events. *N Engl J Med*.
1270 2014;371(7):612-23. PMID: 25119607. <http://www.ncbi.nlm.nih.gov/pubmed/25119607>.
- 1271 17. Pfister R, Michels G, Sharp SJ, Luben R, Wareham NJ, Khaw KT. Estimated urinary
1272 sodium excretion and risk of heart failure in men and women in the EPIC-Norfolk study.
1273 *Eur J Heart Fail*. 2014. PMID: 24464931.
1274 <http://www.ncbi.nlm.nih.gov/pubmed/24464931>.
- 1275 18. Institute of Medicine. Front-of-Package Nutrition Rating Systems and Symbols:
1276 Promoting Healthier Choices. Washington D.C.: The National Academies Press; 2012.

- 1277 19. Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, et al.
 1278 Association of dietary, circulating, and supplement fatty acids with coronary risk: a
 1279 systematic review and meta-analysis. *Ann Intern Med.* 2014;160(6):398-406. PMID:
 1280 24723079. <http://www.ncbi.nlm.nih.gov/pubmed/24723079>.
- 1281 20. Farvid MS, Ding M, Pan A, Sun Q, Chiuve SE, Steffen LM, et al. Dietary Linoleic Acid
 1282 and Risk of Coronary Heart Disease: A Systematic Review and Meta-Analysis of
 1283 Prospective Cohort Studies. *Circulation.* 2014. PMID: 25161045.
 1284 <http://www.ncbi.nlm.nih.gov/pubmed/25161045>.
- 1285 21. Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore HJ, et al. Reduced
 1286 or modified dietary fat for preventing cardiovascular disease. *Cochrane Database Syst*
 1287 *Rev.* 2012;5:CD002137. PMID: 22592684.
 1288 <http://www.ncbi.nlm.nih.gov/pubmed/22592684>.
- 1289 22. Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Bälter K, Fraser GE, et al. Major
 1290 types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort
 1291 studies. *Am J Clin Nutr.* 2009;89(5):1425-32. PMID: 19211817.
 1292 <http://www.ncbi.nlm.nih.gov/pubmed/19211817>.
- 1293 23. Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing
 1294 polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of
 1295 randomized controlled trials. *PLoS Med.* 2010;7(3):e1000252. PMID: 20351774.
 1296 <http://www.ncbi.nlm.nih.gov/pubmed/20351774>.
- 1297 24. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies
 1298 evaluating the association of saturated fat with cardiovascular disease. *Am J Clin Nutr.*
 1299 2010;91(3):535-46. PMID: 20071648. <http://www.ncbi.nlm.nih.gov/pubmed/20071648>.
- 1300 25. Skeaff CM, Miller J. Dietary fat and coronary heart disease: summary of evidence from
 1301 prospective cohort and randomised controlled trials. *Ann Nutr Metab.* 2009;55(1-3):173-
 1302 201. PMID: 19752542. <http://www.ncbi.nlm.nih.gov/pubmed/19752542>.
- 1303 26. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins. A
 1304 meta-analysis of 27 trials. *Arterioscler Thromb.* 1992;12(8):911-9. PMID: 1386252.
 1305 <http://www.ncbi.nlm.nih.gov/pubmed/1386252>.
- 1306 27. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and
 1307 carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and
 1308 apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr.*
 1309 2003;77(5):1146-55. PMID: 12716665. <http://www.ncbi.nlm.nih.gov/pubmed/12716665>.
- 1310 28. Department of Health and Human Services. Food and Drug Administration. Proposed
 1311 definition from FDA in the Proposed Rule to the revision of the Nutrition and
 1312 Supplement Facts Labels (Docket No. FDA-2012-N-1210). March 2014. Available from:
 1313 [https://www.federalregister.gov/articles/2014/03/03/2014-04387/food-labeling-revision-](https://www.federalregister.gov/articles/2014/03/03/2014-04387/food-labeling-revision-of-the-nutrition-and-supplement-facts-labels#p-325)
 1314 [of-the-nutrition-and-supplement-facts-labels#p-325](https://www.federalregister.gov/articles/2014/03/03/2014-04387/food-labeling-revision-of-the-nutrition-and-supplement-facts-labels#p-325).
- 1315 29. Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in
 1316 children and adults: a systematic review and meta-analysis. *Am J Clin Nutr.*
 1317 2013;98(4):1084-102. PMID: 23966427.
 1318 <http://www.ncbi.nlm.nih.gov/pubmed/23966427>.
- 1319 30. Kaiser KA, Shikany JM, Keating KD, Allison DB. Will reducing sugar-sweetened
 1320 beverage consumption reduce obesity? Evidence supporting conjecture is strong, but
 1321 evidence when testing effect is weak. *Obes Rev.* 2013;14(8):620-33. PMID: 23742715.
 1322 <http://www.ncbi.nlm.nih.gov/pubmed/23742715>.

- 1323 31. Mattes RD, Shikany JM, Kaiser KA, Allison DB. Nutritively sweetened beverage
1324 consumption and body weight: a systematic review and meta-analysis of randomized
1325 experiments. *Obes Rev.* 2011;12(5):346-65. PMID: 20524996.
1326 <http://www.ncbi.nlm.nih.gov/pubmed/20524996>.
- 1327 32. The science behind the sweetness in our diets. *Bull World Health Organ.*
1328 2014;92(11):780-1. PMID: 25378738. <http://www.ncbi.nlm.nih.gov/pubmed/25378738>.
- 1329 33. Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, et
1330 al. Association between sugar-sweetened and artificially sweetened soft drinks and type 2
1331 diabetes: systematic review and dose-response meta-analysis of prospective studies. *Br J*
1332 *Nutr.* 2014;112(5):725-34. PMID: 24932880.
1333 <http://www.ncbi.nlm.nih.gov/pubmed/24932880>.
- 1334 34. Romaguera D, Norat T, Wark PA, Vergnaud AC, Schulze MB, van Woudenberg GJ, et
1335 al. Consumption of sweet beverages and type 2 diabetes incidence in European adults:
1336 results from EPIC-InterAct. *Diabetologia.* 2013;56(7):1520-30. PMID: 23620057.
1337 <http://www.ncbi.nlm.nih.gov/pubmed/23620057>.
- 1338 35. Malik VS, Popkin BM, Bray GA, Després JP, Willett WC, Hu FB. Sugar-sweetened
1339 beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes*
1340 *Care.* 2010;33(11):2477-83. PMID: 20693348.
1341 <http://www.ncbi.nlm.nih.gov/pubmed/20693348>.
- 1342 36. Sonestedt E, Overby NC, Laaksonen DE, Birgisdottir BE. Does high sugar consumption
1343 exacerbate cardiometabolic risk factors and increase the risk of type 2 diabetes and
1344 cardiovascular disease? *Food Nutr Res.* 2012;56. PMID: 22855643.
1345 <http://www.ncbi.nlm.nih.gov/pubmed/22855643>.
- 1346 37. Xi B, Li S, Liu Z, Tian H, Yin X, Huai P, et al. Intake of fruit juice and incidence of type
1347 2 diabetes: a systematic review and meta-analysis. *PLoS One.* 2014;9(3):e93471. PMID:
1348 24682091. <http://www.ncbi.nlm.nih.gov/pubmed/24682091>.
- 1349 38. Aeberli I, Gerber PA, Hochuli M, Kohler S, Haile SR, Gouni-Berthold I, et al. Low to
1350 moderate sugar-sweetened beverage consumption impairs glucose and lipid metabolism
1351 and promotes inflammation in healthy young men: a randomized controlled trial. *Am J*
1352 *Clin Nutr.* 2011;94(2):479-85. PMID: 21677052.
1353 <http://www.ncbi.nlm.nih.gov/pubmed/21677052>.
- 1354 39. Aeberli I, Hochuli M, Gerber PA, Sze L, Murer SB, Tappy L, et al. Moderate amounts of
1355 fructose consumption impair insulin sensitivity in healthy young men: a randomized
1356 controlled trial. *Diabetes Care.* 2013;36(1):150-6. PMID: 22933433.
1357 <http://www.ncbi.nlm.nih.gov/pubmed/22933433>.
- 1358 40. Barrio-Lopez MT, Martinez-Gonzalez MA, Fernandez-Montero A, Beunza JJ, Zazpe I,
1359 Bes-Rastrollo M. Prospective study of changes in sugar-sweetened beverage consumption
1360 and the incidence of the metabolic syndrome and its components: the SUN cohort. *Br J*
1361 *Nutr.* 2013;110(9):1722-31. PMID: 23534417.
1362 <http://www.ncbi.nlm.nih.gov/pubmed/23534417>.
- 1363 41. Bernstein AM, de Koning L, Flint AJ, Rexrode KM, Willett WC. Soda consumption and
1364 the risk of stroke in men and women. *Am J Clin Nutr.* 2012;95(5):1190-9. PMID:
1365 22492378. <http://www.ncbi.nlm.nih.gov/pubmed/22492378>.
- 1366 42. Black RN, Spence M, McMahan RO, Cuskelly GJ, Ennis CN, McCance DR, et al. Effect
1367 of eucaloric high- and low-sucrose diets with identical macronutrient profile on insulin

- 1368 resistance and vascular risk: a randomized controlled trial. *Diabetes*. 2006;55(12):3566-
 1369 72. PMID: 17130505. <http://www.ncbi.nlm.nih.gov/pubmed/17130505>.
- 1370 43. Chen L, Caballero B, Mitchell DC, Loria C, Lin PH, Champagne CM, et al. Reducing
 1371 consumption of sugar-sweetened beverages is associated with reduced blood pressure: a
 1372 prospective study among United States adults. *Circulation*. 2010;121(22):2398-406.
 1373 PMID: 20497980. <http://www.ncbi.nlm.nih.gov/pubmed/20497980>.
- 1374 44. Cohen L, Curhan G, Forman J. Association of sweetened beverage intake with incident
 1375 hypertension. *J Gen Intern Med*. 2012;27(9):1127-34. PMID: 22539069.
 1376 <http://www.ncbi.nlm.nih.gov/pubmed/22539069>.
- 1377 45. de Koning L, Malik VS, Kellogg MD, Rimm EB, Willett WC, Hu FB. Sweetened
 1378 beverage consumption, incident coronary heart disease, and biomarkers of risk in men.
 1379 *Circulation*. 2012;125(14):1735-41, S1. PMID: 22412070.
 1380 <http://www.ncbi.nlm.nih.gov/pubmed/22412070>.
- 1381 46. Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, et al. Soft drink
 1382 consumption and risk of developing cardiometabolic risk factors and the metabolic
 1383 syndrome in middle-aged adults in the community. *Circulation*. 2007;116(5):480-8.
 1384 PMID: 17646581. <http://www.ncbi.nlm.nih.gov/pubmed/17646581>.
- 1385 47. Duffey KJ, Gordon-Larsen P, Steffen LM, Jacobs DR, Popkin BM. Drinking caloric
 1386 beverages increases the risk of adverse cardiometabolic outcomes in the Coronary Artery
 1387 Risk Development in Young Adults (CARDIA) Study. *Am J Clin Nutr*. 2010;92(4):954-
 1388 9. PMID: 20702604. <http://www.ncbi.nlm.nih.gov/pubmed/20702604>.
- 1389 48. Eshak ES, Iso H, Kokubo Y, Saito I, Yamagishi K, Inoue M, et al. Soft drink intake in
 1390 relation to incident ischemic heart disease, stroke, and stroke subtypes in Japanese men
 1391 and women: the Japan Public Health Centre-based study cohort I. *Am J Clin Nutr*.
 1392 2012;96(6):1390-7. PMID: 23076619. <http://www.ncbi.nlm.nih.gov/pubmed/23076619>.
- 1393 49. Faeh D, Minehira K, Schwarz JM, Periasamy R, Periasami R, Park S, et al. Effect of
 1394 fructose overfeeding and fish oil administration on hepatic de novo lipogenesis and
 1395 insulin sensitivity in healthy men. *Diabetes*. 2005;54(7):1907-13. PMID: 15983189.
 1396 <http://www.ncbi.nlm.nih.gov/pubmed/15983189>.
- 1397 50. Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB. Sweetened beverage
 1398 consumption and risk of coronary heart disease in women. *Am J Clin Nutr*.
 1399 2009;89(4):1037-42. PMID: 19211821. <http://www.ncbi.nlm.nih.gov/pubmed/19211821>.
- 1400 51. Larsson SC, Akesson A, Wolk A. Sweetened beverage consumption is associated with
 1401 increased risk of stroke in women and men. *J Nutr*. 2014;144(6):856-60. PMID:
 1402 24717367. <http://www.ncbi.nlm.nih.gov/pubmed/24717367>.
- 1403 52. Lê KA, Ith M, Kreis R, Faeh D, Bortolotti M, Tran C, et al. Fructose overconsumption
 1404 causes dyslipidemia and ectopic lipid deposition in healthy subjects with and without a
 1405 family history of type 2 diabetes. *Am J Clin Nutr*. 2009;89(6):1760-5. PMID: 19403641.
 1406 <http://www.ncbi.nlm.nih.gov/pubmed/19403641>.
- 1407 53. Lewis AS, McCourt HJ, Ennis CN, Bell PM, Courtney CH, McKinley MC, et al.
 1408 Comparison of 5% versus 15% sucrose intakes as part of a eucaloric diet in overweight
 1409 and obese subjects: effects on insulin sensitivity, glucose metabolism, vascular
 1410 compliance, body composition and lipid profile. A randomised controlled trial.
 1411 *Metabolism*. 2013;62(5):694-702. PMID: 23363580.
 1412 <http://www.ncbi.nlm.nih.gov/pubmed/23363580>.

- 1413 54. Lowndes J, Sinnett S, Pardo S, Nguyen VT, Melanson KJ, Yu Z, et al. The effect of
 1414 normally consumed amounts of sucrose or high fructose corn syrup on lipid profiles,
 1415 body composition and related parameters in overweight/obese subjects. *Nutrients*.
 1416 2014;6(3):1128-44. PMID: 24642950. <http://www.ncbi.nlm.nih.gov/pubmed/24642950>.
- 1417 55. Lowndes J, Sinnett S, Yu Z, Rippe J. The effects of fructose-containing sugars on weight,
 1418 body composition and cardiometabolic risk factors when consumed at up to the 90th
 1419 percentile population consumption level for fructose. *Nutrients*. 2014;6(8):3153-68.
 1420 PMID: 25111121. <http://www.ncbi.nlm.nih.gov/pubmed/25111121>.
- 1421 56. Maersk M, Belza A, Stødkilde-Jørgensen H, Ringgaard S, Chabanova E, Thomsen H, et
 1422 al. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat
 1423 depot: a 6-mo randomized intervention study. *Am J Clin Nutr*. 2012;95(2):283-9. PMID:
 1424 22205311. <http://www.ncbi.nlm.nih.gov/pubmed/22205311>.
- 1425 57. Raben A, Vasilaras TH, Møller AC, Astrup A. Sucrose compared with artificial
 1426 sweeteners: different effects on ad libitum food intake and body weight after 10 wk of
 1427 supplementation in overweight subjects. *Am J Clin Nutr*. 2002;76(4):721-9. PMID:
 1428 12324283. <http://www.ncbi.nlm.nih.gov/pubmed/12324283>.
- 1429 58. Tasevska N, Park Y, Jiao L, Hollenbeck A, Subar AF, Potischman N. Sugars and risk of
 1430 mortality in the NIH-AARP Diet and Health Study. *Am J Clin Nutr*. 2014;99(5):1077-88.
 1431 PMID: 24552754. <http://www.ncbi.nlm.nih.gov/pubmed/24552754>.
- 1432 59. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and
 1433 cardiovascular diseases mortality among US adults. *JAMA Intern Med*. 2014;174(4):516-
 1434 24. PMID: 24493081. <http://www.ncbi.nlm.nih.gov/pubmed/24493081>.
- 1435 60. Yu Z, Lowndes J, Rippe J. High-fructose corn syrup and sucrose have equivalent effects
 1436 on energy-regulating hormones at normal human consumption levels. *Nutr Res*.
 1437 2013;33(12):1043-52. PMID: 24267044.
 1438 <http://www.ncbi.nlm.nih.gov/pubmed/24267044>.
- 1439 61. Brown RJ, de Banate MA, Rother KI. Artificial sweeteners: a systematic review of
 1440 metabolic effects in youth. *Int J Pediatr Obes*. 2010;5(4):305-12. PMID: 20078374.
 1441 <http://www.ncbi.nlm.nih.gov/pubmed/20078374>.
- 1442 62. Miller PE, Perez V. Low-calorie sweeteners and body weight and composition: a meta-
 1443 analysis of randomized controlled trials and prospective cohort studies. *Am J Clin Nutr*.
 1444 2014;100(3):765-77. PMID: 24944060. <http://www.ncbi.nlm.nih.gov/pubmed/24944060>.
- 1445 63. Wiebe N, Padwal R, Field C, Marks S, Jacobs R, Tonelli M. A systematic review on the
 1446 effect of sweeteners on glycemic response and clinically relevant outcomes. *BMC Med*.
 1447 2011;9:123. PMID: 22093544. <http://www.ncbi.nlm.nih.gov/pubmed/22093544>.
- 1448 64. World Health Organization. Draft guidelines on free sugars released for public
 1449 consultation, 5 March 2014.
 1450 http://www.who.int/nutrition/sugars_public_consultation/en/.
- 1451 65. Van Horn L, Johnson RK, Flickinger BD, Vafiadis DK, Yin-Piazza S, Group ASCP.
 1452 Translation and implementation of added sugars consumption recommendations: a
 1453 conference report from the American Heart Association Added Sugars Conference 2010.
 1454 *Circulation*. 2010;122(23):2470-90. PMID: 21060079.
 1455 <http://www.ncbi.nlm.nih.gov/pubmed/21060079>.
- 1456 66. U.S. Department of Health and Human Services. Office of Disease Prevention and Health
 1457 Promotion. Healthy People 2020. Objective NWS-17.2. Available from:

- 1458 [https://www.healthypeople.gov/2020/topics-objectives/topic/nutrition-and-weight-](https://www.healthypeople.gov/2020/topics-objectives/topic/nutrition-and-weight-status/objectives)
 1459 [status/objectives](https://www.healthypeople.gov/2020/topics-objectives/topic/nutrition-and-weight-status/objectives).
- 1460 67. Barlow SE, Committee E. Expert committee recommendations regarding the prevention,
 1461 assessment, and treatment of child and adolescent overweight and obesity: summary
 1462 report. *Pediatrics*. 2007;120 Suppl 4:S164-92. PMID: 18055651.
 1463 <http://www.ncbi.nlm.nih.gov/pubmed/18055651>.
- 1464 68. American Academy of Pediatrics Committee on School Health. Soft drinks in schools.
 1465 *Pediatrics*. 2004;113(1 Pt 1):152-4. PMID: 14702469.
 1466 <http://www.ncbi.nlm.nih.gov/pubmed/14702469>.
- 1467 69. American Academy of Pediatrics. Sweeteners and Sugar Substitutes. Available from:
 1468 [http://www.healthychildren.org/English/healthy-living/nutrition/Pages/Sweeteners-and-](http://www.healthychildren.org/English/healthy-living/nutrition/Pages/Sweeteners-and-Sugar-Substitutes.aspx)
 1469 [Sugar-Substitutes.aspx](http://www.healthychildren.org/English/healthy-living/nutrition/Pages/Sweeteners-and-Sugar-Substitutes.aspx).
- 1470 70. American Diabetes Association. Sugar and Desserts [November 2014]. Available from:
 1471 [http://www.diabetes.org/food-and-fitness/food/what-can-i-eat/understanding-](http://www.diabetes.org/food-and-fitness/food/what-can-i-eat/understanding-carbohydrates/sugar-and-desserts.html)
 1472 [carbohydrates/sugar-and-desserts.html](http://www.diabetes.org/food-and-fitness/food/what-can-i-eat/understanding-carbohydrates/sugar-and-desserts.html).
- 1473 71. Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, et al.
 1474 Nutrition therapy recommendations for the management of adults with diabetes. *Diabetes*
 1475 *Care*. 2013;36(11):3821-42. PMID: 24107659.
 1476 <http://www.ncbi.nlm.nih.gov/pubmed/24107659>.
- 1477 72. National Heart Lung and Blood Institute. Integrated Guidelines for Cardiovascular Health
 1478 and Risk Reduction in Children and Adolescents. Bethesda, MD: U.S. Department of
 1479 Health and Human Services, National Institutes of Health; 2013.
- 1480
 1481
 1482

Part D. Chapter 7: Physical Activity

INTRODUCTION

The combination of a healthy diet and regular physical activity is central to promoting overall health and preventing many chronic diseases. The *Dietary Guidelines for Americans* first emphasized the importance of physical activity in 1990 and has included the topic in every edition in the two decades since. Although the 1990 and 1995 *Dietary Guidelines for Americans* discussed physical activity as a tool for managing and maintaining a healthy body weight, it broadened this perspective with the 2000 edition. Beginning in 2000, the *Dietary Guidelines for Americans*' physical activity content reflected the growing evidence base on the relationship between physical activity and various health outcomes. This evidence, from a wide range of well-conducted studies, clearly demonstrates that physically active people have improved growth and development, higher levels of fitness, a lower risk profile for developing a number of disabling medical conditions, and lower rates of various chronic diseases than do people who are less active or sedentary.¹

In 2008, the U.S. Department of Health and Human Services issued the first *Physical Activity Guidelines for Americans* (PAG).² The PAG serves as the benchmark and single, authoritative voice for science-based guidance on physical activity, fitness, and health for Americans 6 years and older (Table D7.1). The content of the PAG complements the *Dietary Guidelines for Americans*. Recognizing the dual importance of being physically active and eating a healthy diet to promote good health and reduce the risk of chronic diseases, therefore, the 2015 DGAC included a number of physical activity questions, including several related to body weight.

Table D7.1. 2008 Physical Activity Guidelines for Americans: Key Recommendations**Recommendations for Children and Adolescents Ages 6 to 17 Years**

Children and adolescents should do 60 minutes (1 hour) or more of physical activity daily.

- **Aerobic:** Most of the 60 or more minutes a day should be either moderate- or vigorous-intensity aerobic physical activity, and should include vigorous-intensity physical activity at least 3 days a week.
- **Muscle-strengthening:** As part of their 60 or more minutes of daily physical activity, children and adolescents should include muscle-strengthening physical activity on at least 3 days of the week.
- **Bone-strengthening:** As part of their 60 or more minutes of daily physical activity, children and adolescents should include bone-strengthening physical activity on at least 3 days of the week.
- It is important to encourage young people to participate in physical activities that are appropriate for their age, that are enjoyable, and that offer variety.

Recommendations for Adults Ages 18 Years and Older

- All adults should avoid inactivity. Some physical activity is better than none, and adults who participate in any amount of physical activity gain some health benefits.
- For substantial health benefits, adults should do at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity, or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week.
- For additional and more extensive health benefits, adults should increase their aerobic physical activity to 300 minutes (5 hours) a week of moderate intensity, or 150 minutes a week of vigorous intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity activity. Additional health benefits are gained by engaging in physical activity beyond this amount.
- Adults should also do muscle-strengthening activities that are moderate or high intensity and involve all major muscle groups on 2 or more days a week, as these activities provide additional health benefits.

Recommendations for Older Adults

The PAG recommendations for adults also apply to older adults. In addition, the following Guidelines are just for older adults (ages 65 years and older):

- When older adults cannot do 150 minutes of moderate-intensity aerobic activity a week because of chronic conditions, they should be as physically active as their abilities and conditions allow.
- Older adults should do exercises that maintain or improve balance if they are at risk of falling.
- Older adults should determine their level of effort for physical activity relative to their level of fitness.
- Older adults with chronic conditions should understand whether and how their conditions affect their ability to do regular physical activity safely.

24

25 Despite the consistent public health advice and encouragement to engage in regular physical
 26 activity, the majority of the U.S. population does not meet PAG recommendations. Using self-
 27 reported measures, in 2012 fewer than 21 percent of adults met the PAG recommendations for
 28 aerobic and muscle-strengthening physical activity, with fewer women than men meeting
 29 recommendations.³ As reported in the National Health Interview Survey, physical activity
 30 participation rates are lower in Blacks or African Americans and Hispanic or Latinos than in
 31 White populations. Older adults had the lowest participation rates across all adult age groups.³ In

32 2013, only 27 percent of adolescents met PAG recommendations; again, fewer girls than boys
 33 achieved recommended levels of physical activity.⁴

34

35 It is important to note that self-reported data on physical activity participation rates are likely to
 36 have significant over-reporting bias.⁵ Using objective accelerometer data on a nationally
 37 representative sample, Troiano et al. demonstrated that the percentage of the population meeting
 38 PAG recommendations was much lower than with self-report. For example, when considering
 39 bouts of moderate- to vigorous-intensity aerobic physical activity lasting 8 to 10 minutes or
 40 longer, less than 5 percent of adults met 2008 PAG recommendations.⁵ Nonetheless, some data
 41 indicate that Americans may be increasing their level of physical activity. Over the past six
 42 years, consistent data show a minimal, but positive, trend (Tables D7.2a and D7.2b).^{3,6-8}

43

Table D7.2a. Proportion of adults who self-report meeting the Physical Activity Guidelines for Americans recommendations for aerobic and muscle-strengthening physical activity

Population	2008	2009	2012	2013
Adult Total:	18.2%	19.0%	20.6%	*
Adult Male	21.7%	22.0%	24.3%	
Adult Female	14.9%	16.2%	17.1%	

Table D7.2b. Proportion of adolescents who self-report meeting the Physical Activity Guidelines for Americans recommendations for aerobic physical activity

Adolescent Total:	**	18.4%	**	27.1%
Adolescent Boys		24.8%		36.6%
Adolescent Girls		11.4%		17.7%

* National Health Interview Survey, 2013 data unavailable at time of publication.

** Youth Risk Behavior Surveillance was not conducted in 2008 or 2012.

Sources: Pleis, 2008; Pleis, 2009; Blackwell et al., 2014; CDC, 2010; CDC, 2014

44

45 To ensure sufficient discussion of physical activity for the population across the life cycle, as
 46 well as its relationship with a range of health outcomes, the DGAC reviewed the three major
 47 Federal reports on physical activity and health outcomes and selected specific questions for
 48 inclusion in this chapter. The Committee did not conduct independent formal systematic reviews
 49 of the evidence. This chapter summarizes the key evidence contained in these reports of the
 50 benefits of physical activity on health. Due to the extensive nature and number of evidence
 51 reviews within the three reports, the Committee refers readers to specific information using
 52 hyperlinks in each review of evidence found in this chapter.

53

54 **LIST OF QUESTIONS**

55 **Physical Activity and Health Outcomes in Children and Adolescents**

- 56 1. What is the relationship between physical activity, body weight, and health outcomes in
57 children and adolescents?

58

59 **Physical Activity and Health Outcomes in Adults**

- 60 2. What is the relationship between physical activity and body weight?

- 61 3. What is the relationship between physical activity and cardiorespiratory health?

- 62 4. What is the relationship between physical activity and metabolic health and risk of type 2
63 diabetes?

- 64 5. What is the relationship between physical activity and musculoskeletal health?

- 65 6. What is the relationship between physical activity and incidence of breast and colon cancer?

- 66 7. What is the relationship between physical activity and mental health?

67

68 **Physical Activity and Health Outcomes in People with Disabilities**

- 69 8. What is the relationship between physical activity and health outcomes in people with
70 disabilities?

71

72 **Physical Activity and Health Outcomes During Pregnancy and the Postpartum
73 Period**

- 74 9. Does being physically active during pregnancy and the postpartum period provide health
75 benefits?

76

77 **Physical Activity and Adverse Events**

- 78 10. What is the relationship between the amount and type of physical activity and the risk of
79 adverse events?

80

81 **Physical Activity Dose**

- 82 11. What dose of physical activity is most likely to provide health benefits in children and
83 adolescents?

- 84 12. What dose of physical activity is most likely to provide health benefits in adults?

- 85 13. Are there any special considerations for dose of physical activity for older adults?

86

87 **Physical Activity Interventions in Children and Adolescents**88 14. What is the relationship between physical activity participation and interventions in school-
89 based settings?90 15. What is the relationship between physical activity participation and interventions to change
91 the built environment?92 16. What is the relationship between physical activity participation and interventions based in
93 home settings?94 17. What is the relationship between physical activity participation and interventions based in
95 early care and education centers?96 18. What is the relationship between physical activity participation and interventions based in
97 primary health care settings?

98

99 **METHODOLOGY**

100 The DGAC agreed to use existing systematic reviews and reports to address the physical activity
101 topic area. The Committee used the PAG and two related reports—the *Physical Activity*
102 *Guidelines Advisory Committee Report, 2008* (PAGAC) and the *Physical Activity Guidelines for*
103 *Americans Midcourse Report*—as primary sources of evidence^{1,2,9} and discussed at its public
104 meetings questions that could be developed to frame the reports' key findings. The DGAC
105 reviewed and extracted information on the methodological approaches from each report and
106 identified key findings. The DGAC then carried forward verbatim conclusion statements from
107 the PAGAC Report and PAG Midcourse Report and concurred with 2008 PAG
108 recommendations to answer the questions. The DGAC subsequently assigned strength of
109 evidence grades and, based on the various report findings and conclusions, developed an overall
110 physical activity implications statement. Below is a brief description of each of the three reports.

111

112 [*Physical Activity Guidelines Advisory Committee Report, 2008*](#). In 2007, the Secretary of HHS
113 appointed a 13-member Physical Activity Guidelines Advisory Committee and charged them
114 with reviewing existing scientific literature to identify areas where sufficient evidence existed to
115 develop a comprehensive set of specific physical activity recommendations and highlight areas
116 where further scientific research was needed.¹ The PAGAC conducted systematic searches of the
117 scientific literature on physical activity and selected health outcomes in people ages 5 years and
118 older. Similar to the 2010 and 2015 DGAC, the PAGAC developed analytic frameworks for each
119 question and examined a diverse array of literature representing a number of study designs,
120 including randomized controlled trials (RCTs), non-randomized trials, prospective cohort
121 studies, case-control studies, and other observational studies. For each topic area, the PAGAC
122 used the best available and most appropriate body of evidence to answer specific questions. One

123 of the PAGAC’s major goals was to integrate the scientific information on the relationship
 124 between physical activity and health and to summarize it in a manner that could be used
 125 effectively by HHS to develop the *Physical Activity Guidelines for Americans* and related policy
 126 statements.

127
 128 [*Physical Activity Guidelines for Americans, 2008*](#). In 2008, HHS issued the PAG, which provides
 129 science-based guidance to help Americans ages 6 years and older improve their health through
 130 appropriate physical activity.² The 2008 PAG is designed to provide information and guidance
 131 on the types and amounts of physical activity that provide substantial health benefits. The
 132 primary audiences for the PAG are policymakers, health professionals, and interested members
 133 of the public.

134
 135 [*Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical
 136 Activity Among Youth*](#). In spring 2012, HHS convened a subcommittee of the President’s Council
 137 on Fitness, Sports & Nutrition to review the evidence on strategies to increase youth physical
 138 activity and make recommendations. The *Physical Activity Guidelines for Americans Midcourse
 139 Report*, released in 2013, is intended to identify interventions that can help increase physical
 140 activity in youth across a variety of settings.⁹ The subcommittee used a review-of-reviews
 141 approach to assess the current literature on interventions to increase physical activity in youth
 142 across five selected settings: schools, preschool and childcare centers, community, family and
 143 home, and primary health care. A total of 31 reviews covering 910 studies were examined. In its
 144 report, the subcommittee expanded the PAG’s age focus on those ages 6 years and older to
 145 include children ages 3 to 5 years.

146
 147 Overall, the DGAC concurs with the findings and evidence grades of the *Physical Activity
 148 Guidelines Advisory Committee Report, 2008*; the *2008 Physical Activity Guidelines for
 149 Americans*; and the *Physical Activity Guidelines for Americans Midcourse Report: Strategies to
 150 Increase Physical Activity Among Youth*.^{1,2,9} These reports state that being physically active is
 151 one of the most important steps that people of all ages can take to improve and maintain their
 152 health.

153
 154 **PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN CHILDREN AND
 155 ADOLESCENTS**

156 **Question 1: What is the relationship between physical activity, body weight, and
 157 health outcomes in children and adolescents?**

158 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*
 159

160 **Conclusion**

161 The DGAC concurs with the 2008 PAGAC, which found that strong evidence demonstrates that
 162 the physical fitness and health status of children and adolescents is substantially enhanced by
 163 frequent physical activity. Compared to inactive young people, physically active children and
 164 adolescents have higher levels of cardiorespiratory endurance and muscular strength, and well
 165 documented health benefits include lower body fatness, more favorable cardiovascular and
 166 metabolic disease risk profiles, enhanced bone health, and reduced symptoms of anxiety and
 167 depression. These conclusions are based on the results of prospective observational studies in
 168 which higher levels of physical activity were found to be associated with favorable health
 169 parameters as well as intervention studies in which exercise treatments caused improvements in
 170 physical fitness and various health-related factors. **DGAC Grade: Strong**

171

172 **Review of Evidence**

173 A body of RCTs, non-randomized trials, prospective cohort studies, case-control studies, other
 174 observational studies, and meta-analyses support the relationship between physical activity and
 175 physical fitness (i.e., cardiorespiratory fitness and muscular strength), healthy body weight and
 176 composition, cardio-metabolic health, bone health, and mental health (i.e., anxiety and
 177 depression).

178

179 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
 180 *Guidelines Advisory Committee Report, 2008 at*
 181 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

182 For evidence reviews on:

- 183 • Physical fitness, see Part G. Section 9: Youth
- 184 • Body weight and composition, see Part G. Section 9: Youth
- 185 • Cardio-metabolic health, see Part G. Section 9: Youth
- 186 • Bone health, see Part G. Section 9: Youth
- 187 • Mental health, see Part G. Section 9: Youth

188 **PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN ADULTS**

189 **Question 2: What is the relationship between physical activity and body weight?**

190 **Question 3: What is the relationship between physical activity and**
191 **cardiorespiratory health?**

192 **Question 4: What is the relationship between physical activity and metabolic**
193 **health and risk of type 2 diabetes?**

194 **Question 5: What is the relationship between physical activity and**
195 **musculoskeletal health?**

196 **Question 6: What is the relationship between physical activity and incidence of**
197 **breast and colon cancer?**

198 **Question 7: What is the relationship between physical activity and mental**
199 **health?**

200

201 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

202

203 **Conclusion**

204 The DGAC concurs with the 2008 PAGAC, which found that compared to less active people,
205 physically active adults and older adults exhibit a higher level of cardiorespiratory and muscular
206 fitness, healthier body weight and body composition, and a biomarker profile that is more
207 favorable for preventing cardiovascular disease (CVD) and type 2 diabetes and enhancing bone
208 health. In addition, there is an association between higher levels of physically activity in adults
209 and older adults and lower rates of all-cause mortality, coronary heart disease, high blood
210 pressure, stroke, type 2 diabetes, metabolic syndrome, colon cancer, breast cancer, and
211 depression. High-intensity muscle-strengthening activity enhances skeletal muscle mass,
212 strength, power, and intrinsic neuromuscular activation. Physically active adults who are
213 overweight or obese experience a variety of health benefits that are generally similar to those
214 observed in physically active people of ideal body weight. Physical activity reduces risk of
215 depression and is associated with lower risk of cognitive decline in adults and older adults.
216 Physical activity is associated with higher levels of functional health and a lower risk of falling
217 in older adults. **DGAC Grade: Strong**

218

219 In older adults with existing functional limitations, fairly consistent evidence indicates that
220 regular physical activity is safe and has a beneficial effect on functional ability. Consistent
221 evidence indicates that physically active adults and older adults have better quality sleep and
222 health-related quality of life. **DGAC Grade: Moderate**

223

224 **Review of Evidence**

225 A body of well-designed prospective cohort studies, case-control studies, and other observational
 226 studies exists for the relationship between regular physical activity and lower risk of all-cause
 227 mortality; coronary heart disease (CHD), CVD, and stroke; type 2 diabetes; metabolic syndrome,
 228 body weight, and body composition; bone health; functional health; cancer; and mental health. A
 229 body of RCTs and meta-analyses provides evidence for a positive effect of physical activity on
 230 blood pressure, atherogenic dyslipidemia, and cardiorespiratory fitness; body weight and body
 231 composition; bone health and muscular strength; falls risk; mental health; and type 2 diabetes.

232
 233 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
 234 *Guidelines Advisory Committee Report, 2008 at*
 235 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

236 For evidence reviews on:

- 237 • All-cause mortality, see Part G, Section 1: All-cause Mortality
- 238 • Coronary heart disease (CHD), CVD, and stroke; blood pressure, atherogenic
 239 dyslipidemia, and cardiorespiratory fitness, see Part G, Section 2: Cardiorespiratory
 240 Health
- 241 • Type 2 diabetes, see Part G, Section 3: Metabolic Health
- 242 • Metabolic syndrome, see Part G, Section 3: Metabolic Health
- 243 • Body weight and body composition, see Part G, Section 4: Energy Balance
- 244 • Bone health and muscular strength, see Part G, Section 5: Musculoskeletal Health
- 245 • Functional health and falls risk, see Part G, Section 6
- 246 • Cancer, see Part G, Section 7
- 247 • Mental Health, see Part G, Section 8

248

249 **PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN PEOPLE WITH** 250 **DISABILITIES**

251 **Question 8: What is the relationship between physical activity and health**
 252 **outcomes in people with disabilities?**

253 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

254

255 **Conclusion**

256 The DGAC concurs with the 2008 PAGAC, which found that for people with physical
 257 disabilities, strong evidence shows that exercise can increase cardiorespiratory, musculoskeletal,

258 and mental health outcomes; and for people with cognitive disabilities, strong evidence shows
 259 that exercise can improve musculoskeletal health and select functional health and mental health
 260 outcomes. **DGAC Grade: Strong**

261
 262 For people with physical disabilities, moderate evidence indicates that physical activity improves
 263 a variety of functional health outcomes and reduces the effects of certain types of secondary
 264 conditions (i.e., pain and fatigue associated with the primary disability); and for people with
 265 cognitive disabilities, moderate evidence indicates that physical activity improves
 266 cardiorespiratory health outcomes, musculoskeletal fitness, and metabolic health, and helps
 267 maintain healthy weight. **DGAC Grade: Moderate**

268
 269 For people with physical disabilities, limited evidence suggests physical activity may promote a
 270 healthy weight and improve metabolic health, and for people with cognitive disabilities, limited
 271 evidence suggests that physical activity may reduce secondary conditions. **DGAC Grade:**
 272 **Limited**

273
 274 Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on
 275 physical activity for people with disabilities (Table D7.3). The DGAC concurs with these
 276 recommendations.

277

Table D7.3. PAG Recommendations for Adults with Disabilities

- Adults with disabilities, who are able to, should get at least 150 minutes a week of moderate-intensity, or 75 minutes a week of vigorous-intensity aerobic activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week.
- Adults with disabilities, who are able to, should also do muscle-strengthening activities of moderate or high intensity that involve all major muscle groups on 2 or more days a week, as these activities provide additional health benefits.
- When adults with disabilities are not able to meet the Guidelines, they should engage in regular physical activity according to their abilities and should avoid inactivity.
- Adults with disabilities should consult their health-care provider about the amounts and types of physical activity that are appropriate for their abilities.

278

279 **Review of Evidence**

280 A body of RCTs, meta-analyses, and non-randomized trials provides evidence on physical
 281 activity in people with physical and cognitive disabilities. Non-randomized trials were included
 282 in the review of evidence for this question due to the high variability of physical and cognitive
 283 disabilities considered.

284

285 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
 286 *Guidelines Advisory Committee Report, 2008 at*
 287 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

288 For evidence reviews on:

- 289 • Physical and cognitive disabilities, see Part G, Section 11: Understudied Populations.
290 Review of the Science: Health Outcomes Associated with Physical Activity in People
291 With Disabilities (pages G11-2 to G11-35)

292

293 *For additional details about the PAG recommendations, visit:*

294 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

295

296 **PHYSICAL ACTIVITY AND HEALTH OUTCOMES DURING PREGNANCY** 297 **AND THE POSTPARTUM PERIOD**

298 **Question 9: Does being physically active during pregnancy and the postpartum**
299 **period provide health benefits?**

300 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

301

302 **Conclusion**

303 The DGAC concurs with the 2008 PAGAC, which found that while the benefits of maternal
304 physical activity have clearly been demonstrated, there is a lack of prospective, randomized
305 intervention studies in diverse populations. Based on current evidence, unless there are medical
306 reasons to the contrary, a pregnant woman can begin or continue a regular physical activity
307 program throughout gestation, adjusting the frequency, intensity, and time as her condition
308 warrants. Very little evidence exists for the dose of activity that confers the greatest health
309 benefits to women during pregnancy and the postpartum period. In the absence of data, it is
310 reasonable for women during pregnancy and the postpartum period to follow the moderate-
311 intensity physical activity recommendations set for adults unless specific medical concerns
312 warrant a reduction in activity. **DGAC Grade: Limited**

313

314 Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on
315 physical activity for women who are pregnant or in the postpartum period (Table D7.4). The
316 DGAC concurs with these recommendations.

317

Table D7.4. PAG Recommendations for Women During Pregnancy and the Postpartum Period

- Healthy women who are not already highly active or doing vigorous-intensity activity should get at least 150 minutes of moderate-intensity aerobic activity a week during pregnancy and the postpartum period. Preferably, this activity should be spread throughout the week.
- Pregnant women who habitually engage in vigorous-intensity aerobic activity or who are highly active can continue physical activity during pregnancy and the postpartum period, provided that they remain healthy and discuss with their health care provider how and when activity should be adjusted over time.

318

319 **Review of Evidence**

320 Laboratory investigations and observational studies provide evidence on physical activity during
321 pregnancy and the postpartum period.

322

323 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
324 *Guidelines Advisory Committee Report, 2008 at*
325 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

326

327 For evidence reviews on:

- 328 • Pregnancy and the postpartum period, see Part G, Section 11: Understudied Populations.
329 Review of the Science: Physical Activity During Pregnancy and the Postpartum Period
330 (pages G11-35 to G11-38)

331

332 *For additional details about the PAG recommendations, visit:*
333 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

334

335

336 **PHYSICAL ACTIVITY AND ADVERSE EVENTS**

337 **Question 10: What is the relationship between the amount and type of physical**
338 **activity and the risk of adverse events?**

339 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

340

341 **Conclusion**

342 The DGAC concurs with the 2008 PAGAC, which found that the benefits of regular physical
343 activity outweigh the inherent risk of adverse events. Risk of musculoskeletal injuries is lower
344 for non-contact (e.g., walking) and limited contact (e.g., baseball) activities than for contact (e.g.,
345 basketball) and collision (e.g., football) activities. The usual dose of regular physical activity is
346 directly related to the risk of musculoskeletal injury and inversely related to the risk of sudden
347 adverse cardiac events. The risk of musculoskeletal injuries and sudden cardiac adverse events is
348 directly related to the size of the difference between the usual dose of activity and the new or
349 momentary dose of activity. The most consistently reported risk factor for musculoskeletal
350 injuries and sudden cardiac adverse events is inactivity and low fitness. **DGAC Grade: Strong**

351

352 Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on
353 physical activity and reducing the risk of adverse events (Table D7.5). The DGAC concurs with
354 these recommendations.

355

Table D7.5. PAG Recommendations for Reducing the Risk of Adverse Events

To do physical activity safely and to reduce risk of injuries and other adverse events, people should:

- Understand the risks and yet be confident that physical activity is safe for almost everyone.
- Choose to do types of physical activity that are appropriate for their current fitness level and health goals, because some activities are safer than others.
- Increase physical activity gradually over time whenever more activity is necessary to meet the guidelines or health goals. Inactive people should “start low and go slow” by gradually increasing how often and how long activities are done.
- Protect themselves by using appropriate gear and sports equipment, looking for safe environments, following rules and policies, and making sensible choices about when, where, and how to be active.
- Be under the care of a health care provider if they have chronic conditions or symptoms. People with chronic conditions and symptoms should consult their health care provider about the types and amounts of activity appropriate for them.

356

357 **Review of Evidence**

358 A body of RCTs, meta-analyses, well-designed prospective cohort studies, and case control
359 studies provides evidence on physical activity and risk of adverse events.

360

361 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
362 *Guidelines Advisory Committee Report, 2008 at*
363 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

364 For evidence reviews on:

- Adverse events, see Part G, Section 10: Adverse Events

366

367 *For additional details about the PAG recommendations, visit:*
368 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

369

370 **PHYSICAL ACTIVITY DOSE**

371 **Question 11: What dose of physical activity is most likely to provide health**
372 **benefits in children and adolescents?**

373 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

374 **Conclusion**

375 The DGAC concurs with the 2008 PAGAC, which found that substantial evidence indicates
376 important health and fitness benefits can be expected to accrue to most children and adolescents
377 who participate daily in 60 or more minutes of moderate to vigorous physical activity. Also,

378 certain specific types of physical activity should be included in an overall physical activity
 379 pattern in order for children and adolescents to gain comprehensive health benefits. These
 380 include regular participation in each of the following types of physical activity on 3 or more days
 381 per week: resistance exercise to enhance muscular strength in the large muscle groups of the
 382 trunk and limbs, vigorous aerobic exercise to improve cardiorespiratory fitness and
 383 cardiovascular and metabolic disease risk factors, and weight-loading activities to promote bone
 384 health. **DGAC Grade: Strong**

385

386 Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on
 387 physical activity for children and adolescents (Table D7.1). The DGAC concurs with these
 388 recommendations.

389

390 **Review of Evidence**

391 A body of RCTs, meta-analyses, non-randomized trials, well-designed prospective cohort
 392 studies, case-control studies, and other observational studies supports the dose of physical
 393 activity most likely to provide health benefits in children and adolescents.

394

395 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
 396 *Guidelines Advisory Committee Report, 2008 at*
 397 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

398 For evidence reviews on:

- 399 • Children and adolescents, see Part G, Section 9: Youth

400

401 *For additional details about the PAG recommendations, visit:*
 402 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

403

404 **Question 12: What dose of physical activity is most likely to provide health** 405 **benefits in adults?**

406 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

407

408 **Conclusion**

409 The DGAC concurs with the 2008 PAGAC, which found that for overall public health benefit,
 410 data from a large number of studies evaluating a wide variety of benefits in diverse populations
 411 generally support 30 to 60 minutes per day of moderate- to vigorous-intensity physical activity
 412 on 5 or more days of the week. For a number of benefits, including all-cause mortality, coronary
 413 heart disease, stroke, hypertension, and type 2 diabetes in adults and older adults, lower risk is
 414 consistently observed at 2.5 hours per week of moderate- to vigorous-intensity activity. The

415 amount of moderate- to vigorous-intensity activity most consistently associated with
 416 significantly lower rates of colon and breast cancer and the prevention of unhealthy weight gain
 417 or significant weight loss by physical activity alone is in the range of 3 to 5 hours per week. The
 418 available evidence suggests that the major health benefits of physical activity and the dose
 419 needed for major health benefits are similar for all adults, regardless of race or ethnicity. For a
 420 variety of health and fitness outcomes, including chronic disease prevention, improvement of
 421 various disease biomarkers and the maintenance of a healthy weight, reasonably strong evidence
 422 demonstrates that amounts of moderate- to vigorous-intensity activity that exceed 150 minutes
 423 per week are associated with greater health benefits. **DGAC Grade: Strong**

424

425 Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on
 426 physical activity for adults ages 18 years and older (Table D7.1). The DGAC concurs with these
 427 recommendations.

428

429 **Review of Evidence**

430 A body of well-designed prospective cohort studies and case control studies provides evidence
 431 on physical activity dose most likely to provide health benefits in adults.

432

433 *For additional details on this body of evidence, visit: **Appendix E-2.49** and *Physical Activity*
 434 *Guidelines Advisory Committee Report, 2008* at
 435 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.*

436 For evidence reviews on:

- 437 • Adults, see Part E: Integration and Summary of the Science (pages E-23 to E-24)

438

439 *For additional details about the PAG recommendations, visit:*
 440 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

441

442 **Question 13: Are there any special considerations for dose of physical activity for** 443 **older adults?**

444 **Source of Evidence:** *Physical Activity Guidelines Advisory Committee Report, 2008*

445 **Conclusion**

446 The DGAC concurs with the 2008 PAGAC, which found that, because the exercise capacity of
 447 adults tends to decrease as they age, older adults generally have lower exercise capacities than
 448 younger persons. Thus, they may need a physical activity plan that is of lower absolute intensity
 449 and amount (but similar in self-perceived relative intensity and amount) than is appropriate for
 450 more fit people, especially when they have been sedentary and are starting an activity program.

451

452 For older adults at risk of falling, strong evidence exists that regular physical activity is safe and
453 reduces falls by about 30 percent. Most evidence supports a program of exercise with the
454 following characteristics: 3 times per week of balance training and moderate-intensity muscle-
455 strengthening activities for 30 minutes per session and with additional encouragement to
456 participate in moderate-intensity walking activities 2 or more times per week for 30 minutes per
457 session. Some evidence, albeit less consistent, suggests that tai chi exercises also reduce falls.
458 Successful reduction in falls by tai chi interventions resulted from programs conducted from 1 to
459 3 hours or more per week. No evidence indicates that planned physical activity reduces falls in
460 adults and older adults who are not at risk of falls. **DGAC Grade: Strong**

461
462 Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on
463 physical activity for adults ages 65 years and older (Table D7.1). The DGAC concurs with these
464 recommendations.

465

466 **Review of Evidence**

467 A body of RCTs, meta-analyses, and non-randomized trials provides evidence on physical
468 activity dose in older adults.

469

470 *For additional details on this body of evidence, visit: Appendix E-2.49 and Physical Activity*
471 *Guidelines Advisory Committee Report, 2008 at*
472 <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>.

473 For evidence reviews on:

- 474 • Older adults, see Part E: Integration and Summary of the Science (pages E-23 to E-24)

475

476 *For additional details about the PAG recommendations, visit:*
477 <http://www.health.gov/paguidelines/pdf/paguide.pdf>.

478 **PHYSICAL ACTIVITY INTERVENTIONS FOR CHILDREN AND**
 479 **ADOLESCENTS**

480 **Question 14: What is the relationship between physical activity participation and**
 481 **interventions in school-based settings?**

482 **Question 15: What is the relationship between physical activity participation and**
 483 **interventions to change the built environment?**

484 **Question 16: What is the relationship between physical activity participation and**
 485 **interventions based in home settings?**

486 **Question 17: What is the relationship between physical activity participation and**
 487 **interventions based in early care and education centers?**

488 **Question 18: What is the relationship between physical activity participation and**
 489 **interventions based in primary health care settings?**

490 **Source of Evidence:** *Physical Activity Guidelines for Americans Midcourse Report:*
 491 *Strategies to Increase Physical Activity Among Youth*

492

493 **Conclusion**

494 The DGAC concurs with the *Physical Activity Guidelines for Americans Midcourse Report:*
 495 *Strategies to Increase Physical Activity Among Youth*, which found that multi-component school-
 496 based interventions that include strategies such as physical education, active transportation, and
 497 activity breaks can increase physical activity in children and adolescents during school hours.

498 **DGAC Grade: Strong**

499

500 Reasonably consistent evidence suggests that changing the built environment as well as
 501 interventions in early care and education centers can increase physical activity in children and
 502 adolescents. **DGAC Grade: Moderate**

503

504 Evidence to date is insufficient to conclude that intervention strategies in home or primary health
 505 care settings increase physical activity in children and adolescents. **DGAC Grade: Grade Not**
 506 **Assignable**

507

508 **Review of Evidence**

509 A body of systematic reviews and meta-analyses supports interventions to increase physical
 510 activity in children and adolescents.

511

512 **For additional details on this body of evidence, visit: Appendix E-2.49** and *Physical Activity*
 513 *Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among*
 514 *Youth* at <http://www.health.gov/paguidelines/midcourse/pag-mid-course-report-final.pdf>.

515 For evidence reviews on:

- 516 • School-based interventions, see School Setting (pages 9 to 14)
- 517 • Early care and education interventions, see Preschool and Childcare Center Setting (page
518 15)
- 519 • Built environment interventions, see Community Setting (pages 16 to 18)
- 520 • Home-based interventions, see Family and Home Setting (page 19)
- 521 • Primary care interventions, see Primary Health Care Setting (pages 20 to 21)
- 522

523 **IMPLICATIONS**

524 Given the strong evidence for health benefits of regular physical activity as well as the low levels
 525 of adherence to national recommendations, every effort should be made to encourage and
 526 facilitate programs at multiple levels so that children, adults, and older adults can meet the 2008
 527 PAG in combination with the *Dietary Guidelines for Americans*. This can be achieved if
 528 programs, policies, and communication strategies are developed across sectors to increase
 529 opportunities for engaging in physical activity and to improve the built environment. Ultimately,
 530 these actions can create a culture of health that facilitates participation in regular physical
 531 activity. Individuals, communities, schools, health care, and the private and public sectors
 532 should:

- 533 • Ensure that all individuals have access to safe, affordable, and enjoyable modes of physical
534 activity throughout the day in the environments where they live, learn, work, and play.
535 These opportunities must include structured programming and informal modes of
536 transportation and play.
- 537 • Focus particular attention on people with the greatest health disparities, as these individuals
538 have the lowest physical activity participation rates but can gain the most health benefits by
539 being physically active.
- 540 • Support policies and promote programs for children, adolescents, adults, and older adults that
541 help set and reinforce a personal value system that instills a lifetime of physical activity.
- 542 • Enact effective policies and strengthen existing policies within schools, communities, health
543 care settings, housing, and worksites that promote opportunities for regular physical activity.
- 544 • Enact effective policies and strengthen existing policies that promote active transport (e.g.,
545 walking and bicycling) within and between communities.
- 546 • Develop and promote programs to create or enhance access to safe and enjoyable places to be
547 physically active, including public spaces and local, state, and national parks.

- 548 • Develop and implement ongoing physical activity promotion campaigns that involve high-
549 visibility and multiple delivery channels and multiple sectors of influence.
- 550 • Coordinate efforts between numerous Federal and non-Federal initiatives, such as the
551 President’s Council on Fitness, Sports and Nutrition, *Let’s Move!*, the National Physical
552 Activity Plan, and Active Schools Acceleration Project.

553
554
555

CHAPTER SUMMARY

556 The findings outlined in this chapter provide strong evidence supporting the importance of
557 regular physical activity for health promotion and disease prevention in the U.S. population.
558 Physical activity is important for all people—children, adolescents, adults, older adults, women
559 during pregnancy and the postpartum period, and individuals with disabilities. The findings
560 further provide guidance on the dose of physical activity needed across the lifecycle to realize
561 these significant health benefits.

562
563 Future Physical Activity Guidelines Advisory Committees will be asked to carefully review the
564 most recent evidence so that the Federal government can fully update the PAG. Given the
565 exceedingly low physical activity participation rates in this country, it will be critically important
566 for the next PAGAC to identify proven strategies and approaches to increase population-level
567 physical activity across the lifespan.

568
569

NEEDS FOR FUTURE RESEARCH

- 571 1. Evaluate best practices in programming at the community and national level and identify
572 which local and national policies in the public and private sector have demonstrated the
573 greatest effect on increasing physical activity participation across the lifespan, especially in
574 populations with the greatest health disparities.

575 **Rationale:** Physical activity participation rates are exceptionally low across all age groups,
576 and are especially low in individuals with the greatest health disparities. Many different
577 initiatives are currently underway in the private and public sector to help increase physical
578 activity on a population level. Understanding which programs and policies are having the
579 greatest impact will help focus valuable resources and national recommendations for
580 maximum public health benefit.

- 581
582 2. Identify the dose of physical activity needed to achieve health benefits, as well as appropriate
583 growth and development, for children younger than age 6 years.

584 **Rationale:** Until recently, very little effort has been focused on understanding the health
585 benefits of physical activity for young children. Given that this is a critical age of growth and
586 development, considerable research should be focused on this age group.

587
588
589

3. Evaluate the effects of various modes and doses of physical activity on health outcomes in older adults.

590
591
592
593
594

Rationale: Older adults are the fastest growing segment of the population. They also have the greatest burden of disease and functional (mental and physical) limitations. To reduce burden of disease and related economic impacts, research regarding mode and dose of physical activity should be focused on this age group.

595
596

4. Further evaluate the importance of light activity, short bouts of physical activity (i.e., 10-minutes or less) and modes of activity on health outcomes across the lifespan.

597
598
599
600
601
602

Rationale: The review of the evidence in the 2008 PAGAC Report focused primarily on moderate- and vigorous-intensity activity. Emerging research highlights the positive effects of light activity as well as shorter bouts of vigorous activity on health outcomes. Understanding the health impact of the full range of mode, intensity, duration, frequency, and setting will help to further refine the PAG to support maximum public health benefit.

603
604

5. Further investigate the effects of sedentary behaviors on health outcomes, including duration, frequency, and mode of sedentary activities.

605
606
607
608

Rationale: Increasing evidence demonstrates the negative health consequences of sedentary behaviors. Clarity on the types and duration of sedentary behaviors that have the most negative health impact would help to identify meaningful evidence-based public health recommendations.

609

REFERENCES

610
611

612
613
614
615
616
617
618
619
620
621
622
623
624
625
626
627
628
629

1. Physical Activity Guidelines Advisory Committee. Physical Activity Guidelines Advisory Committee Report, 2008. Washington, DC: U.S. Department of Health and Human Services; 2008. Available from: <http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf>
2. U.S. Department of Health and Human Services. 2008 Physical Activity Guidelines for Americans. Washington, DC: U.S. Department of Health and Human Services; 2008. Available from: <http://www.health.gov/paguidelines/pdf/paguide.pdf>
3. Blackwell DL, Lucas JW, Clarke TC. Summary health statistics for U.S. adults: national health interview survey, 2012. *Vital and health statistics. Series 10, Data from the National Health Survey*. Feb 2014(260):1-161. PMID: 24819891. <http://www.ncbi.nlm.nih.gov/pubmed/24819891>
4. Kann L, Kinchen S, Shanklin SL, Flint KH, Kawkins J, Harris WA et al. Youth Risk Behavior Surveillance — United States, 2013. *MMWR*. 2014;63(4). PMID: 24918634. <http://www.ncbi.nlm.nih.gov/pubmed/24918634>
5. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. *Medicine and science in sports and exercise*. Jan 2008;40(1):181-188. PMID: 18091006. <http://www.ncbi.nlm.nih.gov/pubmed/18091006>
6. Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. *Vital and health statistics. Series 10, Data from the National Health*

- 630 *Survey*. Dec 2009(242):1-157. PMID: 20821903.
631 <http://www.ncbi.nlm.nih.gov/pubmed/20821903>
632 7. Pleis JR, Ward BW, Lucas JW. Summary health statistics for U.S. adults: National Health
633 Interview Survey, 2009. *Vital and health statistics. Series 10, Data from the National Health*
634 *Survey*. Dec 2010(249):1-207. PMID: 21905346.
635 <http://www.ncbi.nlm.nih.gov/pubmed/21905346>
636 8. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, et al. Youth Risk Behavior
637 Surveillance — United States, 2009. *MMWR*. 2010;59(5).
638 <http://www.ncbi.nlm.nih.gov/pubmed/20520591>
639 9. U.S. Department of Health and Human Services. Physical Activity Guidelines for Americans
640 Midcourse Report: Strategies to Increase Physical Activity Among Youth. Washington, DC:
641 U.S. Department of Health and Human Services; 2012.
642 <http://www.health.gov/paguidelines/midcourse/pag-mid-course-report-final.pdf>
643

644

645

Appendix E-1: Needs for Future Research

CHAPTER 1: FOOD AND NUTRIENT INTAKES AND HEALTH: CURRENT STATUS AND TRENDS

1. Expand WWEIA participation to include more respondents from race/ethnic minorities and non-U.S. born residents.

Rationale: Very little is known about the dietary habits of many of the cultural subgroups in the United States. This knowledge is essential to moving forward any nutrition programs for first and second generation immigrants. More data on the impact of acculturation also are needed on food and health behaviors. The number of participants in WWEIA using the derived acculturation variable was too small for any analysis. Finally, “Hispanic” is a very broad term and a better understanding is needed of the nutritional profiles (including shortfalls and excesses) across various Spanish-speaking people in the United States, who come from different cultural backgrounds with distinct eating patterns.

2. Include higher proportion of older Americans as respondents in WWEIA.

Rationale: More data are needed on dietary intake of older adults; the sample sizes in WWEIA were too small for any meaningful analyses for those older than the age of 71 years. In addition to nutrient intake, additional information is needed on whether older adults are able to shop and cook, whether polypharmacy plays a role in nutritional adequacy, and whether co-morbidities, such as poor dentition, musculo-skeletal difficulties, arthralgias and other age-related symptoms, affect their ability to establish and maintain proper nutritional status.

3. Increase the number of pregnant women as respondents in WWEIA.

Rationale: The number of pregnant women in WWEIA is currently too small to properly evaluate the status and trends in food and nutrient intake in pregnant women. Since good nutrition in pregnancy is critical to proper growth development of the infant it is critical to properly evaluate food and nutrient intake, which will inform recommendations and public policies for pregnant women.

4. Conduct research on nutrition transitions from childhood to shed light on how and why dietary intake changes so rapidly from early childhood through pre-adolescence and adolescence, and to identify the driving forces behind dietary intake change in these age groups and what programs are most effective at maintaining positive nutrition habits established in very young children.

Rationale: Young children have better dietary intake than older children and adolescents. It is important to maintain the positive gains made in early childhood and identify factors

38 responsible for the declines in intakes of fruit, dairy, and other food groups and increases in
 39 added sugars and refined grains as children become enter the elementary school age years, as
 40 poor eating patterns in elementary school seem to persist into adolescence and beyond.

41

- 42 5. Evaluate the effects of common variations in dietary patterns in small children on nutrient
 43 intakes.

44 **Rationale:** Children from 2 to 4 years of age have a highly variable diet and often do not fit
 45 readily into the USDA Food Pattern food groups diet pattern analyses. Further information is
 46 needed to understand the broad range of diets and supplement use in small children and how
 47 this relates to nutrient intake and growth. Research is needed to better characterize their diets
 48 so that appropriate guidance can be offered.

49

- 50 6. Increase the quantity and quality of food composition databases available for research.

51 **Rationale:** Accurate assessment of nutrient intake and trends over time in the U.S.
 52 population is dependent upon the quality of food composition data. Tens of thousands of
 53 foods are available for purchase and consumption in the United States, but accurate nutrient
 54 content data are available only for less than 10,000 foods and are almost non-existent for
 55 many ready-to-eat and restaurant-type foods. Analytic values from foods are needed on
 56 specific nutrients and components, such as vitamin D, fiber, added sugars, and sodium.
 57 Improved food composition data also is critical for needed research to better define, identify,
 58 and quantify total grain, whole grain consumption, and refined grain consumption in dietary
 59 studies.

60

- 61 7. Investigate the validity, reliability, and reproducibility of new biomarkers of nutrient intake
 62 and biomarkers of nutritional status.

63 **Rationale:** Limited biomarkers are available and some that are available are difficult to
 64 interpret due to other contributing factors to the biomarker measure (e.g., vitamin D is
 65 obtained in the diet and is also endogenously synthesized).

66

- 67 8. Evaluate effects of fortification strategies and supplement use on consumer behavior related
 68 to the intake of foods and supplements containing key nutrients, including calcium, vitamin
 69 D, potassium, iron, and fiber

70 **Rationale:** The intake of key nutrients of concern is considerably affected by the rapidly
 71 evolving marketplace of food fortification and supplementation. Understanding consumer
 72 behavior related to fortification and supplementation would be important in predicting the
 73 effects of interventions and marketplace changes in content of these nutrients. Special
 74 interest exists regarding fortification strategies of foods, including whole grains and yogurts,
 75 in allowing individuals to reach the RDA for vitamin D without using supplements. Data are
 76 needed on how supplements may help meet nutrients shortfalls and/or how use of

77 supplements may place individuals at risk of overconsumption. Research on effective
78 consumer guidance is needed.

79

80 9. Understand the rationale for and consequences of the use of supplements above the UL for
81 vitamins and minerals. Identify biochemical markers that would indicate the effects of high-
82 dose supplement use.

83 **Rationale:** Consumer use of high-dose supplements has increased. Understanding the
84 influences guiding this use would be helpful in considering how to educate consumers about
85 safe upper intake limits.

86

87 10. Develop a standardized research definition for meals and snacks.

88 **Rationale:** Multiple different criteria are used in studies to define a snack or meal occasion,
89 such as time of day, the types or amounts of food consumed, or subjective assessment by the
90 study respondent. Researchers should work toward a consensus on the use of standard
91 definitions.

92

93 11. Understand better the concept of dietary patterns and design approaches to quantify the diet
94 in large population-based studies.

95 **Rationale:** More methodological work on dietary patterns is needed. For example, food
96 frequency questionnaires, which are used in most diet assessment studies, do not capture data
97 on meal timing, meal frequency, or the types of foods consumed together. Studies using diet
98 recalls and records are better at capturing specific foods and their quantities consumed
99 (portion sizes) and the types of foods eaten together, but often these detailed assessment
100 methods are not feasible for large population-based studies. Quantification of food group
101 intake is needed. In addition, dietary patterns research encompasses a broader scope of issues
102 than can be addressed by diet scores and data drive approaches.

103

104 12. Consistently report the nutrients, foods, and food groups that are used to evaluate dietary
105 patterns in published studies.

106 **Rationale:** The current scientific literature evaluating dietary patterns and health is
107 inconsistent in its provision of dietary patterns composition information. This makes it
108 difficult to compare, across studies, the components of healthful patterns that are associated
109 with health benefits.

110

111 13. Conduct population surveillance on the prevalence and trends of nutrition-related chronic
112 diseases including type 2 diabetes, cardiovascular disease, some cancers osteoporosis and
113 neurocognitive disorders.

114 **Rationale:** Current data on diabetes in adults cannot be stratified by disease type (type I or
115 type II), making it very difficult to monitor incidence and prevalence of type 2 diabetes.

116 Continued population surveillance is needed to effectively link nutritional factors with risk of
 117 these diseases.

118
 119

120 **CHAPTER 2: DIETARY PATTERNS, FOODS AND NUTRIENTS, AND** 121 **HEALTH OUTCOMES**

122 1. Conduct additional dietary patterns research for other health outcomes to strengthen the
 123 evidence beyond CVD and body weight in populations of various ethnic backgrounds and
 124 life course stages in order for future DGACs to draw stronger conclusions.

125 **Rationale:** The NEL systematic reviews demonstrated that considerable CVD research
 126 related to dietary patterns is available. However, it also is important to note, that unlike CVD,
 127 some of the other health outcomes are more heterogeneous and thus may require greater
 128 specificity in the examination of diet and disease risk. There is a clear need for all studies
 129 examining the relationship between dietary patterns and health outcomes to include the full
 130 age spectrum and to take a life course perspective (including pregnancy); insufficient
 131 research is being devoted to children and how diseases may evolve over time. An increased
 132 emphasis should be placed on understanding how the diets of all those in the U.S. population
 133 from various ethnic backgrounds may be associated with health outcomes, thereby
 134 broadening knowledge beyond Hispanics and African Americans to include the diversity that
 135 exists in the United States today. This may require our national nutrition monitoring
 136 programs to over-sample individuals from other national origins to conduct subgroup
 137 analysis.

138

139 2. Improve the understanding of how to more precisely characterize dietary patterns by their
 140 food constituents and the implications of the food constituents on nutrient adequacy through
 141 the use of Food Pattern Modeling. More precise characterization, particularly of protein
 142 foods, is needed.

143 **Rationale:** Researchers are characterizing dietary patterns very differently and yet
 144 sometimes use similar nomenclatures. This makes it difficult to compare results across
 145 studies and as demonstrated in the NEL systematic reviews, can impair the grading of the
 146 body of evidence as strong. The reason why researchers are not replicating others findings in
 147 different populations may be a function of publication bias. It is important for editors of
 148 scientific journals and peer reviewers to appreciate the replication of findings first and then
 149 value a research group's methodological nuance that may improve the examination of the
 150 association between dietary patterns and a health outcomes. Perhaps what should be stressed
 151 is a harmonization of research methods across various cohorts or randomized trials, similar to
 152 what is being done at the National Cancer Institute's Dietary Patterns Methods Project^{9, 220}
 153 led by Drs. Krebs-Smith and Reedy. The use of Food Pattern Modeling as demonstrated in
 154 Chapter 1 allows questions about the adequacy of the dietary patterns given specific food

155 constituents to be addressed and how modifications of the patterns by altering the foods for
 156 specific population groups or to meet specific nutrient targets can be achieved.

157
 158 3. Examine the long-term cardio-metabolic effects of the various dietary patterns identified in
 159 the AHA/ACC/TOS Guidelines for the Management of Overweight and Obesity in Adults
 160 that are capable of resulting in short-term weight loss (see Question 2, above).

161 **Rationale:** Although the research to date demonstrates that to lose weight, a variety of
 162 dietary pattern approaches can be used if a reduction in caloric intake is achieved, the long-
 163 term effects of these diets on cardio-metabolic health are not well known. Emerging research
 164 is exploring health effects of variations of the low-carbohydrate, higher protein/fat dietary
 165 pattern. In some approaches (such as Atkins), the dietary pattern which emphasizes animal
 166 products, may achieve a macronutrient composition that is higher in saturated fat. Others
 167 may emphasize plant-based proteins and fats and may achieve a lower saturated fat content
 168 and may be higher in polyunsaturated fats and dietary fiber. Research is needed to determine
 169 the impact of these alternative approaches, and perhaps others, on CVD risk profiles as well
 170 as other health outcomes. As mentioned in the review of the literature associated with
 171 saturated fat and cardiovascular disease in Chapter 6: Cross-Cutting Topics of Dietary
 172 Guidance and Public Health Importance, substituting one macronutrient for another may
 173 result in unintended consequences. Careful consideration to the types of foods that are used
 174 in these diets and in particular the type of fat and amount of added sugars should be taken
 175 into account.

176
 177
 178 **CHAPTER 3: INDIVIDUAL DIET AND PHYSICAL ACTIVITY BEHAVIOR**
 179 **CHANGE**

180 **Eating Out**

181 1. Develop a standard methodology to collect and characterize various types of eating venues.

182 **Rationale:** This recommendation is fundamental to conducting rigorous research, evaluating
 183 findings from multiple studies, and developing policies to promote healthy eating among
 184 people who frequent eating out venues and/or consume take away meals.

185
 186 2. Conduct rigorously designed research to examine the longitudinal impact of obtaining or
 187 consuming meals away from home from various types of commonly frequented venues on
 188 changes in food and beverage intakes (frequency, quantity, and composition), body weight,
 189 adiposity, and health profiles from childhood to adulthood in diverse (racial/ethnic,
 190 socioeconomic, cultural, and geographic) groups of males and females.

191 **Rationale:** Most groups in the U.S. population regularly consume meals that are prepared
 192 away from home and the landscape of fast food and other types of food procurement and

193 consumption venues is increasingly complex. The potential for eating out and/or take away
 194 meals to influence diet quality, energy balance, body mass and composition, and the risks of
 195 health-related morbidities across the life course among our diverse population underscores the
 196 importance of understanding this issue.

197

198 **Family Shared Meals**

199 3. Conduct studies in diverse populations that assess not only frequency of family shared meals,
 200 but also quality of family shared meals.

201 **Rationale:** Our understanding of the importance of family shared meals in terms of how they
 202 contribute in a positive way to body weight and overall health and well-being requires a
 203 rigorous examination of the dietary quality of these meals compared to other meals consumed
 204 by family members.

205

206 4. Conduct RCTs to isolate the effect of interventions that increase the frequency of family
 207 meals from other health and parenting behaviors that may be associated with dietary intake
 208 and weight status.

209 **Rationale:** Family shared meals are commonly implemented as one component of lifestyle
 210 interventions that include an array of other behavioral and parenting strategies for weight
 211 management. To improve our understanding of the causal pathway of how family shared meals
 212 contributes to maintaining or achieving a health weight, the specific contribution of family
 213 shared meals to weight outcomes independent of other behavioral strategies needs to be
 214 ascertained.

215

216 **Sedentary Behavior**

217 5. Develop improved and better standardized and validated tools to assess sedentary behaviors
 218 and activities that children, adolescents, and adults regularly engage in.

219 **Rationale:** Our understanding of the impact of sedentary behaviors on diet, energy balance,
 220 body mass, adiposity, and health is currently compromised by reliance on subjective
 221 assessments, including self-reports of daily activity patterns, and by inadequate techniques to
 222 document and quantify the array of sedentary activities people engage in (beyond TV viewing
 223 and (or) computer screen time). It also would be beneficial for researchers to document the
 224 potential benefits and implications of reducing one type of sedentary behavior (e.g. screen
 225 time) on other sedentary behaviors (e.g., reading for leisure, arts and crafts, listening to music)
 226 and indices of health (e.g. sleep quality and duration).

227

228 6. Conduct prospective research to examine the effects and mechanisms of the quantity,
 229 patterns, and changes of sedentary behaviors on diet quality, energy balance, body weight,

230 adiposity, and health across the life span in groups within the U.S. population with diverse
231 personal, cultural, economic, and geographic characteristics.

232 **Rationale:** Emerging, but limited, evidence implicates sedentary behaviors with adverse
233 health-related outcomes, especially in children and adolescents as they transition into
234 adulthood. However, an improved understanding of why these relationships exist will help in
235 developing appropriate and effective approaches and policies to reduce the amount of time
236 people spend engaging in sedentary behaviors.

237

238 **Self-Monitoring**

239 7. Evaluate the impact of different types, modalities, and frequencies of self-monitoring on
240 body weight outcomes during both the weight loss intervention and maintenance periods.

241 **Rationale:** Self-monitoring is associated with improved weight management. However, the
242 current practice of recommending daily self-monitoring may represent a barrier to its
243 implementation and/or continued use. Hence, it is important to determine whether lower
244 frequencies of self-monitoring can produce beneficial effects on weight outcomes.

245

246 8. Evaluate the comparative effectiveness of performance feedback from self-monitoring
247 delivered through automated systems versus personal interactions with a counselor.

248 **Rationale:** Automated feedback derived from self-monitoring data and delivered
249 electronically can produce beneficial changes on weight outcomes. However, the comparative
250 effectiveness and cost efficiency of feedback delivered through non-personal modalities versus
251 personal interactions has yet to be determined.

252

253 9. Test the effectiveness of self-monitoring on weight outcomes in understudied groups,
254 including ethnic/racial minorities, low education, low literacy, and low numeracy
255 populations, males, and subjects younger than age 30 years and older than age 60 years.

256 **Rationale:** Evidence regarding the effectiveness of self-monitoring has been derived largely
257 from research conducted on well educated, middle-class, white women. Hence, it is important
258 to determine whether the beneficial effects of self-monitoring on weight outcomes are
259 generalizable to understudied groups.

260

261 10. Conduct RCTs based on sound behavioral change theories that incorporate self-monitoring,
262 employ heterogeneous populations, and are powered for small effect sizes and high attrition
263 rates, to test the short- (e.g., 3 months) and long-term (e.g., 12 months) effects of mobile health
264 technologies on dietary and weight outcomes.

265 **Rationale:** Mobile health technologies have the potential to reach larger portions of the
266 populations than face-to-face interventions, but the effect sizes of mobile technologies may be
267 small and the attrition rates may be large. Larger, more representative study populations and

268 longer study periods will permit an assessment of the generalizability and sustainability of
 269 mobile health technologies.

270

271 **Food and Menu Labeling**

272 11. Develop novel labeling approaches to provide informative strategies to convey caloric intake
 273 values on food items consumed at home and in restaurant settings.

274 **Rationale:** Menu labels can include different types of information in addition to calories.
 275 These include physical activity equivalents, and daily caloric needs. Very few studies have
 276 been designed to examine the optimal combination of menu label information to prevent
 277 excessive caloric intake. This will be very valuable evidence to inform the calorie label policy
 278 that has just been enacted by the FDA.

279

280 12. Compare labeling strategies across various settings, such as restaurants, stores, and the home
 281 to determine their efficacy in altering food selection and health outcomes, including weight.

282 **Rationale:** The great majority of menu labeling RCT's have been conducted under laboratory
 283 conditions. Given the recent FDA regulations, future studies will be able to impact the
 284 effectiveness of these polices across settings as accessed by diverse free living populations.

285

286 13. Evaluate the process and impact of recent FDA menu labeling regulation.

287 **Rationale:** The new FDA regulation provides a unique opportunity to understand the impact of
 288 menu labeling on consumers dietary behaviors in "real world" settings.

289

290 **Household Food Insecurity**

291 14. Conduct prospective cohort studies that cover a wide age range and include children,
 292 families, older adults, and ethnically/racially diverse populations and describe potential effect
 293 modifiers such as gender, ethnic and cultural factors, family structure, area of residence (i.e.,
 294 urban vs. rural), employment, and use of social support systems while examining the
 295 relationship between household food insecurity, dietary intake, and body weight.

296 **Rationale:** Understanding the temporal process of when and how long food insecurity occurs
 297 within a family/individual's lifetime and their response to this economic stressor is critical to
 298 conducting rigorous research and comparing finding across studies in order to develop and
 299 implement intervention studies and policies to alleviate this public health problem.

300

301 15. Standardize research methodology, including developing a consistent approach to measuring
 302 food insecurity and use of measured height and weight to reduce the likelihood of responder
 303 bias.

304 **Rationale:** The measurement error issues related to the use of self-reported weight have been

305 well documented in the literature. In order to conduct rigorous studies in this area that can be
 306 compared and evaluated as to the causal nature of the role of food insecurity on body weight,
 307 standard methodology is warranted both in the measurement of the exposure as well as the
 308 outcome.

309

310 **Acculturation**

311 16. Conduct prospective longitudinal studies, including those that start in early childhood to
 312 track dietary intake, sedentary behaviors, body weight, and chronic disease outcomes across
 313 the life course. Include the diversity of ethnic/racial groups in the United States, including
 314 individuals and families of diverse national origins. Include comparison groups in countries
 315 of origin to rule out, among other things, the potential confounding by internal migration
 316 from rural to urban area within the country of origin.

317 **Rationale:** Acculturation is a time-dependent life course process that requires longitudinal
 318 studies to be properly understood. Because the impact of acculturation on dietary, weight and
 319 health outcomes can be expected to be modified by the life course stage of life when
 320 individuals migrate to the United States, prospective acculturation studies need to start
 321 following individuals from very early childhood.

322

323 17. Develop a standard tool to measure acculturation or validation of multidimensional
 324 acculturation scales in different immigrant groups and in different languages.

325 **Rationale:** Acculturation is a complex construct that is seldom measured with
 326 multidimensional scales that can capture the different paths that migrant scan take with
 327 regards to the acculturation process, including assimilation, integration, segregation, and
 328 marginalization. Although research in acculturation measurement has been conducted among
 329 Hispanic/Latinos, it has been predominantly based on Mexican American populations and
 330 little acculturation measurement research has been conducted among other groups, including
 331 individuals from Asia, Africa, Europe, and the Middle East.

332

333 **Sleep Patterns**

334 18. Conduct prospective studies that start in childhood (including transition to adulthood), to
 335 investigate the longitudinal effect of sleep patterns on diet and body weight outcomes while
 336 accounting for confounders, mediators, and moderators including: physical activity,
 337 socioeconomic variables (such as education, employment, household income), sex, alcohol
 338 intake, smoking status (including new smoker, new non-smoker), media use/screen time, and
 339 depression.

340 **Rationale:** While research associates short sleep duration and disordered sleep patterns with
 341 adverse differences and changes in food and beverage consumption, body weight, and indices
 342 of metabolic and cardiovascular health, less is known about the impact of potential modifying

343 lifestyle factors. This research will help delineate the role of sleep patterns, duration and
 344 quality, i.e., mediator or moderator, on diet and weigh-related outcomes. Research in children
 345 shows that sleep deprivation and weight are related but this relationship is not apparent in adult
 346 studies. This may be due to the fact that energy intake increases during transition to short sleep
 347 duration, but levels off when short sleep duration becomes consistent.

348

349 19. Conduct studies to assess the effects of diet on sleep quality to examine the mechanism by
 350 which dietary intake, energy intake, and energy expenditure may impact sleep.

351 **Rationale:** Most research has focused on sleep quality and duration as modifying factors on
 352 diet, body weight, and health. A paucity of research exists on the potential impact of diet on
 353 sleep-related outcomes. This line of research would use diet as the means to improve indices of
 354 sleep, which in turn may subsequently improve health-related outcomes.

355

356

357 CHAPTER 4: FOOD ENVIRONMENT AND SETTINGS

358 1. Develop more valid and reliable methods for measuring all aspects of the food environment,
 359 including the total food environment of communities. These methods can then be used to
 360 assess the impact of the food environment on community health as well as on economic
 361 development and growth.

362 **Rationale:** The food environment has become more complex, with more and more retail
 363 outlets selling food and beverages. Having valid and reliable methodologies for a variety of
 364 food environments and settings (tools and new analytical approaches) will allow more
 365 meaningful inquiry into the contributions of various settings in supporting or hindering
 366 nutritional health.

367

368 2. Identify, implement, evaluate, and scale up best practices (including private-public
 369 partnerships) for affordable and sustainable solutions to improving the food environment and
 370 increasing food access, especially in those environments of greatest need.

371 **Rationale:** The environments in which people live, work, learn, and play greatly influence
 372 their food intake. To best guide efforts to improve the food environment, research is needed
 373 to identify and evaluate best practices to direct available resources to new programs and scale
 374 up.

375

376 3. Identify, implement, accelerate, evaluate, and scale up programs that improve access to
 377 healthy food and that can be integrated seamlessly with Federal nutrition assistance
 378 programs, such as SNAP, WIC and elder nutrition.

379 **Rationale:** Federal nutrition assistance programs reach individuals and populations with the
 380 greatest health disparities. Identifying and evaluating initiatives that integrate improvements

381 in the food environment with Federal programs will help ensure that Federal nutrition
382 assistance programs have as great an impact as possible.

383

384 4. Conduct additional obesity prevention intervention research in child care settings (e.g., child-
385 care centers, family child-care homes) to: 1) Identify the most potent components of the
386 interventions and the optimal combinations for improving diet quality, physical activity, and
387 weight outcomes; 2) Assess implementation and translation costs and benefits of the
388 intervention, including impact, cost-effectiveness, generalizability and reach, sustainability
389 and feasibility; 3) Develop and evaluate culturally appropriate and tailored interventions for
390 preschool children in low-income and racial/ethnic communities, given the disproportionate
391 impact of obesity in these groups; 4) Explore intervention strategies on how to use child care
392 settings as access points to create linkages to parents, caretakers, and health care providers as
393 partners in health promotion; 5) Evaluate the impact of Federal, state, and local policies,
394 regulations, and support (e.g., provider training and technical assistance) for child care
395 programs on the eating and physical activity practices and behaviors, and weight status of
396 young children.

397 **Rationale:** Early care and education settings are an important venue for interventions
398 targeting young children. A strong evidence base is essential to identify and support
399 evidence-based practices and policies that can be implemented at Federal, state, and local
400 levels and to mobilize efforts to improve healthy eating and physical activity, leading to
401 healthy weight development in these settings. Interventions found to effectively reduce risk
402 of obesity in one setting need to be appropriately adapted for diverse groups and different
403 settings.

404

405 5. Improve intervention research methods by the use of stronger study designs and the
406 development of standardized assessments of body composition, weight status. Develop
407 enhanced validated measures of diet quality, feeding and physical activity practices, and
408 physical activity and eating behaviors and policies. Create standardized measures to assess
409 the nutrition quality of meals and snacks in child care settings, as well as the food and
410 physical activity environments. Create standardized methods for assessing the relationship of
411 child care food, nutrition and physical activity-related measures to similar measures
412 representing non-child care time are needed to provide greater consistency in determining the
413 contributors to the development and progression of childhood overweight and obesity.

414 **Rationale:** Although many of the studies included in these evidence reviews were
415 methodologically strong and were controlled studies, some were limited by small sample
416 size, lack of adequate control for confounding factors, and different outcome measures and
417 different tools used to measure the outcome variables.

418

- 419 6. Examine the effect of the recommended Child and Adult Care Food Program (CACFP)
 420 through ongoing periodic evaluations and fill gaps in the knowledge regarding participation,
 421 demand, food procurement and practices, nutrient intake, and food security.

422 **Rationale:** Improvements in school meals and the school food environment have been
 423 fostered by national data from periodic studies such as the USDA/FNS School Nutrition
 424 Dietary Assessment Studies (SNDA), the HHS/CDC School Health Policies and Practices
 425 Studies (SHPPS) and the HHS/NIH C.L.A.S.S. In contrast, considerably fewer periodic
 426 national studies are conducted of meals and dietary intake in child care settings and their
 427 relation to the child care food and physical activity environment.

- 428
 429 7. Conduct new research to document the types and quantities of foods and beverages students
 430 consume both at school and daily outside of school, before, during, and after school-based
 431 healthy eating approaches and policies are implemented.

432 **Rationale:** Effective school-based approaches and policies to improve the availability,
 433 accessibility, and consumption of healthy foods and beverages, and reduce competition from
 434 unhealthy offerings, are central to improving the weight status and health of children and
 435 adolescents. Accurate quantification of the types and quantities of foods and beverages the
 436 students consume before, during, and after approaches and policies are implemented is
 437 fundamental to assessing effectiveness. However, many of the studies included in the
 438 systematic reviews and meta-analyses used by the DGAC to address this issue did not
 439 comprehensively measure or report dietary information. Although the USDA/FNS-sponsored
 440 School Nutrition Dietary Assessment (SNDA) series collects student dietary intake data
 441 every 10 years, the DGAC recommends more frequent and consistent data collection,
 442 especially before and periodically after implementation of school-based nutrition and
 443 physical activity policy and program changes.

- 444
 445 8. Improve the quality of research studies designed to assess the effects of school-based
 446 approaches and policies on dietary behaviors and body weight control to reduce the risk of
 447 bias, with an emphasis on randomized controlled trials.

448 **Rationale:** Although the methodological quality of the systematic reviews and meta-analyses
 449 used by the DGAC to evaluate school-based approaches and policies on dietary intake and
 450 body weight outcomes was high, the authors of these reviews commented that the scientific
 451 quality of individual studies was generally poor and the risk of bias high. Many of the studies
 452 were done using quasi-experimental (with or without control), pre-post intervention, or cross-
 453 sectional designs. Future research should prioritize using prospective, repeated measures,
 454 randomized controlled trial experimental designs, with randomization at the individual,
 455 classroom, school, or school district level. Pilot feasibility studies also may be helpful to
 456 quickly identify promising novel approaches to improve dietary intake and weight control

457 outcomes.

458

459 9. Conduct post-program follow-up assessments lasting longer than 1 year to determine the
460 long-term retention of the changed nutrition behaviors as well as the usefulness of continuing
461 to offer the programs while children advance in school grade. Also, conduct research is
462 needed in adolescents (grades 9-12).

463 **Rationale:** Literature supports that eating and physical activity behaviors and body weight
464 status of children predict changes over time as they progress into adolescence and adulthood.
465 Ideally, improvements in dietary intake and weight status achieved due to a given school-
466 based approach or policy would be sustained over time and progressive improvements would
467 occur long-term. The vast majority of published research focuses on children in grades K-8,
468 or ages 4-12 years, and new and improved data are needed on adolescents and the transition
469 from childhood to adolescence.

470

471 10. Encourage a wider variety of school-based approaches and policies to develop and evaluate
472 innovative approaches focused on increasing vegetable intakes.

473 **Rationale:** Consumption of non-potato vegetables is below 2010 Dietary Guidelines for
474 Americans recommendations in both children and adolescents. Published research indicates
475 that school-based approaches and policies designed to increase vegetable and fruit intakes are
476 generally more effective at increasing fruit intake, except for –school gardens and economic
477 incentives, which increase vegetable intake among school-aged children. Some past public
478 policies (e.g. the Basic 4) treated fruit and vegetables and as a single food group, which props
479 the need for new research that uses prospective, repeated measures, and randomized
480 controlled trial experimental designs to specifically target increased consumption of healthy
481 vegetables.

482

483 11. Conduct assessments of the effectiveness of worksite interventions that emphasize obesity
484 prevention and weight control among workers across racially/ethnically diverse populations,
485 blue and white collar employees, and at-risk populations. Scientifically rigorous studies
486 (especially randomized controlled trials) addressing the long-term health impact of worksite-
487 based approaches and policies that improve employee diet, physical activity, and body
488 weight control would have public health relevance.

489 **Rationale:** In light of the high rates of obesity and overweight, worksite interventions
490 targeting obesity prevention and weight control through enhanced dietary behaviors and
491 increased physical activity among workers is important. The majority of the studies to date
492 have been conducted for relatively short periods of time, and the long-term impact of these
493 approaches and policies may prove beneficial.

494

495 **CHAPTER 5: FOOD SUSTAINABILITY AND SAFETY**496 **Dietary Patterns and Sustainability**

497 1. Conduct research to determine whether sustainable diets are affordable and accessible to all
 498 sectors of the population and how this can be improved, including how policy strategies
 499 could influence the supply chain (all steps from farm to plate) to affect this improvement.

500 **Rationale:** Ensuring that sustainable diets are accessible and affordable to all sectors of the
 501 population is important to promote food security.

502

503 2. Develop, conduct, and evaluate in-depth analyses of U.S. domestic dietary patterns and
 504 determine the degree to which sustainability practices, domestically and internationally, are
 505 important to food choice and how to increase public awareness of the impact of food choices
 506 on environmental outcomes.

507 **Rationale:** Understanding consumer choice across demographic groups and the degree to
 508 which either health and/or sustainability is a significant decisional criterion as well as the
 509 degree to which choice theory can be used to improve choices will be important to helping
 510 drive change.

511

512 3. Develop a robust understanding of how production practices, supply chain decisions,
 513 consumer behaviors, and waste disposal affect the environmental sustainability of various
 514 practices across the USDA food components of MyPlate.

515 **Rationale:** Developing sustainable production and supply chain practices for all parts of
 516 MyPlate, especially meat and dairy products will be important to reduce their environmental
 517 impact.

518

519 4. Determine the potential economic benefits and challenges to supply chain stakeholders in
 520 relationship to findings in Research Recommendation 3.

521 **Rationale:** Experience demonstrates that many practices over the past few decades that
 522 improve the environmental footprint of, for example, production practices, also have led to
 523 improved profit (e.g., Integrated Pest Management to reduce pesticide use in many fruit and
 524 vegetables). It is important to know how changes will affect profit to help enable future
 525 policy in both the private and public spheres.

526

527 **Seafood Sustainability**

- 528 5. Conduct research on methods to ensure the maintenance of nutrient profiles of high-trophic
529 level farmed seafood and improve nutrient profiles of low-trophic farmed seafood
530 concurrently with research to improve production efficacy.

531 **Rationale:** The evidence supporting healthfulness of seafood consumption is based on
532 consumption of predominantly wild caught species. Many popular low-trophic level farmed
533 seafood have nutrient profiles that depend on feeds. Efficient production of seafood with
534 nutrient profiles that are known to be healthful should be emphasized.

535

- 536 6. Conduct research to develop methods to ensure contaminant levels in all seafood remain at
537 levels similar to or lower than at present. Maintain monitoring of contaminant levels for
538 capture fisheries to ensure that levels caused by pollution do not rise appreciably. This
539 research should include developing effective rapid response approaches if the quality of
540 seafood supply is acutely affected.

541 **Rationale:** Current research findings support the contention that contaminant levels are
542 generally well below those that significantly alter the healthfulness of seafood. As industry
543 naturally improves efficiency, feeds and environmental conditions should be monitored to
544 maintain or reduce priority contaminants and insure significant new contaminants do not
545 enter the seafood supply.

546

547 **Usual Caffeine/Coffee Intake**

- 548 7. Evaluate the effects of coffee on health outcomes in vulnerable populations, such as women
549 who are pregnant (premature birth, low birth weight, spontaneous abortion).

550 **Rationale:** Given the limited evidence of the effects of coffee/caffeine consumption on
551 pregnancy outcomes, future studies need to establish safe levels of coffee/caffeine
552 consumption during pregnancy.

553

- 554 8. Examine the effects of coffee on sleep patterns, quality of life, and dependency and
555 addiction.

556 **Rationale:** Because coffee is a known stimulant, future research should examine the effect of
557 coffee/caffeine on sleep quality, dependency, addiction, and overall quality of life measures.

558

- 559 9. Evaluate the prospective association between coffee/caffeine consumption and cancer at
560 different sites.

561 **Rationale:** Large well-conducted prospective cohort studies that adequately control for
562 smoking (status and dosage) and other potential confounders are needed to understand the
563 association of coffee (caffeinated and decaffeinated) with cancer at different sites.

564
 565
 566
 567
 568
 569
 570
 571
 572
 573
 574
 575
 576
 577
 578
 579
 580
 581
 582
 583
 584
 585
 586
 587
 588
 589
 590
 591
 592
 593
 594
 595
 596
 597
 598
 599
 600

10. Examine prospectively the effects of coffee/caffeine on cognitive decline, neurodegenerative diseases, and depression.

Rationale: Neurodegenerative diseases affect millions of people worldwide and more than five million Americans are living with Alzheimer’s disease. Given the limited evidence of coffee/caffeine on neurodegenerative diseases, well-designed prospective studies should examine the association of coffee/caffeine consumption on cognitive decline, depression, and Alzheimer’s disease.

11. Understand the mechanisms underlying the protective effects of coffee on diabetes and CVD.

Rationale: Evidence for a biological plausibility for coffee on risk of type 2 diabetes and CVD stems primarily from animal studies. Randomized controlled trials in humans should evaluate the effect of coffee/caffeine on measures of glycemia, insulin sensitivity, endothelial dysfunction, and inflammation.

12. Understand the association between coffee and health outcomes in individuals with existing CVD, diabetes, cancer, neurodegenerative diseases, or depressive symptoms.

Rationale: Strong evidence supports a protective effect of moderate coffee consumption on chronic disease risk in healthy adults, but its association among those with existing diseases has been less studied. Given that a substantial number of people suffer from these chronic diseases, the role of coffee in preventing other health outcomes in such groups remains understudied.

High-dose Caffeine Intake

13. Define excessive caffeine intake and safe levels of consumption for children, adolescents, and young adults.

Rationale: Current research on caffeine and health outcomes has focused primarily on adults. Given the increasing prevalence of energy drink consumption among children, adolescents, and young adults, research is needed to identify safe levels of consumption in these groups.

14. Determine the prevalence of excessive caffeine intake in children and adults beyond intake of energy drinks.

Rationale: Data on the sources (other than energy drinks) and doses of caffeine intake in children and adults are limited. Identifying the sources and safe levels of consumption will help in formulating policy and framing recommendations.

601 15. Examine the effect of excessive consumption of caffeine and energy drinks on health
602 outcomes in both children and adults.

603 **Rationale:** Prospective studies of associations of excessive caffeine and energy drink intake
604 with health outcomes in children and adults are necessary, as randomized controlled trials are
605 not be feasible given ethical constraints.

606

607 16. Conduct observational studies to examine the health effects of alcohol mixed with energy
608 drinks.

609 **Rationale:** In recent years, consumption of alcohol energy drinks by adolescents has resulted
610 in emergency room admissions and deaths. No data exist on the prospective association
611 between consumption of alcohol energy drinks and health outcomes in both adolescents and
612 adults.

613

614 **Aspartame**

615 17. Examine the risks of aspartame related to some cancers, especially hematopoietic ones, and
616 pregnancy outcomes.

617 **Rationale:** Limited and inconsistent evidence suggests a possible association between
618 aspartame and risk of hematopoietic cancers (non-Hodgkin lymphoma and multiple
619 myeloma) in men, indicating the need for long-term human studies. Additionally, limited and
620 inconsistent evidence indicates a potential for risk of preterm delivery, which warrants
621 further research.

622

623

624 **CHAPTER 6: CROSS-CUTTING TOPICS OF PUBLIC HEALTH** 625 **IMPORTANCE**

626 1. Design and conduct studies with sufficient power to define the impact of improving dietary
627 quality, including the lowering dietary sodium intake, on hypertension and relevant disease
628 outcomes, including cardiovascular disease, stroke, peripheral vascular disease, kidney
629 disease, and others. The interactions with patterns of therapeutic medication use (e.g.,
630 diuretics, antihypertensives, and lipid-lowering) should be considered.

631 **Rationale:** The current literature is incomplete, limited in power and durations, and often
632 compromised by methodological challenges that must be addressed in well-designed studies
633 with relevant clinical outcomes.

634

635 2. Assess the accuracy of 24-hour urine collections for sodium assessment in populations with
636 different health conditions (e.g., diabetes, chronic kidney disease, heart failure,
637 cardiovascular disease) and interactions with different patterns of medication use (e.g.,
638 diuretics, antihypertensives).

639 **Rationale:** If there is systematic error in sodium assessment because individuals with various
 640 co-morbidities who are taking medications systematically do not provide accurate urine
 641 collections, paradoxical findings between sodium and health outcomes may be observed.

642

643 3. Examine the effect of behavioral interventions, with novel approaches (e.g., flavorful recipes,
 644 cooking techniques) on adherence to dietary sodium recommendations.

645 **Rationale:** For decades, the population has exceeded dietary sodium intake
 646 recommendations. A public health approach that results in reformulation of commercially
 647 processed foods to lower sodium content should be the primary strategy for decreasing
 648 sodium intake in the U.S. population. However, individual support for public health policies
 649 will be needed to further document demand for changes in the sodium food environment. To
 650 this end, interventions that modify individual knowledge, attitudes, and behaviors around
 651 sodium intake should be evaluated.

652

653 4. Examine the effect of low sodium intake on taste preferences for sodium and healthy dietary
 654 patterns.

655 **Rationale:** It has been argued that populations desire higher levels of sodium intake and will
 656 inevitably revert to higher levels of sodium intakes after acute reductions in sodium intake. It
 657 has also been argued that after six weeks of reduced sodium intake, taste preferences are
 658 modified such that higher sodium is no longer desirable. Studies are needed to elucidate the
 659 effects of lowering sodium intake on diet preferences.

660

661 5. Document the relationship between portion size and sodium intake.

662 **Rationale:** These data are needed to inform whether dietary recommendations for sodium
 663 should be adjusted for caloric intake. It is known that the absolute amount of sodium intake is
 664 highly correlated with caloric intake. As a result, the absolute recommended amount of
 665 sodium is harder to achieve for a larger, high energy consuming person than for a smaller,
 666 low energy consuming person. The science to inform whether sodium density confers
 667 different risk than absolute intake of sodium is limited because of methodologic limitations
 668 in surveys where both calories and sodium intake can be calculated. Furthermore, the
 669 existing correlation between sodium and calories may be an artifact of the current food
 670 supply.

671

672 6. Determine the effects of replacement of saturated fat with different types of carbohydrates
 673 (e.g., refined vs. whole grains) on cardiovascular disease risk.

674 **Rationale:** Most randomized controlled trials and prospective cohort studies compared
 675 saturated fat with total carbohydrates. It is important to distinguish different types of
 676 carbohydrates (e.g. refined vs. whole grains) in future studies.

677

- 678 7. Examine the effects replacement of saturated fat with polyunsaturated fat vs.
679 monounsaturated fat on cardiovascular disease risk.

680 **Rationale:** Most existing studies have examined the effects of substituting PUFA for
681 saturated fat on cardiovascular disease risk. Future studies should also examine the potential
682 benefits of substituting monounsaturated from plant sources such as olive oil and nuts/seeds
683 for saturated fat on cardiovascular disease risk.

- 684
685 8. Examine lipid and metabolic effects of specific oils modified to have different fatty acid
686 profiles (e.g. commodity soy oil (high linoleic acid) vs. high oleic soy oil).

687 **Rationale:** As more modified vegetables oils become commercially available, it is important
688 to assess their long-term health effects. In addition, future studies should examine lipid and
689 metabolic effects of plant oils that contain a mix of *n-9*, *n-6*, and *n-3* fatty acids, as a
690 replacement for animal fat, on cardiovascular disease risk factors.

- 691
692 9. Examine the effects of saturated fat from different sources, including animal products (e.g.
693 butter, lard), plant (e.g., palm vs. coconut oils), and production systems (e.g. refined
694 deodorized bleached vs. virgin coconut oil) on blood lipids and cardiovascular disease risk.

695 **Rationale:** Different sources of saturated fat contain different fatty acid profiles and thus,
696 may result in different lipid and metabolic effects. In addition, virgin and refined coconut oils
697 have different effects in animal models, but human data are lacking.

- 698
699 10. Conduct gene-nutrient interaction studies by measuring genetic variations in relevant genes
700 that will enable evaluation of effects of specific diets for individualized nutrition
701 recommendations.

702 **Rationale:** Individuals with different genetic background may respond to the same dietary
703 intervention differently in terms of blood lipids and other cardiovascular disease risk factors.
704 Future studies should explore the potential role of genetic factors in modulating the effects of
705 fat type modification on health outcomes.

- 706
707 11. Identify sources and names of added sugars and low-calorie sweeteners used in the food
708 supply and quantify their consumption levels and trends in the U.S. diet.

709 **Rationale:** It is unclear whether all food and nutrient databases capture all added sugars
710 because: 1) added sugars have varied and inconsistent nomenclature and may not be
711 recognized as added sugars in nutrient analyses; and 2) many foods with added sugars have
712 formulations considered proprietary by the manufacturers and for this reason actual added
713 sugars content is difficult to obtain. Accurate assessment of added sugars in the U.S. diet is
714 needed to quantify the population level exposure and subsequent health risks from added
715 sugars. The lack of information on the various added sugars in the food supply hinders efforts
716 to make policy about consumption.

717

718 12. Conduct prospective research with strong experimental designs and multiple measurements
719 of the consumption of added sugars and low-calorie sweeteners on health outcomes, such as
720 body weight, adiposity, and clinical markers of type 2 diabetes and cardiovascular disease.

721 **Rationale:** High heterogeneity exists among published research with regard to the types and
722 forms of added sugars and low-calorie sweeteners-containing foods/beverages used for
723 interventions, which precludes assessing the effects of specific added sugars and low-calorie
724 sweeteners on body weight, adiposity, and cardio-metabolic health in adults and children.
725 Many studies use single baseline measurements of diet to reflect usual patterns and quantities
726 of intake over time. New research should emphasize assessments within the context of usual
727 dietary intakes and patterns of food and beverage consumption in free-living populations,
728 along with specific added sugars and low-calorie sweeteners, especially those that are
729 currently understudied. Large prospective studies with repeated measurements of low-calorie
730 sweeteners are needed to monitor their long-term effects on cancer and other health
731 outcomes.

732

733 13. Design studies that emphasize assessments of relationships between the intakes of added
734 sugars and low-calorie sweeteners and body weight, adiposity, and cardio-metabolic health in
735 diverse sub-populations who are at high risk of obesity and related morbidities.

736 **Rationale:** Insufficient evidence exists to assess the impact of added sugars and low-calorie
737 sweeteners contained in foods and beverages on individuals from diverse populations who
738 have high risk for adverse health outcomes. These include (but are not limited to) different
739 race/ethnicity groups; low income groups, especially those with food insecurity; groups who
740 live in specific geographic locations with high prevalence of obesity (e.g. inner city, rural,
741 and Southern regions of the United States); and age and sex groups (women, children, and
742 elderly adults).

743

744 14. Assess and improve approaches and policies to reduce the amount of added sugars in the
745 food and beverage supply as well as in school and community settings.

746 **Rationale:** Results from this research would assist policy makers and the private sector in
747 establishing sustainable approaches and policies to limit the availability and consumption of
748 added sugars. These approaches and policies would also be important for multi-component
749 strategies to improve weight control and health among people living in the United States.

750

751 15. Conduct consumer research to identify and test elements of a standardized, easily understood
752 front-of-package label.

753 **Rationale:** Research is needed to provide an evidence base to support the need and identify
754 critical elements of a front of package label. This is particularly important to support the
755 Food and Drug Administration in implementing a front-of-package labeling system.

756 **CHAPTER 7: PHYSICAL ACTIVITY**

757 1. Evaluate best practices in programming at the community and national level and identify
 758 which local and national policies in the public and private sector have demonstrated the
 759 greatest effect on increasing physical activity participation across the lifespan, especially in
 760 populations with the greatest health disparities.

761 **Rationale:** Physical activity participation rates are exceptionally low across all age groups,
 762 and are especially low in individuals with the greatest health disparities. Many different
 763 initiatives are currently underway in the private and public sector to help increase physical
 764 activity on a population level. Understanding which programs and policies are having the
 765 greatest impact will help focus valuable resources and national recommendations for
 766 maximum public health benefit.

767
 768 2. Identify the dose of physical activity needed to achieve health benefits, as well as appropriate
 769 growth and development, for children younger than age 6 years.

770 **Rationale:** Until recently, very little effort has been focused on understanding the health
 771 benefits of physical activity for young children. Given that this is a critical age of growth and
 772 development, considerable research should be focused on this age group.

773
 774 3. Evaluate the effects of various modes and doses of physical activity on health outcomes in
 775 older adults.

776 **Rationale:** Older adults are the fastest growing segment of the population. They also have
 777 the greatest burden of disease and functional (mental and physical) limitations. To reduce
 778 burden of disease and related economic impacts, research regarding mode and dose of
 779 physical activity should be focused on this age group.

780
 781 4. Further evaluate the importance of light activity, short bouts of physical activity (i.e., 10-
 782 minutes or less) and modes of activity on health outcomes across the lifespan.

783 **Rationale:** The review of the evidence in the 2008 PAGAC Report focused primarily on
 784 moderate- and vigorous-intensity activity. Emerging research highlights the positive effects
 785 of light activity as well as shorter bouts of vigorous activity on health outcomes.
 786 Understanding the health impact of the full range of mode, intensity, duration, frequency, and
 787 setting will help to further refine the PAG to support maximum public health benefit.

788
 789 5. Further investigate the effects of sedentary behaviors on health outcomes, including duration,
 790 frequency, and mode of sedentary activities.

791 **Rationale:** Increasing evidence demonstrates the negative health consequences of sedentary
 792 behaviors. Clarity on the types and duration of sedentary behaviors that have the most
 793 negative health impact would help to identify meaningful evidence-based public health
 794 recommendations.

Appendix E-2: Supplementary Documentation to the 2015 DGAC Report

The 2015 DGAC used a variety of scientifically rigorous approaches to address its science-based questions. These approaches are described in *Part C. Methodology*. Slightly more than one-third of the questions were answered using a state-of-the-art systematic review process, and these reviews are publically available in the Nutrition Evidence Library (NEL) at www.NEL.gov.

The remaining questions were answered using existing sources of evidence (including systematic reviews, meta-analyses, or reports), data analyses, and food pattern modeling analyses. These three approaches allowed the Committee to ask and answer its questions in a systematic, transparent, and evidence-based way.

Appendix E-2 provides a list of supplementary documentation related to the existing sources of evidence and data analyses used by the Committee in evidence reviews (see *Appendix E-3 for USDA Food Patterns for Special Analyses*). These sources are publically available online through active links within this document at www.DietaryGuidelines.gov.

CHAPTER 1: FOOD AND NUTRIENT INTAKES AND HEALTH: CURRENT STATUS AND TRENDS

NUTRIENTS OF CONCERN

- Appendix E-2.1 [Usual intake distributions, 2007-2010, by age/gender groups](#)
- Appendix E-2.2 [Usual intake distributions as a percent of energy for fatty acids and macronutrients, 2007-2010, by age/gender groups](#)
- Appendix E-2.3 [Usual intake distributions for individuals age 71 and older, 2007-2010](#)
- Appendix E-2.4 [Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in the U.S. ages 19-50 years](#)
- Appendix E-2.5 Usual intake distributions for [supplement](#) users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/gender groups
- Appendix E-2.6 Usual intake distributions for [non-supplement](#) users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/gender groups

FOOD CATEGORIES

- Appendix E-2.7 [Major categories and subcategories used in DGAC analyses of WWEIA Food Categories](#)
- Appendix E-2.8 [Percent of total food group intake, 2009-2010, for U.S. population ages 2 years and](#)

- [older, from WWEIA Food Categories](#)
- Appendix E-2.9 [Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories](#)
- Appendix E-2.10 [Percent of total energy intake, 2009-2010, for age/sex groups of the U.S. population from WWEIA Food Categories](#)
- Appendix E-2.11 [Percent of total energy intake, 2009-2010, for racial/ethnic groups of the U.S. population, from WWEIA Food Categories](#)
- Appendix E-2.12 [Percent of total energy intake, 2009-2010, for age/income groups of the U.S. population, from WWEIA Food Categories](#)

EATING BEHAVIORS

- Appendix E-2.13 [Percent of energy intake from major points of purchase and location of eating, 2003-04, 2005-06, 2007-08, and 2009-10, for the U.S. population ages 2 years and older](#)
- Appendix E-2.14 [Food group and nutrient content of foods per 1000 calories obtained from major points of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010 for the U.S. population ages 2 years and older](#)
- Appendix E-2.15 [Amount of key nutrients and food groups by age group per 1000 calories from each major point of purchase, 2003-04, 2005-06, 2007-08, and 2009-10](#)

HEALTH CONDITIONS

- Appendix E-2.16 [Body mass index, adults ages 20 years and older, NHANES 2009-2012](#)
- Appendix E-2.17 [Body mass index, children and adolescents ages 2-19 years, NHANES 2009 -2012](#)
- Appendix E-2.18 [Total cholesterol and high density lipoprotein cholesterol \(HDL\), adults ages 20 years and older, NHANES 2009-2012](#)
- Appendix E-2.19 [Low density lipoprotein cholesterol \(LDL-C\) and triglycerides, adults ages 20 years and older, NHANES 2009-2012](#)
- Appendix E-2.20 [Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012](#)
- Appendix E-2.21 [Total diabetes, adults ages 20 years and older, NHANES 2009 -2012](#)
- Appendix E-2.22 [Total cholesterol, high density lipoprotein cholesterol \(HDL\), and non-HDL-cholesterol, children and adolescents ages 6-19 years, NHANES 2009-2012](#)
- Appendix E-2.23 [Low density lipoprotein cholesterol \(LDL-C\) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012](#)

Appendix E-2.24 [Prevalence of high and borderline high blood pressure \(BP\), children and adolescents ages 8-17 years, NHANES 2009-2012](#)

DIETARY PATTERNS

Appendix E-2.25 [Average Healthy Eating Index-2010 scores for Americans ages 2 years and older \(National Health and Nutrition Examination Survey 2009-2010\)](#)

20
21

22 **CHAPTER 2: DIETARY PATTERNS, FOODS AND NUTRIENTS, AND**
23 **HEALTH OUTCOMES**

DIETARY PATTERNS AND RISK OF CARDIOVASCULAR DISEASE

Appendix E-2.26 Evidence Portfolio

DIETARY PATTERNS AND MEASURES OF BODY WEIGHT

Appendix E-2.27 Evidence Portfolio

DIETARY PATTERNS AND RISK OF TYPE 2 DIABETES

Appendix E-2.28 Evidence Portfolio

24
25
26

CHAPTER 4: FOOD ENVIRONMENT AND SETTINGS

SCHOOL-BASED APPROACHES AND DIETARY INTAKE

Appendix E-2.29a Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED POLICIES AND DIETARY INTAKE

Appendix E-2.30 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED APPROACHES AND WEIGHT STATUS

Appendix E-2.31 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED POLICIES AND WEIGHT STATUS

Appendix E-2.32 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

WORKSITE-BASED APPROACHES AND DIETARY INTAKE

Appendix E-2.33a Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED POLICIES AND DIETARY INTAKE

Appendix E-2.34 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED APPROACHES AND WEIGHT STATUS

Appendix E-2.35 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED POLICIES AND WEIGHT STATUS

Appendix E-2.36 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

27

28

29

CHAPTER 5: FOOD SUSTAINABILITY AND SAFETY

DIETARY PATTERNS AND FOOD SUSTAINABILITY

Appendix E-2.37 Evidence Portfolio

SEAFOOD AND SUSTAINABILITY

Appendix E-2.38 Evidence Portfolio

USUAL CAFFEINE CONSUMPTION AND HEALTH

- Appendix E-2.39a Evidence Portfolio
- Appendix E-2.39b Systematic Review/Meta-Analysis Data Table

HIGH-DOSE CAFFEINE CONSUMPTION AND HEALTH

- Appendix E-2.40 Evidence Portfolio

ASPARTAME CONSUMPTION AND HEALTH

- Appendix E-2.41 Evidence Portfolio

30
31
32
33

CHAPTER 6: CROSS-CUTTING TOPICS OF PUBLIC HEALTH IMPORTANCE

SODIUM AND BLOOD PRESSURE IN ADULTS

- Appendix E-2.42 Evidence Portfolio

SATURATED FAT AND RISK OF CARDIOVASCULAR DISEASE

- Appendix E-2.43 Evidence Portfolio

ADDED SUGARS AND LOW-CALORIE SWEETENERS

- Appendix E-2.44 Evidence Portfolio – Added Sugars and Measures of Body Weight
- Appendix E-2.45 Evidence Portfolio – Added Sugars and Risk of Type 2 Diabetes
- Appendix E-2.46 Evidence Portfolio – Added Sugars and Dental Caries
- Appendix E-2.47 Evidence Portfolio – Low-Calorie Sweeteners and Measures of Body Weight
- Appendix E-2.48 Evidence Portfolio – Low-Calorie Sweeteners and Risk of Type 2 Diabetes

34
35
36

CHAPTER 7: PHYSICAL ACTIVITY

PHYSICAL ACTIVITY

- Appendix E-2.49 Existing Report Data Table

37
38

Appendix E-3: USDA Food Patterns for Special Analyses

The 2015 DGAC identified specific questions that they felt could best be addressed through a food pattern modeling approach, using the USDA Food Patterns and the modeling process developed to address similar requests by the 2005 and 2010 DGACs. The approach used for the 2015 DGAC food pattern modeling questions is described in *Part C: Methodology*.

Seven modeling analyses requested by the Committee were completed by staff working closely with Subcommittee 1 members, and provided as reports for the full Committee to consider. The food pattern modeling analyses conducted for the 2015 DGAC are listed below. Full reports for each analysis are available online through active links within this document at www.DietaryGuidelines.gov.

E-3.1 Adequacy of USDA Food Patterns

How well do updated USDA food intake patterns meet IOM Dietary Reference Intakes and 2010 Dietary Guidelines nutrient recommendations? How do the recommended amounts of food groups compare to current distributions of usual intakes for the American population?

E-3.2 Food Group Contributions to Nutrients in USDA Food Patterns and Current Nutrient Intakes

What is the contribution of whole grain foods and fruits and vegetables to (1) total fiber intake and (2) total nutrient intake in the USDA Food Patterns? What is the contribution of fruits and vegetables to current nutrient intake (focus on nutrients of concern, including fiber)?

E-3.3 Meeting Vitamin D Recommended Intakes in USDA Food Patterns

Can vitamin D EARs and/or RDAs be met with careful food choices following recommended amounts from each food group in the USDA Food Patterns? How restricted would food choices be, and how much of the vitamin D would need to come from fortified food products?

E-3.4 USDA Food Patterns—Adequacy for Young Children

How well do the USDA Food Patterns meet the nutritional needs of children 2 to 5 years of age and how do the recommended amounts compare to their current intakes? Given the relatively small empty calorie limit for this age group, how much flexibility is possible in food choices?

E-3.5 Reducing Saturated Fats in the USDA Food Patterns

What would be the effect on food choices and overall nutrient adequacy of limiting saturated fatty acids to 6 percent of total calories by substituting mono- and polyunsaturated fatty acids?

E-3.6 Dairy Group and Alternatives

What would be the impact on the adequacy of the patterns if (1) no Dairy foods were consumed, (2) if calcium was obtained from nondairy sources (including fortified foods), and (3) if the proportions of milk and yogurt to cheese were modified?

What is the relationship between changes in types of beverages consumed (milk compared with sugar-sweetened beverages) and diet quality?

E-3.7 Developing Vegetarian and Mediterranean-style Food Patterns

Using the Food Pattern Modeling process, can healthy eating patterns for vegetarians and for those who want to follow a Mediterranean-style diet be developed? How do these patterns differ from the USDA Food Patterns previously updated for the 2015 DGAs?

13

14

Appendix E-4: NHANES Data Used in DGAC Data Analyses

Most of the DGAC data analyses used the National Health and Nutrition Examination (NHANES) data and its dietary component, What We Eat in America (WWEIA), NHANES (Zipf et al., 2013). These data were used to answer questions about food and nutrient intakes because they provide national and group level estimates of dietary intakes of the U.S. population on a given day as well as usual intake distributions. These data contributed substantially to questions answered using data analyses. This appendix describes the NHANES data in greater detail.

NHANES

NHANES consists of ongoing, comprehensive, cross-sectional, population-based surveys designed to collect data on health, nutritional status, and health behaviors of the non-institutionalized civilian population living in households in the United States. It is conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC). NHANES has had a long history starting in the early 1960s (Zipf et al., 2013); it has been monitoring food and nutrient intake and nutritional status of the U.S. population since 1971, starting with NHANES I. Since then, several cycles of NHANES have been conducted as a series of cross-sectional surveys focusing on different population groups in terms of age and race/ethnicity, or health topics. In 1999, NHANES became a continuous survey, sampling U.S. residents of all ages, with a changing focus on a variety of health and nutrition measurements to meet emerging needs. The goals of the continuous NHANES are to provide prevalence data on selected diseases and risk factors for the U.S. population; to monitor trends in selected diseases, behaviors, and environmental exposures; to explore emerging public health needs; and to maintain a national probability sample of baseline information on health and nutritional status of the U.S. household population (Zipf et al., 2013).

NHANES has a complex, multi-stage, probability sampling design and examines a nationally representative sample of about 5,000 persons each year. In NHANES, certain subgroups have been periodically oversampled. These include low income, older Americans, infants and children, pregnant women and certain race/ethnic groups (e.g., Hispanics, including Mexican Americans, African Americans, and more recently, Asian Americans). The NHANES survey is unique because it combines personal interviews with standardized physical examinations and laboratory tests administered by a specially trained staff that travels with the Mobile Examination Center (MEC) to survey sites selected to represent the U.S. population (Zipf et al., 2013).

38 In the continuous NHANES, dietary intake is assessed through two 24-hr recalls, administered
39 by trained dietary interviewers using the USDA's Automated Multiple Pass Method (AMPM)
40 (Blanton et al., 2006) through What We Eat in America (WWEIA). The first 24-hr recall (day 1)
41 is collected in-person at the MEC and a subsequent 24-hr recall (day 2) is obtained 7 to 10 days
42 later over the telephone. Information on dietary supplements consumed during the 24-hour recall
43 period is also collected. The strengths of the WWEIA, NHANES dietary data include that
44 because two 24-hour recalls are available in WWEIA, NHANES (from 2003 onwards), usual
45 intake distributions can be estimated based on statistical techniques that reduce the effect of
46 intra-individual variation in food and nutrient intakes in 24-hour recalls (Nusser et al. 1996;
47 Tooze et al. 2006; Dodd et al. 2006).

48
49 The WWEIA, NHANES dietary data are one of the few sources that can provide national
50 estimates of total nutrient intake from diet and dietary supplements for the U.S. population.
51 Moreover, dietary intakes can be described by specific socio-demographic groups including
52 race/ethnic groups, income status, and participation in Federal nutrition assistance programs
53 (e.g., Supplemental Nutrition Assistance Program). Dietary data from WWEIA, NHANES can
54 be linked to thorough anthropometric, laboratory, and clinical evaluation data as well health
55 outcomes to examine cross-sectional associations at the national and large subgroup levels. It
56 must be recognized that WWEIA, NHANES dietary data are not designed for individual-level
57 assessment. These data can be useful to inform nutrition policy, but not sufficient by themselves
58 to form policy recommendations.

59
60 No single perfect method for assessing dietary intake information is available in surveys (Willett
61 1998; Gibson 2005; Berdainer et al., 2008) and different methods may be indicated for specific
62 purposes (Willett 1998; Beaton et al., 1983; Berdainer et al., 2008). NCHS has been actively
63 involved in researching and reviewing its data collection methods, including dietary data, over
64 the years internally and in consultation with expert groups (Wright et al., 1994; Briefel &
65 Sempos, 1992). The methods used in NHANES are adapted in light of its large sample size and
66 complex design, cost and feasibility, and respondent burden to ensure a high response rate to
67 derive nationally representative estimates. Some examples of adaptations in methods include the
68 transition to USDA's standardized automated multi-pass method for collection of dietary recalls
69 by trained interviewers that has been evaluated and associated with reduced measurement error
70 (Moshfegh et al., 2008). Other examples include collection of an additional 24-hour dietary
71 recall in NHANES since 2003 (for a total of two 24-hour recalls), coupled with targeted food
72 frequency questionnaires over various NHANES cycles.

73
74 The strengths and shortcomings of these dietary assessment methods have been discussed over
75 time in various meetings (e.g., International Conference on Diet and Activity Methods and
76 American Society for Nutrition/Experimental Biology), workshops, and expert groups. This has
77 also been discussed for several years in the scientific literature (Beaton 1994; Berdainer et al.,

78 2008) and in recent articles (Archer et al., 2013; Hébert et al., 2014; Webb, 2013). No
 79 assessment method is perfect and the choice of dietary method is based on the purpose for which
 80 it is intended. For NHANES, repeated 24-hour recalls remain the backbone of dietary assessment
 81 and monitoring. These data are useful in providing national- and group-level estimates of dietary
 82 intakes of the U.S. population, on a given day as well as in describing usual intake distributions
 83 using appropriate statistical approaches, to inform nutrition policy.

84

85

86 **REFERENCES**

87 Archer E, Hand GA, Blair SN. Validity of U.S. Nutritional Surveillance: National Health and
 88 Nutrition Examination Survey Caloric Energy Intake Data, 1971-2010. PloS one
 89 2013;8(10):e76632.

90 Beaton GH, Milner J, McGuire V, Feather TE, Little JA. Source of variance in 24-hour dietary
 91 recall data: implications for nutrition study design and interpretation. Carbohydrate sources,
 92 vitamins, and minerals. Am J Clin Nutr. 1983;37(6):986-95.

93 Beaton GH. Approaches to analysis of dietary data: relationship between planned analyses and
 94 choice of methodology. Am J Clin Nutr 1994; 59(1Suppl):253S-61S.

95 Berdainer CD, Dwyer J, Feldman EB. Handbook of Nutrition and Food. New York, NY: CRC
 96 Press, 2008

97 Blanton CA, Moshfegh AJ, Baer DJ, Kretsch MJ. The USDA Automated Multiple-Pass Method
 98 accurately estimates group total energy and nutrient intake. J Nutr 2006;136(10):2594-9.

99 Briefel RR, Sempos CT, eds. Dietary methodology workshop for the third National Health and
 100 Nutrition Examination Survey. National Center for Health Statistics. Vital and Health Stat 4(27)
 101 March 1992.

102 Dodd KW, Guenther PM, Freedman LS, Subar AF, Kipnis V, Midthune D, Tooze JA, Krebs-
 103 Smith SM. Statistical methods for estimating usual intake of nutrients and foods: a review of
 104 the theory. J Am Diet Assoc. 2006;106:1640-50.

105 Hébert JR, Hurley TG, Steck SE, Miller DR, Tabung FK, Peterson KE, Kushi LH, Frongillo EA.
 106 Considering the value of dietary assessment data in informing nutrition-related health policy.
 107 Adv Nutr. 2014;5(4):447-55.

108 Gibson R. Principles of Nutritional Assessment. 2nd ed. Oxford: Oxford University Press, 2005.

109 Moshfegh AJ, Rhodes DG, Baer DJ, et al. The US Department of Agriculture Automated
 110 Multiple-Pass Method reduces bias in the collection of energy intakes. *Am J Clin Nutr*
 111 2008;88(2):324-32.

112 Nusser, S. M., Carriquiry, A. L., Dodd, K. W., and Fuller, W. A. (1996). A semiparametric
 113 transformation approach to estimating usual daily intake distributions. *J Am Stat Assoc*
 114 1996;91:1440-49.

115 Toozé JA, Midthune D, Dodd KW, Freedman LS, Krebs-Smith SM, Subar AF, Guenther PM,
 116 Carroll RJ, Kipnis V. A new statistical method for estimating the usual intake of episodically
 117 consumed foods with application to their distribution. *J Am Diet Assoc.* 2006;106:1575-87.

118 Webb D, Leahy MM, Milner JA, Allison DB, Dodd KW, Gaine PC, Matthews RA, Schneeman
 119 BO, Tucker KL, Young SS. Strategies to optimize the impact of nutritional surveys and
 120 epidemiological studies. *Adv Nutr.* 2013 Sep 1;4(5):545-7.

121 Willett W. *Nutritional Epidemiology*. 2nd Edition ed. New York, NY: Oxford University Press,
 122 1998.

123 Wright JD, Ervin B, Briefel RR, eds. *Consensus Workshop on Dietary Assessment: Nutrition*
 124 *Monitoring and Tracking the Year 2000 Objectives*. National Center for Health Statistics.
 125 December 1994.

126 Zipf G, Chiaapa M, Porter K, Ostchega Y, Lewis B, Dostal J. *The National Health and Nutrition*
 127 *Examination Survey: Plan and Operations, 1999-2010*. *Vital Health Stat, 1;56*: National Center
 128 for Health Statistics, 2013

129

130

131

132

133

134

Appendix E-5: Glossary of Terms

Aquaculture—The farming of aquatic organisms, including fish, mollusks, crustaceans, and aquatic plants. Farming includes activities to enhance production, such as regular stocking, feeding, and protection from predators.

Acculturation—The process by which immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a new culture. Acculturation is the gradual exchange between immigrants' original attitudes and behavior and those of the host culture.

Added sugars—Sugars that are either added during the processing of foods, or are packaged as such. They include sugars (free, mono- and disaccharides), syrups, naturally occurring sugars that are isolated from a whole food and concentrated so that sugar is the primary component (e.g., fruit juice concentrates), and other caloric sweeteners. Names for added sugars include: Brown sugar, corn sweetener, corn syrup, dextrose, fructose, fruit juice concentrates, glucose, high-fructose corn syrup, honey, invert sugar, lactose, maltose, malt sugar, molasses, raw sugar, turbinado sugar, trehalose, and sucrose.

Behavioral weight-management program—A structured, multi-component program that encompasses a number of behavior changes, including diet and physical activity with the intent to improve weight (lose weight or maintain weight loss).

Biodiversity—The variety and variability among living organisms and the ecosystems in which they occur. Biodiversity includes the numbers of different items and their relative frequencies; these items are organized at many levels, ranging from complete ecosystems to the biochemical structures that are the molecular basis of heredity. Thus, biodiversity expresses the relative abundance of different ecosystems, species, and genes.

Body mass index (BMI)—A measure defining weight in kilograms (kg) divided by height in meters (m) squared. BMI is an indicator of deficient or excess body tissue, both fat and muscle. BMI status categories include underweight, normal weight, overweight, and obese. (Normal weight is often referred to as “healthy” weight.) Overweight and obese describe ranges of weight that are greater than what is considered healthy for a given height, while underweight describes a weight that is lower than what is considered healthy. Because children and adolescents are growing, their BMI is plotted on growth charts for sex and age. The percentile indicates the relative position of the child's BMI among children of the same sex and age. This is generally referred to as a **BMI z-score**.

	Children and Adolescents (ages 2 to 19 years)	Adults
Underweight	Less than the 5th percentile	Less than 18.5 kg/m ²
Normal weight	5th percentile to less than the 85th percentile	18.5 to 24.9 kg/m ²
Overweight	85th to less than the 95th percentile	25.0 to 29.9 kg/m ²
Obese	Equal to or greater than the 95 th percentile	
Obese class I		30.0 to 34.9 kg/m ²
Obese class II		35.0 to 39.9 kg/m ²
Obese class III		40.0 kg/m ² and greater

34

35 **Built environment**—The physical form of communities, including urban design (i.e., how a city
 36 is designed; its physical appearance and arrangement), land use patterns (i.e., how land is used
 37 for commercial, residential, and other activities), and the transportation system (i.e., the facilities
 38 and services that link one location to another).

39 **Calorie**—A unit commonly used to measure energy content or energy use. It is used as a
 40 convenient measure to relate the energy content of food to the energy needs of the body. A
 41 calorie is equal to the amount of energy required to raise the temperature of one liter of water 1
 42 degree centigrade. Energy, as measured in calories, is required to sustain the body's various
 43 functions, including metabolic processes and physical activity. Carbohydrate, fat, protein, and
 44 alcohol provide all of the energy supplied by foods and beverages.

45 **Carbohydrates**—One of the three classes of macronutrients. Carbohydrates include sugars,
 46 starches, and fibers:

47 • **Sugars**—A simple carbohydrate composed of one unit (a monosaccharide, such as
 48 glucose and fructose) or two joined units (a disaccharide, such as lactose and sucrose).
 49 Sugars include white and brown sugar, fruit sugar, corn syrup, molasses, and honey. (See
 50 **Added sugars**)

51 • **Starches**—Many glucose units linked together. Examples of foods containing starch
 52 include vegetables, dry beans and peas, and grains (e.g., brown rice, oats, wheat, barley,
 53 corn).

54 • **Fiber**—Nondigestible carbohydrates and lignin that are intrinsic and intact in plants.
 55 Fiber consists of dietary fiber, the fiber naturally occurring in foods, and functional fiber,
 56 which are isolated, nondigestible carbohydrates that have beneficial physiological effects
 57 in humans.

58 **Child-care settings**—Locations that include child-care centers and child-care provided in
 59 homes. Early childhood education settings, such as preschool and Head Start programs, also are
 60 included.

61 **Competitive foods**—Foods and beverages offered at schools that are sold or offered outside of
 62 the Federally reimbursed school lunch and breakfast programs. Competitive foods include food
 63 and beverage items sold through à la carte lines, snack bars, student stores, vending machines,
 64 and school fundraisers.

65 **Comprehensive lifestyle intervention**—Interventions that are designed to address chronic
 66 disease risk factors and improve health. They generally include three principal components—a
 67 diet component, a physical activity component, and a program of behavior change to facilitate
 68 adherence to diet and physical activity recommendations.

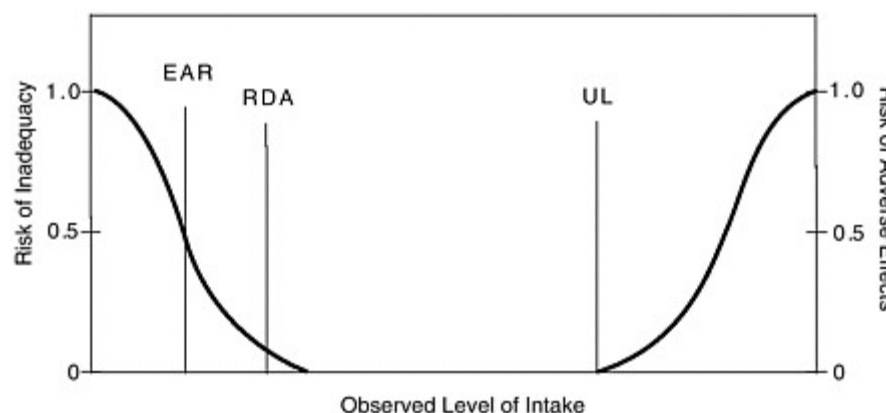
69 **Comprehensive lifestyle intervention team**—A multidisciplinary team of highly trained
 70 professionals, including registered dietitians and nutritionists, exercise specialists, and
 71 behaviorists who work with individuals on weight loss or other lifestyle behavior change to
 72 improve health and reduce chronic disease risk. (See **Interventionist**)

73 **Cross-contamination**—The spread of bacteria, viruses, or other harmful agents from one
 74 surface to another.

75 **Cup equivalent (cup eq)**—The amount of a food product that is considered equal to 1 cup from
 76 the vegetable, fruit, or milk food group. A cup eq for some foods may differ from a measured
 77 cup in volume because (1) the foods have been concentrated (such as raisins or tomato paste),
 78 (2) the foods are airy in their raw form and do not compress well into a cup (such as salad
 79 greens), or (3) the foods are measured in a different form (such as cheese).

80 **Dietary pattern**—The quantities, proportions, variety or combinations of different food and
 81 beverages in diets, and the frequency with which they are habitually consumed.

82 **Dietary Reference Intakes (DRIs)**—A set of nutrient-based reference values that expand upon
 83 and replace the former Recommended Dietary Allowances (RDAs) in the United States and the
 84 Recommended Nutrient Intakes (RNIs) in Canada. They include the values shown in the graphic
 85 (<http://www.dslid.nlm.nih.gov/dslid/dri.jsp>) and described here:



86

87

- 88 • **Acceptable Macronutrient Distribution Ranges (AMDR)**—Range of intake for a
 89 particular energy source that is associated with reduced risk of chronic disease while
 90 providing intakes of essential nutrients. If an individual’s intake is outside of the AMDR,
 91 there is a potential of increasing the risk of chronic diseases and/or insufficient intakes of
 92 essential nutrients.
- 93 • **Adequate Intakes (AI)**—A recommended average daily nutrient intake level based on
 94 observed or experimentally determined approximations or estimates of mean nutrient
 95 intake by a group (or groups) of apparently healthy people. This is used when the
 96 Recommended Dietary Allowance cannot be determined.
- 97 • **Estimated Average Requirements (EAR)**—The average daily nutrient intake level
 98 estimated to meet the requirement of half the healthy individuals in a particular life stage
 99 and sex group.
- 100 • **Recommended Dietary Allowance (RDA)**—The average dietary intake level that is
 101 sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy
 102 individuals in a particular life stage and sex group.
- 103 • **Tolerable Upper Intake Level (UL)**—The highest average daily nutrient intake level
 104 likely to pose no risk of adverse health effects for nearly all individuals in a particular life
 105 stage and gender group. As intake increases above the UL, the potential risk of adverse
 106 health effects increases.
- 107 **Eating out**—A behavior that includes meals eaten outside of the home at a variety of venues and
 108 takeout or ready-to-eat meals purchased and consumed either away from or in the home.
- 109 **Empty calories**—The calories from components of a food or beverage that contribute few or no
 110 nutrients. Major sources of empty calories are solid fats and added sugars. Other sources of
 111 empty calories include refined starches (e.g., corn starch, potato starch) and alcohol. In some
 112 foods, such as soda and many candies, all the calories are empty calories. However, empty
 113 calories also can be found in foods that contain important nutrients. For example, whole milk
 114 contains solid fats (butterfat) and sweetened applesauce contains added sugars, which means that
 115 some of their calories are empty calories.
- 116 **Energy drink**—A beverage that contains caffeine as a major active ingredient, along with other
 117 ingredients, such as taurine, herbal supplements, vitamins, and sugar. It is usually marketed as a
 118 product that can improve perceived energy, stamina, athletic performance, or concentration.
- 119 **Enrichment**—The addition of specific nutrients (iron, thiamin, riboflavin, and niacin) to refined
 120 grain products in order to replace losses of the nutrients that occur during processing.
- 121 **Environmental sustainability**—Long-term maintenance of ecosystem components and
 122 functions for future generations.

123 **Existing reports**—Previously published reports or articles that were used as sources of evidence
 124 to answer some questions posed by the 2015 DGAC. These sources included reports (e.g., *the*
 125 *2013 American College of Cardiology/ American Heart Association (ACC/AHA) Guidelines on*
 126 *Lifestyle Management to Reduce Cardiovascular Risk*), systematic reviews, and meta-analyses.
 127 (See **Meta-analysis**)

128 **Fast food**—Foods designed for ready availability, use or consumption and sold at eating
 129 establishments for quick availability or take-out. Fast food restaurants are also known as quick-
 130 service restaurants.

131 **Fats**—One of the three classes of macronutrients. (See **Solid Fats and Oils**)

132 • **Monounsaturated Fatty Acids**—Monounsaturated fatty acids (MUFAs) have one
 133 double bond. Plant sources that are rich in MUFAs include nuts and vegetable oils that
 134 are liquid at room temperature (e.g., canola oil, olive oil, high oleic safflower and
 135 sunflower oils).

136 • **Polyunsaturated fatty acids**—Polyunsaturated fatty acids (PUFAs) have two or more
 137 double bonds and may be of two types, based on the position of the first double bond.

138 ○ **n-6 PUFAs**—Linoleic acid, one of the n-6 fatty acids, is required because it cannot be
 139 synthesized by humans and, therefore, is considered essential in the diet. Primary
 140 sources are nuts and liquid vegetable oils, including soybean oil, corn oil, and
 141 safflower oil. Also called omega-6 fatty acids.

142 ○ **n-3 PUFAs**—Alpha-linolenic acid is an n-3 fatty acid that is required because it
 143 cannot be synthesized by humans and, therefore, is considered essential in the diet.
 144 Primary sources include soybean oil, canola oil, walnuts, and flaxseed.
 145 Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are very long chain
 146 n-3 fatty acids that are contained in fish and shellfish. Also called omega-3 fatty
 147 acids.

148 • **Saturated fatty acids**—Saturated fatty acids have no double bonds. Major sources
 149 include animal products such as meat and dairy products, and tropical oils such as
 150 coconut or palm oils. In general, fats high in saturated fatty acids are solid at room
 151 temperature.

152 • **trans fatty acids**—*Trans* fatty acids are unsaturated fatty acids that contain one or more
 153 isolated (i.e., nonconjugated) double bonds in a *trans* configuration. Sources of *trans*
 154 fatty acids include partially-hydrogenated vegetable oils that have been used to make
 155 traditional shortening and some commercially prepared baked goods, snack foods, fried
 156 foods, and traditional stick margarine. *Trans* fatty acids also are present in foods that
 157 come from ruminant animals (e.g., cattle and sheep) and are called “natural” or rTFA.
 158 Such foods include dairy products, beef, and lamb.

- 159 **Fight Bac!**[®]—A national public education campaign to promote food safety to consumers and
 160 educate them on how to handle and prepare food safely. In this campaign, pathogens are
 161 represented by a cartoonlike bacteria character named “BAC.” For more information, visit:
 162 <http://www.fightbac.org>.
- 163 **Fishery**—An activity leading to harvesting of fish. It may involve capture of wild fish or the
 164 raising of fish through aquaculture.
- 165 **Food access**—Accessibility to sources of healthy food, as measured by distance to a store or the
 166 number of stores in an area; individual-level resources such as family-income or vehicle
 167 availability; and neighborhood-level indicators of resources, such as average income of the
 168 neighborhood and the availability of public transportation.
- 169 **Food categories**—A method of grouping similar foods in their as-consumed forms, for
 170 descriptive purposes. The USDA/ARS has created 150 mutually exclusive food categories to
 171 account for each food or beverage item reported in What We Eat in America (WWEIA), the food
 172 intake survey component of the National Health and Nutrition Examination Survey (for more
 173 information, visit: <http://seprl.ars.usda.gov/Services/docs.htm?docid=23429>). Examples of
 174 WWEIA Food Categories include soups, nachos, and yeast breads. In contrast to food groups,
 175 items are not disaggregated into their component parts for assignment to food categories. For
 176 example, all pizzas are put into the pizza category.
 177
- 178 **Food environments**—Factors and conditions that influence food choices and food availability.
 179 These environments include settings such as home, child care (early care and education), school,
 180 after-school programs, worksites, food retail stores and restaurants, and other outlets where
 181 children and their families make eating and drinking decisions. The food environment also
 182 includes macro-level factors and includes food marketing, food production and distribution
 183 systems, agricultural policies, Federal nutrition assistance programs, and economic price
 184 structures.
- 185 **Food groups**—A method of grouping similar foods for descriptive and guidance purposes. Food
 186 groups in the USDA Food Pattern are defined as fruits, vegetables, grains, dairy, and protein
 187 foods. Some of these groups are divided into subgroups, such as dark-green vegetables or whole
 188 grains, which may have intake goals or limits (for more information, see *Appendix E3.1 Table*
 189 *AI. USDA Healthy U.S.-Style Food Patterns—Intake Amounts*). For assignment to food groups,
 190 mixed dishes are disaggregated into their major component parts. For example, pizza may be
 191 disaggregated into the grain (crust), dairy (cheese), vegetable (sauce and toppings), and protein
 192 foods (toppings) food groups.
- 193 **Food pattern modeling**—The process of developing and adjusting daily intake amounts from
 194 food categories or groups to meet specific criteria, such as meeting nutrient intake goals, limiting
 195 nutrients or other food components, or varying proportions or amounts of specific food
 196 categories or groups.

- 197 **Food policies**—Regulations, laws, policy-making actions or formal or informal rules established
 198 by formal organizations or government units. Food and nutrition policies are those that influence
 199 the food environment and eating behavior to improve eating and body weight.
- 200 **Food security**—A condition in which all people, now and in the future, have access to sufficient,
 201 safe, and nutritious food to maintain a healthy and active life. (See **Household food insecurity**)
- 202 **Fortification**—The addition of one or more essential nutrients to a food whether or not it is
 203 normally contained in the food for the purpose of preventing or correcting a demonstrated
 204 deficiency of one or more nutrients in the population or specific population groups.
- 205 **Greenhouse gases (GHG)**—Any gas that absorbs infrared radiation in the atmosphere.
 206 Greenhouse gases include carbon dioxide, methane, nitrous oxide, ozone, chlorofluorocarbons,
 207 hydrochlorofluorocarbons, hydrofluorocarbons, perfluorocarbons, and sulfur hexafluoride.
- 208 **Health**—A state of complete physical, mental and social well-being and not merely the absence
 209 of disease or infirmity.
- 210 **Household food insecurity**—Circumstances in which the availability of nutritionally adequate
 211 and safe food, or the ability to acquire acceptable foods in socially acceptable ways, is limited or
 212 uncertain.
- 213 • **Persistent household food insecurity**—Occurs when people are unable to meet their
 214 minimum food requirements over a sustained period of time.
 - 215 • **Progressing household food insecurity**—A change in situation from food secure to food
 216 insecure or from acute or temporary food insecurity to persistent food insecurity.
 - 217 • **Household food insufficiency**—A similar measure to food insecurity considered more
 218 severe than the concept of food security, although not as severe as hunger.
- 219 **Interventionist**—Trained health professionals (e.g., registered dietitians, psychologists, exercise
 220 physiologists, health counselors, or professionals in training) who adhere to formal protocols in
 221 providing healthy lifestyles counseling and treatment, such as for weight management. In a few
 222 cases, lay persons are used as trained interventionists; they received instruction in protocols
 223 (designed by health professionals) for programs that have been validated in high-quality trials
 224 and published in peer-reviewed journals.
- 225 **Isocaloric**—Having the same caloric values. For example, two dietary patterns that vary in
 226 macronutrient proportions but have the same calorie content are isocaloric.
- 227 **Lean meat**—Any meat with less than 10% fat by weight, or less than 10 grams of fat per 100
 228 grams, based on USDA and FDA definitions for food label use. Examples include 95% lean
 229 ground beef, cooked; broiled beef steak, lean only eaten; baked pork chop, lean only eaten;
 230 roasted chicken breast or leg, no skin eaten; and smoked/cured ham, lean only eaten.

- 231
- 232 **Life Cycle Assessment (LCA)**—A technique for assessing the biophysical environmental
- 233 aspects and potential impacts associated with a product, by:
- 234 • Compiling an inventory of relevant inputs and outputs of a product system;
- 235 • Evaluating the potential environmental impacts associated with those inputs and outputs;
- 236 • Interpreting the results of the inventory analysis and impact assessment phases in relation
- 237 to the objectives of the study.
- 238 LCA studies the environmental aspects and potential impacts throughout a product’s life (i.e.,
- 239 cradle to grave), from raw material acquisition through production, use, and disposal. The
- 240 general categories of environmental impacts needing consideration include resource use, human
- 241 health, and ecological consequences.
- 242 **Macronutrient**—A dietary component that provides energy. Macronutrients include protein,
- 243 fats, and carbohydrates. Alcohol also provides energy but, for purposes of the DGAC report, it is
- 244 not considered when discussing macronutrients.
- 245 **Meta-analysis**—The statistical analysis of multiple individual studies for the purpose of
- 246 integrating the findings and deriving conclusions from the body of literature.
- 247 **Mobile Health (mHealth)**—The use of mobile and wireless technologies to support the
- 248 achievement of health objectives.
- 249 **Moderate alcohol consumption**—Average daily consumption of up to one drink per day for
- 250 women and up to two drinks per day for men, with no more than three drinks in any single day
- 251 for women and no more than four drinks in any single day for men. One drink is defined as 12 fl.
- 252 oz. of regular beer, 5 fl. oz. of wine, or 1.5 fl. oz. of distilled spirits.
- 253 **Nutrient-dense foods**—Foods that are naturally rich in vitamins, minerals, and other substances
- 254 that may have positive health effects, and are lean or low in solid fats and without added solid
- 255 fats, sugars, starches, or sodium and that retain naturally-occurring components such as fiber. All
- 256 vegetables, fruits, whole grains, fish, eggs, and nuts prepared without added solid fats or sugars
- 257 are considered nutrient-dense, as are lean or low-fat forms of fluid milk, meat, and poultry
- 258 prepared without added solid fats or sugars. Nutrient-dense foods provide substantial amounts of
- 259 vitamins and minerals (micronutrients) and relatively few calories compared to forms of the food
- 260 that have solid fat and/or added sugars.
- 261 **Nutrition Evidence Library (NEL) systematic review**—A process that uses state-of-the-art
- 262 methodology to search, evaluate, and synthesize food and nutrition-related research. This
- 263 rigorous, protocol-driven methodology is designed to minimize bias, maximize transparency, and
- 264 ensure relevant, timely, and high-quality systematic reviews to inform Federal nutrition-related

265 policies, programs, and recommendations. The NEL is a division of the USDA Center for
266 Nutrition Policy and Promotion. For more detailed information, visit: www.nel.gov.

267 **Oils**—Fats that are liquid at room temperature. Oils come from many different plants and some
268 fish. Some common oils include canola, corn, olive, peanut, safflower, soybean, and sunflower
269 oils. A number of foods are naturally high in oils, such as: nuts, olives, some fish, and avocados.
270 Foods that are mainly made up of oil include mayonnaise, certain salad dressings, and soft (tub
271 or squeeze) margarine with no *trans* fats. Oils are high in monounsaturated or polyunsaturated
272 fats, and lower in saturated fats than solid fats. A few plant oils, termed tropical oils, including
273 coconut oil, palm oil and palm kernel oil, are high in saturated fats and for nutritional purposes
274 should be considered as solid fats. Partially-hydrogenated oils that contain *trans* fats should also
275 be considered as solid fats for nutritional purposes. (See **Fats**)

276 **Ounce equivalent (oz eq)**—The amount of a food product that is considered equal to one ounce
277 from the grain or protein foods food group. An oz eq for some foods may be less than a
278 measured ounce in weight if the food is concentrated or low in water content (nuts, peanut butter,
279 dried meats, flour) or more than a measured ounce in weight if the food contains a large amount
280 of water (tofu, cooked beans, cooked rice or pasta).

281 **Persistent organic pollutants (POPs)**—Toxic chemicals that can adversely affect human health
282 and the biophysical environment. Because they can be transported by wind and water, most
283 POPs generated in one country may affect people and wildlife distant to where they are used and
284 released. They can persist for long periods of and can accumulate and pass from one species to
285 the next through the food chain.

286 **Plant-based foods**—Foods such as vegetables, fruits, whole grains, nuts and seeds.

287 **Point-of-purchase**—A place where sales are made. Various intervention strategies have been
288 proposed to affect individuals' purchasing decisions at the point of purchase, such as board or
289 menu labeling with various amounts of nutrition information or shelf tags in grocery stores.

290 **Portion size**—The amount of a food served or consumed in one eating occasion. A portion is not
291 a standardized amount, and the amount considered to be a portion is subjective and varies.

292 **Processed meat**—Meat, poultry, or seafood products preserved by smoking, curing or salting, or
293 addition of chemical preservatives. Processed meat includes bacon, sausage, hot dogs, sandwich
294 meat, packaged ham, pepperoni, and salami.

295 **Protein**—One of the three macronutrients classes. Protein is the major functional and structural
296 component of every animal cell. Proteins are composed of amino acids, nine of which are
297 indispensable, meaning they cannot be synthesized by humans and therefore must be obtained
298 from the diet. The quality of dietary protein is determined by its amino acid profile relative to
299 human requirements as determined by the body's requirements for growth, maintenance, and
300 repair. Protein quality is determined by two factors: digestibility and amino acid composition.

- 301 • **Animal protein**—Protein from meat, poultry, seafood, eggs, and milk and milk products.
- 302 • **Vegetable protein**—Protein from plants such as dry beans, whole grains, fruit, nuts, and
303 seeds.
- 304 **Refined grains**—Grains and grain products missing the bran, germ, and/or endosperm; any grain
305 product that is not a whole grain. Many refined grains are low in fiber but enriched with thiamin,
306 riboflavin, niacin, and iron, and fortified with folic acid.
- 307 **Screen time**—Time in front of a computer, television, video or computer game system, or smart
308 phone or tablet or related device.
- 309 **Seafood**—Marine animals that live in the sea and in freshwater lakes and rivers. Seafood
310 includes fish, such as salmon, tuna, trout, and tilapia, and shellfish, such as shrimp, crab, and
311 oysters.
- 312 **Sedentary behavior**—Any waking activity predominantly done while in a sitting or reclining
313 posture. A behavior that expends energy at or minimally above a person’s resting level (between
314 1.0 and 1.5 metabolic equivalents), is considered sedentary behavior.
- 315 **Self-monitoring**—Self-monitoring refers to the process by which an individual observes and
316 records specific information about his or her behaviors. For example, in weight management
317 self-monitoring, observations and records would reflect dietary intake, physical activity, and/or
318 body weight.
- 319 **Solid fats**—Fats that are usually not liquid at room temperature. Solid fats are found in animal
320 foods except for seafood, and can be made from vegetable oils through hydrogenation. Some
321 tropical oil plants, such as coconut and palm, are considered as solid fats due to their fatty acid
322 composition. Solid fats contain more saturated fats and/or *trans* fats than liquid oils (e.g.,
323 soybean, canola, and corn oils), with lower amounts of monounsaturated or polyunsaturated fatty
324 acids. Common fats considered to be solid fats include: butterfat, beef fat (tallow, suet), chicken
325 fat, pork fat (lard), stick margarine, shortening, coconut oil, palm oil and palm kernel oil. Foods
326 high in solid fats include: butter, full-fat cheeses, creams, whole milk, full fat ice creams,
327 marbled cuts of meats, regular ground beef, bacon, sausages, poultry skin, and many baked
328 goods made using these products (such as cookies, crackers, doughnuts, pastries, and
329 croissants). The fat component of milk and cream (butter) is solid at room temperature. (See
330 **Fats**)
- 331 **Sugar-sweetened beverages**—Liquids that are sweetened with various forms of added sugars
332 (see **Added Sugars** and **Carbohydrates: Sugars**). These beverages include, but are not limited
333 to, soda, fruitades, and sports drinks. Also called calorically-sweetened beverages.

334 **Sustainable diets**—A pattern of eating that promotes health and well-being and provides food
335 security for the present population while sustaining human and natural resources for future
336 generations.

337 **Trophic level**—A functional classification of species that is based on feeding relationships.
338 Generally, aquatic and terrestrial green plants comprise the first, or lowest, trophic level,
339 herbivores comprise the second, and primary carnivores comprise the third, or highest level.
340 Examples of high trophic fish species are salmon and trout. Low trophic fish species include
341 crayfish and catfish.

342 **Whole grains**—Grains and grain products made from the entire grain seed, usually called the
343 kernel, which consists of the bran, germ, and endosperm. If the kernel has been cracked, crushed,
344 or flaked, it must retain the same relative proportions of bran, germ, and endosperm as the
345 original grain in order to be called whole grain. Many, but not all, whole grains are also sources
346 of dietary fiber.

Appendix E-6: History of Dietary Guidance Development in the United States and the Dietary Guidelines for Americans

In early 1977, after years of discussion, scientific review, and debate, the U.S. Senate Select Committee on Nutrition and Human Needs, led by Senator George McGovern, recommended Dietary Goals for the American people (U.S. Senate Select Committee, 1977). The Goals consisted of complementary nutrient-based and food-based recommendations. The first Goal focused on energy balance and recommended that, to avoid overweight, Americans should consume only as much energy as they expended. Overweight Americans should consume less energy and expend more energy. For the nutrient-based Goals, the Senate Committee recommended that Americans:

- Increase consumption of complex carbohydrates and “naturally occurring sugars;” and
- Reduce consumption of refined and processed sugars, total fat, saturated fat, cholesterol, and sodium.
- For the food-based Goals, the Senate Committee recommended that Americans:
 - Increase consumption of fruits, vegetables, and whole grains;
 - Decrease consumption of:
 - refined and processed sugars and foods high in such sugars;
 - foods high in total fat and animal fat, and partially replace saturated fats with polyunsaturated fats;
 - eggs, butterfat, and other high-cholesterol foods;
 - salt and foods high in salt; and
 - Choose low-fat and non-fat dairy products instead of high-fat dairy products (except for young children).

The Dietary Goals was met with considerable debate and controversy, as industry groups and the scientific community expressed doubt that the science available at the time supported the specificity of the numbers provided in the Dietary Goals. To support the credibility of the science used by the Senate Committee, the U.S. Department of Agriculture and U.S. Department of Health and Human Services (then called the Department of Health, Education, and Welfare) selected scientists from the two Departments and obtained additional expertise from the scientific community throughout the country to address the public’s need for authoritative and consistent guidance on diet and health.

In February 1980, the two Departments collaboratively issued *Nutrition and Your Health: Dietary Guidelines for Americans*, a brochure that, in describing seven principles for a healthful

35 diet, provided assistance for healthy people in making daily food choices (USDA/HHS, 1980).
36 These Guidelines were based, in part, on the 1979 *Surgeon General's Report on Health*
37 *Promotion and Disease Prevention* (DHEW/PHS, 1979) and reflected findings from a study on
38 the relationship between dietary practices and health outcomes (ASCN, 1979). Ideas for
39 incorporating a variety of foods to provide essential nutrients while maintaining recommended
40 body weight were a focus. The brochure also provided guidance on limiting dietary components
41 such as fat, saturated fat, cholesterol, and sodium, which were beginning to be considered risk
42 factors in certain chronic diseases. Both the Dietary Goals and the first Dietary Guidelines for
43 Americans were different from previous dietary guidance in that they reflected emerging
44 scientific evidence and changed the historical focus on nutrient adequacy to also identify the
45 impacts of diet on chronic disease. These documents discussed the concepts of moderation as
46 well as nutrient adequacy.

47
48 Even though the recommendations of the 1980 *Dietary Guidelines for Americans* were presented
49 as innocuous and straightforward extrapolations from the science base, they, too, were met with
50 controversy from a variety of industry and scientific groups.

51
52 The debate about the 1980 *Dietary Guidelines for Americans* led to Congressional report
53 language that directed the two Departments to convene an advisory committee that would ensure
54 that outside advice, both formal and informal, was captured in developing future editions of the
55 Dietary Guidelines. A Dietary Guidelines Advisory Committee composed of scientific experts
56 outside the Federal sector was established shortly after that directive and was very helpful in the
57 development of the 1985 *Nutrition and Your Health: Dietary Guidelines for Americans*
58 (USDA/HHS, 1985). The Departments made relatively few changes from the first edition, but
59 this second edition was issued with much less debate from either industry or the scientific
60 community. The 1985 Dietary Guidelines were widely accepted and were used as the framework
61 for consumer nutrition education messages. They also were used as a guide for healthy diets by
62 scientific, consumer, and industry groups.

63
64 In 1989, USDA and HHS established a second scientific advisory committee to review the 1985
65 Dietary Guidelines and make recommendations for revision. The basic tenets of earlier Dietary
66 Guidelines were reaffirmed, and the 1990 *Nutrition and Your Health: Dietary Guidelines for*
67 *Americans* (USDA/HHS, 1990) promoted enjoyable and healthful eating through variety and
68 moderation, rather than dietary restriction. For the first time, the Guidelines also suggested
69 quantitative goals for total fat and saturated fat, though they stressed that the goals were to be
70 met through dietary choices made over several days, not through choices about one meal or one
71 food.

72
73 The 1980, 1985, and 1990 editions of the Dietary Guidelines were issued voluntarily by the two
74 Departments. With the passage of the 1990 National Nutrition Monitoring and Related Research

75 Act (Section 301 of Public Law 101-445, 7 USC 5341, Title III) (US Congress, 1990), the 1995
76 edition of *Nutrition and Your Health: Dietary Guidelines for Americans* became the first Dietary
77 Guidelines policy document mandated by statute. This Act directed the Secretaries of USDA and
78 HHS to jointly issue at least every 5 years a report entitled *Dietary Guidelines for Americans*.

79
80 A Dietary Guidelines Advisory Committee was established to prepare technical reports that
81 advised the Federal government on the status of the evidence on nutrition and health. These
82 technical reports were used in developing the 1995, 2000, 2005, and 2010 versions of the
83 *Dietary Guidelines for Americans* (HHS/USDA, 1995a; HHS/USDA, 1995b; USDA/HHS,
84 2000a; USDA/HHS, 2000b; HHS/USDA, 2004; HHS/USDA, 2005a; USDA/HHS, 2010;
85 USDA/HHS, 2011). This report of the 2015 Dietary Guidelines Advisory Committee will serve a
86 similar purpose for HHS and USDA as the Departments develop the 2015 edition of *Dietary*
87 *Guidelines for Americans*.

88
89 Since 1980, the Dietary Guidelines have been notably consistent in their recommendations on the
90 components of a healthful diet, but they also have changed in some significant ways to reflect
91 emerging science as well as public health concerns, such as the increasing prevalence of major
92 chronic diseases among the majority of the general population. In keeping with growing
93 emphasis on data quality in developing recommendations, the 2005 Committee used a modified
94 systematic approach for reviewing the scientific literature. This systematic review of the
95 evidence was further realized for the 2010 Dietary Guidelines Advisory Committee with the
96 establishment of the USDA's Nutrition Evidence Library, a process that uses state-of-the-art
97 methodology to search, evaluate, and synthesize food and nutrition-related research. This
98 rigorous, protocol-driven methodology is designed to minimize bias, maximize transparency, and
99 ensure relevant, timely, and high-quality systematic reviews to inform Federal nutrition-related
100 policies, programs, and recommendations. (See **PART C: Methodology** for a brief description of
101 the systematic evidence review process used by the 2015 Dietary Guidelines Advisory
102 Committee and www.NEL.gov for additional information about the Nutrition Evidence Library.)

103
104 Over the past two decades, *Nutrition and Your Health: Dietary Guidelines for Americans* has
105 evolved to become a broadly accepted, science-based document that serves as the Federal
106 nutrition policy on which nutrition standards, nutrition programs, and nutrition education are
107 based. The Dietary Guidelines have proven to be a mechanism for addressing public health
108 concerns by providing focused guidance that can help to promote health and reduce chronic
109 disease risk. As such, while earlier editions of the Dietary Guidelines focused specifically on
110 healthy Americans ages 2 years and older, more recent editions also have included those who are
111 at increased risk of chronic disease. The Dietary Guidelines, however, are not directly intended
112 for disease treatment, but they can be used as a basis for developing clinical guidelines.

113

114 Future editions of the Dietary Guidelines will continue to evolve to address public health
 115 concerns and the nutrition needs of specific populations. For example, a Federal initiative has
 116 been established to develop comprehensive guidance for infants and toddlers from birth to 24
 117 months and women who are pregnant so that by 2020, the Dietary Guidelines will also include
 118 these important populations comprehensively. For now, nutrition and health professionals
 119 actively promote the Dietary Guidelines as a means of encouraging Americans to focus on eating
 120 a healthful diet and being physically active throughout the entire lifespan.
 121

122 **HISTORY OF DIETARY GUIDANCE DEVELOPMENT IN THE UNITED**
 123 **STATES AND THE DIETARY GUIDELINES FOR AMERICANS – A**
 124 **CHRONOLOGY**
 125

- 1977** *Dietary Goals for the United States* (the “McGovern Report”) was issued by the U.S. Senate Select Committee on Nutrition and Human Needs (U.S. Senate Select Committee, 1977). The Dietary Goals reflected a shift in focus from obtaining adequate nutrients to avoiding excessive intake of food components linked to chronic disease. These goals were controversial among some nutritionists and others concerned with food, nutrition, and health.
- 1979** The American Society for Clinical Nutrition formed a panel to study the relationship between dietary practices and health outcomes (ASCN, 1979). The findings, presented in 1979, were reflected in *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention* (DHEW/PHS, 1979).
- 1980** Seven principles for a healthful diet were jointly issued by the then U.S. Department of Health, Education, and Welfare (now HHS) and the U.S. Department of Agriculture (USDA) in response to the public's desire for authoritative, consistent guidelines on diet and health. These principles became the first edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1980). The 1980 Guidelines were based on the most up-to-date information available at the time and were directed to healthy Americans ages 2 and older. The Guidelines generated some concern among consumer, commodity, and food industry groups, as well as some nutrition scientists, who questioned the causal relationship between certain guidelines and health.
- 1980** A U.S. Senate Committee on Appropriations report directed that an external advisory committee be established to review scientific evidence and recommend revisions to the 1980 *Nutrition and Your Health: Dietary Guidelines for Americans* (U.S. Senate, 1980).
- 1983** An external Federal advisory committee of nine nutrition scientists was convened to review and make recommendations in a report to the Secretaries of USDA and HHS about the first (1980) edition of the Dietary Guidelines (USDA/HHS, 1985a).

- 1985** USDA and HHS jointly issued the second edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1985b). This edition was nearly identical to the first, retaining the seven guidelines from the 1980 edition. Some changes were made for clarity, while others reflected advances in scientific knowledge of the associations between diet and chronic diseases. The second edition received wide acceptance and was used as the basis for dietary guidance for the general public as well as a framework for developing consumer education messages.
- 1987** Language in the *Conference Report of the House Committee on Appropriations* indicated that USDA, in conjunction with HHS, “shall reestablish a Dietary Guidelines Advisory Group on a periodic basis. This Advisory Group will review the scientific data relevant to nutritional guidance and make recommendations on appropriate changes to the Secretaries of the Departments of Agriculture and Health and Human Services” (U.S. House of Representatives, 1987).
- 1989** USDA and HHS established a second Federal advisory committee of nine members, which considered whether revisions to the 1985 Dietary Guidelines were needed and made recommendations for revision in a report to the Secretaries (USDA/HHS, 1990a). The 1988 Surgeon General's Report on Nutrition and Health (HHS/PHS, 1988) and the 1989 National Research Council's report *Diet and Health: Implications for Reducing Chronic Disease Risk* were key resources used by the Committee (NAS/NRC, 1989).
- 1990** USDA and HHS jointly released the third edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1990b). The basic tenets of the 1985 Dietary Guidelines were reaffirmed, with additional refinements made to reflect increased understanding of the science of nutrition and how best to communicate the science to consumers. The language of the new Dietary Guidelines was positive, was oriented toward the total diet, and provided specific information regarding food selection. For the first time, quantitative recommendations were made for intakes of dietary total fat and saturated fat.
- 1990** The 1990 National Nutrition Monitoring and Related Research Act (Section 301 of Public Law 101-445, 7 USC 5341, Title III) directed the Secretaries of the USDA and HHS to jointly issue at least every 5 years a report entitled *Dietary Guidelines for Americans* (U.S. Congress, 1990). This legislation also required USDA and HHS to review all Federal publications containing dietary advice for the general public.
- 1993** HHS initiated a charter establishing the 1995 Dietary Guidelines Advisory Committee.
- 1994** An 11-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of HHS and USDA to review the third edition of the Dietary Guidelines and determine whether changes were needed. If so, the Committee was to recommend

suggestions and the rationale for any revisions.

- 1995** The report of the Dietary Guidelines Advisory Committee to the Secretaries of HHS and USDA was published (HHS/USDA, 1995a).
- 1995** Using the 1995 report of the Dietary Guidelines Advisory Committee as the foundation, HHS and USDA jointly developed and released the fourth edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (HHS/USDA, 1995b). This edition continued to support the concepts from earlier editions. New information included the Food Guide Pyramid, Nutrition Facts label, boxes highlighting good food sources of key nutrients, and a chart illustrating three weight ranges in relation to height.
- 1997** USDA initiated the charter establishing the 2000 Dietary Guidelines Advisory Committee.
- 1998** An 11-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the fourth edition of the Dietary Guidelines to determine whether changes were needed and, if so, to recommend suggestions for revision.
- 2000** The Committee submitted its report to the Secretaries of USDA and HHS (USDA/HHS, 2000a). This report contained the proposed text for the fifth edition of *Nutrition and Your Health: Dietary Guidelines for Americans*.
- 2000** The President of the United States spoke of the Dietary Guidelines in his radio address after USDA and HHS jointly issued the fifth edition of *Nutrition and Your Health: Dietary Guidelines for Americans* earlier in the day (USDA/HHS, 2000b). Earlier versions of the Guidelines included seven statements. This version included 10—created by breaking out physical activity from the weight guideline, splitting the grains and fruits/vegetables recommendations for greater emphasis, and adding a new guideline on safe food handling.
- 2003** HHS initiated the charter establishing the 2005 Dietary Guidelines Advisory Committee.
- 2003** A 13-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of HHS and USDA to review the fifth edition of the Dietary Guidelines to determine whether changes were needed and, if so, to recommend suggestions for revision.
- 2003-2004** In keeping with renewed emphasis on data quality, the Committee used a modified “systematic approach” to review the scientific literature and develop its recommendations. Committee members initially posed approximately 40 specific research questions that were answered using an extensive search and review of the scientific literature. Issues relating diet and physical activity to health promotion and chronic disease prevention were included in the Committee’s evidence review. Other

major sources of evidence used were the Dietary Reference Intake (DRI) reports prepared by expert committees convened by the Institute of Medicine (IOM) as well as various Agency for Healthcare Research and Quality (AHRQ) and World Health Organization (WHO) reports. In addition, USDA completed numerous food intake pattern modeling analyses and the Committee analyzed various national data sets and sought advice from invited experts.

- 2004** The Committee submitted its technical report to the Secretaries of HHS and USDA (HHS/USDA, 2004). This 364-page report contained a detailed analysis of the science and was accompanied by many pages of evidence-based tables that were made available electronically. After dropping some questions because of incomplete or inconclusive data, the Committee wrote conclusions and comprehensive rationales for 34 of the 40 original questions.
- 2005** Using the Committee’s technical report as a basis, HHS and USDA jointly prepared and issued the sixth edition of *Dietary Guidelines for Americans* in January 2005 (HHS/USDA, 2005a). This 80-page policy document was the first time the Departments prepared a policy document that was intended primarily for use by policy makers, healthcare professionals, nutritionists, and nutrition educators. The content of this document included nine major Dietary Guidelines messages that resulted in 41 Key Recommendations, of which 23 were for the U.S. population overall and 18 for specific population groups. The policy document highlighted the USDA Food Guide and the DASH Eating Plan as two examples of eating patterns that exemplify the Dietary Guidelines recommendations. A companion, 10-page brochure called *Finding Your Way to a Healthier You* (HHS/USDA, 2005b) was released concurrently with the Dietary Guidelines to provide advice to consumers about food choices that promote health and decrease the risk of chronic disease. Shortly thereafter, USDA released the MyPyramid Food Guidance System, an update of the Food Guide Pyramid, which included more detailed advice for consumers to help them follow the Dietary Guidelines.
- 2008** USDA initiated the charter establishing the 2010 Dietary Guidelines Advisory Committee.
- 2008** A 13-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the sixth edition of *Dietary Guidelines for Americans* to determine whether changes were needed and, if so, to recommend suggestions for revision.
- 2008-2009** USDA’s Center for Nutrition Policy and Promotion established the Nutrition Evidence Library (NEL) to conduct systematic reviews to help inform Federal nutrition policy and programs. The NEL supported the Dietary Guidelines Advisory Committee in

answering approximately 130 of the total 180 diet and health-related questions posed. This was the most rigorous and comprehensive approach used to date for reviewing the science in order to develop nutrition-related recommendations for the public. Other sources of evidence for answering scientific questions included modeling analyses of USDA's Food Patterns, review of reports from various data analyses, as well as other available authoritative reports (e.g., 2005 DGAC Report and IOM reports). An elaborate web-based public comments database was developed and provided a successful mechanism for the public to provide comments and thereby participate in the Committee's evidence review process. The database also allowed the public to read other comments that were submitted. This database eventually included more than 800 public comments related to the DGAC process.

- 2010** The Committee submitted its technical report to the Secretaries of USDA and HHS (USDA/HHS 2010). This 445-page report contained a detailed analysis of the science and was accompanied by additional 230 pages of food pattern modeling appendices made available electronically at www.DietaryGuidelines.gov.
- 2011** Using the Committee's technical report as the basis, HHS and USDA jointly prepared and published the seventh edition of *Dietary Guidelines for Americans* released publically in January 2011 (USDA/HHS, 2011). The 95-page policy document encompassed the overarching concepts of maintaining calorie balance over time to achieve and sustain a healthy weight, and consuming nutrient-dense foods and beverages. The policy document included 23 key recommendations for the general population and six additional key recommendations for specific populations. To assist individuals to build a healthy diet based on the Dietary Guidelines, the USDA Food Patterns were updated and new vegetarian adaptations were included. The DASH Eating Plan also was included as an example of a healthy dietary pattern. This publication will serve as the basis for Federal nutrition policy until the next policy document is released in 2015. In June, USDA released MyPlate, a new visual icon, and the ChooseMyPlate.gov website that provides tools to help consumers of all ages, educators, and health professionals learn about and follow the Dietary Guidelines.
- 2013** HHS initiated the charter establishing the 2015 Dietary Guidelines Advisory Committee.
- 2013** A 15-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the seventh edition of *Dietary Guidelines for Americans* and recommend suggestions for revision. One member resigned due to professional obligations within the first three months after appointment; 14 members served the remainder of the two-year charter. The Committee also added three consultant subcommittee members during its work to address specific issues; these members participated in discussions and decision at the subcommittee level but were not members

of the full Committee.

- 2015** The Committee submitted this technical report to the Secretaries of USDA and HHS in January 2015. This 580-page report contained a detailed analysis of the science and was accompanied by substantial documentation of the process made available electronically at www.DietaryGuidelines.gov and www.NEL.gov.

126

127

128 **REFERENCES**

129

130 American Society for Clinical Nutrition (ASCN). Symposium. Report of the Task Force on the
 131 evidence relating six dietary factors to the nation's health. *Am J Clin Nutr.*
 132 1979;32(Supplement):2621-748.

133

134 Murphy S. Development of the MyPyramid Food Guidance System. *J Nutr Educ Behav.*
 135 2006;38(6S):S77-S162.

136

137 National Academy of Science, National Research Council (NAS, NRC). *Diet and Health:
 138 Implications for Reducing Chronic Disease Risk.* Washington (DC): National Academy Press,
 139 1989.

140

141 U.S. Congress. National Nutrition Monitoring and Related Research Act of 1990, Public Law
 142 445, 101st Cong., 2nd Session, Section 301, 7 USC 5341, October 22, 1990.

143

144 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 145 (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans.* 1st edition.
 146 Washington (DC): USDA/HHS, 1980. Home and Garden Bulletin No. 232.

147

148 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 149 (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
 150 for Americans, 1985.* Washington (DC): USDA, Human Nutrition Information Service, 1985a.

151

152 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 153 (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans.* 2nd edition.
 154 Washington (DC): USDA/HHS, 1985b. Home and Garden Bulletin No. 232.

155

156 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 157 (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
 158 for Americans, 1990.* Washington (DC): USDA, Human Nutrition Information Service, 1990a.

159

160 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 161 (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans.* 3rd edition.
 162 Washington (DC): USDA/HHS, 1990b. Home and Garden Bulletin No. 232.

163

164 U.S. Department of Agriculture and U.S. Department of Health and Human Services
 165 (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
 166 for Americans, 2000.* Washington (DC): USDA, Agricultural Research Service, 2000a.

167

- 168 U.S. Department of Agriculture and U.S. Department of Health and Human Services
169 (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans*. 5th edition.
170 Washington (DC): USDA/HHS, 2000b. Home and Garden Bulletin No. 232.
171
- 172 U.S. Department of Agriculture and U.S. Department of Health and Human Services
173 (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
174 for Americans, 2010*. Washington (DC): USDA, Agricultural Research Service, 2010.
175
- 176 U.S. Department of Agriculture and U.S. Department of Health and Human Services
177 (USDA/HHS). *Dietary Guidelines for Americans*. 7th edition. Washington (DC): U.S.
178 Government Printing Office, 2011.
179
- 180 U.S. Department of Health and Human Services and U.S. Department of Agriculture
181 (HHS/USDA). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
182 for Americans, 1995*. Washington (DC): USDA, Agricultural Research Service, 1995a.
183
- 184 U.S. Department of Health and Human Services and U.S. Department of Agriculture
185 (HHS/USDA). *Nutrition and Your Health: Dietary Guidelines for Americans*. 4th edition. Home
186 and Garden Bulletin No. 232. Washington (DC): HHS/USDA, 1995b.
187
- 188 U.S. Department of Health and Human Services and U.S. Department of Agriculture
189 (HHS/USDA). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines
190 for Americans, 2005*. Washington (DC): USDA, Agricultural Research Service, 2004.
191
- 192 U.S. Department of Health and Human Services and U.S. Department of Agriculture
193 (HHS/USDA). *Dietary Guidelines for Americans*. 6th edition. Washington (DC): U.S.
194 Government Printing Office, 2005a.
195
- 196 U.S. Department of Health and Human Services and U.S. Department of Agriculture
197 (HHS/USDA). *Finding Your Way to a Healthier You*. Washington (DC): U.S. Government
198 Printing Office, 2005b. HHS-ODPHP-205-01-DGA-A; USDA Home and Garden Bulletin No.
199 232.
200
- 201 U.S. Department of Health and Human Services, Public Health Service (HHS, PHS). *The
202 Surgeon General's Report on Nutrition and Health*. Washington (DC): DHHS, PHS, 1988.
203 Publication No. 88-50215.
204
- 205 U.S. Department of Health, Education, and Welfare, Public Health Service (DHEW, PHS).
206 *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention*.
207 Washington (DC): DHEW, PHS, 1979. DHEW Publication No. 79-55071.

- 208 U.S. House of Representatives Conference Committee (U.S. House of Representatives). 100th
209 Cong., 1st sess., H. Rep. 498, 1987.
210
- 211 U.S. Senate Agricultural Appropriations Committee (U.S. Senate). 96th Cong., 1st sess., S. Rep.
212 1030, 1980.
213
- 214 U.S. Senate Select Committee on Nutrition and Human Needs. Dietary Goals for the United
215 States. 2nd edition. Washington (DC): U.S. Government Printing Office, 1977.
216

Appendix E-7: Public Comments

As a government advisory committee, the Dietary Guidelines Advisory Committee (DGAC) is required by the Federal Advisory Committee Act to function in an open process in which the public may participate. This is accomplished through public submission of written comments and oral testimony given to the DGAC.

Federal Register notices alerted the public to DGAC meetings held in-person and/or by webcast. In these notices the public was invited and reminded to submit their comments to an online database at www.DietaryGuidelines.gov. The public comments process opened on May 29, 2013. Comments continued to be submitted throughout the time the DGAC operated. Following the submission of the 2015 DGAC Report to the Secretaries of HHS and USDA, the Federal government will alert the public of its availability through a *Federal Register* notice. This notice also will announce a public comment period and the date of an in-person meeting where the public can provide comments to the Federal government about the DGAC Report.

A public comments online database was developed for the 2015 DGAC process based on the structure and content used for the 2010 process, but with many enhancements that were intended to streamline submission of comments by the public and processing by staff.

When submitting comments, the public selected one or more topic areas into which they felt their comments belonged. Initially, these topic areas were: Food Groups, Eating Patterns-Diets, Energy Balance, Carbohydrates, Protein, Fats, Micronutrients, Water and Nonalcoholic Beverages, Alcoholic Beverages, Food Safety, Behavior and Food Environment, Lifespan Needs, and Other. During their deliberations and at the DGAC's request, the topic area "Behavior and Food Environment" was split into two distinct topic areas, "Behavior" and "Food Environment," and a new topic area, "Sustainability," also was added. Individual submissions were allowed to include up to five attachments, such as journal articles, reports, and other scientific material for the DGAC to consider. The submission page noted that submitters should take care to not violate copyright laws when submitting attachments.

For the first time, the 2015 DGAC requested public comments related to specific topic areas. Subcommittee (SC) 2 requested comments on "steps the food industry is taking or has taken to reduce the nutrients listed below in the food supply, including what nutrients have been increased as a consequence of reductions where applicable: sodium, added sugars, fats (i.e., total fats, saturated fats, *trans* fats, and other individual fatty acids)." SC 5 requested public comments on "a targeted topic on food system sustainability, including comments from both the private and public sectors and addressing local, regional, national, or international scales. Specifically, it seeks approaches and current examples of sustainability in the food system. Comments are encouraged that address: (a) Elements of a whole food system; (b) Information

40 on specific food groups or commodities; and (c) Sustainability metrics that have been
41 implemented or are in development.”

42
43 In addition, for the first time, the Committee also provided specific guidance to the public on
44 “length and timing of public comments.” This guidance was shared through the *Federal Register*
45 and on www.DietaryGuidelines.gov. This guidance stated to “provide a brief summary (approx.
46 250 words) of the points or issues in the comment text box.” It asked that “if providing literature
47 or other resources, one of the following forms is preferred: complete citation, as in a
48 bibliographic entry; abstract; electronic link to full article or report.” The public was encouraged
49 to “provide comments as early as possible in the Committee’s process to increase the opportunity
50 for meaningful impact.” Lastly, as of April 2014, it stated that “a deadline for comment
51 submission prior to each public meeting will no longer be used.”

52
53 For all public comments, submitters were required to provide the following information: topic
54 area(s), the comment itself (5,000 character limit), any accompanying attachments, full name
55 (with option to make it public), affiliation, and organization. They also were required to provide
56 their email address, phone number, and zip code, but this information was not included when the
57 comment was posted on the www.DietaryGuidelines.gov public comments page. Submitters
58 were given the option, but not required, to also provide their business or academic credentials
59 and postal address, including country. This information was not posted on the public website.
60 After the comment was submitted, confirmation was provided to the submitter by e-mail.

61
62 Staff reviewed each submitted comment. Only a few comments were not posted; reasons were:
63 (1) duplicate submission of another comment posted by the same submitter, (2) test submission,
64 (3) partial comment due to the 5,000 character limit, which was corrected by a shorter comment
65 being submitted, and (4) comments that did not pertain to the DGAC.

66
67 At the request of the DGAC, staff generated reports and drafted summaries on each topic area for
68 comments submitted since the previous meeting or since the previous comment summary. On
69 occasion, various Committee members also chose to access the public comments database
70 themselves in order to read comments.

71
72 A total of 972 comments were submitted from May 29, 2013 through the closing of the public
73 comments database on December 30, 2014. Of these, 918 were relevant to the DGAC’s work.

74
75 The majority of comments submitted fell into these topic areas: Food Groups; Eating Patterns-
76 Diets; Sustainability; and Energy Balance. However, comments were received in all 18 topic
77 areas and covered a wide range of issues. Comments came from the United States, Australia,
78 India, Spain, Canada, Brazil, France, Belgium, Norway, Iraq, United Kingdom, Pakistan,
79 Indonesia, and Denmark.

80

81 In addition to written comments, oral comments from 53 individuals were presented at the
82 January 2014 public meeting. The list of presenters along with their affiliations is located on
83 www.DietaryGuidelines.gov under Meeting 2 (January 13-14, 2014). These 53 individuals each
84 provided 3 minutes or less of testimony before the Committee, and they submitted a brief outline
85 of their comments when registering to participate in the comment session.

86

87 The oral and written comments provided by the public were valuable in that they helped
88 the Committee gather background information and understand public and professional
89 perceptions. Comments from the public brought new issues to light as well as new approaches to
90 current issues and emerging evidence. They also highlighted and ensured consideration of topics
91 deemed to be important by the submitters, who represented a variety of backgrounds and focus
92 areas. The public comments will remain archived at www.DietaryGuidelines.gov.

Appendix E-8: Biographical Sketches of the 2015 DGAC

Chair: Barbara Millen, DrPH, RD: *Professor, Department of Family Medicine, Boston University School of Medicine, Boston, MA (through 2009).* Dr. Millen is currently the Founder and President of Millennium Prevention, Inc., a U.S.-based start-up company with a public health mission, which develops web-based platforms and mobile applications to encourage healthy preventive lifestyle behaviors for clinical settings and corporate, academic, and community wellness initiatives. Dr. Millen is a nutrition epidemiologist whose academic research career focused on dietary patterns and lifestyle determinants of health and chronic disease risk as well as evidence-based clinical and public health strategies to promote optimal nutrition and well-being in younger and older adults as well as low-income and minority populations. During her 30-year tenure at Boston University, she was the Founding Chairman of the Graduate Programs in Medical Nutrition Sciences, the Associate Dean for Research and Faculty Development of the School of Public Health, the Chairman of the Faculty Council, and Director of Nutrition Research for the internationally-renown Framingham Heart Study. She has advised research groups nationally and globally, including the World Health Organization, and served from 2008 to 2013 on the expert panels for the American Heart Association (AHA)/American College of Cardiology (ACC)/The Obesity Society (TOS) Guideline for the Management of Overweight and Obesity in Adults and the AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk.

Vice Chair: Alice H. Lichtenstein, DSc: *Stanley N. Gershoff Professor of Nutrition Science and Policy, Friedman School of Nutrition Science and Policy, Tufts University, Boston, MA.* Dr. Lichtenstein is also Director and Senior Scientist, Cardiovascular Nutrition Laboratory, Jean Mayer USDA Human Nutrition Research Center on Aging and Professor of Medicine at Tufts University School of Medicine. Dr. Lichtenstein has broad expertise in nutrition and cardiovascular disease risk reduction. She previously served as a member of the 2000 Dietary Guidelines Advisory Committee and as a member of the Institute of Medicine (IOM) Dietary Reference Intake Panel on Macronutrients. Dr. Lichtenstein recently served as the vice-chair of the IOM Committee on Examination of Front-of-Package Nutrient Rating System and Symbols, a member of the IOM Committee on the Consequences of Sodium Reduction in Populations, the vice-chair of the ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults expert panel, a member of the AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk expert work group, and is chair of the American Heart Association's Nutrition Committee. She is currently a member of the IOM Food and Nutrition Board.

Steven Abrams, MD: *Professor of Pediatrics, Baylor College of Medicine, Houston, TX.* Dr. Abrams also is an Adjunct Professor at the University of Texas School of Public Health and the Medical Director for the Neonatal Nutrition Program at Baylor College of Medicine. He is an

40 expert on mineral requirements in children, including calcium, zinc, iron, magnesium, and
41 copper. He has served on the IOM Panels on Calcium and Vitamin D and the Use of Dietary
42 Reference Intakes in Nutrition Labeling, and on the IOM Subcommittee on Upper Safe
43 Reference Levels of Nutrients. Dr. Abrams currently is a member of the American Academy of
44 Pediatrics Committee on Nutrition and the American Society for Bone and Mineral Research.

45
46 **Lucile Adams-Campbell, PhD:** *Professor of Oncology, Georgetown University Medical*
47 *Center, Lombardi Comprehensive Cancer Center, Washington, DC.* Dr. Adams-Campbell also
48 serves as the Associate Director of Minority Health and Health Disparities Research and
49 Associate Dean of Community Health and Outreach at Georgetown University Medical Center
50 Lombardi Comprehensive Cancer Center. Dr. Adams-Campbell is an epidemiologist who
51 specializes in community health research, interventions, and outreach and is a current member of
52 the Institute of Medicine of the National Academies. She has played a leading role in the
53 Washington, DC cancer and public health communities. Her research focuses on energy balance,
54 diet and exercise. Dr. Adams-Campbell has participated in and led several large cohort studies of
55 African-American women, and she played a leading role in bringing the Boston University Black
56 Women's Health Study to the District of Columbia—the largest study of African-American
57 women.

58
59 **Cheryl Anderson, PhD, MPH:** *Associate Professor of Preventive Medicine, Department of*
60 *Family and Preventive Medicine, School of Medicine, University of California, San Diego, La*
61 *Jolla, Calif.* Dr. Anderson also is an Adjunct Associate Professor, Department of Epidemiology
62 at the Bloomberg School of Public Health, Johns Hopkins University. Her research expertise
63 includes evaluating the role of nutritional factors in chronic disease prevention in minority and
64 underserved populations, with emphasis on the role of dietary sodium and potassium intake in
65 cardiovascular disease prevention. Dr. Anderson currently serves as a member of the IOM Food
66 and Nutrition Board and has served on several other IOM committees including the 2013 IOM
67 Committee on the Consequences of Sodium Reduction in Populations.

68
69 **J. Thomas Brenna, PhD:** *Professor of Human Nutrition, of Chemistry and Chemical Biology,*
70 *and of Food Science, Cornell University, Ithaca, NY.* Dr. Brenna also is an Adjunct Professor,
71 Department of Public Health Sciences at the University of Rochester College of Medicine and
72 Dentistry. He is an expert in the field of fatty acid and lipid metabolism and in food fatty acid
73 composition. His research focuses on the role of polyunsaturated fatty acids throughout the life
74 cycle, in particular the effect of intake during pregnancy and lactation on fetal and infant
75 development. Dr. Brenna has served as a panelist and author for the Expert Consultancy on Fats
76 and Fatty Acids in Human Nutrition for the Food and Agriculture Organization and the World
77 Health Organization.

78

79 **Wayne Campbell, PhD:** *Professor, Department of Nutrition Science, Purdue University, West*
 80 *Lafayette, IN.* Dr. Campbell also is an Adjunct Faculty in the Department of Health and
 81 Kinesiology, Purdue University. He is the Director of the Indiana Clinical Research Center at
 82 Purdue, which is a component of the NIH-supported Indiana Clinical and Translational Science
 83 Institute at the Indiana University School of Medicine. Dr. Campbell's expertise includes
 84 determining the dietary protein requirements of old and very old adults and evaluating the effects
 85 of protein, carbohydrate, and energy intakes and exercise training on macronutrient metabolism,
 86 body composition, and muscle strength and function. In addition, his research endeavors include
 87 studying the effects of food form, portion size, and dietary patterning on appetite and weight
 88 control with a special emphasis on the aging population.

89
 90 **Steven Clinton, MD, PhD:** *John B. and Jane T. McCoy Chair of Cancer Research, The Ohio*
 91 *State University Comprehensive Cancer Center, and Professor, Division of Medical Oncology,*
 92 *Department of Internal Medicine, The Ohio State University School of Medicine, Columbus, OH.*
 93 Dr. Clinton also holds appointments in the Department of Human Nutrition in the College of
 94 Education and Human Ecology and in the Division of Environmental Health Sciences in the
 95 College of Public Health. He is a physician-scientist who has devoted his career to research in
 96 cancer etiology and prevention. Dr. Clinton's research focuses on epidemiology, clinical trials,
 97 community research, and experimental models, as well as cell and molecular systems. He has
 98 published extensively on the role of dietary energy balance and obesity in cancer risk, on a
 99 variety of foods associated with cancer prevention properties, as well as on several nutrients
 100 including vitamin D, calcium, omega-3 fatty acids, and vitamin E. He served on the IOM
 101 Committee on Dietary Reference Intakes for Vitamin D and Calcium.

102
 103 **Frank Hu, MD, PhD, MPH:** *Director, Harvard Transdisciplinary Research in Energetics and*
 104 *Cancer Center, Department of Nutrition, Harvard School of Public Health, Boston, MA.* Dr. Hu
 105 also serves as Director, Boston Nutrition and Obesity Research Center Epidemiology and
 106 Genetics Core, a Professor of Nutrition and Epidemiology at the Harvard School of Public
 107 Health, and a Professor of Medicine at Harvard Medical School and Channing Division of
 108 Network Medicine, Brigham and Women's Hospital. Dr. Hu is an epidemiologist and an expert
 109 in the areas of dietary and lifestyle determinants of obesity, type 2 diabetes, and cardiovascular
 110 disease. He is the principal investigator for the diabetes component of the Nurses' Health Study.
 111 Dr. Hu has served as an academic leader in a variety of roles, including on the National Heart,
 112 Lung, and Blood Institute Obesity Guidelines Expert Panel and the IOM Committee on
 113 Preventing the Global Epidemic of Cardiovascular Disease.

114
 115 **Miriam Nelson, PhD:** *Associate Dean, Jonathan M. Tisch College of Citizenship and Public*
 116 *Service, Tufts University, Boston, MA.* Dr. Nelson also is a Professor in the Friedman School of
 117 Nutrition Science and Policy. Dr. Nelson is an expert on nutrition and physical activity, with
 118 extensive research experience integrating the science of energy balance into national-scale

119 approaches. Her work combines civic engagement, public policy, communications, and systems
 120 thinking to create change. Dr. Nelson is Founder of the Strong Women Initiative and Co-Founder
 121 of ChildObesity180 at Tufts University. Dr. Nelson served as Vice Chair of the Physical Activity
 122 Guidelines Advisory Committee in 2008 and was a member of the 2010 Dietary Guidelines
 123 Advisory Committee.

124

125 **Marian Neuhouser, PhD, RD:** *Full Member, Cancer Prevention Program, Division of Public*
 126 *Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA.* Dr. Neuhouser also is
 127 an Affiliate Professor in the Department of Epidemiology and Core Faculty in the Graduate
 128 Program in Nutritional Sciences, School of Public Health, University of Washington. Dr.
 129 Neuhouser is a nutritional epidemiologist with broad experience in large clinical trials, including
 130 the Women's Health Initiative and the Prostate Cancer Prevention Trial, small-scale controlled
 131 dietary interventions, and large observational cohorts. She has expertise in the role of numerous
 132 dietary components in cancer risk, including carbohydrates, dietary fiber, and vitamin D. Her
 133 research focuses on methods to improve diet and physical activity assessment, diet and physical
 134 activity in relation to energy balance, diet-related health disparities, and dietary factors related to
 135 breast and prostate cancer prevention and survivorship.

136

137 **Rafael Pérez-Escamilla, PhD:** *Professor of Epidemiology and Public Health, Yale School of*
 138 *Public Health, New Haven, CT.* Dr. Pérez-Escamilla also serves as Director, Office of Public
 139 Health Practice and the Global Health Concentration at the Yale School of Public Health. He is
 140 an internationally recognized scholar in the area of community nutrition. Dr. Pérez-Escamilla has
 141 specialized experience with Hispanic and low-income Americans, as well as populations in low
 142 and middle income countries. His research program seeks to understand how best to protect,
 143 promote, and support breastfeeding, causes and consequences of food insecurity, and how to
 144 improve diabetes self-management through community health workers. Dr. Pérez-Escamilla has
 145 published numerous articles that have led to improvements in breastfeeding outcomes, iron
 146 deficiency anemia among infants, household food security measurement, and community
 147 nutrition education programs worldwide. He is past-chair of the Global Nutrition Council of the
 148 American Society for Nutrition and is a member of the IOM Food and Nutrition Board.
 149 Previously, Dr. Pérez-Escamilla served as a member of the IOM Committee to Re-examine IOM
 150 Pregnancy Weight Guidelines and was a member of the 2010 Dietary Guidelines Advisory
 151 Committee.

152

153 **Anna Maria Siega-Riz, PhD, RD:** *Associate Dean for Academic Affairs and Professor,*
 154 *Departments of Epidemiology and Nutrition, Gillings School of Global Public Health, University*
 155 *of North Carolina at Chapel Hill, Chapel Hill, NC.* Dr. Siega-Riz serves as the Program Leader
 156 for the Reproductive, Perinatal, and Pediatric Program in the Department of Epidemiology. Dr.
 157 Siega-Riz has focused her research on maternal nutritional status, including maternal obesity and
 158 gestational weight gain and their effect on birth outcomes as well as the determinants of early

159 childhood obesity. She studies dietary patterns among Hispanic adults and children, in general,
160 and served on the Scientific Advisory Panel for the Feeding Infants and Toddlers Study. Dr.
161 Siega-Riz has served on multiple committees for the IOM, examining topics from the WIC food
162 packages to standards for systematic reviews in health care and currently serves on the advisory
163 council of the National Heart, Lung, and Blood Institute.

164

165 **Mary Story, PhD, RD:** *Professor, Community and Family Medicine and Global Health, Duke*
166 *University, Durham, NC.* Before coming to Duke in January 2014 she was Senior Associate
167 Dean for Academic and Student Affairs and Professor in the Division of Epidemiology and
168 Community Health in the School of Public Health, University of Minnesota. Dr. Story
169 concurrently serves as Director of the National Program Office for the Robert Wood Johnson
170 Foundation Healthy Eating Research Program that supports research on environmental and
171 policy strategies to promote healthy eating among children to prevent childhood obesity. She has
172 conducted numerous school and community-based environmental intervention and obesity
173 prevention studies for children, adolescents, and families. Dr. Story was elected to the IOM in
174 2010 and is currently a member of the IOM Food and Nutrition Board and vice co-chair of the
175 IOM Roundtable on Obesity Solutions.

176

177 **Consultant Subcommittee Members to the 2015 DGAC**

178 **Timothy S. Griffin, PhD:** *Director, Agriculture and Environment Program, Friedman School of*
179 *Nutrition Science and Policy, Tufts University, Boston, MA.* Dr. Griffin also is an Associate
180 Professor at Tufts University where he serves on the Water: Systems, Science and Society
181 faculty steering committee and is a Faculty Co-Director for the Tufts Institute for the
182 Environment. His research expertise and interests include the intersection of agriculture and the
183 environment, and the development and implementation of sustainable production systems.
184 Previously he worked as a Research Agronomist and Lead Scientist with USDA-ARS New
185 England Plant Soil and Water Lab, and as Extension Sustainable Agriculture Specialist at the
186 University of Maine.

187

188 **Michael W. Hamm, PhD:** *Director, Center for Regional Food Systems, Michigan State*
189 *University, East Lansing, MI.* Dr. Hamm is also the C.S. Mott Professor of Sustainable
190 Agriculture in the Department of Community Sustainability in the College of Agriculture and
191 Natural Resources and has appointments in the Department of Food Science Human Nutrition
192 and the Department of Plant, Soil and Microbial Sciences at Michigan State University. His
193 research expertise and interests include regional and sustainable food systems and food security.

194

195 **Michael G. Perri, PhD, ABPP:** *Dean, College of Public Health and Health Professions*
196 *University of Florida; Gainesville, FL.* Dr. Perri is also the Robert G. Frank Endowed Professor
197 of Clinical and Health Psychology. His research focuses on health promotion and disease
198 prevention through changes in diet and physical activity. His NIH-funded studies involve the
199 translation, dissemination, and implementation of effective programs for the management of
200 obesity in underserved rural communities. Dr. Perri has served as a member on NIH data and
201 safety monitoring boards, including serving as chair of the recent NIH/NHLBI Data and Safety
202 Monitoring Board for the “EARLY Weight Loss Trials.”

203

Appendix E-9: Work Structure and Member Organization

Work Group Structure

Work structure from inception through fall 2013.

Work Group 1: Environmental Determinants of Food, Diet, and Health

Miriam Nelson (Lead)

Steven Abrams

Lucile Adams-Campbell

Mary Story

Work Group 2: Dietary Patterns and Quality and Optimization through Lifestyle Behavior Change

Rafael Pérez-Escamilla (Lead)

Cheryl Anderson

Gary Foster*

Frank Hu

Anna Maria Siega-Riz

Work Group 3: Foods, Beverages, and Nutrients and Their Impact on Health Outcomes

Alice H. Lichtenstein (Lead)

J. Thomas Brenna

Wayne Campbell

Steven Clinton

Marian Neuhouser

* Dr. Gary Foster assumed a new position shortly after being appointed as a member of the 2015 DGAC. Due to the significant demands of the new position, it became necessary for Dr. Foster to resign his appointment to the 2015 DGAC (August 2013).

Subcommittee Structure

Work structure from fall 2013 through completion of the report.

Science Review Subcommittee *(In place from inception through completion of the report.)*

Barbara Millen (Chair)
Alice H. Lichtenstein (Vice Chair)
Miriam Nelson (2010 and 2015 DGAC member)
Rafael Pérez-Escamilla (2010 and 2015 DGAC member)

SC 1: Food and Nutrient Intakes, and Health: Current Status and Trends

Marian Neuhouser (Chair)
Alice H. Lichtenstein (Chair/Vice Chair Representative)
Steven Abrams
Cheryl Anderson
Mary Story

SC 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes

Anna Maria Siega-Riz (Chair)
Alice H. Lichtenstein (Chair/Vice Chair Representative)
Cheryl Anderson
Tom Brenna
Steven Clinton
Frank Hu
Rafael Pérez-Escamilla
Marian Neuhouser

SC 3: Diet and Physical Activity Behavior Change

Rafael Pérez-Escamilla (Chair)
Barbara Millen (Chair/Vice Chair Representative)
Wayne Campbell
Steven Clinton
Anna Maria Siega-Riz
Lucile Adams-Campbell
Michael Perri (Consultant)

SC 4: Food and Physical Activity Environments

Mary Story (Chair)

Barbara Millen (Chair/Vice Chair Representative)

Lucile Adams-Campbell

Wayne Campbell

Mim Nelson

SC 5: Food Sustainability and Safety

Mim Nelson (Chair)

Barbara Millen (Chair/Vice Chair Representative)

Steven Abrams

Tom Brenna

Frank Hu

Michael Hamm (Consultant)

Tim Griffin (Consultant)

Working Group Structure

Work structure developed as need identified from spring 2014 through completion of report.

Added Sugars Working Group

Miriam Nelson (Co-Lead)

Mary Story (Co-Lead)

Cheryl Anderson

Wayne Campbell

Frank Hu

Alice H. Lichtenstein

Barbara Millen

Marian Neuhouser

Sodium Working Group

Cheryl Anderson (Lead)

Wayne Campbell

Steven Clinton

Alice H. Lichtenstein

Saturated Fat Working Group

Frank Hu (Lead)

Tom Brenna

Alice H. Lichtenstein

Barbara Millen

Physical Activity Writing Group

Miriam Nelson (Lead)

Wayne Campbell

Alice H. Lichtenstein

Appendix E-10: Dietary Guidelines Advisory Committee Report Acknowledgments

Invited Expert Speakers

Steven Abrams, MD	Alanna Moshfegh, MS, RD
Dawn Alley, PhD	Suzanne Murphy, PhD, RD
Sonia Angell, MD, MPH	Catherine Oakar, MPH
Amelia Arria, PhD	Rafael Pérez-Escamilla, PhD
Robert Brackett, PhD	Barry M. Popkin, Ph.D
Laurel Bryant,	John Ruff, MA
Kathryn B. H. Clancy, PhD	Michael B. Rust, PhD
William H. Dietz, MD, PhD	Jill Reedy PhD, MPH, RD
Linda Duffy, PhD, MPH	Donna H. Ryan, MD
Robert H. Eckel, MD	Marie-Pierre St-Onge, PhD, FAHA
Lorraine Gunzerath, PhD, MBA	Pam Starke-Reed, PhD
Van Hubbard, MD, PhD	Patrick Stover, PhD
Susan M. Krebs-Smith, PhD, MPH, RD	Deborah F. Tate, PhD
Antonia Mattia, PhD	Katherine Tucker, PhD
J. Michael McGinnis, MD, MA, MPP	Connie M. Weaver, PhD
Kathleen Merrigan, PhD	CAPT Andrew Zajac

Staff, Contract, and/or Technical Support

Nadine S. Braunstein, PhD, RD	Olga Nelson
Eileen Dykes, MS, RD	Andrea Popp
Janet East	Cikena Reid
Janie Fleming	Ronnie Rogers
Yolande Gary	Ken Ryland
Vibhuti Giltrap	India Taylor
Joseph Goldman, MA	Jennifer Wilkinson
Hazel Hiza, PhD, RDN	Miyuki Shimizu, PhD Candidate
Joy Jackson Farrar	Jeff Steele
Ashlee Johnson	Teresa T. Fung, ScD, RD

National Service Volunteer Evidence Abstractors

Brian R. Barrows, PhD
Samantha Berger, MS, MPH
Cynthia Blanton, PhD, RD
Stacy Blondin, MSPH
Eric Calloway, PhD
Sheau Ching Chai, PhD, RD
Sarah Forrestal, PhD
Carol C. Giesecke, PhD, RD, LDN
Maryam S. Hamidi, MSc, PhD
Heidi L. Himler, MPH, RD, LD
Tiffany Huang, MS
Sarah A. Johnson, PhD, RD, CSO
J. Philip Karl MS, RD
Lydia Kaume Ph.D

Alexandra Kazaks, PhD, RDN
Kathryn Lawson, MS, RDN, CD
Annie W. Lin, MS, RD, CDN
Dalia Majumdar, PhD
Melissa A. Masters, PhD, RDN
Kevin Pietro, MS, RD, LDN
Maja Redzic, MS
Amy Steffey, DVM, MPH
Maria L. Stewart, PhD
Libo Tan, PhD
Alison K. Ventura, PhD
Ding Ding Wang, MPH
Marquitta C. Webb, Ph.D., LN
Sahar Zaghoul, MBBS, PhD