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Diet Quality

An Evidence-Based Approach

Volume 1

Humana Press

Nutrition and Health Series

Adrianne Bendich, PhD, FACN, FASN, Series Editor

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An Evidence-Based Approach, Volume 1

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Preface

It is well known that nutritional well-being relates to the ingestion of specific micro- and macronutrients. The optimum intakes of these nutrients are subjected to national or international guidelines such as the Dietary Reference Values or the Recommended Daily Allowances. However there is an increasing awareness that well-being is also associated with the qualitative nature of diets.

Diet quality is a broad term that encapsulates both perceived and actual practices, personal preferences, and cultural diversity. Its imprecision contrasts sharply with information derived from quantitative measures of micro- and macronutrients intakes or status. For quantitative measures, the analytical tools range from dietary recall studies and weighed intakes to laboratory measures of circulating nutrients in blood or clinical signs of disease. Measuring dietary quality is more problematic and includes investigating food types, the number or size of portions or their frequency. Diet quality may also be related to the type of food being ingested, snacking, and other eating habits. The inclusions of manufactured beverages (carbonated drinks) or take-away meals may also be included within the measures of diet quality, albeit as a negative measure. It may also include microbiological quality and detailed composition of single food items such as meats, pulses, or vegetables. However, hitherto finding out information on food quality and diet has been fragmentary.

In this book *Diet Quality*: *An Evidence-Based Approach* we cover all of the major facets of diet quality. The book adopts a holistic approach to diet quality and imparts information on the major areas of concern or knowledge. Chapters link in measurable indices of health such as obesity, cardiovascular disease, pregnancy outcomes, mortality, diseases of organs, and cancer.

Contributors are authors of international and national standing, leaders in the field and trendsetters. Emerging fields of science and important discoveries are also incorporated in *Diet Quality*: *An Evidence-Based Approach*.

This book is designed for nutritionists and dietitians, public health scientists, doctors, epidemiologists, healthcare professionals of various disciplines, policy makers, and marketing and economic strategists. It is suitable for teachers and lecturers, undergraduates and graduates, researchers and professors.

London, UK — Victor R. Preedy
Maidenhead, UK — Lan-Anh Hunter
London, UK — Vinood B. Patel

Series Editor Page

The great success of the Nutrition and Health Series is the result of the consistent overriding mission of providing health professionals with texts that are essential because each includes (1) a synthesis of the state of the science, (2) timely, in-depth reviews by the leading researchers in their respective fields, (3) extensive, up-to-date fully annotated reference lists, (4) a detailed index, (5) relevant tables and figures, (6) identification of paradigm shifts and the consequences, (7) virtually no overlap of information between chapters, but targeted, inter-chapter referrals, (8) suggestions of areas for future research, and (9) balanced, data-driven answers to patient as well as health professionals questions which are based upon the totality of evidence rather than the findings of any single study.

The Series volumes are not the outcome of a symposium. Rather, each editor has the potential to examine a chosen area with a broad perspective, both in subject matter as well as in the choice of chapter authors. The editor(s), whose trainings are both research and practice oriented, have the opportunity to develop a primary objective for their book, define the scope and focus, and then invite the leading authorities to be part of their initiative. The authors are encouraged to provide an overview of the field, discuss their own research, and relate the research findings to potential human health consequences. Because each book is developed de novo, the chapters are coordinated so that the resulting volume imparts greater knowledge than the sum of the information contained in the individual chapters.

"Diet Quality: An Evidence-Based Approach," edited by Professor Victor R. Preedy, PhD, DSc, FRSPH, FRSC, FSB, FRCPath, Dr. Lan-Anh Hunter, BSc, MBBS, MRCGP, and Vinood B. Patel, PhD clearly exemplifies the goals of the Nutrition and Health Series. The major objective of this comprehensive two volume text is to review the body of research on the growing importance of the role of diet quality in assuring adequate intake of the essential nutrients, sufficient calories to maintain normal weight, and improve indices of good health. The book includes 50 up-to-date informative reviews of the current thinking about the role of diet quality in the health of individuals during different stages of life, determinants of diet quality around the world and examines the consequences of diet quality on diseases, public health, and international nutrition program development. Practicing health professionals, researchers, and academicians can rely on the chapters in this volume for objective data-driven sources about essential vitamins and minerals, proteins and fats as well as other dietary components that have been determined to be required in healthy diets. This new comprehensive review of the science behind the assessment of diet quality is of great importance to the nutrition community as well as for health professionals who have to answer patient, client, or graduate student questions about the newest clinical research on the nutritional interactions of foods and the health consequences.

"Diet Quality: An Evidence-Based Approach" represents the most comprehensive compilation of the recent data on the critical drivers of food consumption independent of the nutrient density of the foods being consumed. It is to the credit of Drs. Preedy, Hunter, and Patel that they have organized this volume so that it provides an in-depth overview of the critical issues involved in the determination of the best nutritional strategies for infants, toddlers, school-age children, and adult populations whether they were born in developing nations or in developed nations. The volumes' editors provide their in-depth knowledge and expertise to help the reader to understand the value of diet quality in the development of national dietary recommendations. Professor Preedy is a senior member of King's College London where he is a Professor of Nutritional Biochemistry and is also a Professor of Clinical Biochemistry at King's College Hospital. He is also Director of the Genomics Centre and a member of the School of Medicine. He is a member of The Royal College of Pathologists, a Fellow of the Society of Biology, The Royal College of Pathologists, The Royal Society for the Promotion of Health, The Royal Institute of Public Health, The Royal Society for Public Health and in 2012 a Fellow of the Royal Society of Chemistry. Dr. Hunter received her medical degree from Guys, King's & St Thomas' Medical School, London. Her research focuses on the psychological and cultural aspects of diet, nutrition, and anorexia. She also has a background in diabetes research. Dr. Patel is a Senior Lecturer in Clinical Biochemistry at the University of Westminster and honorary fellow at King's College London. Dr. Patel obtained his degree in Pharmacology from the University of Portsmouth, his PhD in protein metabolism from King's College London and completed postdoctoral research at Wake Forest University School of Medicine.

Each of the two volumes contains about 25 comprehensive chapters. The first volume contains four related parts. The first part provides an overview and perspective on national and international diet quality scoring systems and the potential nutritional and health benefits of adopting high quality diets. The quality of diets consumed at home, in the military, hospitals, or other institutions can be dependent upon the costs associated with diets of different levels of quality. Health economics can help to determine the cost-effectiveness of dietary choices. The four chapters in the first part examine the complexities of developing standards and scoring systems, initiatives to determine the science behind food preferences, and use examples of successful and not so successful dietary programs which focus on health as well as cost-effectiveness. The first chapter presents a comprehensive review and tabulation of the current diet quality indexes including, but not limited to the Healthy Eating Index (HEI), Diet Quality Index (DQI), Healthy Diet Indicator (HDI),the Mediterranean Diet Score (MDS), the Diet Quality Index-International (DQI-I), and the Food-Based Quality Index (FBQI). The second chapter examines the biological drive to eat and the complex neural and hormonal systems that control food consumption. The factors affecting the selection of one food over another, including food availability, social and cultural norms, economic, biologic, and cognitive elements, are reviewed. Food preferences are examined including both the learned and innate and those stemming from environmental, psychosocial, biologic, and genetic influences. The environmental and psychosocial elements influencing food choice and preference includes food's perceived value, culture, religion, cost, access, and exposure to food advertisements are discussed. Several of the assessment tools to quantify food intake and preference including food frequency questionnaires, hedonic food scales, feeding studies, food records, and open-ended questionnaires are also reviewed.

The next unique chapter examines the interaction between emotional attachments between parents and children and the quality of foods consumed in the home compared to meals eaten outside the home. The authors take an evidence-based approach to examine the underlying mechanisms contributing to the protective effects of the home on healthy eating. It is widely observed that the nutritional quality of home consumed meals is healthier than in away-from-home settings. The authors present the results of their recent studies that found that the more anxious the children and parents, the poorer the diet quality. The last chapter in this part reviews the literature that examines the cost-effectiveness of healthy eating and describes the association seen between lowered healthcare costs and improved diet quality using data from studies in Australia.

The second part looks at the importance of diet quality for men and women of reproductive age, during pregnancy and in the growth of children. The first two chapters clearly indicate that the effects of maternal diet, as a composite measure of total diet quality on pregnancy outcomes have only recently been studied. There is an overall review of the nutritional requirements of pregnancy that includes a discussion of recent data on dietary fatty acid and folate requirements using the Mediterranean diet as an example. There are important tables and figures that summarize the evidence showing that dietary intake (and diet quality as determined using any of the three quality indexes that have a pregnancy component), influences pregnancy outcomes such as fetal growth, preterm delivery, or neural tube defects. However, the totality of epidemiological evidence regarding the association of diet quality and pregnancy outcomes is weak overall; nonetheless it is suggestive of a beneficial effect of a high quality diet on pregnancy outcomes even in populations that are assumed to be well nourished. Of importance, a study in the USA found that a maternal diet of high quality as measured by the DQI and the MDS during the preconceptional period was associated with a reduced risk of neural tube birth defects.

The importance of maternal choices of foods for their offspring is reviewed in the next chapter. There is evidence that the foods consumed in the first 2 years of life can determine a child's food preferences until adulthood. Poor dietary quality during childhood has been associated with micronutrient deficiencies, overweight, and cardiovascular disease risk factors. The chapter discusses the formation of feeding habits in early childhood, the importance and influence of nutritional programs and guidance for mothers and the impact of diet quality on children's health. Several studies on educational interventions with mothers are reviewed and provide evidence about the positive effects of such interventions on children's diet quality and nutritional conditions. The final chapter in this part examines the diet quality of women from Africa, Asia, and Latin America and concludes that from a biological standpoint as well as a cultural one, women are at greater risk for micronutrient deficiencies than men and that this equates into a number of adverse health outcomes for women and their offspring.

The third part of this volume contains eight chapters that describe the diet quality of children and seniors. Currently, there are no studies that are specifically designed to look at diet quality in children and/or adolescents. The intervention studies that have been done are reviewed and suggest modest improvements in fruit intakes from school-based intervention programs. Two studies from Norway are described that provided a free fruit to school-age children in controlled settings. Free fruit provided over a year resulted in increased fruit intake after the program concluded and reduced consumption of unhealthy snacks. In the next chapter, studies of dietary patterns and their associations with growth and weight parameters in children and adolescents are examined. Using the Youth HEI scores of over 16,000 children and teens, it was reported that children with higher diet quality had lower body mass indexes. Other studies reviewed showed that lower percent body/abdominal fat in children was associated with higher diet quality and conversely, childhood obesity prevalence decreased significantly with increasing diet quality. Diet quality can be significantly influenced by nonfood components.

The next chapter summarizes the data linking TV viewing in children and video game playing in adolescents with lowered diet quality. There is even a large, WHO collaborative cross-national study that was conducted in a number of European countries and regions, and also in the USA, Canada, and Israel. A common identical questionnaire was used. Increased TV viewing time was statistically significantly associated with higher rates of daily consumption of sugared drinks and sweets and was significantly negatively associated with the consumption of vegetables and fruit indicating a common non-healthy diet quality pattern related to TV viewing. The next chapter examines the complex role of parents in the diet quality of their children. As an example, diet quality is negatively associated with not eating regular family meals and the frequent consumption of fast food and meals in front of the television. Unfortunately, there is limited research on parental perceptions of their children's eating behaviors and diet quality, with only one study of the direct comparison between maternal perceptions of the child's diet quality with the actual quality of the child's diet.

Many parents rely on convenience foods, defined in this chapter as foods where preparation has occurred outside the home and the foods are consumed in the home, to complement their children's meal. Use of convenience foods is growing and is often initiated in the second half of infancy and continues through adolescence. Recent research suggests that there is an inverse relationship between convenience food consumption and diet quality; however, producers are reducing salt and fat content to improve the convenience food's quality profile. Changing dietary habits is challenging, as documented in the next chapter that describes an intervention study in high school students in Spain. There were significant, but small, increases in fruit consumption and reductions in body weight in this intensive educational program. Recommendations for follow-up studies include initiation in elementary grades and a more prolonged program of at least 1 year to better reinforce new dietary habits. The last chapter in this part reviews the rationales behind the development of dietary recommended intakes for individuals 70 years old and older in the USA and Canada. The recommendations include an increase in the nutrient density of foods consumed as caloric intake decreases with aging, yet essential nutrient requirements either stay the same or increase. Additionally, there are decreased levels of physical activity, decreased metabolic rates, and increased proportions of fat to lean muscle mass, as well as a greater risk of chronic degenerative diseases and use of many drugs that can affect energy requirements and appetite.

The fourth part, entitled "Foods and Dietary Components" contains five chapters that examine key nutrients such as salt, sweet potatoes, meats, orange juice, and whole grains and how these individual dietary components can alter the quality of diets consumed in different parts of the world. The chapter that describes the beneficial effects of the Mediterranean diet on blood pressure examines current data on the higher than expected salt intake of children and adolescents who are consuming this diet by including a number of processed foods, such as bread, pizza, and cheeses that are high in salt. The authors warn that the beneficial effects on blood pressure may be lost for the next generation if salt concentrations in processed foods are not reduced. The next four chapters describe nutrient sources that can enhance the overall diet quality for populations that may be at risk for nutrient deficiencies. The sweet potato is not one plant, but many different varieties that provide valuable nutrients shown to have health benefits. Sweet potatoes are the seventh most commonly consumed carbohydrate-rich food source in the world. Varieties have been bred that contain higher concentrations of phenolic compounds, β-carotene, anthocyanins, and proteins called sporamins. Health benefits associated with sweet potato consumption include reductions in risk of diabetes, cancer and hypertension, reduction in vitamin A deficiency, improving hepatic functions, and enhanced memory.

Meat, discussed in the next chapter, is an important source of high quality protein, providing all the essential amino acids needed for growth and development. Meat is also an excellent source of essential micronutrients (vitamins, minerals, and trace elements), which are often limited in the diet, including vitamin A, iron, and zinc. Cooking methods and their effects on the nutritional quality of meats are reviewed in this detailed, well-referenced chapter. Another important food that is consumed globally and has high nutrient density is orange juice. Brazil is South America's largest producer of orange juice and orange derivatives. The second largest producer is the USA, followed by China, Costa Rica, Mexico, and the Mediterranean region. Orange juice is a major contributor to vitamin C intake for children and also contains high quantities of flavonoids and carotenoids. Whole grains are also important contributors to diet quality. In addition to fiber and protein, whole grains are rich sources of B vitamins and vitamin E, minerals including iron, magnesium, potassium, zinc and selenium, beta-glucan, inulin, numerous phytochemicals, phytosterols, phytin, and sphingolipids. Of importance, as described in the final chapter in this part, these compounds exert an additive and/or synergistic effect on health when consumed together. Individuals who consume diets containing higher than average intakes of whole grains have significantly reduced risk of cardiovascular disease and diabetes. Dietary guidelines from nations around the world are tabulated and consistently recommend the daily consumption of whole grains.

The second volume of "Diet Quality: An Evidence-Based Approach" also contains four related parts. The first part examines the data linking diseases and health conditions that are associated with aspects of diet quality. The six chapters in this part review the associations between diet quality and body weight, strokes, menstrual irregularities and pain, breast cancer and nonalcoholic fatty liver disease (NAFLD). Obesity is a major global problem and there are a number of studies that have examined the association between diet quality and body mass index (BMI). Overall, as reviewed in the first chapter in this part, there is a consistent inverse association between the healthiness of dietary intake and prospective weight gain. Poor diet quality is also strongly associated with increased cardiovascular and cerebrovascular diseases. The next chapter describes the detailed data that corroborates the association of low diet quality with significant increased risk of stroke in the male Korean population. C-reactive protein is an accepted biomarker of inflammation associated with increased cardiovascular risk. Preliminary data, summarized in a unique chapter, indicates that C-reactive protein can also serve as an indicator of a poorer diet in children. One nutrient that has been linked to cardiovascular health is calcium. Low calcium intakes, often seen in children and young women, have been associated with increased blood pressure and newer research has also linked low calcium intakes with menstrual pain that may be linked to blood vessel and/or muscle contractions. This chapter examines the potential for numerous nutrients to reduce prostaglandin secretions from uterine tissue that is considered the major source of menstrual pain. Diets that are rich in antioxidants and anti-inflammatory nutrients have been associated with reduced pain during menstruation.

Breast cancer is the leading cancer to affect women and is related to genetic as well as environmental components including dietary factors. Several of the diet quality indexes have been used to determine if dietary factors are predictive of breast cancer incidence, progression, or mortality. The data, tabulated and reviewed, point out the lack of consistent findings between studies that may be in part due to the strong genetic component of this disease. The next chapter describes a relatively new liver disease, NAFLD. The majority of NAFLD patients have metabolic risk factors such as visceral obesity, type II diabetes, and dyslipidemia. As the prevalence of metabolic syndrome, obesity and diabetes continue to increase globally, the prevalence of NAFLD has increased. This very serious disease is mainly treated with weight loss diets and/or surgery. Diet quality, including the composition of the macronutrients in the diet of NAFLD patients may help to reduce the adverse effects of hepatic fat and inflammation. A diet that is low in carbohydrates and saturated fat and high in fiber and omega-3 fatty acids may be beneficial.

As indicated earlier, there are many critical factors that determine an individual's diet quality. The five chapters in the next part examine the roles of eating behavior and psychology on food choices. Survey data suggest that certain behavioral factors can improve diet quality. As an example, the next chapter reviews the association between consuming meals in a family setting vs. eating alone. Family meal frequency is associated with more healthy food intake and behaviors, including more fruits and vegetables, whole grains, calcium rich foods, and protein; less-fried food and sugar-sweetened beverages and more micronutrients from food. The next chapter discusses the motivations for eating. Motivations to eat are related to weight status and predict food intake. Environmental motivations to eat include eating in response to the environment, food availability, and social influences; biological cues including taste and hunger, and psychological factors such as emotional eating and dietary restraint. Obese individuals have less availability of healthy foods, eat in response to environmental cues, and are more comfortable with their weight when surrounded by obese peers. Perceptions of taste and hunger are associated with weight status. Greater self-efficacy and dietary restraint, and reduced vulnerability to eating in response to emotional states are linked to a healthier weight status. Another chapter examines the importance of breakfast to diet quality. A nutrient dense breakfast influences the composition of subsequent meals, regulates energy intake, boosts physical activity levels, and provides a crucial source of dietary micronutrients. The benefits are consistently observed in the USA and around the world when the breakfast includes fortified ready-to-eat cereals. Consistent cereal consumption has been shown to predict improvements in body composition, including BMI in children, adolescents, and adults.

Most animals have biological controls that determine their normal food intake and obesity is not an issue. In contrast, in humans, non-homeostatic or psychological factors may play a more important role in food selection and energy intake than do homeostatic or biological factors. Foods that are high in energy density are often preferred and more reinforcing, but these foods are typically lower in nutrient density. Finding ways to reduce motivation to eat unhealthier food while increasing motivation to eat healthier food to improve diet quality and reduce obesity is the major focus of the next chapter. The final chapter in this part confirms the association between fast food consumption and reduced diet quality. Studies in the USA showed that fast food consumers have a lower overall dietary intake quality score, higher intake of energy, fat, saturated fat, sodium and soft drinks, and lower intake of vitamins A and C, milk, fruits and vegetables than those who did not reported eating fast foods. In addition, a growing number of studies showed an association between fast food consumption and obesity in children and adults.

The third part of this volume examines, from and international perspective, the public health initiatives that have been undertaken by governments to improve the diet quality of their citizens. The ten chapters review recommendations from Germany, Norway, the USA and Puerto Rico, Arabs in Israel, developing countries, sub-Saharan Africa and South Africa specifically. In the first chapter, we learn that in Germany, the German Nutrition Society establishes the nation's dietary recommendations. Two graphical representations are used. The Nutrition Circle as a concrete representation of quantitative food-based dietary guidelines for adults representing the quantity relationships of different food groups in a balanced diet. The 3D Food Guide Pyramid with four triangle sides is used to illustrate qualitative recommendations for adequate food choices within four major food groups: plant foods, animal foods, fat and oil as well as beverages. There is an interesting chapter that compares the dietary components of the Nordic vs. Mediterranean diets. The Nordic climate does not support the growth of a great variety of fresh vegetables, olives and citrus fruits, but does include plant-derived foods including root vegetables, berries, cabbages, plums, apples, and pears. Rye, oats, barley, and rapeseed oil are also readily available and the Nordic waters contain fish that are rich in long-chain omega-3 fatty acids. These foods are regarded as traditional Nordic foods with favorable health effects.

For over a century, the US Department of Agriculture has issued dietary recommendations for its population. The history of these recommendations and the rationale for the foods that are recommended are reviewed in several chapters. Awareness of these guidelines is also examined in a separate chapter. Awareness of US dietary guidelines has varied by sex, age, race-ethnicity, income, and education. Over 80 % of adults have heard of at least one of the three sets of federal dietary guidance with the greatest awareness of the Food Guide Pyramid. Only about half had heard of the Dietary Guidelines for Americans or the 5 A Day program. There was a significant linear trend of increasing awareness with increasing education, and a similar significant trend of increasing awareness with increasing income level. The Hispanic population of the USA is growing rapidly and represents the largest minority population in the USA. The next chapter describes the process of dietary acculturation which is defined as a minority group adopting the eating patterns or food choices of the host country. Traditional Hispanic foods are fresh and rich in complex carbohydrates, micronutrients, fiber, and phytochemicals. These foods are being replaced by highly processed foods with higher amounts of salt, sugar, and fat. Thus, food acculturation may not change the percentage of the contribution that a specific food group has on total calorie intake, but it can have an impact on the kind of foods that are selected. A unique chapter describes in detail, using nine tables and figures, the melding of the US dietary guidelines with the foods usually consumed in Puerto Rico, a commonwealth and territory of the USA.

Dietary acculturation of nomadic Arabs into Israeli society has been associated with significant changes in all aspects of daily life including diet quality and increased prevalence of chronic diseases. Among traditionally semi-nomadic Bedouin Arabs, the transition away from whole wheat bread to white bread as the main dietary staple was associated with reductions in diet quality. This chapter confirms that dietary acculturation, in addition to many other changes in lifestyle among Arabs in Israel often negatively affects their diet quality, and may increase the risk for the development of chronic diseases. Determination of diet quality in developing countries and/or developing populations, such as discussed above, can be improved with the use of tools that capture the diversity of the foods consumed. Dietary diversity is defined as the number of foods or food groups consumed, and it is considered as a key dimension of dietary quality. Diverse diets increase the likelihood of adequate intake of essential nutrients. The chapter on diet diversity summarizes new evidence regarding their potential use as a proxy for dietary quality in developing countries. The new studies confirm a positive association between dietary diversity indicators and anthropometric measures in both children and women. As evidence of the importance of diet diversity for measuring diet quality, the next chapter endorses this tool for use in sub-Saharan Africa. Currently, data-driven dietary patterns and dietary diversity may be more appropriate to assess dietary quality in Africa. The dietary transition seen in sub-Saharan Africa reflects globalization of food culture. The chapter describes diet quality of South Africans who have recently moved to cities and uses diet diversity as a tool to determine overall diet quality. In South Africa, the characteristics of malnutrition are changing together with the nutrition transition. In 2007 a health survey found that underweight was uncommon, with less than 5 % of women and 8 % of men with a BMI of <18.5. The survey also found that 50 % of young women and 30 % of young men were either overweight or obese. Research has shown that both hunger and obesity are often present within the same household, especially in low-income households. Higher obesity rates are, furthermore, associated with low-income and education levels and low diet diversity mainly among women.

The final part in the volume includes seven chapters dealing with methods, indexes, and scoring systems and their applications and analysis. The first chapter describes the development of the DQI. The original DQI was developed in the early 1990s for the US population and included food and nutrient-based recommendations. A number of revisions and additions for subpopulations have been developed over time. An adapted version is described that was exclusively based on Food Based Dietary Guidelines (FBDG) and was designed for use in Flemish preschoolers. Although the FBDG-based DQI was designed to compare and monitor dietary changes in populations rather than individuals, it has also served as an effective evaluation tool for intervention studies and clinical trials.

The next chapter describes the development of diet quality indices that reflected the nutritional status of participants in the Framingham Heart Study (FHS) cohorts. The FHS has investigated cardiovascular disease and other health outcomes among Framingham residents in Massachusetts since 1948. A second-generation cohort of 5,124 FHS offspring and their spouses was enrolled in 1971 and comprise the Framingham Offspring/Spouse Study (FOS). Members of the FOS cohort participate in standardized clinical assessments approximately every 4 years. The Framingham Nutrition Studies were initiated in 1984–1988. All men and women completed the Framingham Food Frequency Questionnaire, a single 24-h recall, and a nutrition behavior questionnaire; two-thirds of these participants also completed 3-day dietary records. The Framingham Nutritional Risk Score (FNRS) is a validated 19-nutrient index for evaluating diet quality. The index was developed specifically to assess cardiovascular disease, which is a focus of the FHS. The

FNRS has been shown to predict weight change, overweight and obesity, abdominal obesity, and metabolic syndrome. The FNRS is the only index that has shown long-term stability, further validating the utility of diet quality indices. In addition to the FNRS, there are more than 40 published papers that have examined the diet quality in seniors, mainly in developed nations. The systematic review by Freisling et al. evaluates the major criteria for determining factors that affect the diet quality of the elderly. The chapter reminds us that the elderly population is more heterogeneous in terms of functional capacity, physical conditions, social, economic, and lifestyle situations than any other age group, and individual diversity tends to increase with age. This diversity adds an additional dimension in identifying factors that are associated with diet in older adults.

Consistent findings among studies indicate that more efforts are needed to improve diet among elderly, particularly men, overweight and obese individuals, and those with lower educational levels. Health conditions may directly affect food selection, including impaired oral health and/or gastrointestinal problems. Poor diets tend to be clustered with other unhealthy behaviors such as smoking or being physically inactive.

The Overall Nutritional Quality Index (ONQI™) is another index, designed in 2005, to quantify the nutritional and health consequences of certain diet patterns. The objectives of the index are (1) to develop a (the) definitive algorithm to stratify food items within any category, and across food categories, based on overall nutrition quality; (2) to develop a novel metric for the nutritional quality of individual foods based on the relationship among nutrients in foods, the overall quality of the diet, and associations with health outcomes; (3) to place the ability of top nutrition experts to discriminate among food choices on the basis of nutrition into the hands of every consumer; (4) to provide the public a powerful and empowering means to improve dietary intake patterns, and thereby health, one food choice at a time. The authors indicate that the ONQI™ is the only system with published data demonstrating a direct correlation between score variation, and variation in health outcomes including all-cause mortality. Another important component of diet quality is the actual data capture from individuals. There is a unique chapter that describes the use of newly developed electronic data capture tools especially to affect weight loss. Self-monitoring is a behavior change strategy that is central in behavioral weight loss interventions. The process of self-regulation involves self-monitoring, self-evaluation, and self-reinforcement, where self-monitoring occurs first followed by self-evaluation of progress made toward one's goal and self-reinforcement for the progress made. From a practical perspective, self-monitoring consists of identifying a behavior and recording details about its occurrence. It helps individuals learn when and how often a behavior occurs and the circumstances that surround that behavior and the necessary actions to be taken to change desired behaviors.

Another index has been developed to capture food purchasing and its impact on diet quality. The chapter reviews a system of indicators for the nutritional quality of the marketing and food environments in terms of product quality, availability, affordability, and promotion. Indicators in the system were constructed by observing marketing activities at the stock keeping unit (SKU) level and by geo-coding the stores and media through which the marketing activities occurred. The diagnostic tool assessed the nutritional quality of the food environment created by marketing activities at community, city, provincial/state, national, and global levels. This index has the potential to provide an understanding of how food marketing and nutrition influences individual food choices. Continuous monitoring of manufacturers' and retailers' marketing activities over time can provide vital information to decision-makers regarding whether there is an opportunity to improve the food environment and hence achieve food supply chain transformations toward healthy eating.

The Mediterranean Diet Pattern (MDP) has been shown in numerous studies to be consistently associated with a lower prevalence of ischemic heart disease and lower overall mortality and morbidity. The pattern is characterized by a diet rich in fruit, vegetables, legumes, whole grains, fish, and low-fat dairy products. The next chapter reviews the data and also comments on the changes seen recently in the diets of populations living in the Mediterranean regions. New indexes have been developed to help capture these changes. The Mediterranean Diet Quality Index (KIDMED) was developed to assess the adherence to MDP in school children and adolescents. Based on a new study using the KIDMED score of students, only 6 % were classified as high adherers to a Mediterranean diet and about 42 % were classified as having very low diet quality that correlated with clinically relevant levels of urinary albumin.

The logical sequence of the parts in each volume as well as the chapters within each part enhances the understanding of the latest information on the current standards of practice in diet quality assessment in different countries around the world. This comprehensive two volume resource has great value for academicians involved in the education of graduate students and postdoctoral fellows, medical students, allied health professionals, and public health nutritionists who plan to improve diet quality especially for populations at risk for macro and/or micronutrient deficiencies. Moreover, the final chapter in the volume provides a comprehensive resource to websites, professional organizations, books, journals, and other resources that are of value to any health professional interested in diet quality.

The two volumes contain over 250 detailed tables and figures that assist the reader in comprehending the complexities of food choices, quantification of intake and availability of essential nutrients, composition of diet indexes across the world, comparisons of the nutritional needs of infants and children, pregnant women, other healthy adults and seniors. There are in-depth discussions of the behavioral aspects of eating as well as cultural, genetic, and emotional health inputs into food choices. The overriding goal of this volume is to provide the health professional with balanced documentation and awareness of the newest research and technical approaches including an appreciation of the complexity of the interactions between genetics, health and disease, nutrient deficiencies, and new issues of psychological aspects to food choice in this relatively new field of investigation of diet quality. Hallmarks of the 50 chapters include key words and bulleted key points at the beginning of each chapter, complete definitions of terms with the abbreviations fully defined for the reader, and consistent use of terms between chapters. There are over 2,300 up-to-date references; all chapters include a conclusion to highlight major findings. The volume also contains a highly annotated index.

This unique text provides practical, data-driven resources based upon the totality of the evidence to help the reader understand the basics of determining diet quality, historic perspectives, and descriptions of the most widely used indexes as well as updates that are specialized to capture the diet quality of children, seniors, and other population groups. New research using novel sources of data such as information concerning food purchase choices and uses of electronic personal data collection are described as these are being implemented in the most at risk populations in developed as well as developing nations across the world. Of equal importance, critical issues that involve food preferences, food choice interactions that affect nutrient absorption, and regulatory and public health perspectives in developing national dietary recommendations based upon diet quality data are included in well-referenced, informative chapters. The overarching goal of the editors is to provide fully referenced information to health professionals so they may have a balanced perspective on the value of measuring diet quality to assure governments that their food fortification options improve the nutrient density of consumed diets, as one example.

In conclusion, "Diet Quality: An Evidence-Based Approach," edited by Professor Victor R. Preedy, PhD, DSc, FRSPH, FRSC, FSB, FRCPath, Dr. Lan-Anh Hunter, BSc, MBBS, MRCGP, and Vinood B. Patel, PhD provides health professionals in many areas of research and practice with the most up-to-date, well-referenced, and comprehensive volumes on the current state of the science and medical practice guidelines with regard to the value of dietary quality assessment programs. The two volumes will serve the reader as the most authoritative resource in the field to date and are very welcome additions to the Nutrition and Health Series.

Morristown, NJ, USA Adrianne Bendich, PhD, FACN, FASN
Series Editor

About Series Editor

Dr. Adrianne Bendich, PhD, FACN, FASN has recently retired as Director of Medical Affairs at GlaxoSmithKline (GSK) Consumer Healthcare where she was responsible for leading the innovation and medical programs in support of many well-known brands including TUMS and Os-Cal. Dr. Bendich had primary responsibility for GSK's support for the Women's Health Initiative (WHI) intervention study. Prior to joining GSK, Dr. Bendich was at Roche Vitamins Inc. and was involved with the groundbreaking clinical studies showing that folic acid-containing multivitamins significantly reduced major classes of birth defects. Dr. Bendich has coauthored over 100 major clinical research studies in the area of preventive nutrition. Dr. Bendich is recognized as a leading authority on antioxidants, nutrition and immunity and pregnancy outcomes, vitamin safety and the cost-effectiveness of vitamin/mineral supplementation.

Dr. Bendich, who is now President of Consultants in Consumer Healthcare LLC, is the editor of ten books including "Preventive Nutrition: The Comprehensive Guide for Health Professionals, Fourth Edition" coedited with Dr. Richard Deckelbaum, and is *Series Editor of* "Nutrition and Health" for Springer/Humana Press (http://www.springer.com/series/7659). The Series contains over 50 published volumes—major new editions include *Vitamin D*, *Second Edition* edited by Dr. Michael Holick; "Dietary Components and Immune Function" edited by Dr. Ronald Ross Watson, Dr. Sherma Zibadi, and Dr. Victor R. Preedy; "Bioactive Compounds and Cancer" edited by Dr. John A. Milner and Dr. Donato F. Romagnolo; "Modern Dietary Fat Intakes in Disease Promotion" edited by Dr. Fabien DeMeester, Dr. Sherma Zibadi, and Dr. Ronald Ross Watson; "Iron Deficiency and Overload" edited by Dr. Shlomo Yehuda and Dr. David Mostofsky; "Nutrition Guide for Physicians" edited by Dr. Edward Wilson, Dr. George A. Bray, Dr. Norman Temple, and Dr. Mary Struble; "Nutrition and Metabolism" edited by Dr. Christos Mantzoros and "Fluid and Electrolytes in Pediatrics" edited by Leonard Feld and Dr. Frederick Kaskel. Recent volumes include "Handbook of Drug-Nutrient Interactions" edited by Dr. Joseph Boullata and Dr. Vincent Armenti; "Probiotics in Pediatric Medicine" edited by Dr. Sonia

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Dr. Bendich served as Associate Editor for "Nutrition" the International Journal; served on the Editorial Board of the Journal of Women's Health and Gender-based Medicine, and was a member of the Board of Directors of the American College of Nutrition.

Dr. Bendich was the recipient of the Roche Research Award, is a *Tribute to Women and Industry* Awardee and was a recipient of the Burroughs Wellcome Visiting Professorship in Basic Medical Sciences, 2000–2001. In 2008, Dr. Bendich was given the Council for Responsible Nutrition (CRN) Apple Award in recognition of her many contributions to the scientific understanding of dietary supplements. Dr. Bendich holds academic appointments as Adjunct Professor in the Department of Preventive Medicine and Community Health at UMDNJ and has an adjunct appointment at the Institute of Nutrition, Columbia University P&S, and is an Adjunct Research Professor, Rutgers University, Newark Campus. She is listed in Who's Who in American Women.

About Volume Editors

Victor R. Preedy, BSc, **PhD**, **DSc**, **FSB**, **FRCPath**, **FRSPH**, **FRSC** is a senior member of King's College London (Professor of Nutritional Biochemistry) and King's College Hospital (Professor of Clinical Biochemistry). He is attached to both the Diabetes and Nutritional Sciences Division and the Department of Nutrition and Dietetics. He is also Director of the Genomics Centre and a member of the School of Medicine. Professor Preedy graduated in 1974 with an Honors Degree in Biology and Physiology with Pharmacology. He gained his University of London PhD in 1981. In 1992, he received his Membership of the Royal College of Pathologists and in 1993 he gained his second doctoral degree, for his outstanding contribution to protein metabolism in health and disease. Professor Preedy was elected as a Fellow to the Institute of Biology in 1995 and to the Royal College of Pathologists in 2000. Since then he has been elected as a Fellow to the Royal Society for the Promotion of Health (2004) and The Royal Institute of Public Health (2004). In 2009, Professor Preedy became a Fellow of the Royal Society for Public Health and in 2012 a Fellow of the Royal Society of Chemistry. In his career Professor Preedy has carried out research at the National Heart Hospital (part of Imperial College London) and the MRC Centre at Northwick Park Hospital. He has collaborated with research groups in Finland, Japan, Australia, USA, and Germany. He is a leading expert on the science of health. He has lectured nationally and internationally. To his credit, Professor Preedy has published over 570 articles, which includes 165 peer-reviewed manuscripts based on original research, 100 reviews, and over 50 books and volumes.

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Part I

Introductory Chapters

1 Diet Quality: Setting the Scene

Najeeba F. Alamir and Victor R. Preedy

Key Points

- Diet quality is a subjective term that is often used within the context of a deficiency or excess of nutrients or foods.
- Attempts have been made to translate such subjectivity into objective measures, namely via scoring systems. Such scoring systems comprise nutrients and foods or food groups that are assumed to be either healthy or detrimental.
- This chapter briefly describes a few scoring systems, namely the Healthy Eating Index (HEI), Diet Quality Index (DQI), Diet Quality Index-International (DQI-I), Healthy Diet Indicator (HDI), and the Mediterranean Diet Score (MDS).
- These selective examples illustrate the nature of the scoring systems, their attributes, and applications.
- Some of these scoring systems correlate with biomarkers, mortality, cognitive impairment, and other variables.
- On the other hand some studies are negative, suggesting that the scoring systems may not be applicable to all situations.

Keywords

Diet quality • Health Eating Index (HEI) • Diet Quality Index (DQI) • Healthy Diet Indicator (HDI) • Mediterranean Diet Score (MDS) • Diet Quality Index-International (DQI-I)

Abbreviations

AHEI	Alternative Healthy Eating Index
AI	Adequate Intake
ATP	Adenosine triphosphate
CHO	Carbohydrate
CVD	Cardiovascular disease
DGI	Dietary Guidelines Index
DQI	Diet Quality Index

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DQI-I	Diet Quality Index-International
DQI-R	Diet Quality Index Revised
FBQI	Food-Based Quality Index
FPI	Food Pyramid Index
Hb	Hemoglobin
HDI	Healthy Diet Indicator
HDL	High density lipoprotein
HEI	Healthy Eating Index
HFI	Healthy Food Index
MAR	Mean Adequacy Ratio
MDQI	Mediterranean Diet Quality Index
MDS	Mediterranean Diet Score
MUFA	Monounsaturated fatty acid
NAR	Nutrient Adequacy Ratio
PRO	Protein
PUFA	Polyunsaturated fatty acid
RDA	Recommended Dietary Allowance
RNI	Recommended Nutrient Intake
SFA	Saturated fatty acid

Introduction

The human diet is composed of many different nutrients, which contribute to maintaining normal bodily functions. Components of the diet may be used to provide metabolic fuels (e.g., the breakdown of macronutrients to form adenosine triphosphate (ATP) which may be used to maintain the sodium potassium balance across cells), building blocks (e.g., amino acids for cytoskeletal proteins), or components of various cellular constituents (e.g., selenium as part of glutathione peroxidase).

In nutritional epidemiology, the focus has mainly been directed towards the role of such single dietary components [1]. This "reductionist" approach can reveal the relationship of individual nutrients or foods in disease development [1, 2]. However, this approach disregards the true complexity of the human diet, and therefore the true relationship between diet and disease [3]. This is because the food matrix is a composite mixture of individual components, many of which do not appear in the established lists of dietary reference values or recommended daily intakes. Additionally, within the body, there are many nutrient-to-nutrient interactions that further complicate the associations between single dietary components and disease [4]. For this matter, a "holistic" approach is usually undertaken to evaluate diet quality based on patterns of intake [3].

Table 1.1 Characteristics of predefined indices of overall diet quality

Created beforehand ("a priori")
Researcher selects attributes: nutrients, foods or food groups, and/or an indicator of dietary variety
Built upon current nutrition knowledge
Often represents nutrition guidelines or recommendations
Or based on diet that has proven healthful (e.g., Mediterranean diet)

Source: Waijers and Feskens [3]

Dietary patterning can be defined as theoretically defined dietary patterns or empirically derived dietary patterns [1]. The latter is examined in "a posteriori" approach, where statistical methods such as factor and cluster analyses are used to generate patterns from collected food consumption data [1, 3, 5]. Theoretically defined dietary patterns (dietary indices/scores), on the other hand, are created "a priori." They are made up of nutritional variables (foods and/or nutrients) and based on current nutrition knowledge [1, 3, 5]. These variables are quantified and summed to provide an overall measure of diet quality [1, 5]. The ensuing material will focus on predefined indices of overall diet quality. Unfortunately, the term *diet quality* has been defined and used in different ways. However, there have been various attempts to make the concept of diet quality more objective, quantitative, and measurable. Table 1.1 provides a basic breakdown of the attributes and key issues in the construction of predefined indices of diet quality [3].

A variety of diet indices or tools have been developed to assess overall diet quality [6]. Typically, these indices are constructed on the basis of dietary recommendations, such as "servings" of food items in the US Department of Agriculture Food Guide Pyramid [4, 7].

Existing Indices of Diet Quality

There are several systems of scoring that have been validated by relating the index to health outcome. Table 1.2 provides an overview of existing diet quality indices and studies in

Table 1.2 Overview of existing diet quality measures and studies in which they have been used and/or evaluated

Index	Authors
Based on dietary guidelines	
Diet Quality Index (DQI)[a]	**Patterson et al.** [9] Seymour et al. [13] Dubois et al. [14]
Diet Quality Index Revised (DQI-R)	**Haines et al.** [15] Newby et al. [16] Fung et al. [17]
Diet Quality Index International (DQI-I)	**Kim et al.** [12]
Other indices adapted from the DQI	
DQI-a I	Drewnowski et al. [18]
DQI-a II	Drewnowski et al. [19]
DQI-a III	Lowik et al. [20]
Healthy Eating Index (HEI)[a]	**Kennedy et al.** [8] McCullough et al. [21] McCullough et al. [22] Dubois et al. [14] Kennedy et al. [23] Hann et al. [24] McCullough et al. [25] Weinstein et al. [26] Fung et al. [17]
Alternative Healthy Eating Index (AHEI)	**McCullough et al.** [25] Fung et al. [17]
Healthy Diet Indicator (HDI)[b]	**Huijbregts et al.** [10, 27] Huijbregts et al. [28] Dubois et al. [14] Haveman-Nies et al. [29]
Dietary Guidelines Index (DGI)	**Harnack et al.** [30]
Based on Mediterranean diet	
Mediterranean Diet Score (MDS)	**Trichopoulou et al.** [11] Osler and Schroll [31] Kouris-Blazos et al. [32] Lasheras et al. [33] Woo et al. [34] Haveman-Nies et al. [29] Bosetti et al. [35]
Mediterranean Diet Quality Index (MDQI)	**Gerber et al.** [36] Scali et al. [37]
MDS + fish (MDS-f)	**Trichopoulou et al.** [38] Knoops et al. [39] Trichopoulou et al. [40]
Other indices adapted from the MDS	
MDS-a I	**Haveman-Nies et al.** [41]
MDS-a II	Schroder et al. [42]
MDS-a III	Fung et al. [17]
MDS-a IV	Pitsavos et al. [43]
Food-based	
Food-Based Quality Index (FBQI)	**Lowik et al.** [20]
Healthy Food Index (HFI)	**Osler et al.** [44] Osler et al. [45]
Food Pyramid Index (FPI)	**Massari et al.** [46]
Nutrient-based	
Nutrient Adequacy Ratio (NAR/MAR)[c]	**Madden and Yoder** [47]

Source: Waijers et al. [1]

Publications in which the index was first published are shown in bold

[a]Based on US dietary recommendations

[b]Based on 1990 WHO dietary guidelines

[c]Nutrient Adequacy Ratio (NAR) is the ratio of intake of a nutrient relative to its Recommended Dietary Allowance (RDA). The Mean Adequacy Ratio (MAR) is computed by averaging the sum of the NAR. These scores have been used in several studies, and also to evaluate diet quality scores

which they have been used and/or evaluated [3]. Some of the most common of these indices are the Healthy Eating Index (HEI) [8], Diet Quality Index (DQI) [9], Healthy Diet Indicator (HDI) [10], and the Mediterranean Diet Score (MDS) [11]. The Diet Quality Index-International (DQI-I) [12], which is a derivative of the DQI, is a fairly recent predefined measure created for global monitoring and exploring diet quality across countries. Prior to the development of this index, cross-national comparison of diet quality had rarely been attempted [12].

There are some indices, such as the Food-Based Quality Index (FBQI), that consist solely of foods or food groups. Other indices like the adapted DQIs consist of just nutrients. The majority of indices, however, comprise both food groups and nutrients [3]. Table 1.3 includes an overview of the attributes found in the scoring systems mentioned below [8–47].

Healthy Eating Index

The HEI is a 10-component, 100-point measure of diet quality that assesses conformance to US dietary guidelines [48]. It is based on five different food groups (grains, vegetables, fruits, milk, and meat), four nutrients (total fat, saturated fatty acids (SFAs), cholesterol, and sodium), and a measure of the variety in food intake [8].

A HEI score of 80 or more indicates a good diet; scores between 50 and 80 suggest that a diet needs improvements, and scores less than 50 consider a diet to be poor [49]. Table 1.4 shows an overview of the HEI.

Hann et al. [24] and Weinstein et al. [26] found that the HEI was associated with a wide range of nutritional biomarkers of micronutrients, i.e., alpha-carotene, beta-carotene, beta-cryptoxanthin, and vitamin C. They explain that consumption of these nutrients is a common indicator of fruits and vegetables, and therefore consumption of these food groups [24]. Both studies found no significant correlation between HEI score and cholesterol [24, 26]. A study by Dubois et al. [14] analyzed three different methods to measuring overall diet quality. They found that the HEI had a higher correlation with the Mean Adequacy Ratio (MAR = 0.287) of several nutrients, compared to the DQI and HDI.

Data on the relationship between the HEI score and mortality is lacking. However, there are four studies that have examined the relationship between HEI and disease risk [21, 22, 25, 30]. Harnack et al. [30] found no significant association between the HEI score with cancer incidence [30]. In the McCullough et al. [21, 22] studies, a weak inverse association between HEI score and chronic disease risk (cardiovascular disease (CVD) and cancer) was reported [21]. They did not report such an association with overall chronic disease risk in women, and only a weak inverse association with CVD risk [22].

The HEI is based on US dietary guidelines and, to a certain extent, measures how individuals follow these guidelines. However, further work is needed to firmly establish the HEI as a good predictor of health outcome. Nevertheless, the HEI shows correlations with plasma biomarkers such as alpha-carotene, beta-carotene, beta-cryptoxanthin, and vitamin C [24].

Table 1.3 Overview of attributes included in theoretically defined indices of diet quality

Nutrients
Fat-related variables: total fat, saturated fat, cholesterol, MUFA/SFA
Carbohydrates: (complex) carbohydrates, mono- and disaccharides, sucrose
Dietary fiber
Protein
Micronutrients: sodium, calcium, iron, vitamin C
Alcohol
Foods of food groups
Vegetables and fruit: vegetables, vegetables and fruit, fruit, fruit and nuts, legumes, legumes and nuts, etc.
Meats (and meat products)
Cereals or grains
Milk (and dairy)
Others: fish, olive oil, cheese
Dietary diversity or dietary variety
Dietary moderation

Source: Waijers and Feskens [3]
MUFA monounsaturated fatty acid, *SFA* saturated fatty acid

Table 1.4 Health Eating Index (HEI)

	Scoring		
Component	Criteria for score 0	Criteria for score 10[a]	Range
Grains	0 servings	6–11 servings	0–10
Vegetables	0 servings	3–5 servings	0–10
Fruits	0 servings	2–4 servings	0–10
Milk	0 servings	2–3 servings	0–10
Meat	0 servings	2–3 servings	0–10
Total fat	>45 energy %	<30 energy %	0–10
Saturated fatty acids	>15 energy %	<10 energy %	0–10
Cholesterol	>450 mg	<300 mg	0–10
Sodium	>4,800 mg	<2,400 mg	0–10
Variety	≤6 different food items/3 days	16 different food items/3 days	0–10

Source: Kennedy et al. [8]
[a]Depending on energy intake

Healthy Diet Indicator

The HDI (Table 1.5) was developed in the Netherlands [3] and is based on the World Health Organisation's dietary recommendations for the prevention of chronic disease [50]. The HDI is made up of nine micro- and macronutrient components [3, 10]. It uses a 9-point measure of four nutrients (SFAs, polyunsaturated fatty acids (PUFAs), mono- and disaccharides, and cholesterol) and five food groups (complex carbohydrates, dietary fiber, fruits and vegetables, and pulses, nuts, and seeds). Typically, the higher the overall HDI score, the better the diet quality.

Huijbregts et al. [10, 27] reported the HDI to be inversely associated with all-cause mortality in men [10] but not women [27]. Huijbregts et al. [28] also suggested a healthy HDI score to correlate with better cognitive function in elderly men. Dubois et al. [14] reported that the HDI score only slightly correlated with MAR (0.079). Moreover, Haveman-Nies et al. [29] found no association between HDI score and albumin, hemoglobin (Hb), or waist circumference.

Diet Quality Index and Diet Quality Index-International and Health Outcome

The DQI is made up of eight components (Table 1.6) and is based on the US recommendations from *Diet and Health* [51]. The DQI uses a 16-point measure, where a high score is indicative of a poor diet quality (unlike the previously mentioned dietary indices).

Dubois et al. [14] showed the DQI to only marginally correlate with nutrient adequacy. One study by Seymour et al. [13] found that a high DQI score was positively associated with all-cause mortality. Moreover, persons with a high DQI score had lower CVD-mortality, but no association was found between cancer mortality and the DQI score [13].

The DQI-I is an adapted version of the DQI [12]. It is divided into four major components, including, variety, adequacy, moderation, and empty calorie foods (Table 1.7). The total DQI-I score ranges from 0 to 100, where a score of "0" reflects an extremely poor diet, and a score of "100" (highest possible score) indicates a high quality diet [12]. The differences between the original DQI and the DQI-I indices are significant. The DQI-I not only is more extensive than

Table 1.5 Healthy Diet Indicator (HDI)

Nutrient or food group	Scoring	
SFA	0–10 energy %	1 (else: 0)
PUFA	3–7 energy %	1 (else: 0)
Protein	10–15 % energy	1 (else: 0)
Complex carbohydrates	50–70 energy %	1 (else: 0)
Dietary fiber	27–40 g/day	1 (else: 0)
Fruits and vegetables	>400 g/day	1 (else: 0)
Pulses, nuts, and seeds	>30 g/day	1 (else: 0)
Mono- and disaccharides	0–10 energy %	1 (else: 0)
Cholesterol	0–300 mg/day	1 (else: 0)

Source: Huijbregts et al. [10]
SFA saturated fatty acid, *PUFA* polyunsaturated fatty acid

Table 1.6 Diet Quality Index (DQI[a])

Component	Scoring	
Total fat	<30 energy %	0
	30–40 energy %	1
	>40 energy %	2
Saturated fatty acids	<10 energy %	0
	10–13 energy %	1
	>13 energy %	2
Cholesterol	<300 mg	0
	300–400 mg	1
	>400 mg	2
Fruits and vegetables	5+ servings	0
	3–4 servings	1
	0–2 servings	2
Complex carbohydrates	6+ servings	0
	4–5 servings	1
	0–3 servings	2
Protein	≤100 % RDA	0
	100–150 % RDA	1
	>150 % RDA	2
Sodium	<2,400 mg	0
	2,400–3,400 mg	1
	>3,400 mg	2
Calcium	≥RDA	0
	2/3 RDA	1
	<2/3 RDA	2

Source: Patterson et al. [9]
RDA Recommended Dietary Allowance
[a]Based on US recommendations from Diet and Health (National Research Council, Committee on Diet and Health) [50]

Table 1.7 Diet Quality Index-International (DQI-I)

Component	Score (points)	Scoring criteria
Variety	0–20	
Overall food group variety (meat/poultry/fish/eggs, dairy/beans, grain, fruit, vegetable)	0–15	≥1 serving from each food group/day = 15 Any 1 food group missing/day = 12 Any 2 food groups missing/day = 9 Any 3 food groups missing/day = 6 ≥4 food groups missing/day = 3 None from any food groups = 0
Within-group variety for protein source (meat, poultry, fish, diary, beans, eggs)	0–5	≥3 different sources/day = 5 2 different sources/day = 3 From 1 source/day = 1 None = 0
Adequacy	0–40	
Vegetable group	0–5	≥3–5 servings/day = 5, 0 servings/day = 0
Fruit group	0–5	≥2–4 servings/day = 5, 0 servings/day = 0
Grain group	0–5	≥6–11 servings/day = 5, 0 servings/day = 0
Fiber	0–5	≥20–30 g/day = 5, 0 g/day = 0
Protein	0–5	≥10 % of energy/day = 5, 0 % of energy/day = 0
Iron	0–5	≥100 % RDA (AI)/day = 5, 0 % RDA (AI)/day = 0
Calcium	0–5	≥100 % (AI)/day = 5, 0 % (AI)/day = 0
Vitamin C	0–5	≥100 % RDA (RNI)/day = 5, 0 % RDA (RNI)/day = 0
Moderation	0–30	
Total fat	0–6	≤20 % of total energy/day = 6 >20–30 % of total energy/day = 3 >30 % of total energy/day = 0
Saturated fat	0–6	≤7 % of total energy/day = 6 >7–10 % of total energy/day = 3 >10 % of total energy/day = 0
Cholesterol	0–6	≤300 mg/day = 6 >300–400 mg/day = 3 >400 mg/day = 0
Sodium	0–6	≤2,400 mg/day = 6 >2,400–3,400 mg/day = 3 >3,400 mg/day = 0
Empty calorie foods	0–6	≤3 % of total energy/day = 6 >3–10 % of total energy/day = 3 >10 % of total energy/day = 0
Overall balance	0–10	
Macronutrient ratio (CHO:PRO:FAT)	0–6	55–65:10–15:15–25 = 6 52–68:9–16:13–27 = 4 50–70:8–17:12–30 = 2 Otherwise = 0
Fatty acid ratio (PUFA:MUFA:SFA)	0–4	P/S = 1–1.5 and M/S = 1–1.5 = 4 Else if P/S = 0.8–1.7 and M/S = 0.8–1.7 = 2 Otherwise = 0

Source: Kim et al. [12]
RDA Recommended Dietary Allowance, *AI* Adequate Intake, *RNI* Recommended Nutrient Intake, *CHO* carbohydrate, *PRO* protein, *PUFA* polyunsaturated fatty acid, *MUFA* monounsaturated fatty acid, *SFA* saturated fatty acid

the DQI but also has attributed different weights to the individual components [3]. Thus, the DQI-I incorporates both nutrient and food perspectives of the assessed diet, providing a more grounded tool to describe the diversity of consumption observed from country to country [12]. Kim et al. [12] have suggested that the DQI-I can indentify dietary problem areas. Like the DQI, a high score on the DQI-I, suggests a good diet quality.

Mediterranean Diet Score

The MDS is an eight-component, 8-point measure of diet quality (Table 1.8). It is based mainly on food groups, and is supplemented with a ratio of the fatty acid composition of the diet (monounsaturated fatty acids (MUFAs) and SFAs) [1]. A high MDS score indicates a good diet quality.

A study by Lasheras et al. [33] evaluated the relationship between MDS and mortality among Spanish elderly. They found that the MDS was only significantly associated with a reduced risk of death in persons under 80 years [33]. Another study evaluating French adults was also reported to exhibit lower mortality following a Mediterranean diet in intervention studies [52, 53]. Furthermore, Osler and Schroll [31] found an association between plasma carotene and MDS. However, no association was reported between plasma cholesterol, high density lipoprotein (HDL), or vitamin E with the score [31].

Dietary patterns are influenced greatly by cultural differences. Thus, it is important to take into consideration these differences when choosing diet quality indices to measure diet quality of a certain population. Though the MDS may seem pertinent in predicting mortality, especially in European Mediterranean populations, it may be better to adapt or develop a score that tailors to local diets in Western populations, like the UK [3].

Subjective and Non-scoring Systems

It is important to emphasize that many published studies employ the term *diet quality* or other aspects of *quality* without using indices or numerical scoring systems. Such studies may be subjective but based on a firm scientific foundation. For example, diets low in iodine may be deemed as *poor* or *low quality* as the consequences of iodine deficiency can be devastating. Iodine deficiency, for example, is well known to be associated with increased rates of stillbirths, spontaneous abortions, cretinism, hypothyroidism, impaired cognitive function, etc. This does not mean to say that the terms *poor* or *low quality* have been misused but rather they have been used within a different context. However, it is increasingly likely that the use of diet quality scoring systems will gain wider usage, and new ones developed as the scientific dialogue between diet and disease progresses.

Table 1.8 Mediterranean Diet Score (MDS)

Nutrient or food group	Scoring	
MUFA:SFA	>Median	1 (else: 0)
Legumes	>Median	1 (else: 0)
Cereals	>Median	1 (else: 0)
Fruits and nuts	>Median	1 (else: 0)
Vegetables	>Median	1 (else: 0)
Meat and meat products	<Median	1 (else: 0)
Milk and dairy products	<Median	1 (else: 0)
Alcohol	<Median	1 (else: 0)

Source: Trichopoulou et al. [11]
MUFA monounsaturated fatty acid, *SFA* saturated fatty acid

Conclusion

Diet quality is a subjective term that is often used within the context of a deficiency or excess of nutrients or foods. Attempts have been made to translate such subjectivity into objective measures, namely via scoring systems. Some of these scoring systems correlate with biomarkers, mortality, cognitive impairment, and other variables. On the other hand some studies are negative suggesting that the scoring systems may not be applicable to all situations or populations.

References

1. Waijers PM, Feskens EJ, Ocke MC. A critical review of predefined diet quality scores. Br J Nutr. 2007;97(2):219–31.
2. Willett W. Nutritional epidemiology. 2nd ed. Oxford: Oxford University Press; 1998.
3. Waijers PM, Feskens E. Indexes of overall diet quality. A review of the literature. Bilthoven: The National Institute for Public Health and the Environment. Ministry of Health, Welfare and Sports; 2005.
4. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol. 2002; 13(1):3–9.
5. Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. Nutr Rev. 2004;62(5):177–203.
6. Kant AK. Indexes of overall diet quality: a review. J Am Diet Assoc. 1996;96(8):785–91.

7. Homes and Garden Bulletin No. 252. The food guide pyramid. Washington, DC: US Department of Agriculture, Human Nutrition Information Service; 1992.
8. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: design and applications. J Am Diet Assoc. 1995;95(10):1103–8.
9. Patterson RE, Haines PS, Popkin BM. Diet quality index: capturing a multidimensional behavior. J Am Diet Assoc. 1994;94(1):57–64.
10. Huijbregts P, Feskens E, Rasanen L, et al. Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and The Netherlands: longitudinal cohort study. BMJ. 1997;315(7099):13–7.
11. Trichopoulou A, Kouris-Blazos A, Wahlqvist ML, et al. Diet and overall survival in elderly people. BMJ. 1995;311(7018):1457–60.
12. Kim S, Haines PS, Siega-Riz AM, Popkin BM. The Diet Quality Index-International (DQI-I) provides an effective tool for cross-national comparison of diet quality as illustrated by China and the United States. J Nutr. 2003;133(11):3476–84.
13. Seymour JD, Calle EE, Flagg EW, et al. Diet Quality Index as a predictor of short-term mortality in the American Cancer Society Cancer Prevention Study II Nutrition Cohort. Am J Epidemiol. 2003;157(11):980–8.
14. Dubois L, Girard M, Bergeron N. The choice of a diet quality indicator to evaluate the nutritional health of populations. Public Health Nutr. 2000;3(3):357–65.
15. Haines PS, Siega-Riz AM, Popkin BM. The Diet Quality Index revised: a measurement instrument for populations. J Am Diet Assoc. 1999;99(6):697–704.
16. Newby PK, Hu FB, Rimm EB, et al. Reproducibility and validity of the Diet Quality Index Revised as assessed by use of a food-frequency questionnaire. Am J Clin Nutr. 2003;78(5):941–9.
17. Fung TT, McCullough ML, Newby PK, et al. Diet-quality scores and plasma concentrations of markers of inflammation and endothelial dysfunction. Am J Clin Nutr. 2005;82(1):163–73.
18. Drewnowski A, Henderson SA, Shore AB, Fischler C, Preziosi P, Hercberg S. Diet quality and dietary diversity in France: implications for the French paradox. J Am Diet Assoc. 1996;96(7):663–9.
19. Drewnowski A, Henderson SA, Driscoll A, Rolls BJ. The Dietary Variety Score: assessing diet quality in healthy young and older adults. J Am Diet Assoc. 1997;97(3):266–71.
20. Lowik MR, Hulshof KF, Brussaard JH. Food-based dietary guidelines: some assumptions tested for The Netherlands. Br J Nutr. 1999;81 Suppl 2:S143–9.
21. McCullough ML, Feskanich D, Rimm EB, et al. Adherence to the Dietary Guidelines for Americans and risk of major chronic disease in men. Am J Clin Nutr. 2000;72(5):1223–31.
22. McCullough ML, Feskanich D, Stampfer MJ, et al. Adherence to the Dietary Guidelines for Americans and risk of major chronic disease in women. Am J Clin Nutr. 2000;72(5):1214–22.
23. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. J Am Diet Assoc. 2001;101(4):411–20.
24. Hann CS, Rock CL, King I, Drewnowski A. Validation of the Healthy Eating Index with use of plasma biomarkers in a clinical sample of women. Am J Clin Nutr. 2001;74(4):479–86.
25. McCullough ML, Feskanich D, Stampfer MJ, et al. Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr. 2002;76(6):1261–71.
26. Weinstein SJ, Vogt TM, Gerrior SA. Healthy Eating Index scores are associated with blood nutrient concentrations in the third National Health And Nutrition Examination Survey. J Am Diet Assoc. 2004;104(4):576–84.
27. Huijbregts P, de Vegt F, Feskens E, Bowles C, Kromhout D. Dietary patterns and mortality in an elderly population in the Netherlands. A comparison between cluster analysis and the healthy diet indicator. In: Huijbregts P, editor. Dietary patterns and health in the elderly. Wageningen: Wageningen University; 1997. p. 33–42.
28. Huijbregts PP, Feskens EJ, Rasanen L, et al. Dietary patterns and cognitive function in elderly men in Finland, Italy and The Netherlands. Eur J Clin Nutr. 1998;52(11):826–31.
29. Haveman-Nies A, Tucker KL, de Groot LC, Wilson PW, van Staveren WA. Evaluation of dietary quality in relationship to nutritional and lifestyle factors in elderly people of the US Framingham Heart Study and the European SENECA study. Eur J Clin Nutr. 2001;55(10):870–80.
30. Harnack L, Nicodemus K, Jacobs Jr DR, Folsom AR. An evaluation of the Dietary Guidelines for Americans in relation to cancer occurrence. Am J Clin Nutr. 2002;76(4):889–96.
31. Osler M, Schroll M. Diet and mortality in a cohort of elderly people in a north European community. Int J Epidemiol. 1997;26(1):155–9.
32. Kouris-Blazos A, Gnardellis C, Wahlqvist ML, Trichopoulos D, Lukito W, Trichopoulou A. Are the advantages of the Mediterranean diet transferable to other populations? A cohort study in Melbourne, Australia. Br J Nutr. 1999;82(1):57–61.
33. Lasheras C, Fernandez S, Patterson AM. Mediterranean diet and age with respect to overall survival in institutionalized, nonsmoking elderly people. Am J Clin Nutr. 2000;71(4):987–92.
34. Woo J, Woo KS, Leung SS, et al. The Mediterranean score of dietary habits in Chinese populations in four different geographical areas. Eur J Clin Nutr. 2001;55(3):215–20.
35. Bosetti C, Gallus S, Trichopoulou A, et al. Influence of the Mediterranean diet on the risk of cancers of the upper aerodigestive tract. Cancer Epidemiol Biomarkers Prev. 2003;12(10):1091–4.
36. Gerber MJ, Scali JD, Michaud A, et al. Profiles of a healthful diet and its relationship to biomarkers in a

population sample from Mediterranean southern France. J Am Diet Assoc. 2000;100(10):1164–71.
37. Scali J, Richard A, Gerber M. Diet profiles in a population sample from Mediterranean southern France. Public Health Nutr. 2001;4(2):173–82.
38. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348(26):2599–608.
39. Knoops KT, de Groot LC, Kromhout D, et al. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. JAMA. 2004;292(12):1433–9.
40. Trichopoulou A, Orfanos P, Norat T, et al. Modified Mediterranean diet and survival: EPIC-elderly prospective cohort study. BMJ. 2005;330(7498):991.
41. Haveman-Nies A, de Groot LP, Burema J, et al. Dietary quality and lifestyle factors in relation to 10-year mortality in older Europeans: the SENECA study. Am J Epidemiol. 2002;156(10):962–8.
42. Schroder H, Marrugat J, Vila J, Covas MI, Elosua R. Adherence to the traditional Mediterranean diet is inversely associated with body mass index and obesity in a Spanish population. J Nutr. 2004;134(12): 3355–61.
43. Pitsavos C, Panagiotakos DB, Tzima N, et al. Adherence to the Mediterranean diet is associated with total antioxidant capacity in healthy adults: the ATTICA study. Am J Clin Nutr. 2005;82(3): 694–9.
44. Osler M, Heitmann BL, Gerdes LU, Jorgensen LM, Schroll M. Dietary patterns and mortality in Danish men and women: a prospective observational study. Br J Nutr. 2001;85(2):219–25.
45. Osler M, Helms Andreasen A, Heitmann B, et al. Food intake patterns and risk of coronary heart disease: a prospective cohort study examining the use of traditional scoring techniques. Eur J Clin Nutr. 2002;56(7):568–74.
46. Massari M, Freeman KM, Seccareccia F, Menotti A, Farchi G, Research Group of the RP. An index to measure the association between dietary patterns and coronary heart disease risk factors: findings from two Italian studies. Prev Med. 2004;39(4):841–7.
47. Madden JP, Yoder MD. Program evaluation: food stamps and commodity distribution in rural areas of central Pennsylvania. University Park, PA: Department of Agricultural Economics and Rural Sociology, The Pennsylvania State University, College of Agriculture; 1972.
48. US Department of Agriculture, Center for Nutrition Policy and Promotion. The Healthy Eating Index. Washington, DC: US Department of Agriculture; 1995.
49. Pick ME, Edwards M, Moreau D, Ryan EA. Assessment of diet quality in pregnant women using the Healthy Eating Index. J Am Diet Assoc. 2005;105(2):240–6.
50. WHO Study Group on Diet Nutrition and Prevention of Noncommunicable Diseases. World Health Organization. Diet, nutrition and the prevention of chronic diseases: report of a WHO study group [meeting held in Geneva from 6–13 March 1989]. Geneva: World Health Organization; 1990.
51. Committee on Diet and Health, National Research Council. Diet and health. Implications of reducing chronic disease risk. Washington, DC: National Academy Press; 1989.
52. deLorgeril M, Salen P, Martin JL, Monjaud I, Boucher P, Mamelle N. Mediterranean dietary pattern in a randomized trial: prolonged survival and possible reduced cancer rate. Arch Intern Med. 1998;158(11): 1181–7.
53. deLorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. Circulation. 1999;99(6):779–85.

Food Intake and Food Preference

2

Stephanie Stein, Elizabeth Lamos, Michael Quartuccio, Sruti Chandraskaran, Nicole Ionica, and Nanette Steinle

Key Points

- Perceived health benefits of foods strongly influence food selection, although perceived health benefits influence women's food choices more than the food choices of men.
- Men's food choices are strongly influenced by social norms. Cultural and social norms also determine customary potion sizes of meals and influence the amount of food consumed at a meal.
- At least in part, an individual's food intake and preferences may have cultural and/or religious roots.
- Food cost and accessibility play a role in food choice and preference.
- Commercial advertising influences food intake.
- The presence of nutrients in the gut stimulates the secretion of several gut-derived hormones including cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), oxyntomodulin (OXM), neurotensin (NT), and peptide YY (PYY), as well as glucose-dependent insulinotrophic peptide (GIP), all which attenuate continued food ingestion. The adipose tissue-derived hormone, leptin also promotes satiety signals.
- The gut-derived peptide hormone, ghrelin, stimulates food intake. The neuropeptides NPY and agouti-related peptide are produced in the central nervous system (CNS) and stimulate food intake.
- The CNS is important in regulating food intake. The hypothalamus and brainstem are key in regulating energy intake and expenditure through neuronal and hormonal networks. The limbic and prefrontal regions

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regulate reward systems. Brain centers involved in pleasure and reward are activated when we eat, and the neurotransmitter dopamine plays a significant role in the neuronal control of feeding behavior. Centers processing cognitive behaviors include the prefrontal cortex and cingulated regions. Cognitive control can be enhanced with practice.

- Obesity is associated with attenuated satiety signaling, leptin resistance, and abnormalities in the CNS dopamine reward network.
- Taste and olfaction strongly affect eating behavior and nutrient intake. Enhanced bitter perception is associated with reduced vegetable intake and reduced preference for sweet.
- Genetic studies suggest genetic variation to play a role in modulating food intake and preference.
- Many different modalities are used to measure food preference, including food frequency questionnaires (FFQs), hedonic food scales, direct observational feeding studies, 3 day and 24 h food records. FFQs are widely used to estimate food preference and intake. Hedonic scales are used to measure food preference or liking. Both food frequency and hedonic scales link food intake and preference to risk of chronic disease.

Keywords

Food intake • Eating behavior • Taste • Genetics • Hormone • Nervous system • Food preference

Abbreviations

AACE	American Association of Clinical Endocrinologists
ADA	American Diabetes Association
ARP	Agouti-related peptide
BMI	Body mass index
CCK	Cholecystokinin
CNS	Central nervous system
DASH	Dietary approaches to stop hypertension
FCI	Food craving inventory
FFQ	Food Frequency Questionnaire
fMRI	Functional magnetic resonance imaging
GABA	γ-Aminobutyric acid
GAD	Glutamic acid decarboxylase
GI	Gastrointestinal
GIP	Glucose-dependent insulinotrophic peptide
GLP-1	Glucagon-like peptide 1
IAPP	Islet amyloid polypeptide
NPY	Neuropeptide Y
NT	Neurotensin
POMC	Proopiomelanocortin
PYY	Peptide YY
SES	Socioeconomic status
TFEQ	Three-Factor Eating Questionnaire
TNF-α	Tumor necrosis factor α
vmPFC	Ventral medial pre frontal cortex

Introduction

The biologic drive to eat results from complex neural and hormonal systems. The selection of one food over another is also a complex process involving many factors including food availability, social and cultural norms, economic, biologic, and cognitive elements. Factors determining food preference are both learned and innate, stemming from environmental, psychosocial, biologic, and genetic influences. The environmental and psychosocial elements influencing food choice and preference include food's perceived value, culture, religion, cost, access, and exposure to food advertisements. Many modalities have been used to quantify food intake and preference including food frequency questionnaires (FFQs), hedonic food scales, feeding studies, food records, and open-ended questionnaires. In this chapter, we will discuss environmental, psychological, social, and economic factors impacting food intake and preference as well as biologic factors shaping eating behaviors, and lastly discuss scientific methods of quantifying and qualifying food preference and intake.

Perceived Health Value and Food Intake and Preference

The perceived health value of food impacts food intake and preference. Both real and perceived health risks and benefits of food play a role in food intake. Many consumers purchase organic foods due to perceived health or environmental benefits over conventional foods. Fresh foods have been promoted as being beneficial over processed foods due to absence of chemical preservatives and other food additives. Data from questionnaires aimed at determining attitudes and behaviors toward organic foods indicated that perceived health benefits of these foods most strongly determined whether respondents chose foods labeled organic [1]. Whole grains, lean protein, low-fat dairy and fresh fruits and vegetables are promoted due to health benefits associated with habitual consumption of these foods. Whole grains may be consumed rather than refined grains by individuals who hope to lower fasting insulin concentrations and improve insulin sensitivity, and/or to reduce risk of cardiovascular diseases, diabetes, and colon cancer [2]. The American Association of Clinical Endocrinologists (AACE) and the American Diabetes Association (ADA) recommend a reduced energy, low glycemic index, and low-fat diet as a therapeutic intervention for persons with diabetes mellitus [3]. Low-fat dairy and fresh vegetables and fruits are mainstays of the dietary approaches to stop hypertension (DASH) diet as well, which promotes blood pressure control. Health conscious individuals who desire to prevent or ameliorate the consequences of hypertension, diabetes, obesity, and cardiovascular disease through dietary interventions may select these types of foods for their dietary portfolio. Evidence from tools used to assess eating behavior suggests that women tend to select foods based on their perceived health value more so than do men.

Cultural and Religious Influences

Cultural and religious practices also influence food intake. Western culture, for example, traditionally shuns the consumption of insects, foods which are served as delicacies in Thailand, New Zealand, and South America. Dogs, socially perceived as part of the family unit in most Western cultures are not consumed as food; whereas, in some Chinese and Pacific Island cultures dogs are viewed as serviceable animals and are included in the dietary portfolio of many individuals. Many religions include a dietary code of conduct. Pork is often avoided by followers of both Judaism and Islam, and vegetarianism is promoted by some Christian, Buddhist, and Hindu sects. Hindu practice also rejects the consumption of cow based on the caste system inherent to their religious faith. Celebratory events are associated with particular foods such as the traditional birthday or graduation cake in Western Culture or the roasted turkey and the American Thanksgiving holiday. The Indian culture's celebration of the Festival of Lights involves the preparation of sweet foods like rice kheer or rice pudding. At least in part, an individual's food intake and preferences may have cultural or religious roots.

Environmental, Economic, and Social Factors

Historically, people consumed foods supported by local natural environments. Those living in regions with fertile soil enjoyed a variety of agricultural products, while desert nomads thrived on the plants and animals that survive harsh environments. With advances in transportation, a variety of foods produced globally are available in many communities; however, food deserts continue to exist due to political conflict and poverty. Food cost and accessibility play a role in food choice and preference, especially in today's urban environments. Energy dense foods, for example, pastries and snack foods tend to have a lower price compared to nutrient rich, low energy foods such as lean meat, fruits, and vegetables. Education about eating healthy may influence food intake toward more nutritious options, but focus groups of women in low income households (75 % of whom were overweight or obese) show that despite wanting to choose healthy options, the price and availability of these foods often limits their use. Most of these women report that the corner stores are most convenient and shopping at these stores curbs the cost and inconvenience of traveling to larger markets. However, these stores often do not stock lean meats or fresh produce which, therefore, limits food selections [4]. In addition, women of lower socioeconomic status (SES) often do not have role models to teach them how to cook healthy foods and therefore feel uncomfortable preparing a nutritious meal. Cultural and social norms also determine customary potion sizes of meals which influence the amount of food consumed at a meal. Additionally, some individuals from lower SES perceive healthy eating as equivalent to dieting and do not value the benefits outside of weight loss. In some cultures, being overweight or obese is without negative social stigma [5].

Exposure to advertisements also appears to have an influence on food choice and intake among children and adults. One study showed that when men were exposed to information about social norms (specifically that high percentages of the population agreed with eating a high vegetable/fruit diet and claimed that healthy eating was important to them), these men had a significant increase in their vegetable and fruit consumption. This social norm information caused a greater effect than information on both health benefits and cost effectiveness of healthy eating. This study suggests that alternate marketing campaigns aimed at stressing the popularity of healthy eating might be effective in influencing food intake [6]. Exposure to advertising may impact both the amount and type of food chosen by consumers. One study showed that children ate 45 % more goldfish crackers while watching TV with food advertising compared to children who watched nonfood advertising [7].

Table 2.1 Hormones associated with regulation of food intake

Stimulate food intake	Source	Inhibit food intake	Source
Ghrelin	Stomach	CCK	GI tract
		GLP-1	
		GIP	
		Glucagon	
		IAPP	
		Insulin	
		OXM	
		Pancreatic peptide	
		NT	
NPY	Central nervous system	PYY	Central nervous system
ARP		POMC	
		Leptin	Adipose tissue

Biologic Factors Impacting Food Preference and Intake

Biologic and genetic influences also play a role in our food preferences and food intake. Hunger drives food intake, and satiety signals bring about cessation of eating behavior. Both short term, meal to meal behaviors as well as long term energy intake and expenditure are regulated by the interaction of a variety of hormones and the nervous system (Tables 2.1 and 2.2), and coordinated via homeostatic mechanisms. Brain centers regulating food intake include reward centers (striatum, orbitofrontal cortex, insula), centers of emotion and memory (amygdala, hippocampus), sensory and motor processing regions (insula, precentral gyrus), centers regulating cognitive control and attention (prefrontal cortex, cingulate) and the center of homeostatic regulation of food intake regulating hunger and appetite (hypothalamus and brainstem) [8]. Gut-derived hormones and signals

Table 2.2 Central nervous system pathways involved in regulating food intake

Pathway	Associated brain area
Reward	Limbic region and prefrontal regions
Cognitive control, attention,	Prefrontal cortex, cingulate
Integration of appetite; homeostasis	Hypothalamus, brain stem
Activate ingestive behavior	Amygdala, orbitofrontal cortex, anterior insula, and striatum
Centers of emotion and memory	Amygdala, hippocampus
Sensory (taste, olfaction) and motor processing regions	Insula, precentral gyrus

provide input to brain centers forming networks modulating feeding behavior. It is beyond the scope of this chapter to provide a detailed explanation of the hormonal and brain networks regulating food intake; however, an overview of main concepts is presented.

Gut Hormones

Nutrients detected in the gut provide feedback to the central nervous system (CNS) and peripheral tissues to regulate food intake. The presence of nutrients in the gut stimulates the secretion of several gut-derived hormones including cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), oxyntomodulin (OXM), neurotensin (NT), peptide YY (PYY), and glucose-dependent insulinotrophic peptide (GIP), all which attenuate continued food ingestion [9–12]. These hormones provide input to the CNS via the vagus nerve. GLP-1, released by the intestines after a meal, slows gastric emptying and promotes satiety. Obese individuals appear to have a blunted release of GLP-1 following a meal. Gut endocrine L cells express the G protein coupled receptors (GPCRs) TAS1R and TAS2R which are also present in taste tissue and throughout the gastrointestinal (GI) tract. This family of GPCRs act as nutrient detectors by sensing sugars and amino acids [10]. When activated, these GPCRs signal the release of GLP-1 and PYY from L cells. Hormones produced by the pancreas in response to the detection of nutrients in the gut include insulin, pancreatic peptide, glucagon, and islet amyloid polypeptide (IAPP) also known as amylin, and have been shown to inhibit ingestive behavior [9–12].

Hypoglycemia is a potent stimulus of hunger and is mediated by glucose sensitive neurons in the brain and brainstem. Hypothalamic input stimulating initiation of food intake is also mediated by the gut-derived peptide hormone ghrelin, an agonist of the growth hormone secretagogue receptor 1a. Ghrelin levels rise in response to an overnight fast and are significantly reduced with meal ingestion [10].

Brain Centers Regulate Food Intake

The hypothalamus and brainstem are central regions regulating food intake, energy expenditure, appetite, and satiety, and are often referred to as the homeostasis center. The paraventricular nucleus of the hypothalamus receives input from neurons that secrete neuropeptide Y (NPY) and agouti-related peptide (ARP), hormones that stimulate appetite, and from proopiomelanocortin (POMC), which promotes satiety. The hypothalamus provides output signals to the pituitary gland, cerebral cortex, motor and autonomic neurons to coordinate endocrine, neuronal, and behavioral responses to maintain food intake and energy balance. Neurons releasing melanocortin receptor ligands reside in the hypothalamic arcuate nucleus and in the nucleus tractus solitarius of the brainstem and are regulated by nutrient sensors and other metabolic signals [13]. Neurons of the arcuate nucleus express receptors for gut hormones and the adipose tissue-derived hormone leptin. Leptin inhibits the NPY/ARP system while stimulating the POMC system, strongly favoring appetite suppression [14]. Leptin resistance is argued to be one of the main features of obesity, suggesting appetite is not suppressed sufficiently by this peptide [14]. Endogenous cannabinoids stimulate both NPY and ARP while amphetamines and cocaine analogs stimulate the melanocortin system. The gut-derived hormone

PYY competes with NPY for the same receptor, stimulating satiety. Integrative neural processing in response to food stimuli takes place in several brain centers including bilateral insular and opercular cortices, the left orbitofrontal region, and putamen [15]. The homeostasis center primarily regulates food intake to maintain energy balance.

The brain centers involved in pleasure and reward are activated when we eat, and the neurotransmitter dopamine plays a significant role in the neuronal control of feeding behavior. Feeding is associated with dopamine release [16], and the amount of dopamine release positively correlates with perceived food pleasure [17]. In laboratory experiments, dopamine deficient animals die of starvation. Weight gain brought on by overeating is thought to be in part due to an imbalance between the homeostatic and reward networks, where reward seeking overrides satiety. Palatable rewarding food can motivate intake even in a state of energy sufficiency and satiety. Obese individuals have lower striatal concentrations of the D2 dopamine receptor [17], findings suggesting that the lower concentrations of this GPCR may evoke overeating in obese individuals in order to produce a reward response. An alternative interpretation is that dopamine receptors may be down-regulated in response to excessive food stimuli.

The pursuit of pleasure influences both food intake and preference. Humans and animals eat the foods they find to be palatable and rewarding. Brain reward processes likely evolved in response to natural rewards, such as the presence of food and the pleasant sensations derived from olfaction, taste, and texture. Animal studies suggest that even simple behaviors motivated by reward, such as returning to a palatable food, are a complicated interplay of multiple processes including sensory perception, motor ability, learning, and memory. The endogenous opioid system also plays a central role in driving food reward-related behavior, and broad expression of opioid precursor proteins and receptors are found throughout the brain [18]. There are extensive functional interactions between the dopamine and opioid systems within the reward circuitry, suggesting a role of these molecules in regulating food intake and preference [19]. Opioid activity in the brain enhances liking for sweet taste, and opioid agonists increase food intake. Over-activation of reward networks by palatable foods may be involved in food cravings.

In addition to reward and homeostatic networks, cognitive control plays a central role in controlling food intake. Cognitive control involves decision making, and is exhaustible in the short term [20], can be enhanced with practice [21], and is correlated with measures of intelligence [22]. Novel approaches including functional magnetic resonance imaging (fMRI) have been used to study self-control, neural activity, and eating behavior. Hare et al. measured activity in the brain while self-reported dieters made real decisions about which foods to eat. Participants performed three tasks during brain imaging. They rated 50 different food items for taste and health value, and were asked to choose between these foods and a reference food which subjects had rated neutral in both taste and health. Participants assigned a goal value term to refer to the amount of expected reward associated with consuming the food. Because participants were selected because they were dieting, the research model relied on the hypothesis that participants should be concerned with both the perceived healthiness as well as their taste preference, and that decision making involves integrating both these concerns and that individuals exercise self-control when making a choice toward a food rated more highly for health verses taste. Results of the study showed that activity in an area of the brain called the ventral medial pre frontal cortex (vmPFC) was correlated with integration of competing factors (health and taste) and with short term goal values (taste) regardless of the amount of self-control, while activity in the dorsal lateral pre frontal cortex (dlPFC) increased when subjects exercised self-control. The study authors concluded that the vmPFC drives short term choices and reward regardless of the degree of self-control while a different brain area, the dlPFC plays a critical role in higher order reasoning involving long term considerations and the exercise of self-control [23].

Other eating behavior studies utilizing fMRI have been used to identify brain loci regulating food intake behaviors in response to a variety of conditions.

Studies indicate that fasting increases cortical activation among lean individuals [15], increases preference for high calorie foods in obese individuals [15, 24, 25], and that obese men have attenuated postprandial brain reactions to satiety which may explain excess caloric intake [26]. In addition to increasing subjective hunger, ghrelin infusion in normal-weight volunteers produced increased fMRI signal intensity in the amygdala, orbitofrontal cortex, anterior insula, and striatum in response to food pictures, areas of the brain involved in activating ingestive behavior [27]. Likewise, subjects with lower leptin levels secondary to weight loss or genetic leptin deficiency have increased fMRI signal activity in brain areas involved in emotional, cognitive, and sensory control of food intake in response to food stimuli, which subsequently normalize with leptin infusion [28, 29]. Martin et al. demonstrated that brain regions involved in pathways of food reward in obese individuals exhibited increased fMRI signal activity, specifically the limbic region, and prefrontal regions, both which have high concentrations of dopamine receptors [30]. Obese individuals also had greater memory for foods in the fasted state. Fasted obese individuals have also been shown to exhibit higher premeal activation of the anterior cingulated cortex and medial prefrontal cortex, areas of the brain implicated in motivational processing [30]. Increased fMRI signal in the prefrontal and limbic regions have also been observed in response to taste stimuli among fasting obese individuals [31].

Taste and olfaction strongly affect eating behavior and nutrient intake. The density of taste papillae on the tongue, genetic differences in taste receptors or sensitivity of taste receptors, constituents of saliva, and other factors all contribute to an individual's taste perception and subsequent food preferences [32]. An individual's sensitivity to the tastes evoked by certain compounds in foods (i.e., sweet, salty, sour, bitter) is strongly affected by genetic variation [36–38]. Gustatory and olfactory function tends to decline with age and impacts appetite and food preference in older individuals. Total caloric intake and food variety are also generally reduced with aging.

Bitter taste perception affects food preference and intake [33–37]. Bitter taster and nontaster phenotypes have been associated with preference for sucrose and sweet tasting foods and beverages, with tasters having reduced preference for sweets [38, 39]. Differences in bitter taste perception have also been associated with preferences for high-fat food [36], avoidance of specific fruits and vegetables [37, 40, 41], alcohol intake, and tobacco use [42, 43]. The principal genetic determinants of phenotypic variation in bitter taste sensitivity are alleles of the gene *TAS2R38* [44]. In a study reported by Wang et al., a common haplotype in *TAS2R38* which confers greater sensitivity to the bitter compound propothiouracil, "PAV" was significantly associated with lower mean alcohol consumption compared with other haplotypes [45]. The *TAS2R38* bitter taster haplotype "PAV" is also associated with smoking quantity in African American smokers. It has been suggested that heightened oral sensitivity may confer protection against nicotine dependence [46].

Family and twin studies provide evidence for a genetic component to eating behavior and food preference [47, 48]. The Three-Factor Eating Questionnaire (TFEQ) is a tool often used to qualify eating behaviors and quantifies eating behaviors in normal-weight and obese persons as well as in subjects with eating disorders such as anorexia nervosa, bulimia nervosa, and binge eating disorders. The three eating behavioral traits assessed by the TFEQ are the cognitive restraint of eating, disinhibition, and susceptibility to hunger [49]. Restraint involves avoidance of eating to control body weight, while disinhibition is loss of restraint that results in overeating. Hunger measures the perceived need for food. Heritability estimates for hunger from family and twin studies using the TFEQ reach approximately 8–28 %; estimates for disinhibition, 18–45 %, and 6–58 % for restraint [50–54]. Disinhibition and hunger are generally positively correlated with obesity, while restraint is negatively correlated with body mass index (BMI) and body fat [52–61]. In general, a high level of restraint or a decrease in disinhibition is associated with greater weight loss during dieting [62, 63] and better weight maintenance after weight loss [64, 65]. Persons who binge eat, have bulimia nervosa, or anorexia nervosa are characterized by dysfunctional levels of cognitive dietary restraint, disinhibition, and susceptibility to hunger compared with normal subjects [66–68].

Genome-wide linkage analyses provide an opportunity to identify chromosomal regions (loci) harboring genes influencing complex traits, including eating behaviors and have revealed several functional candidate genes affecting eating behavior. The first linkage analysis of eating behavior was performed in an Amish cohort, and identified four chromosomal regions or quantitative trait loci for eating behaviors assessed by the TFEQ. Of the genetic loci identified, the strongest evidence was found with the traits restraint and disinhibition [52]. Modest evidence for linkage on chromosome 3 with both restraint as well as obesity-related traits was identified in the Amish [52, 69]. A chromosome 6 loci in proximity to the genes encoding the GLP-1 receptor, tumor necrosis factor α (TNF-α), and lymphotoxin was also associated with restraint scores. GLP-1 has been shown to be involved in eating behavior in human and animal models [70]. GLP-1 receptors are found in the hypothalamus and are the target for hypothalamic neurons containing glucose transporter 2 and glucokinase, proteins involved in glucose sensing and metabolism. TNF-α has been implicated in the development of insulin resistance and obesity [71, 72]. In the Amish study, analysis of disinhibition scores yielded suggestive evidence for linkage to a region on chromosome 7 in proximity to the leptin gene [52]. In a similar study, Bouchard et al. identified a loci on chromosome 15 and implicated *neruomedin beta* to be associated with disinhibited eating behavior and obesity [50].

GAD (glutamic acid decarboxylase) has also been linked to eating behavior. GAD decarboxylates glutamate into GABA (γ-aminobutyric acid), a major inhibitory neurotransmitter in the brain which interacts with NPY in the paraventricular nucleus to stimulate food intake [73]. Two specific *GAD* variants, rs7908975 and rs992990 have been reported to be associated with disinhibition and disordered food intake, specifically increased carbohydrate intake, in women [74]. Additionally, disinhibition scores in Amish women have been linked to genetic variation in the GPCR *TAS2R38* [75].

Polymorphisms in genes including those encoding hormones that regulate food intake and or their receptors have been associated with food intake behaviors. Monogenic forms of obesity attributed to hyperphagia involve functional changes in genes encoding leptin [76], the leptin receptor [77], the melanocortin 4 receptor [78], the *NTRK2* gene [79], genes at the chromosome 15q11.2 locus resulting in Prader-Willi syndrome [80], prohormone convertase 1/3 [81], and the *MBD5* gene [82]. Genetic variants in brain-derived neurotrophic factor have been strongly linked to the eating disorders anorexia and bulimia [83].

Measuring Food Preference and Intake

There are many different modalities used to measure food preference such as FFQs, hedonic food scales, feeding studies, food records, and open-ended questionnaires; and these tools are often used to study the links between food intake, preference, and disease. There are both limitations and benefits to using these tools in research. FFQs are the traditional method for determining food preference but hedonic food scales have increasingly become popular because self-reported food intake frequency may not reflect true preference. In 1982, Dr. Gladys Block reviewed four different self-report systems of food intake: dietary history approach, 24-h recall, 7-day recall, and 7-day record method [84]. The dietary history method includes any method which consists of an extensive interview designed to elicit the usual or customary diet of that individual. In the 24-h and 7-day recall methods, the subject is asked to recall exact intake in the last 24 h or 7 days, respectively. In the 7-day record method the subject records food intake over 7 days. Each of these methods has its own advantages and disadvantages but overall these methods were found to be time consuming and required training on the part of the interviewer. FFQs were established with the goal of being less time consuming and improving efficiency, and were created by compiling a comprehensive predefined food lists which are often linked to nutrient databases. Subjects identify food items from a standardized list and record consumption frequency for a specified period of time. The nutrient totals are a sum of the frequency of consumption multiplied by the portion size and then by nutrient density. FFQs are beneficial in that they are representative of a person's overall typical food intake, especially in those with high day-to-day variability, are efficient, and do not require much training of the subject or interviewer. They are most useful in retrospective case–control studies [85], and are often used in many epidemiologic studies linking food intake with disease risk and risk phenotypes. However, FFQs do rely on the responder's memory, and as such are subject to reduced reliability compared to direct feeding studies. Standard lists may not include certain foods popular to ethnic groups, and therefore introduce possible error due to inaccurate reporting. Both of these factors can contribute to reduced accuracy in research data obtained from FFQs. Using a FFQ linked to a nutrient database simplifies research aimed at identifying nutrients associated with phenotypes being studied. Data from epidemiologic studies is used, at least in part by scientific and professional organizations (for example, AACE and ADA) and governments in making dietary recommendations.

The hedonic scale is another modality used to measure food preference. This was developed at the Quartermaster Food and Container Institute of the US Armed Forces in 1949. This scale was initially used to measure the food preferences of soldiers in order to design rations for the Armed Forces but was then quickly adopted by the food industry [86]. The hedonic scale uses actual food names or samples presented singly to the subject. The subject then rates each item on a scale, for instance, from "dislike extremely" to "like extremely," based on the assumption that there is a continuum of preferences. It has been noted that longer scales, up to 9 intervals, are more discriminating than shorter scales [87]. Food preferences using a 9-point scale have been shown to be a predictor of dietary intake [88]. The basis of the hedonic scale, in comparison with FFQs, is the realization that how often a person eats a certain food may not reflect that person's true preference for that particular food. Peryam et al. reported advantages to the hedonic scale to include its utility among a wide range of populations, ability to produce meaningful results which indicate a general level of preference for the subject, and the fact that no prior experience is needed to perform the test [87]. This scale has been adapted across a variety of age groups and cultures. Facial hedonic scales have been developed by a number of investigators for use in children under the age of 5. Birch et al. initially performed the most widely used 3-point facial hedonic scale among children. In this scale "like" corresponded to "smile," "just ok" corresponded to "neutral" and "dislike" corresponded to "frown" [89]. Yeh et al. compared the use of a 9-point hedonic scale between American, Korean, Chinese, and Thai subjects regarding preference for various foods. The scale was translated into their respective languages and food samples were able to be designated to cover the entire range of hedonic ratings for each culture [90]. Bartoshuk et al. have reported evidence that suggest the sensory and hedonic properties of sweet and fat vary with BMI. Their data indicate that obese individuals live in different orosensory and orohedonic worlds than do the non-obese. Using a food hedonic scale that involves matching the magnitude of the food sensory scale with magnitude of a non food sensation, these authors report that obese participants experience reduced sweetness, which may intensify fat sensations, and that obese participants like both sweet and fat more than the non-obese do [91].

Tools to measure both food intake and preference have been used in dietary studies, which are useful in examining the role of diet and nutrients in relation to disease and disease development. For example, 422 non-Hispanic white men were evaluated using a FFQ to assess intake of saturated fats, fruits, vegetables, whole grains, and alcohol in order to assess cardiovascular disease risk. Fat preference, intake of fiber-rich foods, and alcohol consumption were the best dietary determinants of cardiovascular disease risk factors like adiposity and waist circumference [92]. Increased waist circumference and BMI were associated with a higher intake of sugar-sweetened carbonated beverages in 10–19 year old Saudi boys [93]. Murphy et al. used self-reported food nutrient intake to reaffirm the inverse association between vitamin C intake and esophageal adenocarcinoma as well as reduced risk of reflux esophagitis seen in other studies [94]. A Chinese group used a structured questionnaire to evaluate dietary intake in patients with known gastric cancer and compared them in a 1:2 matched control group. They hypothesized that an increased salt intake would be associated with gastric cancer based on studies showing that high salt intake produces atrophy and metaplasia of the gastric mucosa. Consumption of fresh vegetables had a mild protective effect with an OR of 0.92 (95 % CI 0.58–0.98) and fruit with an OR of 0.87 (95 % CI 0.67–0.93). The investigators also looked at the salt taste sensitivity threshold of the participants. A higher score was correlated with increased salt food preference and intake (OR 1.57, CI 1.15–2.93) and a diagnosis of gastric cancer with an OR of 5.71 (95 % CI 3.18–6.72) [95]. Ikeda et al. investigated food preferences in dementia patients using a four point questionnaire geared toward the caregiver of the patient. Altered food preference toward sweet foods was a prominent and early feature in the fronto-temporal dementia groups [96]. A 24-h dietary recall was used to assess the relationship between dietary protein intake and microproteinuria. Results showed that dietary protein intake was not associated with microproteinuria in healthy patients. However, patients with both diabetes and hypertension had an increased prevalence of microproteinuria with higher protein intake [97].

Laboratory methods have been developed to directly quantify food intake. The initial laboratory methods predominantly assessed intake of liquids. In 1980, Kissileff et al. developed a Universal Eating Monitor (UEM) which weighed the plate of food using a concealed electronic balance. The device generates precisely the amount of food consumed with the help of a computer and digital program. This tool has been useful in assessing total food intake, rate food intake, deceleration of food intake, and can assess both solids and liquids [98]. Several limitations of using a device such as the UEM or other direct feeding methods exist. Special equipment is costly, often bulky, and must be confined to the laboratory. Subjects must travel to the laboratory for observation, therefore representing an unnatural environment, which may introduce bias into experiments.

Food craving also plays a role in food preference and intake, and tools have been developed to measure this trait as well. Studies have used self-report questionnaires to record cravings; for example, in one study of 1,000 male and female college students, chocolate was reported to be the food that was craved the most. However, there were limitations to this study as is typically the case with self-reported data, as this method can lead to inadequate validation [99, 100]. Developed by White et al. in 2001, the food craving inventory (FCI) is a reliable and valid tool to assess cravings for high-fat foods, sweets, carbohydrates/starches, and fast food fats [101]. The FCI has been used in various studies that examine food craving behavior and has also been useful in assessing obesity and binge eating.

Conclusions

In summary, food intake and preference is a complicated interplay of multiple factors, those which are innate, and likely have a genetic basis, and those that are learned and are a function of our environment. CNS mechanisms responsible for control of food intake have evolved to sense the nutrient and energy levels and to coordinate appropriate responses to adjust energy intake and expenditure. The energy and nutritional value as well as hedonic and emotional aspects of feeding impact food intake and preference which in turn influence health status including BMI. Improved understanding of eating behaviors as well as having valid but efficient and cost-effective means to measure food preference are important to further our understanding of why people choose the foods they do. A better understanding of the basis for food preference is important in developing strategies to prevent the development of nutrition-related diseases including obesity, hypertension, cardiovascular disease, and diabetes.

References

1. Magnusson MK, Arvola A, Hursti UK, et al. Choice of organic foods is related to perceived consequences for human health and to environmentally friendly behavior. Appetite. 2003;40:109–17.
2. McKeown NM, Meigs JB, Liu S, et al. Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham offspring study. Am J Clin Nutr. 2002;76:390–8.
3. Handelsman Y, Blonde L, Bloomgarden ZT, et al. American association of clinical endocrinologists guidelines for clinical practice for developing a diabetes mellitus comprehensive care plan. Endocr Pract. 2011;17:1–53.
4. Dammann KW, Smith C. Factors affecting low-income women's food choices and the perceived impact of dietary intake and socioeconomic status on their health and weight. J Nutr Educ Behav. 2009; 41:242–53.
5. Lawrence W, Skinner C, Haslam C, et al. Why women of lower educational attainment struggle to make healthier food choices: the importance of psychological and social factors. Psychol Health. 2009;24:1003–20.
6. Croker H, Whitaker KL, Cooke L, et al. Do social norms affect intended food intake? Prev Med. 2009; 49:190–3.
7. Harris JL, Bargh JA, Brownell KD. Priming effects of television food advertising on eating behavior. Health Psychol. 2009;28:404–13.
8. Carnell S, Gibson C, Benson L, et al. Neuroimaging and obesity: current knowledge and future directions. Obes Rev. 2012;13:43–56.
9. Sam AH et al. The role of the gut/brain axis in modulating food intake. Neuropharmacology. 2012;63(1): 46–56.
10. Margolskee RF, Dyer J, Kokrashvill Z, et al. T1R3 and gustducin in gut sense sugars to regulate expression of Na^+-glucose cotransporter 1. Proc Natl Acad Sci U S A. 2007;104:15075–80.
11. Cummings DE et al. A preprandial rise in plasma ghrelin levels suggest a role in meal initiation in humans. Diabetes. 2001;50:1714–9.
12. Batterham RI et al. Gut hormone PYY (3–36) physiologically inhibits food intake. Nature. 2002;418: 650–4.
13. Begriche K, Sutton GM, Butler AA. Homeostatic and non-homeostatic functions of melanocortin-3 receptors in the control of energy balance and metabolism. Physiol Behav. 2011;104:546–54.
14. Dietrich MO, Horvath TL. Feeding signals and brain circuitry. Eur J Neurosci. 2009;30:1688–96.
15. Porubska K, Veit R, Preissl H, et al. Subjective feeling of appetite modulates brain activity: an fMRI study. Neuroimage. 2006;32:1273–80.
16. Siep N, Roefs A, Roebroeck A, et al. Hunger is the best spice: an fMRI study of the effects of attention, hunger and calorie content on food reward processing in the amygdala and orbitofrontal cortex. Behav Brain Res. 2009;198:149–58.
17. Stice E, Spoor S, Bohon C, et al. Relation between obesity and blunted striatal response to food is moderated by TaqIA A1 allele. Nutrition. 2009;25: 132–3.
18. Le Merrer J, Becker JA, Befort K, et al. Reward processing by the opioid system in the brain. Physiol Rev. 2009;89:1379–412.
19. Stinus L, Cador M, Le Moal M. Interaction between endogenous opioids and dopamine within the nucleus accumbens. Ann N Y Acad Sci. 1992;654: 254–73.
20. Vohs KD, Baumeister RF, Schmeichel BJ, et al. Making choices impairs subsequent self-control: a limited-resource account of decision making, self-regulation, and active initiative. J Pers Soc Psychol. 2008;94:883–98.
21. Siegel E, Rachlin H. Soft commitment: self-control achieved by response persistence. J Exp Anal Behav. 1995;64:117–28.
22. Tangney JP, Baumeister RF, Boone AL. High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. J Pers. 2004;72:271–324.
23. Hare TA, Camerer CF, Rangel A. Self-control in decision-making involves modulation of the vmPFC valuation system. Science. 2009;324:646–8.
24. Goldstone AP, de Hernandez CG, Beaver JD, et al. Fasting biases brain reward systems towards high-calorie foods. Eur J Neurosci. 2009;30:1625–35.
25. Stoeckel LE, Weller RE, Cook EW, et al. Widespread reward-system activation in obese women in response to pictures of high-calorie foods. Neuroimage. 2008;41:636–47.
26. Le DS, Pannacciulli N, Chen K, et al. Less activation of the left dorsolateral prefrontal cortex in response to a meal: a feature of obesity. Am J Clin Nutr. 2006;84:725–31.
27. Malik S, McGlone F, Bedrossian D, Dagher A. Ghrelin modulates brain activity in areas that control appetitive behavior. Cell Metab. 2008;7:400–9.
28. Ahima RS. Revisiting leptin's role in obesity and weight loss. J Clin Invest. 2008;118:2583–91.
29. Baicy K, London ED, Monterosso J, et al. Leptin replacement alters brain response to food cues in genetically leptin-deficient adults. Proc Natl Acad Sci U S A. 2007;104:18276–9.
30. Martin LE, Holsen LM, Chambers RJ, et al. Neural mechanisms associated with food motivation in obese and healthy weight adults. Obesity. 2010;18: 254–60.
31. Haase L, Cerf-Ducastel B, Murphy C. Cortical activation in response to pure taste stimuli during the physiological states of hunger and satiety. Neuroimage. 2009;44:1008–21.
32. Rolls ET. Taste, olfactory, and food texture processing in the brain, and the control of food intake. Physiol Behav. 2005;85:45–56.
33. Bell KI, Tepper BJ. Short-term vegetable intake by young children classified by 6-n-propylthoiuracil

bitter-taste phenotype. Am J Clin Nutr. 2006;84(1): 245–51.
34. Dinehart ME, Hayes JE, Bartoshuk LM, Lanier SL, Duffy VB. Bitter taste markers explain variability in vegetable sweetness, bitterness, and intake. Physiol Behav. 2006;87:304–13.
35. Drewnowski A, Rock CL. The influence of genetic taste markers on food acceptance. Am J Clin Nutr. 1995;62:506–11.
36. Duffy VB, Bartoshuk LM. Food acceptance and genetic variation in taste. J Am Diet Assoc. 2000; 100:647–55.
37. Yackinous CA, Guinard JX. Relation between PROP (6-n-propylthiouracil) taster status, taste anatomy and dietary intake measures for young men and women. Appetite. 2002;38:201–9.
38. Mennella JA, Pepino MY, Reed DR. Genetic and environmental determinants of bitter perception and sweet preferences. Pediatrics. 2005;115:e216–22.
39. Tepper BJ, Nurse RJ. PROP taster status is related to fat perception and preference. Ann N Y Acad Sci. 1998;855:802–4.
40. Drewnowski A. The science and complexity of bitter taste. Nutr Rev. 2001;59(6):163–9.
41. Sandell MA, Breslin PA. Variability in a taste-receptor gene determines whether we taste toxins in food. Curr Biol. 2006;16:R792–4.
42. Duffy VB, Peterson JM, Bartoshuk LM. Associations between taste genetics, oral sensation and alcohol intake. Physiol Behav. 2004;82:435–45.
43. Cannon DS, Baker TB, Piper ME, et al. Associations between phenylthiocarbamide gene polymorphisms and cigarette smoking. Nicotine Tob Res. 2005;7: 853–8.
44. Myerhof W, Betram C, Kuhn C, et al. The molecular receptive ranges of human TAS2R bitter taste receptors. Chem Senses. 2009;35:157–70.
45. Wang JC, Hinrichs AL, Bertelsen S, et al. Functional variants in TAS2R38 and TAS2R16 influence alcohol consumption in high-risk families of African-American origin. Alcohol Clin Exp Res. 2007;31: 209–15.
46. Mangold JE, Payne TJ, Ma JZ. Bitter taste receptor gene polymorphisms are an important factor in the development of nicotine dependence in African Americans. J Med Genet. 2008;45:578–82.
47. Reed DR, Bachmanov AA, Beuchamp GK, et al. Heritable variation in food preferences and their contribution to obesity. Behav Genet. 1997;27:373–87.
48. Falciglia GA, Norton PA. Evidence for a genetic influence on preference for some foods. J Am Diet Assoc. 1994;94:154–8.
49. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res. 1985;29:71–83.
50. Bouchard L, Drapeau V, Provencher V, et al. Neuromedin beta: a strong candidate gene linking eating behaviors and susceptibility to obesity. Am J Clin Nutr. 2004;80:1478–86.
51. de Castro JM, Lilenfeld LR. Influence of heredity on dietary restraint, disinhibition, and perceived hunger in humans. Nutrition. 2005;21:446–55.
52. Steinle NI, Hsueh WC, Snitker S, et al. Eating behavior in the Old Order Amish: heritability analysis and a genome-wide linkage analysis. Am J Clin Nutr. 2002;75:1098–106.
53. Neale BM, Mazzeo SE, Bulik CM. A twin study of dietary restraint, disinhibition and hunger: an examination of the eating inventory (three factor eating questionnaire). Twin Res. 2003;6:471–8.
54. Tholin S, Rasmussen F, Tynelius P, Karlsson J. Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study. Am J Clin Nutr. 2005;81:564–9.
55. Klem ML, Wing RR, McGuire MT, et al. Psychological symptoms in individuals successful at long-term maintenance of weight loss. Health Psychol. 1998;17:336–45.
56. Hakala P, Rissanen A, Koskenvuo M, et al. Environmental factors in the development of obesity in identical twins. Int J Obes Relat Metab Disord. 1999;23:746–53.
57. Bryant EJ, King NA, Blundell JE. Disinhibition: its effects on appetite and weight regulation. Obes Rev. 2008;9(5):409–19.
58. Carmody TP, Brunner RL, St Jeor ST. Dietary helplessness and disinhibition in weight cyclers and maintainers. Int J Eat Disord. 1995;18(3):247–56.
59. Maus N, Paul T, Pudel V, Westenhöfer J. Behavioral factors involved in the control of food intake in man. Int J Vitam Nutr Res. 1988;58(3):356–66.
60. Provencher V, Drapeau V, Tremblay A, et al. Eating behaviors and indexes of body composition in men and women from the Quebec family study. Obes Res. 2003;11:783–9.
61. Drapeau V, Provencher V, Lemieux S, et al. Do 6-y changes in eating behaviors predict changes in body weight? Results from the Quebec family study. Int J Obes Relat Metab Disord. 2003;27:808–14.
62. Westenhoefer J, Pudel V, Maus N. Some restrictions on dietary restraint. Appetite. 1990;14(2):137–41; discussion 142–3.
63. Pekkarinen T, Takala I, Mustajoki P. Two year maintenance of weight loss after a VLCD and behavioural therapy for obesity: correlation to the scores of questionnaires measuring eating behavior. Int J Obes Relat Metab Disord. 1996;20:332–7.
64. Westerterp-Plantenga MS, Kempen KP, Saris WH. Determinants of weight maintenance in women after diet-induced weight reduction. Int J Obes Relat Metab Disord. 1998;22:1–6.
65. Foster GD, Wadden TA, Swain RM, et al. The eating inventory in obese women: clinical correlates and relationship to weight loss. Int J Obes Relat Metab Disord. 1998;22:778–85.
66. Gendall KA, Sullivan PF, Joyce PR, et al. Food cravings in women with a history of anorexia nervosa. Int J Eat Disord. 1997;22:403–9.
67. Faith MS, Johnson SL, Allison DB. Putting the behavior into the behavior genetics of obesity. Behav Genet. 1997;27:423–39.
68. Bulik CM, Sullivan PF, Fear JL, et al. Outcome of anorexia nervosa: eating attitudes, personality, and parental bonding. Int J Eat Disord. 2000;28:139–47.

69. Hsueh WC, Mitchell BD, Schneider JL, et al. Genome-wide scan of obesity in the old order Amish. J Clin Endocrinol Metab. 2001;86:1199–205.
70. Gutzwiller JP, Drewe J, Goke B, et al. Glucagon-like peptide-1 promotes satiety and reduces food intake in patients with diabetes mellitus type 2. Am J Physiol. 1999;276(5):R1541–4.
71. Fernandez-Real JM, Gutierrez C, Soler J, et al. The TNF-alpha gene *NCO* I polymorphism influences the relationship among insulin resistance, percent body fat and increased serum leptin levels. Diabetes. 1997;46:1468–72.
72. Hotamisligil GS, Arner P, Caro JF, et al. Increased adipose tissue expression of tumor necrosis factor alpha in human obesity and insulin resistance. J Clin Invest. 1995;95:2409–15.
73. Boutin P, Dina C, Vasseur F, et al. GAD2 on chromosome 10p12 is a candidate gene for human obesity. PLoS Biol. 2003;1:E68.
74. Choquette AC, Lemieux S, Tremblay A, et al. GAD2 gene sequence variations are associated with eating behaviors and weight gain in women from the Quebec family study. Physiol Behav. 1998;4:505–10.
75. Dotson CD, Shaw HL, Mitchell BD, et al. Variation in the gene TAS2R38 is associated with the eating behavior disinhibition in Old Order Amish women. Appetite. 2010;54:93–9.
76. Strobel A, Issad T, Camoin L, et al. A leptin missense mutation associated with hypogonadism and morbid obesity (letter). Nat Genet. 1998;18:213–5.
77. Clement K, Vaisse C, Lahlou N, et al. A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction. Nature. 1998;392:398–401.
78. Yeo GH, Farooqi IS, Aminian S, et al. A frameshift mutation in MC4R associated with dominantly inherited human obesity. Nat Genet. 1998;20:111–2.
79. Yeo GH, Hung CC, Rochford J, et al. A de novo mutation affecting human TrkB associated with severe obesity and developmental delay. Nat Neurosci. 2004;7:1187–9.
80. Magenis RE, Toth-Fejel S, Allen LJ, et al. Comparison of the 15q deletions in Prader-Willi and Angelman syndromes: specific regions, extent of deletions, parental origin, and clinical consequences. Am J Med Genet. 1990;35:333–49.
81. Farooqi IS, Volders K, Stanhope R, et al. Hyperphagia and early-onset obesity due to a novel homozygous missense mutation in prohormone convertase 1/3. J Clin Endocrinol Metab. 2007;92:3369–73.
82. Talkowski ME, Mullegama SV, Rosenfeld JA, et al. Assessment of 2q23.1 microdeletion syndrome implicates MBD5 as a single causal locus of intellectual disability, epilepsy, and autism spectrum disorder. Am J Hum Genet. 2011;89:551–63.
83. Ribases M, Gratacos M, Fernandez-Aranda F, et al. Association of BDNF with anorexia, bulimia and age of onset of weight loss in six European populations. Hum Mol Genet. 2004;13:1205–12.
84. Block G. A review of validations of dietary assessment methods. Am J Epidemiol. 1982;115(4): 492–505.
85. Kristal AR, Shattuck AL, Williams AE. Food frequency questionnaires for diet intervention research. http://www.nutrientdataconf.org/PastConf/NDBC17/5-5_Kristal.pdf. Retrieved 1 Oct 2011.
86. Jones LV, Peryam DR, Thurstone LL. Development of a scale for the measuring of soldiers' food preferences. J Food Sci. 1955;20(5):512–20.
87. Peryam DR, Pilgram FJ. Hedonic scale method of measuring food preferences. Food Technol. 1957;11:9–14.
88. Drewnowski A, Hann C. Food preferences and reported frequencies of food consumption as predictors of current diet in young women. Am J Clin Nutr. 1999;70:28–36.
89. Birch L, McPhee L, Steinberg L, et al. Conditioned flavor preferences in young children. Physiol Behav. 1990;47:501–5.
90. Yeh LL, Kim KO, Chompreeda P, et al. Comparison in the use of the 9-point hedonic scale between Americans, Chinese, Koreans, and Thai. Food Qual Pref. 1998;9:413–9.
91. Bartoshuk LM, Duffy VB, Hayes JE, et al. Psychophysics of sweet and fat perception in obesity: problems, solutions and new perspectives. Philos Trans R Soc Lond B Biol Sci. 2006;361:1137–48.
92. Duffy VB, Lanier SA, Hutchins HL, et al. Food preference questionnaire as a screening tool for assessing dietary risk of cardiovascular disease within health risk appraisals. J Am Diet Assoc. 2007;107:237–45.
93. Collison KS, Zaidi MZ, Subhani SN, et al. Sugar-sweetened carbonated beverage consumption correlates with BMI, waist circumference, and poor dietary choices in school children. BMC Public Health. 2010;10:1–13.
94. Murphy SJ, Anderson LA, Ferguson HR, et al. Dietary antioxidant and mineral intake in humans is associated with reduced risk of esophageal adenocarcinoma but not reflux esophagitis or Barrett's esophagus. J Nutr. 2010;140:1757–63.
95. Yang WG, Chen CB, Wang ZX, et al. A case–control study on the relationship between salt intake and salty taste and risk of gastric cancer. World J Gastroenterol. 2011;170:2049–53.
96. Ikeda M, Brown J, Holland AJ, et al. Changes in appetite, food preference, and eating habits. J Neurol Neurosurg Psychiatry. 2002;73:371–6.
97. Wrone EM, Carnethon MR, Palaniappan L, et al. Association of dietary protein intake and microalbuminuria in healthy adults: Third National Health and Nutrition Examination Survey. Am J Kidney Dis. 2003;41:580–7.
98. Kissileff HR, Klingsberg G, Van Itallie TB. Universal eating monitor for continuous recording of solid versus liquid consumption in man. Am J Physiol. 1980;238:R14–22.
99. Weingarten HP, Elston D. The phenomenology of food cravings. Appetite. 1990;15:231–46.
100. Weingarten HP, Elston D. Food cravings in a college population. Appetite. 1991;17:167–75.
101. White MA, Whisenhunt BL, Williamson DA, et al. Development and validation of the food craving inventory. Obes Res. 2002;10:107–14.

3 An Evidence-Based Approach to the Nutritional Quality of Home Meals: Exploring Emotional Reinforcement and Attachment Style as Underlying Mechanisms

Ji Lu, Aida Faber, and Laurette Dubé

Key Points

- To better understand the superior nutritional quality of home meals vs. away-from-home meals, two protective mechanisms are proposed and empirically explored: emotional reinforcement and attachment style.
- Beyond the biological reinforcement process, in which eating is naturally rewarded by sensory and hedonic experiences, emotions can be associated with eating, adding emotional reinforcing properties to food consumption.
- Home may be a place of foremost importance for emotional reinforcement in that, compared to away-from-home settings, more positive emotions are experienced and can be attached to food consumed in this setting. As such, emotional reinforcement processes particularly benefit healthy foods, because they are generally lacking biological reinforcing properties.
- It is highly plausible that emotional reinforcement as a protective mechanism of home eating starts from early stage of life through the parent/child attachment relationship where both primary caregivers and food are the two most central objects in a child's life.
- By providing a secure environment to explore and learn—mainly the home environment—nurturing and caring parents establish a secure attachment with their children. Such secure attachment styles promote emotional reinforcement and become internalized, forming the base for life-long healthy eating patterns.

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- This mechanism was empirically explored in a survey about attachment style and high fat/high sugar food consumption. For children and adults, more trusting bonds (lower avoidance) and lower anxiety and worry about their relationship with their own parents (lower anxiety) were linked with lower consumption of unhealthy foods.
- Potential interventions and future studies are discussed.

Keywords
Eating behavior • Food choices • Reinforcement • Emotions • Home meals • Attachment style • Avoidant attachments • Anxious attachment • Food consumption

Introduction

It is widely observed that food consumption, in terms of its nutritional quality, is typically healthier at home than in away-from-home settings [1, 2]; supporting evidences were extensively documented in the chapter by Woodruff and Hanning showing an important association between family meals and diet quality. This leads to the present chapter's theoretical interest of taking an evidence-based approach to further explore the underlying mechanisms contributing to the protective effects of home on healthy eating. Using theoretical rationales and empirical examinations, this chapter proposes that emotional reinforcement and parent/child attachment style are potential protective mechanisms accounting for healthier eating patterns occurring at home. Particularly, we will first examine evidence revealing that positive moods experienced at home can be associated with food consumption, especially with healthy food intake, therefore increasing the emotional reinforcing value of healthy meals at home. We will then demonstrate that a trusting and caring parent/child relationship starting as early as birth and internalized as a secure attachment style may facilitate such an emotional reinforcement and lead to a life-long healthier eating habits.

Closely tied to the pleasure brought by the taste of food, eating is generally rewarded by biological signals sent by the sensory and hedonic systems. This biological reinforcing property of food is particularly salient for unhealthy foods (high in sugar and/or fat) because of their superior palatability compared to healthy foods (low in sugar and/or fat) [3]. Beyond biological pleasure, emotional experiences can also be associated with eating through an emotional reinforcement process [4]. It has been shown that the home as opposed to the outside environment (work places, schools, etc.) is a more intense source of positive emotional experiences [5]. Through an associative learning process, the positive affective states experienced at home can become attached to food consumed in this setting, assigning home meals a superior emotional reinforcing value. Moreover, because healthy food lacks a strong biological reinforcing property, emotional reinforcement tends to manifest its protective effect particularly on this type of food which is low in sugar and/or fat. Interestingly, these associations between positive emotions and food start as early as birth; mostly within home settings, where sensitive and caring parents provide important opportunities to infants to experience with food and eating in a calm and safe environment [6]. Over time, these experiences are internalized and constitute the cornerstone for individuals to interpret emotions and assign emotional value to eating experiences [6]. Therefore, the home becomes a haven where positive emotions are more likely to arise and, through powerful and early conditioning, to become associated with healthier food consumption which lacks the high biological reinforcement of unhealthy food alternatives.

Identifying processes through which eating at home becomes associated with healthy eating habits is essential, not only because home meals still represent the bulk of food consumption worldwide, but also because food habits are formed in the first 5 years of life (mostly at home) and serve as a foundation for eating throughout the life course [7]. Understanding these mechanisms will allow for the development of interventions that may impact eating and healthy lifestyles, and that can be implemented as early as birth.

Emotional Reinforcement as a Protective Mechanism of Home

Reinforcement and Food Consumption

Reinforcement is a process of associating a behavior with contingent positive consequences or the alleviation of negative states as rewards [8]. Food is a powerful primary reinforcer in that (1) eating is repeatedly paired with biological positive outcomes, such as pleasant taste and the decrease of hunger feelings; and (2) the learned association between food and such positive occurrences motivates further behaviors to obtain it, therefore, food consumption can be triggered by physiological signals of hunger and/or olfactory and visual food cues that suggest opportunities to get reward.

Furthermore, foods vary in their biological reinforcing qualities, with highly reinforcing foods being favored over less reinforcing ones [9]. The biological reinforcing quality of a specific food is closely related to the sensory and hedonic signals triggered by the subjective pleasure of taste which conceptually incorporates palatability, smell, and food texture. It has been shown that individuals inherently prefer unhealthy foods, which are high in sugar and/or fat (e.g., French fries, pizza, or ice cream), finding these foods tastier and therefore more biologically reinforcing [10, 11], compared to healthy foods (e.g., whole grains, low fat yogurt, or vegetables). Moreover, individuals are biologically programmed to avoid bitter taste as it is associated with poison. This makes some vegetables such as Brussels sprouts and broccolis not only less preferred because of their lack of biological reinforcement qualities but sometimes outright rejected regardless of their beneficial nutritional qualities [12].

Emotions are often at the core of the reinforcement process influencing motivated behavior [8]. For eating, immediate taste evaluation along the dimension of positive–negative food acceptability activates the entire affective system, triggering not only simple hedonic experiences (like/dislike) but also more differentiated affective states such as enjoyment, accomplishment, sadness, anxiety, melancholy, or guilt [13]. Furthermore, food consumption is a complex behavior; beside the biological signals related to the appreciation of palatability, eating can be reinforced by emotional experiences and motivated by emotional regulation needs. As shown in our published studies, the consumption of comfort food can result in increased positive emotions or provide alleviation from negative moods [14]. Such associations between food and emotional comfort, carved in the mind as a schema, assigns emotional reinforcing properties to food consumption [15]. We further showed in these studies that individual emotional experiences associated with comfort food are critically moderated by social-cultural factors, such as gender and cultural background. This evidence points to the importance of postnatal environment (e.g., the context that individuals consistently experience both food and certain emotions) in the emotional reinforcement process.

Home Environment and the Emotional Reinforcement of Food

Focusing on positive emotions, we propose that emotions arising from the consumption context can be associated with the food consumed, and can be integrated into the reinforcing value as a contingent consequence rewarding consumption or as an antecedent state triggering eating. It has been shown that enjoyable social interactions and pleasant environments [16], if stably paired with eating, may become associated with food consumption, altering its reinforcing properties and ultimately influencing food choices [13]. As a unique set of social-cultural and physical-environmental factors, the home can greatly influence the reinforcing quality of food through an associative learning process. Individuals typically experience more intense positive emotions and less intense negative emotions at home than in work places and other away-from-home contexts [5, 17] and tend to associate such ambient affects with the ongoing activities they perform in this environment [18]. Through repeated interactions with food at home in the presence of positive emotions, people may come to associate these positive affective states with food consumed at home and attach an emotional reinforcing value to home meals.

We further suggest that one of the protective mechanisms of the home environment is that the higher emotionally reinforcing value attached to food eaten at home should be particularly salient for healthy meals rather than unhealthy ones. As previously mentioned in this chapter, unhealthy food is biologically rewarded primarily by its desirable taste. Because of the powerful influence of biological signals [19], when consuming high fat/sugar foods, individuals tend to focus their attention to the hedonic experiences brought by the food and are less likely process contextual information, including emotional signals, from their surrounding environment [20]. Therefore, positive emotional states tied to the home are less likely to be actually experienced and hence are hard to attach to foods rich in sugar and fat. On the other hand, healthy meals lack an inherent biological reinforcing value effectively leaving space for the emotional reinforcing quality to be attached to them. Hence, without the attention-narrowing effect derived from biological reward, the superior emotional states of home, compared to away-from-home settings, can be more saliently experienced when consuming healthy food. If stably paired with each other, the superior value of positive contextual emotions becomes associated with healthy meals consumed in the same environment, adding an emotional reinforcing value to healthy food.

As a result of such an associative learning process, which involves a set of emotions, healthy foods, and the home as a physical-psychological context, pre-consumption positive emotions that align with the rewarding property of healthy foods may serve as triggering cues to motivate behavior to pursue healthy eating, especially at home. This proposition is consistent with studies exploring the effects of positive emotions, as antecedent states, on food intake. For example, it has been shown that positive emotions, such as happiness, calm, and joy, can increase an individual's motivation to eat [21] particularly triggering healthy food consumption and healthful food preferences [14].

To empirically explore the proposed protective mechanism of home on healthy eating, we will shortly review our published empirical study [22] to illustrate how meal location (home vs. away-from-home) and emotions (positive vs. negative) interact to influence food choice (healthy vs. unhealthy) and discuss some methodological issues related to home meal research.

An Empirical Examination of Food, Emotions, and Location

Baseline Meal Food Choice Pattern

The primary goal of this study was to test the hypothesis that home is a central setting, where eating, especially healthy eating, is emotionally reinforced. In the midst of everyday life, contextual factors affect individual choice and behavior in each occasion; while across occasions (e.g., breakfast, lunch, and dinner), stable patterns of behavior can be identified and individual differences regarding such stable patterns can be manifested [23]. It has been consistently shown that food choices vary systematically across daily meals, with low-caloric foods most likely being chosen at breakfast, while the tasty, caloric-intense food more likely being chosen at dinner [24]. Such habitual eating patterns were referred as baseline habits, and individuals were found generally to exhibit these baseline habits in their food choices [23]. Such baseline habits are mainly shaped by social-cultural forces [25] and are relatively independent of emotional reinforcing value. Therefore, an empirical comparison of

Table 3.1 Emotional factor structure

Factor components	Cronbach's alpha	Emotion items			
General positive emotions	0.88	Joyful	Happy	Elated	Amused
		Optimistic	Hopeful	Encouraged	
		Fulfilled	Accomplished		
		Warmhearted	Loving	Sentimental	
Positive emotions—calm	0.78	Serene	Calm	Peaceful	Contended
General negative emotions	0.82	Frustrated	Annoyed	Angry	
		Miserable	Sad	Depressed	
Negative emotions—shame	0.60	Ashamed	Guilty	Embarrassed	
Negative emotions—anxiety	0.74	Nervous	Tense	Worried	

Participants reported their momentary emotional states for every episode on a 28-item scale of emotions adapted from the Consumption Emotions Set. Participants were asked to indicate the degree to which they were feeling each emotion at that present moment by placing a mark on a 15-cm visual analog scale (rated from "not at all" to "very much"). Factor structure was extracted based on Chain-P factor analysis (Factor Extraction Method: Principal Component Analysis. Rotation Method: Varimax with Kaiser Normalization)

emotional reinforcing value across meals with healthy or unhealthy content should be based on each meal's relative deviation from an individual's baseline habit rather than on absolute nutritional quality of a meal.

Emotional Reinforcing Property of Meals

Our empirical study operationalizes emotional reinforcing value as an association between positive mood and food consumption. First, having a home meal, if it is more emotional reinforcing than other meals, should result in a better mood as a positive emotional consequence. Therefore, a better post-meal affective state is one of the indicators suggesting a higher emotional reinforcing value being attached to home meals. Second, within the home setting, if healthier meals are more emotional reinforcing than unhealthy meals, then the healthier meals are expected to trigger more intensive positive and/or less negative post-meal affective states. Third, as an antecedent state, positive mood can serve as a conditioned cue to motivate a reward seeking behavior. Hence, another way to operationalize emotional reinforcing value is to examine the premeal affective states as a trigger of food choice. As a context of associative learning, the home setting activates or enhances the associative link between healthy meals and positive affective states, while the effect of such emotional reinforcement may not be as powerful in away-from-home settings. Hence, healthier meals, especially those consumed at home, should be preceded by more positive pre-meal emotion and/or less negative emotions.

Research Paradigm and Results

We used experience sampling method [26], in which every participant (160 Caucasian adult non-obese women) was observed on a large number of meals occurring over a short period of time. This method allowed us to examine each participant's food choice at a meal level, account for the nutritional quality of each meal in terms of its deviance from one's baseline eating pattern, and to map the relationship between emotional states and food choice. During 10 observation days, participants were asked to report 6 times a day on their momentary emotional states (see Table 3.1 for the measured emotion items) and on their eating behavior. Participants were asked to indicate the nutritional quality for each meal in a relative form compared to their usual (baseline) pattern in the same meal occasion (healthier, less healthy, vs. baseline meal). Participants indicated the location of the meal, that is whether the meal episode had been taken at home or away-from-home (see Table 3.2 for the frequencies of observed meals).

Table 3.2 The frequencies (odds ratios) of reported nutritional quality for home meals and away-from-home meals

	Nutritional quality		
	Healthier[a]	Baseline	Less healthy[b]
Away from home	167 (0.29)	568	174 (0.31)
Home	345 (0.18)	1,903	311 (0.16)
Total	512	2,471	485

Separated for home and away-from-home settings, this table shows the number of episodes that participants reported as having baseline, healthier than usual, and less healthy than usual meals

[a]Odds ratios in this column are based on healthier/baseline

[b]Odds ratios in this column are based on unhealthy/baseline

In the examination of the post-meal affective states, expectations for the higher general reinforcing value of eating at home vs. away-from-home and for a stronger effect in the specific case of healthy meals were supported. Specifically, participants rated higher on calm feeling and less anxiety across all meals at home compared to away-from-home meals. Moreover, only at home, having healthier meals resulted in more positive emotions compared to baseline meals, and no such facilitation effect was found for meals eaten away-from-home (see Fig. 3.1). In fact, for away-from-home meals, consuming healthier meals

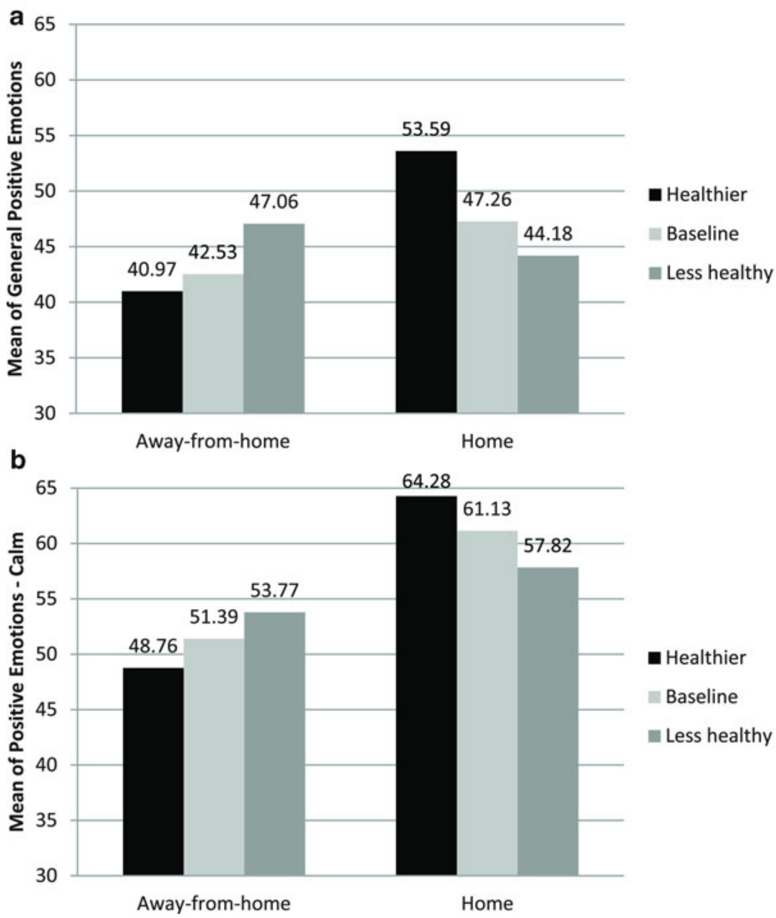

Fig. 3.1 (**a**) General positive emotions. (**b**) Positive emotions—calm. Means of post-meal positive emotions after home vs. away-from-home meals with different nutritional quality. Marginal means reported in this table are for demonstration purpose. Hypotheses were tested by hierarchical linear models, in which the day-centered emotion scores (the deviation from day-level mean) for each meal were explained by the location (home vs. away from home), nutritional quality, and the interaction between them, controlled by social context (eat alone vs. with others) and meal size (larger than usual, baseline, or less than usual)

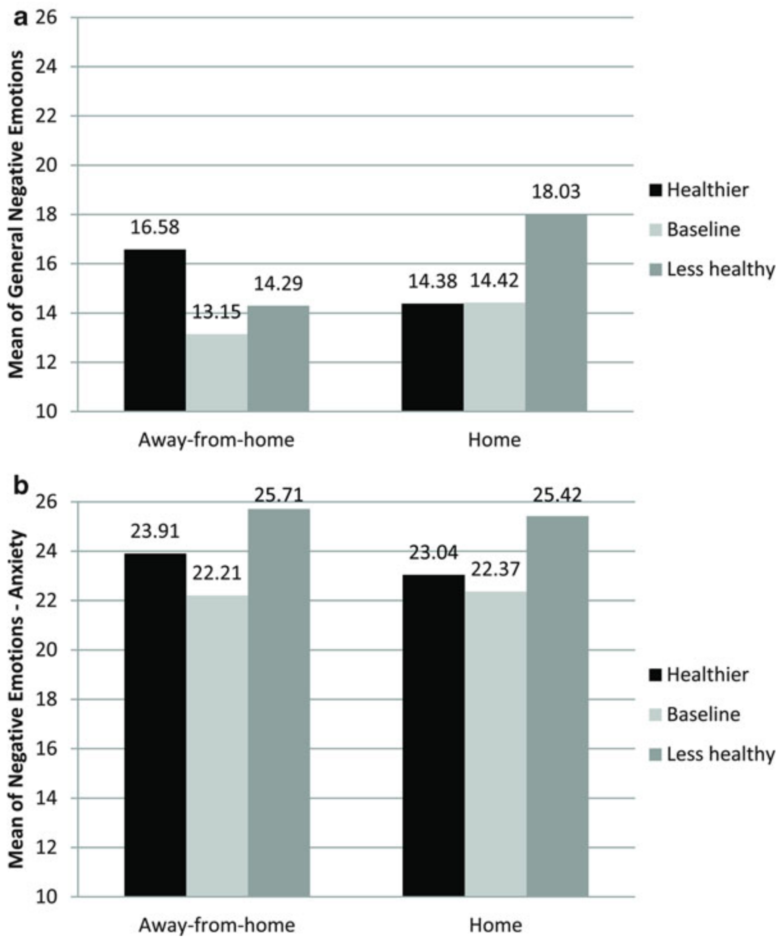

Fig. 3.2 (**a**) General negative emotions. (**b**) Negative emotions—anxiety. Means of post-meal negative emotions after home vs. away-from-home meals with different nutritional quality. Marginal means reported in this table are for demonstration purpose. Hypotheses were tested by hierarchical linear models, in which the day-centered emotion scores (the deviation from day-level mean) for each meal were explained by the location (home vs. away from home), nutritional quality, and the interaction between them, controlled by social context (eat alone vs. with others) and meal size (larger than usual, baseline, or less than usual)

was associated with more negative emotions, compared to baseline meals (see Fig. 3.2). For the analyses of premeal emotional states as a trigger of healthy eating, the relative nutritional quality of a given meal (healthier, baseline, or less healthy) was predicted by meal location and the emotions reported before the meal. Particularly, the results supported the proposition that more positive emotional experiences precede healthier eating choices in a home setting. Specifically at home, more intense premeal positive emotions were predictive of higher likelihood of choosing a healthier meal over baseline meal, while for away-from-home meals, premeal positive emotions showed no effect on the nutritional quality of meals chosen.

As a further source of evidence to paving the way to a more theoretical understanding of positive emotional reinforcing and food, we next present evidence of the importance of parent/child attachment, most of it happening in a home setting, which is also linked to healthier eating.

The Role of Attachment Style in Eating

As shown previously, not only emotional reinforcement is a protective mechanism for healthy eating at home as demonstrated through the above-reported study on emotions and food consumption context, but also it is highly plausible that this association starts from birth through the parent/child attachment relationship where both primary caregivers and food are the two most central objects in a child's life.

Research shows that eating habits are developed early in life and remain imprinted in habits and preferences of adult life [27]. Child/caregiver attachment is the first process through which biological cues (i.e., hunger) become linked with behavior (i.e., feeding) in a social context (parent/child interaction). In the domain of food and food choices (as in all domains), responsive and sensitive parents provide children with a calm and peaceful, albeit challenging, environment where they can explore and learn about food and eating in a safe environment [6]. By encouraging children to master new food experiences in a positive context, nurturing parents promote exploratory behaviors that allow children to become autonomous and knowledgeable about eating and food and that may come to supersede an instinctive preference for high reward/high sugar and or/fatty foods, promoting healthier eating habits. We hence propose that early child/parent attachment relationship is potentially a critical protective/risk factor shaping a life course of food choices and eating behaviors.

Birch has shown that children's food preferences are very much influenced by their early learning experiences, where repeated positive associations with food in an unpressured fashion is key into developing healthy eating habits and accepting new foods [27]. As such, early infancy is a critical stage where most meals are consumed at home [27] and where, positive exploratory experiences with an increased variety of foods in a safe haven context (the home) fostered by the availability of a nurturing caregiver become internalized and contribute to healthier eating patterns. However, from a parent/child attachment perspective, inadequate parental caring, through lack of consistency and supportiveness or outright rejection, does not allow for optimal exploration and positive associations with food and eating in a calm and safe context. Consequently, these children are more likely to show an unhealthy food consumption pattern. Accordingly, although the home is linked with more positive emotions, quality of caretaker/child experience within the home context can be more or less optimal and influence differentially food consumption. This theorizing is also in line with Woodruff and Hanning chapter about family meals and diet quality, showing that within the home, particular patterns are more (high family cohesion, authoritarian parenting style) or less (television watching) conducive to healthy eating.

To understand the differential role of parent/child attachment style and food consumption, we will first shortly review attachment style formation together with its typology, followed by the role of attachment style in eating and evidence of how attachment style is linked with food consumption in adults and children.

Attachment Style Theory and Typology

Pioneered by Bowlby [28], the attachment theory is a general theory of personality that aims to explain how the quality of early caretaker/child relationship interacts with the social and physical environment to become the cornerstone for organizing affect, cognitions and behaviors and guiding distress responses throughout the life course [29]. Bowlby's theory assumes that all human beings have an innate attachment system which, when activated by physical or psychological distress, works to maintain proximity and communication with nurturing caregivers (attachment figures) [29]. When danger is felt, children stop their ongoing activities and seek to be comforted and reassured. Once their security needs are met, children resume their attachment-unrelated activities (exploration or play) which allow them to interact with and master their environment and develop their personalities [29, 30]. The attachment and exploratory systems are complementary in that exploratory behaviors can take place only when the attachment system is quiescent. Through repeated patterns of interaction, parental responsiveness to a child's felt distress becomes internalized, generalizing to his/her future expectations of parental availability in these situations and impacting exploratory behaviors [31].

Using observational studies, three attachment styles have been documented: secure, anxious, and avoidant attachment styles [32]. These attachment styles are based on differential patterns of exploration and reactions to distress observed in children. For instance, mothers of securely attached children were consistently sensitive and responsive to their children attention-seeking bids which allowed them to confidently explore their environment [30]. Upon a brief separation from their mothers, secure children looked to reestablish some contact with them. Mothers of anxiously attached children showed inconsistent patterns of responsiveness towards their children, being at times unavailable and at other times intrusive. When exploring, anxiously attached children were continuously preoccupied with their mothers' whereabouts, trying to keep tabs on them rather than focusing on actual exploration [33]. Upon a brief separation/reunion episode, these children showed mixed reactions of anger and attachment towards their mothers [31]. Lastly, mothers of insecure-avoidant children rejected their bids for proximity, especially for close body contact [30]. When left to explore, avoidant-attached children showed a lot of passiveness and low interest in the exploration task at hand [33]. When reunited with their mothers after a brief separation, they did not seek close contact with them but rather continued with their exploration, thus displacing their attachment needs [30]. A fourth category, named fearful/disorganized, has been added by Main and Solomon [34] to describe a pattern of attachment where the child oscillates between an anxious and an avoidant type of attachment style.

These differential parent/child relationship patterns become the cornerstone for organizing a child's eating experiences and expectations, influencing their acceptance of new foods [6]. As can be seen from the description of attachment patters, nurturing parents provide children with a place where they can safely explore because comfort is readily available in case of distress. Secure children are thus more active and curious explorers [35, 36]. On the other hand, parents of anxiously attached children are unreliable and so the child cannot solely focus on learning about food and eating because they are unsure of the caretaker's availability making the child more prone to anxious reactions in new environments. Lastly, avoidant-attached children have caregivers who do not provide comfort when distressed and hence the exploratory experience and learning about food becomes very passive. Since these parent/child attachment styles become *internalized* and, through more or less optimal emotional experiences, are likely to shape a lifetime of food attitudes and preferences, we next shortly review evidence of the role of attachment style in eating.

Attachment and Eating

There is significant evidence pointing to the unique role of attachment style in eating and its correlates. For instance, a recent study done in the US with 6,650 preschool aged children showed that the prevalence of obesity in children classified as insecurely (anxious, avoidant, and fearful) attached (fourth lowest quartile of security score) was 23.1 % compared to 16.6 % for their securely attached counterparts (quartile 1, 2, and 3) [37]. Moreover, even after controlling for mother/child interaction during play, parental practices related to obesity, maternal body mass index and socio-demographic characteristics, insecurely attached children were 1.3 times more likely to be obese than children categorized as securely attached [37]. Both attachment anxiety and avoidance have been associated with eating disorders prevalence in general [38]. For example, Sharpe et al. [39] found that insecurely attached girls aged between 10 and 14 years old had significantly more weight concerns and lower self-esteem, compared to their secure counterparts, and were more at risk for eating disorders.

Table 3.3 French and English attachment questionnaire adapted to children aged 8–12

Attachment dimension	Item description—French	Item description—English
Avoidant	Je suis mal à l'aise de devoir compter sur mes parents	I am not comfortable having to depend on my parents
	Je suis mal à l'aise lorsque mes parents comptent sur moi	I'm not comfortable when my parents depend on me
	Je suis mal à l'aise lorsque mes parents tentent de se rapprocher de moi	I don't like when my parents try to get too close to me
	J'ai peur de devenir trop proche de mes parents	I am scared to become too close to my parents
	Ça m'est difficile de faire entièrement confiance à mes parents	I find it difficult to trust my parents completely
Anxious	Je voudrais que mes parents soient plus proches de moi	I would like my parents to be closer to me
	Je pense que mes parents ne m'aiment pas réellement	I think my parents do not really love me
	Mon désir de me rapprocher fait fuir mes parents	My desire of being close to my parents scares them away
	Je m'inquiète d'être abandonné(e) par mes parents	I worry about being abandoned by my parents

French and English versions of the Attachment Questionnaire as administered to both children and adults. Cronbach's alpha for the avoidant index for children was 0.80 and for adults 0.82. The reliability for the anxious index was 0.55 for children and 0.65 for adults

Moreover, children in third to fifth grade who reported loss of control over eating, a variable prevalent in overweight children and adolescents seeking weight loss treatments, were more likely to be insecurely attached to both parents, compared with children who reported no such control loss [40]. In a sample of undergraduate students, Wilkinson et al. [41] found that attachment anxiety was linked with disinhibited eating, a strong predictor of high BMI, while, in adults, a study of 5,692 participants showed that an insecure-anxious attachment style was positively associated with cardiovascular diseases, i.e., stroke, high blood pressure, and heart disease, whereas a secure attachment style was not correlated with any poor health conditions [42].

Attachment Style and Nutrition: A Study of Child and Adult Unhealthy Food Consumption

The aim of the present study was to investigate how attachment style influenced actual food consumption from a life-course perspective since attachment styles are based on early parent/child interactions starting from birth and later become internalized as mental schemas. To examine this, 148 parent/child dyads were recruited from a major French-speaking North-American city. Because, as shown before, humans have an inherent preference for unhealthy foods which is linked to negative health consequences [43], this study focused particularly on unhealthy food consumption. As such, children aged 8–12 and one of their parents filled out an online questionnaire. Brennan et al. [44] showed that attachment styles were better conceptualized as dimensions rather than categorical attachment styles with the anxiety and avoidant dimensions encompassing attachment measures in both adults and children. As a result, adult and child attachment was measured using an age-adapted version of the Adult Attachment Questionnaire [45] which allows for the construction of an avoidant/secure and anxious index. Because as stated before, the authors were interested in the life-course influence of attachment, both adults and children were asked think about their own parents when filling out the attachment questionnaire and rate their experience on nine statements with their respective caregivers (see Table 3.3). Higher scores on the avoidant/secure index reflected greater avoidance or not desiring closeness or dependency with one's parents while higher scores on the anxiety index were indicative of more anxiety or worries about being rejected by one's parents. To measure unhealthy food consumption an average of 14 commonly eaten high sugar and/or fat food items was computed. These food items were shown to constitute major sources of calorie, carbohydrate, and fat in children's and adults' diets.

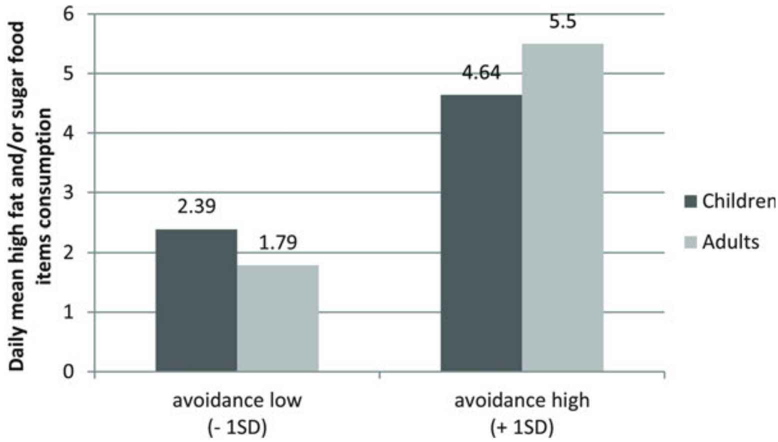

Fig. 3.3 Means of high sugar and/or fat food consumption in children and adults for low and high avoidant index scores. Scores of the avoidant index are based on an adapted version of the Attachment Questionnaire [45] where higher scores reflect higher avoidance or lower trust in parental figures. Least square means of daily high sugar and/or fat food consumption in children and adults ($N=148$ parent/child dyads) were calculated for low avoidance (M − 1 SD) and high avoidance (M + 1SD) groups. Estimates were obtained using PROC REG (SAS 9.1 version)

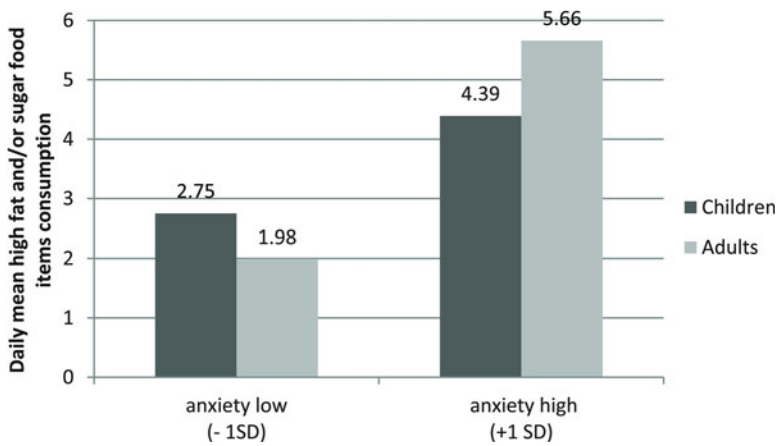

Fig. 3.4 Means of high sugar and/or fat food consumption in children and adults for low and high anxious index scores. Scores of the anxious index are based on an adapted version of the Attachment Questionnaire [45] where higher scores reflect higher worries and fear of abandonment from parental figures. Least square means of daily high sugar and/or fat food consumption in children and adults ($N=148$ parent/child dyads) were calculated for low anxiety (M − 1SD) and high anxiety (M + 1SD) groups. Estimates were obtained using PROC REG (SAS 9.1 version)

Items included sugar-added cereals, red meat, crisps, fried chicken, hot dogs, fries, and poutine, ice cream, etc. To separate the effects of attachment style from that of other variables known to influence food consumption an optimal model was elaborated where, for each attachment dimension, number of hours spent in front of a computer, TV or video game, age, BMI and level of education of parent were used as control variables for both children and adults. In addition, parental unhealthy food consumption was also controlled for in the children's model.

Our results indicate that children and adults who had parental relationships marked by higher avoidance and anxiety consumed more high sugar and/or high fat foods compared to their counterparts who showed lower avoidance and anxiety in their parental relationships (see Figs. 3.3 and 3.4).

As such, the internalization of more unreliable and more unsupportive parental behaviors based on children's and adults' reported attachment styles with their own caregivers was found to be positively associated with higher intake of unhealthy food items. These results provide preliminary evidence in line with the authors' theorizing that internalized parent/child attachment styles formed in early infancy have the potential of influencing a life-long course of food attitudes and choices.

Conclusions

Strong evidence shows that meals eaten at home are healthier than those eaten away-from-home; this chapter uses empirical explorations to investigate the role of emotional reinforcement and parent/child attachment as two potential underlying protective mechanisms contributing to the superior nutritional quality of home meals. While eating cannot only be reinforced by sensory and hedonic pleasure but also by emotions arisen from the context in which eating takes place, research has shown that more positive emotions are experienced at home than in away-from-home settings. Through an associative learning process, the positive emotions experienced at home may become saliently attached to home meals especially to healthy foods which lack the biologically reinforcing property of unhealthier foods. The results of our first study show that, specifically at home, healthier meals had a superior emotional reinforcing value than less healthy meals as indicated by more positive and less negative emotions being reported before and after home healthy meals than less healthy meals consumed in the same setting. Moreover, away-from-home settings did not show the similar protective effects on the nutritional quality of food choice.

Furthermore, this associative learning between the emotional experiences and eating may start at birth when nurturing and sensitive parents provide children with a safe haven (mostly at home) where they can learn to explore their environment and master tasks at hand, eating being one of these tasks. This parent/child attachment is internalized and becomes the cornerstone guiding one's relationship with food and eating throughout life. Nonetheless, some parents do not provide a safe and calm environment for children to explore, learn, and master food experiences. Consequently, these less optimal internalized experiences of parental caring and environment exploration characterized either by high anxiety and worries or lack of exploratory interest may influence food consumption. As such, findings from our second study with children aged 8–12 and one of their parents showed that both children and adults who showed more anxiety and avoidance in their relationships with their own parents to be more likely to consume unhealthy foods, i.e., foods that were high in sugar and/or fat content.

In an effort to bridge both literatures and acquire a more solid understanding of how emotional reinforcement and attachment styles are related, future studies should assess how emotional reinforcement varies with different attachment patterns. For instance we propose that, because a secure attachment style is based on trusting bonds with caregivers and positive exploratory experiences, positive emotions will be more saliently attached to healthy food consumption for securely attached individuals. On the other hand, for insecurely attached individuals (avoidant/anxious/fearful) who have internalized less positive experiences with caregivers, positive emotions will be less likely to saliently attach to healthy meals. Also, insecure individuals will be more likely to experience negative emotions within the home context after an eating episode than securely attached people. Hence, different attachment patterns are likely to influence directly the emotional reinforcement likely to occur within a home setting, ultimately influencing food consumption.

The present chapter underscores the family and home as a vital setting to promote healthy eating. We focus on two potential underlying mechanisms of home healthy eating, emotional reinforcement, and attachment style, paving the way to important prevention strategies. For instance, systematic parental interventions should emphasize further the emotional consequences attached to healthy eating at home. This may be a powerful strategy to help establish in children a lifetime network of memory associations between healthy food and positive affect, as a complement to providing adequate education in matters of nutritional values of foods and the consequences of unhealthy eating habits. Moreover, parents can receive sensitivity training to enhance nurturing behaviors and hence promote positive emotional food experiences for their children within a safe haven context [46]. Lastly, parents can also be taught how to use home design and atmospheric cues, such as music, dining landscape, and kitchen equipment, all media through which positive emotions in everyday and laboratory contexts can be induced [47], to promote healthy eating at home.

References

1. Lin B-H, Frazao E, Guthrie JF. Contribution of away-from-home foods to American diet quality. Fam Economics Nutr Rev. 1999;12(3&4):85–9.
2. Popkin BM, Duffey K, Gordon-Larsen P. Environmental influences on food choice, physical activity and energy balance. Physiol Behav. 2005;86: 603–13.
3. Broberger C. Brain regulation of food intake and appetite: molecules and networks. J Intern Med. 2005; 258:301–27.
4. Canetti L, Bachar E, Berry EM. Food and emotion. Behav Processes. 2002;60:157–64.
5. Côté S, Moskowitz DS. On the dynamic covariation between interpersonal behavior and affect. Prediction from neuroticism, extraversion, and agreeableness. J Pers Soc Psychol. 1998;75(4):1032–46.
6. Satter E. Feeding dynamics: helping children to eat well. J Pediatr Health Care. 1995;9(4):178–84.
7. Savage JS, Fisher JO, Birch LL. Parental influence on eating behavior: conception to adolescence. J Law Med Ethics. 2007;35(1):22–34.
8. Epstein LH, Leddy JJ, Temple JL, Faith MS. Food reinforcement and eating: a multilevel analysis. Psychol Bull. 2007;133(5):884–906.
9. Levine AS, Kotz CM, Gosnell BA. Sugar and fats: the neurobiology of preference. J Nutr. 2003;133:831S–4.
10. Drewnowski A, Hann C. Food preferences and reported frequencies of food consumption as predictors of current diet in young women. Am J Clin Nutr. 1999;70:28–36.
11. Rosenstein D, Oster H. Differential facial responses to four basic tastes in newborns. Child Dev. 1988; 59(6):1555–68.
12. Mennella JA, Pepino MY, Reed DR. Genetic and environmental determinants of bitter perception and sweet preferences. Pediatrics. 2005;115(2):e216–22.
13. Desmet PMA, Schifferstein HNJ. Sources of positive and negative emotions in food experience. Appetite. 2008;50(2–3):290–301. doi:10.1016/j.appet.2007.08.003.
14. Dubé L, LeBel JL, Lu J. Affect asymmetry and comfort food consumption. Physiol Behav. 2005;86:559–67.
15. LeBel JL, Lu J, Dubé L. Weakened biological signals: highly-developed eating schemas amongst women are associated with maladaptive patterns of comfort food consumption. Physiol Behav. 2008;94:384–92.
16. Macht M, Meininger J, Roth J. The pleasure of eating: a qualitative analysis. J Happiness Stud. 2005;6: 137–60.
17. Moskowitz DS, Côté S. Do interpersonal traits predict affect? A comparison of three models. J Pers Soc Psychol. 1995;69(5):915–24.
18. Garg N, Wansink B, Inman JJ. The influence of incidental affect on consumers' food intake. J Market. 2007;71:194–206.
19. Sclafani A. Psychobiology of food preferences. Int J Obes. 2001;25 Suppl 5:S13–6.
20. Lu J. Maladaptive eating and the dynamic interactions between component processes in "cold" and "hot" systems. Unpublished doctoral dissertation, McGill University; 2010, p. 132.
21. Macht M. How emotions affect eating: a five-way model. Appetite. 2008;50(1):1–11.
22. Lu J, Huet C, Dubé L. Emotional reinforcement as a protective factor for healthy eating in home settings. Am J Clin Nutr. 2011;94(1):254–61.
23. Khare A, Inman JJ. Habitual behavior in American eating patterns: the role of meal occasions. J Consumer Res. 2006;32:567–75.
24. de Graaf C. Nutritional definitions of the meal. Gaithersburg, MD: Aspen; 2000.
25. Sweeting H, Anderson A, West P. Socio-demographic correlates of dietary habits in mid to late adolescence. Eur J Clin Nutr. 1994;48(10):736–48.
26. de Vries M. The experience of psychopathology in natural settings: introduction and illustration of variables. In: de Vries M, editor. The experience of psychopathology—investigating mental disorders in their natural settings. Cambridge, MA: Cambridge University Press; 1992.
27. Birch LL. Children, parents and food. Br Food J. 1993;95(9):11–5.
28. Bowlby J. Attachment and loss. Vol. 1, 2nd ed. New York, NY: Basic Books; 1969/1999.
29. Aspelmeier JE, Kerns KA. Love and school: attachment/exploration dynamics in college. J Soc Pers Relat. 2003;20(1):5–30.
30. Hazan C, Shaver PR. Love and work: an attachment-theoretical perspective. J Pers Soc Psychol. 1990; 59(2):270–80.
31. Mikulincer M, Florian V, Weller A. Attachment styles, coping strategies, and posttraumatic psychological distress: the impact of the Gulf War in Israel. J Pers Soc Psychol. 1993;64(5):817–26.
32. Ainsworth MDS, Blehar MC, Waters E, Wall S. Patterns of attachment: a psychosocial study of the strange situation. Hillsdale, NJ: Lawrence Erlbaum Associates; 1978.
33. Hazen NL, Durrett ME. Relationship of security of attachment to exploration and cognitive mapping abilities in 2-year-olds. Dev Psychol. 1982;18:751–9.

34. Main M, Solomon J. Procedures for identifying infants as disorganized/disoriented during the Ainsworth Strange Situation. In: Greenberg MT, Cicchetti D, Cummings EM, editors. Attachment in the preschool years. Chicago: University of Chicago Press; 1990. p. 121–60.
35. Arend R, Gove FL, Sroufe LA. Continuity of individual adaptation from infancy to kindergarten: a predictive study of ego–resiliency and curiosity in preschoolers. Child Dev. 1979;50(4):950.
36. Sroufe LA. The coherence of individual development: early care, attachment, and subsequent developmental issues. Am Psychol. 1979;34(10):834–41.
37. Anderson SE, Whitaker RC. Attachment security and obesity in US preschool-aged children. Arch Pediatr Adolesc Med. 2011;165(3):235–42.
38. Ward A, Ramsay R, Turnbull S, Benedettini M, Treasure J. Attachment patterns in eating disorders: past in the present. Int J Eat Disord. 2000;28(4): 370–6.
39. Sharpe TM, Killen JD, Bryson SW, et al. Attachment style and weight concerns in preadolescent and adolescent girls. Int J Eat Disord. 1998;23(1):39–44.
40. Goossens L, Braet C, Decaluwé V. Loss of control over eating in obese youngsters. Behav Res Ther. 2007;45(1):1–9.
41. Wilkinson LL, Rowe AC, Bishop RJ, Brunstrom JM. Attachment anxiety, disinhibited eating, and body mass index in adulthood. Int J Obes. 2010;34(9): 1442–5.
42. McWilliams LA, Bailey SJ. Associations between adult attachment ratings and health conditions: evidence from the National Comorbidity Survey Replication. Health Psychol. 2010;29(4): 446–53.
43. Behan, Donald F., Samuel H, et al. “Obesity and its relation to mortality and morbidity costs.” Society of Actuaries. Consulted on August 5 (2010): 2011.
44. Brennan KA, Clark CL, Shaver PR. Self-report measurement of adult attachment: an integrative overview. In: Simpson JA, Rholes WS, editors. Attachment theory and close relationships. New York, NY: Guilford Press; 1998. p. 46–76.
45. Simpson JA. Influence of attachment styles on romantic relationships. J Pers Soc Psychol. 1990;59(5): 971–80.
46. Broberg AG. A review of interventions in the parent–child relationship informed by attachment theory. Acta Paediatr. 2000;89:37–42.
47. Morin S, Dubé L, Chebat JC. Role of pleasant music in service spaces: test of a dual model of environmental perception. J Retail. 2007;83(1):115–30.

4 Diet Quality and Its Potential Cost Savings

Clare Collins, Alexis Hure, Tracy Burrows, and Amanda Patterson

Key Points

- Limited studies have determined the relationship between diet quality and health care costs. The current analysis investigates the relationship between Medicare health care costs and claims in Australia with diet quality, in a nationally representative sample of mid-aged Australian women over the 10-year period from 2001 to 2010. Health care utilization data was obtained from Medicare and diet quality was calculated using the Australian Recommended Food Score (ARFS). The ARFS is derived from Food Frequency Questionnaire (FFQ) responses to foods that are consistent with national recommendations detailed in the Dietary Guidelines for Australian Adults.
- It was found that consuming a greater variety of vegetables predicted lower 10-year cumulative Medicare Charges, Benefit, Gap and a lower number of claims. However, for fruit, dairy and healthy fats component scores this relationship was in the opposite direction. Further evaluations in other studies are needed and will provide the basis for modelling future cost savings and may help to identify appropriate dietary targets associated with reduced health care costs.

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Diet index • Diet quality • Diet score • Health costs • Health service usage • Medicare • Nutrition survey

Abbreviations

AIHW	Australian Institute of Health and Welfare
ALSWH	Australian Longitudinal Study on Women's Health
ARFS	Australian Recommended Food Score
DALY	Disability-adjusted life years
DQES	Dietary Questionnaire for Epidemiological Studies
DQI	Diet Quality Index
DQS	Diet Quality Score
EPC	Enhanced Primary Care
FFQ	Food Frequency Questionnaire
HDI	Healthy Diet Indicator
HEI	Healthy Eating Index
HFI	Healthy Food Index
HMO	Health Maintenance Organization
MDS	Mediterranean Diet Score
RFS	Recommended Food Score
SENECA	Survey in Europe on Nutrition and the Elderly; a Concerted Action

Introduction

Surprisingly, only limited studies have examined the relationship between diet quality and the health care costs to government or to individuals. Additionally, very few studies have examined whether diet quality is able to predict future health care costs.

Optimal dietary quality in the context of this review has been defined as eating patterns that reflect greater adherence to national dietary recommendations. Diet quality can be measured by diet quality or diet index scores. There are over 25 indexes to date that have been developed to assess overall diet quality and/or variety. Wirt and Collins [1] reviewed these in 2009 and found that the major indexes included the Healthy Eating Index (HEI) [2], the Healthy Diet Indicator (HDI) [3], the Healthy Food Index (HFI) [4], the Recommended Food Score (RFS) [5], the Diet Quality Index (DQI) [6], the Diet Quality Score (DQS) [7], and the Mediterranean Diet Score (MDS) [8]. Construction of these diet quality indexes follows three major scoring approaches: (1) based on food groups or specific foods, (2) based on nutrient intakes or (3) derived from combinations of foods and nutrient intakes [9].

A review of the relationship between these diet quality scores and health-related outcomes demonstrated that higher quality eating patterns were associated with lower self-reported indices of health care usage and lower morbidity and mortality from chronic disease [1, 10, 11].

However, whether higher diet quality translates into lower health care costs over time has rarely been examined. One key Australian report evaluated the relationship between fruit and vegetable intake (as a measure of diet quality) and burden of disease. This Australian Institute of Health and Welfare report (AIHW-2003) estimated that 2.1 % of the total burden of disease could be attributed to inadequate fruit and vegetable consumption, making it seventh in a ranking of 14 risk factors studied [12], which together explained 32.2 % of the total burden of disease and injury. Eating an adequate amount of fruits and vegetables has been shown to prevent chronic diseases such as cardiovascular disease, type 2 diabetes and some cancers [12]. The AIHW report found that 69 % of the burden from low fruit and vegetable consumption was due to ischaemic heart disease and that two-thirds of this was experienced by males. Further, the report estimated that 81 % of the burden from low fruit and vegetable consumption was due to mortality, and that the absolute burden from low consumption peaked between 60 and 80 years of age [12].

We previously reported the results of the relationship between diet quality and health service use in a cross-sectional analysis of the Australian Longitudinal Study on Women's Health (ALSWH) for over 9,000 mid-aged women [13]. Among these women, a higher Australian Recommended Food Score (ARFS, the index of diet quality used) was associated with better self-reported health status (excellent, very good, good or fair/poor), a lower number of visits to a general medical practitioner in the previous year and fewer consultations with a specialist medical practitioner in the previous year. Those with a higher ARFS also had higher intakes of micronutrients and a lower percentage of energy from total or saturated fat [13].

Kant and Schatzkin [11] examined the 10-year association between diet quality (measured by nutrient intakes) with self-reported doctor-advised chronic medical conditions, for a nationally representative sample of adults (>25 years, $n = 7{,}207$) in the Continuing Survey of Food Intakes. A greater number of self-reported medical conditions were associated with an increased risk of not consuming 100 % of the Recommended Dietary Allowance (RDA) for *some* nutrients (vitamins E, B12 and calcium, zinc and iron, $P < 0.05$). Women were more likely to report poor nutrient intakes than men [11].

In the SENECA (Survey in Europe on Nutrition and the Elderly; a Concerted Action) study on older persons from seven European countries, Haveman-Nies et al. [14] examined the health status and lifestyle behaviours of 216 men and 264 women born between 1913 and 1918, for the 10-year period (1988–1999). Self-rated health and self-care ability declined in men and women with both healthy and unhealthy lifestyle habits during follow-up. There was no relationship between having a healthy, Mediterranean-style diet and deterioration in health status. Inactive and smoking persons did have an increased risk for a decline in health status, compared with active and non-smoking people, and the authors concluded that both physical activity and being a non-smoking delayed deterioration in health status for older people.

Cost-effectiveness studies have modelled the potential cost savings of implementing strategies to promote healthful eating patterns and prevent disease development. Cobiac et al. [15] evaluated the cost-effectiveness of interventions to promote fruit and vegetable consumption to assess the degree to which promotion strategies could potentially reduce population disease burden. They identified 23 interventions that promoted fruit and vegetable intake in adults that also had sufficient information to model the health impacts in disability-adjusted life years (DALY). They modelled the intervention costs and potential cost savings from averting disease treatment, and the cost-effectiveness over the population's lifetime, from a health sector perspective. They found that only five of the 23 interventions had less than a $50,000 per DALY cost-effectiveness threshold and that the most effective intervention could avert only 5 % of the disease burden attributed to insufficient fruit and vegetable intake. They concluded that further evaluation of population level interventions was needed. While observational studies and modelling estimates do provide a clear rationale for trialling interventions to improve diet quality as a strategy to reduce health care and medical costs, evaluation in real-life settings and databases provides important data in relation to true health care costs.

Others have assessed the cost-effectiveness of interventions to improve diet in specific at risk populations groups. Patrick et al. [16] randomized about 5,000 health maintenance organization (HMO) older Medicare beneficiaries to receive a 2-year preventive service benefit package that included one component aimed at improving diet quality, as measured by percentage energy from fat and grams from daily fibre intakes, or to usual care. At the 2- and 4-year follow-ups, the treatment group reportedly participated in more exercise and reduced their percentage fat intake more than the control group. Although they observed an unexpected greater number of deaths in the treatment group, which they attributed to more individuals over the age of 75 years being randomized to the intervention group, the surviving treatment group participants reported higher satisfaction with health, less decline in self-rated health status, and fewer depressive symptoms than surviving control participants. However, the intervention did not yield a lower cost per quality adjusted life year.

Wolf et al. [17] evaluated, in a 12-month randomized controlled trial, the costs associated with provision of a lifestyle intervention programme on health care expenditure in a high-risk obese population ($n = 147$) with type 2 diabetes. The intervention group received individual and group education, support and referrals to registered dietitians, while the usual-care group received educational materials only. The net cost of the intervention was $328 per person per year, but the overall mean health plan costs (including the $328) was $3,586 lower in intervention group compared to the usual care group (95 % CI −$8,036 to −$25, $P < 0.05$). The difference was driven by group differences in medical costs (−$3,316, 95 % CI −$7,829 to −$320, $P < 0.05$), with significantly fewer inpatient admissions for the intervention group (prevalence 2.8 % in the intervention group vs. 22.5 % in controls, $P < 0.001$). Authors concluded that adding a modest-cost, registered dietitian-led lifestyle case-management intervention to usual medical care did not increase health care costs, but resulted in modest cost savings for obese patients with type 2 diabetes.

Pavlovich et al. conducted a systematic review of randomized controlled trials on the cost-effectiveness of the provision of outpatient nutrition services [18]. Of the 13 studies included there was relatively consistent support for the cost-effectiveness of nutrition services. Provision of nutrition services was cost-effective in reducing serum cholesterol levels (for example, $20–1,268/mmol/L decrease in serum low-density lipoprotein level), for weight loss ($2.40–10 per pound lost), and in reducing blood glucose ($5/mmol/L decrease). This was evident in target populations with diabetes mellitus and hypercholesterolemia as well as for those without such diagnoses. The authors noted that due to limitations in trial quality and differences in cost perspectives though, that more randomized controlled trials addressing these omissions were needed.

While these approaches provide important insights into potential cost savings through optimizing dietary quality, we have only been able to locate a limited number of studies internationally that have prospectively examined the relationship between diet quality and health care costs. Two studies have examined the national government Medicare charges in relation to aspects of dietary intake. Daviglus et al. [19] looked at US Medicare charges over a 15-year period in relation to fruit and fruit plus vegetable intakes in mid-aged men from the Chicago Western Electric Study, while we examined Australian Medicare costs incurred over a 6-year period by mid-aged women ($n > 10{,}000$) from the ALSWH in relation to diet quality as measured by the ARFS [20].

Daviglus et al. demonstrated that in 1,063 men from the Chicago Western Electric Study, those in the highest tertile of fruit and fruit plus vegetable intakes during middle age had lower mean annual and cumulative Medicare charges over a 15-year period [19]. They found that, after adjusting for baseline age, education, total energy intake and baseline risk factors, high fruit and vegetable intakes were associated with lower mean annual and cumulative Medicare charges, including total charges and cardiovascular disease and cancer-related charges, although the P value for trend ranged from 0.019 to 0.862. While many of the results were not statistically significant, the trends do suggest that having higher fruit and vegetable intakes earlier in adulthood has the potential to lower health care costs in older age, as well as improve health status.

While we hypothesized lower costs would be associated with higher diet quality, our Australian data demonstrated a positive association between having a higher ARFS (i.e. better diet quality) and higher cumulative Medicare (Australia) costs. Although those in the highest quintile of ARFS incurred higher Medicare costs, the number of Medicare claims for this quintile was lower than for the lowest quintile of ARFS for the period 2002–2007, $P = 0.002$.

Firstly, it is important to note that there are some differences between the national Medicare programmes in the USA and Australia. In the USA, Medicare is a social insurance programme administered by the government, which provides health insurance cover to people who are aged 65 and over, or who meet specific criteria. Medicare

(USA) covers hospital-related inpatient and outpatient services, skilled nursing facility services and charges related to outpatient services, including emergency room visits, clinic and ambulatory surgery, laboratory tests, radiography, rehabilitation therapy, radiation therapy and renal dialysis. This contrasts with Medicare Australia, who administer a universal health care programme called Medicare for the Australian government. Medicare (Aus) is funded by income tax and an income-related Medicare levy. Medicare (Aus) is the largest source of primary health care spending and covers scheduled fees for out-of-hospital services for doctors (including specialists), tests and examinations by doctors, X-rays and pathology tests, eye tests performed by optometrists, most surgical and other therapeutic procedures performed by doctors, some surgical procedures performed by approved dentists, specified items under the Cleft Lip and Palate Scheme, and specified items for allied health services as part of the Enhanced Primary Care (EPC) programme. In-hospital services are only covered by Medicare (Aus) for treatment as a public patient by doctors and specialists nominated by the hospital. Medicare (Aus) also administers a separate Pharmaceutical Benefits Scheme (PBS), which makes a range of prescription medicines available at affordable prices to Australian residents, through government subsidy.

While the above two studies are the only ones we have found that actually calculate costs, van Baal et al. [21], in three cohorts from the Netherlands, estimated the annual and lifetime medical costs attributed to obesity for people aged 20 years at baseline. They compared obese people to those who were smokers and those with a healthy lifestyle (including following a high quality diet). The obese group were modelled as having the highest annual health expenditure, followed by smokers, but lifetime health expenditure was highest among healthy-living individuals, due to their longer life expectancy. The authors concluded that while obesity prevention is an important and cost-effective way of improving public health, it is not the only strategy for reducing health expenditures.

Other than these studies, we were not able to locate any other studies that have examined associations between diet quality and subsequent cost of health service use. This relationship needs further study with both costs and claims monitored over time to examine patterns of health care usage and costs in the longer term for those with the dietary patterns that adhere more closely to national recommendations.

Evaluating the Association Between Diet Quality and 10-Year Cumulative Medicare Costs

On this basis, we have recently extended the 1946–1951 (mid-aged) ALSWH cohort follow-up period to 10 years, to examine whether higher diet quality, as measured by the ARFS was still associated with higher health care costs but lower number of claims.

The data for this analysis comes from ALSWH, which was established to investigate multiple factors affecting the health and wellbeing of women over a 20-year period. Women in three age groups (“younger” 18–23, “mid-aged” 45–50 and “older” 70–75 years) were randomly selected from the national health insurance database, Medicare (Aus) that includes all permanent residents of Australia, with over-representation of women living in rural and remote areas. The methods have been previously published [22–24].

Diet Quality and Health Care Costs in Mid-Aged Women in Australia

Data used in this analysis have been derived from the mid-aged cohort of the ALSWH. Survey 1 ($n = 13{,}716$) was conducted in 1996 and the respondents have been shown to be broadly representative of the national population of women in the target age groups [22]. Survey 2 ($n = 12{,}338$) was conducted in 1998 and Survey 3 ($n = 11{,}228$) was conducted in 2001. The response rate for Survey 3 of the mid-aged cohort was 83 % of women who had completed Survey 1 and had not died or become too ill to complete further surveys. The non-respondents included those who did not complete Survey

3 (7.4 %), withdrew from the study completely (2.8 %) or could not be contacted (6.8 %) [24]. Of the women who completed Survey 3 (then aged 50–55 years), 11,194 completed a usable Food Frequency Questionnaire (FFQ).

Assessment of Dietary Intake

Dietary intake was assessed using the Dietary Questionnaire for Epidemiological Studies (DQES) FFQ. The DQES asks respondents to report their usual consumption of 74 foods and six alcoholic beverages over the preceding 12 months using a 10-point frequency option from "never" up to "three or four times per day". Portion size photographs are used to adjust the serve size for vegetables, meat and casseroles. Additional questions are asked about total number of daily serves of fruits, vegetables, bread, dairy products, eggs, fat spreads and sugar, as well asking the type of bread, dairy products and fat spreads used. Nutrient intakes are computed from NUTTAB 1995, a food composition database of Australian foods [25], using software developed by the Cancer Council of Victoria. Both the development of the DQES [26] and its validation in mid-aged Australian women have been previously reported [27]. FFQs with greater than four missing values were discarded.

Australian Recommended Food Score

The ARFS was modelled on the RFS by Kant and Thompson [28] and has been previously described [13]. Briefly, it is calculated based on DQES items consistent with national recommendations in the Dietary Guidelines for Australian Adults [29]. In brief, items consumed less than once a week are scored zero and those consumed once a week or more score one. Additional points are awarded for type and variety of core foods consistent with national dietary intake recommendations. A maximum of two points was added for alcohol consumption: one point for moderate frequency and the second point for moderate quantity, when they drank alcohol. The maximum ARFS is 74.

Medicare Data

The health services utilization and cost data was provided by Medicare Australia. In the third survey of the mid-aged cohort, 7,225 out of the 11,226 women (64 %) gave consent for linkage of their Medicare data to their survey. There were significant but small differences between consenters and non-consenters by area of residence, and those consenting to Medicare linkage tended to be better educated, more likely to be able to manage on their available income and more likely to say their health was excellent, very good or good [23, 24]. Only ALSWH respondents who consented to link their Medicare data were used in this analysis. There were 6,781 women who had given both Medicare consent and had an ARFS.

All Medicare data were collected for the 10 years from 2001 to 2010. Annual data was totalled for each woman to determine the amount the women spent on Medicare health care and the "Number of claims". Women with no claims for a particular year had values of zero imputed into their claims and cost fields for that year, as Medicare Australia only provides data for women for which there are charges recorded. The "Charge" item is the total cost of the treatment. There are two other Medicare variables, the "Benefit" and the "Gap". The Benefit is what was paid back to the patient, while the Gap is the difference between the Charge and the Benefit (i.e. what the patient paid out of their own pocket). The Charge is almost always higher than the Benefit.

Those with the highest (n=67) and lowest 1 % (n=68) of charges were excluded from data analysis to avoid extreme values (<$634 and >$49,395). Therefore, the sample included data for n=6,646 women. Level of education was recorded at Survey 1 and was missing for n=39, hence a total sample of n=6,607 are included in the multivariate regressions with adjustment for baseline area of residence and education. The Medicare data used in this study were from the Medicare Benefits Schedule and did not include data from the PBS.

The analysis followed the same approach as that described previously [20]. Data manipulation and statistical analyses were performed using

Table 4.1 Median 10-year (2001–2010) cumulative Medicare claims and costs ($AUS) for mid-aged Australian women by quintile of ARFS, where 1 = lowest and 5 = highest quintile

ARFS quintile	*n*	ARFS median	IQR	Number of claims	IQR	Charge ($)	IQR	Benefits ($)	IQR	Gap ($)	IQR
1	1,362	21	6	148	137	7,366	8,694	5,732	6,404	1,206	2,507
2	1,487	29	3	143	124	7,452	7,886	5,605	5,786	1,572	2,706
3	1,239	33	2	147	128	7,745	8,903	5,744	6,327	1,702[a]	2,967
4	1,244	38	2	140	120	7,683	8,368	5,704	5,849	1,764	2,830
5	1,314	44	5	138[a]	118	7,835	8,481	5,625	5,983	1,704[a]	3,062
All	6,646	33	12	143	126	7,608	8,432	5,682	6,040	1,590	2,845

[a]Generalized linear modelling with adjustment for area of residence and education at baseline shows ARFS quintile is a significant ($P < 0.01$) predictor of number of Medicare Claims (quintile 5) and Gap expenses (quintiles 3 and 5), compared to quintile 1

Intercooled Stata, version 11 (StataCorp LP, College Station, TX, USA). The distribution of the Medicare data was highly skewed to the right; therefore, median values have been presented. The ARFS was normally distributed. ARFS quintiles were generated using the xtile function in Stata. Generalized linear modelling was performed with area of residence and level of education at baseline used as covariates, to adjust for the sampling frame and for socioeconomic status. *P*-values <0.01 were considered statistically significant, due to the large sample size.

The ARFS was normally distributed while the Medicare costs and claims were not. The median 10-year (2001–2010) cumulative Medicare Claims and Charge, Benefit and Gap expenses were examined by quintiles of ARFS at Survey 3, with a median (interquartile range, IQR) ARFS of 21 (6) for quintile 1 and 44 (5) for quintile 5 (Table 4.1). After adjustment for area of residence and education at baseline, ARFS quintile 5 had significantly fewer Claims ($P = 0.008$) compared to ARFS quintile 1. ARFS quintiles 3 and 5 had significantly higher Gap expenses compared to quintile 1; $P = 0.004$ and $P < 0.001$, respectively. ARFS quintile was not a significant predictor of Charge ($P = 0.583$) or Benefit ($P = 0.369$).

ARFS Component Scores

The individual ARFS component scores were then tested as the predictors of 10-year cumulative Medicare claims and costs. Table 4.2 shows that the number of Medicare Claims and Charges was inversely associated with consuming a greater variety of vegetables, independent of total ARFS at Survey 3, and area of residence and education at baseline. There was a positive linear relationship between Medicare claims and charges and the ARFS component scores for fruit, dairy and fat. All other ARFS component scores were not associated with Medicare claims and costs ($P > 0.01$).

How Diet Quality Impacts on Health Care Costs

This analysis extends the cumulative Medicare costs and claims in Australia in relation to diet quality in mid-aged Australian women to 10 years. Unlike our previous 5-year cumulative data, which demonstrated higher Medicare costs associated with higher diet quality, this 10-year cumulative data show that Medicare charge and benefit are not related to ARFS (Table 4.1). However, the Medicare Gap, the amount paid by an individual, does increase significantly with higher diet quality (ARFS). This relationship is in the opposite direction to what you would expect, and the 10-year cumulative mean difference for Gap payments from the bottom to the top quintile is substantially greater at $AUS500. This relationship is likely to be confounded by charges incurred for routine screening services related to breast and/or cervical cancer, as those likely to have higher diet quality may also have more routine screening procedures. Interestingly,

Table 4.2 Coefficients and 95 % confidence intervals for ARFS components that significantly predict[a] 10-year (2001–2010) cumulative Medicare claims or costs ($AUS) for mid-aged Australian women ($n = 6{,}607$)

		Coefficients (95 % confidence intervals)			
ARFS component	Maximum points	Number of claims	Charge ($)	Benefits ($)	Gap ($)
Vegetable	22	−2.3 (−3.5, −1.1)	−154 (−233, −76)	−100 (−157, −43)	−54 (−82, −36)
Fruit	14	2.2 (0.8, 3.6)	162 (72, 252)	112 (47, 177)	50 (18, 82)
Dairy	7	6.4 (3.3, 9.4)	397 (202, 593)	268 (126, 409)	129 (60, 198)
Nuts/beans/soy/egg[b]	7				−98 (−164, −33)
Fat	1	9.1 (2.8, 15.4)	672 (270, 1,073)	421 (131, 712)	250 (108, 393)

[a]Generalized linear modelling with adjustment for area of residence and education at baseline, and total ARFS at Survey 3. All significant at $P < 0.01$

[b]Non-significant Medicare items and ARFS components are not shown

as for the 5-year data, the total number of claims for health care services over the 10-year period is significantly lower in the highest quintile of ARFS compared to the lowest, by a total of 10 claims. It will be important to revisit this relationship in another 5 years to examine whether there is a point at which having consumed a higher quality diet over an extended period that it does lead to Medicare savings in terms of cumulative health care costs.

When the ARFS sub-scales were examined, there was a statistically significant relationship with many of the sub-scales scores (Table 4.2). While having greater variety of vegetables predicted lower 10-year cumulative Medicare Charges, Benefit, Gap and a lower number of claims, this was in the opposite direction for fruits, dairy and healthy fats. This finding is different to the results from the Western Electric company study of male employees [19] where after 25 years of follow-up there were lower Medicare (USA) costs for those with the highest reported fruits and vegetables intakes. Although the Medicare costs do represent different things in the USA and Australia, there is now some synergy with the longer follow-up in the Australian data set. Limitations in the analysis include that baseline health status was not adjusted for, although the top and bottom 1 % of extreme Medicare cost values were removed, and not all costs related to medical treatments are captured by the Medicare (Aus) data.

It will be critically important to continue to examine the relationship between diet quality and Medicare costs for the ALSWH, and to also examine these relationships in other cohort studies where data are available to conduct similar analyses. This is particularly so because of a previous review in which we demonstrated that across a range of diet quality scores and studies that higher diet quality is consistently inversely related to all-cause mortality, with a protective effect of moderate magnitude [1], and with associations seemingly stronger for men.

Mid-aged women with lower dietary variety and quality, as measured by the ARFS, do not have increased cumulative Medicare costs, but do have a greater number of Medicare claims. This suggests a need to monitor both costs and number of claims over time to determine whether higher diet quality does eventually lead to monetary savings.

Conclusions

It is surprising that so few economic evaluations of the relationship between diet quality and health care costs have been undertaken. Further evaluations will provide the basis for modelling the cost savings in future interventions and may help to identify appropriate dietary targets associated with reduced health care costs.

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References

1. Wirt A, Collins C. Diet quality—what is it and does it matter? Public Health Nutr. 2009;12:2473–92.
2. Hann C et al. Validation of the Healthy Eating Index with use of plasma biomarkers in a clinical sample of women. Am J Clin Nutr. 2001;74:479–86.
3. Huijbregts P et al. Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and the Netherlands: longitudinal cohort study. Br Med J. 1997;315:13–7.
4. Osler M et al. Dietary patterns and mortality in Danish men and women: a prospective observational study. Br J Nutr. 2001;85:219–25.
5. Kant A et al. A prospective study of diet quality and mortality in women. J Am Med Assoc. 2000; 283(16):2109–15.
6. Seymour J et al. Diet Quality Index as a predictor of short-term mortality in the American Cancer Society Cancer Prevention Study II Nutrition Cohort. Am J Epidemiol. 2003;157(11):980–8.
7. Fitzgerald A, Dewar R, Veugelers P. Diet quality and cancer incidence in Nova Scotia, Canada. Nutr Cancer. 2002;43(2):127–32.
8. Trichopoulou A et al. Diet and survival in elderly people. Br Med J. 1995;311:1457–60.
9. Kant A. Indexes of overall diet quality. J Am Diet Assoc. 1996;96:785–91.
10. Kant A. Dietary patterns and health outcomes. J Am Diet Assoc. 2004;104:615–35.
11. Kant A, Schatzkin A. Relation of age and self-reported chronic medical condition status with dietary nutrient intake in the US population. J Am Coll Nutr. 1999;18:69–76.
12. Begg S, et al. The burden of disease and injury in Australia 2003. Canberra: Australian Institute of Health and Welfare; 2007.
13. Collins C, Young A, Hodge A. Diet quality is associated with higher nutrient intake and self-rated health in mid-aged women. J Am Coll Nutr. 2008;27:146–57.
14. Haveman-Nies A, De Groot L, Van Staveren W. Relation of dietary quality, physical activity, and smoking habits to 10-year changes in health status in older Europeans in the SENECA study. Am J Public Health. 2003;93(2):318–23.
15. Cobiac L, Vos T, Veerman J. Cost-effectiveness of interventions to promote fruit and vegetable consumption. PLoS One. 2010;5(11):e14148.
16. Patrick D et al. Cost and outcomes of Medicare reimbursement for HMO preventive services. Health Care Financ Rev. 1999;20(4):25–43.
17. Wolf A et al. Effects of lifestyle intervention on health care costs: Improving Control with Activity and Nutrition (ICAN). J Am Diet Assoc. 2007; 107(8):1365–73.
18. Pavlovich W et al. Systematic review of literature on the cost-effectiveness of nutrition services. J Am Diet Assoc. 2004;104(2):226–32.
19. Daviglus M et al. Relationship of fruit and vegetable consumption in middle-aged men to Medicare expenditures in older age: the Chicago Western Electric Study. J Am Diet Assoc. 2005;105: 1735–44.
20. Collins C, Patterson A, Fitzgerald D. Higher diet quality does not predict lower Medicare costs but does predict number of claims in mid-aged Australian women. Nutrients. 2011;3(1):40–8.
21. van Baal P et al. Lifetime medical costs of obesity: prevention no cure for increasing health expenditure. PLoS Med. 2008;5(2):e29.
22. Brown W et al. Women's Health Australia: recruitment for a national longitudinal cohort study. Women Health. 1998;28:23–40.
23. Brown W, Young A, Byles J. Tyranny of distance? The health of mid-age women living in five geographical areas of Australia. Aust J Rural Health. 1999;7: 148–54.
24. Lee C et al. Cohort profile: the Australian Longitudinal Study on Women's Health. Int J Epidemiol. 2005;34: 987–91.
25. National Food Authority. NUTTAB95 database. Canberra: National Food Authority; 1995.
26. Giles G. Dietary Questionnaire for Epidemiological Studies (version 2). Melbourne: The Cancer Council Victoria; 1996.
27. Hodge A et al. The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle-aged women in a study of iron supplementation. Aust N Z J Public Health. 2000;24:576–83.
28. Kant A, Thompson F. Measures of overall diet quality from a food frequency questionnaire: National Health Interview Survey. Nutr Res. 1992;17:1443–56.
29. NHMRC. Dietary guidelines for Australian adults. Canberra: National Health and Medical Research Council; 2003.

Part II

Reproduction, Pregnancy, and Women

5 Reproduction, Pregnancy, and Women: Diet Quality and Dysmenorrhea

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Key Points

- Dysmenorrhea or menstrual pain is one of the most common gynecologic complaints among adolescent and young females.
- Prostaglandin (PG) is the major underlying cause of painful menstruation.
- Management of dysmenorrhea would have an important social influence for this chronic problem.
- There is a wide range of treatment options available to relief the symptoms of primary dysmenorrhea, however these approaches showed variable effectiveness benefits and are troubled by some negative side effects.
- A recent study showed a strong association between intake of dietary calcium and risk of dysmenorrhea.
- When adolescent and young females who experience dysmenorrhea considered dietary calcium approach, the majority of participants documented some improvement in dysmenorrheal symptoms and fewer participants reported the need for medications for pain relief.
- Therefore, dietary calcium may be considered as a promising nutritional therapy for the relief of pain and symptoms associated with menstruation, which can significantly influence the quality of life of adolescent girls and young women during the reproductive age.

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Dysmenorrheal • Dietary factors • Lifestyle modification • Calcium • Dairy products

Abbreviations

25(OH)D	25-Hydroxyvitamin D
CAM	Complementary and Alternative Medicines
IRB	Institutional Review Board
NSAIDs	Nonsteroidal anti-inflammatory drugs
OCs	Oral contraceptives
PGs	Prostaglandins
PTH	Parathyroid hormone

Introduction

Dysmenorrhea is a Greek word that means painful or difficult monthly menstrual flow. It is also defined as menstrual cramps of uterine origin. Dysmenorrhea is one of the most common gynecologic complaints among adolescent and young females [1, 2]. Different types of studies from both developed and developing countries have found a consistently high prevalence of dysmenorrhea in women of different ages and nationalities. Dysmenorrhea is more prevalent in adolescent women. It occurs with an estimated prevalence of up to 97 % of menstruating females [3].

Dysmenorrhea is commonly subcategorized into two distinct types based on pathophysiology: primary and secondary. Primary dysmenorrhea is defined as painful menses in women with normal pelvic anatomy. It usually begins during adolescence, typically in the first few years after menarche with an age range between 17 and 22 years.

Secondary dysmenorrhea becomes more common as woman ages, many years to decades after menarche and menstrual pain is associated with organic pelvic pathology such as endometriosis, chronic pelvic inflammatory disease, fibroid tumors, adenomyosis, ovarian cyst, uterine polyps, and the use of intrauterine contraceptive devices [4, 5].

Risk Factors

The severity of dysmenorrhea is associated with several risk factors including early onset of puberty, long and heavy menstrual flow, low body mass index or dieting, stress, depression, anxiety, smoking, obesity, and alcohol consumption.

However, some of the risk factors such as smoking, obesity, and alcohol consumption were not found to be consistently associated with primary dysmenorrhea [5–8].

Symptoms

Dysmenorrhea is characterized by lower abdominal pain that occurs during menstruation, but may start 2 or more days before menstruation and persists up to 72 h. The pain may also radiate to the lower back or inner thighs [9, 10]. Frequently, pain is associated with several types of symptoms including headache, backache, general weakness, nausea, vomiting, dizziness, sweating, abdominal bloating, loose stool, depression, excitability, and irritability [6, 9–11].

Pathogenesis of Primary Dysmenorrhea

The pathogenesis of primary dysmenorrhea is not fully understood. Current evidence suggests that prostaglandins (PGs) are the major underlying cause of painful menstruation [12]. Evidence that support this theory is the presence of high level of prostaglandins PGF2 and PGE2 in menstrual fluid that correlate with symptoms of dysmenorrhea [13]. PGs are potent compounds that induce a wide range of physiologic and pathologic responses. PGs are synthesized mainly from arachidonic acid, a C20:4 omega-6 fatty acid which is found in membrane phospholipids. Before the start of menstruation and after the drop in progesterone level, arachidonic acid is released from cellular membrane phospholipids by phospholipase A2 for the synthesis of prostaglandins (PGs) in the uterus, which leads to constriction of arterioles, and reduction in uterine blood flow and pain level [14, 15].

Treatment of Dysmenorrhea

A wide range of treatment options for primary dysmenorrhea are available [16]. Virtually all treatment options are directed toward correction of the risk factors or the etiology that produced dysmenorrhea with minimal side effects. However, these approaches showed variable benefits in the management of menstrual pain.

Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most common pharmacologic treatment for primary dysmenorrhea [10, 17]. Several randomized controlled trials demonstrated the superiority of NSAIDs in treating of primary dysmenorrhea compared with placebo but no superiority was established among different NSAIDs in the treatment options of dysmenorrhea [15, 18]. However, despite the considerable efficacy of NSAIDs, not all adolescents with dysmenorrhea respond to NSAIDs. For many women, NSAID therapies offer inadequate treatment with a failure rate of 10–25 % [19]. This failure was attributed to different mediators or causes that have been implicated to either the pathogenesis of primary dysmenorrhea or the secondary causes of dysmenorrhea [1, 20]. Moreover, some women have intolerable side effects to current therapies such as gastric upset and even infertility [21]. Simple analgesics such as paracetamol and aspirin can be considered for pain relief in women when other treatments are contraindicated or intolerable [1]. NSAIDs affect the physiological mechanisms behind menstrual pain, inhibiting prostaglandin production through reducing the activity of the cyclooxygenase pathway, which is the rate-limiting factor in the conversion of arachidonic acid to PGs [1].

Oral Contraceptive Pills

Treatment of dysmenorrhea also includes a well-accepted, off-label use of oral contraceptives (OCs) [6, 22]. Results from multiple studies indicate that low-dose oral contraceptive pills can effectively reduce the incidence and severity of pain and symptoms associated with primary dysmenorrhea [23]. A recent randomized controlled study demonstrated a significant improvement of primary dysmenorrhea and its associated symptoms in response to combined low-dose oral contraceptives ethinyl estradiol and norethisterone (IKH-01) treatment [24].

Reduced menstrual pain severity was attributed to reduction in uterine contractions and volume of menstrual flow through suppression of PG synthesis [25]. However, some women prefer not to use OCs because of side effects or cultural reasons. Adverse effects of combined OCs including headache, nausea, and weight gain have been reported [24].

Complementary and Alternative Medicines

A number of alternative therapies have been studied for the treatment of dysmenorrhea and have been reported to be effective in relieving menstrual cramps. However, some reports have small number of participates and only short-term follow-ups, and sometimes are limited to only one trial.

Herbal Medicine

Complementary and Alternative Medicines (CAM) research has focused on the possible beneficial effects of herbal preparations. The most widely studied formulations are the traditional Chinese herbal preparations. Chinese herbal preparations are found to be effective for the treatment of dysmenorrhea with minimal side effects [26]. The effectiveness of Chinese herbals in dysmenorrhea therapies could be related to several mechanisms including decrease of PG levels, modulating nitric oxide, elevation of plasma beta-endorphin levels, blocking of calcium-channels, and improvement of microcirculation [26].

Dietary Factors

A variety of nutrients have been the focus of number of researches that examined the possible beneficial effects in management of primary dysmenorrhea including vitamin B1 (thiamine), vitamin B3 (niacin), vitamin B6 (pyridoxine), vitamin E, fish oil supplement, calcium, and magnesium.

Vitamin B1 and B6

In a single study, supplementation of 100 mg of vitamin B1 daily for 3 months to 556 Indian adolescent girls with moderate to very severe dysmenorrhea was shown to be an effective therapy. The majority of participants (87 %) reported no menstrual pain as compared to placebo [27]. Some evidence also exists for the benefit of 100 mg/day supplementation of vitamin B6 taken with or without magnesium in management of dysmenorrhea. However, magnesium can have adverse effects such as constipation.

Vitamin E

Three trials have demonstrated the effectiveness of vitamin E in relieving menstrual pain of primary dysmenorrhea.

In the first trial, 100 adolescent girls who suffered from primary dysmenorrhea received supplements of 500 IU/day vitamin E (about 333 mg) 2 days before the onset of menses and continued through the first 3 days of menstruation for 2 months. Vitamin E supplementation significantly improved menstrual pain as compared to placebo [28]. Also the intake of 500 mg vitamin E 10 days before the menses and continued until the fourth day of menses for 3 months was reported to improve menstrual pain as compared to placebo (68 % vs. 18 %) [29]. Similar result was obtained in another randomized controlled trial when vitamin E (200 U/day) was administered 2 days before the menses and continued through the first 3 days of menses for 3 months [30]. In this trial, participants also reported reduced blood loss.

Fish Oil

The protective effect of fish oil on dysmenorrhea is supported by several crossover design clinical trials. In the first trial, 42 girls who experience dysmenorrhea were placed into two groups of 21 each. One group received 6 g of fish oil (1,080 mg EPA, 720 mg DHA, and 1.5 mg vitamin E) daily for 2 months followed by a placebo for an additional 2 months, while the second group received placebo for the first 2 months followed by fish oil for 2 more months. The results show marked reduction in symptoms of dysmenorrhea due to fish oil treatment [31]. In the second trial, 36 girls aged 18–22 were randomly allocated into two groups. The treated group received 15 mL of fish oil per day for 3 months while the control group received placebo. In that trial, fish oil was proven to be more effective for relief of menstrual pain than placebo [32]. In another double blind, randomized, placebo-controlled trial, 78 Danish women who experience dysmenorrhea [33] supplemented with 2.5 g/day fish oil with or without vitamin B12, seal oil (5 g/day), or placebo for three menstrual cycles. Women taking fish oil plus B12 reported a significant reduction in the intensity of menstrual pain and some associated symptoms.

The beneficial effect of fish oil is apparently due to its content of omega-3 fatty acids (eicosapentaenoic acid and decosahexaenoic acid). Several mechanisms have been proposed for the anti-inflammatory effect of omega-3 fatty acids including first: fewer PGs are made from omega-3 fatty acids, second: omega-3 can inhibit the formation of arachidonic acid from C18 fatty acids at the level of δ^6 desaturase enzyme, third: binding of omega-3 fatty acids to cyclooxygenase reduces the conversion of omega-6 to PGs, and fourth: PGs derived from omega-3 fatty acids are less potent than those formed from the omega-6 fatty acids [34]. Therefore, high omega-3 intake is expected to be associated with milder menstrual pain. Adverse effects of fish oil have been reported, including stomach upset, slight nausea and bad taste, and acne.

Lifestyle Modification

Few studies have examined the effect of lifestyle modification with respect to ameliorating symptoms of dysmenorrhea, including low-fat vegetarian diet [35] and exercise [36]. Low-fat vegetarian diet was reported to be associated with reduction in dysmenorrhea duration and intensity as compared with baseline in a crossover design which involved 33 women for two menstrual cycles. The effect was attributed to diet influences on estrogen activity. A low-fat vegetarian diet was associated with increased serum sex hormone-binding globulin concentration, which inactivates estrogens. Estrogen and progesterone stimulate the endometrium, which is the source of the PGs which induce ischemia and uterine muscle contraction [37].

Dietary Habits and Primary Dysmenorrhea

Although the prevalence and severity of dysmenorrhea in adolescents and young females are high, many girls either do not ask for medical advice or are undertreated [17]. The severity of dysmenorrheal pain varies among different females; however, it could be severe enough to cause a substantial negative impact on women's daily activities. Pain may inconvenience a woman during holidays, social activities, or sometimes when high performance is required. Chronic recurrent pain of dysmenorrhea leads to high rate of school or work absences as well as limits their class concentration, which affects their academic performance [12, 38]. Besides the negative impact on the society, dysmenorrhea causes significant cost to the health care system and imposes significant socioeconomic consequences [39].

Although there is a wide range of treatment options available to relief dysmenorrheal pain, these approaches showed variable benefits in the management of menstrual pain. Therefore, finding an effective and safe nonpharmacologic intervention for management of dysmenorrhea would have an important social influence on females.

Knowledge of beneficial food-related practices can provide better inexpensive nonpharmacologic alternative treatment for primary dysmenorrhea. It can also limit several negative side effects of the current pharmacological treatments. In addition, it will contribute positively to the general health of adolescent girls and young women.

Few studies had examined the relationship between dietary habits and menstrual pain.

Higher intake of fruit, eggs, and animal and fish products was found to correlate with milder menstrual pain [34, 40]. The protective effect of fruit and eggs was attributed to their calcium and magnesium contents. However, the role of calcium in the treatment of primary dysmenorrhea was subjected to controversial results. A study with a relatively small sample size (only ten women) conducted by Penland et al. [41] found that increased dietary intake of calcium (587 or 1,336 mg/day) and magnesium (1.0 or 5.6 mg/day) reduced menstrual pain and improved some psychological symptoms. In contrast, Di Cintio et al. [42] reported no clear relationship between the intake of 50 various food items (grains, vegetables, fruits, meat, poultry and fish, milk and milk products, and oil) and risk of dysmenorrhea with the exception of cheese and eggs consumption, which showed a little positive association. The finding of Di Cintio et al. does not support previous suggestions that calcium [41] and even omega-3 fatty acids [34, 40] intake reduces risk for dysmenorrhea.

The effectiveness of magnesium in the treatment of primary dysmenorrhea has been also evaluated in three small randomized controlled trials [43–45]. The results suggest that magnesium supplementation is effective in treatment of dysmenorrhea. The appropriate dose of magnesium for treatment of dysmenorrhea is not clear due to different magnesium dose and formulation used in different trials. The improvement of dysmenorrhea symptoms was attributed to reduction in the $PGF2\alpha$ level and to direct muscle relaxant and vasodilatory

Table 5.1 Dysmenorrhea in relation to daily intake of dairy products

	Total number of dairy servings/day				
Parameter	0	1	2	3	4
Number (%) of female students who experience dysmenorrheic pain (total number = 111)	36(32.4)	26(23.4)	26(23.4)	11(9.9)*	12(10.8)**
Number of female students who experience no dysmenorrheic pain (total number = 16)	1(6.3)	3(18.7)	7(43.8)	4(25)	1(6.3)
Total number	37	29	33	15	13

There was significant reduction in dysmenorrheic pain in participants who took three daily servings (*P value = 0.000) or four daily servings (**P value = 0.000) of dairy products compared to participants who took none. Cited from ref. [10] with permission from John Wiley & Sons Ltd.

Table 5.2 The association between intake of daily dairy servings and dysmenorrhea-associated symptoms

	Total number of daily servings (*n* %)				
Symptom	0	1	2	3	4
Nausea and vomiting	7(26.92)	5(19.23)	8(30.77)	3(11.54)	3(11.54)
Sweat	13(35.14)	10(27.03)	8(21.62)	4(10.81)*	2(5.41)
Abdominal bloating	32(32.32)	24(24.24)	23(23.23)	10(10.10)**	10(10.10)
Stool	17(34.00)	16(32.00)	12(24.00)	4(8.00)***	1(2.00)
Dizziness	17(37.78)	9(20.00)	11(24.44)	3(6.67)****	5(11.11)
Others	7(26.92)	5(19.23)	7(26.92)	2(7.69)	5(19.23)

Cited from ref. [10] with permission from John Wiley & Sons Ltd.
Significant reduction in dysmenorrhea-associated symptom was found in participants who took three compared with participants who took no daily servings of dairy products
n number of students expressing the symptom
*P value = 0.009
**P value = 0.000
***P value = 0.001
****P value = 0.000

effect of magnesium [43]. However, treatment with magnesium reported to be either free of side effect [43, 44] or associated with intestinal discomfort and other minor adverse effects [45].

Although the available data provides little direct scientific evidence to the relationship between dietary habits and risk factor for dysmenorrhea, consistent observations indicate the presence of an association between frequency of dietary calcium intake and the risk of dysmenorrhea. A study by Abdul-Razzak et al. [10] showed the possible relationship between daily dairy products intake and risk of dysmenorrhea and other associated symptoms among university female students. A detailed description of field data collection can be found elsewhere. In this study, a strong correlation between dairy products intake and dysmenorrhea and its associated symptoms among participants was found. A significantly lower percentage of female students expressing dysmenorrhea were observed when their intakes of dairy products were three or four servings per day as compared to female who took none (Table 5.1). Similar influence pattern of dairy products on most dysmenorrhea-associated symptoms was also observed as symptoms frequency was analyzed against the number of daily serving of dairy products (Table 5.2). On the other hand the severity of primary dysmenorrhea decreased with increasing the number of daily dairy products intake (Fig. 5.1).

This study appears to elucidate the relationship between dietary intake of dairy products and the risk of dysmenorrhea.

Fig. 5.1 The relationship between daily intake of dairy serving and pain severity. Cited from ref. [10] with permission from John Wiley & Sons Ltd. *$P=0.028$, **$P=0.002$

Dietary Calcium Is a Promising Nutritional Therapy for Primary Dysmenorrhea

In another study 35 participants of those who were involved in a previous study [10] and experienced dysmenorrhea and their daily intake of dairy products was none were asked to take three daily servings of dairy products, 7–10 days before the onset of menstruation. To evaluate the overall improvement in dysmenorrhea after including dairy products in the diet, participants were requested to fill out another questionnaire to assess any changes in the usual pattern of their menstruation, if any improvement to pain or the other symptoms that are usually associated with menstruation were noticed, and if daily activities were continued as usual with less or without need to administer medications or herbs.

The overall improvement in dysmenorrheal pain and associated symptoms after considering the dairy products therapy were evaluated as "excellent, very good, good, and slight." After considering dietary calcium management, 83 % of participates had documented some degree of improvement regarding their menstrual pain. About 11 % of participants reported an excellent response to the dietary approach with complete resolution of pain, while 26 %, 44 %, and 19 % of students have reported very good, good, and slight improvement, respectively. Students also reported improvement of some symptoms associated with dysmenorrhea in different degrees (11 % for nausea and vomiting, 14 % for sweating, 31 % for abdominal bloating, 9 % for loose stools, and 23 % for dizziness). Interestingly and after considering the dietary calcium approach, fewer participants had reported the need for medications to relieve pain associated with dysmenorrhea (46 % vs. 68.57 %, $P=0.047$). Additionally, lower percentage of participating students continued to report a negative impact of dysmenorrhea on their daily activities (46 % vs. 74.3 %, $P=0.01$).

The finding of this single cohort study clearly elucidated that dietary calcium is a promising nutritional therapy for the relief of pain and symptoms associated with menstruation, thus can significantly influence the quality of life of adolescent girls and young women. Besides calcium can contribute positively to general health including reduction of the risk of osteomalacia and osteoporosis in older age [46], since more than 90 % of peak bone mass is achieved at the end of adolescence [47] and the majority of the cases of primary dysmenorrhea usually begin.

Evaluation of Nutritional Intervention on Dysmenorrhea

A total of 185 healthy university students between the ages of 18 and 24 years and who experience dysmenorrhea were involved in a study, which aimed to assess the association between daily intake of dairy products, vitamin D status, parathyroid hormone (PTH) level, and dysmenorrhea symptoms for the period from January to April 2010. Participants were allowed to complete a guided self-filling questionnaire to serve as a baseline. The questionnaire was generally prepared with reference to previous study [10] and consisted of three parts. The first part of the questionnaire was about participants' demographics. The second contained information regarding menstruation; possible symptoms associated with dysmenorrhea and daily consumption of dairy products.

Pain severity was measured using numerical rating scale (NRS) ranged from 1 to 10 and also graded as mild, severe, and very severe as previously described [10].

All participants donated venous blood sample, which was assayed for plasma 25-hydroxyvitamin D (25(OH)D) level and intact PTH level.

Vitamin D status was divided into three diagnostic categories according to plasma 25(OH)D levels; vitamin D sufficiency, vitamin D insufficiency, and vitamin D deficiency.

Besides, participants were advised to consume three servings of dairy products daily and increase the exposure to direct sunlight. Participants with severe vitamin D deficiency were advised to visit family physician at University Teaching Primary Health Care Center for appropriate treatment. The effectiveness of dietary calcium as a therapy to relief pain and symptoms associated with menstruation was evaluated after at least three consecutive menstruations in 84 out of the 185 subjects who followed our recommendation to increase daily dietary intake to three servings. In those participants, there was a significant reduction ($P < 0.05$) in pain severity as compared to baseline. Pain severity decreased from 5.98 ± 2.1 to 3.6 ± 2.3. The overall improvement in dysmenorrheal symptoms is shown in Fig. 5.2. A highly significant improvement in all dysmenorrheal symptoms was noted among participants after the intervention ($P < 0.001$). The most improved symptom was abdominal bloating followed by dizziness, sweating, nausea, and losing stool. Also the results showed that after the dietary calcium intervention, there was a significant reduction in analgesic drugs use ($P < 0.001$). Out of 67 participants who reported taking analgesic drugs, 45 of them decreased or discontinued the drug use (Fig. 5.3).

The finding of this single cohort study shows that dietary calcium is a promising nutritional therapy for the relief of pain and symptoms associated with menstruation thus can significantly influence the quality of life of adolescent girls and young women.

Possible Role of Calcium in the Etiology of Primary Dysmenorrhea

Current understanding of the pathogenesis in primary dysmenorrhea implicates excessive PGs that are released from the endometrium [12]. The level of PGs can be reduced to below normal with NSAIDs, and because of that, these medications are considered effective treatments for the management of menstruation pain. In the female body, the uterus is a muscle that contracts frequently and rhythmically [48]. Strong uterine contractions are painful. Uterine hypercontractility is associated with reduced blood supply to the uterus and eventually leads to increased peripheral nerve hypersensitivity causing pain [49]. In another word, contractility of the uterus appears to be the major source of pain during menstruation.

Calcium is the most abundant mineral in the human body. Calcium has several important physiological functions. Extracellular calcium is the ultimate source of intracellular calcium, therefore, alterations in calcium are associated with many disturbances including its effect on neuromuscular activity. Normal calcium level controls the contractility, tone, and relaxation of smooth muscle, while reduction in calcium level may increase neuromuscular excitability,

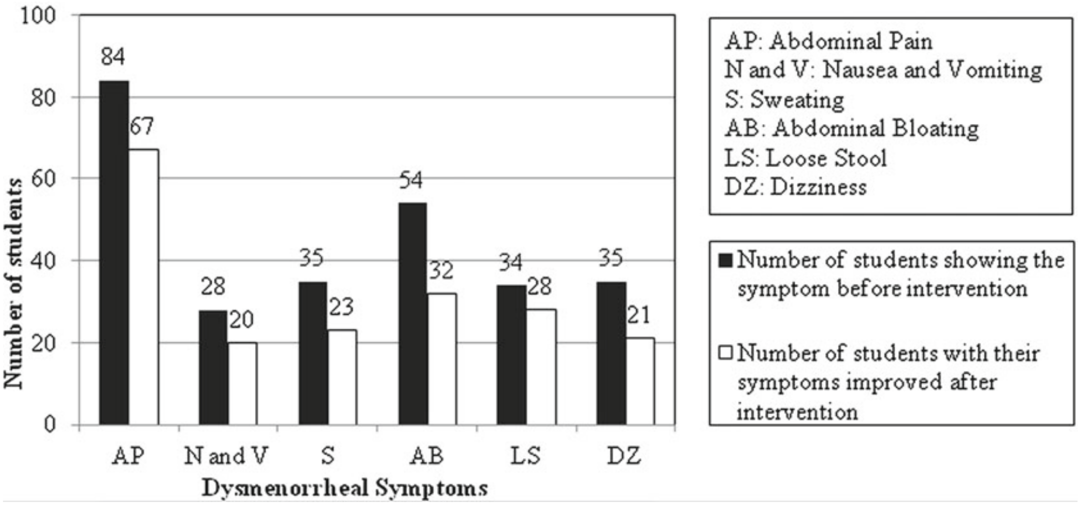

Fig. 5.2 Participants' dysmenorrheal symptoms before and after dietary calcium intervention. (Unpublished) Significant improvement in all dysmenorrheal symptoms was observed among participants after including dairy products in the diet ($P<0.001$). *AP* abdominal pain, *N and V* nausea and vomiting, *S* sweating, *AB* abdominal bloating, *LS* loose stool, *DZ* dizziness. Non-parametric analysis using McNemar test was used to find out the improvement in dysmenorrheal symptoms. $P<0.05$ considered statistically significant

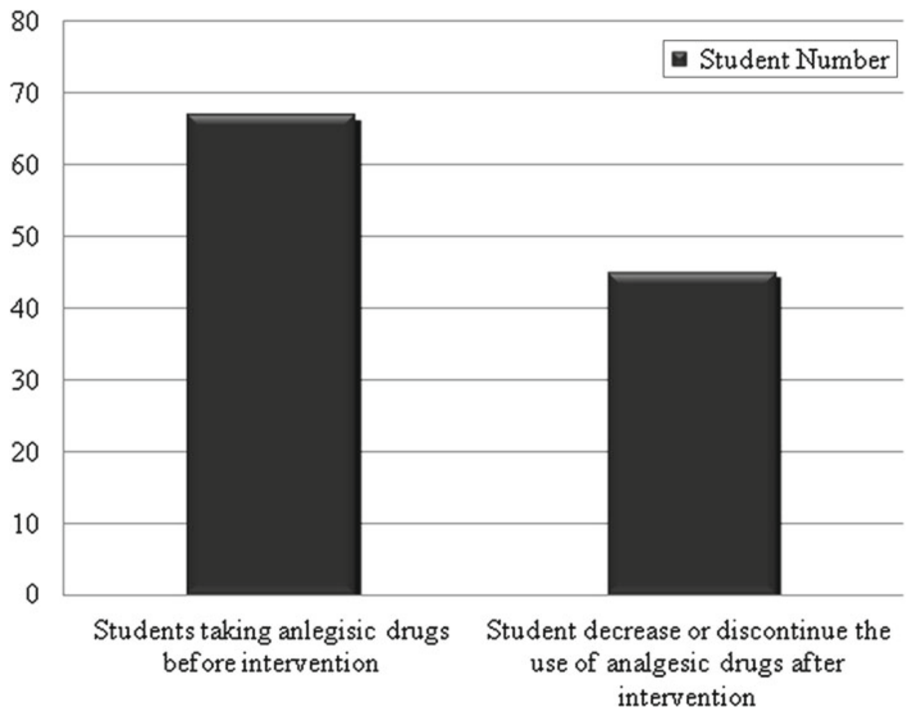

Fig. 5.3 Participants' medication use before and after dietary calcium intervention. (Unpublished) Significant reduction in pain severity combined with significant reduction in analgesic drugs use was observed among participants after including dairy products in the diet ($P<0.001$). Non-parametric analysis using McNemar test was used to find out the change in analgesic drugs use. $P<0.05$ considered statistically significant

resulting in increased spasms and muscular contractions [49]. However, mild hypocalcemia is often asymptomatic [50]. Therefore, low intake of dietary calcium may be a predisposing factor for increased uterine cramping and pain among women with primary dysmenorrhea.

The finding of this research may also explain the high prevalence of dysmenorrhea among adolescent females, since most of the world population do not satisfy their calcium needs as calcium is mostly found in one class of food, namely milk, and in adolescents and adults, three dairy servings per day are recommended [47].

Conclusions

Dysmenorrhea is one of the most common gynecologic complaints among adolescent and young females. Generally, dysmenorrhea has a negative impact on the quality of life in young women during their reproductive age. Although there is a wide range of treatment options available to relief the symptoms of primary dysmenorrhea, these approaches showed variable benefits in the management of menstrual pain and are troubled by some negative side effects.

The results of these studies suggest that dietary calcium has a functional role in etiology of dysmenorrhea and is a promising nutritional therapy for the relief of dysmenorrheal pain and associated symptoms.

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References

1. Harel Z. Dysmenorrhea in adolescents and young adults: etiology and management. J Pediatr Adolesc Gynecol. 2006;19:363–71.
2. Jamieson DJ, Steege JF. The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. Obstet Gynecol. 1996;87:55–8.
3. Latthe P, Latthe M, Say L, Gülmezoglu M, Khan KS. WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. BMC Public Health. 2006;6:177.
4. Morrow C, Naumburg EH. Dysmenorrhea. Prim Care. 2009;36:19–32, vii.
5. Proctor ML, Farquhar C. Diagnosis and management of dysmenorrhea. BMJ. 2006;332:1134–8.
6. French L. Dysmenorrhea. Am Fam Physician. 2005; 71:285–91.
7. Wang L, Wang X, Wang W, Chen C, Ronnennberg AG, Guang W, et al. Stress and dysmenorrhoea: a population based prospective study. Occup Environ Med. 2004;61:1021–6.
8. Latthe P, Mignini L, Gray R, Hills R, Khan K. Factors predisposing women to chronic pelvic pain: systematic review. BMJ. 2006;332:749–55.
9. Agarwal AK, Agarwal A. A study of dysmenorrhea during menstruation in adolescent girls. Indian J Community Med. 2010;35:159–64. doi:10.4103/0970-0218.62586.
10. Abdul-Razzak KK, Ayoub NM, Abu-Taleb AA, Obeidat BA. The influence of dietary intake of dairy products on dysmenorrhea. J Obstet Gynaecol Res. 2010;36:377–83.
11. Dawood MY. Dysmenorrhea. Clin Obstet Gynecol. 1990;33:168–78.
12. Willman EA, Collins WP, Clayton SG. Studies in the involvement of prostaglandins in uterine symptomatology and pathology. Br J Obstet Gynaecol. 1976;83:337–41.
13. Chan WY, Hill JC. Determination of menstrual prostaglandin levels in non-dysmenorrheic and dysmenorrheic subjects. Prostaglandins. 1978;15:365–75.
14. Simopoulos AP. Omega-3 fatty acids in health and disease and in growth and development. Am J Clin Nutr. 1991;54:438–63.
15. Zahradnik HP, Hanjalic-Beck A, Groth K. Nonsteroidal anti-inflammatory drugs and hormonal contraceptives for pain relief from dysmenorrhea: a review. Contraception. 2010;81:185–96.
16. Wong CL, Farquhar C, Roberts H, Proctor M. Oral contraceptive pill for primary dysmenorrhoea. Cochrane Database Syst Rev. 2009;(4):CD002120.
17. Harel Z. Dysmenorrhea in adolescents. Ann N Y Acad Sci. 2008;1135:185–95.
18. Marjoribanks J, Proctor M, Farquhar C, Derks RS. Nonsteroidal anti-inflammatory drugs for dysmenorrhoea. Cochrane Database Syst Rev. 2010;(1):CD001751.
19. Proctor ML, Murphy PA. Herbal and dietary therapies for primary and secondary dysmenorrhoea. Cochrane Database Syst Rev. 2001;(3):CD002124.
20. Nigam S, Benedetto C, Zonca M, Leo-Rossberg I, Lubbert H, Hammerstein J. Increased concentrations of eicosanoids and platelet-activating factor in menstrual blood from women with primary dysmenorrhea. Eicosanoids. 1991;4:137–41.

21. Coco AS. Primary dysmenorrhea. Am Fam Physician. 1999;60:489–96.
22. Proctor ML, Farquhar CM. Dysmenorrhoea. Clin Evid. 2006;15:2429–48.
23. Hendrix SL, Alexander NJ. Primary dysmenorrhea treatment with a desogestrel-containing low-dose oral contraceptive. Contraception. 2002;66:393–9.
24. Harada T, Momoeda M, Terakawa N, Taketani Y, Hoshiai H. Evaluation of a low-dose oral contraceptive pill for primary dysmenorrhea: a placebo-controlled, double-blind, randomized trial. Fertil Steril. 2011;95:1928–31.
25. Kido A, Togashi K, Kataoka M, Maetani Y, Nakai A, Kataoka ML, et al. The effect of oral contraceptives on uterine contractility and menstrual pain: an assessment with cine MR imaging. Hum Reprod. 2007;22: 2066–71.
26. Jia W, Wang X, Xu D, Zhao A, Zhang Y. Common traditional Chinese medicinal herbs for dysmenorrhea. Phytother Res. 2006;20:819–24.
27. Gokhale LB. Curative treatment of primary (spasmodic) dysmenorrhea. Indian J Med Res. 1996;103:227–31.
28. Ziaei S, Faghihzadeh S, Sohrabvand F, et al. A randomised placebo-controlled trial to determine the effect of vitamin E in treatment of primary dysmenorrhoea. BJOG. 2001;108:1181–3.
29. Fugh-Berman A, Kronenberg F. Complementary and alternative medicine (CAM) in reproductive-age women: a review of randomized controlled trials. Reprod Toxicol. 2003;17:137–52.
30. Ziaei S, Zakeri M, Kazemnejad A. A randomised controlled trial of vitamin E in the treatment of primary dysmenorrhea. BJOG. 2005;112:466–9.
31. Harel Z, Biro FM, Kottenhahn RK, Rosenthal SL. Supplementation with omega-3 polyunsaturated fatty acids in the management of dysmenorrhea in adolescents. Am J Obstet Gynecol. 1996;174:1335–8.
32. Moghadamnia AA, Mirhosseini N, Abadi MH, Omranirad A, Omidvar S. Effect of Clupeonella grimmi (anchovy/kilka) fish oil on dysmenorrhoea. East Mediterr Health J. 2010;16:408–13.
33. Deutch B, Jorgensen EB, Hansen JC. Menstrual discomfort in Danish women reduced by dietary supplements of omega-3 PUFA and B12 (fish or seal oil capsules). Nutr Res. 2000;20:621–31.
34. Balbi C, Musone R, Menditto A, et al. Influence of menstrual factors and dietary habits on menstrual pain in adolescence age. Eur J Obstet Gynecol Reprod Biol. 2000;91:143–8.
35. Barnard ND, Scialli AR, Hurlock D, Bertron P. Diet and sex-hormone binding globulin, dysmenorrhea, and premenstrual symptoms. Obstet Gynecol. 2000;95:245–50.
36. Brown J, Brown S. Exercise for dysmenorrhoea. Cochrane Database Syst Rev. 2010;(2):CD004142, review.
37. Jaster B. Suggest vegetarian diet to patients with dysmenorrhea [letters to the editor]. Am Fam Physician. 2005;72(8). http://www.aafp.org/afp/2005/1015/ol2.html. Accessed 20 Nov 2011.
38. Sanfilippo J, Erb T. Evaluation and management of dysmenorrhea in adolescents. Clin Obstet Gynecol. 2008;51:257–67.
39. Dawood MY. Ibuprofen and dysmenorrhea. Am J Med. 1984;77:87–94.
40. Deutch B. Menstrual pain in Danish women correlated with low n-3 polyunsaturated fatty acid intake. Eur J Clin Nutr. 1995;49:508–16.
41. Penland JG, Johnson PE. Dietary calcium and manganese effects on menstrual cycle symptoms. Am J Obstet Gynecol. 1993;168:1417–23.
42. Di Cintio E, Parazzini F, Tozzi L, Luchini L, Mezzopane R, Marchini M, et al. Dietary habits, reproductive and menstrual factors and risk of dysmenorrhea. Eur J Epidemiol. 1997;13:925–30.
43. Seifert B, Wagler P, Dartsch S, Schmidt U, Nieder J. [Magnesium—a new therapeutic alternative in primary dysmenorrhea]. Zentralbl Gynakol. 1989; 111:755–60.
44. Benassi L, Barletta FP, Baroncini L, Bertani D, Filippini F, Beski L, et al. Effectiveness of magnesium pidolate in the prophylactic treatment of primary dysmenorrhea. Clin Exp Obstet Gynecol. 1992;19: 176–9.
45. Fontana-Klaiber H, Hogg B. [Therapeutic effects of magnesium in dysmenorrhea]. Schweiz Rundsch Med Prax. 1990;79:491–4.
46. Dwyer J. Starting down the right path: nutrition connections with chronic diseases of later life. Am J Clin Nutr. 2006;83(2):415S–20.
47. Fulgoni VL, Huth PJ, DiRienzo DB, Miller GD. Determination of the optimal number of dairy servings to ensure a low prevalence of inadequate calcium intake in Americans. J Am Coll Nutr. 2004;23: 651–9.
48. Akerlund M. Modern treatment of dysmenorrhea. Acta Obstet Gynecol Scand. 1990;69:563–4.
49. Johnson PE, Lykken GI. Dietary calcium and manganese effects on menstrual cycle symptoms. Am J Obstet Gynecol. 1993;168:1417–23.
50. Marshall WJ, Bangert SK, editors. Clinical chemistry. 6th ed. London: Mosby; 2008.

6 Maternal Diet Quality and Pregnancy Outcomes

Clara L. Rodríguez-Bernal, Marisa Rebagliato, Leda Chatzi, Clara Cavero Carbonell, Carmen Martos, and Ferran Ballester

Key Points

- There is evidence showing that dietary intake during pregnancy influences pregnancy outcomes such as fetal growth, preterm delivery or neural tube defects.
- Adverse pregnancy outcomes are important predictors of infant and adult mortality and morbidity.
- Assessment of the relationship between diet during gestation and pregnancy outcomes has been based mainly on nutrient deficiencies; moreover mainly in industrialized countries, the results for certain nutrients or food groups are still controversial.
- The assessment of overall diet represents nutrient and food intake more accurately and takes into account nutrient synergies and interactions, and from the public health perspective would facilitate health education on the effects of maternal diet on the health of the offspring.
- Maternal diet as a composite measure, its quality and its effects on pregnancy outcomes has been scarcely studied.
- The body of epidemiological evidence regarding the association of diet quality and pregnancy outcomes is weak overall nonetheless suggestive of a beneficial effect of a high-quality diet even in industrialized settings where populations are assumed to be well nourished.
- Well-designed randomized controlled trials are needed in order to assess the effect of high-quality diets during the gestational period on pregnancy outcomes.

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- Multinational studies in this context would help to support policy measures regarding diet quality at an international level.
- The existing evidence in relation to pregnancy outcomes adds importance to the current public health messages recommending balanced healthy diets containing fruit and vegetables, starchy carbohydrates from cereals and legumes and unsaturated fats.

Keywords
Diet quality • Dietary patterns • Pregnancy outcomes • Nutrition • Fetal growth • Preterm birth • Neural tube defects • Dietary indexes

Abbreviations

AHEI	Alternate Healthy Eating Index
BMI	Body mass index
DGI	Dietary Glycemic Index
DQI	Diet Quality Index
FGR	Fetal growth restriction
LGA	Large for gestational age
MD	Mediterranean diet
MDS	Mediterranean Diet Scale
MUFA	Monounsaturated fatty acids
NTD	Neural tube defects
PUFAS	Polyunsaturated fatty acids
RCT	Randomized controlled trial
SGA	Small for gestational age
USA/US	United States of America

Introduction

Dietary intake during the periconceptional and pregnancy period has been shown to influence pregnancy outcomes such as fetal growth, preterm delivery, and congenital anomalies, particularly, but not exclusively, in nonindustrialized/low-income populations [1–3]. In the context of congenital anomalies, strong evidence on the effect of nutrient intake is available mainly for neural tube defects (NTD) [4].

Adverse pregnancy outcomes, in turn, are important predictors of the risk of infant mortality and morbidity [5, 6] and might determine a higher susceptibility to developing chronic diseases in adulthood [7, 8]. This concept, known as early nutrition programming or metabolic programming, has gained broad recognition among researchers as evidence increasingly shows that diet during pregnancy, breastfeeding, and complementary feeding could have an irreversible impact on child development and long-term health [9].

When considering the effect of maternal dietary intake on pregnancy outcomes it is important to acknowledge that many micronutrient requirements increase during pregnancy in relation to the inherent physiological changes and in order to satisfy the nutrient supply to the fetus. A complex relationship exists between maternal nutrient intake and fetal nutrient uptake and the timing of adequate maternal nutrition is extremely important, as developing organ systems respond directly to the availability of nutrients during critical periods of rapid development [10].

Research on the child health effects of maternal nutrition has focused largely on nutrient deficiencies or the intake of specific dietary components, and there is still controversy about such effects on pregnancy outcomes in populations assumed to be well nourished [1].

The study of specific dietary components may have the drawback of not taking into account the interaction between nutrients and food synergies [11]. Alternatively, assessment of overall dietary intake and its quality in order to examine its effect on disease risk has grown in interest over the last years [12]. Evaluation of diet as a whole might represent food and nutrient intake and its global effect on health outcomes more accurately [13].

There are few data available describing the prevailing patterns of dietary intake among pregnant women in industrialized countries, the quality of overall maternal diet and its effects on pregnancy outcomes. From a public health perspective, it might be more useful to have evidence on the relationship between the quality of diet as a whole, rather than a specific dietary component and health outcomes.

We aimed in this chapter to examine the relationship between maternal diet quality and important pregnancy outcomes, gathering information from recent evidence, summarizing the main findings, and establishing needs for future research.

Pregnancy Outcomes Considered

Fetal Growth Restriction

Fetal growth restriction (FGR), which represents the pathological inhibition of fetal growth and the failure of the fetus to attain its growth potential [14], is an important predictor of an infant immediate and long-term health. Fetuses who do not reach their full growth potential or babies who are born with a low weight have an increased risk of infant mortality and morbidity [5], including poor cognitive and neurological development [15] and a higher risk of developing chronic diseases such as coronary heart disease and type 2 diabetes in adulthood [16, 17].

Anthropometrical measurements at birth (i.e., birth weight, birth length, or head circumference) have been used to assess fetal growth. Small for gestational age (SGA)—defined as a baby whose weight at birth is below the tenth percentile for a given reference growth chart according to their gestational age—is a proxy measure of FGR [18]. However, size at birth can be influenced by factors such as infant gender and parental anthropometric variables, race/ethnicity, or parity [18]. Customized birthweight percentiles have been designed to differentiate better between infants who are small because their in-utero growth has been restricted and infants who are constitutionally small but have reached their full growth potential [19]. The normative values in customized percentiles at younger gestational ages are based on the distribution of the best estimate of intrauterine weights, whereas conventional birthweight charts are based on the weights of live births [20].

Although few studies have examined the effect of diet during pregnancy on intrauterine growth in Western industrialized countries, various aspects of maternal diet are hypothesized to affect fetal growth, including fruit and vegetables [21, 22], fish [3, 23], and dairy products [24].

Other nutritional factors that have been related to FGR are low pre-pregnancy body mass index (BMI) [25], and low concentration or intakes of nutrients such as iron [26], folic acid [2], and vitamins D and E [24, 27]. However, results for nutrient or food intake are not always conclusive [1].

Evidence on Maternal Diet Quality and Fetal Growth Restriction

Recently, interest has shifted to place greater emphasis on dietary patterns that represents a broader picture of food and nutrient consumption and may therefore be more predictive of health outcomes. Nevertheless, the evaluation of dietary patterns and diet quality in pregnant populations and its effect on fetal growth is rare.

Table 6.1 shows a summary of evidence on the relationship between maternal diet quality and fetal growth.

Evidence from observational studies: Two cohort studies have assessed the effect of diet quality on fetal growth [28, 29] using “a posteriori” techniques (which obtain dietary patterns based on observed correlations among dietary variables) [30, 31]. The Danish National Birth Cohort showed that a diet rich in red and processed meat and high-fat dairy products and low intake of fruit and vegetables during pregnancy increased the risk of giving birth to SGA babies [28]. Another birth cohort in New Zealand revealed that the “traditional” diet (meat, fruit, green and root vegetables and dairy products) had a protective effect against SGA births [29].

Only three birth cohorts have assessed the effect of diet quality on fetal growth through the assessment of indexes/scales of diet quality [32–34],

Table 6.1 Studies assessing the relationship between diet quality during pregnancy and fetal growth

Cohort, country	Author (Year)	*N* women	Main exposure	Main growth outcome	Time when diet was measured	Type of study	Main results
Fetal growth							
DNBC, Denmark	Knudsen et al. (2008) [28]	44,612	Dietary patterns	SGA	25 wk	Prospective birth cohort	A diet rich in red meat, high-fat dairy and low intakes of fruit and vegs. increased risk of SGA
Australia	Thompson et al. (2010) [29]	1,714	Dietary patterns	SGA	After delivery (referred to diet at first and third trimesters)	Case–control study	A "traditional" diet (meat, fruit, vegs. and dairy products) reduced risk of SGA
Project Viva, USA	Rifas–Shiman et al. (2009) [32]	1,777	Maternal diet quality (measured by AHEI)	SGA BW	First and second trimester	Prospective birth cohort	No significant association found
INMA, Spain	Rodrıguez-Bernal et al. (2010) [33]	787	Maternal diet quality (measured by AHEI)	FGR BW, BL, HC	12 wk	Prospective birth cohort	Higher AHEI scores increased BW and BL and reduced risk of FGR
Rhea, Greece and INMA, Spain	Chatzi et al. (2012) [34]	889 (Rhea) 2,461 (INMA)	Mediterranean diet (MD)	FGR BW, BL, HC	14–18 wk, Rhea ~14 wk, INMA	Prospective birth cohort	High MD adherence reduced risk of FGR in Mediterranean Spanish populations only
Finland	Kinnunen et al. (2007) [42]	105 (48, IG; 56, CG)	Dietary advice (≥5 fruit and vegs, high-fiber bread and ≤1 portion high-sugar snacks)	High birth weight (HBW) >4,000 g LBW	16–18 wk (3 more visits up to 37 wk)	Intervention	Intervention associated with higher rates of appropriate BW and lower rates of HBW
Finland	Aaltonen et al. (2008) [41]	256 (85, IG1; 86, IG2; 85, CG)	Dietary counseling (more vegs, fruit, vegetable fats and less butter)	BW, BL, HC	14, 24, 34 wk	Intervention	No significant differences found between intervention and control groups

SGA small for gestational age, *wk* week of gestation, *vegs* vegetables, *BW* birth weight, *BL* birth length, *HC* head circumference, *AHEI* Alternate Healthy Eating Index, *FGR* fetal growth restriction, *LBW* low birth weight, *IG* intervention group, *CG* control group

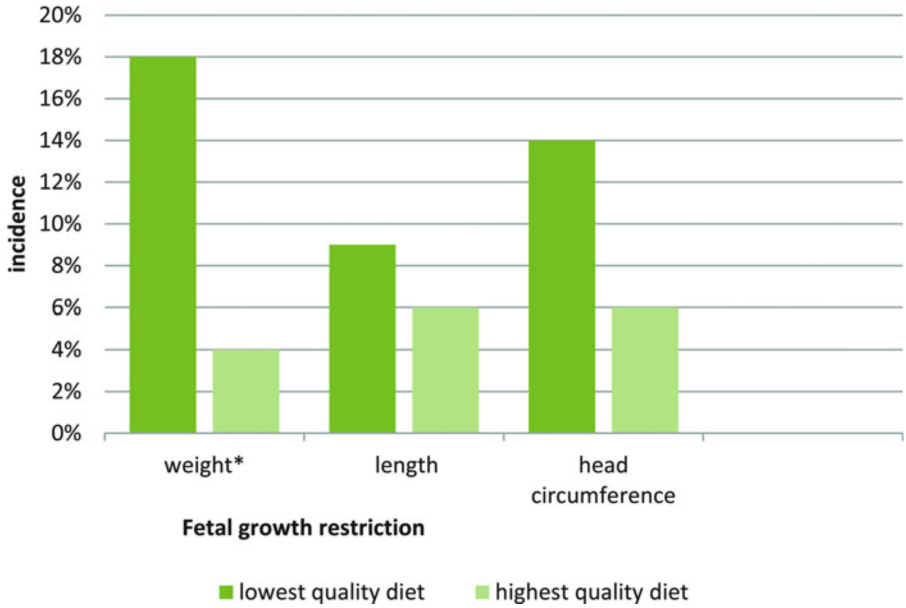

Fig. 6.1 Frequency of fetal growth restriction (FGR) according to diet quality in the INMA-Valencia cohort. Frequency of FGR (in weight, length, and head circumference) between extreme categories (lowest vs. highest) of maternal diet quality during pregnancy, as measured by the AHEI modified for pregnancy. *p* values obtained by using chi-squared test, *statistically significant differences found ($p < 0.05$). INMA-Valencia cohort (unpublished)

which have been defined as "a priori" techniques. Both, "a priori" and "a posteriori" approaches have strengths and drawbacks. One advantage of the "a priori" approach is that it is based on previous knowledge in relation to the health effects of various dietary constituents [30]. The aforementioned cohorts are Project Viva, a birth cohort from the USA [32], and INMA [33] and Rhea [34] mother–child cohorts, from Spain and Greece, respectively. The indexes/scales used were the Alternate Healthy Eating Index (AHEI) [35] and the Mediterranean Diet Scale (MDS) [36], both of which were modified for pregnancy [32–34].

The AHEI modified for pregnancy awarded better scores for higher intakes of vegetables, fruits, nuts, and soy (this item was included in the INMA cohort only), cereal fiber, the preference of white over red meat and that of polyunsaturated over saturated fat, adequate dietary intakes of folate, iron and calcium, and for reduced intake of *trans* fats [32, 33]. Regarding this index, for the USA cohort Rifas-Shiman et al. [32] reported a nonsignificant trend towards a decreased risk of SGA with increasing maternal diet quality, whereas in the Spanish cohort (from the Valencia region), Rodríguez-Bernal et al. [33] observed a higher frequency of FGR in offspring of women with the poorest diet compared to those with the best diet quality, as measured by the AHEI modified for pregnancy (Fig. 6.1). After adjusting for potential confounders, a significant increase in mean birth weight and birth length and a reduced risk of delivering a FGR infant—according to a customized model—in women with better diet quality compared with women who obtained the lowest scores on the AHEI was also observed. It has been suggested that the different results observed in these cohorts could be explained in part by differences in the outcomes used as indicators of FGR and by the different modifications made to the AHEI [33].

The MDS awards higher scores to individuals whose intake of vegetables, legumes, fruits and nuts, fish and seafood and dairy products, as well as the ratio of monounsaturated to saturated lipids, is above the median; and that of any type of meat below the median of the study population [36].

Chatzi et al., who studied pregnant women from Atlantic and Mediterranean areas in Spain, and from Greece, found that higher dietary quality (measured by the MDS) was protective against the risk of FGR in the Spanish Mediterranean populations only [34]. The authors state that this fact could be explained in part by the differences in the Mediterranean dietary pattern found across the different regions. The pattern of intake associated with a lower risk of FGR included a higher consumption of vegetables, legumes and monounsaturated fatty acids (MUFA) and a moderate consumption of fish and dairy products compared to the other populations studied. Stratified analysis by smoking revealed that higher Mediterranean diet adherence increased birth weight and length in smoking mothers, suggesting a high fetal exposure to several antioxidant compounds, and their property to counter the effect of oxidative stress damage of fetal tissues [34].

The AHEI and the MDS have similar components, some of which have been related to fetal growth and are included in dietary guidelines for pregnant women. Studies examining diet quality in women have found that higher scores of dietary indexes are related to increased intakes of nutrients such as fiber, folate, calcium, iron, and zinc [37, 38]. On the other hand, it is important to bear in mind that intakes of micronutrients considered important for fetal growth, such as folic acid, iron, calcium, and zinc, have been found to be inadequate in populations considered well nourished [39, 40]. Therefore, as proposed earlier [33], it is possible that an improvement in diet quality leads to higher intakes of such nutrients, benefitting fetal growth even in women who live in industrialized countries.

Evidence from intervention studies: There is limited evidence from intervention studies assessing the effect of diet quality on fetal growth. To the best of our knowledge, only one randomized clinical trial (RCT)—including 256 Finnish women randomized into two intervention groups of 85 women each, and one control group of 86 women—has examined the effect of diet quality on anthropometric outcomes at birth which might reflect fetal growth impairment [41]. Women in intervention group 1 were advised to increase their intake of fruit and vegetables, wholegrain bread and cereals and to consume leaner meat products. Women in intervention group 2 received the same advice as group 1 and were encouraged to consume a probiotic drink. Women in the control group were told to carry on with their usual diet. When dietary intake was assessed, women in the intervention groups reported consuming more fruit and vegetables, and vegetable fats, and less butter compared with the control group. The study did not show significant differences in birth weight, birth length, or head circumference between women in the intervention and the control groups [41]. One nonrandomized study, also among Finnish women ($n = 105$) in their second trimester of gestation [42], assessed the effect of a dietary intervention on fetal growth. Women in the intervention group were advised to follow a diet considered healthy (which included a regular meal pattern and the consumption of ≥5 portions of fruit and vegetables, high-fiber bread, and ≤1 portion of high-sugar snacks). A higher intake of vegetables, fruits, and high-fiber bread was reported by women in the intervention group compared to those in the control group. Women in the intervention group had higher rates of appropriate weight at birth and decreased rates of high birth weights (>4,000 g). No differences were found regarding LBW. It is noteworthy that women in the intervention group were younger, less educated, more often smokers and on average had higher pre-pregnancy weight and BMI than the women in the control group; besides, the intervention aimed to reduce the incidence of excessive gestational weight gain and gestational diabetes. Methodological issues such as lack of randomization [42], or lack of power due to small sample size [41, 42], hinder the interpretation of the results derived from these intervention studies.

Preterm Birth

Preterm birth, defined as a pregnancy ending at less than 37 completed weeks of gestation, is a primary risk factor for infant morbidity and mortality [43]. Rates of premature delivery have

Khoury. Cholesterol-lowering diet.

- Fruit and vegetables ≥ 6 per day.
- Red meat twice per week and the rest of the days fatty fish, poultry, legumes or vegetables as main dishes.
- Olive or rape-seed oil for dressing and cooking.
- Nuts.
- Skimmed or low-fat dairy.
- Coffee ≤ 2 cups a day.

Mikkelsen. Mediterranean-type diet.

- Fruit and vegetables ≥ 5 per day.
- Red meat ≤ 2 servings per week.
- Fish ≥ 2 servings per week.
- Olive or canola (rape seed) oil for dressing and cooking.
- Coffee ≤ 2 cups a day.

Haugen. Mediterranean-type diet.

- Fruit and vegetables ≥ 5 per day.
- Red meat ≤ 2 servings per week.
- Fish ≥ 2 servings per week.
- Olive or canola (rape seed) oil for dressing and cooking.
- Coffee ≤ 2 cups a day.

Fig. 6.2 Criteria for dietary patterns used in the studies assessing the relationship between overall diet during pregnancy and preterm birth. Criteria used to define overall maternal diet in the intervention study (Khoury et al. 2005) [45] and the two observational studies [46, 47] assessing the relationship between diet during pregnancy and preterm birth (unpublished)

increased in recent years in different settings. The worldwide incidence of preterm birth has been estimated about 9 %, with the highest rates in Africa and North America (10–12 %), followed by Asia and Latin America and the Caribbean regions. Europe has the lowest incidence at around 6 %. Other industrialized countries have rates which range from 5 to 9 % [43, 44].

The associated mortality, and short- and long-term morbidity as well as the economic consequences for healthcare systems make preterm delivery an important perinatal problem at the global level [43].

Spontaneous preterm births are the result of multiple causes, including infection or inflammation, vascular disease, and uterine overdistension. Among the maternal factors that have been associated with premature delivery are some socio-demographic characteristics, pregnancy history (i.e., previous preterm birth), characteristics of current pregnancy (i.e., short interpregnancy interval), and maternal nutritional status. Pre-pregnancy BMI and low concentrations or intakes of certain nutrients such as iron, folate, or zinc have been related to preterm birth [44].

Evidence of the Association Between Diet Quality and Preterm Birth

There is limited evidence on the effect of diet as a whole on preterm birth. The literature published has examined a diet resembling the Mediterranean dietary pattern (MD) as a measure of diet quality [45–47]. Available evidence comes from Nordic populations, and includes one RCT [45], whose purpose was to see the effect of a cholesterol-lowering diet on pregnancy outcomes, and two observational studies [46, 47].

In the RCT [45], women in the intervention group were advised to adhere to a diet resembling the Mediterranean dietary pattern. The criteria for this pattern are shown in Fig. 6.2. The control group was asked to follow their usual diet. Compared with the control group, the intervention group consumed significantly less energy,

Table 6.2 Studies assessing the association between diet quality during pregnancy and preterm birth

Population, country	Author (Year)	*N*	Main exposure	Main outcome	Time when diet was measured	Type of study	Main results
Preterm birth							
Healthy pregnant women, Norway	Khoury et al. (2005) [45]	290 (141, IG; 149, CG)	IG: diet resembling the MD CG: usual diet	Preterm birth	Wk 17 until delivery	Randomized control trial	Risk of preterm delivery greatly reduced in women following the MD
Women belonging to a nationwide birth cohort, Denmark	Mikkelsen (2008) [46]	35,530	Diet resembling the MD	Early preterm birth (<35 weeks) and preterm birth (<37 weeks)	Wk 21–24	Prospective study	Risk of early preterm birth but not preterm birth reduced in women who fulfilled the criteria defined for MD
Women belonging to a nationwide birth cohort, Norway	Haugen et al. (2008) [47]	26,563	Diet resembling the MD	Early preterm birth (<35 weeks) and late preterm birth (<37 weeks)	Wk 17–24	Prospective study	No effect of MD was found for early or late preterm birth

IG intervention group, *CG* control group, *Wk* week of pregnancy, *MD* mediterranean diet

saturated and total fat, and cholesterol. The authors report this was due to a lower intake of fatty milk and meat products. Mean dietary intake of fatty fish and derivative products was significantly higher in the intervention compared to the control group. As the intervention was aimed at reducing cholesterol intake, the consumption of other food groups is not reported, but results also show that the control group consumed more proteins, carbohydrates (but less sugar), vitamin C, tocopherol, magnesium, and vitamin D, which suggests higher intakes of their food sources (such as fruit, vegetables, or legumes). Regarding pregnancy outcomes, the incidence of preterm delivery was significantly reduced and mean gestational age at delivery was increased in the intervention group.

In the observational studies [46, 47], in which a Mediterranean dietary pattern was defined as shown in Fig. 6.2; results were contrasting, with a Mediterranean-like diet being associated with a reduced risk of preterm delivery in one of the studies [46] and an absence of association in the other [47].

Table 6.2 shows a summary of evidence on the relationship between quality of maternal diet and preterm birth.

Results from these studies are not conclusive, although evidence points towards the possible beneficial effect of a high-quality diet (resembling the Mediterranean diet) on duration of gestation. The only difference between the study finding no effect of a dietary pattern similar to the Mediterranean on preterm delivery [47] and those which did find a reduced risk [45, 46] is that the latter, apart from showing evidence of higher intakes of nutrients such as carbohydrates (but not sugar), vitamin C, α-tocopherol, magnesium, vitamin D, and lower intakes of saturated fat, also found a reduction in cholesterol levels. That is not the case in the former, in which although a higher intake of vitamin C, folate and fiber was observed in the "Mediterranean diet group," a higher intake of cholesterol and a lower intake of mono- and polyunsaturated fats was also found.

These findings might indicate that the composition of fat in the diet together with the action of other beneficial foods or nutrients composing the Mediterranean dietary pattern are likely to reduce the risk of preterm delivery. Khoury et al. proposed various mechanisms which could explain the association [45]: In brief, diets with a low content of cholesterol and high polyunsaturated fatty acids (PUFAS) may have an effect on cytokine

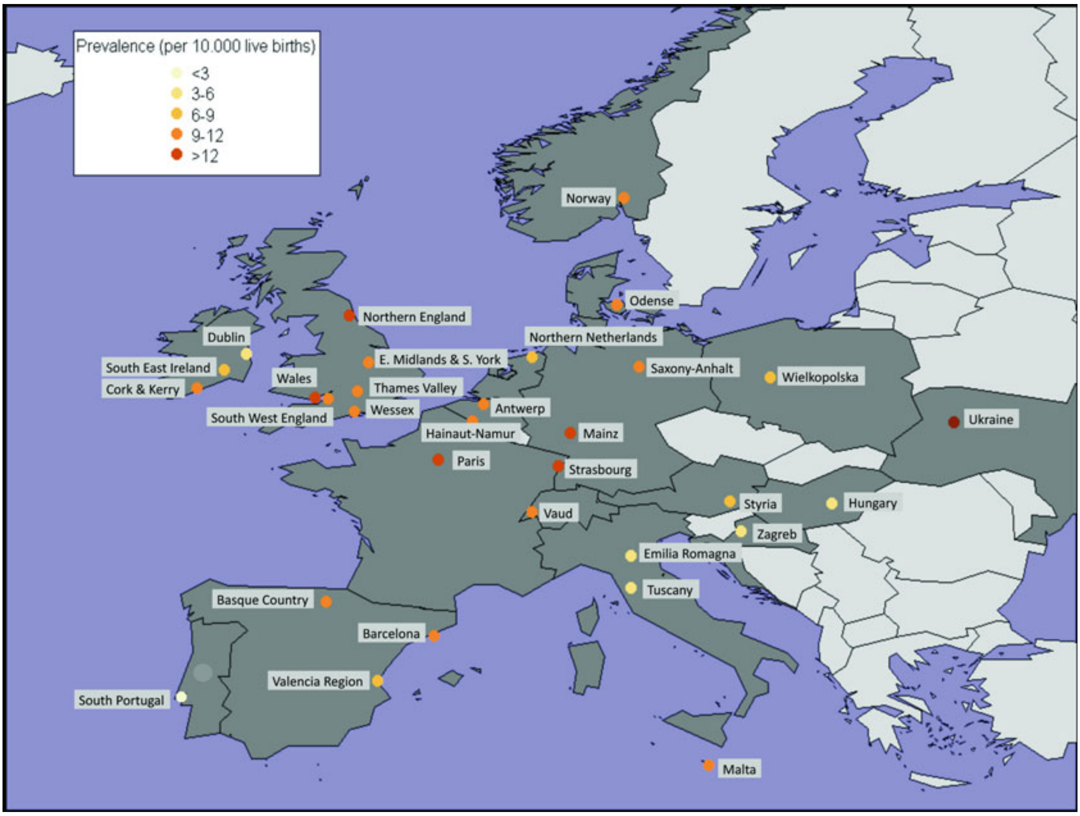

Fig. 6.3 Prevalence of neural tube defects (NTD) according to EUROCAT registries, 2005–2009. Prevalence of NTD per 10,000 live births between 2005 and 2009 in different places across Europe, which belong to the EUROCAT registry. *Circles* represent cities, regions, or countries which provide data on congenital anomalies to the registry. Source of information: Prevalence tables of EUROCAT. This map is production of the authors of this chapter and has not been published previously

levels, which together with stress hormone levels are thought to be involved in mechanisms of preterm delivery. Moreover, it has been proposed that long-chain n-3 PUFAS might have an antiarrythmic effect on the myometrium. Furthermore, authors stated that higher intakes of some micronutrients such as vitamin C and E could increase the length of gestation according to available evidence [45]; nevertheless, associations between supplementation with these nutrients and preterm delivery warrant further investigation [48].

Neural Tube Defects

NTD are a group of anomalies that might occur due to a failure in the closure of the neural tube, usually at day 28 of pregnancy. They are one of the most frequent types of birth defects; they might lead to spontaneous abortion or intrauterine death and are an important cause of infant mortality or lifelong disabilities [6, 49]. The frequency of such defects varies between geographical regions, with less than 1 per 10,000 in North America [50], 9.7 per 10,000 in Europe [51] and can be more than 10 per 10,000 in South America [52]. There is also variation within geographical regions, Fig. 6.3 shows a map representing differences in prevalence in the European region, as an example.

NTD are multifactorial disorders resulting from the combination of genetic and environmental factors; the wide range of differences in their occurrence, related also to socio-demographic factors, reflects the combined nature of their aethiology [49]. As of today no clinical or experimental study has provided unequivocal evidence for a definitive role for any of the genes (the genes involved in the Folic Acid, vitamin B12, and homocysteine metabolism during neural tube development) in the causation of NTD, suggesting that a multitude of genes, growth factors, and receptors interact in controlling neural tube development by yet unknown mechanisms [53].

Nutritional or metabolic factors with very strong evidence for their association with NTD are maternal diabetes and obesity—both related to glucose metabolism—possibly due to a sustained state of hyperglycemia and/or hyperinsulinemia [54].

Regarding the relationship between nutrient intakes and NTD, only folate intake has been consistently found to be strongly related to these congenital anomalies. Following the publication of a randomized trial showing that folate reduces the recurrence risk of NTD [4], recommendations about periconceptional folic acid supplementation have been established [55].

Evidence on Pre-conceptional Diet Quality and Neural Tube Defects

Recently, researchers have started to assess the effect of diet during the pre-conceptional period, as a composite measure, on NTD. Evidence is scarce and, due to the nature of the anomalies, comes from case–control studies.

Table 6.3 summarizes evidence on pre-conceptional diet quality and its effect on NTD.

One study in the USA [56] found that a periconceptional diet containing foods that might induce an elevation in serum glucose, and therefore a rise in demand for insulin and hyperinsulinemia increased the risk (5–6-fold) of NTD only in obese women (it is noteworthy that those women were nondiabetic). The aforementioned study was aimed at measuring the metabolic response to certain foods using the Dietary Glycemic Index (DGI), but not at measuring diet quality as such, although the index measures diet quality indirectly. Foods with a high DGI value (highly processed grains, monosaccharides, and disaccharides) were found to increase the risk of NTD compared to those with a low DGI (fruit, vegetables, unprocessed whole grains, beans, and some dairy products) [56]. Another case–control study found that a diet like the Mediterranean dietary pattern (composed of intakes of fruit, vegetables, vegetable oil, legumes and cereals, fish, and low intakes of potatoes and sweets) was associated with a reduction in the risk of Spina Bifida in the offspring [57]. Similarly, a study in the US found that a maternal diet of high quality as measured by two different indexes—The Diet Quality Index (DQI) and the MDS—during the pre-conceptional period was associated with a reduced risk of NTD [58]. Both indexes awarded higher scores to women consuming more adequate amounts of fruit, vegetables, and grains. Some other components differed between the two indexes; however, overall, they reflect the intake of some macro and micronutrients according to recommendations (i.e., fat, calcium).

In the light of the available evidence, diets with a low content of fruit, vegetables, legumes, and cereals—especially whole grains—and vegetable oils and high intakes of sweets—including monosaccharides and disaccharides—might increase the risk of NTD. Diverse mechanisms have been suggested for this association. One of them is thought to be the result of elevated serum glucose, either through an excess received by the fetus at a stage in which it is unable to regulate glucose levels due to the lack of pancreatic function [59], or through hypoglycemia following elevated glucose concentrations, which has been associated with teratogenity in vitro [56].

Moreover, experimental studies have shown that elevated glucose concentrations lead to embryonic depletion of inositol, which in turn is related to an abnormal closure of the neural tube [60]. Another explanation is that a high-quality diet contains enough natural folate to reach a similar protective effect as supplementation with folic acid [57]. It is noteworthy that in the study by Vujkovic et al., the MD was found to be significantly associated with increased serum folate, red blood cell folate, and serum vitamin B12, and with decreasing levels of total homocysteine [57], markers with a documented association with NTD [61].

The effect of folic acid supplements in the periconceptional period on reducing the incidence

Table 6.3 Studies assessing the association between maternal diet quality and neural tube defects

Population, country	Author (Year)	*N*	Main exposure	Main outcome	Time when diet was measured	Type of study	Main results
Neural tube defects (NTD)							
Mothers of children with NTD and controls, USA	Shaw et al. (2003) [56]	454 cases 452 controls	Dietary glycemic index (DGI)	Spina bifida and anencephaly	3 mo before pregnancy	Case control study	Higher DGI associated with higher risk of NTD in obese women only
Mothers of children with spina bífida and controls, Netherlands	Vujkovic et al. (2009) [57]	50 cases 81 controls	Dietary patterns: Mediterranean dietary pattern (MDP) identified	Spina bifida (SB)	3 mo before pregnancy	Case–control study	Higher risk of SB in women with weak use of MDP
Mothers of children with NTD and orofacial clefts and controls, USA	Carmichael et al. (2012) [58]	936 cases 6,147 controls	Maternal diet quality measured by Diet Quality Index (DQI) and Mediterranean diet score (MDS)	Spina bifida and anencephaly (AE)	3 mo before pregnancy	Case–control study	Higher diet quality measured by either DQI or MDS reduced the risk of NTD. The strongest association found with AE

mo month

of NTD has been proved by different RCT's. However, periconceptional supplementation is problematic and evidence shows that doses and timing of supplementation in pregnant women are inadequate if the aim is the prevention of NTD [62, 63]. As an alternative, food fortification with folic acid has been implemented in diverse settings in a compulsory manner [63]. There is consistent evidence on the reduction of NTD after the inclusion of compulsory fortification policies in North America [64, 65]. As of November 2009, compulsory food fortification with folic acid had not been implemented in any European country, although it is now widespread in North and South America and in several countries in the Middle East [66].

Nevertheless, this public health measure is controversial because of potential health risks and the issue of freedom of choice [63].

Food voluntarily fortified with folic acid (mainly breakfast cereals) is available in many European countries. In a study investigating the effects of consumption of folic acid-fortified bread compared with folic acid tablets, bread was found to be equally effective in increasing folate status as indicated by both increased red cell and serum folate concentrations [67]. However, it may be difficult for women to identify foods fortified with folic acid and to determine the amount in relation to their needs due to limitations/restrictions on food labeling [66]. In this context, promoting a high-quality diet in women of childbearing age would be a helpful complementary strategy aimed at reducing the risk of NTD; however, stronger evidence is needed to specify its effect on the incidence of this adverse outcome. Meanwhile, it is necessary to acknowledge that adequate periconceptional supplementation with folic acid is the most effective measure known so far.

Summary of Evidence on the Effect of Maternal Diet Quality on Pregnancy Outcomes

Overall, we consider that the body of evidence regarding the association between diet quality and pregnancy outcomes is methodologically too weak to inform prevention decisions but suggests the important role of a high-quality diet during pregnancy, including higher intakes of vegetables, fruits, legumes, and unsaturated fats, moderate consumption of fish and dairy products, and low intake of red meat and *trans* fats, in preventing undesired pregnancy outcomes such as FGR or preterm birth. Similarly, diets with a high content of fruit, vegetables, legumes and cereals—especially whole grains—and vegetable oils and low intakes of sweets—including monosaccharides and disaccharides—might decrease the risk of NTD.

Unfortunately, due to the nature of NTD, it is difficult to study their association with maternal diet quality using methodological designs different from the retrospective designs used so far, where recall bias could play an important role, weakening the evidence quality. Prospective studies would be a good option to overcome this limitation; however, very big sample sizes would be necessary.

The limitations of this overview of evidence stem primarily from the scarcity of randomized intervention studies, the heterogeneity of study designs, small sample sizes, and differing outcome parameters in the case of FGR (i.e., birth weight, SGA, and large for gestational age [LGA]).

Large multicentre studies could have an important role in contrasting the hypotheses related to diet quality and pregnancy outcomes outlined in this chapter. Population-based prospective cohort studies that started early in pregnancy or at birth should be encouraged to avoid the risk of recall bias to which retrospective approaches are prone. Multinational studies in this context are important for scientific reasons, including the ability to study the effects of a wider range of diets and the ability to test for the consistency of results in different environments [68]. However, because diet is still very specific to each country, multinational studies are also important for the application of scientific research to the development of food policy, which is bound to need strong arguments at a multinational level for implementation.

Conclusions

The body of epidemiological evidence supporting the recommendation for high-quality diet for the prevention of FGR, preterm birth and NTD is weak overall, but nonetheless suggestive of the beneficial effect of a high quality diet including higher intakes of vegetables, fruits, unrefined grains, legumes, and unsaturated fats.

Existing evidence in relation to pregnancy outcomes adds importance to the current public health messages recommending balanced healthy diets containing fruit and vegetables, starchy carbohydrates from cereals and legumes and unsaturated fats.

There is a need for well-designed randomized controlled trials as well as multinational studies assessing high-quality diets during pregnancy and their effect on pregnancy outcomes.

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References

1. Abu-Saad K, Fraser D. Maternal nutrition and birth outcomes. Epidemiol Rev. 2010;32:5–25.
2. Scholl TO, Johnson WG. Folic acid: influence on the outcome of pregnancy. Am J Clin Nutr. 2000;71: S1295–303.
3. Halldorsson TI, Meltzer H, Thorsdottir I, Knudsen V, Olsen S. Is high consumption of fatty fish during pregnancy a risk factor for fetal growth retardation? A study of 44,824 Danish pregnant women. Am J Epidemiol. 2007;166:687–96.
4. Medical Research Council Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. Lancet. 1991;338:131–7.
5. McIntire DD, Bloom S, Casey B, Leveno K. Birth weight in relation to morbidity and mortality among newborn infants. N Engl J Med. 1999;340:1234–8.
6. Eurocat. The status of health in the European Union. Congenital malformations. Eurocat: Ulster; 2009. p. 1–34.
7. Barker DJ, Eriksson J, Forsén T, Osmond C. Fetal origins of adult disease: strength of effects and biological basis. Int J Epidemiol. 2002;31:1235–9.
8. Yajnik CS. Early life origins of insulin resistance and type 2 diabetes in India and other Asian countries. J Nutr. 2004;134:205–10.
9. Demmelmair H, von Rosen J, Koletzko B. Long-term consequences of early nutrition. Early Hum Dev. 2006;82(8):567–74.
10. Wu G, Bazer F, Cudd T, Meininger C, Spencer T. Maternal nutrition and fetal development. J Nutr. 2004;134:2169–72.
11. Jacobs Jr DR, Steffen LM. Nutrients, foods, and dietary patterns as exposures in research: a framework for food synergy. Am J Clin Nutr. 2003;78:508S–13.
12. Waijers PMC, Feskens E, Ocké M. A critical review of predefined diet quality scores. Br J Nutr. 2007; 97:219–31.
13. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol. 2002; 13:3–9.
14. Mandruzzato G, Antsaklis A, Botet F, Chervenak FA, Figueras F, Grunebaum A, et al. Intrauterine restriction (IUGR). J Perinat Med. 2008;36(4):277–81.
15. Richards M, Hardy R, Kuh D, Wadsworth M. Birthweight, postnatal growth and cognitive function in a national UK birth cohort. Int J Epidemiol. 2002; 31:342–8.
16. Barker DJ. Fetal origins of coronary heart disease. Acta Paediatr Suppl. 1997;422:78–82.
17. Barker DJ. Adult consequences of fetal growth restriction. Clin Obstet Gynecol. 2006;49(2):270–83.
18. Imdad A, Yakoob MY, Siddiqui S, Bhutta ZA. Screening and triage of intrauterine growth restriction (IUGR) in general population and high risk pregnancies: a systematic review with a focus on reduction of IUGR related stillbirths. BMC Public Health. 2011;11 Suppl 3:S1.
19. Gardosia J, Figuerasc F, Clausson B, Francisa A. The customised growth potential: an international research tool to study the epidemiology of fetal growth. Paediatr Perinat Epidemiol. 2011;25:2–10.
20. Mamelle N, Boniol M, Rivière O, et al. Identification of newborns with fetal growth restriction (FGR) in weight and/or length based on constitutional growth potential. Eur J Pediatr. 2006;165:717–25.
21. Mikkelsen TB, Osler M, Orozova-Bekkevold I, et al. Association between fruit and vegetable consumption and birth weight: a prospective study among 43 585 Danish women. Scand J Public Health. 2006;34: 616–22.
22. Ramon R, Ballester F, Iniguez C, et al. Vegetable but not fruit intake during pregnancy is associated with newborn anthropometric measures. J Nutr. 2009;139:561–7.
23. Ramon R, Ballester F, Aguinagalde X, et al. Fish consumption during pregnancy, prenatal mercury exposure, and anthropometric measures at birth in a prospective mother–infant cohort study in Spain. Am J Clin Nutr. 2009;90:1047–55.
24. Mannion CA, Gray-Donald K, Koski KG. Association of low intake of milk and vitamin D during pregnancy with decreased birth weight. CMAJ. 2006;174: 1273–7.

25. Mardones F, García-Huidobro T, Ralph C, Farías M, Domínguez A, Rojas I, et al. Combined influence of preconception body mass index and gestational weight gain on fetal growth. Rev Méd Chile. 2011; 139(6):710–6.
26. Rodríguez-Bernal CL, Rebagliato M, Ballester F. Maternal nutrition and fetal growth: the role of iron status and intake during pregnancy. Nutr Diet Suppl. 2012;4:25–37.
27. Scholl TO, Chen X, Sims M, Stein T. Vitamin E: maternal concentrations are associated with fetal growth. Am J Clin Nutr. 2006;84:1442–8.
28. Knudsen VK, Orozova-Bekkevold IM, Mikkelsen TB, et al. Major dietary patterns in pregnancy and fetal growth. Eur J Clin Nutr. 2008;62:463–70.
29. Thompson JM, Wall C, Becroft DM, Robinson E, Wild CJ, Mitchell EA. Maternal dietary patterns in pregnancy and the association with small-for-gestational-age infants. Br J Nutr. 2010;103(11):1665–73.
30. Trichopoulos D, Lagiou P. Dietary patterns and mortality. Br J Nutr. 2001;85:133–4.
31. Chatfield C, Collins AJ. Introduction to multivariate analysis. London: Chapman & Hall; 1995.
32. Rifas-Shiman SL, Rich-Edwards J, Kleinman K, Oken E, Gillman M. Dietary quality during pregnancy varies by maternal characteristics in Project Viva: a US cohort. J Am Diet Assoc. 2009;109:1004–11.
33. Rodriguez-Bernal CL, Rebagliato M, Iniguez C, et al. Diet quality in early pregnancy and its effects on fetal growth outcomes: the Infancia y Medio Ambiente (Childhood and Environment) Mother and Child Cohort Study in Spain. Am J Clin Nutr. 2010;91: 1659–66.
34. Chatzi L, Mendez M, Garcia R, et al. Mediterranean diet adherence during pregnancy and fetal growth: INMA (Spain) and RHEA (Greece) mother-child cohort studies. Br J Nutr. 2012;107(1):135–45.
35. McCullough ML, Feskanich D, Stampfer M, et al. Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr. 2002;76:1261–71.
36. Trichopoulou A. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348(26):2599–608.
37. Bodnar LM, Siega-Riz A. A diet quality index for pregnancy detects variation in diet and differences by socio-demographic factors. Public Health Nutr. 2002;5:801–9.
38. Hann CS, Rock C, King I, Drewnowski A. Validation of the Healthy Eating Index with use of plasma biomarkers in a clinical sample of women. Am J Clin Nutr. 2001;74:479–86.
39. Mouratidou T, Ford F, Prountzou F, Fraser R. Dietary assessment of a population of pregnant women in Sheffield, UK. Br J Nutr. 2006;96:929–35.
40. Rodríguez-Bernal CL, Ramón R, Quiles J, et al. Dietary intake in pregnant women in a Spanish Mediterranean area: as good as it is supposed to be? Public Health Nutr. 2012;1–11. [Epub ahead of print].
41. Aaltonen J, Ojala T, Laitinen K, Piirainen TJ, Poussa TA, Isolauri E. Evidence of infant blood pressure programming by maternal nutrition during pregnancy: a prospective randomized controlled intervention study. J Pediatr. 2008;152(1):79–84, 84.e1–2.
42. Kinnunen TI, Pasanen M, Aittasalo M, et al. Preventing excessive weight gain during pregnancy—a controlled trial in primary health care. Eur J Clin Nutr. 2007;61(7):884–91.
43. Beck S, Wojdyla D, Say L, et al. The worldwide incidence of preterm birth: a systematic review of maternal mortality and morbidity. Bull World Health Organ. 2010;88(1):31–8.
44. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. Lancet. 2008;371(9606):75–84.
45. Khoury J, Haugen G, Tonstad S, Frøslie KF, Henriksen T. Effect of a cholesterol-lowering diet on maternal, cord, and neonatal lipids, and pregnancy outcome: a randomized clinical trial. Am J Obstet Gynecol. 2005;193:1292–301.
46. Mikkelsen TB, Osterdal ML, Knudsen VK, et al. Association between a Mediterranean-type diet and risk of preterm birth among Danish women: a prospective cohort study. Acta Obstet Gynecol Scand. 2008;87(3):325–30.
47. Haugen M, Meltzer HM, Brantsaeter AL, et al. Mediterranean-type diet and risk of preterm birth among women in the Norwegian Mother and Child Cohort Study (MoBa): a prospective cohort study. Acta Obstet Gynecol Scand. 2008;87(3):319–24.
48. Hauth JC, Clifton RG, Roberts JM, et al. Vitamin C and E supplementation to prevent spontaneous preterm birth. Obstet Gynecol. 2010;116(3):653–8.
49. Detrait ER, George TM, Etchevers HC, Gilbert JR, Vekemans M, Speer MC. Human neural tube defects: developmental biology, epidemiology, and genetics. Neurotoxicol Teratol. 2005;27(3):515–24.
50. Tolmie J. Neural tube defects and other congenital malformations of the central nervous system. In: Rimoin DL, Connor JM, Pyeritz RE, editors. Emery and Rimoin's principles and practice of medical genetics. New York: Churchill Livingston; 1996. p. 2152.
51. EUROCAT. Website database (2005–2009) [uploaded 06/12/2011]. European surveillance of congenital anomalies [web site]. Ulster [accessed 29 Jan 2012]. Prevalence tables. Disponible en:http://www.eurocat-network.eu.
52. Pachajoa H, Ariza Y, Isaza C, Méndez F. [Major birth defects in a third-level hospital in Cali, Colombia, 2004–2008]. Rev Salud Publica (Bogotá). 2011; 13(1):152–62 (Spanish).
53. Padmanabhan R. Etiology, pathogenesis and prevention of neural tube defects. Congenit Anom (Kyoto). 2006;46(2):55–67.
54. Yazdy MM, Liu S, Mitchell AA, Werler MM. Maternal dietary glycemic intake and the risk of neural tube defects. Am J Epidemiol. 2010;171(4):407–14.
55. Eurocat. A review of environmental risk factors for congenital anomalies. Ulters: Eurocat; 2004.
56. Shaw GM, Quach T, Nelson V, et al. Neural tube defects associated with maternal periconceptional

dietary intake of simple sugars and glycemic index. Am J Clin Nutr. 2003;78(5):972–8.

57. Vujkovic M, Steegers EA, Looman CW, Ocké MC, van der Spek PJ, Steegers-Theunissen RP. The maternal Mediterranean dietary pattern is associated with a reduced risk of spina bifida in the offspring. BJOG. 2009;116(3):408–15.
58. Carmichael SL, Yang W, Feldkamp ML, et al. Reduced risks of neural tube defects and orofacial clefts with higher diet quality. Arch Pediatr Adolesc Med. 2012;166(2):121–6.
59. Jovanovic-Peterson L, Peterson CM. Abnormal metabolism and the risk of birth defects with emphasis on diabetes. Ann N Y Acad Sci. 1993;678:228–43.
60. Wentzel P, Wentzel CR, Gareskog MB, Eriksson UJ. Induction of embryonic dysmorphogenesis by high glucose concentration, disturbed inositol metabolism, and inhibited protein kinase C activity. Teratology. 2001;63:193–201.
61. van der Put NM, van Straaten HW, Trijbels FJ, Blom HJ. Folate, homocysteine and neural tube defects: an overview. Exp Biol Med. 2001;226:243–70.
62. Sanfélix-Gimeno G, Ferreros I, Librero J, Peiró S. [Characterization of folate supplementation in pregnancy, based on a combination of health information systems]. Gac Sanit. 2012;26(6):512–18.
63. Eichholzer M, Tönz O, Zimmermann R. Folic acid: a public-health challenge. Lancet. 2006;367:1352–61.
64. Ray JG, Meier C, Vermeulen MJ, Boss S, Wyatt PR, Cole DE. Association of neural tube defects and folic acid food fortification in Canada. Lancet. 2002; 360(9350):2047–8.
65. Dietrich M, Brown CJ, Block G. The effect of folate fortification of cereal-grain products on blood folate status, dietary folate intake, and dietary folate sources among adult non-supplement users in the United States. J Am Coll Nutr. 2005;24(4): 266–74.
66. EUROCAT. Prevention of neural tube defects by periconceptional folic acid supplementation in Europe. Ulster: EUROCAT; 2009.
67. Armstrong NC, Pentieva K, McPartlin J, Strain JJ. Comparison of the homocysteine-lowering effect of folic acid-fortified bread versus folic acid tablets. In: Homocysteine metabolism 3rd international conference, Naples; 2001. p. 162.
68. Tricon S, Willers S, Smit HA, Burney PG, Devereux G, Frew AJ, et al. Nutrition and allergic disease. Clin Exp Allergy Rev. 2006;6(5):117–88.

7 Diet Quality in Pregnancy: A Focus on Requirements and the Protective Effects of the Mediterranean Diet

Miguel Mariscal-Arcas, Celia Monteagudo, and Fátima Olea-Serrano

Key Points

- Major anabolic activity increases nutritional requirements during the preconception period, and nutritional research has demonstrated that a balanced diet has a positive effect on the course of pregnancy, preventing premature births, and even on the development of the newborn, reducing the risk of weight or size loss and lower resistance to infections, among other problems.
- After reviewing the nutritional requirements of a healthy woman during the pregnancy, it is appropriate to discuss healthy diet patterns. Recent observational studies suggested that a higher intake of antioxidant vitamins, tocopherols, zinc and selenium during pregnancy and childhood reduces the likelihood of childhood asthma, wheezing and eczema. The generalized use of olive oil may explain the relationship detected between the daily intake of monounsaturated fatty acids (MUFAs) and vitamin E in maternal milk during the first month after delivery in a Mediterranean population.
- There is considerable scientific evidence on the beneficial effects of PUFAs of either vegetable (seed oil) or animal (fish) origin during pregnancy. It was reported that the consumption of two portions/week of salmon achieves the minimum recommended intake of EPA and docosahexaenoic acid (DHA) and increases their levels in the foetus.
- Diet Quality Indexes (DQIs) consider the foods and/or nutrients usually named in dietary guidelines or nutritional objectives. Some indexes assess the variability of the diet, which is normally measured by considering

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the amount of different foods or food groups consumed over a given period of time. Other systems of diet quality assessment are based on comparison with a specific diet pattern that has proven to be healthy, such as the Mediterranean Diet or the Dietary Guidelines for Americans. We reviewed published studies on the applications in populations of pregnant women of the adapted DQI the DQI-P, of the Healthy Eating Index (HEI) and adaptation (HEI-P) and of the Mediterranean Diet Score (MDS) and adaptation (MDS-P).
- Dietary recommendations and DQIs for healthy pregnant women published in the scientific literature to date are all based on international healthy diet models, and there is little difference among them. Indexes are needed that take account of the sensitivity of this population to environmental pollutants contained in foods, such as heavy metals, persistent OCs, synthetic hormone disruptors (e.g., phthalates and bisphenols) and anti-oxidizing agents.

Keywords

Mediterranean diet • Pregnant diet • Diet quality index pregnancy • Protective effects • Pregnancy

Abbreviations

ALA	α-Linoleic acid
BMI	Body mass index
DHA	Docosahexaenoic acid
DRI	Dietary reference intakes
HEI	Healthy Eating Index
LC-PUFAs	Long-chain polyunsaturated fatty acids
MD	Mediterranean diet
MDS	Mediterranean diet score
MUFA	Monounsaturated fatty acid
PTWI	Provisional tolerable weekly intake
SFA	Saturated fatty acid

Introduction

Pregnancy is one of the periods of highest nutritional vulnerability a woman's life. Major anabolic activity increases nutritional requirements during the preconception period, and nutritional research has demonstrated that a balanced diet has a positive effect on the course of pregnancy, preventing premature births, and even on the development of the newborn, reducing the risk of weight or size loss and lower resistance to infections, among other problems [1].

Nutritional Needs During Pregnancy

Some of the nutritional requirements established for the healthy adult are modified under special situations, including pregnancy (Table 7.1).

1. *Energy requirements*. The energy intake during pregnancy should guarantee the growth of the foetus, placenta and associated maternal tissue to permit a full-term, healthy newborn of adequate size. It should maintain the mother at her appropriate weight and provide sufficient energy reserves for the lactation period. The average weight gain for a healthy, well-nourished female should be around 12 kg in order to increase the likelihood of a full-term newborn with an average weight of 3.3 kg and to reduce the risk of foetal and maternal complications. An inadequate weight of the mother is related to low weight of the newborn and delay in intrauterine growth, and a low body

Table 7.1 Recommended intakes of energy and nutrients [5, 8–11] during pregnancy

Nutrients	Recommendation		
	First trimester	Second trimester	Third trimester
Energy	–	1.5 MJ/day (360 kcal/day)	2.0 MJ/day (475 kcal/day)
Proteins	1.1 g/kg/day		
Lipids	<30–35 % total energy		
Carbohydrates	>50–55 % total energy		
Iron	27 mg/day		
Calcium	1,300[a] to 1,000 mg/day		
Iodine	220 mg/day		
Folic acid	600 mg/day		
Vitamin D	5 mg/day		
Vitamin A	750[a] to 770 mg/day		

[a]Recommended intake for pregnant women under 18 years old

mass index (BMI) before pregnancy is associated with premature delivery [2].

It has been estimated, by factorial calculations based on a theoretical model, that there is an accumulated deposit of 925 g protein and of 825 g fat over the entire pregnancy period, with 90 % energy use efficiency and an accumulated increase of 150 MJ in the basal metabolic rate. Hence, the total energy expenditure of pregnancy is 320.2 MJ (76,530 kcal), although it is not distributed equally over the gestation period, and the accumulation of protein and fat deposits largely occurs during the second and third trimester. The increase in energy is estimated to be 0.35 MJ/day in the first trimester, 1.2 MJ/day in the second trimester and 2.0 MJ/day in the third trimester. It is therefore recommended to increase the calorie intake by 1.5 MJ/day (360 kcal/day) in the second trimester and by 2.0 MJ/day (475 kcal/day) in the third [2].

2. *Fat*. The recommended fat intake during pregnancy is the same as for the general population, i.e., no more than 30–35 % of total energy intake [3]. However, the intake of long-chain polyunsaturated fatty acids (LC-PUFAs) is of interest, especially docosahexaenoic acid (DHA), due to its beneficial effects on cognitive and ocular development, sleep patterns, motor function, immune system, body composition and sensitivity to insulin in the newborn. The intake of at least 200 mg DHA/day is recommended, and direct DHA intake is more effective to deposit DHA in the foetal brain in comparison to the intake of precursors, such as α-linolenic acid. An increase in the supply of α-linolenic and linoleic acid is not recommended during pregnancy [4].
3. *Proteins*. Although the need for additional protein for the synthesis of maternal and foetal tissues is acknowledged, the required magnitude of this increase is uncertain. The efficiency of protein utilization in pregnant women is approximately 70 %, the same as observed in breastfed infants. Requirements vary over the gestation period and are higher during the second and third trimester. The protein recommendation is 1.1 g/kg/day, i.e., 0.3 g/kg/day above the daily recommended intake for healthy adults [5]. A balance between energy and protein supplies has a positive effect on the mother and also on the foetus and newborn, and it is associated with optimal weight gain in both and a significant reduction in the risk of a small-for-gestational-age baby [6]. Protein supplements and low protein:energy ratios do not confer a benefit in pregnant women who are overweight, and they may even be detrimental for the newborn.
4. *Carbohydrates*. The intake of carbohydrates should be >50–55 % of the total energy intake, and a minimum intake of 175 g/day is recommended [5]. Consideration should be given to the quality and distribution of carbohydrate

intake over the day in order to avoid episodes of hyper- or hypo-glycemia. Slow-release sugars (fruit, cereals and pulses) and fast-release sugars (sugar, honey, etc.) are recommended, mainly at breakfast and dinner. It is also recommended to consume both types at all meals.

In general, an appropriate supply of essential micronutrients is provided by a balanced diet, which is generally accessible in developed countries. However, some population groups are more vulnerable, including pregnant women, who may have an inadequate vitamin and mineral intake, especially of iron, iodine, folic acid, vitamin D and/or vitamin B_{12} [7].

5. Minerals

Iron. Iron needs double during pregnancy for iron storage in the newborn (270 mg), for placenta formation, and for the increase in red blood cells required by the greater blood volume and the blood loss during delivery. Iron requirements range from 18 mg/day for adult women to 27 mg/day for pregnant women [8–11]. Iron deficiency and ferropenic anaemia during pregnancy are risk factors for premature delivery and low gestational weight. It has been hypothesized that it may have a negative effect on the intelligence and behaviour development of children [12].

Iron supplementation is common during pregnancy. However, excessive iron intake may be toxic through its capacity to generate reactive oxygen species and induce cell damage. The most appropriate approach is to apply selective prophylaxis if low ferritin levels are detected during the first stage of pregnancy [7].

Dietary sources of iron during pregnancy include meat, viscera, fish and eggs (hemo iron) and pulses and cereals (non-hemo iron).

Calcium. Epidemiologic studies have demonstrated an inverse relationship between calcium intake and hypertension development during pregnancy. However, the effects of calcium supplementation have only been observed in populations with an inadequate calcium intake [6]. No scientific evidence has been published that relates calcium supplementation to foetal growth. The recommended calcium intake during pregnancy is 1,000 mg/day, reaching 1,300 mg/day when the woman is under 18 years old [8–11].

The consumption of fortified foods and/or supplements is an alternative way to enhance the intake of calcium, whose main sources are milk and milk derivatives such as cheese and yoghurt.

Iodine. Iodine requirements are increased during pregnancy because of (1) an increase in the maternal production of thyroxine to maintain maternal euthyroidism and thyroid hormone transfer to the foetus, (2) iodine transfer to the foetus and (3) an increase in the renal excretion of iodine [13]. The recommended iodine intake during pregnancy is 220 μg/day [8–11].

A low iodine intake during pregnancy is associated with a higher incidence of miscarriage, small-for-gestational-age foetus, mental retardation and hearing loss [13]. Thyroxine intervenes in the development of the nervous system and an adequate iodine intake is especially important during the first trimester.

6. Vitamins

Folic acid. There is a considerable increase in the requirement for folic acid during pregnancy due to the creation of new tissue and the growth and development of the foetus. There is scientific evidence that folic acid is vitally important during the preconception period (8–12 weeks before conception) and during pregnancy to protect the foetus against neural tube defects [14]. As a preventive measure dietary reference intakes (DRI) of folic acid was recently increased to 400 μg/day for women of childbearing age and to 600 μg/day for pregnant females [8–11]. The incidence of neural tube defects is correlated with the intake of folic acid and vitamin B_{12}, which are both involved in the metabolism of homocysteine [7]. Folic acid supplementation is appropriate, given the impossibility of achieving this intake by diet alone. Folic acid is present in green-leaf vegetables, pulses, fruit and citric juices, liver and fortified foods such as bread, cereals, flour, corn flour, pasta, rice, etc.

Vitamin D. Observational studies suggested that maternal vitamin D levels are related to the bone mass of newborns, with low levels predisposing them to neonatal hypocalcaemia and rickets. Various authors have also reported that low vitamin D concentrations during prenatal life increase the risk of developing multiple sclerosis, cancer, insulin-dependent diabetes mellitus, schizophrenia and respiratory problems in subsequent life stages [15]. The recommended vitamin D intake for pregnant women is the same as for nonpregnant women (5 μg/day) [8–11].

Vitamin A. Vitamin A contributes to the maintenance and development of tissues and has essential functions in sight, bone growth, and the immune and nervous systems. Foetal vitamin A levels depend on the vitamin A status of the mother; therefore, an adequate maternal supply is essential to ensure normal foetal growth and development [16], and recommendations range from 750 to 770 μg/day of vitamin A [8–11]. Due to the possible teratogenic effect associated with this vitamin, intakes >3 mg (10,000 IU) preformed vitamin A/day are not recommended in women that are or may be pregnant, following the precaution principle [16]. Consequently, during the first weeks of pregnancy, women should avoid the intake of foods that may contain high doses of this vitamin, such as liver and/or supplements [7].

Mediterranean Diet and Pregnancy

After reviewing the nutritional requirements of a healthy woman during the pregnancy, it is appropriate to discuss healthy diet patterns, including the Mediterranean diet (MD), and consider their possible benefits diet during pregnancy. The characteristics of the MD [17] are exhibited in Table 7.2.

Table 7.2 Characteristics of the MD [17]

Consumption of olive oil as main lipid source, favouring a high ratio of monounsaturated to saturated fatty acids (MUFA/SFA)
High consumption of fruit, dried fruit and vegetables; high consumption of legumes
High consumption of cereals and derivatives and potatoes, among other sources of carbohydrates
Moderate-to-high consumption of fish; moderate consumption of milk and dairy products
Low consumption of meat and meat products
Moderate consumption of alcohol (mainly wine), usually with meals

This eating pattern is characterized by an elevated content of total fats (30–40 % of total energy, according to the geographic region) but low content of saturated fats (≤7–8 % of total energy) The high consumption of vegetable products and moderate consumption of animal products results in an elevated intake of fibre, vitamins, minerals and phytochemicals. However, this dietary pattern does not meet iron recommendations during pregnancy, and the deficiency must therefore be covered by nutritional supplements.

Recent studies have demonstrated the protective role of MD for the health of the mother and child, who suffer from fewer asthmatic and atopic symptoms due to the antioxidants supplied by components of this diet, such as olive oil, fruit, vegetables, fish and legumes [18]. It has been observed that females with low adherence to the MD are threefold more likely to have a child with *spina bifida*. This protective effect is associated with the MD pattern in its totality and not solely with the folic acid that it supplies. Moreover, given the beneficial effect of the MD on fertility, the need has been proposed for nutritional education programmes to promote this dietary pattern in women wishing to become pregnant [19].

Importantly, the MD does not merely represent a list of foods, because its beneficial effects on health are also influenced by other relevant attributes of Mediterranean society, culture and lifestyle, including the use of fresh seasonal and minimally processed foods, unhurried cooking, consumption of meals in company, with no time pressure, sharing dishes and conversation, a moderate level of physical activity and even the habit of "siesta" [20].

Foods that are sources of calcium, iron and folic acid should be included in the diet of pregnant women, which should also provide B complex vitamins, vitamin A, chrome and PUFAs. These nutrients are supplied by the intake of whole or semi-skimmed milk and milk derivatives (e.g., yogurt and fresh cheese), lean meat, eggs, white and blue fish, dried fruits, fruit and fruit juice, vegetables, pulses and wholemeal cereals, which are all characteristic of the MD and other healthy diets.

Wine is consumed as part of the traditional MD pattern and is a source of anti-oxidizing agents. Nevertheless, it is recommended that pregnant women exclude all alcoholic drinks from their diet, including wine, due to the risk of exposing the foetus and the possibility of foetal alcohol syndrome [21, 22]. On the other hand, there is no published evidence to associate the usual wine intake in the traditional MD with any alteration in the foetus. A recent article reported the absence of Italian or Spanish statistics on ethanol consumption during pregnancy and the lack of data on the prevalence of foetal alcohol syndrome or any foetal alcohol spectrum disorders [23]. The authors considered that neonatologists and paediatricians should be adequately informed in order to accurately diagnose ethanol use during pregnancy and inform pregnant women appropriately about the consequences for the newborn.

Main Lipid Sources in the Mediterranean Diet

The health benefits of the MD have in part been attributed to its low saturated fatty acid (SFA) content. However, numerous studies have also demonstrated the crucial role played by the antioxidant nutrients and non-nutrients in the foods it contains, including vegetables, virgin olive oil, fish and wine [20, 24]. Nevertheless, the overall health impact of the MD is not produced by one or various individual components but by synergism among them all.

Although the MD concept was initially interpreted as a diet low in total fats, there is now scientific evidence that it is the quality and not quantity of the fats that protects against disease. In fact, the nutritional policies of many countries appear to have changed, and they now promote the consumption of high quality fats from olive oil and blue fish to the detriment of *trans* fats and saturated fats. An additional advantage of the MD in this regard is that the benefits of olive oil are enhanced by the practice of consuming it along with large amounts of vegetables/vegetable products, fibre and antioxidant nutrients. In olive oil, obtained from the fruit of *Olea europaea L.*, oleic acid comprises 60–85 % of total triglyceride fatty acids, whereas linoleic acid makes up only 3–21 %. Oleic acid constitutes around 29 % of the daily caloric intake in many Mediterranean countries. Lipoproteins rich in monounsaturated fatty acid (MUFA)-rich lipoproteins, due to the long-term consumption of olive oil, have proven less susceptible to oxidation in comparison to PUFA-rich particles [25].

Phenolic compounds and waxes represent only a small fraction of olive oil (1–2 %), but they exert important biological activities [26]. Virgin olive oil is a source of valuable antioxidant substances such as polyphenols and vitamin E. Salvini et al. [27] studied the reduction of DNA oxidative damage and recommended the consumption of virgin olive oil, characterized by higher concentrations of phenolic compounds and hence of antioxidant substances. The antioxidant potential of phenolic compounds is higher in olive oil than in other vegetable oils. In addition, the phenols of olive oil may also possess anti-inflammatory and antithrombotic properties. Recent observational studies suggested that a higher intake of antioxidant vitamins, tocopherols, zinc and selenium during pregnancy and childhood reduces the likelihood of childhood asthma, wheezing and eczema [28].

A change in maternal dietary fat intake has a rapid effect on the fatty acids in breast milk, and the vitamin E content of mothers' milk during the first month of breastfeeding was significantly correlated with their total fat intake, although it was not correlated with their intake of PUFAs, which are accompanied by vitamin E in many foods, in the study of a population following the MD. The generalized use of olive oil may explain the relationship detected between the daily intake of MUFAs and vitamin E in maternal milk during the first month after delivery in a Mediterranean population [29].

Oxidative stress has been implicated in many diseases that arise during pregnancy, delivery and puerperium, and the possible preventive or therapeutic effects of antioxidants are therefore of great interest. Systematic reviews on the effects of supplementary antioxidant vitamins during pregnancy have not been conclusive about the benefits of exceeding recommended intakes. However, an adequate supply of antioxidants during pregnancy was associated with a reduction in the risk of preeclampsia and the delivery of newborns small for gestational age, although supplementation with high doses of vitamin C and E was not associated with a reduced incidence of preeclampsia [4].

The MD appears to offer an ideal dietary supply of antioxidant molecules, attributable to the large amount of polyphenols and other antioxidant components, both nutrients (e.g., vitamins A and C from vegetables) and phytochemicals (e.g., fruit and vegetables).

Nutritional Sources of n-3 Fatty Acids

There is considerable scientific evidence on the beneficial effects of PUFAs of either vegetable (seed oil) or animal (fish) origin during pregnancy. Thus, the intake of linoleic acid (omega [n-] 6), α-linolenic acid (n-3) and essential long-chain acids is important for neonatal growth and development and reduces the incidence of premature births. The supply of these acids via maternal milk has been positively associated with increased weight, height and head circumference of newborns, whereas oleic acid intake was only associated with increased head circumference. Given the benefits of n-3 long-chain fatty acids for sight and their relationship with a lower incidence of premature delivery, pregnant women should be advised to increase their intake of foods rich in these fatty acids. However, the intake of α-linoleic acid (ALA) and DHA is often low among pregnant and breastfeeding women, who should increase their intake of ALA by consuming vegetable oil. Improvements in infant growth and development become evident before the child is 2 years old. No significant effect was observed after supplementation with DHA and ALA [30].

Recent studies suggested that the intake of seafood and n-3 fatty acids favours foetal growth and infant development. A high n-3 fatty acid/PUFA ratio before pregnancy may sustain foetal growth in overweight women. Blue fish contain long-chain fatty acids that are considered important for the growth, development, and health of foetus and newborn. It was reported that the consumption of two portions/week of salmon achieves the minimum recommended intake of EPA and DHA and increases their levels in the foetus [31]. Habitual fish consumption is one of the characteristics of the population of the Mediterranean region, especially in coastal areas. Numerous studies have addressed the association between fish/fish oil consumption and the protection and development of the newborn. Currently, the intake of PUFA n-3 in the Mediterranean area from the consumption of fish and derivatives is appreciable but not high. Consequently, it is necessary to develop strategies to redefine and promote the consumption of fish and seafood in the context of the Mediterranean pattern of healthy diet. The ethnic group and country of origin are both related to DHA + EPA intake. A wide study in the USA found a significantly higher monthly intake of DHA + EPA by people of African origin than by Hispanics or Caucasians. Among cultural reasons for this difference would be the preservation of habits from their historic origins in coastal areas despite living in regions with lesser fish availability. An investigation for the European Commission into recommendations on fat intake during pregnancy and breastfeeding concluded that women of childbearing age should consume 1–2 portions of fish a week, including fish fat [4].

There may be a causal relationship between the intake of n-6 PUFA and allergic diseases that involves biologically plausible mechanisms in which eicosanoid mediators of n-6 PUFA arachidonic acid participate. The n-3 PUFA chain is considered to protect against atopic sensitization and against the clinical manifestations of atopy. Epidemiologic studies on the effects of fish intake during infancy and childhood on atopy risk in breastfed infants or children have not been conclusive, although most of them found fish to have a protective effect. The supply of fish oil during pregnancy may diminish sensitization to the most common food allergens and reduce the prevalence and severity of atopic dermatitis during the first year of life. This effect may persist until adolescence, decreasing the prevalence and/or severity of eczema, hay-fever and asthma [32].

Fish and Pollution

Although the consumption of fish is beneficial as a source of LC-PUFAs, there is increasing evidence that it may be an important route for human pollution. This is because heavy metals, such as mercury and persistent organochlorines (OCs), can be stored in their muscle and adipose tissue. A study in the USA confirmed that a large number of pregnant women do not consume enough DHA, an essential nutrient contained in fish, because of a reduction in fish intake to avoid exposure to substances toxic for mother and even more so for the foetus. Uncertainty has grown about the appropriate consumption of fish, but a wide study concluded that women would be willing to consume more fish if it were recommended by their obstetrician, as long as data were made available to them on the types of fish that are safe [33].

A recent study in Norway analyzed ten chemical pollutants, including mercury (Hg), and found fish consumption to be a robust positive predictor for Hg; they also reported an inverse association between parity and blood Hg levels. The consumption of certain foods, such as seafood, may affect the blood concentrations of Hg and arsenic (As), among other elements, although the authors concluded that the levels of toxic elements (As, Hg, cadmium [Cd] and lead [Pb]) observed were relatively low and not clinically relevant [34].

The FAO/WHO [35] established 1.6 μg/kg body weight as the provisional tolerable weekly intake (PTWI) of methyl mercury (MeHg). The most vulnerable groups are women of childbearing age, pregnant women, breastfeeding women and children. In foetuses, neurotoxicity has been related to chronic exposure to low Hg concentrations. Because it is impossible to minimize the risk solely by establishing stricter maximum Hg contents for fish, the European Commission [36] urged member States to formulate recommendations to protect the health of consumers.

Among all of the chemical species of Hg present in food, the most toxic is the organic component MeHg, which is mainly found in fish and seafood, in which it can represent over 90 % of the total Hg. The highest concentrations of MeHg appear in large fish at the highest level of the food chain in both freshwater and seawater species.

Based on average fish consumption values, the Spanish Food Safety Agency (Spanish initials, AESAN) estimated that the average MeHg intake by children was 98.7 % of the Provisional Tolerable Daily Intake (PTDI) [35] and that the intake by adults was 80.7 % of the PTDI [37]. Larger fish (swordfish, tuna and bonito) are known to accumulate greater Hg contents, and an intake of less than 100 g/week of large predator fish meat was recommended by the European Commission [36] for populations at risk. Risk management options considered by the WHO include promoting the consumption of non-predator fish or smaller species and the reduction of intake by heavy fish consumers [38].

Foods are considered a constant source of exposure, despite compliance with maximum permitted residue levels. The diet of the breastfeeding mother affects the quality and quantity of the milk that she feeds her child. Milk can be a vehicle for toxins that can harm the health of the breastfeeding child, including drugs and their metabolites, viruses, nicotine, caffeine, alcohol and OC molecules (e.g., PCBs, DDT, HCB, HCH and dioxins). A statistically significant relationship was found between the consumption of fatty foods and some OC molecules (*p,p′* DDD, ppDDT and methoxychlor) in the milk of breastfeeding mothers in the south of Spain [39].

Our group studied the relationship between the nutritional habits of women in Southeast Spain and their serum concentrations of OCs. The pregnant group showed significantly higher serum concentrations of all OCs studied with the exception of DDE. The mean serum concentration of OCs (*p*,*p*-DDT, *o*,*p*-DDT, aldrin and p-p´DDE) was significantly associated with the intake of milk/yoghurt, red meat, eggs and poultry [40]. We investigated the presence of nine OC residues in the umbilical cord blood of newborns in Southern Spain and analyzed the relationship of this exposure with maternal and pregnancy variables, including maternal adherence to the MD. OCs were detected in 95 % of umbilical cord blood samples from 318 mothers, who had a mean degree of adherence to the MD of 56.77 (SD: 16.35) (range, 0–100). The MD prioritizes consumption of vegetable and fruit over that of meat and dairy products, and OCs are generally lipophilic molecules that accumulate in foods of animal origin. We found a significant association between the consumption of meat, fish and dairy products and DDE in umbilical cord serum, between the intake of dairy products and lindane, between vegetable and lindane and between fruit intake and endosulphan I. No significant association was found between adherence to the MD and serum OC levels, but closer adherence to the MD may offer greater protection against OC exposure through a lesser intake of meat and dairy products [41].

Diet Quality Indexes: Considerations for Pregnant Women

DQIs consider the foods and/or nutrients usually named in dietary guidelines or nutritional objectives, including food groups such as vegetables and fruit, cereals and meat products, among others. The most frequently assessed nutrients in these indexes are total fat intake, SFA intake and/or MUFA:SFA ratio, and many also assess cholesterol and/or alcohol intakes. Some indexes assess the variability of the diet, which is normally measured by considering the amount of different foods or food groups consumed over a given period of time. Other systems of diet quality assessment are based on comparison with a specific diet pattern that has proven to be healthy, such as the MD [20] or the Dietary Guidelines for Americans [42].

Table 7.3 Diet Quality Indexes for pregnancy

Quality index	Items	Score range
DQI-P	Cereals, vegetables, fruit, folic acid, iron, calcium, percentage of calories from fats and food model score	0–80
HEI-P	Vegetables, fruit, white meat:red meat ratio, *trans* fats, PUFA:SFA ratio, folic acid, calcium and iron	0–90
MDS-P	High consumption of vegetables, fruit, nuts, pulses, cereals and fish, a high MUFA:SFA ratio, low consumption of meat and dairy products and adequate intake of iron, calcium and folic acid	0–11

A diet assessment index for pregnant women should consider the supply of the specific nutrients discussed above in this chapter. In summary, pregnancy implies an extra supply of (1) energy (250–300 kcal/day during second half of pregnancy); (2) calcium; (3) folic acid; (4) iron and vitamin C, which favours the absorption of iron from vegetables.

The foods that provide these nutrients come from animals (dairy products, meat, liver, eggs, fish, seafood, vegetables (dark green-leaf vegetables, peppers, tomatoes, cabbage, etc.), fruit (citric fruits, strawberries, etc.), cereals (bread, fortified and wholemeal cereals), pulses and dried fruits (walnuts, peanuts, hazelnuts, etc.). Almost without realizing it, we have listed the foods that define the MD pattern on which Trichopoulou et al. [20] and other groups [43, 44] based the Mediterranean Diet Score (MDS).

We reviewed published studies on the applications in populations of pregnant women of the adapted DQI, the DQI-P [45–48], of the Healthy Eating Index (HEI) and adaptation (HEI-P) [49, 50] and of the adapted MDS (MDS-P) [51], whose characteristics are summarized in Table 7.3. In all of these studies, regardless of the healthy diet model followed, the majority of the population was deficient in iron, folic acid and occasionally in

Ca intake according to the appropriate international recommendations. The DQI-P index has been used by various authors. Briefly, it is constructed from a food consumption frequency questionnaire (FFQ) with items on the intake of cereals, vegetables, fruit, folic acid, iron and calcium, on the percentage of calories from fats, and on the food model score. Each of the eight items is scored from 0 to 10, giving a maximum score of 80 for adherence to recommendations. The first three evaluate the grain, fruit and vegetable intake according to Dietary Guidelines for Americans [42]. The next three items assess the intake of folic acid, iron and calcium, which are especially important in pregnancy. The following item scores the percentage energy from fat according to the Dietary Guidelines for Americans [42]. Finally, item 8 considers eating patterns, based on recommendations by the Institute of Medicine for pregnant women to have a minimum of three meals and two snacks per day. This index has been used by various research groups, notably by Laraia et al. [47, 48].

Various groups found the food-based HEI to be an inadequate instrument for measuring important nutrients during pregnancy [49, 50]. The HEI uses the portions recommended in the Food Guide Pyramid servings for pregnant women in the USA [52]. It assesses the global amount of consumption and identifies ten components (score of 0–10 for each): the intakes of five food groups (cereals, vegetables, fruit, milk and meat), total fat, SFA, cholesterol, and sodium, and the variety of the diet. The index score ranges from 0 to 100. In the modification of the HEI for pregnant women, the HEI-P [53], the diet quality score ranges from 0 to 90 and considers nine components: vegetables, fruit, white meat:red meat ratio, *trans* fats, PUFA:SFA ratio, folic acid, calcium and iron.

The MDS-P [51] is based on the MDS [20] and takes account of the specific need for Fe, Ca and folic acid during pregnancy. The MDS is based on the eight typical components of the MD, i.e., a high consumption of vegetables, fruit, nuts, pulses, cereals, and fish, a high MUFA:SFA ratio and a low consumption of meat and dairy products. A moderate alcohol supply, also typical of the MD, was not considered in the index for pregnant women for reasons discusses earlier in the chapter. Three new components (Fe, Ca and folic acid) were included in the MDS, scoring positively if they met the DRIs for pregnant women. The final MDS-P score ranges from 0 to 11.

Conclusions

Dietary recommendations and DQIs for healthy pregnant women published in the scientific literature to date are all based on international healthy diet models, and there is little difference among them. Indexes are needed that take account of the sensitivity of this population to environmental pollutants contained in foods, such as heavy metals, persistent OCs, synthetic hormone disruptors (e.g., phthalates and bisphenols) and anti-oxidizing agents.

References

1. Scholl TO. Maternal nutrition before and during pregnancy. Nestle Nutr Workshop Ser Pediatr Program. 2008;61:79–89.
2. Human energy requirements. Report of a joint FAO/WHO/UNU expert consultation, Rome; 17–24 Oct 2001.
3. European Food Safety Authority (EFSA). Scientific opinion on dietary reference values for fats, including saturated fatty acids, polyunsaturated fatty acids, monounsaturated fatty acids, trans fatty acids, and cholesterol. EFSA J. 2010;8(3):1461.
4. Koletzko B, Cetin I, Brenna JT. Dietary fat intakes for pregnant and lactating women. Br J Nutr. 2007; 98(5):873–7.
5. Dietary Reference Intakes for Energy, Carbohydrate. Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (2002/2005). This report may be accessed via www.nap.edu.
6. Imdad A, Bhutta ZA. Effect of balanced protein energy supplementation during pregnancy on birth outcomes. BMC Public Health. 2011;11 Suppl 3:S17.
7. Berti C, Biesalski HK, Gärtner R, Lapillonne A, Pietrzik K, Poston L, et al. Micronutrients in pregnancy: current knowledge and unresolved questions. Clin Nutr. 2011;30(6):689–701.
8. Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride. 1997. The reports may be accessed via www.nap.edu. Copyright 2001 by The National Academies.

9. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. 1998. The reports may be accessed via www.nap.edu. Copyright 2001 by The National Academies.
10. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. 2000. The reports may be accessed via www.nap.edu. Copyright 2001 by The National Academies.
11. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. 2001. The reports may be accessed via www.nap.edu. Copyright 2001 by The National Academies.
12. Beard JL. Why iron deficiency is important in infant development. J Nutr. 2008;138:2534–6.
13. Zimmermann MB. Iodine deficiency. Endocr Rev. 2009;30(4):376–408.
14. Greenberg JA, Bell SJ, Guan Y, Yu YH. Folic acid supplementation and pregnancy: more than just neural tube defect prevention. Rev Obstet Gynecol. 2011; 4(2):52–9.
15. Kovacs CS. Vitamin D, in pregnancy and lactation: maternal, fetal, and neonatal outcomes from human and animal studies. Am J Clin Nutr. 2008;88:520S–8.
16. FAO/WHO Report. Vitamin and mineral requirements in human nutrition. 2nd ed. Geneva: World Health Organization and Food and Agriculture Organization of the United Nations; 2004.
17. Mediterranean Diet Foundation. http://www.fdmed.org/.
18. Castro-Rodriguez JA, Garcia-Marcos L, Sanchez-Solis M, Pérez-Fernández V, Martinez-Torres A, Mallol J. Olive oil during pregnancy is associated with reduced wheezing during the first year of life of the offspring. Pediatr Pulmonol. 2010;45(4):395–402.
19. Toledo E, Lopez-del Burgo C, Ruiz-Zambrana A, Donazar M, Navarro-Blasco I, Martínez-González MA, et al. Dietary patterns and difficulty conceiving: a nested case–control study. Fertil Steril. 2011;96(5): 1149–53.
20. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348: 2599–608.
21. Bull World Health Organ. Fetal alcohol syndrome: dashed hopes, damaged lives 2011;89:398–9. doi:10.2471/BLT.11.020611.
22. Jones KL. The effects of alcohol on fetal development. Birth Defects Res C Embryo Today. 2011;93(1):3–11. doi:10.1002/bdrc.20200.
23. Vagnarelli F, Palmi I, García-Algar O, Falcon M, Memo L, Tarani L, et al. A survey of Italian and Spanish neonatologists and paediatricians regarding awareness of the diagnosis of FAS and FASD and maternal ethanol use during pregnancy. BMC Pediatr. 2011;11:51.
24. Papamichael CM, Karatzi KN, Papaioannou TG, Karatzis EN, Katsichti P, Sideris V, et al. Acute combined effects of olive oil and wine on pressure wave reflections: another beneficial influence of the Mediterranean diet antioxidants? J Hypertens. 2008; 26(2):223–9.
25. Binkoski AE, Kris-Etherton PM, Wilson TA, Mountain ML, Nicolosi RJ. Balance of unsaturated fatty acids is important to a cholesterol-lowering diet: comparison of mid-oleic sunflower oil and olive oil on cardiovascular disease risk factors. J Am Diet Assoc. 2005;105(7):1080–6.
26. Gambacorta G, Faccia M, Previtali MA, Pati S, La Notte E, Baiano A. Effects of olive maturation and stoning on quality indices and antioxidant content of extra virgin oils (cv. Coratina) during storage. J Food Sci. 2010;75(3):C229–35.
27. Salvini S, Sera F, Caruso D, Giovannelli L, Visioli F, Saieva C, et al. Daily consumption of a high-phenol extra-virgin olive oil reduces oxidative DNA damage in postmenopausal women. Br J Nutr. 2006;95(4): 742–51.
28. Patelarou E, Giourgouli G, Lykeridou A, Vrioni E, Fotos N, Siamaga E, et al. Association between biomarker-quantified antioxidant status during pregnancy and infancy and allergic disease during early childhood: a systematic review. Nutr Rev. 2011; 69(11):627–41.doi:10.1111/j.1753-4887.2011.00445.x.
29. Antonakou A, Chiou A, Andrikopoulos NK, Bakoula C, Matalas AL. Breast milk tocopherol content during the first six months in exclusively breastfeeding Greek women. Eur J Nutr. 2011;50(3):195–202.
30. Huffman SL, Harika RK, Eilander A, Osendarp SJ. Essential fats: how do they affect growth and development of infants and young children in developing countries? A literature review. Matern Child Nutr. 2011;7 Suppl3:44–65.doi:10.1111/j.1740-8709.2011.00356.x.
31. Miles AM, Noakes PS, Kremmyda LS, et al. The Salmon in pregnancy study: study design, subject characteristics, maternal fish and marine n-3 fatty acid intake, and marine n-3 fatty acid status in maternal and umbilical cord blood. Am J Clin Nutr. 2011; 94(suppl):1986S–92.
32. Calder PC, Kremmyda LS, Vlachava M, Noakes PS, Miles EA. Is there a role for fatty acids in early life programming of the immune system? Proc Nutr Soc. 2010;69(3):373–80.
33. Bloomingdale A, Guthrie LB, Price S, Wright RO, Platek D, Haines J, et al. A qualitative study of fish consumption during pregnancy. Am J Clin Nutr. 2010;92(5):1234–40.
34. Hansen S, Nieboer E, Sandanger TM, Wilsgaard T, Thomassen Y, Veyhe AS, et al. Changes in maternal blood concentrations of selected essential and toxic elements during and after pregnancy. J Environ Monit. 2011;13(8):2143–52.
35. FAO/WHO. Food and Agriculture Organization of the Unite Nations. World Health Organization. Joint FAO/WHO expert committee on food additives. sixty-first meeting, Rome, 10–19 June 2003. Summary and conclusions. Disponible en: http://whqlibdoc.who.int/trs/WHO_TRS_922.pdf.
36. Comisión Europea. Information note. Methyl mercury in fish and fishery products; 2008. http://ec.

europa.eu/food/food/chemicalsafety/contaminants/information_note_mercury-fish_21-04-2008.pdf.
37. AESAN. Agencia Española de Seguridad Alimentaria y Nutrición. Modelo de dieta española para la determinaciónde la exposición del consumidor a sustancias químicas; 2006. http://www.aesan.msps.es/AESAN/docs/docs/docs/notas_prensa/modelo_dieta_espanola.pdf.
38. WHO. Organización Mundial de la Salud. Guidance for identifying populations at risk from mercury exposure. Issued by UNEP DTIE Chemicals Branch and WHO Department of Food Safety, Zoonoses and Foodborne Disease. Geneva; 2008. http://www.who.int/foodsafety/publications/chem/mercuryexposure.pdf.
39. Moreno Frías M, Jiménez Torres M, Garrido Frenich A, Martínez Vidal JL, Olea-Serrano F, Olea A. Determination of organochlorine compounds in human biological samples by GC-MS/MS. Biomed Chromatogr. 2004;18(2):102–11.
40. Rivas A, Cerrillo I, Granada A, Mariscal-Arcas M, Olea-Serrano F. Pesticide exposure of two age groups of women and its relationship with their diet. Sci Total Environ. 2007;382(1):14–21.
41. Mariscal-Arcas M, Lopez-Martinez C, Granada A, Olea N, Lorenzo-Tovar ML, Olea-Serrano F. Organochlorine pesticides in umbilical cord blood serum of women from Southern Spain and adherence to the Mediterranean diet. Food Chem Toxicol. 2010;48(5):1311–5.
42. US. Department of Health and Human Services, U.S. Department of Agriculture. Dietary guidelines for Americans; 2005. http://www.health.gov/dietaryguidelines/dga2005/documento/pdf.
43. Kant AK. Indexes of overall diet quality: a review. J Am Diet Assoc. 1996;96:785–91.
44. Pitsavos C, Panagiotakos DB, Tzima N, Chrysohoou C, Economou M, Zampelas A, et al. Adherence to the Mediterranean diet is associated with total antioxidant capacity in healthy adults: the ATTICA study. Am J Clin Nutr. 2005;82:694–9.
45. Fowles ER, Bryant M, Kim S, Walker LO, Ruiz RJ, Timmerman GM, et al. Predictors of dietary quality in low-income pregnant women: a path analysis. Nurs Res. 2011;60(5):286–94.
46. Bodnar LM, Siega-Riz AM. A Diet Quality Index for pregnancy detects variation in diet and differences by sociodemographic factors. Public Health Nutr. 2002;5(6):801–9.
47. Laraia BA, Siega-Riz AM, Kaufman JS, Jones SJ. Proximity of supermarkets is positively associated with diet quality index for pregnancy. Prev Med. 2004;39:869–75.
48. Laraia BA, Bodnar LM, Siega-Riz AM. Pregravid body mass index is negatively associated with diet quality during pregnancy. Public Health Nutr. 2007;10(9):920–6.
49. Pick ME, Edwards M, Moreau D, Ryan EA. Assessment of diet quality in pregnant women using the Healthy Eating Index. J Am Diet Assoc. 2005;105(2):240–6.
50. Tsigga M, Filis V, Hatzopoulou K, Kotzamanidis C, Grammatikopoulou MG. Healthy Eating Index during pregnancy according to pre-gravid and gravid weight status. Public Health Nutr. 2011;14(2):290–6.
51. Mariscal-Arcas M, Rivas A, Monteagudo C, Granada A, Cerrillo I, Olea-Serrano F. Proposal of a Mediterranean diet index for pregnant women. Br J Nutr. 2009;102(5):744–9.
52. American Dietetic Association. Position of the American Dietetic Association: nutrition and lifestyle for a healthy pregnancy outcome. J Am Diet Assoc. 2002;102:1479–90.
53. Rifas-Shiman SL, Rich-Edwards JW, Kleinman KP, Oken E, Gillman MW. Dietary quality during pregnancy varies by maternal characteristics in Project Viva: a US cohort. J Am Diet Assoc. 2009;109(6):1004–11.

8 Maternal Dietary Counselling and Children's Diet Quality

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Key Points

- The first 2–3 years of life play an important role in the future food habits.
- Maternal attitudes and beliefs most directly affect children's food intake.
- Maternal dietary counselling is effective to improve children diet quality even in low income population.
- In childhood, diet quality influences health condition since it is a critical period for the prevention of both deficiency-related and excess-related nutritional disorders.
- Interventions to promote healthy feeding practices at the beginning of life have the potential to influence health conditions throughout the course of life.

Keywords
Diet • Counselling • Food habits • Infant • Child • Health

Abbreviations

ALSPAC	Avon Longitudinal Study of Parents and Children
HDL	High-density lipoprotein
HEI	Healthy Eating Index
LDL	Low-density lipoprotein

Introduction

The early years of childhood are characterized by accelerated growth and huge acquisitions in the development process, including the ability to

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receive, chew and digest foods besides breast milk as well as ingestive self-control. There is evidence that the foods that children receive in the first 2 years of life will determine their preferences and that these preferences can last until adulthood [1–3]. Consequently, inadequate feeding practices during childhood constitute a public health problem, being associated with the occurrence of micronutrient deficiencies [4], overweight [5] and later cardiovascular disease risk factors [6]. Thus, this chapter will discuss the formation of feeding habits in early childhood, the importance and influence of nutritional programs and guidance for mothers and the impact of diet quality on children's health.

The Development of Children's Eating Habits

Food habits are a result of genetic factors; environmental and family influences; psychosocial and cultural factors; and especially individual preferences [2, 7]. Children are genetically predisposed to accept sweet-tasting substances, reject acidic and bitter ones and associate food intake with its psychophysiological consequences [7–9]. Beginning at age two in particular, children are also predisposed to food neophobia—a phase in which the rejection of new foods is manifested [9]. A survey of approximately 600 children between the ages of 2 and 6 years demonstrated that neophobia was associated with lower consumption of fruits, vegetables and meats but did not influence the intake of dairy products, cakes, cookies and other flour products [10].

Surveys conducted with children aged 2–14 in different countries have demonstrated a transcultural pattern regarding food preferences: foods with a high concentration of sugar and fat such as ice cream, pizza, cake and French fries were indicated as favourites while vegetables were named as the least-preferred food group [11–13].

Despite the genetic and biological basis in determining food preferences, studies indicate that they are strongly modified by the intensity of children's experiences with food [9–14]. Food exposure frequency, the physiological consequences caused by the intake of different food types and the social context of eating are factors that can affect food acceptance patterns in the first years of life [14].

In the first years of life, parents also play an important role throughout the development of eating habits since they influence food familiarity by controlling a child's food environment, exposure and availability [15, 16]. Although there are innumerable external factors that influence eating habits—such as family income, food prices and advertising [17, 18]—evidence indicates that maternal attitudes and beliefs most directly affect children's food intake [15].

Inappropriate feeding practices in the first years of life are associated with parents' or caregivers' lack of knowledge and information as well as restrictions imposed by traditions, beliefs and myths [19]. Thus interventions to disseminate appropriate knowledge and information to mothers are essential for behavioural changes and improved infant feeding [20].

Maternal Education Programs to Improve Infant Feeding Practices

There are many limiting factors that can interfere with an educational program's success—such as intervention intensity, the complexity of behaviour to be changed and the existence of external constraints such as a lack of access to food [21]. The ideal design for each intervention depends on local culture, resources, infrastructure and communication channels as well as the potential to modify them [22]. The most effective educational interventions use a limited number of messages aimed at feasible practices to be adopted by the target population, besides being based on local health problem data related to feeding practices that can plausibly be modified [22].

Although increasing the duration of exclusive breastfeeding is still a global challenge—including in countries with high breastfeeding rates—studies conducted in countries with different cultures and socioeconomic conditions demonstrate the effectiveness of maternal counselling on the promotion of this practice [23]. Considering the fact that the main reasons for the low prevalence of exclusive breastfeeding are the absence of social group and health professional support; the mother's stress; maternal perception that breast milk does not adequately meet the baby's needs; and family pressure to introduce new foods [24], programs that provide mothers with information, counselling and support appear essential in reversing this situation. Ibanez et al. [25] evaluated ten studies conducted with 1,445 women of low socioeconomic status and concluded that educational programs conducted with mothers were effective in stimulating breastfeeding initiation and continuing breastfeeding until 3 months after birth. The studies assessed had various forms of intervention, but the most effective ones conducted multiple yet short orientation sessions (~30 min).

Shi and Zang [20] performed a systematic review of studies published since 1998 that assess the effectiveness of dietary interventions with educational components related to complementary feeding in developing countries. The authors described 15 studies in different countries (including nine randomized clinical trials) and concluded that the evidence supports the hypothesis that educational interventions are effective in improving the quality of complementary feeding but that the analysis and comparison of the studies' results must take into consideration the different local profiles relating to socioeconomic conditions, disease prevalence and children's nutritional status (Table 8.1). In a meta-analysis, Imdad et al. [26] assessed the impact of experimental and quasi-experimental studies that use educational strategies with mothers (with or without the provision of fortified foods) on children's growth in developing countries. Likewise, the authors reported that maternal nutritional counselling (with or without the concurrent provision of fortified foods) resulted in greater height and weight gain between 6 and 24 months of age. Most of the studies described were conducted in communities with a moderate or high prevalence of child malnutrition and limited food availability and demonstrated that maternal dietary counselling can be effective in increasing energy intake, food consumption frequency and diet quality as well as accelerating height and weight gain in the first 2 years of life.

However, the current epidemiological situation in the majority of developed and developing countries (characterized by high childhood obesity rates and early onset of cardiovascular risk factors) highlights the need for action strategies to also be focused on problems related to excessive weight gain and associated risk factors. The few studies that have been published in the scientific literature have assessed the effectiveness of infant interventions with this objective.

In a recent systematic review, Ciampa et al. [27] described only 12 articles (representing the results of ten studies) that aimed to test educational interventions conducted with the parents of children under 2 years old for obesity prevention. Among the studies described, only three were randomized clinical trials and seven were evaluated as being "of low quality" due to methodological limitations. No intervention caused any significant impact on the children's weight.

In a short-term assessment, Kavanagh et al. [28] conducted a randomized clinical trial with children aged between 3 and 10 months who were artificially breastfed in the city of Sacramento, CA, USA with the objective of preventing excessive weight gain. The parents of children belonging to the intervention group participated in a single orientation session about infant feeding in which they were advised on how to recognize signs of satiety and limit the volume of bottle milk per meal. The results showed no change in bottle-feeding practices among the groups, stressing the difficulty of modifying this behaviour among children.

The *Strip Study* is a randomized clinical trial that begun in Finland in 1990 in which families with 5-month-old children were recruited and randomized to receive home-based dietary counselling at an interval of 1–3 months when the children were between 8 and 24 months of age and twice a year from the age of 2 years. The intervention was carried out individually and was based on families' experiences during the well-child care visits and aimed mainly to ensure the quantity and quality of fat intake. The results showed that, between 8 months and 2 years of age, the intervention group had a lower intake of total fat (a 2 % reduction in relation to total energy consumed) and saturated fat (a 3 % reduction in

Table 8.1 Nutrition education intervention programs to improve infant complementary feeding in developing countries [20]

Site	Subjects	Intervention strategies	Main findings
Bhandari et al. (2004), India	Five hundred and fifty-two in the intervention (four communities) and 473 in the control (four communities). Followed from birth to 18 months of age	Nutrition counselling, monthly home visits, group training, feeding demonstration, community mobilization	Children in the intervention group got greater length gain (0.32 cm, 95 % CI: 0.03–0.61) than did controls. But there was no difference in weight. The intervention group had higher energy intakes (531 kJ/day at 9 months and 1,230 kJ/day at 18 months)
Kilaru et al. (2005), India	Sixty-nine infants aged 5–11 months each from the intervention and control were followed till 24 months	Monthly nutrition education, growth chart	Girls in the intervention group had a weight velocity 77 g per month greater than controls. The intervention increased feeding frequency, dietary diversity and consumption of bananas
Roy et al. (2005), Bangladesh	Two hundred and eighty-two moderately malnourished children aged 6–24 months from 15 communities were randomized to two intervention groups and one comparison group	INE group received nutrition education (group training and demonstration of preparing khichuri), the INE + SF group received nutrition education and food supplementation	The WAZ score was 0.28 higher in the INE group and 0.43 higher in the INE + SF group compared to the comparison group. No significant difference in nutritional status between the INE and INE + SF groups
Penny et al. (2005), Peru	One hundred and eighty-seven children from six communities in intervention and 190 from six communities in control. Followed from birth to 18 months of age	Nutrition counselling, group training, demonstration of recipe preparation, recommended recipes: thick puree, adding liver, egg, fish to infant diet	The intervention group was 1.07 cm (95 % CI: 0.49–1.65) taller and 0.30 kg (95 % CI: 0.06, 0.53) heavier than the controls. The increment in WAZ score of the intervention group was 0.29 (95 % CI: 0.1–0.47) higher, and increment in HAZ was 0.39 (95 % CI: 0.21–0.56) higher
Santos et al. [21], Brazil	Twenty-eight health centres were paired and randomized to treatment. Two hundred and eighteen children <18 months in intervention and 206 controls were visited 8, 45 and 180 days after initial consultation	Nutrition education on IMCI feeding guidelines (feeding frequency, increase energy and nutrient density, add animal protein and micronutrients)	The intervention group had higher daily fat, energy and nutrient intake and better knowledge. Among those children entered the intervention after 1 year of age, weight gain was higher in intervention group (1.48 vs. 1.14 kg); WAZ and WHZ score gain was higher in intervention group (0.3 vs. 0.1; and 0.4 vs. 0.1)
Salehi et al. (2004), Iran	Four hundred and six children aged 0–59 months from intervention group and 405 controls. Intervention lasted for 12 months	Training influential people, girls and tribal teachers to disseminate use of eggs, vegetables and legumes	The intervention group achieved 0.42 kg heavier in weight, 1.6 cm longer in length, 0.5 cm longer MAC, 0.45 greater WAZ, 0.41 greater HAZ and 0.266 greater WHZ than controls ($p < 0.05$)

(continued)

Table 8.1 (continued)

Site	Subjects	Intervention strategies	Main findings
Mackintosh et al. (2002) Viet Nam	Forty-six households were selected from four PANP communities and 25 from comparisons. Two children from each household, one in PANP program, another not	Nutrition counselling, group training, community mobilization	The WAZ was 0.63 higher and the WHZ was 0.74 higher in the intervention group than the comparison. The intervention group performed better in feeding frequency and hand-washing practices
Pacho et al. (2002), Viet Nam	Twelve communities were randomized to treatment. One hundred and nineteen children 5–25 months each in intervention and comparison. Followed for 6 months	Nutrition education, growth monitoring	The intervention group had higher consumption of all food groups and higher nutrient and energy intakes
Gardener et al. (2007), West Africa	Two cross-sectional surveys (before/after intervention): 1,807 children 6–35 months at baseline and 1,676 at the second survey	Nutritional education, growth monitoring and food supplementation	The wasting rate of the intervention group decreased from 13.7 to 8.6 %, compared to from 11.3 to 10.8 % in the control group
Hotz and Gilson (2005), Malawi	Eighty-seven and 42 breastfed children aged 9–23 months from three intervention villages and one control village	Four nutrition education sessions, individual counselling, use of soaked pounded maize flour, enriching maize porridge with egg, banana, oil, etc.	The intervention group had larger quantity of complementary foods and higher diet intake of energy, animal protein and micronutrient. Did not report growth data
Guldan et al. [19], China	Two hundred and fifty infants from two intervention townships and 245 from two control townships, recruited at birth and evaluated at 4–12 months	Monthly growth monitoring, nutrition counselling to pregnant women, complementary food recipes	The intervention group had higher WAZ (−1.17 vs. −1.93) and HAZ (−1.32 vs. 1.96), lower anaemia rate (22 % vs. 32 %) and higher breast-feeding rate (83 % vs. 75 %) at 12 months
Li et al. (2007), China	Three hundred and fifty-two newborns of Dai minority in Yunnan Province	Nutrition education, growth monitoring, demonstration of preparing weaning food, integrated management of childhood diseases	Weight was increased by 0.83 kg among male infants aged 4–5 months, 0.64 and 0.42 kg between 12–13 and 15–16 months. Prevalence of underweight decreased from 20.5 to 13.7 % between 6 and 11 months, from 39.0 to 26.4 % at 12–17 months
Bhandari et al. (2001), India	Four hundred and eighteen infants 4 months of age were randomized to intervention or comparison groups and followed until 12 months	Two intervention groups: food supplementation (milk cereal supplement packet) and nutritional counselling	Food supplementation increased energy intakes (1,212–2,257 kJ) and weight increment (250 g, 95 % CI: 20–480 g); no effects on length; nutritional counselling alone had no impacts on weight and length, but increased energy intakes (280–752 kJ)

(continued)

Table 8.1 (continued)

Site	Subjects	Intervention strategies	Main findings
Aboud et al. (2008), Bangladesh	One hundred children 12–24 months of age were in the intervention group and 102 comparable children were enrolled in control group. They were followed until 5 months post-intervention	Weekly education sessions on child nutrition, child self-feeding and parent's responsive feeding	Children in intervention group gained 0.34 kg more weight than controls. Child self-feeding behaviour, diet variety and mother's knowledge were better in the intervention than control group
Campbell et al. (1998), Pakistan	Three hundred and seventy-five children aged 6–24 months were recruited from 36 health centres and assigned to intervention or control. They were followed for 180 days post-intervention	Health care providers received training on feeding counselling using the Integrated Management of Childhood Illness (IMCI) module, then they discussed with mothers regarding the recommended foods and frequency of feeding	The intervention group had significantly higher weight-for-age score than the control group (−1.14 vs. −1.65). Mothers in the intervention group achieved better knowledge

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PANP poverty alleviation and nutrition program, WAZ weight-for-age z score, WHZ weight for height z score, HAZ height for age z score, INE group intensive nutrition education, INE+SF group intensive nutrition education and additional supplementary feeding, MAC mean arm circumference

relation to total energy consumed) and greater intake of polyunsaturated fat (a 1 % increase in relation to total energy consumed)—without differences in total energy, vitamins and minerals consumed between the groups [29]. Monitoring of the children showed that the intervention continued to positively influence the children's diets between 4 and 10 years of age, with the intervention group having 2–3 % lower intake of total and saturated fat and 0.5–1 % higher intake of polyunsaturated fat in relation to total energetic intake [30]. In regard to nutritional status, the intervention demonstrated a protective role against the development of overweight from 2 to 10 years of age, but only among girls [31]. Total and LDL serum cholesterol levels were lower in the intervention group compared to the control group during all years of the follow-up, while HDL levels showed no significant differences [32]. At 9 years of age, the intervention group demonstrated better insulin sensitivity compared to the control group [33].

The Brazilian Health Ministry and the Pan-American Health Organization published the first edition of the "Feeding Guide for Children Under Two" in Brazil in 2002 [34]. The publication was based on feeding guidelines proposed by the World Health Organization for this life cycle and was prepared according to local socioeconomic and cultural contexts and health problems related to feeding—after extensive data collection in the country. Using this study, recommendations were prepared and published in the technical manual "Ten Steps for Healthy Feeding for Brazilian Children from Birth to Two Years of Age", aiming to guide the content of messages to be passed on to the target population. Its main objectives are the promotion of exclusive breastfeeding until the child's sixth month of age, complementary feeding until 2 years of age and appropriate introduction of complementary foods [35].

The "Ten Steps for Healthy Feeding for Brazilian Children From Birth to Two Years of Age" are: (1) Exclusive breastfeeding up to 6 months; (2) After 6 months, mothers should continue breastfeeding up to 2 years and gradually introduce complementary foods; (3)

Table 8.2 Effectiveness of a home-based maternal dietary counselling on breastfeeding practices [36]

		Intervention		Control			
		n	%	*n*	%	RR	95 % CI
Feeding practices							
Exclusive breastfeeding	<1 month	54	33.3	111	48.0	0.69	0.54–0.90
	>=4 months	73	45.1	66	28.6	1.58	1.21–2.06
	=6 months	31	19.1	19	8.2	2.34	1.37–3.99
Breastfeeding	At 6 months	114	66.3	134	55.6	1.19	1.02–1.39
	At 12 months	86	52.8	98	41.9	1.26	1.02–1.55

Adapted from Vitolo MR, Bortolini GA, Feldens CA, Drachler ML. [Impacts of the 10 Steps to Healthy Feeding in Infants: a randomized field trial]. Cad Saúde Pública. September-October 2005;21(5):1448–1457. Article in Portuguese

Table 8.3 Effectiveness of a home-based maternal dietary counselling on energy-dense foods consumption

	Intervention $n = 161$	Control $n = 229$	
	n (%)	*n* (%)	RR (95 % CI)
Consumption in the prior month			
Candies	102 (63.4)	171 (74.7)	0.85 (0.74–0.98)*
Soft drink	118 (73.3)	190 (83.0)	0.88 (0.79–0.99)*
Table sugar	151 (93.8)	219 (95.6)	0.98 (0.93–1.03)
Honey	52 (32.3)	114 (49.8)	0.65 (0.50–0.84)*
Cookies	112 (69.6)	201 (87.8)	0.79 (0.71–0.89)*
Chocolate	81 (50.3)	160 (69.9)	0.72 (0.60–0.86)*
Salty snacks	113 (70.2)	187 (81.7)	0.86 (0.76–0.97)*
LD foods group	57 (35.4)	129 (56.3)	0.62 (0.49–0.80)*
SD foods group	27 (16.8)	83 (36.2)	0.46 (0.31–0.68)*
Early introduction			
Honey (<6 months)	32 (19.9)	98 (42.6)	0.47 (0.33–0.66)*
Table sugar (<6 months)	97 (60.2)	167 (72.0)	0.84 (0.72–0.97)*
Soft drink (<10 months)	74 (45.7)	138 (60.0)	0.76 (0.62–0.93)*

Adapted from Vitolo MR, Campagnolo PD, Hoffman DJ. Maternal dietary counseling reduces consumption of energy-dense foods among infants: a randomized controlled trial J Nutr Educ Behav. Mar 2012, 44(2):140–147
SD group: children who had eaten all the following foods, candies, soft drinks, table sugar and honey during the last month. LD group: Children who had eaten all the following foods, salty snacks, cookies and chocolate during the last month
*$p < 0.05$

Complementary food should be given 3 times a day (two fruits and a meal at lunch) if the child was being breastfed; 5 times, if not and the mothers were advised not to use bottles or breastfeeding as pacifiers; (4) Mealtimes should be adjusted to the child's internal hunger and satiety cues; (5) New foods should gradually get thicker up to the time the child is able to eat a family meal and complementary foods should not be liquefied as a soup; (6) A wide variety of healthy foods should be given every day to ensure intake of different nutrients; (7) Daily intake of different fruits and vegetables; (8) Avoid offering sugar, sweets, soft drinks, salty snacks, cookies, processed and fried foods; (9) Good hygienic practices in preparing and handling food; (10) Adequate feeding patterns during child illness [35].

The impact of these Brazilian feeding guidelines through maternal counselling during home visits in the first year of life showed that the intervention was associated with a higher occurrence of exclusive breastfeeding and breastfeeding in general (Table 8.2) [36] and lower intake of high-energy foods in the first year of life (Table 8.3) [37]. In the same group of children, a longitudinal study showed that at preschool age (3–4 years),

Table 8.4 Effectiveness of a home-based maternal dietary counselling on diet quality at 3–4 years old [38]

	Intervention group $n=145$	Control group $n=200$	Difference 95 % CI
HEI score			
Mean ± SD	67.7 ± 10.5**	64.2 ± 11.6	3.52 (1.18–5.88)
Minimum–maximum	44.4–89.8	31.6–91.0	
Median (Q1-Q3)	68.6 (59.3–75.3)	65.2 (57.3–72.2)	
Children with poor diet (HEI score < 50)			
n (%)	6 (4.1)*	28 (14.0)	9.9 (4.1–15.7)
RR (95 % CI)	0.30 (0.13–0.70)	1.00	
NNT (95 % CI)	10.1 (6.3–24.3)		
Children with a good diet (HEI score > 80)			
n (%)	20 (13.8)*	13 (6.5)	7.3 (0.8–13.8)
RR (95 % CI)	2.12 (1.09–4.12)	1.00	

Adapted from Vitolo MR, Rauber F, Campagnolo PD, Feldens CA, Hoffman DJ. Maternal dietary counseling in the first year of life is associated with a higher healthy eating index in childhood. J Nutr. November 2010;140(11):2002–2007

*Different from control, $p<0.05$

**Different from control, $p<0.01$. HEI scores: poor diet (HEI score, 50 points); diet needs improvement (HEI score between 51 and 80 points); good diet (HEI score, 80 points)

Table 8.5 Effectiveness of a home-based maternal dietary counselling on diet quality at 3–4 years old [38]

	Intervention ($n=145$)		Control ($n=200$)		
	Min–Max	Mean ± SD	Min–Max	Mean ± SD	Difference, 95 % CI
Grains[a]	0.83–10.0	5.07 ± 2.22	0.0–10.0	5.18 ± 2.34	–0.11 (–0.60 to 0.38)
Meat group[a]	0.00–10.0	6.19 ± 3.05	0.00–10.0	6.09 ± 3.07	0.10 (–0.56 to 0.75)
Vegetables[b]	0.00–10.0	1.53 ± 2.10	0.00–10.0	1.00 ± 1.84	0.53 (0.10–0.95)**
Fruits[a]	0.00–10.0	4.43 ± 3.56	0.00–10.0	3.56 ± 3.29	0.87 (0.15–1.59)*
Milk[a]	0.00–10.0	8.95 ± 2.34	0.00–10.0	8.60 ± 2.77	0.34 (20.20–0.88)
Variety[a]	0.00–10.0	7.15 ± 2.71	0.00–10.0	6.35 ± 2.95	0.80 (0.19–1.42)*
Total fat[b,c]	0.68–10.0	9.15 ± 1.80	0.00–10.0	9.08 ± 1.85	0.07 (–0.32 to 0.46)
Sodium[b,c]	0.00–10.0	8.64 ± 3.22	0.00–10.0	7.73 ± 3.90	0.91 (0.15–1.66)
Cholesterol[b,c]	0.00–10.0	9.39 ± 1.91	0.00–10.0	9.70 ± 1.50	–0.31 (–0.69 to 0.07)*
Saturated fat[a,c]	0.00–10.0	7.25 ± 3.51	0.00–10.0	6.926 ± 3.59	0.33 (–0.43 to 1.09)

Adapted from Vitolo MR, Rauber F, Campagnolo PD, Feldens CA, Hoffman DJ. Maternal dietary counseling in the first year of life is associated with a higher healthy eating index in childhood. J Nutr. November 2010;140(11):2002–2007

[a]Student's *t* test

[b]Mann–Whitney test

[c]Lower scores indicate greater intake:

*$p<0.05$; **$p<0.01$

the prevalence of children with low-quality diets (as assessed by the Healthy Eating Index, HEI) was lower in those who participated in the intervention group compared to those who belonged to the control group (Table 8.4) [38]. Children in the intervention group presented higher intake of fruits and vegetables as well as greater dietary variety (Table 8.5). On the other hand, the intervention group also presented higher cholesterol consumption at age 3–4—a result that can be explained by the incentive given to mothers in the first year of life to offer meat and entrails in order to prevent anaemia. However, the intervention group's higher cholesterol consumption did not have a negative impact on the children's lipid levels at school age, as we could observe in the reassessment performed on the same children at age 7–8. The only changes observed in the serum

Table 8.6 Effectiveness of a home-based maternal dietary counselling on the lipid profile at 7–8 years old [39]

	7–8 years				
	Intervention		Control		
	Mean	SD	Mean	SD	Difference (95 % CI)
Boys (*n* = *172*)					
TC (mmol/L)[a]	4.16	0.72	4.22	0.67	–0.04 (–0.27 to 0.14)
LDL (mmol/L)[a]	2.55	0.63	2.57	0.57	–0.02 (–0.19 to 0.17)
HDL (mmol/L)[a]	1.24	0.27	1.30	0.29	–0.06 (–0.14 to 0.03)
TC/HDL[a]	3.46	0.84	3.35	0.65	0.11 (–0.11 to 0.34)
Triglycerides (mmol/L)[a]	0.75[b]	0.29	0.74[b]	0.31	0.01 (–0.07 to 9.10)
Girls (*n* = *132*)					
TC (mmol/L)[a]	4.41	0.76	4.28	0.69	0.13 (–0.11 to 0.39)
LDL (mmol/L)[a]	2.78	0.66	2.68	0.60	0.10 (–0.32 to 0.11)
HDL (mmol/L)[a]	1.30	0.31	1.19	0.27	0.11 (0.00–0.20)[c]
TC/HDL[a]	3.53	0.75	3.69	0.87	–0.16 (–0.44 to 0.13)
Triglycerides (mmol/L)[a]	0.69[b]	0.24	0.82[b]	0.42	–0.13 (–0.25 to –0.01)[c]

Adapted from Louzada MLC, Campagnolo PD, Rauber F, Vitolo MR. Long-term effectiveness of maternal dietary counseling in a low-income population: a randomized field trial. Pediatrics. Jun 2012, 129(6):e1477–1484

TC total cholesterol, *LDL* low-density lipoprotein, *HDL* high-density lipoprotein, *TC/HDL* total-to-HDL cholesterol ratio

[a]Student *t* test was used

[b]These variables were log-transformed for the analyses

[c]Values from intervention group were significantly different from those of the control group ($p<0.05$)

lipid levels between the control and intervention groups were favourable for girls in the intervention group in that their serum HDL levels were 0.11 mmol/L higher (IC 95 % 0.00–0.20) and triglyceride levels were 0.13 mmol/L lower (IC95% –0.25 to –0.01) (Table 8.6) [39].

Importance of Diet Quality in the First Years of Life

The transcendence of the problems related to inappropriate feeding practices during childhood is widely discussed in the literature. The relationship between micronutrient intake and deficiency-related diseases such as anaemia and vitamin A deficiency has been known for years [5, 40]. More recently the literature has described the importance of child nutrition for the development of non-transmissible chronic diseases [6].

Nutrition quality in the prenatal period influences health conditions later in life [41]. Children who had restricted intrauterine nutritional intake tend to present poor growth in childhood and a higher prevalence of long- and short-term nutritional disorders [42–44]. Some of these associations can be attributed to the Barker programming theory in which it is suggested that children who suffered intrauterine growth retardation were submitted to metabolic adjustments in foetal life in order to survive in inadequate circumstances [45, 46]. Such adjustments would be responsible for hormonal and metabolic changes which would result in an increase in insulin resistance, hypertension and cardiovascular diseases in adulthood.

In the first years of life, food quality also influences health conditions since the ages of 6–24 months represent a critical period for the prevention of both deficiency-related and excess-related nutritional disorders [22]. Inadequate complementary feeding practices are associated with diarrhoea, growth retardation and micronutrient deficiencies. Brion et al. [47], using data from the Avon Longitudinal Study of Parents and Children (ALSPAC), showed that sodium intake at 4 months of age was positively associated with systolic blood pressure at 7 years of age after adjusting for age, sex and energy consumption, and Herbst et al. [48] argued that the consumption of added sugar between 1 and 2 years of age was related to higher body mass index values at 7 years of age. In older children, the intake of low-quality foods such as French fries, potato chips, candies and sugary drinks was associated with higher risk of overweight and obesity [49–51]. These results may have even more relevant practical implications considering that childhood eating habits tend to persist into adulthood [52].

Therefore, interventions to promote healthy feeding practices at the beginning of life have the potential to influence health conditions throughout the course of life.

Conclusions

Considering the importance of mothers in shaping children's eating habits, the implementation of educational interventions that aim to improve knowledge and change attitudes and practices in this regard becomes fundamental in changing behaviour and improving feeding quality for children. From the data presented, it can be seen that several studies on educational interventions with mothers have been published in the last decade and have added evidence about the effects of such interventions on children's diet quality and nutritional conditions.

References

1. Beauchamp GK, Moran M. Acceptance of sweet and salty tastes in 2-year-old children. Appetite. 1984;5:291–305.
2. Skinner JD, Carruth BR, Bounds W, Ziegler P. Children's food preferences: a longitudinal analysis. J Am Diet Assoc. 2002;102:1638–47.
3. Leathwood P, Maier A. Early influences on taste preferences. Nestle Nutr Workshop Ser Pediatr Program. 2005;56:127–38.
4. Jiang JX, Lin LM, Lian GL, Greiner T. Vitamin A deficiency and child feeding in Beijing and Guizhou, China. World J Pediatr. 2008;4:20–5.
5. Jennings A, Welch A, van Sluijs EM, Griffin SJ, Cassidy A. Diet quality is independently associated with weight status in children aged 9–10 years. J Nutr. 2011;141:453–9.
6. Lanigan J, Singhal A. Early nutrition and long-term health: a practical approach. Proc Nutr Soc. 2009;68:422–9.
7. Birch LL. Development of food acceptance patterns in the first years of life. Proc Nutr Soc. 1998;57:617–24.
8. Birch LL, Deysher M. Caloric compensation and sensory specific satiety: evidence for self regulation of food intake by young children. Appetite. 1986;7:323–31.
9. Birch LL, Fisher JA. Appetite and eating behavior in children. Pediatr Clin North Am. 1995;42:931–53.
10. Cooke L, Wardle J, Gibson EL. Relationship between parental report of food neophobia and everyday food consumption in 2–6-year-old children. Appetite. 2003;41:205–6.
11. Pérez-Rodrigo C, Ribas L, Serra-Majem L, Aranceta J. Food preferences of Spanish children and young people: the enKid study. Eur J Clin Nutr. 2003;57 Suppl 1:45S–8.
12. Cooke L, Wardle J. Age and gender differences in children's food preferences. Br J Nutr. 2005;93:741–6.
13. Bellisle F, Rolland-Cachera MF. Three consecutive (1993, 1995, 1997) surveys of food intake, nutritional attitudes and knowledge, and lifestyle in 1000 French children, aged 9–11 years. J Hum Nutr Diet. 2007;20:241–51.
14. Birch LL. The role of experience in children's food acceptance patterns. J Am Diet Assoc. 1987;87 Suppl 9:36S–40.
15. Ventura AK, Birch LL. Does parenting affect children's eating and weight status? Int J Behav Nutr Phys Act. 2008;15:15–27.
16. Cooke L, Wardle J, Gibson EL, Sapochnik M, Sheiham A, Lawson M. Demographic, familial and trait predictors of fruit and vegetable consumption by pre-school children. Public Health Nutr. 2004;7:295–302.
17. Claro RM, Monteiro CA. Family income, food prices, and household purchases of fruits and vegetables in Brazil. Rev Saude Publica. 2010;44:1014–20.
18. Taylor JP, Evers S, McKenna M. Determinants of healthy eating in children and youth. Can J Public Health. 2005;96 Suppl 3:20–9.
19. Guldan GS, Fan HC, Ma X, Ni ZZ, Xiang X, Tang MZ. Culturally appropriate nutrition education improves infant feeding and growth in rural Sichuan, China. J Nutr. 2000;130:1204–11.
20. Shi L, Zhang J. Recent evidence of the effectiveness of educational interventions for improving complementary feeding practices in developing countries. J Trop Pediatr. 2010;57:91–8.
21. Santos I, Victora CG, Martines J, Gonçalves H, Gigante DP, Valle NJ, et al. Nutrition counseling increases weight gain among Brazilian children. J Nutr. 2001;131:2866–73.
22. Dewey KG, Adu-Afarwuah S. Systematic review of the efficacy and effectiveness of complementary feeding interventions in developing countries. Matern Child Nutr. 2008;4 Suppl 1:24S–85.

23. Imdad A, Yakoob MY, Bhutta ZA. Effect of breastfeeding promotion interventions on breastfeeding rates, with special focus on developing countries. BMC Public Health. 2011;11 Suppl 3:S24.
24. Erkkola M, Salmenhaara M, Kronberg-Kippilä C, Ahonen S, Arkkola T, Uusitalo L, et al. Predictors of the duration of exclusive breastfeeding among first-time mothers. Res Nurs Health. 2008;31:428–41.
25. Ibanez G, de Reynal de Saint Michel C, Denantes M, Saurel-Cubizolles MJ, Ringa V, Magnier AM. Systematic review and meta-analysis of randomized controlled trials evaluating primary care-based interventions to promote breastfeeding in low-income women. Fam Pract. 2012;29(3):245–54.
26. Imdad A, Yakoob MY, Bhutta ZA. Impact of maternal education about complementary feeding and provision of complementary foods on child growth in developing countries. BMC Public Health. 2011;11(Suppl):3S–25.
27. Ciampa PJ, Kumar D, Barkin SL, Sanders LM, Yin HS, Perrin EM, et al. Interventions aimed at decreasing obesity in children younger than 2 years: a systematic review. Arch Pediatr Adolesc Med. 2010; 164:1098–104.
28. Kavanagh KF, Cohen RJ, Heinig MJ, Dewey KG. Educational intervention to modify bottle-feeding behaviors among formula-feeding mothers in the WIC program: impact on infant formula intake and weight gain. J Nutr Educ Behav. 2008;40:244–50.
29. Lagström H, Jokinen E, Seppänen R, Rönnemaa T, Viikari J, Välimäki I, et al. Nutrient intakes by young children in a prospective randomized trial of a low-saturated fat, low-cholesterol diet. The STRIP Baby Project. Special Turku Coronary Risk Factor Intervention Project for Babies. Arch Pediatr Adolesc Med. 1997;151:181–8.
30. Talvia S, Lagström H, Räsänen M, Salminen M, Räsänen L, Salo P, et al. A randomized intervention since infancy to reduce intake of saturated fat: calorie (energy) and nutrient intakes up to the age of 10 years in the Special Turku Coronary Risk Factor Intervention Project. Arch Pediatr Adolesc Med. 2004;158:41–7.
31. Hakanen M, Lagström H, Kaitosaari T, Niinikoski H, Näntö-Salonen K, Jokinen E, et al. Development of overweight in an atherosclerosis prevention trial starting in early childhood. The STRIP study. Int J Obes (Lond). 2006;30:618–26.
32. Niinikoski H, Lagström H, Jokinen E, Siltala M, Rönnemaa T, Viikari J, et al. Impact of repeated dietary counseling between infancy and 14 years of age on dietary intakes and serum lipids and lipoproteins: the STRIP study. Circulation. 2007;116: 1032–40.
33. Niinikoski H, Lagström H, Jokinen E, Siltala M, Rönnemaa T, Viikari J, et al. Low-saturated fat dietary counseling starting in infancy improves insulin sensitivity in 9-year-old healthy children: the Special Turku Coronary Risk Factor Intervention Project for Children (STRIP) study. Diabetes Care. 2006;29: 781–5.
34. Ministério da Saúde. Dez passos para uma alimentação saudável: guia alimentar para crianças menores de dois anos. Brasília: Ministério da Saúde/Organização Pan-Americana da Saúde; 2002.
35. Ministério da Saúde. Dez passos para uma alimentação saudável: guia alimentar para crianças menores de dois anos. Brasília: Ministério da Saúde; 2010.
36. Vitolo MR, Bortolini GA, Feldens CA, Drachler ML. [Impacts of the 10 steps to healthy feeding in infants: a randomized field trial]. Cad Saude Publica. 2005; 21:1448–57. Article in Portuguese.
37. Vitolo MR, Campagnolo PD, Hoffman DJ. Maternal dietary counseling reduces consumption of energy-dense foods among infants: a randomized controlled trial. J Nutr Educ Behav. 2012;44(2):140–7.
38. Vitolo MR, Rauber F, Campagnolo PD, Feldens CA, Hoffman DJ. Maternal dietary counseling in the first year of life is associated with a higher healthy eating index in childhood. J Nutr. 2010;140:2002–7.
39. Louzada MLC, Campagnolo PD, Rauber F, Vitolo MR. Long-term effectiveness of maternal dietary counseling in a low-income population: a randomized field trial. Pediatrics. 2012;129(6):e1477–84.
40. Osorio MM. [Determinant factors of anemia in children]. J Pediatr (Rio J). 2002;78:269–78. Article in Portuguese.
41. Brion MJ, Ness AR, Rogers I, Emmett P, Cribb V, Davey Smith G. Maternal macronutrient and energy intakes in pregnancy and offspring intake at 10 y: exploring parental comparisons and prenatal effects. Am J Clin Nutr. 2010;91:748–56.
42. Barker DJ, Fall CH. Fetal and infant origins of cardiovascular disease. Arch Dis Child. 1993;68:797–9.
43. Drachler ML, Macluf SPZ, Leite JCC, Aerts DR, Giugliani ER, Horta BL. [Risk factors for overweight in children from Southern Brazil]. Cad Saude Publica. 2003;19:1073–81. Article in Portuguese.
44. Lian WC, Muda WA, Hussin ZA, Ching Thon C. Factors associated with undernutrition among children in a rural district of Kelantan, Malaysia. Asia Pac J Public Health. 2012;24(2):330–42.
45. Barker DJ, Gelow J, Thornburg K, Osmond C, Kajantie E, Eriksson JG. The early origins of chronic heart failure: impaired placental growth and initiation of insulin resistance in childhood. Eur J Heart Fail. 2010;12:819–25.
46. Barker DJ, Osmond C, Forsen TJ, Kajantie E, Eriksson JG. Trajectories of growth among children who have coronary events as adults. N Engl J Med. 2005;353:1802–9.
47. Brion MJ, Ness AR, Smith GD, Emmett P, Rogers I, Whincup P, et al. Sodium intake in infancy and blood pressure at 7 years: findings from the Avon Longitudinal Study of Parents and Children. Eur J Clin Nutr. 2008;62:1162–9.
48. Herbst A, Diethelm K, Cheng G, Alexy U, Icks A, Buyken AE. Direction of associations between added sugar intake in early childhood and body mass index at age 7 years may depend on intake levels. J Nutr. 2011;141:1348–54.

49. Receveur O, Morou K, Gray-Donald K, Macaulay AC. Consumption of key food items is associated with excess weight among elementary-school-aged children in a Canadian first nations community. J Am Diet Assoc. 2008;108:362–6.
50. Fiorito LM, Marini M, Francis LA, Smiciklas-Wright H, Birch LL. Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. Am J Clin Nutr. 2009;90:935–42.
51. O'Neil CE, Fulgoni VL, Nicklas TA. Association of candy consumption with body weight measures, other health risk factors for cardiovascular disease, and diet quality in US children and adolescents: NHANES 1999–2004. Food Nutr Res. 2011;55:5794.
52. Northstone K, Emmett PM. Are dietary patterns stable throughout early and mid-childhood? A birth cohort study. Br J Nutr. 2008;100:1069–76.

9 Diet Quality, Micronutrient Intakes and Economic Vulnerability of Women

Liv Elin Torheim and Mary Arimond

Key Points

- Women are physiologically vulnerable to inadequate micronutrient intakes; they require a more nutrient-dense diet (nutrients per 100 kcal) due to higher needs and (usually) lower food intakes than men.
- Few studies from resource-poor settings report intakes separately for men and women; among these, several document lower or less adequate micronutrient intakes for women than for men.
- Gender differences in micronutrient intakes or adequacy are more pronounced in South Asian countries, and are least prominent in studies from South America; African studies show varying results.
- Studies on gender bias in intra-household food allocation generally confirm the same pattern of differences between continents.
- Both diet quality and micronutrient intakes vary with socioeconomic status in developing countries; food security is an important determinant of diet quality.
- Limited evidence suggests that gender differences in micronutrient intake are not exacerbated by poverty.
- Women might nevertheless be harder hit by poverty and economic crises than men because of their physiological vulnerability and need for nutrient-dense diets.

Keywords

Micronutrient intake • Micronutrient adequacy • Diet quality • Gender • Physiological vulnerability • Intra-household food allocation • Poverty • Food insecurity

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Abbreviations

BMI	Body mass index
BMR	Basal metabolic rate
FAO	Food and Agriculture Organization of the United Nations
MAR	Mean adequacy ratio
NAR	Nutrient adequacy ratio
PA	Prevalence of adequacy
WHO	World Health Organization

Introduction

Women's micronutrient status is important for their current and future health. During pregnancy and lactation, their micronutrient status will also impact on their offspring's health, development and survival [1]. The childbearing role of women also leads to increased requirements for many micronutrients through pregnancy, lactation and menstruation [2]. While being physiologically vulnerable to micronutrient deficiencies, women are also often socially and economically vulnerable. This chapter aims to describe women's vulnerability to low diet quality in terms of micronutrient intake and to investigate how economic disadvantage may further exacerbate this vulnerability, based on a literature review.

This chapter focuses on adequacy of micronutrient intakes, which is both one dimension of diet quality and one determinant of micronutrient status. The chapter also has a geographic focus, on populations in Africa, Asia and Latin America. Micronutrient intakes, like dietary intakes in general, can be assessed using a variety of measurement approaches including weighed records of food intake, 24-h recalls, and validated food frequency questionnaires. Micronutrient adequacy is quantitatively assessed by comparing estimated intakes to reference levels set for individuals and populations [3]. In addition, at population level simple dietary diversity indicators have been shown to be strongly associated with micronutrient intakes and adequacy, as explained in the chapter "Diet quality in developing countries", and thus may serve as proxy indicators of this dimension of diet quality for populations. Other diet quality indicators, which are more geared towards reflecting the risk of chronic diseases, are not considered in this chapter but have been described in several other chapters in this book. The aim of this chapter is to describe and summarize the specific vulnerability of women to micronutrient-poor diets. The PubMed database (years 1990–2011) was searched for relevant literature on gender differences in micronutrient intakes and allocation of resources within households.

Are Women Vulnerable to Low Diet Quality and Low Micronutrient Intakes?

Nutritional Needs of Women and Men

Women are physiologically vulnerable to inadequate nutrient intakes as compared with men. This physiological vulnerability relates mainly to women's reproductive role, since menstruation, pregnancy and lactation increase needs for nutrients, to varying degrees (see Table 9.1). Iron needs, in particular, fluctuate with physiological status. Compared to men, women have an increased need for iron from the moment they reach menarche until menopause. Women who suffer heavy losses are in particular at risk of iron deficiency [4]. During pregnancy, iron requirements increase significantly to cover the needs of the growing foetus.

Requirements for a range of other nutrients also increase during pregnancy and lactation (Table 9.1). Requirements for protein, vitamin A, vitamin C, thiamine, riboflavin, niacin, vitamin B6, vitamin B12, folate, iodine, selenium and zinc all increase. For most of these nutrients, the recommended daily intake for women who are pregnant or lactating exceeds the recommended daily intake for men. In addition, since women often consume less food and have lower energy intakes than men [5], they require diets with a higher nutrient density (Fig. 9.1).[1] Negative

[1]In Fig. 9.1, energy intakes used for calculating the required nutrient density (per 100 kcal) have been set to the same level for pregnant and lactating women as for nonpregnant, non-lactating women, since, in communities with restricted food access, there is evidence that pregnant and lactating women do not increase their energy intakes [6–10].

Table 9.1 Recommended Dietary Allowances[a] according to gender and physiological status

Life stage	Protein (g/day)	Vitamin A (μg/day)	Vitamin C (mg/day)	Vitamin D (μg/day)	Vitamin E (mg/day)	Thiamin (mg/day)	Riboflavin (mg/day)	Niacin (mg/day)	Vitamin B6 (mg/day)	Folate (μg/day)	Vitamin B12 (μg/day)	Calcium (mg/day)	Iodine (μg/day)	Iron (mg/day)	Selenium (μg/day)	Zinc (mg/day)
Women, 19–50 years	46	700	75	15	15	1.1	1.1	14	1.3	400	2.4	1,000	150	18	55	8
Pregnancy	71	770	85	15	15	1.4	1.4	18	1.9	600	2.6	1,000	220	27	60	11
Lactation	71	1,300	120	15	19	1.4	1.6	17	2.0	500	2.8	1,000	290	9	70	12
Men, 19–50 years	56	900	90	15	15	1.2	1.3	16	1.3	400	2.4	1,000	150	8	55	11

This table shows that the daily recommended intake of various nutrients (as recommended by the Food and Nutrition Board, The Institute of Medicine of the National Academy of Sciences, United States) vary between nonpregnant, non-lactating women, pregnant women, lactating women and men

[a]*Source*: Food and Nutrition Board, Institute of Medicine, National Academies: Dietary Reference Intakes. Available at: http://iom.edu/Activities/Nutrition/SummaryDRIs/~/media/Files/Activity%20Files/Nutrition/DRIs/RDA%20and%20AIs_Vitamin%20and%20Elements.pdf and http://www.iom.edu/Global/News%20Announcements/~/media/C5CD2DD7840544979A549EC47E56A02B.ashx. Accessed 19th April 2012

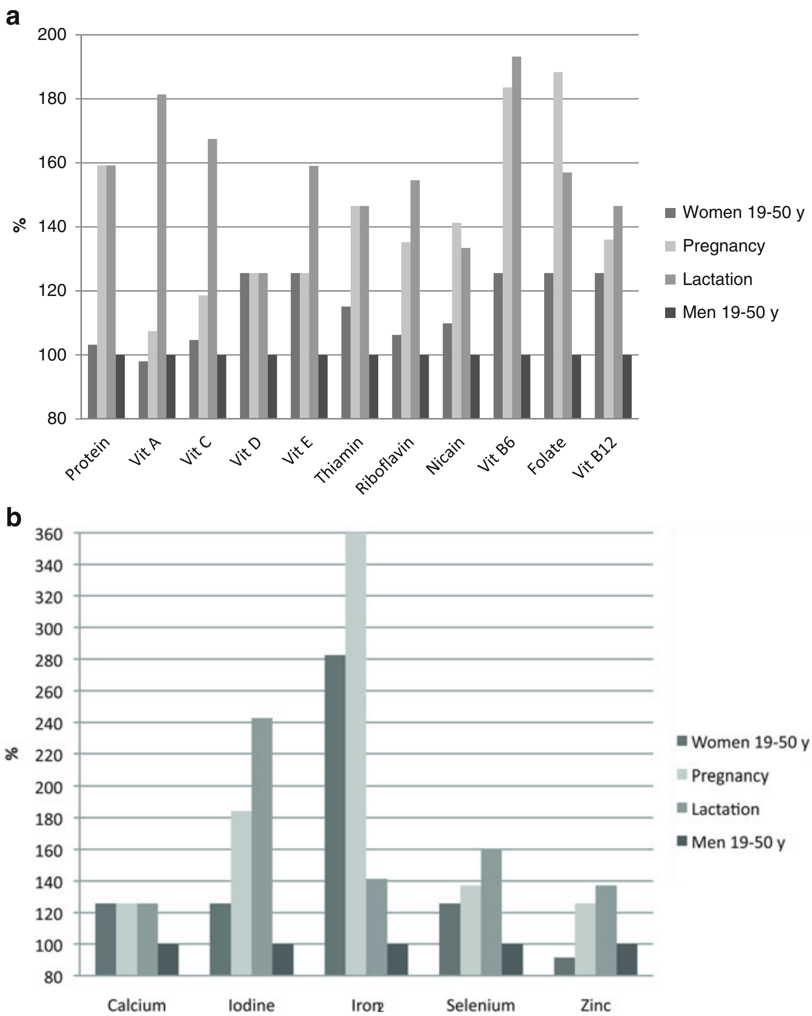

Fig. 9.1 Women's required nutrient densities for meeting recommended micronutrient intakes as percentage of men's required nutrient densities. Nutrient densities based on Recommended Dietary Allowances presented in Table 9.1, and an energy intake of 2,150 kcal for women and 2,700 for men (based on energy needs as presented in FAO/WHO [5] using a body weight of 60 kg for females and 70 for men and a BMR factor (or Physical Activity Level) of 1.65). Required iron density during pregnancy is 424 % higher than the required iron density for men. This figure shows for (**a**) protein and vitamins and (**b**) minerals that in the case of several nutrients, women in general, and in particular pregnant and lactating women require higher nutrient density (mg or µg nutrients per 100 kcal) than men do

consequences of inadequate micronutrient intake during pregnancy are well documented; however, there remain important knowledge gaps [2, 11, 12]. Breast milk micronutrient concentrations are affected by the maternal diet. The micronutrients most affected by low maternal intakes and stores are thiamine, riboflavin, vitamin B6, vitamin B12, vitamin A and iodine [2].

Micronutrient Intakes of Women and Men

Studies from developing countries describing micronutrient intake are scarce. In particular, studies with representative samples of populations, and studies comparing micronutrient intakes of men to those of women are extremely rare [13].

Identified studies from low-resource countries using dietary recall methods of acceptable quality[2] generally show similar results (Table 9.2). Most studies show lower intake and/or less adequate intakes of micronutrients among women as compared to men; however, there are differences between countries and continents. Gender differences are stronger in South Asian countries, whereas they are least prominent in the studies from South America (Table 9.2). In the studies from Africa, the Malian and Gabonese examples showed distinct differences in adequacy of micronutrient intake between men and women, whereas the difference was smaller in South Africa. It is important to note, however, that these studies are few, and representative neither of their respective countries nor of the continents. The studies also present a mix of absolute micronutrient intakes and measures of micronutrient adequacy.

Intra-household Food Allocation

Most studies dealing with nutritional aspects of gender bias within households assessed patterns of food allocation. There have been a number of publications focusing on gender differences and possible gender discrimination in developing countries [15–20]. An extensive review was conducted on the subject by Haddad et al. in 1996 [15]. Fourteen studies that compared food intake (primarily energy intake only) of adult men and women were included in the review. Among the 14, males were favoured in seven studies, females in two, and five studies showed no difference. Only seven studies adjusted energy requirements for activity and body weight. Among these, males were favoured in two studies (South Asia), females were favoured in one study (Southeast Asia) and four studies showed no difference (three from South Asia and one from Southeast Asia). In this review, no studies from Africa or Latin America adjusted energy requirements for activity and body weight [15]. Later studies identified on this topic, seem to confirm the general view. One study by Sudo et al. from Bangladesh found evidence of gender bias, showing that women often received a lower share of nutritious foods than men [19]. Two studies from South America did not find evidence of this [16, 21].

In Nepal, Gittelsohn et al. [22] found that whereas staple foods were equally distributed, side dishes—usually containing a higher proportion of micronutrients—were often preferentially allocated to valued household members, including adult males and small children (of both sexes).

Studies of meal customs in African rural subsistence households typically describe a pattern where men eat separately from women and small children, whereas older children may or may not eat separately from women [23, 24]. In one study from Benin, van Liere et al. found that men got more of the most nutritionally valued foods such as animal-source foods, if available, and larger portions as compared to women [24]. A recent study among East African Pastoralists showed no difference in dietary diversity between adult men and women, whereas adult daughters seemed to be favoured as compared to adult sons [25]. A contrasting finding was reported in a study examining experiences of food insecurity among adolescents in Ethiopia [26]. They found that a larger proportion of girls experienced food insecurity compared to boys, and food insecure girls also reported more illness compared to food insecure boys [26].

A review by Nube and van den Boom examined the prevalence of undernutrition (BMI <18.5 kg/m^2) among women and men in developing countries, with an underlying theoretical concept that a difference in undernutrition could indicate a bias in intra-household food allocation [27]. Their review showed that there are regional variations in the difference in rates of undernutrition between women and men. In communities in Sub-Saharan Africa, prevalence of low BMI was on average a few percentage points higher in men than in women; in South/Southeast Asia, the reverse was the case. Undernutrition rates in Latin America were low and slightly higher among women than men. These results reinforce the findings from Table 9.2, that there might be regional differences in any gender bias of intra-household food allocation.

[2]Either multiple-day records, validated food frequency questionnaires or repeated 24-h recalls.

Table 9.2 Examples of dietary assessment studies from low-resource countries comparing micronutrient intakes of women and men

Source	Country	Sample type	Measure of food consumption	Main conclusion
Africa				
Macintyre et al. [38]	South Africa	Cross-sectional study in an African population from the North West Province of South Africa; $n=1,008$ females and 743 males	Validated quantitative food frequency questionnaire	Intakes of most micronutrients were higher (not statistically tested) for men compared with women within each stratum of urbanization. Exceptions were intakes of vitamin A and C which were in general higher in women
Torheim et al. [14]	Mali	Cross-sectional study in a rural Malian population; $n=275$ females and 227 males	Validated quantitative food frequency questionnaire	Mean adequacy ratio (MAR)[a] was significantly higher in men (0.91, SD 0.08) as compared with women (0.84, SD 0.11, $p<0.001$ in multiple regression model adjusting for other variables)
Blaney et al. [52]	Gabon	Repeated cross-sectional study; $n=194$ adults	7-day food consumption survey	Global nutrient adequacy[b] was significantly higher in men (3.3 (SE 0.1)) as compared with women (2.9 (SE 0.1), $p<0.001$)
Asia				
Gittelsohn et al. [22]	Nepal	Ethnographic study of 767 individuals in 105 house holds (numbers of adult women and men were not given)	Direct observation of meals, and visual estimation of amounts	Women had significantly lower intakes and lower nutrient adequacy ratios of energy, beta-carotene, riboflavin and vitamin C as compared with men.
Venkaiah et al. [53]	India	Cross-sectional study in nine states in India; $n=2,579$ females and males, 10–18 years	24-h recall	Intake of energy and all the nine nutrients analyzed were significantly higher in boys as compared with girls (*p*-values not presented)
Chiplonkars et al. [54]	India	A cross-sectional study in rural and urban communities in India; $n=56$ females and 168 males	Validated quantitative food frequency questionnaire	Intakes of most micronutrients were higher ($p<0.01$) for men compared with women with the exception of vitamin C and ß-carotene
Sudo et al. 2006 [18]	Nepal	Cross-sectional study in Hindi communities in lowland Nepal; $n=195$ females and 122 males	Food frequency questionnaire	Intakes of energy and the seven nutrients analyzed were significantly higher in men as compared with women ($p<0.001$)

(continued)

Table 9.2 (continued)

Source	Country	Sample type	Measure of food consumption	Main conclusion
Latin America				
Barquera et al. [37]	Mexico	Nationally representative cross-sectional survey; $n=9\ 848$ females and 5 898 males	Food frequency questionnaire	Prevalence of adequacy (PA) of iron, folate and calcium was higher in men than in women, whereas PA of vitamin A, vitamin C and zinc was higher in women
Berti and Leonard [55]	Ecuador	Cross-sectional study in one village in Ecuador; $n=41$ females and 39 males >20 years	Repeated 24-h recalls (on average 3.6 days)	There were no significant differences in intake of micronutrients

This table presents examples of published studies from resource-poor countries comparing micronutrient intakes of women and men. It shows a general pattern of larger differences in micronutrient intakes between men and women in South Asia than in Sub-Saharan Africa and Latin America

[a]The mean adequacy ratio (MAR) was calculated as the mean of nutrient adequacy ratios (NARs) for the intake of energy and nine nutrients, each truncated at one. The NAR for a given nutrient was calculated as the ratio of a subject's intake to the current recommended allowance. Thus, maximum score was 1

[b]Global nutrient adequacy was calculated by adding the ratio of the subjects' nutrient intakes to the Estimated Average Requirement for the nutrient, each truncated at one, for four nutrients. Thus, maximum score was 4

Underlying Causes of Gender Differences in Food Intake

Women are disadvantaged in many ways in all countries in the world, as is reflected in the United Nation's Gender Index [28]. This index comprises indicators related to women's reproductive health, and indicators of differences in empowerment and labour market access by men and women. An ideal figure of zero would indicate no gender difference. Whereas Europe has an average score of 31 %, South Asia and Sub-Saharan Africa are at the bottom with average scores of 60 % and 61 %, respectively. Sweden, the Netherlands and Denmark have the lowest scores (5–6 %) and Yemen, Chad and Niger the highest scores (72–77 %).

In her overview of intra-household allocation of food and health care, Messer claims that male-biased "investment" is common in patrilineal, patrilocal societies where inheritance is from father to son and sons establish homes close to or within the father's family [20]. In particular in South Asia, strong gender-based differentials in status are reported [29–31]. Research from India finds that women are often regarded as economic burdens [32]. There is a strong preference for sons in India because they are expected to care for aging parents. Daughters, on the other hand, leave their parents once they are married, and their families incur high dowry costs. Discrimination in allocation of food is consistent with broader gender bias, at least in some South Asian societies [20].

Also in South Asia, in Nepal Gittelsohn et al. explained the observed pattern of biased intra-household food distribution as a reflection of a complex food belief system guiding food selection, preparation, serving and consumption. This system also included normative patterns of favouring/disfavouring household members based on their age and gender. Beginning in early adulthood, adult females were disfavoured [22].

However Messer [20] also suggests that food or care may be apportioned to individuals in the household to meet outcome goals such as income generation, healthiness, social insurance or security. In a study from Burkina Faso, Sauerborn et al. found that sick children were allocated fewer resources for health care compared to that of sick adults [33]. There were no gender differences observed—neither for children nor adults. While children were nonproductive, women were shown to contribute as much to household production as men, and their health appeared to be valued equally with that of men. As a consequence, according to Messer, intra-household food allocations are non-egalitarian; they tend to be biased categorically toward those perceived to be more physically and economically active and therefore to have greater nutritional needs, and not towards those who are most vulnerable nutritionally [20].

Two studies from Latin America are consistent with this view of the primacy of economic roles. In Guatemala, Engle and Nieves studied intra-household distribution to examine two hypothesized patterns: a "Contributors Rule", where individuals considered in the culture to have higher economic value would receive a higher percentage of the family's food; and a "Needs Rule", in which those considered to have a greater need (but not contribution) would receive a higher percentage of the family's food [34]. Findings showed no gender or age differences in nutrient adequacy scores, however, wage earners and heads of households (whether male or female) received a higher proportion of energy and/or protein as compared to the rest of the household. Engle and Nieves concluded that observed patterns of intra-household distribution were better explained by the "Contributors Rule". Berti et al. also found no differences in food intake between men and women in the Bolivian Andes [21]. The authors speculated that rather than reflecting an idea of equity, this related to the economic role of women, which involved heavy physical work [21].

How Does Economic Vulnerability Effect Women's Diet Quality and Micronutrient Intake?

Socioeconomic Status, Food Security and Micronutrient Intake

We previously documented widespread inadequacies in micronutrient intakes among women living in resource-poor settings [10]. These results were confirmed by another study from Burkina Faso, Mali, Mozambique, Bangladesh and the Philippines [35].

Economic vulnerability as assessed by socioeconomic status has been shown to have strong links with diet quality and micronutrient intakes in developing countries. Studies comparing intakes across socioeconomic groups find, in general, that diet quality and micronutrient adequacy increase with increasing socioeconomic status [36]. For example, the Mexican National Health and Nutrition Survey finds a significantly increasing probability of adequacy with increasing socioeconomic status for all micronutrients tested [37]. This has also been found in studies from Africa [14, 38, 39] and Asia [40]. Dietary diversity has also repeatedly been shown to have a positive association with socioeconomic status [41–44]. However, Gittelsohn et al. noted that in Nepal, whereas wealthier households do better nutritionally than poorer households, higher castes eat a decreased variety of food and run a greater risk of certain nutrient deficiencies [22].

The relationship between poverty and diet quality is mediated by food security, in its broadest sense. According to the definition adopted at the World Food Summit in 1996 [45], food security encompasses "physical and economic access to sufficient, safe, and nutritious food to meet … dietary needs and food preferences for an active and healthy life". Thus, food security requires access not only to sufficient energy, but also to foods that will provide micronutrient adequacy.

Previously, most diet-based indicators of food insecurity estimated energy adequacy, but not micronutrient adequacy. However, energy is relatively cheap to obtain compared to micronutrients which require the consumption of animal foods and/or fruits and vegetables [46, 47]. Bouis et al. showed how increases in staple food prices (mainly rice) in Bangladesh led to a slight decrease in energy intake (5–15 %) but a much larger reduction in iron intake (10–30 %) [47]. They hypothesize that poor people adjust to increasing food prices by reducing non-staple food expenditures, and consequently reducing their diet quality and micronutrient intake. The priority given to purchase of staple foods reflects both the need for assuring energy intake and the fact that non-staple foods typically are more expensive.

High levels of concurrent overweight and micronutrient deficiency in the form of anaemia have been described in Mexico, Egypt and Peru [48], and anaemia was more prevalent in lower socioeconomic groups. This might reflect a situation in which food insecurity in countries in economic transition is increasingly accompanied by overconsumption of energy in the form of cheap oil and refined products, leading to concurrent obesity and micronutrient deficiencies. This underscores the importance of considering diet quality—not just energy sufficiency—when assessing food security [46, 47].

Gender and Food Insecurity

The body of evidence presented above shows that women are at high risk of inadequate micronutrient intake during economic stress, particularly when they are disadvantaged in intra-household allocation of nutrient-dense foods. Regardless of allocation, however, in poor households, a further drop in diet quality will result in increased risks of micronutrient inadequacy for women.

Note also that intra-household gender differences are not necessarily exacerbated by poverty. Filmer et al. explored the relationship between per capita income and gender disparity and found no relationship in either South Asian or non-South Asian contexts [49]. They conclude that low income is not one of the explanations of the high levels of gender disparity in South Asia. From a review of Indian case studies, Miller comes to similar conclusions for India [50]: "Daughter discrimination does not characterize the poor, anywhere in India. If anything, greater disparities and malnutrition are found among propertied groups and among the more educated".

Besides inadequate diet quality, poverty and food insecurity may affect women disproportionally in several other ways [46]. Women are usually the primary caretakers of children and play a main role in obtaining and preparing food. In times of crises, they may carry a heavier burden than male household members. This often includes sacrificing their own food for their family, taking up extra work and even high-risk strategies to secure the food intake of the family. For example, in Indonesia mothers buffered the effects of an economic crisis on children by eating less, but at the expense of their own nutritional status [51].

Conclusions

This chapter documents that women are physiologically vulnerable to inadequate micronutrient intakes; they require a more nutrient-dense diet (nutrients per 100 kcal) due to higher needs and (usually) lower food intakes than men. Although documentation is scarce, there is evidence that in many settings, women have lower or less adequate micronutrient intakes than men. These gender differences in micronutrient intakes or adequacy are most pronounced in South Asian countries, and are least prominent in studies from South America; African studies show varying results. Studies on gender bias in intra-household food allocation generally confirm the same pattern of differences between continents. Biased allocation of food may be cultural, linked to a lower status of women, and/or it may be linked to economic roles, with more food allocated to household members based on actual or perceived economic contributions to the household. Both diet quality and micronutrient intakes vary with socioeconomic status in developing countries, and food security is an important determinant of diet quality. It is important to note that available evidence, though limited, suggests that gender differences in micronutrient intake are not exacerbated by poverty. However, globally, and regardless of the presence or absence of biased allocation of resources, poor women are very vulnerable to micronutrient deficiencies associated with low quality diets in combination with high needs. Risks of inadequacy increase when households are food insecure, whether due to seasonal shortages, economic shocks or other household crises.

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References

1. Bartley KA, Underwood BA, Deckelbaum RJ. A life cycle micronutrient perspective for women's health. Am J Clin Nutr. 2005;81:1188S–93.
2. Allen LH. Multiple micronutrients in pregnancy and lactation: an overview. Am J Clin Nutr. 2005;81: 1206S–12.
3. Murphy SP, Poos MI. Dietary Reference Intakes: summary of applications in dietary assessment. Public Health Nutr. 2002;5:843–9.
4. IOM (Institute of Medicine). Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington DC: National Academy Press; 2000.
5. FAO/WHO/UNU. Human energy requirement. Report of a Joint FAO/WHO/UNU Expert Consultation, Rome, Italy, 17–24 Oct 2001. Rome: FAO/WHO/UNU; 2001.
6. Dufour DL, Reina JC, Spurr GB. Food and macronutrient intake of economically disadvantaged pregnant women in Colombia. Am J Hum Biol. 1999;11: 753–62.
7. Hutter I. Reduction of food intake during pregnancy in rural south India. Trop Med Int Health. 1996;1: 399–405.
8. Huybregts LF, Roberfroid DA, Kolsteren PW, Van Camp JH. Dietary behaviour, food and nutrient intake of pregnant women in a rural community in Burkina Faso. Matern Child Nutr. 2009;5:211–22.
9. Prentice AM, Whitehead RG, Roberts SB, Paul AA. Long-term energy balance in child-bearing Gambian women. Am J Clin Nutr. 1981;34:2790–9.
10. Torheim LE, Ferguson EL, Penrose K, Arimond M. Women in resource-poor settings are at risk of inadequate intakes of multiple micronutrients. J Nutr. 2010;140:2051S–8.
11. Black RE, Allen LH, Bhutta ZA, et al. Maternal and child undernutrition: global and regional exposures and health consequences. Lancet. 2008;371:243–60.
12. Christian P, Stewart CP. Maternal micronutrient deficiency, fetal development, and the risk of chronic disease. J Nutr. 2010;140:437–45.
13. Webb P, Nishida C, Darnton-Hill I. Age and gender as factors in the distribution of global micronutrient deficiencies. Nutr Rev. 2007;65:233–45.
14. Torheim LE, Ouattara F, Diarra MM, et al. Nutrient adequacy and dietary diversity in rural Mali: association and determinants. Eur J Clin Nutr. 2004;58: 594–604.
15. Haddad L, Peña C, Nishida C, Quisumbing A, Slack A. Food security and nutrition implications of intrahousehold bias: a review of literature. Washington DC: International Food Policy Research Institute; 1996.
16. Graham MA. Food allocation in rural Peruvian households: concepts and behavior regarding children. Soc Sci Med. 1997;44:1697–709.
17. Gittelsohn J, Thapa M, Landman LT. Cultural factors, caloric intake and micronutrient sufficiency in rural Nepali households. Soc Sci Med. 1997;44:1739–49.
18. Sudo N, Sekiyama M, Maharjan M, Ohtsuka R. Gender differences in dietary intake among adults of Hindu communities in lowland Nepal: assessment of portion sizes and food consumption frequencies. Eur J Clin Nutr. 2006;60:469–77.
19. Sudo N, Sekiyama M, Watanabe C, Bokul AT, Ohtsuka R. Gender differences in food and energy intake among adult villagers in northwestern Bangladesh: a food frequency questionnaire survey. Int J Food Sci Nutr. 2004;55:499–509.
20. Messer E. Intra-household allocation of food and health care: current findings and understandings—introduction. Soc Sci Med. 1997;44:1675–84.
21. Berti PR, Jones AD, Cruz Y, Larrea S, Borja R, Sherwood S. Assessment and characterization of the diet of an isolated population in the Bolivian Andes. Am J Hum Biol. 2010;22:741–9.
22. Gittelsohn J, Thapa M, Landman LT. Opening the box: intrahousehold food allocation in rural Nepal. Soc Sci Med. 1991;33:1141–54.
23. Hudson GJ. Food intake in a West-African village. Estimation of food intake from a shared bowl. Br J Nutr. 1995;73:551–69.
24. van Liere MJ, Brouwer ID, den Hartog AP. A food ethnography of the Otammari in north-western Benin: a systematic approach. Ecol Food Nutr. 1992;34: 293–310.
25. Villa KM, Barrett CB, Just DR. Whose fast and whose feast? Intrahousehold asymmetries in dietary diversity response among East African pastoralists. Am J Agr Econ. 2011;93:1062–81.
26. Belachew T, Hadley C, Lindstrom D, et al. Gender differences in food insecurity and morbidity among adolescents in southwest Ethiopia. Pediatrics. 2010; 127:e398–405.
27. Nube M, Van Den Boom GJ. Gender and adult undernutrition in developing countries. Ann Hum Biol. 2003;30:520–37.
28. United Nations Development Programme. Human Development Report 2011. Sustainability and equity: a better future for all. New York: UNDP; 2011.
29. Osmani S, Sen A. The hidden penalties of gender inequality: fetal origins of ill-health. Econ Hum Biol. 2003;1:105–21.
30. Winkvist A, Akhtar HZ. God should give daughters to rich families only: attitudes towards childbearing among low-income women in Punjab. Pak Soc Sci Med. 2000;51:73–81.
31. Haddad L. Women's status: levels, determinants, consequences for malnutrition, interventions, and policy. Asian Dev Rev. 1999;17:96–131.
32. Saha UC, Saha KB. A trend in women's health in India—what has been achieved and what can be done. Rural Remote Health. 2010;10:1260.
33. Sauerborn R, Berman P, Nougtara A. Age bias, but no gender bias, in the intra-household resource allocation

for health care in rural Burkina Faso. Health Trans Rev. 1996;6:131–45.

34. Engle PL, Nieves I. Intra-household food distribution among Guatemalan families in a supplementary feeding program: behavior patterns. Soc Sci Med. 1993; 36:1605–12.
35. Arimond M, Wiesmann D, Becquey E, et al. Simple food group diversity indicators predict micronutrient adequacy of women's diets in five diverse, resource-poor settings. J Nutr. 2010;140:2059S–69.
36. Vlismas K, Stavrinos V, Panagiotakos DB. Socio-economic status, dietary habits and health-related outcomes in various parts of the world: a review. Cent Eur J Public Health. 2009;17:55–63.
37. Barquera S, Hernandez-Barrera L, Campos-Nonato I, et al. Energy and nutrient consumption in adults: analysis of the Mexican National Health and Nutrition Survey 2006. Salud Publica Mex. 2009; 51:S562–73.
38. MacIntyre U, Kruger H, Venter C, Vorster H. Dietary intakes of an African population in different stages of transition in the North West Province, South Africa: the THUSA study. Nutr Res. 2002;22:239–56.
39. Kamau-Mbuthia E, Elmadfa I. Diet quality of pregnant women attending an antenatal clinic in Nakuru, Kenya. Ann Nutr Metab. 2007;51:324–30.
40. Hartini TN, Winkvist A, Lindholm L, et al. Nutrient intake and iron status of urban poor and rural poor without access to rice fields are affected by the emerging economic crisis: the case of pregnant Indonesian women. Eur J Clin Nutr. 2003;57: 654–66.
41. Clausen T, Charlton KE, Gobotswang KS, Holmboe-Ottesen G. Predictors of food variety and dietary diversity among older persons in Botswana. Nutrition. 2005;21:86–95.
42. Hatloy A, Hallund J, Diarra MM, Oshaug A. Food variety, socioeconomic status and nutritional status in urban and rural areas in Koutiala (Mali). Public Health Nutr. 2000;3:57–65.
43. Ponce X, Ramirez E, Delisle H. A more diversified diet among Mexican men may also be more atherogenic. J Nutr. 2006;136:2921–7.
44. Savy M, Martin-Prével Y, Sawadogo P, Kameli Y, Delpeuch F. Use of variety/diversity scores for diet quality measurement: relation with nutritional status of women in a rural area in Burkina Faso. Eur J Clin Nutr. 2005;59:703–16.
45. Food and Agriculture Organization of the United Nations. Rome declaration on world food security and world food summit plan of action. World Food Summit 13–17 November 1996. Rome: FAO; 1996.
46. Ivers LC, Cullen KA. Food insecurity: special considerations for women. Am J Clin Nutr. 2011;94:1740S–4.
47. Buois HE, Eozenou P, Rahman A. Food prices, household income, and resource allocation: socioeconomic perspectives on their effects on dietary quality and nutritional status. Food Nutr Bull. 2011;32: S14–23.
48. Eckhardt CL, Torheim LE, Monterrubio E, Barquera S, Ruel MT. The overlap of overweight and anaemia among women in three countries undergoing the nutrition transition. Eur J Clin Nutr. 2008;62: 238–46.
49. Filmer D, King EM, Pritchett L. Gender disparity in South Asia: comparisons between and within countries. Washington DC: World Bank; 1998.
50. Miller BD. Social class, gender and intrahousehold food allocations to children in South Asia. Soc Sci Med. 1997;44:1685–95.
51. Block SA, Kiess L, Webb P, et al. Macro shocks and micro outcomes: child nutrition during Indonesia's crisis. Econ Hum Biol. 2004;2:21–44.
52. Blaney S, Beaudry M, Latham M, Thibault M. Nutritional status and dietary adequacy in rural communities of a protected area in Gabon. Public Health Nutr. 2009;12:1946–59.
53. Venkaiah K, Damayanti K, Nayak MU, Vijayaraghavan K. Diet and nutritional status of rural adolescents in India. Eur J Clin Nutr. 2002;56:1119–25.
54. Chiplonkars SA, Agte VV, Tarwadi KV, Paknikar KM, Diwate UP. Micronutrient deficiencies as predisposing factors for hypertension in lacto-vegetarian Indian adults. J Am Coll Nutr. 2004;223: 239–47.
55. Berti PR, Leonard WR. Demographic and socioeconomic determinants of variation in food and nutrient intake in an Andean community. Am J Phys Anthropol. 1998;105:407–17.

Part III

Life Stages: Children and Seniors

10 Interventions to Improve Dietary Quality in Children

Melanie Hingle

Key Points

- Dietary patterns research of children and adolescents demonstrates relationships between diet quality and specific behaviors related to food consumption and adiposity.
- Improvement in diet quality was not an explicit intervention goal of any of the included studies.
- The majority of intervention studies designed to improve diet quality in children take place in school-based settings.
- Attempts to intervene on diet quality of children and adolescents have been met with extremely modest effects.
- Currently, it is not possible to determine whether observed behavioral changes within intervention studies are attributable to specific intervention components or to other unidentified factor(s).

Keywords

Diet quality • Children • Adolescents • Interventions • Disease prevention • Health promotion • Fruit and vegetable • Nutrient dense

Abbreviations

BRFSS	Behavioral risk factor surveillance survey
C	Control
DGA	Dietary Guidelines for Americans
f/u	Follow-up
FJV	Fruit, juice, and vegetables
FV	Fruit and vegetables
HBM	Health Behavior Model
I	Intervention
PA	Physical activity
SCT	Social Cognitive Theory
SEM	Social Ecological Model
SLT	Social Learning Theory
svgs	Servings
TTM	Trans-theoretical model of behavior change
yr	Year
yrs	Years

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Introduction

Nutrient intake recommendations have been established to provide guidelines for nourishment and health in humans. Although obtaining adequate amounts of nutrients to meet basic needs remains a challenge for a significant proportion of the world's population [1], a growing number of individuals are facing a different type of nutritional challenge—selected nutrient inadequacy combined with too *many* calories. This dietary profile is often referred to as "overnutrition," and when prolonged, can lead to the development of overweight and obesity.

As obesity reaches epidemic levels around the world [2], nutrition and health professionals seek methods by which to characterize and promote nutrient-dense diets that at the same time, support a healthy weight. The construct of *diet quality* represents one such way to do so. Diet quality implies a qualitative measure, however, this construct has been used to quantitatively evaluate different aspects of dietary intake, including: adequacy of nutrient intake (i.e., compliance with dietary recommendations), nutrient density (ratio of total nutrient content in grams to total energy content in kilocalories or joules), dietary energy density (available dietary energy per unit of food) [3], dietary variety (number of different food items consumed over a given period) [4], and dietary diversity (number of different food items within same food group).

Relationships between diet quality and disease outcomes. To health professionals who wish to intervene on health and weight in specific populations, the most useful diet quality indexes are those that may be linked to specific health outcomes. In adults, a high quality diet (in general, consisting of high vegetable, fruit and whole grain intake, moderate consumption of mono- and polyunsaturated fatty acids, and low-fat dairy and no or lean animal meat intake) has been associated with lower risk for coronary heart disease [5], cancer [6], ischemic stroke [7], Type 2 diabetes mellitus [8], and reduced adiposity gain [9].

In children, relationships between diet and disease are less clear, in part, because diet-mediated chronic diseases (e.g., Type 2 diabetes or cardiovascular disease) typically do not manifest during childhood. However, there are cross-sectional and longitudinal data that provide insight into the typical dietary patterns of children and adolescents, and importantly, demonstrate relationships between diet quality and specific behaviors related to food consumption, as well as intermediate health indicators such as biomarkers (e.g., blood glucose, insulin) and weight status. These relationships provide a foundation and rationale for interventions designed to impact the diet quality of children.

Trends in food consumption and correlates of diet quality in children. Child and adolescent food and beverage choices have shifted substantially over the past 3 decades [10]. Arguably, some of the most influential factors behind this shift in dietary choices have been the increasing availability of high-calorie, low-nutrient-dense foods and the number of foods prepared and eaten away from the home [11]. Between the late 1970s and the late 1990s, the proportion of US adolescents' food consumption from restaurants tripled (from 6.5 to 19.3 %) [12]. Data from the US National Health and Nutrition Examination Survey (NHANES) in 2003–2006 indicated that major contributors to the diets of children and adolescents (ages 2–18 years) were grain desserts, pizza, and soft drinks. In this same sample of youth, 40 % of total daily calories (~800 kcal/day) consumed were comprised of these "empty calories" (i.e., high-calorie, low-nutrient-dense foods)—half of these calories were from solid fats (consumed as part of whole milk products, pizza, and grain/dairy desserts), and the other half from added sugars (primarily from soft drinks and sugar-sweetened fruit drinks) [13]. At the same time, over 80 % of American children and adolescents did not meet recommended amounts of fruit and vegetable consumption [14]. Taken together, these data highlight nutrients and food groups in need of improvement.

In order to guide the behavioral targets of dietary interventions for this age group, it is important to identify specific behaviors associated with diet quality in children. Ostensibly, interventions designed to impact diet quality should when possible, focus on encouraging behaviors that have the most potential to impact diet quality while discouraging those that negatively impact diet quality. Significant correlates of diet quality in children include family meals (+diet quality) [15], consumption of fast foods or foods away from home (−diet quality) [16], availability of fruits and vegetables in the home (+diet quality) [17, 18], snacking frequency (−diet quality for sugar and fats but +diet quality for fruit) [19], and meal skipping (−diet quality) [20].

A closer look at published studies of child dietary interventions is warranted to:

1. Examine how diet quality has been defined and operationalized within the context of interventions targeting the dietary behavior of children and adolescents.
2. Identify successful elements that exist across interventions.
3. Suggest additional research, evaluation, tools, or data that are needed to inform the design of effective diet quality interventions for this population.

Interventions to Improve Diet Quality in Children

The child dietary intervention literature is expansive. However, diet quality per se has not been a review topic, perhaps because interventions that seek to impact diet behaviors by design improve diet quality.

A literature search was performed using electronic databases such as PubMed, ERIC, PsychINFO, and Google Scholar to identify interventions that sought to improve diet quality in children. The search was limited to primary research articles, systematic reviews, guidelines and recommendations and policy reports published up to December 2011 that reported on dietary interventions that enrolled children and adolescents (2–18 years). This review was focused to include only human, English, clinical trial, or randomized controlled trials conducted at schools or in the community, recognizing that other types of interventions (e.g., one-on-one counseling, or broad public health or environmental campaigns) are important but beyond the scope of this chapter. Studies were limited to those whose primary aim was to increase fruit, vegetable, and whole grain intake, and/or reduce the intake of high-calorie, low-nutrient foods. Studies were also limited to those which employed designs that could be categorized as "large pragmatic trials," as these types of interventions are implemented with relatively unselected participants and under flexible conditions. In theory, these studies should provide the most practical, generalizable information about behavior change strategies that result in improved diet quality in children.

Summary of Findings

A total of 19 studies met the inclusion criteria. The majority of studies enrolled youth between 6 and 12 years of age (Table 10.1).

Study outcomes. Twelve of 19 interventions focused only on dietary outcomes [21–33] and seven on diet plus physical activity or media screen time use [34–40]. Eleven studies had a positive impact on diet [22, 25, 26, 29–32, 34, 35, 38, 40]. These were very modest effects, ranging from increases of 0.3–0.94 servings of fruit and vegetables daily. Four studies reported mixed effects—that is, the intervention had a greater impact on girls but not boys [21, 23, 36], children who started out with lowest intakes of fruits/vegetables made the most gains (compared to those who already met guidelines) [24], and, normal weight but not overweight children improved [33]. Three studies reported no significant effects on dietary outcomes [27, 37, 39]. No studies reported detrimental effects on diet quality.

Definitions of diet quality. Improvement in diet quality was not an explicit intervention goal of any of the included studies. For the majority of these studies, disease prevention (e.g., prevention of cancer, obesity, CVD) was the explicit goal, and to achieve this, investigators typically promoted

Table 10.1 Summary of studies

Author, program, location	Population	Intervention	Behavioral targets and theory or constructs	Dietary measures and time points	Findings
Sahota et al., APPLES, Leeds, UK	$N=636$, 8–10-year-olds 10 schools $I=314$, $C=322$ RCT at school level	*I*: 1-year APPLES health promotion program: teaching training, school meal modification, curriculum development, PE, tuck shops, playground activities; involvement of parents, teachers, catering staff *C*: Standard of care	↓Fat and sugar; ↑FV; ↑PA *Health promoting schools* (population-based obesity prevention approach)	24-h recall and 3-day diet diary at baseline, 12 months	*I*: 0.3 svg (95 % CI 0.2–0.4) ↑V intake post-study than *C*
Friel et al., NEAPS, Ireland	$N=821$, 8–10-year-olds 8 schools $I=453$, $C=368$ Quasi-experimental, randomization of schools	Pilot dietary education program *I*: 20 sessions × 10 weeks worksheets, homework and an exercise regimen; "parent packs" mailed home to parents with assignments *C*: None	↑FV Not specified	5-day food diary at baseline and 3 months	No significant change in *C* Small ↑ in number of *I* children consuming recommended amount of FV
Cullen et al., Girl Scouts Eat 5, USA	$N=300$ 22 Girl Scout troops randomized pre-, posttest *C* group	4 weekly sessions *I*: ↑FV exposure, preparation skills, knowledge, and skills in self-evaluation, self-monitoring, goal setting and problem solving Parent information sheets sent home and parents encouraged to promote FV consumption *C*: None	↑FV SCT	Food recognition form at baseline, 3 months; 12-item FFQ Pre-post questionnaire assessing determinants of food behavior	Baseline: 20 % girls consumed 5 + FV svg/day; 17 % reported 0 svg/day *I*: Significant differences between pre- and posttest ($p<0.002$); mean FV intake of *I* girls was 3.02 (SD 2.21) at baseline and 3.39 (SD 1.93) at posttest; mean FV intake of *C* girls was 2.21 (SD 1.96) and posttest 2.06 (SD 1.71) 96 not available for posttest or follow-up testing 3-month f/u shows no maintenance of effect

Perry et al., 5-a-Day-Power-Plus Program, USA	*N* = 1,612 Fourth and fifth grades *N* = 657 selected for random dietary measurements	2 consecutive school years *I*: 16 classroom sessions of 40–45 min twice weekly for 8 weeks: skill-building, problem solving, snack preparation, taste-testing, comic books, team competitions; parental involvement/education; school food service changes; industry involvement and support *C*: Delayed *I*	↑FV SLT	Group-administered health behavior questionnaires measuring a variety of factors related to FV intake pre-post; random sample self-completed 24-h food record; observation of students in lunch room	*I* students reported ↑ mean intake of FV at f/u (1.53 vs. 1.06 svg, $p < 0.01$); greater effect in girls in *I* compared to *C*; health behavior questionnaires suggested greater perceived teacher support for eating FV, more reports of asking, more reported daily FV
Foerster et al., 5-a-Day Power Play! California, USA	*N* = 2,684 cases matched pre- and post-study 49 schools and 151 classrooms Fourth and fifth grade *I*1: 19 schools *I*2: 15 schools *C*: 15 *C* schools	8 weeks *I*1: Power play! activities conducted in school *I*2: Power play! activities conducted in schools, community youth organizations, supermarkets, farmers' markets, and mass media *C*: None	↑FV Social marketing initiative, resiliency theory, SLC, reciprocal determinism	24-h recall self-reported administered pre-post (California children's food survey)	Both *I* sites reported significant ↑FV intake compared to *C*, but not to one another: *I*2: ↑0.4 svg from 2.9 to 3.3 *I*1: ↑0.2 svg from 2.7 to 2.9 *C*: consumption ↓
Baranowski et al., "Gimme 5" fruit, juice, and vegetables for fun and health, USA	*N* = 1,253 Fourth and fifth grades 16 schools RCT	6-week, 12-session program *I*: Gimme 5 curriculum—teachers provided handouts, posters, worksheets, newsletters, videos Point-of-purchase education conducted at shops near school that parents frequented *C*: None	↑FV SCT	7-day food record each year for 3 years	↑ Mean consumption occurred in the 2 lowest quintile groups and were highest in the *I* group (lowest quintile: +0.47 svgs and +0.82 svgs for *C* and *I* groups, respectively); ↓ occurred in top three quintiles (highest quintile: −1.59 and −0.88 svgs for *C* and *I*, respectively)

(continued)

Table 10.1 (continued)

Author, program, location	Population	Intervention	Behavioral targets and theory or constructs	Dietary measures and time points	Findings
Perry et al., 5-A-Day Cafeteria Power Plus Project, USA	$N = 1{,}668$ First to third grades 26 elementary schools randomized to intervention or control groups (35 students per grade and per school)	Two consecutive school years *I*: Daily activities ↑availability of FV, emphasizing changes in lunch line and school snack cart and special events *C*: Delayed *I*	↑FV SCT and HBM	Direct observation of FV consumption at lunch by trained observers; recording of FV eaten from school snack cart Baseline and 2-year f/u	After 2 years, significant difference between groups for the intake of F without J (*I*: 0.37 vs. *C* 0.21 svg, $p < 0.01$), F with J (*I*: 0.79 vs. 0.63, $p = 0.01$), FV without potatoes and J (0.64 vs. 0.50, $p = 0.02$), and FV without potatoes, with J (1.06 vs. 0.92, $p = 0.03$) *I* schools had significantly ↑ encouragement to eat FV by food service staff as well as FV on the lunch line ($p < 0.01$) Overall change was F rather than V consumption
Gortmaker et al., Eat Well and Keep Moving, USA	$N = 2{,}103$ *I*: 6 public elementary schools in Baltimore *C*: 8 matched schools Quasi-experimental field trial	Two consecutive school years *I*: 13 lessons delivered by teachers and integrated into math, science, language arts, social studies classes, with links to school food service, PE, teacher and other staff wellness programs, families and classroom-based campaigns *C*: Standard of care	↑FV and PA; ↓ foods high in total and sat fat; ↓TV viewing SCT and behavioral choice	FFQ administered to all students Stratified random subsample of 336 students in Grade 5 completed repeated 24-h recall interviews post-intervention	↑FV consumption (0.36 svgs, 95 % CI 0.10–0.62, $p = 0.01$) (equivalent to an increase of 0.73 svgs/day)

Reynolds et al., High 5 Project, USA	$N=1,698$ families Fourth and fifth graders (69 % of approached) 28 schools pair-matched based on ethnic composition and proportion of students receiving free or reduced price meals	Two consecutive school years *I*: 14-lesson curriculum taught by nine coordinators employed by the High-5 project Curriculum delivered on 3 consecutive day/week, 30–45 min lessons: modeling, self-monitoring, problem solving, reinforcement, taste-testing, other methods; parent component; food service component *C*: Standard of care	↑FV SCT	One 24-h recall Cafeteria observation (sample of 425 students were observed during school lunch) Parent FFQ (FV items from Health Habits and History Questionnaire) 2-year follow-up	No difference at baseline in F, V, or FV *I* group had higher intakes at 1 year (F 1.71 vs. 0.83, $p<0.0001$; V 1.84 vs. 1.15, $p<0.0001$; FV 3.96 vs. 2.26, $p<0.0001$), and at 2 years (fruit 1.21 vs. 0.65, $p<0.0001$; V 1.6 vs. 1.25, $p<0.09$; FV 3.2 vs. 2.21, $p<0.0001$)
Birnbaum et al., Lytle et al., Teens Eating for Energy and Nutrition at School, USA	$N=3,503$ 16 schools, ≥20 % of students approved for a free and reduce-priced lunch program; schools pair-matched based on proportion of seventh graders expected to receive all school-based intervention components	Two consecutive school years *I*1: School environment only, promotion of FV and low-fat snacks *I*2: *I*1 + classroom curriculum = 10 sessions and 3 "parent packs" activities and intervention messages *I*3: *I*2 + peer leaders trained to help teachers to deliver curriculum *C*: None	↑FV; ↓fat Multicomponent interventions	BRFSS—FV items 2-year follow-up	No significant changes in FV intakes
Gortmaker et al., Planet Health, Boston, MA, USA	$N=1,560$ Grades 6–8 Ten public schools from four communities in Boston, MA randomly assigned to either *I* ($n=5$) or *C* ($n=5$)	*I*: 32 lessons total in Year 1 and year 2 Teacher training workshops, classroom lessons, physical education materials, wellness sessions and fitness funds *C*: None	↓Total dietary fat; ↑FV, PA; decreased TV viewing SCT	Food and activity survey and youth FFQ 2-year follow-up	Statistically significant changes in FV intake occurred in girls only (0.32 svgs/day, 95 % CI, 0.14–0.50 svgs/day, $p=0.003$)

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Table 10.1 (continued)

Author, program, location	Population	Intervention	Behavioral targets and theory or constructs	Dietary measures and time points	Findings
Nicklas et al., Gimme 5, USA	*N*=2,213 12 schools New Orleans, LA RCT (6 *I*, 6 *C*)	4-year intervention delivered Sept 1994–April 1996 *I*: School-wide media marketing campaign, classroom activities, school meal modification, and parental involvement *C*: None	↑FV Poly-theoretical	Baseline to intervention end (3 years) in same cohort of students 3-year follow-up (9th to 12th grade)	FV intake significantly ↑ in *I* schools compared to *C* schools in year 1 and year 2, but not year 3 *I* group reported 14 % ↑ (+0.37 svgs) in FV consumption after 2 years of *I* from 2.63 svgs at baseline to 3.00 svgs at 2 years. In year 3, consumption remained stable in *I* group with ↑ in *C* group (so no difference between the 2 at year 3)
Perry et al., CATCH, USA	*N*=1,186 among 5,106 students in original CATCH study (i.e., those who agreed to participate in 24-h recall)	96 schools in four states from 1991 to 1994 *I*: school food service, physical education, classroom curricula, and parental involvement *C*: None	↓Dietary fats and dietary sodium; ↑physical activity; ↑FV None specified	Subsample of 5,106 students participated in single 24-h recalls at baseline and 1,186 of these students were followed-up 2 years later	No significant differences between conditions in FV consumption at f/u Baseline: 2.12 svgs of F, and 2 svgs of V in 24 h At f/u, *I* group $n=707$ consumed 4.17 svgs (SE 0.19) of FV, 2.25 svgs (SE 0.16) of F, 1.90 (SE 0.10) of V *C* group $n=479$ consumed 4.10 svgs (SE 0.23) of FV, 2.20 svgs (SE 0.19) of F; and 1.89 svgs (SE 0.13) of V
Thompson et al., Boy Scout 5-a-Day Badge, USA	*N*=473 10–14-year-old Boy Scouts 42 troops in Houston >68 % non-Hispanic White RCT	9 weeks with f/u measures *I*: 5-a-Day badge achievement with nine activities including group and internet components *C*: Same *I* structure but focused on PA	↑FJV SCT	Modified FFQ with 21 F and 17 V line items measured at baseline, 9 weeks, and 6 months	Group by time interaction for FJ intake $p=0.03$; mean ↑ of 0.94 ± 0.0 svgs/day, *I*, 0.56 ± 0.0 svgs/day ↓, *C* Low-fat V intake did not show change or differ by group Not sustained over time

DiNoia et al., Computer-mediated intervention, USA	*N* = 507 African Americans 11–14 years-old 27 youth service organizations in NE states 61 % female; 85 % non-Hispanic White RCT	4 weekly sessions *I*: Computer-based, consisting of 4, 30-min CD-ROM lessons tailored for individual stage of change *C*: None	↑FV TTM	Single item: "About how many servings of fruits and vegetables do you usually eat each day" at baseline and 2 week (post-intervention)	FV svgs at 2 weeks *I*, 3.25 svgs; *C*, 2.46 svgs Post-intervention showed a +0.9 svg/day increase with no change in *C* group
Mauriello et al., Health in Motion, USA	*N* = 1,800 Eight high schools in RI, MA, NY, TN, USA 50.8 % female; 70.1 % non-Hispanic White, 20.7 % African American School is unit of randomization; assigned, 2:1 (*I*:*C*)	Three intervention sessions at baseline, 1 month and 2 months *I*: Stage-matched and tailored feedback *C*: None	↑FV (5-a-day); ↑PA; Limited TV viewing TTM	Single item: "How many servings of FV do you usually eat each day?" Assessed at 2, 6 and 12 months	*I* group reported consuming ↑FV compared to *C* *I*: 2.25 at baseline, 3.86 at 2 months, 3.55 at 6 months, and 3.67 at 12 months *C*: 2.43 at baseline, 3.0 at 2 months, 2.73 at 6 months, and 2.97 at 12 months
Dzewaltowski et al., Healthy Youth Places, USA	*N* = 1,582 Seventh and eighth graders 16 schools 54 % female RCT nested cohort design, school as unit of randomization	Two consecutive school years *I*: environmental change in school lunch and afterschool programs; group goal setting; internet support *C*: None	↑FV; ↑PA SCT	Youth adolescent FFQ measured at baseline, year 1, and year 2	No significant change or difference in or between study groups over time
Haire-Joshu et al., High 5 for Kids, USA	*N* = 1,658 parents and their preschool age children in SE Missouri Group randomized controlled nested cohort design	Four home visits conducted over 6–11 months *I*: Parent educators, home visits (≥5) plus on-site group activities and tailored, mailed newsletters *C*: standard parenting program	↑FV PAT (parent as teachers) training based on SCT and SEM	St. Louis for kids FFQ; intake of 27 FV over past 7 days measured at baseline and post-intervention	Results in FV svgs/day *I*: 4.91 at baseline, 4.92 post-intervention *C*: 4.79 at baseline, 4.74 post-intervention Not significant except subgroup findings wherein normal weight *I* children showed significant ↑ Change in intake correlated with parent intake

(continued)

Table 10.1 (continued)

Author, program, location	Population	Intervention	Behavioral targets and theory or constructs	Dietary measures and time points	Findings
Gentile et al., Switch What You Do, View, and Chew, USA	*N* = 1,323 Third to fifth graders Ten elementary schools in Midwestern states 53 % female RCT school is the unit of randomization	8 months (Oct–May) *I*: Family intervention with school reinforcement—behavioral and environmental strategies *C*: None	↑FV and PA; ↓screen time SEM	FV items from BRFSS—parent report for previous week, child, previous 24 h Measured at baseline, 9 months, and 6 months post-intervention	Child report *I*: 4.9 svgs FV per day (3.9 at baseline, 4.4 ± 0.2 at 9 months) *C*: 4.1 ± 2.9 at baseline, 4.2 ± 0.1 at 9 months Parent report *I*: 25.4 ± 14.1 at baseline; 24.9 ± 0.7 at 9 months *C*: 23.0 ± 12.8 at baseline; 22.6 ± 0.4 at 9 months Parent report suggests post intake lower in *C* than *I* but change not reported

increased fruit and vegetable intake and decreased fat intake [23, 25, 27, 29, 30, 32, 33, 35, 36, 39, 40]. Several other studies focused efforts on helping child participants to meet general nutrition guidelines [21, 24], while still others did not provide a clear rationale for targeting a particular food or food groups [22, 26, 31, 34, 37, 38].

Intervention framework. An a priori theoretical framework or theory-based approach is believed to contribute to the effectiveness of dietary behavior interventions [41]. Theoretical frameworks can help investigators organize their approach and test hypothesized mechanisms through which behavioral changes might take place (e.g., self-efficacy related to healthy eating, or availability/accessibility of healthy foods in the home). Eleven of 19 studies in this review specified a single theoretical framework upon which their intervention was based [21, 24, 26, 29, 30, 32–34, 36–38, 40], while five studies specified multiple theories or approaches [23, 25, 27, 31, 35]. The Social Cognitive Theory was the most frequently mentioned framework, followed by the Social Learning Theory and the Transtheoretical Model. Three studies did not specify any framework.

Location of intervention activities. The majority of studies in this review were conducted or based in a school setting, although 9 of these 14 also included outreach to the home through "parent packs," parent newsletters, and homework assignments that were intended to engage parents in supporting their children in health behavior change [21–23, 27, 29, 31, 34, 35, 39]. The rationale for school-based interventions is simply that children spend a lot of time in school, and interventionists have access to a "captive audience" who are primed to learn—factors that *should* support significant increases in knowledge and perhaps even behavior. While schools are places where youth spend a large part of their time, the home environment exerts significant influence on youth behaviors (i.e., healthy food availability and accessibility and parent role modeling and encouragement) and therefore, youth behavior change, and should be considered as equally important venues for health behavior change interventions.

Delivery of intervention content. In the majority of school-based studies [22–27, 29, 31, 34–37, 39, 40], teachers were trained by research staff to deliver intervention content as a part of classroom curriculum and activities. Three studies used research staff to deliver the intervention directly to children—one of these was in a school setting, and the other two were conducted at Boy Scout and Girl Scout troop meeting locations [21, 29, 32]. In two of the school-based studies, children were guided through intervention materials via tailored software applications, and not supervised by research staff or teachers [30, 40]. Two studies recruited from schools and childcare locations, but bypassed these settings during the intervention to work directly with parents in the home to change family habits and behaviors [33, 38].

Mediators of dietary behavior change. Knowledge is necessary but not sufficient to change dietary behavior [41]. In young children, exposure to foods within a positive social context and healthy eating behavior modeling by peers and adults is important in promoting and sustaining behavior change, while in older children and adolescents, skills such as self-assessment, goal setting and monitoring, and seeking out and creating supportive environments (e.g., increasing availability/accessibility of healthy choices in home) are equally critical. Targeting mediators of dietary behavior such as these has been shown to impact child fruit and vegetable within the context of dietary behavior interventions [42], and have been mentioned as desirable elements of interventions by participants of focus groups [43, 44].

In this review, five studies focused on impacting mediators of dietary change, which included role modeling, self-monitoring, problem solving/reinforcement, taste-testing and preferences, increased awareness, attitudes and knowledge, self-efficacy, and goal setting related to dietary outcomes [29–32, 38]. Although these studies are too few in number to draw any formal conclusions with regard to whether or not targeting mediators results in improved outcomes compared to those that do not include these behavioral targets, it is worth noting that all five of these studies were modestly successful in achieving dietary change.

Duration and frequency of intervention activities and follow-up assessments. Duration of each of the active intervention periods were highly variable, ranging from 4 weeks [21] to 4 years [31]. Several of the school-based studies delivered intervention curriculum spread out over several school semesters or even several years, with curriculum building each year as students progressed through grades. Within the active intervention period, the *frequency* of activities was also highly variable—some programs were very intense, e.g., 20 sessions over 10 weeks [22], while others were more diffuse, e.g., four home visits conducted over 6–11 months [33].

Follow-up measurements (i.e., those conducted beyond the duration of the intervention activities) were reported by only four studies [21, 29, 32, 40], and maintenance of the intervention effect beyond the conclusion of the active intervention occurred only in two of these studies [29, 40].

Methods of dietary assessment. Dietary assessment in children remains a difficult undertaking under the best of circumstances. For a successful study outcome, a valid and reliable instrument or dietary assessment approach is as important as other aspects of the study including reach, implementation, and effectiveness. The lack of observable effects in a large percentage of the child diet intervention literature suggests more work is needed in order to ensure the choice of instruments and approaches reliably captures diet, weight, and behavioral outcomes. Without reliable outcome measurements, the ability to detect a true relationship between participation in the intervention and changes in diet quality is hindered [45]. While selection of a specific dietary assessment approach largely depends on the study design and research question(s), the sheer number and diversity of diet assessment approaches used by investigators remains surprisingly high. Studies included in this review provide a good example of this phenomenon: one study used direct observation to assess dietary behavior [26], two used 24-h recall protocols [25, 39], two used food diaries [22, 24], two used single- or limited-item screeners [30, 38, 40], eight used food frequency questionnaires [21, 23, 27, 31–33, 36, 37], and three used some combination of these methods [29, 34, 35]. Of note, no study used exactly the same instrument or approach to assess dietary intake.

Identifying elements contributing to success. Although several studies reported specific data with regard to process evaluation variables of interest—e.g., fidelity, reach, and dose of the intervention—it was not possible (other than through speculation provided by some of the investigators) to determine whether observed behavioral changes in studies reporting positive outcomes were attributable to specific intervention components, or whether they were due to other unidentified factor(s). In part, this may because the majority of studies targeted multiple and related behaviors (e.g., diet, physical activity, and screen media use) or targeted one behavior using multiple strategies all at once, which made untangling mechanisms of effect difficult.

Process evaluation data were reported by more than two-thirds of studies in this review. In general, these data were intended to capture specific aspects of the intervention implementation including reach and "dose" (i.e., how many people participated, and the extent to which they participated), fidelity to intervention procedures (i.e., how much of intended content was actually delivered to youth), acceptability of materials to the target audience, and any resulting environmental/policy changes (e.g., number of youth reporting point-of-purchase nutrition education). When available, data were only briefly reported and with not enough detail to determine what factors influenced study outcomes. However, these data did emphasize the high amount of variability in process outcomes—e.g., ranging from very low participation in intervention activities (e.g., 12 % of eligible youth) and low fidelity to intervention activities (only 22 % of activities identified as crucial to changing behavior were performed) [24] to very high fidelity and greater than 90 % observed participation [40].

Conclusions

There remains no standard definition of diet quality in the child dietary intervention literature. Fruits and vegetables represent healthy low-calorie, nutrient-dense options to improve diet quality, and as such, remain popular targets for dietary behavior change interventions. Although the majority of studies included in this review had favorable impacts on dietary behavior, these effects were extremely modest. The largest pre-post change was +0.94 servings/day fruit and vegetables [32]—the clinical significance of which remains unclear. The modest impact of interventions on diet behavior should not be surprising to anyone in the field of diet behavior research, but should stimulate thinking regarding the overall approach. One consideration is the dose of the intervention that is likely to have an impact on behavior—programs must be of sufficient duration and intensity to have an impact in the short-term, and then enough momentum and/or built-in behavioral monitoring in order to demonstrate that behavior change is maintained over the long term. Currently, there is no guidance on what dose will successfully promote or maintain behavior change. It is also highly likely that in addition to dose, there are many other factors (e.g., the delivery method, specificity and relevance of the intervention content to participants' lives, support from home and school environments), that together influence whether or not dietary change occurs, and whether or not there is long-term adherence to change (Table 10.2).

Table 10.2 Promising intervention strategies to improve diet quality in children and adolescents

Choosing behavioral targets associated with both adiposity and diet quality, and focusing intervention efforts on encouraging these behaviors
Interventions that are of sufficient dose and duration to impact dietary behavior over the short and long term
Outcomes assessed using reliable and valid measures of dietary change
Adequate collection and reporting of process evaluation data
Inclusion of appropriate biomarkers that may be linked to dietary and health outcomes
Use popular technologies to engage historically underserved populations, effectively broadening and deepening reach and effectiveness of interventions
Address issues of food access through policy change as well as through interventions that improve availability and access to healthy foods
Involve parents and guardians in intervention activities so that the home becomes a supportive environment for healthy food consumption

Designing the optimal intervention. What does an effective intervention look like? To inform future efforts, the childhood dietary intervention literature would benefit from studies reporting more detailed and thorough process evaluation outcomes—perhaps published as companion publications to outcomes papers.

Effective programs would be of sufficient dose and duration to significantly impact dietary behavior over the short and long term. These programs would be assessed using reliable and valid measures of dietary change for pediatric populations, and should include adequate collection and analyses of process evaluation data so that influences (whether these are mediators, moderators, or other factors) may be identified. The clinical significance of the intervention should be quantifiable—whether this is by a biomarker that is related to weight or disease outcomes, or simply through a clear relationship with disease prevention. Related to this, research should focus on identifying appropriate biomarkers in youth that are linked to dietary and health outcomes, and follow participants over the longer term to determine whether dietary change and biomarker changes in childhood impacts health in adulthood.

Mobile technology-based interventions present several opportunities to broaden and deepen the reach and effectiveness of dietary interventions, thereby helping to reduce disparities in access to healthy eating information and opportunities. In addition to penetrating into populations that historically have not participated in health behavior interventions, the use of mobile technologies also provides the opportunity to engage participants with tailored health information, thereby, increasing engagement and interest with intervention content. Of note, low-income populations have numerous resource constraints, which may make choosing healthy foods difficult. The issue of food access needs to simultaneously be addressed through policy change as well as through interventions to improve availability and access to healthy foods. The CDC has highlighted the importance of understanding obesity prevention from an ecological perspective, and have recommended that obesity prevention should address multiple levels and sectors—all the way from federal government to families and individuals [46].

Where people spend the majority of their time significantly influences their dietary behavior—therefore, interventions to improve diet quality should also consider context of eating and food decisions. For example, in the United States, the National School Lunch and Breakfast Programs are available in 99 and 85 % of public schools. This represents a significant opportunity influence the dietary quality of participants in these programs. A recent systematic review that evaluated the effect of dietary education and physical activity interventions on changes in BMI, obesity prevalence, and weight gain in children concluded that strategies most likely to impact weight in school settings included improvements in *nutritional quality of the food supply*, as well as support for teachers and other staff to implement health promotion strategies, increased opportunity for physical activity, and a school curriculum that included healthy eating, physical activity, and body image [47].

In the home, parental modeling and home activities that support consumption of healthier foods, being more active, and spending less time in screen-based activities represent the most promising intervention strategies for child diet- and weight-related outcomes [47]. In fact, parent involvement in general appears to be an "added value" to the success of dietary interventions [48]. This is unsurprising, as parents exert significant influence over the types of foods that are available and accessible in the home, provide opportunities for their children to learn about healthy lifestyle behaviors, and are influential role models for their children. Parents therefore, represent one end of a continuum of support as interventions extend from the community to the school, and into the home.

Making healthy food choices should be the easy, default choice to make. Achieving and sustaining healthy eating habits will require sustained behavior interventions that are strategically combined with other approaches, including reducing barriers to healthy food access, and increasing skills related to healthy food choices, preparation and consumption. In order for interventions to be maximally effective, individual level behavior changes must be supported by community/national policies (e.g., providing subsidies which offset the cost of healthier, higher quality choices). As obesity will most certainly continue to be a major health challenge in the coming decade, interventionists should focus their efforts on influencing significant correlates of adiposity in children. Since specific dietary behaviors have been associated with both adiposity and diet quality in youth, focusing intervention efforts on encouraging behaviors related to increased diet quality and healthy weight (e.g., fruits and vegetables and family meals) while discouraging behaviors that detract from diet quality (e.g., sugar-sweetened beverages and fast food) represent evidence-based, consumer-friendly approaches to nutrition education that are simple for health professionals to talk about with their patients, and easy for children and parents alike to understand and operationalize in the "real world."

References

1. Food and Agriculture Organization. The state of food insecurity in the world. 2010. Available at: http://www.webcitation.org/65WqM7jFP. Accessed 21 March 2012.
2. Seidell JC, Bouchard C. Visceral fat in relation to health: is it a major culprit or simply an innocent bystander? Int J Obes. 1997;21:626–31.
3. Ledikwe JH, Blanck HM, Khan LK, et al. Low-energy-density diets are associated with high diet quality in adults in the United States. J Am Diet Assoc. 2006;106(8):1172–80.
4. Drewnowski A, Henderson SA, Driscoll A, Rolls BJ. The dietary variety score: assessing diet quality in healthy young and older adults. J Am Diet Assoc. 1997;97(3):266–71.
5. Nikolic M, Nikic D, Petrovic B. Fruit and vegetable intake and the risk for developing coronary heart disease. Cent Eur J Public Health. 2008;16(1):17–20.

6. World Cancer Research Fund/American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: World Cancer Research Fund/ American Institute for Cancer Research; 2007.
7. Joshipura KJ, Ascherio A, Manson JE, et al. Fruit and vegetable intake in relation to risk of ischemic stroke. JAMA. 1999;282(13):1233–9.
8. Carter P, Gray LJ, Troughton J, Khunti K, Davies MJ. Fruit and vegetable intake and incidence of type 2 diabetes mellitus: systematic review and meta-analysis. BMJ. 2010;341:c4229.
9. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 2011;364(25):2392–404.
10. Sebastian RS, Cleveland LE, Goldman JD, Moshfegh AJ. Trends in the food intakes of children 1977–2002. In: Proceedings: annual conference of the American council on consumer interests; 2006, vol 52. p. 433–4. URL: http://www.webcitation.org/65Wrj2Qyr
11. Jekanowski MD, Binkley JK, Eales J. Convenience, accessibility, and the demand for fast food. J Agric Resour Econ. 2006;26(1):58–74.
12. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in food locations and sources among adolescents and young adults. Prev Med. 2002;35(2):107–13.
13. Reedy J, Krebs-Smith SM. Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. J Am Diet Assoc. 2010;110(10):1477–84.
14. Krebs-Smith SM, Guenther PM, Subar AF, Kirkpatrick SI, Dodd KW. Americans do not meet federal dietary recommendations. J Nutr. 2010;140(10):1832–8.
15. Gillman MW, Rifas-Shiman SL, Frazier AL, et al. Family dinner and diet quality among older children and adolescents. Arch Fam Med. 2000;9(3):235–40.
16. Poti JM, Popkin BM. Trends in energy intake among US children by eating location and food source, 1977–2006. J Am Diet Assoc. 2011;111(8):1156–64.
17. Cullen KW, Baranowski T, Owens E, Marsh T, Rittenberry L, de Moor C. Availability, accessibility, and preferences for fruit, 100 % fruit juice, and vegetables influence children's dietary behavior. Health Educ Behav. 2003;30(5):615–26.
18. Hanson NI, Neumark-Sztainer D, Eisenberg ME, Story M, Wall M. Associations between parental report of the home food environment and adolescent intakes of fruits, vegetables and dairy foods. Public Health Nutr. 2005;8(1):77–85.
19. Sebastian RS, Cleveland LE, Goldman JD. Effect of snacking frequency on adolescents' dietary intakes and meeting national recommendations. J Adolesc Health. 2008;42(5):503–11.
20. Nicklas TA, Baranowski T, Cullen KW, Berenson G. Eating patterns, dietary quality and obesity. J Am Coll Nutr. 2001;20(6):599–608.
21. Cullen KW, Bartholomew LK, Parcel GS. Girl scouting: an effective channel for nutrition education. J Nutr Educ Behav. 1997;29:86–91.
22. Friel S, Kelleher C, Campbell P, Nolan G. Evaluation of the nutrition education at primary school (NEAPS) programme. Public Health Nutr. 1999;2(4):549–55.
23. Perry CL, Bishop DB, Taylor G, et al. Changing fruit and vegetable consumption among children: the 5-a-Day Power Plus program in St. Paul, Minnesota. Am J Public Health. 1998;88(4):603–9.
24. Baranowski T, Davis M, Resnicow K, et al. Gimme 5 fruit, juice, and vegetables for fun and health: outcome evaluation. Health Educ Behav. 2000;27(1): 96–111.
25. Foerster SB, Gregson J, Beall DL, et al. The California Childrens 5 A Day-Power Play! Campaign: evaluation of a large-scale social marketing initiative. Fam Community Health. 1998;21(1):46–64.
26. Perry CL, Bishop DB, Taylor GL, et al. A randomized school trial of environmental strategies to encourage fruit and vegetable consumption among children. Health Educ Behav. 2004;31(1):65–76.
27. Birnbaum AS, Lytle LA, Story M, Perry CL, Murray DM. Are differences in exposure to a multicomponent school-based intervention associated with varying dietary outcomes in adolescents? Health Educ Behav. 2002;29(4):427–43.
28. Lytle LA, Murray DM, Perry CL, et al. School-based approaches to affect adolescents' diets: results from the TEENS study. Health Educ Behav. 2004;31(2):270–87.
29. Reynolds KD, Franklin FA, Binkley D, et al. Increasing the fruit and vegetable consumption of fourth-graders: results from the high 5 project. Prev Med. 2000;30(4):309–19.
30. Di Noia J, Contento IR, Prochaska JO. Computer-mediated intervention tailored on transtheoretical model stages and processes of change increases fruit and vegetable consumption among urban African-American adolescents. Am J Health Promot. 2008;22(5):336–41.
31. Nicklas TA, Johnson CC, Myers L, Farris RP, Cunningham A. Outcomes of a high school program to increase fruit and vegetable consumption: Gimme 5-a fresh nutrition concept for students. J Sch Health. 1998;68(6):248–53.
32. Thompson D, Baranowski T, Baranowski J, et al. Boy Scout 5-a-Day Badge: outcome results of a troop and internet intervention. Prev Med. 2009;49(6):518–26.
33. Haire-Joshu D, Elliott MB, Caito NM, et al. High 5 for kids: the impact of a home visiting program on fruit and vegetable intake of parents and their preschool children. Prev Med. 2008;47(1):77–82.
34. Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH, Cade J. Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. BMJ. 2001;323(7320):1029–32.
35. Gortmaker SL, Cheung LW, Peterson KE, et al. Impact of a school-based interdisciplinary intervention on diet and physical activity among urban primary school children: eat well and keep moving. Arch Pediatr Adolesc Med. 1999;153(9):975–83.
36. Gortmaker SL, Peterson K, Wiecha J, et al. Reducing obesity via a school-based interdisciplinary intervention

among youth: planet health. Arch Pediatr Adolesc Med. 1999;153(4):409–18.
37. Dzewaltowski DA, Estabrooks PA, Welk G, et al. Healthy youth places: a randomized controlled trial to determine the effectiveness of facilitating adult and youth leaders to promote physical activity and fruit and vegetable consumption in middle schools. Health Educ Behav. 2009;36(3):583–600.
38. Gentile DA, Welk G, Eisenmann JC, et al. Evaluation of a multiple ecological level child obesity prevention program: switch what you do, view, and chew. BMC Med. 2009;7:49.
39. Perry CL, Lytle L, Feldman H, et al. Effects of the Child and Adolescent Trial for Cardiovascular Health (CATCH) on fruit and vegetable intake. J Nutr Educ Behav. 1998;30(6):354–60.
40. Mauriello LM, Ciavatta MM, Paiva AL, et al. Results of a multi-media multiple behavior obesity prevention program for adolescents. Prev Med. 2010;51(6):451–6.
41. Contento I, Black G, Bronner Y, et al. The effectiveness of nutrition education and implications for nutrition education policy, programs and research: a review of research. J Nutr Educ Behav. 1995;17(6):277–418.
42. Baranowski T, Baranowski J, Cullen KW, et al. 5 a day achievement badge for African-American Boy Scouts: pilot outcome results. Prev Med. 2002;34(3):353–63.
43. Baranowski T, Domel S, Gould R, et al. Increasing fruit and vegetable consumption among 4th and 5th grade students: results from focus groups using reciprocal determinism. J Nutr Educ. 1993;25:114–20.
44. Cullen KW, Baranowski T, Rittenberry L, Olvera N. Social-environmental influences on children's diets: results from focus groups with African-, Euro- and Mexican-American children and their parents. Health Educ Res. 2000;15(5):581–90.
45. Baranowski T, Baranowski J, Cullen K. et al. Problems and possible solutions for interventions among children and adolescents. In: O'Dea JA, Eriksen M, editors. Childhood obesity prevention international research, controversies and interventions. Oxford University Press; 2010.
46. Lemieux S, Prud'homme D, Nadeau A, Tremblay A, Bouchard C, Despres JP. Seven-year changes in body fat and visceral adipose tissue in woman. Associations with indexes of plasma glucose-insulin homeostasis. Diabetes Care. 1996;19(9):983–91.
47. Waters E, de Silva-Sanigorski A, Hall BJ, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev. 2011;12:CD001871.
48. Hingle MD, O'Connor TM, Dave JM, Baranowski T. Parental involvement in interventions to improve child dietary intake: a systematic review. Prev Med. 2010;51(2):103–11.

11 Free Fruit for School Children to Improve Food Quality

Elling Bere and Knut-Inge Klepp

Key Points

- Free school fruit is much more effective in increasing school children's FV intake than a subscription programme.
- Free school fruit might result in a sustained elevated FV intake also after the period of free fruit.
- An increased intake of FV might reduce the consumption of unhealthy snacks.
- The nationwide subscription programme in Norway might contribute to increased social disparities in FV intake.
- Altogether a free school fruit programme appears as an effective effort in order to improve food quality of school children.

Keywords

Free school fruit • Intervention • Fruits and vegetables • Adolescents • Unhealthy snacks • Long-term effects • Social disparities

Abbreviations

FV	Fruits and vegetables
FVMM	Fruits and Vegetables Make the Marks
SES	Socioeconomic status

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Introduction

A diet high in fruits and vegetables (FV) is inversely related to several chronic diseases [1], and an increased intake would improve diet quality and global public health [2]. In Norway, children and adolescents consume only about half of the national five-a-day recommendation [3]. As food preferences and habits established in childhood to a large extent tend to be maintained into adulthood [4, 5], and in order to achieve maximum prevention potential, it is important to get children to eat more FV. It is also important that effective efforts conducted to increase children's FV intake result in sustained elevated

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FV intakes, preferably throughout life, in order to have maximum health prevention potential. However, only a few school-based intervention studies evaluate follow-ups longer than 1 year after the end of the intervention [6]. In addition, eating more FV could mean eating less of something else, and the effect on diet quality of eating more FV will be even more apparent if the increased FV consumption additionally leads to a lower intake of unhealthy snacks high in salt, sugar and fats [7]. Only a few studies have explored whether interventions aimed to increase intake of FV, also decrease consumption of unhealthy snacks.

School Fruit Programmes in Norway

The national Norwegian authorities have conducted considerable efforts to increase school children's fruit and vegetable intake at school the last years. A subscription programme for grades 1–10 was initiated in 1996 and made nationwide in 2003 in collaboration with the Norwegian Marketing Board for Fruits and Vegetables. In this programme the schools initially choose to participate or not, and then the pupils at the participating schools can decide to subscribe or not. The cost for the parents is currently NOK 2.50 per school day (approximately EUR 0.30). The pupils who subscribe receive a piece of fruit or a carrot each school day, usually in connection with their lunch meal. Apples, pears, bananas, oranges, clementines, kiwis, carrots and nectarines are the most common types. The programme is subsidized by the Norwegian Government with NOK 1.00 per pupil per school day.

It is a great challenge to get schools to participate and parents/pupils to subscribe. In the spring of 2006 did 41 % of Norwegian elementary schools participate, and within these schools did 28 % of the pupils subscribe [8], giving a total of only 12 % of all Norwegian school children. Younger pupils tend to subscribe to a greater extend then older pupils.

Despite the Norwegian welfare state and the large Norwegian GDP per-capita, there are large social inequalities in health in Norway [9]. Social inequalities are also seen in health-related behaviours (e.g., in diet quality), and among adolescents in Norway rather large sex [10] and socioeconomic [11] disparities in FV intake have been reported. For example, boys have been reported to eat FV 11.9 times/week, compared to 14.5 times/week for girls [10]. Adolescents of parents with higher education (as an indicator of socioeconomic status) have been reported to eat more FV than adolescents of parents without higher education, and this disparity is increasing with age; 14.0 vs. 12.8 times/week for high and low socioeconomic status (SES) at age 12.5 and 15.1 vs. 12.7 times/week respectively at age 15.5 [11]. There is a governmental desire to reduce these inequalities, and a free school fruit programme has been suggested as an effective means of achieving this goal since all adolescents attend school [12].

From fall 2007, an official free school fruit programme (without parental payment) was implemented in all secondary elementary schools (grades 8–10) and all combined schools (grades 1–10) in Norway. Indeed, it is now legally established that all pupils in secondary schools are entitled to a free piece of fruit or vegetable every school day [13]. The subscription program, with parental payment, still runs in primary/elementary schools (grades 1–7).

The Fruits and Vegetables Make the Marks Project

Within the research project Fruits and Vegetables Make the Marks (FVMM) we have evaluated the subscription programme and the effect of receiving free school fruit in two studies. The present paper is a summary of the results previously presented in international journals [7, 14–19].

Study Design of the FVMM Project

A longitudinal school-based intervention study was initiated in 2001 (Cohort 1, Table 11.1), and a new repeated cross-sectional survey was conducted at the same schools in 2008 (Cohort II, Table 11.1).

Table 11.1 Describes the two different study designs for evaluating different school fruit programmes in Norway (Hedmark and Telemark counties) within the FVMM project: (I) A longitudinal design (Cohort I), and (II) two repeated cross-sectional surveys within the same schools (Cohort I vs. Cohort II)

Cohort I (38 schools, $n = 1{,}950$)[a]	Cohort II (27 schools, $n = 1{,}339$)
Baseline survey: 2001	First survey: 2008
Intervention: subscription programme[b]	Intervention: subscription programme[b]
Free fruit PILOT[c]	Free fruit nationwide[d]
Follow-up surveys: 2002, 2003, 2005, 2009	

[a]Of which 27 schools also participated in the 2008 survey (cohort II). $n = 1{,}488$ in these 27 schools

[b]The subscription programme (grades 1–10) was initiated in 1996 and made nation-wide in 2003. In Hedmark and Telemark counties this programme was initiated in 2001

[c]A free school fruit pilot programme was implemented within nine elementary schools (grades 1–7) during the school year of 2001/2002

[d]A free fruit programme was implemented nation-wide to all secondary elementary schools (grades 8–10) and all combined schools (grades 1–10) in Norway from fall 2007

STUDY 1: In 2001, 48 elementary schools from Hedmark and Telemark counties (24 schools in each county) were randomly selected and invited to participate in the research project FVMM, and 19 schools from each county agreed to participate. These two counties were chosen because the subscription programme was about to start in these counties during the autumn semester of 2001. All sixth and seventh graders (age 10–12), and one of their parents, in the 38 schools were invited to take part in a questionnaire survey in September 2001 (which was the baseline survey for Cohort I, see Table 11.1) [14, 15, 20]. In total 1,950 pupils (85 %) and 1,647 parents participated in the baseline survey. Further two surveys were carried in these schools in May 2002 and May 2003, a fourth survey was carried out in May 2005 (at that time the study population were in ninth and tenth grade in 33 different secondary elementary schools), and a fifth survey in September 2009 (at that time most participants had finished high school).

At the first survey in September 2001 no pupils participated in any school fruit programme. A total of 9 out of the 38 elementary schools were offered to participate in the school fruit programme for free from October 2001 to the end of the school year (i.e., June 2002), and all schools wanted to participate, giving all pupils in these schools free school fruit this school year. Free school fruit for the nine schools was made possible by the Norwegian Fruit and Vegetable Marketing Board through the collective agricultural agreement between Norway's farmers and the agricultural authorities to reduce the price and the administration of the School Fruit Programme. Due to practical reasons only schools from Hedmark could receive free school fruit, and these 9 schools were therefore randomly drawn from the 19 participating schools in this county. Of the remaining 29 schools, 9 schools did chose to participate in the subscription programme starting in these counties this school year (lasting from October to June), while 20 schools opted not to participate.

STUDY 2: The 38 schools were recontacted in 2008 and invited to once more participate in a similar survey [19]. At that time 27 schools agreed to participate, and all sixth and seventh graders in these 27 schools were invited to take part in the survey (Cohort II, see Table 11.1). Pupils at these 27 schools, both from 2001 to 2008, constitute the study sample of STUDY 2. A total of 1,488 pupils (out of 1,727 eligible; 86 %) in 2001 and 1,339 pupils (out of 1,712 eligible; 78 %) in 2008 completed the questionnaire and brought home a parent questionnaire to be completed by one of their parents. In the case of 1,230 and 996 pupils, respectively, one of their parents completed the parent questionnaire.

The 2008 survey was also conducted in September. In September 2001, no schools had any organized school fruit programme at the survey time. In September 2008, 5 schools participated in the free official nationwide school fruit programme (all schools with grades 1–10) (abbreviated FREE FRUIT 08), while 10 schools participated in the subscription programme (abbreviated SUBSCRIPTION 08) and 12 schools did not participate in any official school fruit programme (abbreviated NO PROGRAM 08). The free fruit programme was implemented from fall 2007 in the five schools.

Questionnaires in the FVMM Studies

A written 24-h fruit and vegetable recall was used to assess pupils' FV intake. The 24-h recall was read aloud to the pupils by a project worker. FV intake the previous day was recorded for school days (i.e., the survey was conducted on weekdays, Tuesday through Friday). The 24-h recall separated the day into five time periods (before school, at school, after school, at dinner, and after dinner). The pupils recalled the types of FV they ate at the different time periods in household measures (e.g., one apple, 12 grapes) or in portions (e.g., one portion of mixed green salad). The household measures were coded into portions/day, and one portion was set at about 80 g (ranging from 65 g (one carrot), to 105 g (one apple/one orange)). The conversions from household measures to portions were based on household measures and food weights published by The Norwegian National Association for Nutrition and Health. Juices and potatoes were not included in the fruit and vegetable calculations. F, V and FV intake at school (portions/day) and F, V and FV intake all day (portions/day) were calculated. The 24-h recall is previously presented, and validity and reliability have been reported for FV intake among sixth graders [21].

In addition, a frequency question was included, asking: *How often do you eat fruits and/or vegetables at school*? Response alternatives were: Every school day, 4 days a week, 3 days a week, 2 days a week, 1 day a week, less than once a week and never. The FFQ question was dichotomized into a new variable; Eating FV at school 4 or 5 days/week vs. less. Eating FV 4 or 5 school days per week was interpreted as consuming FV at school on most school days. Based on data from a previous test–retest study involving 114 children from sixth grade [21], 93 % of the children were classified into the same category in the dichotomized variable (4 or 5 days/week vs. less) on two assessments, 14 days apart [19].

A sum-score of unhealthy snacks was made from the following three items: "How often do you drink soda (including sugar)?", "How often do you eat candy (e.g., chocolate, mixed candy)", and "How often do you eat potato chips?" All items had ten response alternatives (never, less than once a week, once a week, twice a week, … , 6 times a week, every day, several times every day), and they were scored (0, 0.5, 1, 2, …, 7, 10), giving the unhealthy snacks scale a range from 0 to 30 times/week. Based on data from a previous test–retest study involving 114 children from sixth grade (16), scores on the unhealthy snacks scale were significantly ($p < 0.001$) correlated (Pearson's correlation coefficient; $r = 0.71$), and mean values were not significantly different (paired samples t-test; 5.2 vs. 5.5 times/week, $p = 0.24$), on two assessments with 14 days in between [7].

The pupils reported their own sex. As an indicator of SES, parents recorded their own level of education (lower: having no college or university education/higher: having attended college or university).

The effect of the interventions is in STUDY 1 reported as the difference between intervention and control groups at the follow-up surveys, adjusted for baseline data (September 2001), or as the difference in FV intake between the intervention group and control group in the changes in FV intake from baseline to the follow-up survey. The effect of the governmental efforts/interventions evaluated in STUDY 2 was assesses as the differences in the changes in FV intake over time for the different groups (FREE FRUIT 08, SUBSCRIPTION 08 and NO PROGRAM 08).

The Impact of Free School Fruit on Diet Quality

1. A free fruit programme is more effective than a subscription programme

   STUDY 1: Only 9 of 29 schools that were not offered free school fruit, did choose to participate in the subscription programme. At these 9 schools did 41 % of the pupils in seventh grade choose to subscribe. This was 11 % of the total number of 7 grade pupils at the 29 schools that were offered the subscription programme. FV intake at school did increase at schools taking part in the subscription programme compared to the schools that chose not to take part in the subscription programme (the effect of the subscription programme was estimated to be 0.2 portions/school day at school, $p=0.003$, and 0.1 portions/day for all day, $p=0.58$) [14]. At the schools that received free school fruit the school year of 2001/2002 there were no seventh graders that did not want to participate (i.e., participation rate in the free fruit programme was 100 %). The effect of free school fruit was estimated to be 0.8 portions/day, both at school ($p<0.001$) and for all day ($p=0.003$), compared to schools not taking part in any school fruit programme. Free school fruit was also significantly more effective in increasing pupils FV intake than the subscription programme (difference was 0.6 portions/day at school ($p<0.001$), and 0.7 portions/day for all day ($p=0.03$)).

STUDY 2: Unadjusted, FV intake at school for sixth and seventh grade pupils at the 27 schools increased from 0.36 to 0.71 portions/school day from 2001 to 2008 [19]. FV intake all day increased from 2.45 to 3.07 portions/day over the same period, and the proportion of pupils reporting to eat FV at school 4 or 5 days a week increased from 29 to 59 %. The increase in FV intake, both at school and all day, was largest within the schools that had been included since 2007 in the national free school fruit programme (Free fruit 08), and smallest within the schools not taking part in any of the governmental efforts to increase FV intake at school. The time * group interaction was significant for FV intake at school ($p=0.02$) but not significant for FV intake all day ($p=0.20$). All effects appear to be due to an increase in F intake only. The time * group interactions for both F intake at school and F intake all day were significant; $p<0.001$ and $p=0.04$, respectively. The increases in F intake at school were 0.49, 0.29 and 0.18 portions/school day, respectively for the Free fruit 08, Subscription 08 and No programme 08 schools, and the increases in F intake all day were 0.74, 0.39 and 0.16 portions/day, respectively. The increase in the proportion reporting to eat FV at school 4 or 5 days a week increased respectively with 61, 34 and 9 percentage points.

2. One year of free school fruit—sustained long-term effect

   STUDY 1: One year (May 03) after the end of the free fruit programme did the pupils at these schools still eat more FV than the pupils at schools not taking part in the free fruit programme, both at school (0.2 portions/school day, $p=0.07$) and all day (0.5 portions/day, $p=0.03$) [15]. This sustained effect was also apparent 3 years after the end of the free fruit intervention (May 05) [16]. The sustained effect after 3 years was estimated to be 0.4 portions/day for all day ($p<0.001$) and 0.1 portions/school day at school ($p<0.001$) [16]. Some, but not all, of the sustained effect could be explained by a higher participation rate in the subscription programme among those receiving free fruit in the school year of 2001/2002 in the following years; e.g., 31 % of those receiving free school fruit in 2001/2002 did subscribe in May 03 compared to 7 % of the pupils in the control schools [15]. Similar figures for May 05, while the participants were in secondary elementary school, were 16 % compared to 1 % [16]. Preliminary analyses from a new follow-up survey conducted in September 2009 indicates sustained effects of the free school fruit programme also 7 years after the end of the free fruit programme [22].

3. Free school fruit reduced consumption of unhealthy snacks

   STUDY 1: Those receiving free school fruit did reduce consumption of unhealthy snacks, but this was only apparent for the children of parents without higher education (i.e., the low SES group) [14]. The pupils of parents without higher education did initially consume unhealthy snacks more often than pupils of parents with higher education (1.1 times/week more often ($p=0.005$)). The reduction in consumption of unhealthy snacks in the group of pupils of parents without higher education for those receiving free school fruit compared to those not receiving free school fruit was estimated to be 1.0 times/week ($p=0.04$). This difference was sustained also 1 year after the end of the free fruit programme [15], but not after 3 years [16].

STUDY 2: From 2001 to 2008 consumption of unhealthy snacks in the study sample of all 27 schools decreased from 6.9 to 4.6 times/week ($p<0.001$) [7]. The decrease of unhealthy snack consumption was largest within the schools that had been included in the national free school fruit programme (−2.6 times/week). The effect of the school fruit programme was again significant in reducing consumption of unhealthy snacks in children of parents without higher education, but not among children of parents with higher education. The decreases in consumption of unhealthy snacks were respectively 3.8, 2.5 and 1.6 times/week, respectively for the Free fruit 08, Subscription 08 and No programme 08 schools.

4. The subscription programme increases social disparities while the free fruit programme is effective among all groups

STUDY 1: For the subscription programme it was observed that at participating schools, those that subscribed were in general healthier than non-subscribers [8, 14, 18]. Compared to the non-subscribers, the subscribers were often more girls, more had plans of taking higher education, they ate more FV, they watched less TV, they were less physical active, they ate less often breakfast, lunch and supper, they had a lower BMI and fewer were on a diet. Also parents to those subscribing were different from parents of non-subscribers. Parents of subscribing pupils were older, more had higher education, had higher income, watched less TV and smoked less than parents of non-subscribers.

In the main analyses of free school fruit we did not observe any difference of the effect of free school fruit on FV intake regarding sex, parental education level, initial habitual FV intake and preferences for FV. That is, the effect of the intervention was not different for different groups. However, we did observe an interaction between sex and the school's participation in the subscription programme the year following the free fruit programme: Girls did sustain their elevated FV intake independent of whether the school participated in the subscription programme or not, while boys only sustained their elevated FV intake if the school participated in the subscription programme the year following the free fruit programme—indicating that boys might need an elevated accessibility of fruit and vegetables more than girls, in order to sustain the increased intake level [15].

STUDY 2: Also in STUDY 2 the effect the free fruit programme appears to be similar for both boys and girls and for children of parents both with and without higher education [19]. For example, the percentage of boys and children of parents without higher education reporting to eat FV at school 4 or 5 days/week within the Free Fruit 08 schools increased from 20 % and 28 % in 2001 to 81 % and 79 % in 2008, respectively, while the same figures for girls and for children of parents with higher education increased from 28 % and 30 % in 2001 to 88 % and 90 % in 2008 [19].

Free School Fruit in a Broader Perspective

Due to low participation rates from schools and low subscription rates from pupils, the subscription programme do only have a limited effect of increasing school children's FV intake. In addition, it is in general a healthier group of pupils that choose to subscribe, a fact that might increase the social disparity seen in children's FV intake. The effect of the free programme on intake of FV is much larger than for the subscription programme, and the free programme appears to increase FV intake among all groups of children—also those that initially eat the least (as boys and pupils in low SES families).

It is difficult to state the health impact of a free school fruit programme. We have assessed the effect of 1 year of free school fruit. What effect would a free fruit programme for all 10 years of elementary school give? Research indicates that repeated exposure for FV increase children's preferences for these foods [23, 24]. That those given free fruit for 1 year also 1 and 3 years later still eat more FV than the control condition gives an indication that 10 years of free school fruit might give a sustained elevated FV intake also in life after elementary school. A Norwegian cost benefit analysis reported that an increase of only 2.5 g/day is needed in order for the Norwegian School Fruit Program to be cost effective, if offered for free for all 10 years of elementary and secondary school [25]. However, an assumption is that the increased FV intake has to be sustained throughout life. One FV portion in the FVMM study is defined to about 80 g [21]. The effect of the free fruit intervention 3 years after the fruit was provided for free can therefore be estimated to be about 30–35 g/day [16]. That is, it is probably a very small permanent increase in FV intake that is needed for a free school fruit programme to also be cost effective.

A positive side effect of free school fruit appears to be a reduction in the consumption of unhealthy snacks. A daily extra fruit every school day gives an extra energy content of about 1 MJ/week (e.g., 5 apples). This is comparable to the energy content in 50 g chocolate, 50 g of potato chips or 0.6 L sugar containing soft drink, which again is rather similar to the reduction observed in consumption of unhealthy snacks (about 1.0 times/week). Norwegian children consume more added sugar than recommended [26], and a reduction in consumption of unhealthy snacks is very welcome. The reduction in unhealthy snacks was observed within pupils of parents without higher education, and those children did initially eat more unhealthy snacks than children of parents with higher education, and therefore also had the greatest benefit of a reduced intake.

Socioeconomic disparities in health behaviours are common, also in a well-developed and homogenous society as Norway [9]. In the FVMM project we see that girls eat about 50 % (1 portion/day) more FV than boys [15]. It has been reported that girls usually is more susceptible to the content of FV interventions [27]. As we did not see any interactions between sex and free school fruit when analyzing the effect of free school fruit, free school fruit therefore appears to have similar effect in increasing FV intake both for boys and girls. However, we did observe a difference in the sustained elevated FV intake the following year, and girls had a sustained elevated FV intake independent of whether the school participated in the subscription programme or not the following year, while boys sustained an elevated FV intake in a higher degree if the school participated in the subscription programme the following year [15]. This indicates that boys might need an extra effort (e.g., a subscription programme as here) in order to sustain an elevated FV intake, than girls do.

There is a clear difference between those that subscribe to the subscription programme and those that don't subscribe, and the subscribers are clearly healthier group. A report on the effect of how the price might regulate the subscription rate showed that a rather large group would not participate if the fruit was not handed out for free [28]. Compared to the energy it contains FV is expensive foods [29], and increasing FV intake according to the recommendations will give a more expensive diet [30], which probably contribute to the explanation of the socioeconomic disparities reported in FV intake (i.e., that parents of subscribing pupils have a higher salary than parents of pupils not subscribing). Experiences from the project showed that none of the schools offered to participate in the school fruit programme for free would not participate, and such a free programme will therefore reach all pupils, also those that need it the most.

Conclusions

Free school fruit is much more effective in increasing school children's FV intake than a subscription programme. Free school fruit might result in a sustained elevated FV intake, also after the period of free fruit. An increased intake of FV might reduce the consumption of unhealthy snacks. The nationwide subscription programme in Norway might contribute to increased social disparities in FV intake. Altogether, a free school fruit programme appears as an effective effort in order to improve food quality of school children.

References

1. World Health Organization. Diet, nutrition and the prevention of chronic diseases. Joint WHO/FAO expert consultation. Technical report series no. 916. Geneva: WHO; 2003.
2. World Health Organization. Global strategy on diet, physical activity and health. Geneva: WHO; 2004.
3. Øverby NC, Andersen LF. Ungkost-2000: Landsomfattende kostholdsundersøkelse blant elever i 4. og 8. klasse i Norge. Oslo: Sosial- og helsedirektoratet; 2002.
4. Kelder SH, Perry CL, Klepp K-I, et al. Longitudinal tracking of adolescent smoking, physical-activity, and food choice behaviours. Am J Public Health. 1994;84: 1121–6.
5. Lien N, Lytle LA, Klepp K-I. Stability in consumption of fruit, vegetables, and sugary foods in a cohort from age 14 to age 21. Prev Med. 2001;33:217–26.
6. Van Cauwenberghe E, Maes L, Spittaels H, et al. Effectiveness of school-based interventions in Europe to promote healthy nutrition in children and adolescents: systematic review of published and 'grey' literature. Br J Nutr. 2010;103:781–97.
7. Øverby NC, Klepp K-I, Bere E. Introduction of a school fruit programme is associated with reduced consumption of unhealthy snacks. Am J Clin Nutr. 2012;96(5):1100–3.
8. Bere E, Klepp K-I. Gratis skolefrukt. Norsk tidsskrift for ernæring, no. 1. 2007;4–8.
9. Sosial- og helsedirektoratet. Sosial- og helsedirektoratets handlingsplan mot sosiale ulikheter i helse: Gradientutfordringen. Oslo: Sosial- og helsedirektoratet; 2005.
10. Bere E, Brug J, Klepp K-I. Why do boys eat less fruit and vegetables than girls? Public Health Nutr. 2008;11:321–5.
11. Bere E, van Lenthe F, Klepp K-I, et al. Why do parents' education level and income affect the amount of fruits and vegetables adolescents eat? Eur J Public Health. 2008;18:611–5.
12. Det kongelige helse- og omsorgsdepartementet. St.meld nr. 20 (2006–2007): Nasjonal strategi for å utjevne sosiale helseforskjeller. Oslo: Det kongelige helse- og omsorgsdepartementet; 2007.
13. Det kongelige kunnskapsdepartement. Ot.prp. nr. 40 (2007–2008): Om lov om endringar i opplæringslova og privatskolelova. Oslo: Det kongelige kunnskapsdepartement; 2008.
14. Bere E, Veierød MB, Klepp KI. The Norwegian School Fruit Programme: evaluating paid vs. no-cost subscriptions. Prev Med. 2005;41:463–70.
15. Bere E, Veierød MB, Bjelland M, et al. Free school fruit—sustained effect 1 year later. Health Educ Res. 2006;21:268–75.
16. Bere E, Veierød M, Skare Ø, et al. Free school fruit—sustained effect three years later. Int J Behav Nutr Phys Act. 2007;4:5.
17. Bere E, Klepp K-I. Free participation in the English School Fruit programme: increased fruit and vegetable intake gives decreased consumption of unhealthy snacks. In: Poster presented at International Conference of Health Benefits of Mediterranean-style diet (EGEA III), Roma; 2005.
18. Bere E, Klepp K-I. Free vs. paid school fruit programme—big difference with respect to social inequality. In: Proceedings of the 6th congress of the European society for agricultural and food ethics: ethics and the politics of food, Oslo; 2006.
19. Bere E, Hilsen N, Klepp K-I. Effect of the nationwide free school fruit scheme in Norway. Br J Nutr. 2010;104:589–94.
20. Bere E, Veierod MB, Bjelland M, et al. Outcome and process evaluation of a Norwegian school-randomized fruit and vegetable intervention: Fruits and Vegetables Make the Marks (FVMM). Health Educ Res. 2006;21:258–67.
21. Andersen LF, Bere E, Kolbjørnsen N, et al. Validity and reproducibility of self-reported intake of fruit and vegetable among 6th graders. Eur J Clin Nutr. 2004;58:771–7.
22. Bere E, Klepp K-I. One year of free school fruit—7 years of follow-up. In: Oral presentation at 11th European nutrition conference (FENS), Madrid; 2011.
23. Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. Pediatrics. 1998;101:539–49.
24. Wardle J, Cooke LJ, Gibson EL, et al. Increasing children's acceptance of vegetables; a randomized trial of parent-led exposure. Appetite. 2003;40: 155–62.
25. Sælensminde K. Frukt og grønnsaker i skolen. Beregning av samfunnsøkonomisk lønnsomhet. Oslo: Sosial- og helsedirektoratet; 2006.
26. Øverby NC, Lillegaard IT, Johansson L, et al. High intake of added sugar among Norwegian children and adolescents. Public Health Nutr. 2004;7: 285–93.
27. Perry CL, Bishop DB, Taylor G, et al. Changing fruit and vegetable consumption among children: the 5-a-day power plus program in St. Paul, Minnesota. Am J Public Health. 1998;88:603–9.
28. Bere E. Skolefrukt: Hvilken betydning har prisen. Oslo: Universitetet i Oslo (report); 2004.
29. Drewnowski A, Darmon N, Briend A. Replacing fats and sweets with vegetables and fruits—a question of cost. Am J Public Health. 2004;94:1555–9.
30. Darmon N, Ferguson E, Briend A. Do economic constraints encourage the selection of energy dense diets? Appetite. 2003;41:315–22.

12 Diet Quality in Childhood: Impact on Growth

Guo Cheng and Anette E. Buyken

Key Points

- Emerging evidence suggests that modifying diet quality in childhood offers preventive potential for health in later life with regard to later body composition or puberty onset.
- To date, evidence from prospective analyses focusing on single nutrients and foods is inconclusive regarding the relevance of few dietary factors in childhood for body composition.
- With regard to puberty timing, current evidence suggests a beneficial influence of higher intakes of vegetable protein and isoflavone, and lower animal protein intakes.
- These beneficial nutrients may be indicative of a specific dietary pattern.
- Dietary pattern analyses, which consider diet as a whole and account for interactions of nutrients and foods, may provide much needed insights into dietary factors implicated in growth during childhood.

Keywords

Diet quality • Body composition • Growth • Puberty timing • Childhood

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Abbreviations

BMI	Body mass index
DGI-CA	Dietary Guideline Index for Children and Adolescents
DQI	Diet quality index
GI	Glycemic index
HDI	Healthy diet indicator
HEI	Healthy eating index
PCA	Principal component analysis
RC-DQI	Revised Children's Diet Quality Index

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RRR	Reduced rank regression
SSB	Sugar-sweetened beverage
YHEI	Youth Healthy Eating Index

Introduction

Nutritional influences have sustained consequences for growth and wellbeing in later life. To date, evidence has emerged that various chronic diseases in adulthood, such as cardiovascular disease or diabetes mellitus, have origins in the period of growth. Overweight during childhood has both immediate and long-term health consequences. Over the last few decades, the obesity prevalence in children has increased dramatically in USA and European countries [1]. Since obesity is taking place in stable populations over a very short period of time, the epidemic of obesity cannot be attributed to a changed genetic background [2]. Although the contribution of hereditary factors has to be acknowledged [3], it is accepted that current obesity trends are driven by changes in exposure to environmental factors. Nutritional factors are thought to play a major role in the rising prevalence of obesity worldwide.

Puberty onset is a milestone of growth. Age of puberty timing is considered to be of general public health relevance, since an early age at puberty onset may be an intermediary factor for a number of diseases in adulthood, including hormone-related cancers [4, 5], earlier all-cause mortality [6], and a higher risk of metabolic syndrome or cardiovascular disease [7]. The influence of modifiable risk factors such as nutrition on puberty onset has thus been addressed by a number of studies.

The purpose of this chapter is to summarize the available observational evidence regarding the role of nutritional factors for growth. The review begins with an introduction of the use of dietary pattern analyses (in particular among children) and an introduction to critical periods during growth, and is then organized into two sections as follows: relevance of diet quality and dietary factors for (1) body composition and (2) puberty timing. The final section discusses public health relevance.

Diet Quality: Nutrients, Foods/Food Groups and Dietary Patterns

As far as nutritional influences on health are concerned, energy intake/energy density and nutrient compositions of diets, i.e., intakes of carbohydrate, fat, protein as well as micronutrients, have been investigated systematically over the last years. Besides nutrients, the unfavorable contribution of several food items or food groups has been discussed, e.g., snacks, French fries, sugar-sweetened beverages (SSBs), fast foods, and sweets, while other frequently consumed food item or food groups have been suggested to have a protective effect, such as fruits, vegetables, legumes, and high-fiber breakfast cereals.

However, analyses which focus on a single or a few nutrients or foods have several conceptual limitations. Meals consist of a variety of foods with complex combinations of nutrients, which are likely to be interactive. A dietary pattern may thus be more relevant to identify associations between nutrition and health outcomes. Since dietary patterns may be easier for the public to interpret or translate into daily diets, studying dietary patterns could also have important public health implications. It is impossible to measure dietary patterns directly. Statistical methods are thus used to analyze the collected dietary information and to characterize dietary patterns. To date, three approaches have been used in the literature: dietary indices, factor analysis and cluster analysis, and reduced rank regression (RRR).

Dietary Indices

Dietary indices build on a priori defined parameters. To date, a variety of dietary indices, which are typically constructed on the basis of current dietary recommendations, have been proposed to assess overall diet quality. Evaluating the health effects of adherence to dietary recommendations by individuals can provide a practical way for the public to translate the recommendations into diets, e.g., the healthy eating index (HEI) [8] is a summary measure of the degree to which an individual's diet conforms to the serving recommendations of the US Department of Agriculture Food Guide Pyramid for five major food groups and to specific recommendations in the US Dietary Guidelines for Americans.

To date, several dietary indices have been developed for children: the Nutritional Quality Index, the Youth Healthy Eating Index (YHEI), the Revised Children's Diet Quality Index (RC-DQI), the Dietary Guideline Index for Children and Adolescents (DGI-CA), the diet quality index (DQI), the healthy diet indicator (HDI), and the Mediterranean diet score (Table 12.1). These measure adhere to different dietary recommendations: nutrients- or food groups-specific recommendations, or a combination of both.

Factor Analysis and Cluster Analysis

Factor analysis includes both common factor analysis and principal component analysis (PCA). PCA is commonly used to define dietary patterns. Factor analysis is a multivariate statistical technique to identify common underlying dimensions (factors or patterns) of food consumption. Factor analysis aggregates specific food items or food groups on the basis of the degree to which food items in the dataset are correlated with one another. Then, a summary score for each pattern is derived and can be used in either correlation or regression analysis to examine associations between various eating patterns and the outcome of interest. Compared with factor analysis, cluster analysis aggregates individuals into relatively homogeneous subgroups (clusters) with similar food consumption. Individuals are classified into distinct clusters or groups. After the identification of cluster procedure, further analyses are used to interpret association of the dietary patterns with outcomes.

Both factor analysis and cluster analysis, which are purely explorative, have been commonly used in adults, while to date few studies among children used these approaches to address the relevance of nutrition for body composition (Table 12.2).

Reduced Rank Regression

PCA is purely explorative, i.e., it explains as much variation in food intake as possible, however, that does not mean that much variation in important nutrients will be explained. Therefore, it might be wiser to focus on the variation in those nutrients that presumably affect the incidence of disease. The statistical method known as reduced rank regression (RRR), which provides the opportunity to determine linear functions of predictors (foods) by maximizing the explained variation in responses (disease-related nutrients), has thus been applied in epidemiology [9]. RRR is neither an a priori nor a purely exploratory statistical method. Since RRR uses both information sources, data from the study and prior information to define responses, it represents an a posteriori method. To date, few studies in children and adolescents have used the RRR method to address diet quality (Table 12.2).

Critical Periods During Growth

Critical periods of development have been well recognized for many behavioral and physiologic developmental processes. The possibility that nutritional alterations during critical periods of development entrain nutritional states, such as obesity, has also been mentioned [10, 11]. In 1994, Dietz defined critical periods for the overweight/obesity development as "a developmental stage, in which physiologic alterations increase the later prevalence of obesity" [12]. According to observational evidence, there appear to be at least three critical periods in childhood for the development of obesity and its complications in late life [11, 13, 14]: gestation and early infancy, the period of preschool and preadolescence, and finally, the period of adolescence (Fig. 12.1). Regarding diet quality and eating behavior, both early life and mid-childhood may represent particularly sensitive phases for their long-term effects in later life. Furthermore, the hormonal changes regulating appetite, satiety, and fat distribution that occur during puberty, especially

Table 12.1 Dietary quality indices developed for children to measure adherences to different dietary recommendations

Dietary quality indices	Recommendation	Components and comments
Nutritional Quality Index (NQI)	Age- and sex-specific dietary reference values issued by the German Nutrition Society, the Austrian Nutrition Society, the Swiss Society for Nutrition Research, and the Swiss Nutrition Association	NQI^+: intake of fat and saturated fatty acids
		NQI^-: intake of water, protein, carbohydrate, fiber, folate, vitamin C, vitamin E, sodium, iron, and calcium
		The NQI^+ or NQI^- scores range from 0 to 100, with a higher score indicating better diet quality
		The NQI determines the extent to which a child meets the nutritional recommendations for particular nutrients
Youth Healthy Eating Index (YHEI)	Dietary Guidelines for Americans	Consumption of grains, vegetables, fruit, dairy, meat, total fat, saturated fat, cholesterol, sodium, and food variety
		Higher YHEI scores indicate the consumption of nutrient-dense, healthy foods and nutrition-promoting eating behaviors
Revised Children's Diet Quality Index (RC-DQI)	US dietary intake recommendations	Intake of added sugar, total fat, linoleic and linolenic fatty acids, docosahexaenoic acid and eicosapentaenoic acid, consumption of total grains, whole grains, vegetables, fruits, excess fruit juice, dairy, and iron, as well as a component representing a proxy for energy balance
		RC-DQI scores range from 0 to 1, with higher scores indicating better diet quality
		The RC-DQI was developed to rate diet quality in relation to the US dietary intake recommendations and healthy-promoting behaviors
Dietary Guideline Index for Children and Adolescents (DGI-CA)	2003 Australian Dietary Guidelines for Children and Adolescents	Consumption of fruit, vegetables, breads and cereals, whole grain, cereals, meat and alternatives, dairy foods, reduced fat dairy, water, extra foods, healthy fats, and variety
		The total DGI-CA score ranges from 0 to 100, with a higher score reflecting greater adherence with the dietary guidelines
Diet quality index (DQI)	US dietary recommendations	Intake of total fat, saturated fat, cholesterol, fruit and vegetables, breads and cereals, protein, sodium, and calcium
		Children's DQI scores range from 0 to 16, with lower scores indicating better quality diets
Healthy diet indicator (HDI)	WHO's dietary recommendations for the prevention of chronic disease	Intake of saturated fat, polyunsaturated fat, carbohydrates, free sugars, protein, cholesterol, sodium, fruit and vegetables, and dietary fiber
		The HDI score ranges from 0 to 9, with a higher score indicating a better quality diet
Mediterranean diet score (MDS)	Traditional Mediterranean diet pattern	Consumption of vegetables, legumes, cereals, fruit, fish, meat, and the ratio of mono- and polyunsaturated saturated fat
		The possible range of scores is 0–8, with a higher score indicating better adherence to a Mediterranean diet pattern

Table 12.2 Observational studies relating dietary pattern in childhood to body composition

Author/study	Outcomes/predictors/covariables	Results/conclusion
Dietary quality indices		
Cheng et al. (2010) [69] Prospective study: 222 German children (119 girls and 103 boys)	*Predictors*: nutrient density-based nutritional quality index (NQI), and food group and nutrient-based Revised Children's Diet Quality Index (RC-DQI) at 2 and 3 years before the onset of pubertal growth spurt (ATO) *Outcomes*: age- and gender-specific *Z*-scores of body mass index (BMI), fat mass/height2 and fat-free mass/height2 at ATO *Assessment_method*: 3-day weighed dietary records *Covariates*: birth weight, full breastfeeding for at least 4 months, rapid weight gain between birth and 24 months, physical activity (for NQI), maternal overweight, maternal education, smoking status of the household, and prepubertal body composition	*Results* Neither NQI nor RC-DQI was independently associated with body composition at ATO *Conclusion* This data suggests that diet quality in childhood was not independently associated with body composition at puberty onset
Principal components factor		
Cutler et al. (2011) [70] Cross-sectional study: 4,746 US children Prospective study: 2,516 US children	*Predictors*: dietary pattern identified by principal components factor analysis *Outcomes*: weight status category according to the classification from Must et al. (overweight/obese, normal weight) *Assessment method*: youth/adolescent questionnaire *Covariates*: socioeconomic status, race/ethnicity, physical activity, weight status at baseline	*Results* In cross-sectional analyses, higher adherence to dietary patterns loading heavily on vegetables was associated with lower risk of overweight/obese weight status in girls, whereas higher adherence to a "sweet and salty snack food" pattern was associated with lower risk in boys In prospective analyses, these associations were no longer significant after adjusting for baseline weight status *Conclusion* According to the authors, there were no consistent or intuitive associations between dietary patterns and weight status Identified patterns may not capture the elements of diet that are truly important in determining adolescent weight, or diet may not be the primary driver in determining weight status at this age Methodological difficulties in assessing diet must also be taken into consideration

(continued)

Table 12.2 (continued)

Author/study	Outcomes/predictors/covariables	Results/conclusion
Reduced rank regression		
Wosje et al. (2010) [71]	*Predictors*: dietary pattern identified by reduced rank regression	*Results*
Multiple cross-sectional study: 325 US children	*Outcomes*: fat mass, bone mass	A dietary pattern characterized by a high intake of dark-green and deep-yellow vegetables was related to low fat mass and high bone mass; high processed-meat intake was related to high bone mass; and high fried-food intake was related to high fat mass
	Assessment method: 3-day diet record	Dietary pattern scores remained related to fat mass and bone mass after all covariables were controlled for
	Covariates: race, sex, height, exact age, annual household income, calcium intake, bone mass, fat mass, counts per minute, television viewing, and outdoor playtime	*Conclusion*
		Beginning at preschool age, diets rich in dark-green and deep-yellow vegetables and low in fried foods may lead to healthy fat and bone mass accrual in young children

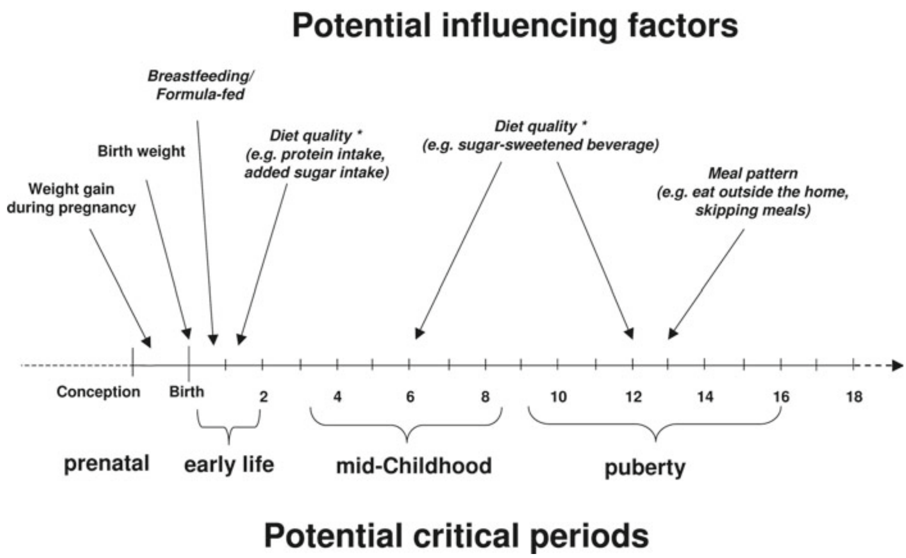

Fig. 12.1 Potential influencing factors during critical periods of growth for body composition development. *Asterisk* current analyses focusing on single nutrients and foods have identified only few dietary factors as potentially relevant for body composition

physiological insulin resistance [15], may put adolescents at increased risk for weight gain [16]. Hence, diet quality in these critical periods may have sustained consequences for growth and wellbeing in later life.

Relevance of Diet Quality for Body Composition

In Early Life

Maternal nutritional status at conception has been associated with obesity or adverse health outcomes in general [17]. Evidence for the role of diet quality during pregnancy is only emerging. Low birth weight is commonly interpreted as a marker of poor fetal nutrition. However, the influences from prenatal period may be modified by the early postnatal period, one of the critical "windows" for growth. The importance of early postnatal life for the body composition development is highlighted by the fact that breastfeeding seems to reduce obesity risk in later childhood [18, 19]. Also, concern has been expressed that higher protein intakes during the period of complementary feeding and early childhood could be associated with both body mass index (BMI) and percentage body fat in later childhood [20, 21]. Recently, added sugar intake at low intake levels during early childhood has been suggested to not be critical for body composition in childhood, however, detrimental effects on body composition development may emerge when added sugar intakes are increased to higher levels [22]. Of interest, the factors discussed as potentially relevant to body composition in early life may be markers for overall diet quality, e.g., breastfeeding is discussed to be confounded by associations with other behavioral factors, and increased intake of added sugar may resemble a marker for other behavioral changes contributing to increases in BMI.

In Mid-Childhood and Puberty

The period of preschool and preadolescence is another important time window for body composition development. Furthermore, adolescents change their lifestyle as they test their autonomy and assert their independence [23], which may affect both their eating behaviors and their physical activity. When addressing the relevance of dietary factors for body composition during these periods, it should be borne in mind that cross-sectional studies do not allow statements on the direction of the observed association, an issue of particular relevance for the outcome overweight/obesity. Therefore, only prospective evidence is reviewed in the following section.

Nutrients and Foods

Energy Intake

Overweight is generally believed to be a consequence of an energy imbalance. Current longitudinal studies, which have addressed the relevance of energy intake in childhood on the body composition, have however not shown significant associations of energy intake with childhood weight gain [24, 25], which may reflect the methodological challenge to accurately measure energy intake. Recently, interest in dietary energy density, i.e., the ratio of the amount of food to total energy intake, has emerged. Higher energy density is commonly interpreted as a marker of diet quality. However, the few prospective studies relating energy density in children to their body composition have yielded mixed results [26–30] (Table 12.3).

Macronutrients

Macronutrients, which are thought to play a role in regulation of body weight, have been investigated systematically (Table 12.3). The evidence from studies analyzing the association between dietary fat and obesity among children is inconsistent [31–38]. In addition, type of dietary fat has received little attention in relation to childhood obesity. The existent prospective studies on protein intake in children and body composition development have yielded mixed results: three studies have shown an association with later overweight/obesity [32, 39, 40], while two other analyses found no relation [34, 37]. In most [24, 39, 41], but not all studies [32], intakes of total carbohydrate among children were not related to the development of body mass.

Table 12.3 Prospective studies focusing on dietary factors in mid-childhood in relation to body composition

Dietary factor	Level of evidence[a] for an association with body composition
Energy intake/energy density	[24–30][b]
Fat intake	[31–38][b]
Protein intake	[32, 34, 37, 39, 40][b]
Total carbohydrate intake	[24, 32, 39, 41][c]
Fiber intake	[43–46][d]
Whole-grain intake	[46][e]
Glycemic index	[45, 46][d]
Sucrose intake	[45, 49, 50][d]
Sugar-sweetened drinks	[51–53, 55–63][a]

[a]Possible evidence of increased risk for overweight/obesity, according to several prospective studies and meta-analyses (please see text explanation)
[b]Inconsistent: several studies suggested an association for body composition; several studies suggested no association for body composition
[c]Probable evidence of no association for body composition, according to all available prospective studies
[d]Possible evidence of no association for body composition, according to several prospective studies
[e]Too little data

Carbohydrate Quality

More recently, increased attention to quality of dietary carbohydrate rather than quantity of carbohydrate intake has begun to clarify the role of dietary carbohydrate in the obesity epidemic. High fiber intake has been reported to yield beneficial effects on objective and subjective measures of satiety in experimental studies conducted in adults [42]. However, among children, only one [43] of the few [44–46] prospective observational studies has demonstrated that dietary fiber intakes were independently associated with measures of obesity (Table 12.3). Additionally, dietary whole grain is supposed to be beneficial for body weight regulation, since whole grains are rich in a myriad of nutrients, including vitamins, minerals, phytochemicals, and other substances that have been related to body weight regulation [47]. To date, the only existent prospective analysis regarding adolescents suggested that whole-grain intake is not relevant for the body composition development during puberty [46] (Table 12.3).

In addition, high dietary glycemic index (GI: calculated as the blood glucose response to a 50 g (or 25 g) carbohydrate portion of food, expressed as a percentage of the same amount of carbohydrate from a reference food, either glucose or white bread [48]) has been proposed as a risk factor for weight gain. However, regarding children and adolescents, two prospective analyses [45, 46] reported no association between dietary GI and BMI or body fatness (Table 12.3).

In particular, the increased consumption of SSB brought up discussions on the relevance of added sugar and SSB for weight gain especially in childhood. However, the results from prospective studies focusing on sucrose intake and added sugar intake in children are inconsistent [45, 49, 50] (Table 12.3). To date, four meta-analyses were conducted [51–54] addressing the relevance of SSB for body composition. Two [52, 53] of them concluded that increased consumption of SSB in children and adolescents is associated with obesity risk, while another meta-analysis [51] judged the effect as almost zero. Mattes et al. did not find any significant effect of lowering the consumption of SSB on BMI, however, in the meta-analysis of a subgroup, the authors found a consistent effect of interventions on the BMI of subjects with higher BMI or obesity at baseline [54]. Since the publication of the meta-analyses, further nine cohort studies in children [55–60] and adolescents [61–63] regarding the relation of SSB with body composition have yielded mixed results. Taken together, according to a number of studies including meta-analysis, an increased consumption of SSB among children and adolescents may be related to later overweight/obesity risk. However, in view of inconsistence, the level of evidence should be judged to indicate a possible association only (Table 12.3). In addition, an increased risk of obesity is especially observed in children and adolescents with initially already increased BMI or already existing overweight.

Dietary Pattern

Overall, the current evidence for dietary factors implicated in body composition development suggests little relevance of diet among childhood except for a potential role of SSB. In this context, it should be noted that consumption of SSB may be strongly related to overall lifestyle and thus be indicative of overall diet quality. It is thus intriguing to consider that analysis of diet quality and/or dietary pattern may provide more insights into the role of diet in the development of body composition during childhood. To date, five cross-sectional studies [64–68] in children and adolescents have directly examined the association between diet quality using dietary quality indices and body composition. Using YHEI scores to characterize the diet quality of 16,452 children aged 9–14 years, Feskanich et al. reported that children with higher YHEI scores (higher diet quality) had a lower BMI [64]. Similarly, Hurley et al. analyzed the diet quality among 317 US children using YHEI scores and they found that lower percent body/abdominal fat was associated with higher diet quality [65]. In addition, using data from the National Health and Examination Survey 1999–2002 from 1,521 children aged 2–5 years, the authors have shown that childhood obesity prevalence decreased significantly with increasing diet quality characterized by RC-DQI score [66]. In line with this analysis, Jennings et al. suggested that children with higher diet quality indices based on the US (DQI) and the WHO (HDI) healthy eating guidelines have lower body weight [67]. However, only a weak positive association was reported in a recent study conducted in 4–10-year-old Australian children [68]: using the age-specific DGI-CA, weak positive associations were observed between DGI-CA score and BMI or waist circumference *Z*-scores. Since cross-sectional data in relation of body composition suffer from potential reverse causality, prospective analyses, which are able to consider the baseline weight status, may be more insightful. To date, only two prospective studies [69, 70] were conducted in children using dietary pattern methods (Table 12.2). The analysis from the DONALD study used two dietary quality indices to identify the diet quality among 222 German children. In this study, diet quality in preadolescents was not independently associated with later body composition. A dietary quality index created on the basis of previous knowledge is an “a priori” dietary pattern [55]. As outlined above, dietary pattern analyses with PCA, which describes the variation in intake of food items, is an alternative approach. Cutler et al. [70] examined associations between dietary patterns identified with PCA based on dietary data in an adolescent cohort and weight status both cross-sectionally and over a 5-year period. In cross-sectional analyses, dietary patterns characterized by higher vegetable intakes were associated with lower risk of overweight in girls, whereas higher adherence to a “sweet and salty snack food” pattern was associated with lower overweight risk in boys. However, these associations were no longer significant in prospective analyses after adjusting for baseline weight status [70].

The fact that in these studies no long-term associations were seen between dietary pattern in children and later overweight may on the one hand relates to the overarching relevance of baseline body composition for later body mass in comparing to current diet quality. Dietary pattern predicting the change of body composition could be of interest in future analyses. On the other hand, the absence of clear relations may be due to the method used for the identification of dietary pattern. Compared with dietary quality indices, PCA is an a posteriori statistical method and solely describes the variation in dietary data. Instead, relating the correlation structure of foods and nutrient intakes to the health outcomes, by methods such as RRR, may reveal more meaningful dietary pattern. This approach has been used in a recent multiple cross-sectional analysis [71] identifying dietary pattern among children predictive of bone and body fat (Table 12.2). This analysis suggested that a dietary pattern characterized by a high intake of dark-green and deep-yellow vegetables was related to low fat mass and high bone mass, while high fried-food intake was related to high fat mass and high processed-meat intake was related to high bone mass. Adjustment for body composition and energy intake at baseline did not influence these associations. Since the patterns identified using RRR in this cohort may change during the time and may not be reproduced in other cohorts, further studies are needed before firm conclusions can be drawn for children. In summary, although an examination of dietary patterns has certain advantages over traditional methods of examining single foods or nutrients, more evidence using advanced approaches is needed to fully elucidate benefits of nutrition for body composition development.

Relevance of Diet Quality for Puberty Timing

Nutrition during early life, which is largely reflected by breastfeeding, has been discussed as a potential determinant of pubertal timing. However, prospective analyses have not found an independent association of breastfeeding with age at menarche [72–75], age at take-off, or age at peak height velocity [74]. Furthermore, protein intake in the first and second year of life may not to be critical for the timing of puberty onset [76].

The influence of various nutrient/food intakes in childhood on puberty timing is increasingly acknowledged in a number of studies. We recently reviewed these data (for details on this summary of observational data please see [77]). To date, energy intake, intakes of fat and fatty acids, fiber, protein (animal protein and vegetable protein), micronutrients, and dietary isoflavone intake have been investigated in various observational studies for their potential relevance to puberty timing. Of them, six analyses suggest notable associations between dietary protein intakes and puberty timing: children in the highest groups of vegetable or animal protein experience their puberty onset up to 7 months later or 7 months earlier, respectively (Table 12.4). One study has shown a strong association between dietary isoflavones and puberty timing among girls, i.e., girls with high isoflavone intakes may experience the onset of breast development and peak height velocity approximately 7–8 months later (Table 12.4). Delays in pubertal timing in response to beneficial dietary habits (higher intakes of vegetable protein and isoflavone, and lower animal protein intakes) may be of substantial public health relevance: a later age at both peak height velocity and menarche is related to a

Table 12.4 Prospective studies on the relation between prepubertal dietary protein and isoflavone intake and puberty timing[a]

Author/Study	Outcomes/predictors/covariables	Results/conclusion
Animal protein intake		
Berkey et al. (2000) [79] Prospective study: 67 US girls	*Predictors*: animal protein intake at ages 1, 2, 3, 4, 5, 6, 7, 8, and 9 years *Assessment method*: dietary history interview *Outcomes*: age at menarche, age at peak height velocity (APHV), peak height growth velocity Covariates: age, energy intake	*Results/conclusion* Girls aged 3–5 with 1 SD higher animal protein (approximately 8 g/day) had their menarche 0.63 years earlier ($p<0.05$)
Gunther et al. (2010) [76] Prospective study: 112 German children (57 girls and 55 boys)	*Predictors*: total protein intake and animal protein intake at ages 1, 1.5–2, 3–4, and 5–6 years; protein intake estimated from 24-h urinary nitrogen excretion at ages 3–4 and 5–6 years *Assessment method*: 3-day weighed dietary records; 24-h urine collection *Outcomes*: ATO, APHV, age at menarche/voice break *Covariates*: sex, birth weight, breastfeeding, rapid weight gain between birth and age 2 years, maternal overweight, parental education, total energy intake, fat intake, fiber intake, prepubertal fat mass index SDS	*Results* According to the life-course plots, total or animal protein intake at age 5–6 years was of particular importance for pubertal timing ($p\leq0.06$) A higher total or animal protein intake at age 5–6 years was related to an earlier ATO. In the highest tertile of animal protein intake, ATO occurred 0.6 years earlier than in the lowest (tertile $(T)_1$: 9.6 (9.4–9.9) vs. T_3: 9.0 (8.7–9.3) years; $p=0.003$) Similar findings were seen for APHV ($p=0.001$) and the age at menarche/voice break ($p=0.02$) *Conclusion* These results suggest that animal protein intake in mid-childhood is related to pubertal timing
Kissinger et al. (1987) [80] Prospective study: 230 white US girls	*Predictors*: protein intake at age 9–15 years; food groups at age 9–15 years: (1) meat, poultry, fish and seafood, (2) dairy products, (3) meat analogues *Assessment method*: multiple 24-h recalls *Outcomes*: age at menarche *Covariates*: No	*Results/conclusion* A significant association was found between meat and age at menarche resulting in a 6-month earlier age of menarche among meat users than vegetarians ($p<0.025$) Total intake of protein was not associated with age at menarche
Remer et al. (2010) [81]	*Predictors*: animal protein intake and adrenal androgen (AA) 1 and 2 years before ATO	*Results*

(continued)

Table 12.4 (continued)

Author/Study	Outcomes/predictors/covariables	Results/conclusion
Prospective study: 109 German children (55 girls and 54 boys)	*Assessment method*: 3-day weighed dietary records; 24-h urine collection	Prepubertal animal protein intake was negatively associated with ATO and APHV ($p<0.05$) and tended to be negatively associated with age at menarche/voice break ($p=0.07$), these associations were independent of AA
	Outcomes: ATO, APHV, age at menarche and age at voice break, age at Tanner stage 2 for breast and genital development (B2_G2) and pubic hair (PH2)	*Conclusion* A higher animal protein intake may be involved independently of AA in an earlier attainment of pubertal growth development
	Covariates: sex, fat mass index at baseline, total energy intake SDS, urine volume related to body surface area, gestational age, birth weight, breastfeeding ≥2 weeks, and maternal overweight	
Rogers et al. (2010) [82]	*Predictors*: nutrients at ages 3, 7, and 10 years: total, animal, and vegetable protein; foods at ages 3, 7, and 10 years: total and oily fish, meat, dairy products, soy meat/textured vegetable protein, legumes	*Results* Total and animal protein intakes were positively associated with age at menarche ≤12 years 8 months (OR for 1 SD increase in protein 1.17 (1.07–1.28))
Prospective study: 3,298 British girls	*Assessment method*: FFQ at ages 3 and 7 years and 3-day dietary record at age 10 years *Outcomes*: age at menarche yes/no at age 12 years 8 months (median age in this cohort) *Covariates*: height and BMI at 7/10 years	Meat intake at age 7 years was strongly positively associated with reaching menarche by 12 years 8 months (OR 1.57 [1.03–2.37] in the highest vs. lowest category of meat consumption) *Conclusion* Higher intakes of protein and meat in early to mid-childhood may lead to earlier menarche
Vegetable protein intake		
Berkey et al. (2000) [79]	*Predictors*: vegetable protein at ages 1, 2, 3, 4, 5, 6, 7, 8, and 9 years	*Results/conclusion* Girls aged 3–5 years with 1 SD higher vegetable protein (approximately 3 g/day) experience their menarche 0.87 years later ($p<0.05$)
Prospective study: 67 US girls	*Assessment method*: dietary history interview	
	Outcomes: age at menarche, age at peak height velocity (APHV), peak height growth velocity	
	Covariates: age, energy intake	
de Ridder et al. (1991) [83]	*Predictors*: total protein intake and vegetable protein intake at age 10 and 12 years	*Results/conclusion*

Prospective study: 63 Dutch girls	*Assessment method*: 7-day food record *Outcomes*: breast development stage, plasma concentrations of gonadotropins, and plasma concentrations of estradiol at age 9–13 years (twice a year from 1986 to 1987) *Covariates*: energy intake, polysaccharides, height, and time of 7-day food record	Girls with higher vegetable protein intake at age 10 years had a lower stage of breast development at age 11.5 years ($p<0.05$)
Gunther et al. (2010) [76] Prospective study: 112 German children (57 girls and 55 boys)	*Predictors*: vegetable protein intake at ages 1, 1.5–2, 3–4, and 5–6 years; protein intake estimated from 24-h urinary nitrogen excretion at ages 3–4 and 5–6 years *Assessment method*: 3-day weighed dietary records; 24-h urine collection *Outcomes*: ATO, APHV, age at menarche/voice break *Covariates*: sex, birth weight, breastfeeding, rapid weight gain between birth and age 2 years, maternal overweight, parental education, total energy intake, fat intake, fiber intake, prepubertal fat mass index SDS	*Results/conclusion* According to the life-course plots, vegetable protein intake at age 3–4 years was of particular importance for pubertal timing ($p \leq 0.06$) Children whose diet was in the highest dietary vegetable protein tertile at age 3–4 years reached their ATO, APHV, and menarche/voice break approximately 0.5 years later than children in the lowest tertile ($p=0.02$–0.04)
Dietary isoflavone intake		
Cheng et al. (2010) [84] Prospective study: 227 German children (119 girls and 108 boys)	*Predictors*: dietary isoflavone and urinary isoflavone at 1 and 2 years before ATO *Assessment method*: 3-day weighed dietary records; 24-h urine collection *Outcomes*: ATO, APHV, age at menarche/voice break, pubertal stage for breast (B2) and gonadal (G2) development *Covariates*: birth weight, maternal overweight, maternal education, smoking in the household, body composition and *Z* score of energy intake in childhood	*Results* Girls in the highest isoflavone tertile experienced their Tanner stage 2 for breast development approximately 0.7 years later and reached PHV approximately 0.6 years later than girls in the lowest isoflavone tertile (age at B2 was 10.7 (10.4–10.9) vs. 10.0 (9.7–10.3) years ($p=0.04$), and age at PHV 11.9 (11.6–12.2) vs. 11.3 (11.0–11.6) years ($p=0.04$)) In boys, no association was found between dietary isoflavone and pubertal markers In both girls and boys, urinary isoflavone was not associated with pubertal markers *Conclusion* Girls, but not boys, with higher prepubertal isoflavone intakes appear to enter puberty at a later age

[a]Table is modified from a review from Cheng et al. [77]

reduced risk of breast cancer, and a later menarcheal age is also associated with a lower total mortality. These nutrients identified as beneficial for later puberty onset may indicate a specific relevance of diet quality. More analyses focusing on the overall diet quality are thus warranted.

Public Health Consideration

Body composition and body fat distribution change rapidly during growth; in addition, puberty onset is to date considered to be of general public health relevance for both sexes. Emerging evidence suggests a relevance of diet quality to both body composition development and puberty timing. Future studies in diverse population are needed to identify which aspects of diet quality or which pattern is of particular relevance. This information could be of major public health relevance, since long-term eating habits are shaped during childhood and adolescence. While it is generally accepted that poor diet quality could conduce overweight/obesity, the importance of diet quality for puberty onset, which is relevant to health in later life, is currently not recognized. Educational interventions focusing on diet quality should not be limited to children and adolescent individuals. Parents with good dietary awareness (or nutritional knowledge) are thought to be more likely to make healthy food choices for their children [78]. Moreover, the intake preference of children is influenced not only by the types of foods present in the household but also by the amount of those foods available to them. The parental influence of food choice should thus be taken into account for the promotion of dietary habits of children. In addition to family dynamics, school factors and the "built environment", which refers to the neighborhoods and communities in which children live, also play a role in influencing the diet quality among youth. Finally, the food industries should be urged to increase their efforts to provide healthier food choices to beneficially influence the diet quality of children and adolescents.

Conclusions

In conclusion, emerging evidence suggests that modifying diet quality in early life could offer preventive potential for health in later life with regard to later body composition or puberty onset. Current approaches focusing on single nutrients and foods have identified only few dietary factors in childhood as potentially relevant for body composition. With regard to puberty timing, current evidence suggests a beneficial influence of dietary protein and isoflavone intake, which may indicate a particular relevance of overall diet quality. The approach of dietary pattern analyses, which considers the interaction of nutrients and foods, may provide new insights into the effect of diet as a whole and should be adapted for this issue.

References

1. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007–2008. JAMA. 2010;303(3):242–9.
2. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. Lancet. 2002;360(9331):473–82.
3. Farooqi IS, O'Rahilly S. Genetic factors in human obesity. Obes Rev. 2007;8 Suppl 1:37–40.
4. Velie EM, Nechuta S, Osuch JR. Lifetime reproductive and anthropometric risk factors for breast cancer in postmenopausal women. Breast Dis. 2005;24:17–35.
5. Garner MJ, Turner MC, Ghadirian P, Krewski D. Epidemiology of testicular cancer: an overview. Int J Cancer. 2005;116(3):331–9.
6. Jacobsen BK, Oda K, Knutsen SF, Fraser GE. Age at menarche, total mortality and mortality from ischaemic heart disease and stroke: the Adventist Health Study, 1976–88. Int J Epidemiol. 2009;38(1):245–52.
7. Remsberg KE, Demerath EW, Schubert CM, Chumlea WC, Sun SS, Siervogel RM. Early menarche and the development of cardiovascular disease risk factors in adolescent girls: the Fels Longitudinal Study. J Clin Endocrinol Metab. 2005;90(5):2718–24.
8. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: design and applications. J Am Diet Assoc. 1995;95(10):1103–8.
9. Hoffmann K, Schulze MB, Schienkiewitz A, Nothlings U, Boeing H. Application of a new

statistical method to derive dietary patterns in nutritional epidemiology. Am J Epidemiol. 2004;159(10):935–44.
10. Godfrey KM, Barker DJ. Fetal nutrition and adult disease. Am J Clin Nutr. 2000;71(5 Suppl):1344S–52.
11. Rolland-Cachera MF, Deheeger M, Guilloud-Bataille M, Avons P, Patois E, Sempe M. Tracking the development of adiposity from one month of age to adulthood. Ann Hum Biol. 1987;14(3):219–29.
12. Dietz WH. Critical periods in childhood for the development of obesity. Am J Clin Nutr. 1994;59(5):955–9.
13. Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. N Engl J Med. 1976;295(7):349–53.
14. Braddon FE, Rodgers B, Wadsworth ME, Davies JM. Onset of obesity in a 36 year birth cohort study. Br Med J (Clin Res Ed). 1986;293(6542):299–303.
15. Hannon TS, Janosky J, Arslanian SA. Longitudinal study of physiologic insulin resistance and metabolic changes of puberty. Pediatr Res. 2006;60(6):759–63.
16. de Ridder CM, Thijssen JH, Bruning PF, Van den Brande JL, Zonderland ML, Erich WB. Body fat mass, body fat distribution, and pubertal development: a longitudinal study of physical and hormonal sexual maturation of girls. J Clin Endocrinol Metab. 1992;75(2):442–6.
17. Robinson SM, Godfrey KM. Feeding practices in pregnancy and infancy: relationship with the development of overweight and obesity in childhood. Int J Obes (Lond). 2008;32 Suppl 6:S4–10.
18. Arenz S, Ruckerl R, Koletzko B, von Kries R. Breastfeeding and childhood obesity—a systematic review. Int J Obes Relat Metab Disord. 2004;28(10):1247–56.
19. Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. Pediatrics. 2005;115(5):1367–77.
20. Günther AL, Buyken AE, Kroke A. Protein intake during the period of complementary feeding and early childhood and the association with body mass index and percentage body fat at 7 y of age. Am J Clin Nutr. 2007;85(6):1626–33.
21. Koletzko B. Long-term consequences of early feeding on later obesity risk. In: Rigo J, Ziegler EE, editors. Protein and energy requirements in infancy and childhood. Basel: Karger AG; 2006. p. 1–18.
22. Herbst A, Diethelm K, Cheng G, Alexy U, Icks A, Buyken AE. Direction of associations between added sugar intake in early childhood and body mass index at age 7 years may depend on intake levels. J Nutr. 2011;141(7):1348–54.
23. Irwin Jr CE, Igra V, Eyre S, Millstein S. Risk-taking behavior in adolescents: the paradigm. Ann N Y Acad Sci. 1997;817:1–35.
24. Bogaert N, Steinbeck KS, Baur LA, Brock K, Bermingham MA. Food, activity and family—environmental vs biochemical predictors of weight gain in children. Eur J Clin Nutr. 2003;57(10):1242–9.
25. Maffeis C, Talamini G, Tato L. Influence of diet, physical activity and parents' obesity on children's adiposity: a four-year longitudinal study. Int J Obes Relat Metab Disord. 1998;22(8):758–64.
26. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children. Int J Obes (Lond). 2008;32(4):586–93.
27. Kral TV, Berkowitz RI, Stunkard AJ, Stallings VA, Brown DD, Faith MS. Dietary energy density increases during early childhood irrespective of familial predisposition to obesity: results from a prospective cohort study. Int J Obes (Lond). 2007;31(7):1061–7.
28. Gunther AL, Stahl LJ, Buyken AE, Kroke A. Association of dietary energy density in childhood with age and body fatness at the onset of the pubertal growth spurt. Br J Nutr. 2011;106(3):345–9.
29. Butte NF, Cai G, Cole SA, Wilson TA, Fisher JO, Zakeri IF, et al. Metabolic and behavioral predictors of weight gain in Hispanic children: the Viva la Familia Study. Am J Clin Nutr. 2007;85(6):1478–85.
30. McCaffrey TA, Rennie KL, Kerr MA, Wallace JM, Hannon-Fletcher MP, Coward WA, et al. Energy density of the diet and change in body fatness from childhood to adolescence; is there a relation? Am J Clin Nutr. 2008;87(5):1230–7.
31. Klesges RC, Klesges LM, Eck LH, Shelton ML. A longitudinal analysis of accelerated weight gain in preschool children. Pediatrics. 1995;95(1):126–30.
32. Skinner JD, Bounds W, Carruth BR, Morris M, Ziegler P. Predictors of children's body mass index: a longitudinal study of diet and growth in children aged 2–8 y. Int J Obes Relat Metab Disord. 2004;28(4):476–82.
33. Lee Y, Mitchell DC, Smiciklas-Wright H, Birch LL. Diet quality, nutrient intake, weight status, and feeding environments of girls meeting or exceeding recommendations for total dietary fat of the American Academy of Pediatrics. Pediatrics. 2001;107(6):E95.
34. Carruth BR, Skinner JD. The role of dietary calcium and other nutrients in moderating body fat in preschool children. Int J Obes Relat Metab Disord. 2001;25(4):559–66.
35. Robertson SM, Cullen KW, Baranowski J, Baranowski T, Hu S, de Moor C. Factors related to adiposity among children aged 3 to 7 years. J Am Diet Assoc. 1999;99(8):938–43.
36. Newby PK, Peterson KE, Berkey CS, Leppert J, Willett WC, Colditz GA. Dietary composition and weight change among low-income preschool children. Arch Pediatr Adolesc Med. 2003;157(8):759–64.
37. Magarey AM, Daniels LA, Boulton TJ, Cockington RA. Does fat intake predict adiposity in healthy children and adolescents aged 2–15 y? A longitudinal analysis. Eur J Clin Nutr. 2001;55(6):471–81.

38. Jago R, Baranowski T, Baranowski JC, Thompson D, Greaves KA. BMI from 3–6 y of age is predicted by TV viewing and physical activity, not diet. Int J Obes (Lond). 2005;29(6):557–64.
39. Rolland-Cachera MF, Deheeger M, Akrout M, Bellisle F. Influence of macronutrients on adiposity development: a follow up study of nutrition and growth from 10 months to 8 years of age. Int J Obes Relat Metab Disord. 1995;19(8):573–8.
40. Gunther AL, Remer T, Kroke A, Buyken AE. Early protein intake and later obesity risk: which protein sources at which time points throughout infancy and childhood are important for body mass index and body fat percentage at 7 y of age? Am J Clin Nutr. 2007;86(6):1765–72.
41. Klesges RC, Isbell TR, Klesges LM. Relationship between dietary restraint, energy intake, physical activity, and body weight: a prospective analysis. J Abnorm Psychol. 1992;101(4):668–74.
42. Pereira MA, Ludwig DS. Dietary fiber and body-weight regulation. Observations and mechanisms. Pediatr Clin North Am. 2001;48(4):969–80.
43. Davis JN, Alexander KE, Ventura EE, Toledo-Corral CM, Goran MI. Inverse relation between dietary fiber intake and visceral adiposity in overweight Latino youth. Am J Clin Nutr. 2009;90(5):1160–6.
44. Iqbal Kring SI, Heitmann BL. Fiber intake, not dietary energy density, is associated with subsequent change in BMI z-score among sub-groups of children. Obes Facts. 2008;1(6):331–8.
45. Buyken AE, Cheng G, Gunther AL, Liese AD, Remer T, Karaolis-Danckert N. Relation of dietary glycemic index, glycemic load, added sugar intake, or fiber intake to the development of body composition between ages 2 and 7 y. Am J Clin Nutr. 2008;88(3): 755–62.
46. Cheng G, Karaolis-Danckert N, Libuda L, Bolzenius K, Remer T, Buyken AE. Relation of dietary glycemic index, glycemic load, and fiber and whole-grain intakes during puberty to the concurrent development of percent body fat and body mass index. Am J Epidemiol. 2009;169(6):667–77.
47. Slavin JL, Martini MC, Jacobs Jr DR, Marquart L. Plausible mechanisms for the protectiveness of whole grains. Am J Clin Nutr. 1999;70(3 Suppl):459S–63.
48. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr. 1981;34(3):362–6.
49. Williams CL, Strobino BA. Childhood diet, overweight, and CVD risk factors: the Healthy Start project. Prev Cardiol. 2008;11(1):11–20.
50. Ruottinen S, Niinikoski H, Lagstrom H, Ronnemaa T, Hakanen M, Viikari J, et al. High sucrose intake is associated with poor quality of diet and growth between 13 months and 9 years of age: the special Turku Coronary Risk Factor Intervention Project. Pediatrics. 2008;121(6):e1676–85.
51. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. Am J Clin Nutr. 2008;87(6):1662–71.
52. Malik VS, Willett WC, Hu FB. Sugar-sweetened beverages and BMI in children and adolescents: reanalyses of a meta-analysis. Am J Clin Nutr. 2009;89(1):438–9; author reply 439–440.
53. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. Am J Public Health. 2007;97(4):667–75.
54. Mattes RD, Shikany JM, Kaiser KA, Allison DB. Nutritively sweetened beverage consumption and body weight: a systematic review and meta-analysis of randomized experiments. Obes Rev. 2011;12(5): 346–65.
55. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. Is sugar-sweetened beverage consumption associated with increased fatness in children? Nutrition. 2007;23(7–8):557–63.
56. Dubois L, Farmer A, Girard M, Peterson K. Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. J Am Diet Assoc. 2007;107(6):924–34; discussion 934–925.
57. Fiorito LM, Marini M, Francis LA, Smiciklas-Wright H, Birch LL. Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. Am J Clin Nutr. 2009;90(4):935–42.
58. Kral TV, Stunkard AJ, Berkowitz RI, Stallings VA, Moore RH, Faith MS. Beverage consumption patterns of children born at different risk of obesity. Obesity (Silver Spring). 2008;16(8):1802–8.
59. Lim S, Zoellner JM, Lee JM, Burt BA, Sandretto AM, Sohn W, et al. Obesity and sugar-sweetened beverages in African-American preschool children: a longitudinal study. Obesity (Silver Spring). 2009;17(6): 1262–8.
60. Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA. Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study. Int J Obes (Lond). 2006;30(7): 1091–3.
61. Libuda L, Alexy U, Sichert-Hellert W, Stehle P, Karaolis-Danckert N, Buyken AE, et al. Pattern of beverage consumption and long-term association with body-weight status in German adolescents–results from the DONALD study. Br J Nutr. 2008;99(6): 1370–9.
62. Laurson K, Eisenmann JC, Moore S. Lack of association between television viewing, soft drinks, physical activity and body mass index in children. Acta Paediatr. 2008;97(6):795–800.
63. Vanselow MS, Pereira MA, Neumark-Sztainer D, Raatz SK. Adolescent beverage habits and changes in weight over time: findings from Project EAT. Am J Clin Nutr. 2009;90(6):1489–95.
64. Feskanich D, Rockett HR, Colditz GA. Modifying the Healthy Eating Index to assess diet quality in children and adolescents. J Am Diet Assoc. 2004;104(9): 1375–83.

65. Hurley KM, Oberlander SE, Merry BC, Wrobleski MM, Klassen AC, Black MM. The healthy eating index and youth healthy eating index are unique, non-redundant measures of diet quality among low-income. African American adolescents. J Nutr. 2009;139(2):359–64.
66. Kranz S, Findeis JL, Shrestha SS. Use of the Revised Children's Diet Quality Index to assess preschooler's diet quality, its sociodemographic predictors, and its association with body weight status. J Pediatr (Rio J). 2008;84(1):26–34.
67. Jennings A, Welch A, van Sluijs EM, Griffin SJ, Cassidy A. Diet quality is independently associated with weight status in children aged 9–10 years. J Nutr. 2011;141(3):453–9.
68. Golley RK, Hendrie GA, McNaughton SA. Scores on the dietary guideline index for children and adolescents are associated with nutrient intake and socio-economic position but not adiposity. J Nutr. 2011;141(7):1340–7.
69. Cheng G, Gerlach S, Libuda L, Kranz S, Gunther AL, Karaolis-Danckert N, et al. Diet quality in childhood is prospectively associated with the timing of puberty but not with body composition at puberty onset. J Nutr. 2010;140(1):95–102.
70. Cutler GJ, Flood A, Hannan PJ, Slavin JL, Neumark-Sztainer D. Association between major patterns of dietary intake and weight status in adolescents. Br J Nutr. 2012;108(2):349–56.
71. Wosje KS, Khoury PR, Claytor RP, Copeland KA, Hornung RW, Daniels SR, et al. Dietary patterns associated with fat and bone mass in young children. Am J Clin Nutr. 2010;92(2):294–303.
72. Moisan J, Meyer F, Gingras S. A nested case–control study of the correlates of early menarche. Am J Epidemiol. 1990;132(5):953–61.
73. Merzenich H, Boeing H, Wahrendorf J. Dietary fat and sports activity as determinants for age at menarche. Am J Epidemiol. 1993;138(4):217–24.
74. Karaolis-Danckert N, Buyken AE, Sonntag A, Kroke A. Birth and early life influences on the timing of puberty onset: results from the DONALD (DOrtmund Nutritional and Anthropometric Longitudinally Designed) Study. Am J Clin Nutr. 2009;90(6):1559–65.
75. Blell M, Pollard TM, Pearce MS. Predictors of age at menarche in the newcastle thousand families study. J Biosoc Sci. 2008;40(4):563–75.
76. Gunther AL, Karaolis-Danckert N, Kroke A, Remer T, Buyken AE. Dietary protein intake throughout childhood is associated with the timing of puberty. J Nutr. 2010;140(3):565–71.
77. Cheng G, Buyken AE, Shi L, Karaolis-Danckert N, Kroke A, Wudy S, et al. Beyond overweight: nutrition as an important lifestyle factor influencing the puberty timing. Nutr Rev. 2012;70(3):133–52.
78. Variyam JN, Blaylock J, Lin B, Ralston K, Smallwood D. Mother's nutrition knowledge and children's dietary intakes. Am J Agric Econ. 1999;81:373–84.
79. Berkey CS, Gardner JD, Frazier AL, Colditz GA. Relation of childhood diet and body size to menarche and adolescent growth in girls. Am J Epidemiol. 2000;152(5):446–52.
80. Kissinger DG, Sanchez A. The association of dietary factors with the age of menarche. Nutr Res. 1987;7:471–9.
81. Remer T, Shi L, Buyken AE, Maser-Gluth C, Hartmann MF, Wudy SA. Prepubertal adrenarchal androgens and animal protein intake independently and differentially influence pubertal timing. J Clin Endocrinol Metab. 2010;95(6):3002–9.
82. Rogers IS, Northstone K, Dunger DB, Cooper AR, Ness AR, Emmett PM. Diet throughout childhood and age at menarche in a contemporary cohort of British girls. Public Health Nutr. 2010;13(12):2052–63.
83. de Ridder CM, Thijssen JH. Van 't Veer P, van Duuren R, Bruning PF, Zonderland ML, Erich WB. Dietary habits, sexual maturation, and plasma hormones in pubertal girls: a longitudinal study. Am J Clin Nutr. 1991;54(5):805–13.
84. Cheng G, Remer T, Prinz-Langenohl R, Blaszkewicz M, Degen GH, Buyken AE. Relation of isoflavones and fiber intake in childhood to the timing of puberty. Am J Clin Nutr. 2010;92(3):556–64.

13 Diet Quality, in Relation to TV Viewing and Video Games Playing

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Key Points

- An unbalanced diet is a very important risk factor for several chronic diseases such as obesity, stroke, cancer, and type 2 diabetes mellitus.
- Television (TV) viewing and video gaming have emerged as significant factors contributing to the current obesity epidemic and the corresponding low diet quality.
- The assessment of diet quality is performed with the assessment of the necessary micro- and macronutrient elements in children's and adolescents' diet or with the use of composite health quality indices.
- Children are more prone to poor diet quality which is attributed to the vulnerability of their developmental phase to food advertising on TV and also to the poor diet quality of their parents or caregivers.
- In adolescence, besides food advertising, snacking while watching TV plays also a significant role in poor diet quality food choices.
- Another possible way in which TV may influence fast food intake is through hidden food consuming messages embedded within program content.
- Important food markers for assessing the relation between TV watching and poor diet quality are snacks and soft drinks or sugar-added beverages.
- Video gaming has emerged as a rapidly evolving favorite screen-related activity in childhood recently and its effect on diet quality is poorly studied.
- Video gaming has been positively related with food overconsumption and increased caloric intake, indicating an association with poor diet quality choices.

Keywords

Television viewing • Video gaming • Diet • Quality • Children • Adolescents

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Abbreviations

AMQI	Adolescent Micronutrient Quality Index
DQI	Diet Quality Index
DQI-I	Diet Quality Index-International
HEI	Healthy Eating Index
TV	Television
USA	United States of America
USDA	Department of Agriculture of the USA
WHO	World Health Organization

Introduction

An unbalanced diet is a very important risk factor for several chronic diseases such as obesity, stroke, cancer, and type 2 diabetes mellitus. These diseases are contributing to the rise of the proportion of preliminary deaths, to the limitation of general life quality and are considered as major public health problems. Thus, it is of primary importance to monitor the population's diet and to help improve dietary habits by providing the necessary nutritional information and the corresponding education. Moreover, it is vital to identify groups or individuals whose consumption of certain nutrients is systematically too high or too low and their diet quality is characterized as poor. These individuals are in greater danger of developing nutrient-related conditions, thus early recognition of the population in danger could lead to interventions that could prevent the arise of such pathology.

Time spent engaged in sedentary behaviors, such as television (TV) viewing and video gaming, is considered to be one of the factors underlying the increasing prevalence of overweight and obesity observed around the world [1]. Positive relations between TV viewing and adiposity are constantly found, with several studies reporting the fact that men and women watching the most TV have a 2–4-fold increased risk of obesity compared with those watching the least TV [2, 3].

Video games have vast mass call and are present in the daily schedule of most children and teenagers. As of 1999, video games account for approximately 30 % of the USA toy market, which helped the video game industry to earn between $6 and $9 billion, thus making more profit than even the motion picture industry [4]. A 2004 estimate of media consumption showed that video game play and nonschool-related computer access occupy approximately 2 h of a typical child's day [5]. The increased prevalence of electronic game play (computer and video games) has alarmed the researchers and urged them to investigate the effect of this media type on various aspects of health [6].

The purpose of this chapter is to review the current literature in order to summarize the effect of the aforementioned sedentary activities (TV viewing and video gaming) in the diet quality of children and adolescence. Furthermore, a summary of the indexes of assessing diet quality will be further discussed.

Definition and Assessment Methods of Diet Quality

Good nutrition is a very important part of every child's healthy life. Diet quality reflects a dietary pattern in accordance with all the necessary micro and macronutrient elements needed to maintain health and to support growth and development. As a reference value, the most commonly used in the international literature are the Reference Daily Intake Tables used from the Department of Agriculture of the USA (USDA) [7].

Diet quality indices are often used to provide a summary of diet quality rather than observing single food and nutrient items [8]. There are several indexes proposed for assessing diet quality in various age groups which are summarized in Table 13.1. The Revised Children's Diet Quality Index (DQI) has been developed by Kranz et al., using the USA dietary recommended intake guidelines for preschool-aged children (aged 2–5 years old). This index evaluates 12 dietary components (added sugar, total fat, linoleic and linolenic fatty acids, docosahexaenoic acid,

Table 13.1 Summary of the main indices used in the diet quality research in children and adolescents

Health index	Components
The Revised Children's Diet Quality [8]	Added sugar
	Total fat
	Linoleic and linolenic fatty acids docosahexaenoic acid
	Eicosapentaenoic acid
	Total grains
	Whole grains
	Vegetables
	Fruits
	100 % fruit juice
	Diary and iron intake
Diet Quality Index (DQI) [9]	Dietary diversity (eight food groups)
	Dietary quality (preferred foods within a food group are given higher scores)
	Dietary equilibrium (higher scores if adequate but moderate intake of each food element is reassured)
	Meal index (number of main meals per day)
Healthy Eating Index (HEI) [10]	Total and whole fruit
	Total vegetables
	Dark, green and orange vegetables and legumes
	Total and whole grains
	Milk
	Meat
	Beans
	Oils and calories from solid fats
	Alcoholic beverages
	Added sugars
	Saturated fats
	Sodium
Adolescent Micronutrient Quality Index (AMQI) [12]	Cereals and millets
	Legumes
	Milk and milk products
	Vegetables (green leafy vegetables, other vegetables, roots and tubers)
	Fruits
	Sugar
	Fats/oils (visible)
	At least 50 % grains as whole grains
	At least 50 % legumes being micronutrient-dense
	Food variety based on all subgroups and weekly variety in vegetables and fruits
	Sprouts/fermented foods/salads
	Tea/coffee with meals
	Fried foods

(continued)

Table 13.1 (continued)

Health index	Components
Diet Quality Index-International (DQI-I) [13]	Variety
	Overall food group variety
	Within-group variety from protein source
	Adequacy
	Vegetable group
	Fruit group
	Grain group
	Fiber
	Protein
	Iron
	Calcium
	Vitamin C
	Moderation
	Total fat
	Saturated fat
	Cholesterol
	Sodium
	"Empty calorie food"
	Overall balance
	Macronutrient ratio (carbohydrate:protein:fat)
	Fatty acid ratio (PUFA + MUFA/SFA)

PUFA polyunsaturated fatty acids, *MUFA* monounsaturated fatty acids, *SFA* saturated fatty acids

eicosapentaenoic acid, total grains, whole grains, vegetables, fruits, 100 % fruit juice, diary and iron intake) as well as total energy intake and television time spent with an interaction term [8]. For the same age group another DQI had been developed as a tool for assessing the compliance of Flemish preschoolers with the Flemish Food-Based Dietary Guidelines for preschool children. This index consists of four major components: dietary diversity (eight food groups), dietary quality (preferred foods within a food group are given higher scores), dietary equilibrium (higher scores if adequate but moderate intake of each food element is reassured), and meal index (number of main meals per day). The DQI has been validated by comparing the DQI scores derived from 3 days' records with nutrient intake profiles and has been found to be a reliable quality index [9]. An index that was not specifically developed for children but can be applied to them, the

Healthy Eating Index (HEI), was revised in 2005 after the publication of the 2005 Dietary Guidelines in order to measure diet quality in terms of compliance with the key diet-related recommendations of the 2005 Dietary Guidelines. The components of the HEI include all the major food groups (total and whole fruit, total vegetables, dark, green and orange vegetables and legumes, total and whole grains, milk, meat, beans, oils and calories from solid fats, alcoholic beverages, added sugars, saturated fats, and sodium) [10]. A modified version of the previous HEI designed to assess diet quality in children aged 9–14 years of age had also been published [11]. Also diet quality indexes for specific population has been formulated, like the Adolescent Micronutrient Quality Index (AMQI) for assessing the micronutrient adequacy in adolescent girls consuming a lacto-vegetarian diet [12] and the Mediterranean adaptation of the Diet Quality Index-International (DQI-I) [13].

Since validated diet quality indexes are lacking from the literature, research has focused on the consumption of specific food groups or nutrients, as a proxy to the overall children's diet quality. The selection of these food groups and nutrients is based on the previous knowledge about the protective or un-protective effect in various aspects of childhood or adult life. For example, sugar-sweetened beverage intake is associated with weight gain in children [14, 15]. Also, calcium intake is associated with decreased blood pressure among children as well as increased bone mineral density among them [16, 17]. Fruit and vegetables may play a protective role against the development of cancer and coronary heart disease while *trans*- and saturated fat consumption has a negative association with it [18, 19]. However, the approach of assessing single nutrients or food items, instead of assessing dietary patterns, could introduce some bias since children do not consume isolated nutrients, but eat meals consisting of a variety of foods with complex combinations of nutrients. This way of dietary assessment is indicated when there is lack of age-appropriated, validated diet quality indexes to capture the whole eating patterns and is often observed in recent studies.

TV Viewing and Diet Quality in Childhood and Young Adulthood

Up to 17 years of age, a US child has spent more hours in watching television than going to school (15,000–18,000 compared to 12,000 h). Thus, TV viewing has been connected early to the various aspects of children's diet patterns. Table 13.2 summarizes several studies regarding the association of TV viewing with diet quality during childhood. The focus of the studies in this age group is attributed to the venerability of this developmental phase to food advertising on TV. There is evidence supporting that TV watching is closely related to snacking and their snacking preferences are most of the time affected by advertised foods which are more likely to have high concentrations of fat, sugar, and sodium [20]. Batada et al. have reported that 91 % of food advertisements during Saturday morning children's television programming in the USA were for foods or beverages high in fat, sodium, or added sugars or were low in nutrients and 18 % of food advertisements were about snack foods [21]. Moreover, children exposed to food advertisements are more prone to request that their parents purchase the dietary products that are most advertised [22, 23]. Exposure to as little as one or two times to short food commercials is enough to alter a preschool-age child's food preferences [24]. During the development of eating patterns the role of environment has a great influence. Children become developmentally capable of making the transition to family foods, after the age of one and their internal regulatory mechanism for hunger and satiety is influenced by cultural patterns. The preschool period is essential to the origins of healthy eating patterns and due to the sensitivity of the developmental immaturity of children it is being thoroughly investigated by the researchers. Also young children's TV and eating habits often reflect those of their parents or caregivers. For instance, children who watch greater amounts of TV may do so because those caring for them may frequently watch TV. Therefore, the association we found between young children's television/video viewing and

Table 13.2 Summary of studies assessing TV viewing and diet quality in childhood and young adulthood

Study	Study design	Sample	Exposure	Estimates of association
Miller et al. (2008) [30]	Cross-sectional	3-Year-old 613 boys 590 girls from USA	1 h TV viewing per day	0.06 servings/day [95 % CI 0.03, 0.10] for sugar-sweetened beverages 0.32 servings/month [95 % CI 0.16, 0.49] for fast food 0.06 servings/day [95 % CI 0.02, 0.09] for red/processed meat 48.7 kcal/day [95 % CI 18.7, 78.6] for total energy intake 0.05 [95 % CI 0.03, 0.07] for % energy intake from trans −0.18 servings/day [95 % CI −0.32, −0.05] for fruits and vegetables 24.6 mg/day [95 % CI −41.0, −8.1] for calcium −0.44 g/day [95 % CI −0.65, −0.22] for dietary fiber
Taveras et al. (2006) [37]	Cross-sectional	2–6 years old 240 parents of children USA	1 h increase of TV/ video watched per day	Adjusted OR for consuming fast food ≥1 time per week: 1.55 (95 % CI, 1.04–2.31)
Parvanta et al. (2010) [33]	Cross-sectional	6–18 years old $n = 1,552$ China	TV watching hours per week Reported paying attention to TV commercials (yes vs. no)	aOR: 1.00; 95 % CI = 0.98–1.02 per TV watching hour for requesting snacks seen on TV aOR: 3.42; 95 % CI = 2.55–4.60 of requesting snacks seen on TV when children paid attention to the commercials aOR 1.60; 95 % CI = 1.23–2.07 for eating snacks seen on TV when children paid attention to the commercials
Liang et al. (2011) [39]	Cross-sectional	11 years old students $n = 4,966$ Canada	Hours of television watching (1–2 h/d, 3–4 h/d, ≥5 h/d vs. <1 h/d) Supper in front of television (1–2 times/ week, 3–4 times/week, ≥5 times/week vs. <1 time/week)	OR for soft drinks: 2.46 95 % CI = 1.54–3.93 for ≥5 h/d vs. <1 h/d 0.57; 95 % CI 0.24–0.89 % of sugar energy from carbohydrate energy for 3–4 h/d vs. <1 h/d 2.20; 95 % CI 0.29–4.10 % of energy from snack foods for ≥5 h/d vs. <1 h/d −0.09; 95 % CI = −0.16, −0.03 daily servings of fruits and vegetables for 3–4 h/d vs. <1 h/d −1.73; 95 % CI = −3.35, −0.10 mean difference in Diet Quality Index for ≥5 h/d vs. <1 h/d OR for overweight: 2.42 95 % CI = 1.54–3.79 for ≥5 h/d vs. <1 h/d

(continued)

Table 13.2 (continued)

Study	Study design	Sample	Exposure	Estimates of association
Manios et al. (2009) [41]	Cross-sectional	1–5 years old $n = 2{,}374$ Greece	≥2 h/d watching TV vs. ≤2 h/d	46.5; 95 % CI = 12.2–80.8 kcal of total energy intake OR: 1.31; 95 % CI = 1.00–1.69 of ≥5 exchanges of fat OR: 1.51; 95 % CI 1.14–2.00 of exchanges of meat OR: 1.31; 95 % CI 1.01–1.67 if exchanges of other carbohydrate
Dubois et al. (2008) [42]	Longitudinal	Birth to 4.5 years of age $n = 2{,}013$ Canada	Frequency of eating while watching TV (meal or lunch using eat while-TV-watching index)	OR: 3.57; $p < 0.05$ for drinking soft drinks when eating snacks while watching TV every day vs. never OR:2.34; $p < 0.05$ for drinking soft drinks while eating when watching TV three of four times daily vs. less than once daily
Jackson et al. (2009) [58]	Cross-sectional	2–6 years $n = 89$ UK	1 h watching TV per day	1.095 kg increase in weight per extra hour of TV viewing
Jago et al. (2005) [43]	Cohort	Start age: 3–4 years $n = 149$ Follow-up time: 3 years USA	Minutes of TV per day	Beta = 0.046 kg/m^2 ($p < 0.001$) increase in per minute increase of TV watching
Fieldman et al. (2007) [34]	Cross-sectional	Mean age: 14.9 years $n = 4{,}746$ USA	Watch TV while eating family meals	1.3 median daily servings of vegetables for family meals with TV vs. 1.4 with no TV, $p < 0.001$ 0.40 median daily servings of dark green/yellow vegetables for family meals with TV vs. 0.43 with no TV, $p < 0.001$ 5.1 median daily servings of grains for family meals with TV vs. 5.3 with no TV, $p = 0.02$ 0.56 median daily servings of fried foods for family meals with TV vs. 0.54 with no TV, $p < 0.001$ 2.4 median daily servings of snack food for family meals with TV vs. 2.2 with no TV, $p = 0.002$
Fitzpatrick et al. (2007) [61]	Cross-sectional	1–4 years $n = 1{,}478$ USA	Number of days per week the television was on during dinner	aOR = 0.95; 95 % CI 0.91–0.99 for serving fruits per night of watching television with dinner aOR = 0.94; 95 % CI 0.90–0.98 for serving vegetables per night of watching television with dinner

(continued)

Table 13.2 (continued)

Study	Study design	Sample	Exposure	Estimates of association
Francis et al. (2006) [59]	Experimental	3–5 years old preschoolers $n=24$ USA	(a) 22-min cartoon video on TV (b) Parental reporting of children's eating during TV viewing at home	Significant higher intake for both snack and lunch meals in the no TV condition in the experimental condition ($t(24)=3.1$, $p<0.01$ and $t(24)=4.2$, $p<0.001$, respectively) Significant higher intake for lunch intakes ($r=0.56$, $p<0.05$) for children reported watching more daily hours of TV at home
Francis et al. (2003) [44]	Longitudinal	Start age: 5-year-old girls Assessment: at 7 and 9 years of age $n=173$ USA	Hours of watching TV per day	Beta = 0.30 kg/m^2 ($p<0.001$) for BMI at age 9 in girls with at least one overweight parent
Boynton-Jaret et al. (2003) [31]	Cohort	Mean start age: 11.7 years Follow-up time = 19 months $n=548$ USA	Hours of watching TV per day	Mean reduction of daily fruit and vegetables consumption: 0.14 serving/day per additional hour of television viewed, adjusted for several confounders
Wiecha et al. (2006) [45]	Cohort	Mean start age: 11.7 years Follow-up time = 18 months $n=548$ USA	Hours of watching TV per day	Mean increase in all foods commonly advertised on television (FCAT-soft drinks, fried foods, and snacks): 0.60 servings/day per 1 h/d increase ($p=0.001$)
Vereecken et al. (2007) [40]	Cross-sectional	11–15 years old $n=162{,}305$ children Several countries from Europe, USA, and Israel	Hours of watching TV per day	Statistically significant increase for the effect of TV viewing to the daily consumption of soft drinks, sweets and decrease in the daily consumption of vegetables and fruit
Pearson et al. (2011) [28]	Longitudinal	Start age: 12–15 years old (T1) Follow-up time = 2 years (T2) $n=1{,}729$ Australia	Eating snacks while watching TV	Beta: 0.26; 95 % CI (0.21–0.31) for energy-dense snack consumption in T2 when adolescents eating snacks while watching TV Beta: 0.14; 95 % CI (0.09–0.20) for energy-dense drink consumption in T2 when adolescents eating snacks while watching TV Beta: –0.06; 95 % CI (–0.10 to 0.03) for fruit consumption in T2 when adolescents eating snacks while watching TV
French et al. (2001) [38]	Cross-sectional	Mean age: 14.9 years $n=4{,}682$ USA	Hours of watching TV on average per weekday and weekend day	More than 3 times/week eating in fast food restaurants was significantly positively associated with weekday television viewing in boys and girls ($p<0.001$) and only with weekend television viewing in girls ($p<0.001$)

(continued)

Table 13.2 (continued)

Study	Study design	Sample	Exposure	Estimates of association
Barr-Anderson et al. (2009) [29]	Longitudinal	Cohort 1: $n=564$ middle school students Cohort 2: $n=1,366$ high school students Follow-up time: 5 years USA	Heavy television viewers (≥5 h/d) vs. moderate (2–5 h/d) and limited (<2 h/d)	Younger cohort: 1.72 (0.11) fruit servings/d for heavy TV viewers vs. 1.9 (0.07) and 2.06 (0.09) for moderate and limited, respectively ($p=0.009$) Older cohort: 1.25 (0.07) fruit servings/d for heavy TV viewers vs. 1.60 (0.04) and 1.70 (0.05) for moderate and limited, respectively ($p<0.001$)
Utter et al. (2006) [32]	Cross-sectional	5–14 years $n=3,275$ New Zealand	≥2 h/d TV watching vs. <1–2 h/d or <1 h/d	Aged 5–10 years: Statistically significant less likely to be high consumers of fruits and vegetables and more likely to be high consumers of soft drinks ($p=0.029$), hamburgers ($p=0.016$), and French fries ($p<0.001$) than children watching less than an hour Aged 10–14 years: Statistically significant more likely to be high consumers of soft drinks ($p=0.036$), hamburgers ($p=0.018$), French fries ($p=0.003$), and chocolate sweets ($p=0.004$) than adolescents watching TV for less than an hour Statistical more likely to be obese ($p=0.04$) than adolescents watching TV for less than an hour
Ranjit et al. (2010) [52]	Cross-sectional	Mean age: 15(1.6) years $n=15,283$ USA	Hours of watching television per day	Statistically significant increase for mean times soda consumption per day ($p<0.001$)

aOR (adjusted) odds ratio, *CI* confidence intervals, *h/d* hours per day

poorer diet quality could, in fact, reflect an association between parents' television viewing and diet quality. Figure 13.1 presents the conceptual framework of the relation between TV watching and low diet quality in children.

The other age group that has been extensively researched is that of adolescents. Adolescence is another critical time during child development in which lifetime behaviors are formed or reshaped and eating patterns developed at this age are more likely to characterize later eating patterns and diet quality [25]. Adolescents have better cognitive abilities than younger children and they provide better quality information to researchers. Also the participation of most of the adolescents in schools makes them an easily accessible age group to longitudinal dietary assessment and evaluation. Like younger children, food advertisement plays a significant role in the negative effect of TV viewing in adolescents' diet quality. Adolescents eat more unhealthy foods such as pizza, hamburgers, snack foods, and soda consistent with the foods advertised on television [26, 27]. However, snacking while watching TV is a significant contributing factor in mediating the association between television viewing and adolescents' eating behaviors. Pearson et al. in a longitudinal

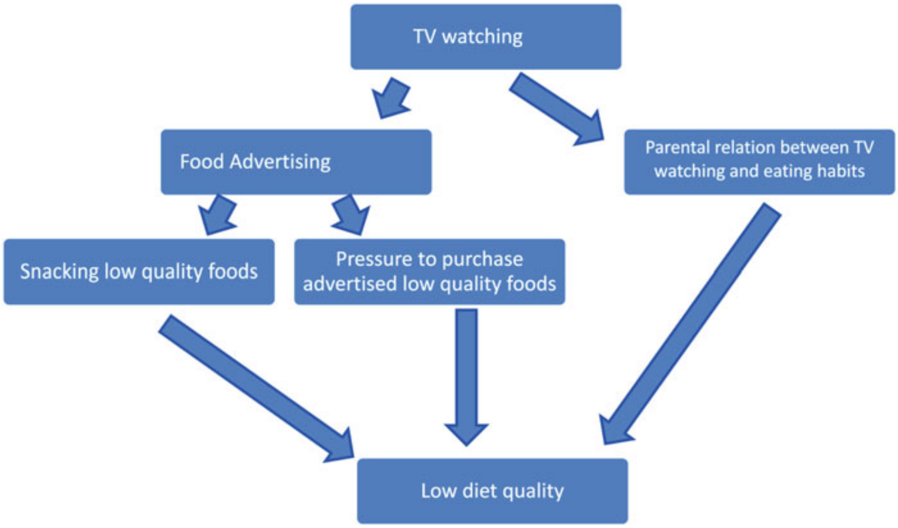

Fig. 13.1 Conceptual framework for the causal association between TV watching and low diet quality in children

Table 13.3 Food and nutrients commonly used in the studies of the association between TV viewing and diet quality in children and adolescents

High diet quality indicators	Low diet quality indicators
Fruits	Snacks
Vegetables	Soft drinks
Meat	Fast food
	Red/processed meat
	Sweets

study of 1,729 adolescents from Australia with start age of 7 and 9 years, who were assessed 2 years later, documented that the prediction of energy-dense drinks, energy-dense snacks, and fruit consumption was mediated by the effect of eating snacks while watching TV and the reinforcing factor of TV viewing, and among them eating snacks while watching TV had the greatest mediating role [28]. Furthermore, the most critical period during adolescence for forming and maintaining bad diet habits to young adulthood is the high school period rather than the middle school period, as demonstrated by the study of Barr-Anderson et al. [29].

There is also a great variability in the outcomes used in the diet quality–TV viewing association studies, which are summarized in Table 13.3. Some studies use for indicators selected food and nutrient intakes, categorizing them as healthy and unhealthy. Greater intake of healthy foods and nutrients is indicative of a better diet quality whereas a lesser intake with the opposite one [30, 31]. The most common unhealthy food categories are snacks and soft drinks or sugar-added beverages. Snacking is a food pattern influenced mostly by advertising than inactivity. Many studies use the duration of TV viewing time as a proxy estimation of the children's exposure to food advertisements and they have reported statistically significant positive associations with advertised snacks and TV viewing [32]. However, in their study Parvanta et al. assessed both TV viewing time and paying attention to the TV commercials in Chinese children and adolescents and found significant positive correlations only between snacking and attention to TV commercials, suggesting that a measure of attention is a better indicator of exposure to food advertisements on TV than is the overall amount of time spent watching TV [33]. Sugar-added beverages and soft drinks are also used as indicators of a bad diet quality with TV viewing. Feldman et al. reported that when family meals are accompanied by television the likelihood of consuming soft drinks increases for both adolescent boys and girls, attributing this effect to the food and soft drink advertisements [34]. This is particularly important since sweetened soft drink consumption could be considered as an overall unhealthful diet quality marker in childhood. As seen in many studies, over consuming sugar-sweetened beverages is associated with less consumption of essential nutrients such as calcium, vitamin D, folate, and iron [35, 36]. Limiting TV viewing time could decrease sugar-sweetened soft drink consumption and positively affect diet quality in childhood. Finally, fast food consumption has also been used as a proxy outcome for assessing overall diet quality in children. There are studies which report positive correlation between TV viewing and fast food consumption both for children and adolescents [37, 38].

Besides food advertising, another possible way in which TV may influence fast food intake is through hidden food consuming messages embedded within program content. TV content analysis focusing of food messages made by Story and Faulkner reported that the average reference to food occurred approximately 4.8 times per 30 min of program content, while one half of them were about foods that are low in beneficial nutrients. These findings are further supported by Kotz and Story who reported that 43.6 % of the advertised foods in children's Saturday morning television programming promote foods predominantly high in fat and/or sugars with relatively low nutritional value [22].

Only one of the recently published studies evaluating the effect of TV watching to diet quality has used a complex DQI. Liang et al. have used DQI as an overall assessment tool for the estimation of the diet quality in Canadian children while at the same time they also report TV watching effects at a macronutrient (servings/day of fruits and vegetables and soft drinks) or at a micronutrient effect (% energy delivered from fat, carbohydrate energy, and snack foods, all of which are related with increased or decreased risk for obesity and chronic diseases) [39]. Since there is lack of evidence for the valid application of diet health indices in childhood populations, most researchers have been avoiding using a single DQI and have preferred using specific indicators, which can be easily studied and can provide results for within-country and longitudinal comparisons.

In the context of 2001/2002 HBSC survey, a WHO collaborative cross-national study was conducted in a number of European countries and regions, and also in the USA, Canada, and Israel. A common identical questionnaire was applied to the participating countries, with items related to sociodemographics, TV viewing, and eating habits, thus allowing for between-countries comparisons. In all participating countries beside Greenland, increased TV viewing time (as per hour increase) was statistically significant associated with higher rates of daily consumption of sugared drinks and sweets. On the other hand, increased TV viewing time was significantly negatively associated with the consumption of vegetables and fruit (in 20 and 19 countries, respectively), revealing a common non-healthy diet quality pattern related to TV viewing. The majority of the countries that no statistically significant associations were found belong to the Central and Eastern Europe. Authors attributed this finding to the differences in the TV food advertisements across these countries, but stressed the possible presence of cultural-specific confounders or other confounders related to family food culture, parental practices, and the availability of access to unhealthy snack foods and beverages [40]. From the existing body of evidence, most of the recent studies concern populations from the USA and Canada, while there is emerging evidence from Greece [41] and China [33].

Concerning the study design of the recent research about TV watching and diet quality, most of the studies are cross-sectionals and very few are longitudinal (Fig. 13.2). Furthermore, more longitudinal studies are referring to adolescents rather than younger children. Longitudinal studies due to their prospective design are able to demonstrate temporal associations between television viewing and diet quality indicators, providing thus evidence regarding the causal relationship of TV watching with bad diet quality. Moreover, they are able to control for baseline exposures as well as for other empirically and theoretically relevant factors. All recent longitudinal studies have documented TV watching as a risk factor for unhealthy diet quality or increase in adiposity at later childhood and adolescence, either as total minutes or hours of TV watched per day or eating while watching TV [28, 29, 31, 42–45]. Their findings are in concordance with many cross-sectional studies published in the literature [30].

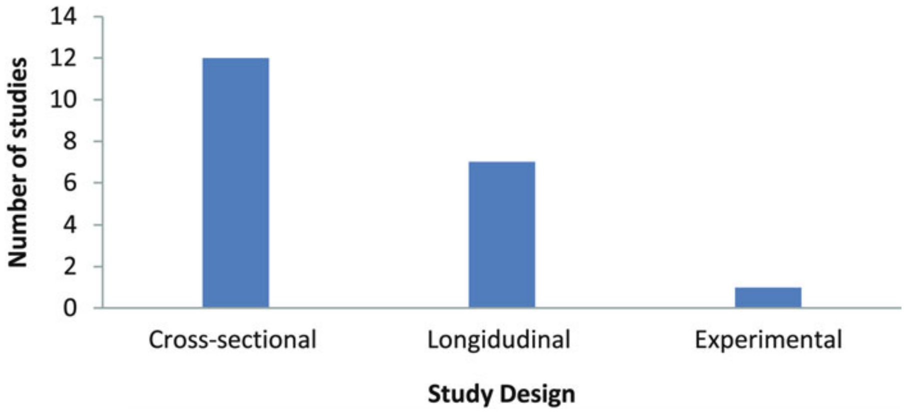

Fig. 13.2 Distribution of the recent studies assessing the association between diet quality and TV viewing according to their study design

Even though cross-sectional design suffers from the phenomenon of reverse causality, due to the nature of the exposure, it is likely that television viewing could lead to poor diet quality rather than the opposite. However, it should be noted that cross-sectional and longitudinal studies of the effect of TV watching suffer also from measurement errors in both exposure and outcome variables. The assessment of TV watching is usually self-reported or in children below 5 years of age is reported by their parents and diet quality assessment is done mostly by food frequency questionnaires, which are prone to recall bias. Also the possibility of the presence of confounding from other factors, such as socioeconomic status (which may not have always been controlled for), needs to be taken into consideration. Taking account of the possible measurement over- or underestimation and the confounding bias, the consistency in the findings of both types of studies argues in favor of the negative effect of TV viewing in children's diet quality.

Video Games and Diet Quality in Childhood and Young Adulthood

American Academy of Pediatrics in the most recent guidelines about the recommended screen time consumption per day for children and adolescents refers to daily total media time consumption and makes no discrepancy between the types of media use in which children are engaging [46]. Although television viewing is still the preferred leisure time activity for most children, in recent studies video gaming has emerged as a rapidly evolving favorite screen-related activity [47]. Furthermore, it has been reported that more than 50 % of children report eating when playing computer video games, besides when watching TV [48]. However, there are rather few studies addressing the unique effect of video gaming in the diet quality of children and adolescents (Table 13.4). Video gaming is considered a part in children's everyday sedentary activities and most researchers have not attempted to assess it as a sole factor, but with other passive activities like TV watching and doing homework. Recent evidence has acknowledged the role of knowledge-based work as a contributor to the current obesity epidemic [49]. The only studied outcome in the relevant literature is food intake, expressed as energy intake. In their study, Chaput et al. reported that 1-h video game played by male adolescents is accompanied by increased caloric intake at a following ad libitum meal compared to the control condition consisting of relaxing on a comfortable chair. Authors also added that overconsumption of food after playing video games was observed without increased subjective sensations of hunger and appetite. The authors justified their finding about the association between overeating and video gaming to non-homeostatic mechanisms but to a rewarding feeding behavior associated with the practices of video games that

Table 13.4 Summary of studies assessing video gaming and diet quality in childhood and young adulthood

Study	Study design	Sample	Exposure	Estimates of association
Chaput et al. (2011) [60]	Randomized crossover clinical trial	Mean age 16.7 ± 1.1 years $n = 22$ Canada	1-h of video gaming	Mean difference in ad libitum energy intake after video gaming vs. after rest: 335 kJ ($p < 0.05$), adjusted for sensation of hunger
Mellecker et al. (2010) [51]	Experimental	Mean age: 11.0 ± 1.0 $n = 30$ USA	1-h free choice video gaming sessions	Mean difference in snacking energy intake between seated and activity-enhanced video game condition 374 (192) vs. 383 (266) cal ($p = 0.844$)
Ranjit et al. (2010) [52]	Cross-sectional	Mean age: 15(1.6) years $n = 15{,}283$ USA	Hours of video gaming per day	Statistically significant increase for mean times soda consumption per day: 0.015
Baranowski et al. (2011) [53]	Randomized clinical trial	10–12 years old $n = 133$ USA	Intervention group: 9 sessions of at least 40 min of playing video games designed to lower risks of type 2 diabetes and obesity Control group: playing general healthy lifestyle video games	Statistically significant higher treatment vs. control fruit and vegetables intake (mean increase = 0.67 servings/day, $p = 0.018$)
Baranowski et al. (2003) [54]	Randomized clinical trial	10–11 years old $n = 1{,}578$ USA	Intervention group: 10 sessions of engaging to a psychoeducational multimedia game for 25 min Control group: not engaging in the multimedia game	Statistically significant higher treatment vs. control fruit and vegetables intake (mean increase = 1.0 servings/day, $p = 0.018$)

impair satiety signal capacity [50]. Moreover, the activity type of video game does not seem to alter food intake. In their study Mellecker et al. demonstrated no statistically significant alteration in snacking between children participating in activity-enhanced video gaming or seated video gaming [51]. Finally, as one of the factors examined in the survey by Ranjit et al., video gaming has been associated with increased consumption of sugar-sweetened beverages, as a part of the assessment of several screen-related sedentary behaviors (hours spent watching television and using the computer), adjusted for several confounders for both sexes [52].

However, video games have been used as an effective means of improving diet quality in healthy children. Barranowski et al. have documented in several works that video games can effectively promote dietary change among youth [53, 54]. Moreover, there is some evidence that active video games, which are electronic games that allow players to physically interact (by using arm, leg, or whole-body movements), could have a beneficial effect against childhood obesity which is mediated by the increase in the physical activity of the participating children [55–57].

Conclusions

Childhood is a very sensitive developmental period where the impact of various factors has lifetime influence on several aspects of children's lives. This period is also a crucial time for the development of future food habits. Hence, sufficient energy and nutrient intake from a balanced diet and healthy snacks should be provided for optimal growth and the acquisition of early healthy diet habits. TV and videogames have become an important part of children's everyday life and their effect in various behavioral patterns has taken a great concern in the recent literature. The majority of the published evidence is about the negative relationship between TV exposure and healthy eating habits. However, most of the studies are cross-sectional which cannot establish causal relationships between TV viewing and several healthy diet quality markers like vegetables and fruit consumption or unhealthy markers like increased snacking and sugar-sweetened beverages. As far as computer gaming, there is very limited data available regarding the correlation between gaming and diet quality. More intervention studies are needed in order to provide sufficient evidence to support that decreasing television viewing time and video gaming can help in obesity prevention and the maintenance of normal weight in childhood.

References

1. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. Science. 1998;280:1371–4.
2. Tucker LA, Bagwell M. Television viewing and obesity in adult females. Am J Public Health. 1991;81:908–11.
3. Tucker LA, Friedman GM. Television viewing and obesity in adult males. Am J Public Health. 1989;79: 516–8.
4. Dickinson A. Video playground. Time. 2000;19:98.
5. Rideout VG, Roberts DF, Foehr UG. Generation M: media in the lives of 8–18 year-olds. Menlo Park, CA: Kaiser Family Foundation; 2005.
6. Wack E, Tantleff-Dunn S. Relationships between electronic game play, obesity, and psychosocial functioning in young men. Cyberpsychol Behav. 2009;12:241–4.
7. USDA. http://www.iom.edu/Activities/Nutrition/SummaryDRIs/~/media/Files/Activity%20Files/Nutrition/DRIs/5_Summary%20Table%20Tables%201-4.pdf. Assessed 18 Oct 2011.
8. Kranz S, Hartman T, Siega-Riz AM, Herring AH. A diet quality index for American preschoolers based on current dietary intake recommendations and an indicator of energy balance. J Am Diet Assoc. 2006;106: 1594–604.
9. Huybrechts I, Vereecken C, De Bacquer D, et al. Reproducibility and validity of a diet quality index for children assessed using a FFQ. Br J Nutr. 2010;104: 135–44.
10. Guenther PM, Reedy J, Krebs-Smith SM. Development of the Healthy Eating Index-2005. J Am Diet Assoc. 2008;108:1896–901.
11. Feskanich D, Rockett HR, Colditz GA. Modifying the Healthy Eating Index to assess diet quality in children and adolescents. J Am Diet Assoc. 2004;104: 1375–83.
12. Chiplonkar SA, Tupe R. Development of a diet quality index with special reference to micronutrient adequacy for adolescent girls consuming a lacto-vegetarian diet. J Am Diet Assoc. 2010;110:926–31.
13. Mariscal-Arcas M, Romaguera D, Rivas A, et al. Diet quality of young people in southern Spain evaluated by a Mediterranean adaptation of the Diet Quality Index-International (DQI-I). Br J Nutr. 2007;98: 1267–73.
14. Welsh JA, Cogswell ME, Rogers S, et al. Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999–2002. Pediatrics. 2005;115:e223–9.
15. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet. 2001;357:505–8.
16. Johnston Jr CC, Miller JZ, Slemenda CW, et al. Calcium supplementation and increases in bone mineral density in children. N Engl J Med. 1992;327: 82–7.
17. Gillman MW, Oliveria SA, Moore LL, Ellison RC. Inverse association of dietary calcium with systolic blood pressure in young children. JAMA. 1992;267: 2340–3.
18. Gillman MW. Enjoy your fruits and vegetables. BMJ. 1996;313:765–6.
19. Hu FB, Stampfer MJ, Manson JE, et al. Dietary fat intake and the risk of coronary heart disease in women. N Engl J Med. 1997;337:1491–9.
20. Halford JC, Boyland EJ, Hughes G, Oliveira LP, Dovey TM. Beyond-brand effect of television (TV) food advertisements/commercials on caloric intake and food choice of 5–7-year-old children. Appetite. 2007;49:263–7.
21. Batada A, Seitz MD, Wootan MG, Story M. Nine out of 10 food advertisements shown during Saturday morning children's television programming are for

foods high in fat, sodium, or added sugars, or low in nutrients. J Am Diet Assoc. 2008;108:673–8.
22. Kotz K, Story M. Food advertisements during children's Saturday morning television programming: are they consistent with dietary recommendations? J Am Diet Assoc. 1994;94:1296–300.
23. Coon KA, Tucker KL. Television and children's consumption patterns. A review of the literature. Minerva Pediatr. 2002;54:423–36.
24. Borzekowski DL, Robinson TN. The 30-second effect: an experiment revealing the impact of television commercials on food preferences of preschoolers. J Am Diet Assoc. 2001;101:42–6.
25. Story M, Neumark-Sztainer D, French S. Individual and environmental influences on adolescent eating behaviors. J Am Diet Assoc. 2002;102:S40–51.
26. Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? Am J Public Health. 1998;88:277–80.
27. Coon KA, Goldberg J, Rogers BL, Tucker KL. Relationships between use of television during meals and children's food consumption patterns. Pediatrics. 2001;107:E7.
28. Pearson N, Ball K, Crawford D. Mediators of longitudinal associations between television viewing and eating behaviours in adolescents. Int J Behav Nutr Phys Act. 2011;8:23.
29. Barr-Anderson DJ, Larson NI, Nelson MC, Neumark-Sztainer D, Story M. Does television viewing predict dietary intake five years later in high school students and young adults? Int J Behav Nutr Phys Act. 2009;6:7.
30. Miller SA, Taveras EM, Rifas-Shiman SL, Gillman MW. Association between television viewing and poor diet quality in young children. Int J Pediatr Obes. 2008;3:168–76.
31. Boynton-Jarrett R, Thomas TN, Peterson KE, et al. Impact of television viewing patterns on fruit and vegetable consumption among adolescents. Pediatrics. 2003;112:1321–6.
32. Utter J, Scragg R, Schaaf D. Associations between television viewing and consumption of commonly advertised foods among New Zealand children and young adolescents. Public Health Nutr. 2006;9:606–12.
33. Parvanta SA, Brown JD, Du S, et al. Television use and snacking behaviors among children and adolescents in China. J Adolesc Health. 2010;46:339–45.
34. Feldman S, Eisenberg ME, Neumark-Sztainer D, Story M. Associations between watching TV during family meals and dietary intake among adolescents. J Nutr Educ Behav. 2007;39:257–63.
35. Keller KL, Kirzner J, Pietrobelli A, St-Onge MP, Faith MS. Increased sweetened beverage intake is associated with reduced milk and calcium intake in 3- to 7-year-old children at multi-item laboratory lunches. J Am Diet Assoc. 2009;109:497–501.
36. Frary CD, Johnson RK, Wang MQ. Children and adolescents' choices of foods and beverages high in added sugars are associated with intakes of key nutrients and food groups. J Adolesc Health. 2004;34:56–63.
37. Taveras EM, Sandora TJ, Shih MC, et al. The association of television and video viewing with fast food intake by preschool-age children. Obesity (Silver Spring). 2006;14:2034–41.
38. French SA, Story M, Neumark-Sztainer D, Fulkerson JA, Hannan P. Fast food restaurant use among adolescents: associations with nutrient intake, food choices and behavioral and psychosocial variables. Int J Obes Relat Metab Disord. 2001;25:1823–33.
39. Liang T, Kuhle S, Veugelers PJ. Nutrition and body weights of Canadian children watching television and eating while watching television. Public Health Nutr. 2009;12:2457–63.
40. Vereecken CA, Todd J, Roberts C, Mulvihill C, Maes L. Television viewing behaviour and associations with food habits in different countries. Public Health Nutr. 2006;9:244–50.
41. Manios Y, Kondaki K, Kourlaba G, et al. Television viewing and food habits in toddlers and preschoolers in Greece: the GENESIS study. Eur J Pediatr. 2009;168:801–8.
42. Dubois L, Farmer A, Girard M, Peterson K. Social factors and television use during meals and snacks is associated with higher BMI among pre-school children. Public Health Nutr. 2008;11:1267–79.
43. Jago R, Baranowski T, Baranowski JC, Thompson D, Greaves KA. BMI from 3-6 y of age is predicted by TV viewing and physical activity, not diet. Int J Obes (Lond). 2005;29:557–64.
44. Francis LA, Lee Y, Birch LL. Parental weight status and girls' television viewing, snacking, and body mass indexes. Obes Res. 2003;11:143–51.
45. Wiecha JL, Peterson KE, Ludwig DS, et al. When children eat what they watch: impact of television viewing on dietary intake in youth. Arch Pediatr Adolesc Med. 2006;160:436–42.
46. American Academy of Pediatrics. Children, adolescents, and television. Pediatrics. 2001;107:423–6.
47. Christakis DA, Ebel BE, Rivara FP, Zimmerman FJ. Television, video, and computer game usage in children under 11 years of age. J Pediatr. 2004;145:652–6.
48. Moag-Stahlberg A, Miles A, Marcello M. What kids say they do and what parents think kids are doing: the ADAF/Knowledge Networks 2003 Family Nutrition and Physical Activity Study. J Am Diet Assoc. 2003;103:1541–6.
49. Tremblay A, Therrien F. Physical activity and body functionality: implications for obesity prevention and treatment. Can J Physiol Pharmacol. 2006;84:149–56.
50. Chaput JP, Klingenberg L, Astrup A, Sjodin AM. Modern sedentary activities promote overconsumption of food in our current obesogenic environment. Obes Rev. 2011;12:e12–20.
51. Mellecker RR, Lanningham-Foster L, Levine JA, McManus AM. Energy intake during activity enhanced video game play. Appetite. 2010;55:343–7.

52. Ranjit N, Evans MH, Byrd-Williams C, Evans AE, Hoelscher DM. Dietary and activity correlates of sugar-sweetened beverage consumption among adolescents. Pediatrics. 2010;126:e754–61.
53. Baranowski T, Baranowski J, Thompson D, et al. Video game play, child diet, and physical activity behavior change a randomized clinical trial. Am J Prev Med. 2011;40:33–8.
54. Baranowski T, Baranowski J, Cullen KW, et al. Squire's Quest! Dietary outcome evaluation of a multimedia game. Am J Prev Med. 2003;24:52–61.
55. Maddison R, Foley L, Ni Mhurchu C, et al. Effects of active video games on body composition: a randomized controlled trial. Am J Clin Nutr. 2011;94:156–63.
56. Adamo KB, Rutherford JA, Goldfield GS. Effects of interactive video game cycling on overweight and obese adolescent health. Appl Physiol Nutr Metab. 2010;35:805–15.
57. Maddison R, Foley L, Mhurchu CN, et al. Feasibility, design and conduct of a pragmatic randomized controlled trial to reduce overweight and obesity in children: the electronic games to aid motivation to exercise (eGAME) study. BMC Public Health. 2009;9:146.
58. Jackson DM, Djafarian K, Stewart J, Speakman JR. Increased television viewing is associated with elevated body fatness but not with lower total energy expenditure in children. Am J Clin Nutr. 2009;89:1031–6.
59. Francis LA, Birch LL. Does eating during television viewing affect preschool children's intake? J Am Diet Assoc. 2006;106:598–600.
60. Chaput JP, Visby T, Nyby S, et al. Video game playing increases food intake in adolescents: a randomized crossover study. Am J Clin Nutr. 2011;93:1196–203.
61. Fitzpatrick E, Edmunds LS, Dennison BA. Positive effects of family dinner are undone by television viewing. J Am Diet Assoc. 2007;107(4):666–71.

14 Parental Perceptions and Childhood Dietary Quality: Who Holds the Reins?

Kristi B. Adamo and Kendra E. Brett

Key Points

- Diet quality is imperative for appropriate child growth and development.
- There are many complex and integrated factors that influence diet quality and how a parent perceives their child's diet quality.
- Parents are their child's primary role model and thus bear considerable responsibility for shaping diet quality and feeding behaviours.
- Parenting style can strongly influence diet quality and eating behaviours in children.
- A parent's perception of child's weight status, depending on its reflection of reality, can positively or negatively affect diet quality.
- Family structure can significantly influence diet quality.
- In general, children's diet quality does not meet recommendations for optimal growth and development. However a parent's perception of their child's diet may not accurately reflect this reality.

Keywords

Parental perception • Diet quality • Healthful eating • Eating behaviours • Children

Abbreviations

BMI	Body mass index
CDC	Center for Disease Control
SES	Socioeconomic status
UK	United Kingdom
US	United States of America
WHO	World Health Organization

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Introduction

This chapter will focus on diet quality in children and specifically the parental perception of diet quality and how this might impact eating behaviours.

There are two interrelated factors to be described:

1. The impact of parental perceptions (knowledge, beliefs, attitudes, self-efficacy) on diet quality of a child
2. The actual parental perception of diet quality (i.e. realism vs. idealism)

Why We Care

The early years represent a critical period of growth and development of health behaviours. The global increase in child obesity and comorbid conditions [1], phenotypes once reserved only for the adult population, indicates that health behaviours have changed over time. While there is a complex set of factors associated with optimal growth and development, one cannot deny the role of eating habits and diet quality. In the case of children, it is universally accepted that parents/guardians are primarily responsible for food procurement, choices and meal preparation, and conventional wisdom suggests that parents want to provide them with the necessary tools to ensure healthy growth and development. There are of course many contributors, both non-modifiable such as biological, societal, environmental, and socioeconomic as well as modifiable contributors to healthy growth and development which include dietary and physical activity or sedentary behaviours.

Parents can be a strong positive influence (good model, encourage high quality choices, exposure to a wide array of healthful choices), or conversely, a powerful negative influence (setting a poor example, making the easy and often less healthy choice, food rewards of low quality, inappropriate snacking, lack of control or heavy restriction) on diet quality. Like any relationship, the one between parents and children is complex and multilayered. Most people, regardless of whether they are a parent or not, have been caught not 'practicing what they preach' and it is not uncommon for children to hold back information, or hide behaviours from their parent(s). This may start as early as the preschool years. Therefore, it should not be surprising that a parent's perception of a child's behaviour, in this case eating behaviour and diet quality, may not be reflective of the true situation. We know from other work that there exists a lack of awareness for various health-related concepts, such as parents not recognizing obesity in their own children [2]. There are many potential reasons and biases related to this concept which will be touched on later (see Table 14.1). Additionally, the commonly held parental perception that they have little or no control over their child's eating behaviour may also lead to detrimental diet quality.

Importance of Diet Quality

Diet quality is strongly related to the physical and cognitive development of children [3], and thus a proper diet with adequate nutrition is of the utmost importance during critical developmental stages. In the early 1900s in Europe and North America, malnutrition was a salient child-health concern due to inadequate supplies of healthy foods (high in protein and vitamins), and the focus of healthy eating was related to achieving adequate growth and maintaining strength in the children. Currently, in developed countries, the problem is quite the opposite; increased exposure to poor-quality food due to an overabundance of low nutrient, energy dense foods including nutritionally unbalanced snacks, convenience and fast foods [4] resulting in high calorie, but nutrient-poor diets (i.e. diets that are not meeting current dietary recommendations known to reduce disease risk). This nutrition transition is being experienced by many developing nations as well [5] and is a major health concern as it has led to an increase in so-called 'diseases of affluence' in children. While conditions such as obesity, high blood pressure, and type 2 diabetes have been linked to caloric excess and diets high in fat, sugar and salt and low in fibre, many children are not meeting the recommended intakes of certain

Table 14.1 Common parental misperceptions about dietary behaviours and diet quality and their associated dangers

What the parent perceives	What is the 'true' situation
Child to have a high quality diet because of parental misconceptions of what constitutes healthy eating	Lack of healthy eating due to misguided unhealthy choices, despite believing that what they are doing is healthy
	May have self perceptions about healthy eating that are not in keeping with the actual guidelines
	Parents may be mislead by advertising and serve low nutrient energy dense foods, over processed foods
In order to improve their child's diet quality (i.e. encourage them to eat their veggies) or ensure their child is eating enough, they must pressure the child to eat or use 'preferred' food as a reward	Pressure feeding is detrimental to development of appropriate satiety cues and may lead to undesirable eating practices
	Rewarding with preferred food or treats may have short term successes but is not beneficial in the long term
Their child has a diet as good as it is going to get given they are working within their current limitations (budget, accessibility)	Choices being made are not ideal and thus child's diet is less than satisfactory
They have no control over their child's eating i.e. fatalistic perception/external locus of control	May results in the provision of very limited variety of food items without the recognition that they do have control and have many more options or choices regarding food that can increase their child's diet quality
They are serving healthy food of high quality, therefore portion control is unnecessary	The over consumption of even 'healthy' foods can lead to excess calorie consumption and weight gain over time
That restrictive eating will produce healthy children with good eating habits	Child may develop restrictive eating style and may suppress feelings of hunger
	Being too health conscious and restrictive, without moderation can be counter productive, resulting in children who may binge on treats outside of the home
	Restrictive eating focuses children's attention on restricted foods, increasing their desire to obtain and consume the foods
That a healthy weight child means that the child is eating healthy (i.e. high diet quality)	A thin child may not have healthy eating habits; thin $\neq$ healthy
	What a child eats is still important for macronutrients and fibre, and can have major impacts on health
That their child has adequate diet quality because the parent packed the child's lunch for school and limits snacks at home (assumption)	Once at school the child does not necessarily eat what is provided, they may trade the unwanted items, selectively eat only the unhealthy treats provided possibly throwing out unwanted items, older kids might buy food in the vending machines, the cafeteria or local convenience stores or food establishments
	Children of a certain age are able to help themselves to food and/or snacks without the parental awareness
That their child does not notice 'adult snacks' or parental unhealthy eating patterns	There should not be unhealthy adult-only food
	Children may learn to seek these forbidden foods
That telling a child that certain food is 'good' for them or 'bad' for them will lead to high quality diet	This may not be the case since simply stating good for you or bad for you may not be enough. There needs to be an explanation or reasoning behind why a child needs to eat more of one food and less of another
	Parents must also model good eating behaviour. Encouraging moderation and decision making is important (i.e. if you are going to have that cookie now, you cannot have cake later on)
Their child to be a 'fussy eater' because they dislike healthy foods	Parent might not want to take responsibility for their child's poor eating habits and may only provide preferred foods to avoid conflict thereby not encouraging their child to try new foods or provide the opportunities for their child to expand their palate
Parents perceive their child as too immature or incompetent to make healthy nutritional choices	Although children may choose to make 'unhealthy' choices, they may also choose 'healthy' options if they are given the opportunity to decide for themselves
Parents perceive their child's diet quality to be high because they eat everything that is served	This can be a positive behaviour if the food served is of high quality but if the parent's perception is skewed, the diet quality may be suboptimal

This chart describes dietary misperceptions that are commonly held by parents and the potential detrimental effects that these misperceptions may have on the child's diet and dietary behaviours micronutrients, such as calcium and fibre, which are important for future disease prevention [6].

It is essential for children to establish healthy eating behaviours early, as experts posit that attitudes towards food choice develop during childhood and play an important role in the maintenance of eating habits which influence health across the lifespan [7, 8]. It is therefore important that an unhealthy or suboptimal diet is identified early in childhood, in order for the proper changes to be made to improve the child's diet and prevent future disease risk. An underlying problem with this approach is the dependence on parental perception, and thus a parent must first be able to recognize when a child's diet is poor, and know how to make the necessary changes. While this chapter will focus on contributors to parental perception and how such perceptions affect child diet quality, there is a paucity of research that has examined parental perceptions and misperceptions of their child's diet [9–11]. To our knowledge only one study to date has directly compared maternal perception of the child's diet quality with the actual quality of the child's diet [12] and this study will be discussed in detail in a later section.

> …although people may want to eat a healthy diet, people of different ages, genders and educational attainment levels have different views as to what constitutes a healthy diet [13]

Parental Role

Parents are their child's first and most important dietary role models. Parents generally hold the greatest responsibility when it comes to establishing diet quality and shaping their child's eating behaviours and thus lifelong eating habits [14]. There is considerable causal evidence that parenting affects child eating; however this relationship is not unidirectional, as there is also substantial correlational evidence that child eating and weight status influence parenting [14]. There is no denying that parents need to not only provide consistent messaging, but to supply appropriate role modelling through their own behaviour; that is influence children to 'do as I do' rather than 'do as I say' [15]. Conner and Armitage [13] argue that a child's attitudes to certain foods are learned by modelling what their parents do; for example parent's fruit and vegetable consumption has been shown to be the strongest predictor of the child's fruit and vegetable consumption [16]. While parents can exert a strong level of control over what their young children eat (particularly in their home), this is age dependent and as children mature and start making their own decisions they become less reliant on their parents, and a portion of this control is lost, thus why the early years are an important time point to target with respect to behaviour adoption/adaptation.

Families are the primary setting for feeding children, and parents and other family members have a substantial influence on a child's eating patterns [17]. Therefore it is important for parents to recognize not only the potential health consequences of a poor diet, but also recognize what constitutes a healthy diet and an unhealthy diet. Knowing that food behaviour in children can be altered, depending on the environment in which they live, parental perception is key to defining and determining diet quality. Children emulate both positive and negative parental behaviours, and if parents are disinterested in nutrition or are against making positive lifestyle (i.e. dietary) changes, then it is likely that the children's attitudes will reflect that of their parents [18], and thus compromise diet quality. This may be further complicated if parental backgrounds or belief systems differ within the same family unit, and thus result in opposing views/perceptions [19]. It is therefore important for parents to be able to decipher when cultural or ethnic customs or traditions are not in accordance with healthy eating recommendations.

Additionally it is important for parents to be able to recognize whether their child is actually *consuming* a healthy diet or a poor diet, and the ability to make this distinction is a combination of what the parent perceives the child to be consuming and their understanding of a healthy diet. If a parent does not comprehend what constitutes a nutritionally balanced diet, than they will be less likely to perceive their child's diet as poor, and they will not be able to make the changes to improve diet quality and prevent future disease.

Contributors to Parental Perceptions and Diet Quality

Overview

The diet quality of children is highly influenced by a wide variety of characteristics from both their physical and social environments, and includes factors such biology, behaviour, availability of food, family food preferences and practices, portion sizes, socioeconomic factors, and cultural beliefs. Figure 14.1 is designed to demonstrate the multidimensional nature of a child's dietary behaviours and the factors that can influence parental perceptions and thus shape a child's dietary quality. Obviously the interplay between contributing factors is complex and interdependencies are high. We do not claim that this figure includes every possible factor but rather it is meant to illustrate the various intricacies.

Things to consider when reading this chapter:

- How does a parent quantify if a food is healthy or not? (sugar, fat, salt, vitamins?)
- How does one determine the healthfulness? (labels, advertising, peers, professionals?)
- Is there a parenting style associated with better diet quality?
- Why are mothers and their perceptions so important to diet quality?
- What are some of the barriers (perceived or otherwise) to diet quality in children?
- What factors contribute to the perception of diet quality? Do they create biases?
- Are there commonly held stereotypes/attitudes that can hamper child's diet quality?
- What influence might family structure have on a child's diet quality?
- Does the child's weight impact how the parents feed a child?
- Does the perception of a child's weight undermine diet quality?

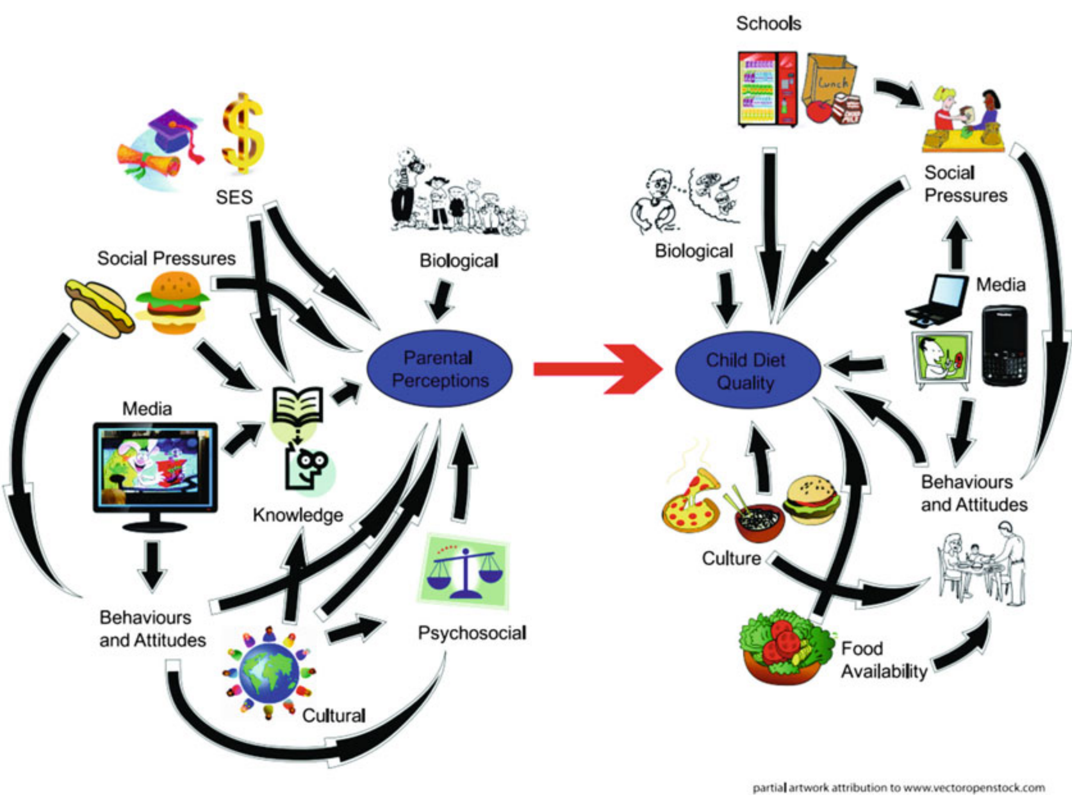

Fig. 14.1 The interrelationships of different influences on parental perceptions and diet quality in children. This diagram demonstrates the complexity of the interrelationships of the different influences on a parent's perception of their child's diet as well as the influences on a child's diet

Parental Knowledge, Awareness, Beliefs, and Action

Parental beliefs and perceptions play a part in childhood growth and development because they shape parental feeding behaviours. Research has shown that the similarities and differences between parent's and children's food preferences are, in part, related to the family environment, and not simply due to a shared cultural experience [20]. Moreover, it is important to recognize that family influences on food preferences are not unidirectional. As mentioned earlier, it is well known that parents have a strong influence on their child's eating habits; however it is not uncommon for children to also influence their parent's eating behaviours [20]. Parental food preferences and beliefs about what constitutes healthy foods are known to influence how a parent feeds their child. For example, Dennison et al. found that parents with young children were more likely to serve their children whole milk if the parent had never tried reduced fat milk or if they believed that whole milk was healthier for their child [21]. Parental eating restraint could also impact a child's diet. Roberts et al. found that many parents felt it was necessary to control their children's snacking; however some parents admitted to being unable to make 'healthy' snack choices when more appealing options were available [11]. This questions whether the parent is always competent to make healthy choices for their child when they are unable to do so for themselves. In addition, when parents restrict certain food items from their children, such as cookies, this may increase desires in their children to obtain and consume the 'forbidden' foods [22], and potentially cause the children to binge on these items should they ever be available for consumption.

The diet quality of the children may also be impacted by whoever holds the grocery purchasing power within the household. While the mother typically has the most authority over food purchases for the family, many parents will admit that the decision of what foods to purchase is often the result of negotiations with their children, who will repeatedly request snack items, in the hopes that one day their parent will capitulate and buy these items [11]. Research has shown that about 50 % of parents believe that their child influences their food purchases [9, 23] and the influence of children on parental purchasing patterns seems to be increasing. Parents may find it difficult to deny their children treats in the grocery store due to increasing advertising and marketing to children; with over 65 % of parents feeling that their children are influenced by advertisements on television [24]. Due to this 'pester power' of children asking for specific foods, such as those featuring their favourite animated character, most parents reported having conflicts when deciding what food to purchase and 60 % of parents agreed or strongly agreed that they gave in to their children's demands in the supermarket [24].

It is also possible that parents may not perceive diet *quality* but rather diet *quantity* as being important. Absolute caloric intake is still a contributing factor to diet quality and research has shown that the most powerful determinant of the amount of food consumed at meals by children between the ages of 4 and 6 years was amount served [25]. Furthermore, this study reported that children do not compensate for a previous large meal or snack prior to their meal. Portion size is important and children can in fact have too much of a good thing signifying that parents need to be aware of the dietary guidelines and what qualifies as an appropriate portion for their child's age.

Information Sources

Parents rely on a wide variety of information, strategies, and personal beliefs when qualifying and trying to improve their child's diet, some of which are beneficial, while others are counter productive. Parental knowledge and beliefs specifically about nutrition undoubtedly impact a child's diet quality. In an attempt to draw in the health conscious shopper, supermarkets commonly pitch presumably 'healthier' versions of popular foods such as baked potato chips, and fat free ice cream, in addition to the traditional item. Functional foods are another recent trend in nutritional advertising. These are foods that boast specific health benefits on the basis of one ingredient (e.g. juice made from pomegranates that are high in antioxidants to protect against cancer); however these products may not always be nutritionally well rounded (e.g. high in sugar and low in fibre juice). These marketing strategies may cause parents to believe they are making 'healthy' choices for their children, when in fact are only making 'slightly less bad' choices.

While a mother's nutrition knowledge and diet-health awareness is significantly associated with improved diet quality in young children, the influence decreases as children age [26]. Beydoun and Wang found that the positive association between socioeconomic status (SES) and diet quality indicators varied across levels of nutrition knowledge and belief; in adults with strong nutrition knowledge, education level was positively associated with healthy eating, while in adults with low nutrition knowledge, the relationship between education and healthy eating was nonsignificant [27]. This indicates that parental education can have an influence on dietary choices and diet quality only for those who have the necessary knowledge and beliefs about nutrition.

Ethnicity and culture can also have a considerable impact on diet quality of children, with cultural inheritance accounting for 30–40 % of the variance in dietary intake in children [28], due to socio-demographic differences or cultural preferences in food choices and the preparation methods [17]. Additionally, many cultural traditions and customs include food as a major component, which may shape attitudes towards food. It is not uncommon for parental knowledge and beliefs to be formed from cultural and intergenerational opinions from trusted family members, rather than from health professionals [19]. However the cultural impact on parental perceptions and diet quality can be influenced by education and income.

It may be difficult for some parents to recognize what constitutes a healthy diet, for in addition to information provided by health professionals, they may receive contradictory advice about health and nutrition from informal sources, notably television, magazines, newspapers, internet, advertisements on buses, friends, relatives, and retailers. Unfortunately the majority of the information on health and nutrition of which parents are inundated comes from commercial or media sources, whose main interest is profit (e.g. increased sales or attracting viewers), rather than the best interest of the consumer. With this constant barrage of information, some adults have reported reaching a saturation point concerning new information on food and health, and tend to ignore new advice [29]. Many adults prefer to rely on nutritional knowledge based on their personal experiences with food and health or information from their friends or relatives, rather than formalized advice [29]. Some have also reported that they find that information on food and health is often contradictory and in a constant state of flux, and generally regard the advice as 'worthless', rather than a result of scientific advances [29]. Due to this distrust of the ever changing nutritional facts, parents may not incorporate the most recent nutritional advice into their notion of a 'healthy diet' when deciding what to feed their children.

Parenting Style

Parents can vary considerably with regard to how they socialize their children, otherwise known as parenting style, and this can dramatically alter their child's development [30]. There are four recognized parenting styles; Authoritative, Authoritarian, Permissive, and Neglectful (Fig. 14.2). While some degree of control is important, authoritarian feeding styles, which are associated with high levels of parental control, are not necessarily the most desirable [31]. For example, in an attempt to prevent overweight or negative health consequences, parents who are concerned about their children's diet may adopt very controlling child-feeding practices [32]. While this may be perceived as an appropriate mechanism to improve diet quality, research has suggested that strict parental control practices may have the opposite effect. A body of research lead by Birch and Fisher has shown that parental control efforts may potentiate children's preference for restricted foods as well as their intake of these unhealthful foods [33], while diminishing self-control in eating [34]. Instead there appears to be consensus in the literature that authoritative parents, who are both demanding and responsive, (i.e. parental warmth, emotional support, appropriate granting

	High Demandingness	Low Demandingness
High responsiveness	**Authoritative** Empathetic and respectful of child's opinions, but maintains clear boundaries/expectations	**Permissive** Empathetic, but indulgent without discipline
Low responsiveness	**Authoritarian** Low warmth and strict discipline	**Neglectful** Emotionally uninvolved and does not set rules or have expectations

Fig. 14.2 Parenting style. This chart demonstrates four different parenting styles and their level of responsiveness and demandingness. Table adapted, with permission from Elsevier, from Berge JM, Wall M, Neumark-Sztainer D, Larson N, Story M. Parenting style and family meals: cross-sectional and 5-year longitudinal associations. *J Am Diet Assoc* 2010 July;110(7):1036–42

of autonomy, and clear, bidirectional communication) have the most beneficial impact on children's developmental outcomes [30, 35] and, relevant to this chapter, diet quality. Additionally, research that has explored associations between children's perceived parental parenting style and dietary behaviour also indicates that authoritative parenting is the most balanced and effective [36, 37] since authoritative feeding styles are associated with higher parental responsiveness dimensions such as nurturing and reasoning. For instance, perceived maternal nurturing has been associated with lower calorie consumption and lower saturated fat intake while paternal nurturing was associated with lower sodium intake [38].

Both maternal and paternal authoritative parenting style is positively associated with frequency of family meals [39], known to be related to healthy diet quality [40]. Authoritative mothers are less reliant on fast food [41], which one would assume is related to more healthful diet quality. On the other hand, neglectful parenting is associated with increased consumption of fast food [41]. Inconsistencies between parents and parenting style or the delivery of contradictory messages may negatively affect diet quality [31], meaning congruency in healthful verbal messaging and action will support high diet quality.

Parents who have a more relaxed or permissive relationship with their child are more likely to make choices to avoid conflict and give children what they want rather than making a responsible decision. While inconsistencies exist in the literature, data has shown that adolescents who reported more permissiveness in their family ate more fat and sweet foods and more snacks, and reported less healthy food choices in the family, while those who reported more food rules in the family consumed less soft drinks [42, 43]. In summary, parents need to exert some level of control as to not indulge their children but not so much that it negatively impacts eating behaviour and food choices.

Fatalists vs. Self-Believers

Parental attitude in the fundamental approach to their child's eating and their perception of control can have a substantial influence on the quality of their child's diet. The UK's Office of Communications report on food advertising in the context of childhood obesity identified two types of mothers in respect to their attitudes to children's eating: fatalists and self-believers [44]. Fatalist mothers lack confidence in their own judgement and have a more relaxed approach to their children's diet. They believe that they have little or no control over their child's diet, and make minimal or no effort to encourage healthier food choices in their children. They often believe that they have little power against the constant barrage of advertising and tempting foods [45]. These mothers are more likely to abandon the idea of trying to control their child's diet since they cannot control or influence 100 % of their

Fig. 14.3 Perceptions of items that contribute to an ideal diet for a child. This diagram demonstrates the differences in food choices that have been selected as contributors to an ideal diet for children by fatalist and self-believer mothers. Figure 14.3a represents the picture of an 'ideal' diet for a child as reported by a fatalist mother. This diet is very sparse, with a high reliance on name brand foods. Figure 14.3b represents a picture of an 'ideal' diet for a child created by a self-believer mother. This diet is more comprehensive, and is more reliant on whole foods and whole cooked meals. Figure reprinted from the 2004 Ofcam report

child's diet. Families with fatalist mothers often have a lower SES, rely heavily on unhealthy processed foods, and readily allow the children to make their own unhealthy food choices.

Conversely, 'self-believers' are more confident in their own judgement and ability to make informed decisions, they feel they have more control over their child's diet, and have a vested interest in choosing healthier options for their family. Families with self-believer mothers are often better educated and have a higher income, serve mainly fresh foods and home-cooked meals, and the mother has the most control over food choices for the family.

When asked to identify the 'ideal diet' for children, both subsets of mothers had a broad understanding of a healthy diet (e.g. convenience foods are less healthy than home-cooked meals and the importance of fruits and vegetables), however, they differed in their ability to conceptualize a healthy diet. Fatalist mothers are thought to have difficulty interpreting information on healthy eating and determining how to provide this diet to their children in an attractive and affordable manner, and may feel that they have to reject whole categories of foods rather than supplying items in moderation. The picture of an 'ideal' diet for a child from a fatalist mother is very sparse, with a greater reliance on brand name foods (Fig. 14.3a). These mothers are also likely to perceive dietary guidelines as unattainable, and consequently do not bother trying to eat healthy. In addition, fatalist mothers are more likely to have a reactive rather than a proactive approach to health and healthy eating; if their child is normal weight, than they may not make any effort to improve the diet. Self-believer mothers are more concerned with the long term consequences of an unhealthy diet (e.g. diabetes, heart disease), and have a more inclusive and more attainable idea of what constitutes a healthy diet. The picture of an 'ideal' diet for a child from a self-believer mother is much more comprehensive, with a greater reliance on home-cooked and whole foods (Fig. 14.3b). These mothers are unlikely to exclude whole categories of food, but rather they limit the consumption of certain foods, and if they do purchase convenience food, they prefer options such as prepackaged salads and chilled foods.

Mothers as the Epicentre of Control

Within the family, the behaviours and knowledge of mothers have appealed to researchers more so than that of fathers in regards to children's eating habits, as mothers continue to spend significantly more time caring for their children, specifically the physical care activities such as feeding [46]. Each family tends to only have one 'nutritional gatekeeper' who controls the majority of the family food [45], and despite more women holding full time jobs, this role still tends to belong to mothers. Given this, mothers tend to be in charge of grocery shopping and consequently their attitudes to food and approach to their children's diet is crucial. It is in no way meant to underplay the role of fathers, some of whom now fill this role, but fathers are seldom included in studies and often report knowing little about their children's eating habits. In general, women tend to be more aware of food and health information, and they become more interested in nutrition and health when they are pregnant or raising young children [29]; however they may still find it difficult to interpret what nutritional information is correct and what constitutes healthy and unhealthy diets.

There is also research to suggest that fear often bolsters mothers' perceptions and attitudes regarding their child's eating and can work to undermine diet quality. Fearing a child is not getting enough to eat for normal development can be a powerful influence and such anxiety can lead a parent to provide their children with only foods they enjoy rather than providing healthier items that they may refuse to eat [44]. A recent focus group study assessed maternal beliefs and practices regarding child feeding among mothers [47]; and three themes emerged. First, the mothers believed that a heavy infant was a healthy infant and reflected successful feeding and parenting. Second, they frequently perceived that their infants were not satiated, which led to the introduction of solids before recommended ages. Finally, the mothers described situations in which food was used to shape child behaviour. These findings highlight how certain maternal biases and beliefs can influence the child-feeding relationship.

Mothers who are preoccupied with their own weight and eating behaviours can negatively impact their children's eating habits, especially if they perceive their children, in particular their daughters, as overweight, by restricting intake and encouraging weight loss [15]. This could influence the child's diet quality if calories are highly restricted and the child does not eat enough healthy foods to obtain adequate macro- and micronutrients for healthy development. This could also result in food preoccupation in the children and putting them at risk for developing problematic eating behaviours.

Contribution of the Individual Child

> Eating greens is a special treat, it makes long ears and great big feet. But it sure is awful stuff to eat (Thumper-Bambi circa 1942)

A child's food intake will be strongly determined by their own personal preferences which may be altered by many factors. It must be noted that there is a difference between preferences and liking of food. While 'liking' a food involves the taste, texture, and smell of the food, ones 'preference' for a food can be impacted by factors such as perceived healthiness, convenience, availability, and cost [20].

A recent systematic review of the qualitative research pertaining to parental perceptions of healthy behaviours found that the child's food likes and dislikes was a commonly identified barrier to healthy eating [19]. Many parents will often defer to their children's food preferences, and serve foods high in fat, sugar, and salt. Additionally, children may attribute different values to 'healthy' and 'unhealthy' food than adults in their lives [11]. Young children are often neophobic about food, that is they are afraid to try new and unfamiliar foods [15], and will often need to be exposed to a new food multiple times before they will try it. Of interest, is that neophobia in children is related to the consumption of fruit, vegetables, and meat, but is unrelated to consumption of sweet, fatty snack foods, or starchy staples [48]. The greater the child's exposure to a food item, the more likely they are to consume it; simply put, if foods of high diet quality are offered most often, a child will tend to enjoy and prefer these foods.

Parental attitudes and food preferences can also substantially impact a child's food intake; food consumption patterns are often similar between children and their parents and if a child observes their parents consuming a new food item, then they will be more apt to try the unfamiliar food [17]. Similarly, if parents dislike a certain food, than their child will not get a chance to try it because it is not offered at home, or their dislike for the item will influence their child's perceptions of that food, and dissuade them from trying it [11]. Thus acknowledging the fact that *children are unable to eat foods that are not made available to them.*

Convenience, Time, and Cost

The term 'convenience food culture', refers to the demand for foods which are quick and easy to prepare, a trend that has considerable negative impact on children's diet quality [44]. In a systematic review, Pocock et al. identified that parents recognized their important role in modelling healthy eating behaviours for their children; however a perceived lack of time often prevented such positive actions from occurring [19]. While the nutritional impact is often considered, convenience and taste have been shown to be very important determinants of food selection [49, 50]. The impact on weight control is often quite low on the list of food choice motives [49, 50]. Unfortunately the prioritization of convenience is positively associated with intake of energy-rich foods and negatively associated with nutrient-rich (healthful) foods [50]. We know that food away from home is now a major contributor to total food expenditures and this is related to perceived lack of time for meal planning and preparation and thus parents with busy work schedules frequently rely on fast food [41]. Fathers use of fast food restaurants was found to be strongly associated with their children's use of these restaurants [41], likely to the detriment of diet quality.

It is not uncommon for parents to have misperceptions about the costs associated with a healthy diet, and the cost of food is frequently listed as a barrier to a healthy diet [19, 51]. Parents may have the perception that all high quality foods are more expensive, particularly when multiple ingredients are needed to prepare a healthy meal. When money is an issue, parents may choose to purchase individually packaged prepared items that appear cheaper, but in reality are lower in quality, non-satiating, and cost more overall [52]. Thus while parents may appreciate the importance of healthful food, if they perceive that all such choices are too expensive they are unlikely to risk their food budget on healthier foods that may not be accepted by their children [53]. This issue is of particular importance in families of lower SES, (see section on 'Socioeconomics'), who must feed their families on a restricted budget.

Perception of Child's Weight

Parental perception of their child's weight is influenced by a number of factors, including the parent's weight, the weight of their child's peers, and stereotypes perpetuated by the media. Given that many Canadian parents are oblivious to the state of their children's health [54], it is not surprising that there is a growing body of research indicating that the majority of parents do not accurately perceive when their children are overweight [2, 55, 56].

Parents have identified that having children of different weights within a family can create difficulties for feeding their children, because it is sometimes necessary to restrict the amount and types of food in the household due to an overweight child; however this was thought to be unfair to the other healthy weight children in the family [19]. The fact that parents perceive that they need only be mindful about the eating behaviours of their overweight children is cause for concern. Providing treats or unhealthful foods is not a necessity and should not be the norm for the lean child and a taboo for the overweight child.

The way in which a parent perceives their child's weight status can potentially have a large impact on the diet quality of the child. There are three possible scenarios through which a misperception of weight could negatively impact the child's diet quality. First, the child is incorrectly perceived to be underweight, a concern that is still common amongst parents [19], and is thus overfed with calorie rich foods, in order to reach the parent's perception of a 'healthy weight'. This malpractice could turn a healthy weight child into an overweight child, and increases the risk of future disease. Secondly, a parent may misperceive a healthy weight child as overweight, and therefore restrict access or availability of food. This is a dangerous practice as it may lead to nutrient deficiencies or the development of disordered eating. Finally, a parent may misperceive their child as having a normal weight, and thus believes that their child's eating habits and diet quality do not matter. This final scenario is particularly problematic, as the child is potentially being over- or under-fed, resulting in an over- or underweight child, respectively. Either way the diet may be of poor quality as the parents do not recognize the need for healthy nutrition if they believe their child has a healthy weight. These scenarios are a grave concern as parents have repeatedly been shown to underestimate their child's true weight status, particularly that of overweight and obese children [2, 55, 56].

As rates of obesity increase in children and larger body sizes become the norm, parents may not be aware that their child's weight is unhealthy because they appear similar to their peers, who may also have an unhealthy body weight. This shift from the norm that is occurring in parental perceptions of a healthy body weight may also be impacting their perceptions of a healthy diet. As prepackaged foods increase in popularity, and grocery stores become filled with products that are highly processed and highly marketed (e.g. 'whole grain' sugary cereals, or 100 % 'juice blends' that boast a full serving of fruit but contain few vitamins and no fibre), parents may be unaware that their child's eating habits are suboptimal as they are similar to others in their social environment.

Peer and Social Environment

As children enter the school environment, peers become a large influence on their food preferences; in particular adolescent eating behaviours [57]. While peers can be both a positive and negative influence on a child's eating habits, a UK-based study reported that 60 % of parents believed peer pressure was associated with children demanding high fat, sugary or salty snacks, while only 44 % of parents believed peer pressure influenced the demand for fruit and healthy foods [44].

Swapping of school lunches may also impact the quality of a child's diet, as a parent may prepare a well balanced lunch for their child; however the child may not consume all or parts of that meal. Children admit to not eating or giving away parts or all of their lunches because they want to go outside to play, and some children have admitted to swapping their lunch even if they did not want to so as to avoid disappointing their peers [11]. Swapping of school lunch items also occurs in elementary students participating in school lunch programs despite there being 'no trading rules', with older students being more likely to trade items than younger students [58]. This implies that an empty lunch tray does not necessarily mean that a child has eaten all of the lunch served to him/her. Most parents understand that it is likely that their child will swap parts or all of their lunches, and thus they cannot monitor what their child eats at school as there is no guarantee that it was their child who consumed the food that was sent in the first place [11].

Family Structure and Dynamics

Mealtime structure is another important factor influencing diet quality in children [59]. Diet quality is negatively associated with not eating regular family meals and the frequent consumption of fast food and meals in front of the television [6]. As previously mentioned, time constraints of parents and busy families also impact the quality of a child's diet. With both parents in many families working outside of the home, it becomes difficult for parents to plan and prepare healthy, home-cooked meals daily, and may opt to serve convenience foods (i.e. fast food or prepackaged meals) that are typically high in calories, fat, and sodium. Working mothers in particular are more likely to have reduced time for meal planning, shopping, and preparation, and may choose the greater convenience of premade or processed foods [60]. Additionally, parents may not have time to prepare healthy lunches for their children, which increases the consumption of convenience foods and lunches purchased at school, which has been associated with reduced diet quality [6].

In fact, in a national health survey it was found that a quarter of all Canadians over the age of 4 years had eaten something that had been prepared in a fast food outlet the day before the survey was conducted [61]. This may be related to a cultural shift in work-home priorities. For example mothers who consider their work more important than other roles, including parenthood, had children who spent more time eating in fast food restaurants [41]. This same research study suggests that a father's perception of family meals as an important ritual is associated with children spending less time in fast food restaurants. This is important because children who eat fast food, compared with those who do not, consume more total energy, more energy per gram of food, more total fat, more total carbohydrate, more added sugars, more sugar-sweetened beverages, less fibre, less milk, and fewer fruits and non-starchy vegetables [62].

Family structure is also important in determining child diet quality. Single parent households are known to rely heavily on time saving, microwaveable and unhealthy convenience foods [63], and if parents are divorced children may receive different foods depending on which parent they are living with or visiting. Some mothers report that although they believe they are feeding their child healthy food, they perceive that their child does not eat as healthily while at the father's house [11]. The guilt that is often felt by divorced, single or nonresident parents due to a lack of time spent with the child can hamper diet quality as this perception results in frequent compensation (e.g. money or food treats), as well as more freedom and independent decision making [64]. Additionally, a UK-based study reported that most families eat together only on weekends and there has been a trend over the last decade for children to eat alone, without parental chaperone or oversight [44]. Another survey indicated that only 46 % of children aged 8–15 years report eating with their family most days [44] and thus the children are responsible for food selection on the other days which may compromise diet quality considering their preferences for unhealthy foods.

A parent's control over their child's diet quality can often be undermined by other members of the family, such as grandparents who always 'treat' the child to sweets, or unintentionally sabotaged by others who interact with the child, such as cookies from the grocery store clerk [11]. Opposing views or inconsistency regarding food within a family may also influence the child's diet quality, with mothers reporting that fathers often interfere with their efforts to control the child's diet by repeatedly giving in when the children ask for treats (permissive parenting) [65].

Socioeconomics

The SES of the parents can also have a large influence on a child's diet quality. Low income parents may be faced with many problems attaining healthy food for their children including lack of access to cheaper food retailers (due to neighbourhood or transportation), an inability to buy bulk, limited cooking facilities, and limited time and money for experimenting with new foods. Moreover, while parents with low incomes may understand the importance of a healthy diet, they must spend within a budget and their priority may be to avoid food waste [24, 66]. A meta-analysis of 11 studies in seven European countries found a positive association between SES of adults and fruit and vegetable consumption [66], and more recently Beydoun and Wang found that SES factors were consistently and positively related to all dietary factors in the Healthy Eating Index in US adults [27]. This suggests that unhealthy eating habits are more likely in adults with lower SES in Europe and North America, and as parents have the greatest influence on their children's eating patterns, that it is likely that the diet quality of their children is also affected. A systematic review illustrated that, compared to their higher SES counterparts, children from lower SES families had less desirable eating behaviours. Among an array of unhealthy factors, these children eat fewer family meals, less fruits and vegetables, are more likely to eat fast food and drink sugar-sweetened beverages, have parents who are more likely to use food as a reward, are more likely to live in neighbourhoods without grocery stores, and are more likely to skip breakfast [60]. A family's access to resources, such as healthy grocery stores, may also impact the diet quality of their child. Barriers to healthy foods include not only monetary resources, but also the physical environment of the area in which the family resides, and whether they can access the necessary foods.

On the other side, family wealth may also negatively impact a child's diet quality. Wealthier families who can afford to feed their children a healthy diet may decide instead to treat their children more frequently because they can afford to do so. Turner et al. found that 60 % of parents feel guilty about not spending time with their children due to work commitments, and that 69 % of parents would give their children treats if they had the money [24]. This suggests that parents may want to ease their guilt by giving their children treats and, that wealthy families, who may spend more time at work, may try to compensate by buying unhealthy treats for the children.

Parental Perceptions and Misperceptions of Diet Quality

Perception vs. Reality: Current Research

The way in which a parent perceives their child's diet quality is very important in ensuring a healthful diet for optimal development, while preventing the development of disease. As most parents believe that they are the primary influence on their child's eating habits [11], it is critical that they have accurate perceptions of their child's diet quality, as it is up to them to make the necessary dietary changes to ensure optimal health. Despite the importance of this issue, it is surprising that there is such limited research on parental perceptions of their children's eating behaviours and diet quality, with only one direct comparison between the maternal perceptions of the child's diet quality with the actual quality of the child's diet [12].

While there are certain views about diet quality and healthy eating that the majority of parents tend to support (e.g. homemade lunches are healthier than store bought, and that parents need to monitor and control the quality and quantity of their child's diet) [11], parents do not share all of the same views about healthy eating. To demonstrate, Roberts et al. found that perceptions of 'healthy eating' differed across parents, with some parents believing that their family was eating healthier because they has started eating 'oven baked chips instead of fried chips', whereas others thought chips were unhealthy altogether [11]. Parents also hold different beliefs in regard to snacking. Some parents believe it is necessary to monitor what their children are eating by having them ask permission for snacks, while others believe that by having snacks freely available, children would be less tempted by the items, rather than creating forbidden and more enticing foods [11]. Conversely, parents may not feel that it is necessary to monitor the intake of all snacks, but rather that it is important to limit certain unhealthy snacks (e.g. cookies or chocolate), while allowing ad libitum access to healthy options (e.g. fruit or vegetables) [11]. Additionally, a parent's perceived responsibility for their child's diet may change over time as the quality of the diet changes. There is evidence to suggest that parents of teenagers who disliked 'healthy' food tend distance themselves from the responsibility of ensuring that their child consumed a healthy diet by calling them 'fussy eaters', inferring they were difficult to care for [67].

Despite reports of unhealthy eating habits of Canadian children [61], a large portion of parents perceive their child's diet to be healthy. For instance, in a large ($n = 1,940$) survey of parents with children aged 4–12 years in the national capital region of Canada, more than 80 % of respondents considered their child to have 'good' or 'excellent' eating habits [9]. While these parents perceived their children to be eating healthy, when questioned about their child's fruit and

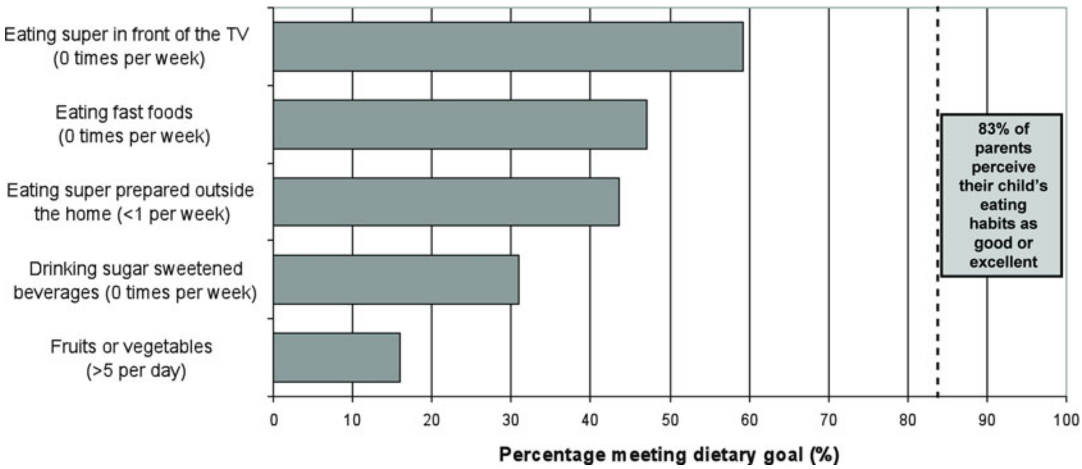

Fig. 14.4 Parental reported eating behaviours of their children. The bar graph demonstrates the percentage of children meeting select dietary goals as reported by their parents. Note the inconsistency between parental perception of eating habits, and percentage meeting high quality dietary goals set by health professional organizations. Adapted from Adamo et al. Using path analysis to understand parents' perceptions of their children's weight, physical activity and eating habits in the Champlain region of Ontario. Paediatrics & Child Health 2010;15(9):e33–e41

vegetable consumption, only a fraction were actually meeting the recommendations and many reported high sugar-sweetened beverage consumption (see Fig. 14.4). This suggests that parents do not apply the same criteria as health experts when assessing the diet quality and eating habits of their children. Similarly, Dammann et al. found that low income minority and homeless mothers perceived their child's diet and health status to be 'fair' (15 %) or 'good' (44 %), and rated their own diet similarly, however most of these families were homeless and receiving food stamps, both of which have been linked to poor dietary intake [10]. It is likely that the perceived 'good' diets of the children would be considered 'fair' or 'poor' according to health experts.

As one might anticipate, parents were more likely to rate their children's eating habits more favourably if they report preparing home cooked meals and eating meals as a family at the dinner table [9], as they believe that these practices are related to more positive eating habits. By the same token, the parent's perception of their child's eating habits was positively related to perceived fruit and vegetable consumption, and negatively related to perceived soft drink consumption [9], implying that parents are aware of the necessity of the former and the dangers of the latter (Fig. 14.5). Finally, the parent's perception of their child's eating habits was positively related to the parent's perceived influence over the children's weight and negatively related to the parent's concern about their children's weight status [9].

To the best of our knowledge, there has only been one study to date that has compared the maternal perception of the child's diet quality with the actual quality of the child's diet as estimated by the Healthy Eating Index [12]. This large scale study that included 1,759 Greek children aged 2–5 years, found that 83 % of mothers overestimated the quality of their child's diet (Fig. 14.6). Specifically, mothers whose main concern when choosing their child's foods was having a healthy diet were more likely to overestimate the quality of their child's diet, with an 86 % overestimation rate [12]. Since these mothers do not recognize their child's diet as 'poor' or 'needs improvement', then they will not make the necessary changes to improve the child's diet, thus increasing the child's risk for nutrition-related diseases of both dietary excess (i.e. diabetes) and nutrient deficiencies (i.e. poor bone health). It is apparent that although mothers may believe that they are providing their child with a healthy diet, this may not be true, and may be a result of limited nutritional knowledge of healthy

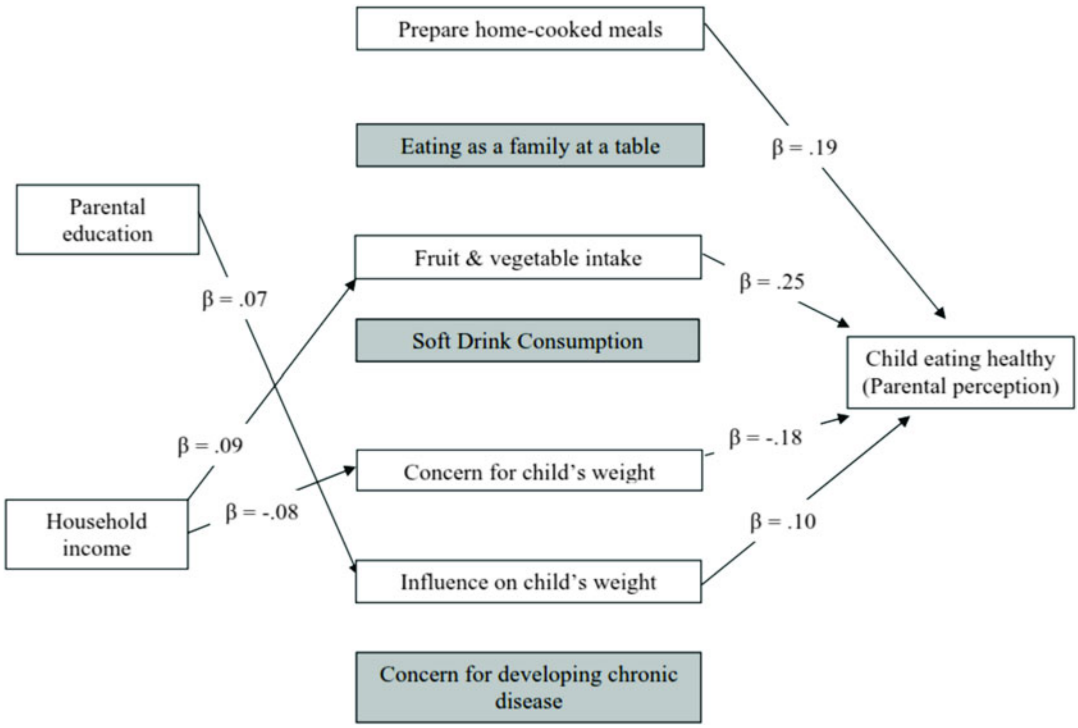

Fig. 14.5 Path analysis predicting parental perception of their child's eating habits. β or beta-(path) correlation coefficients, measure the relative strength and sign of the effect from a causal variable to an outcome variable in the model and help to describe the amount of variance in parental perceptions explained by any given variable. *Shaded boxes* represent variables that were identified in the original correlation analyses but did not have a significant beta-coefficient. This path analysis demonstrates that parents with higher education levels believed they had greater influence over their child's weight which in turn increased their perception that their child was eating healthily. Parents with higher household income believed their child was eating more servings of fruits and vegetables, which was associated with an increased perception that their child was consuming a healthy diet. Conversely, the path analyses indicated that parents from households with lower incomes were more concerned with their child's weight, which was correlated with a decreased perception that their child diet was healthy. This information was originally published in Adamo et al. Using path analysis to understand parents' perceptions of their children's weight, physical activity and eating habits in the Champlain region of Ontario. Paediatrics & Child Health 2010;15(9):e33–e41

foods and appropriate quantities for children. Of interest, was that despite the well documented significant associations between parental SES and a child's diet [27, 66], Kourlaba et al. found no significant association between maternal education, employment status, and BMI and the maternal overestimation of their child's diet quality (Fig. 14.6) [12].

Actual Diet Quality of Children

As previously mentioned, one of the strongest predictors of a child's diet quality is parental beliefs, and role modelling. These may inform a parent's perception of not only their own diet quality, but that of their children. We know that eating habits have changed over time and that the nutritional transition has played a large role in this instigating this change.

The 2004 Canadian Community Health Survey found that *on average* Canadian children were meeting the recommended daily servings of most food groups [61]; however *average* consumption patterns of a population disguise the fact that substantial portions of the population are not meeting the recommended daily servings of the food groups, and do not have a healthy diet.

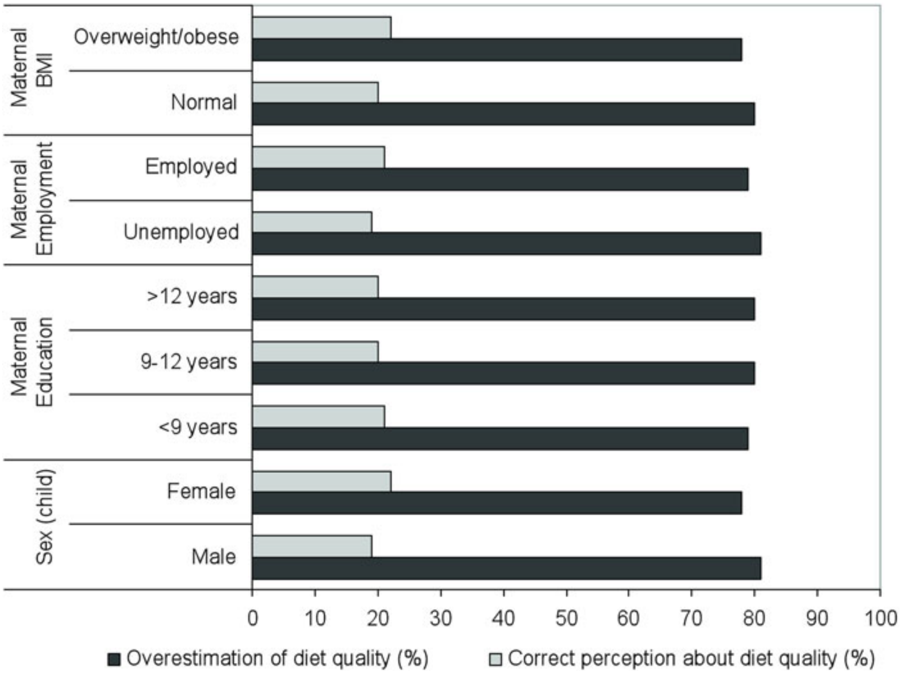

Fig. 14.6 The prevalence of maternal overestimation of diet quality in their children by maternal BMI, maternal level of employment, education, and child sex. This bar graph demonstrates the high percentage of mothers who overestimate their child's diet quality. The likelihood of overestimating their child's diet quality was not impacted by maternal BMI, employment, education or the sex of the child. Adapted from Kourlaba et al. Diet quality of preschool children and maternal perceptions/misperceptions: the GENESIS study. *Public Health* 2009 November;123(11):738–42

Fruits and vegetables, and milk products and alternatives provide vital macro- and micronutrients for developing children, including protein, calcium, fibre, vitamin C and D, and despite the well-documented benefits of these foods, large portions of Canadian children are failing to meet the minimum daily recommended servings of these products [61].

Fruits and Vegetables

Over 60 % of Canadian children aged 9–13 years and 70 % of children aged 4–8 years did not meet the minimum recommendation of five and six servings per day, respectively [61]. These data are not unlike those published by the WHO, which indicated that just over 30 % of European children 11–13 years report eating fruit daily [68], and about 25 % of surveyed children report consuming fruit once per week or less. The vegetable consumption numbers are more dismal and consumption rates decrease with age. Similarly, a report by the Center for Disease Control (CDC) illustrated that only 22 % of US adolescents reported meeting their guidelines of five or more fruits or vegetables per day [69].

Dairy, Grains, Meat, and Alternatives

In the Canadian sample, 37 % of kids aged 4–9 years and 61 % of boys and 83 % of girls aged 10–16 years did not meet the minimum of two and three daily servings of milk and alternative products, respectively [61]. Likewise, only 14.5 % of US high school students reported having consumed three or more servings of milk per day in the 7 days prior to the survey [69]. Canadian children were more likely to meet the recommended servings of meat and alternatives and grain products [61].

Other Foods

In today's grazing culture, a major problem contributing to the failure of children to meet the

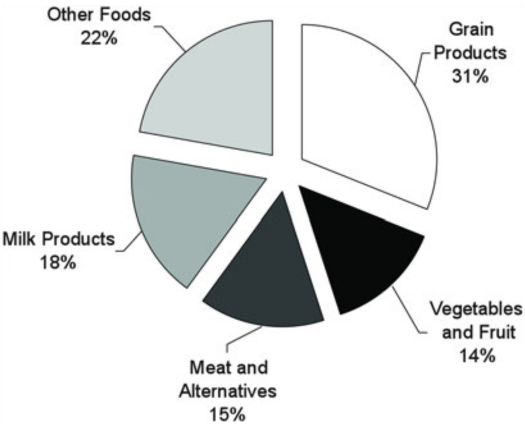

Fig. 14.7 Percentage distribution of sources of calories, by food group in Canadians aged 4–18 years. This pie chart demonstrates the high percentage of calories (22.3 %) that Canadian children are obtaining from 'other' foods that do not fit within Health Canada's Food Guide to Healthy Eating. Adapted from Garriguet D. Canadians' eating habits. *Health Rep* 2007 May;18(2):17–32

minimum recommended servings for healthy eating is the consumption of food items that fall under the 'other' category of the food guide. These 'other' foods are represented by soft drinks, salad dressings, sugars/sweets/preserves, fruit drinks, and oils/fats, which cannot be classified by the four food groups. Consumption of 'other' foods accounts for almost a quarter of their total intake (Fig. 14.7) [61]. Excessive advertising of nutrient-poor, high-sugar foods aimed at children [70] (e.g. breakfast cereals, granola bars), and the lower cost of calorically dense, less nutritionally valuable food choices [51, 70] (e.g. potato chips, soft drinks), are presumed to be major contributors to this problem. Additionally, 41 % of the calories that Canadians consumed as snacks were considered 'other' foods [61], and in the US, children obtain approximately 20 % of their calories from snacks [71], suggesting that snacking could be an area for improved dietary choices and where children and adults could incorporate some of their missing servings of fruits and vegetables. Data from the 2009 CDC Youth Risk Behaviour Surveillance report indicates that almost 30 % of high school students reported drinking at least one regular soft drink per day in the previous 7 days [69] and similar statistics are reported for Europe (Fig. 14.8) [68].

Who Is in Charge?

Although parents have the greatest influence on their child's eating habits, not all parents perceive that they have the same level of control or influence over their child's dietary behaviours. According to the work done by Wansink et al., parents and nutrition educators believe that, on average, the 'nutritional gatekeeper' of the family has direct or indirect control over 72 % their child's diet [45]. While 72 % does not account for the entire child's diet, it is important for parents to recognize that they do have a substantial influence over their child's nutritional habits, and that they may have even more control than they perceive they do (particularly fatalist mothers, see section on 'Fatalists vs. Self-Believers'). While there are a plethora of other factors that may influence a child's diet, a parent must not ignore their influence and it is important that they are aware of how they are influencing the development of their child's dietary habits.

While the majority of parents believe that children are too young or irresponsible to make dietary decisions and that it is the parents' responsibility to determine what the child should consume, others believe that children should know what they should be eating and place the responsibility on the child to make the appropriate choices and changes in their eating habits [11]. This is where the authoritative parenting style is particularly useful in that the need for parental control lessens as a child ages in these households [30] and parents who provide the structure and support needed for children to internalize and maintain positive behaviours have a better chance of producing children capable of taking responsibility for themselves.

Consumer research and academic research have shown that, in general, children are having more say in what they eat [72] and young children, who often accompany their parents to supermarkets, are influencing their parents' purchases [23]. For example, one UK-based study found that 48 % of parents believed that their child influenced their food purchases [23]. Similarly, 50 % of parents surveyed in the capital region of Canada reported that their child

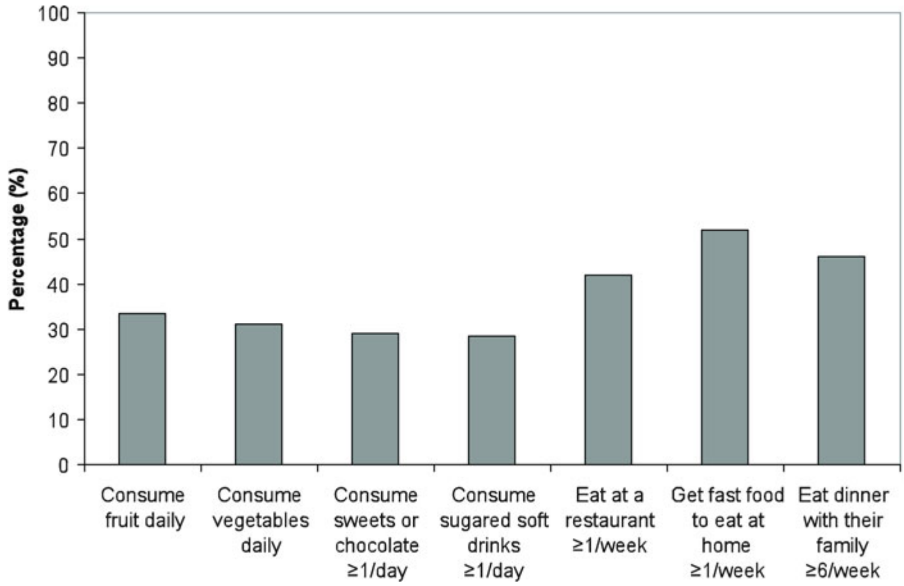

Fig. 14.8 Diet Quality and Food Behaviours of European children. This bar graph demonstrates the percentage of European children who report engaging in select good and bad dietary behaviours. Adapted from the World Health Organization. *Health Behaviour in School-aged Children* (*HBSC*) *study*: International report from the 2001/2002 survey, Eating habits. 2004

influenced their food purchases [9]. This suggests that parents are losing some control over the food procurement process to their children and that parents are consistently finding it more challenging to deny children; a phenomenon thought to be related to advertising and marketing [73]. There has been a gradual shift in the child's role within the family context and many parents involve children in most of the family decisions; including food purchases [74]. Children's own spending power is also providing increasing control over their diet quality. As children get older, spend more time outside of parental supervision and have money of their own, they may choose to spend it on food items of poor quality. Children learn to be consumers initially through observing their parents, then by making requests and choices of their own which leads to making assisted and finally independent purchases. It has been suggested that another potential factor contributing to increasing influence of the child is the high rate of divorce which may provide a child with leverage to put pressure on parents to purchase their preferred products [24].

Conclusions

Diet quality is influenced by many factors including parental perceptions. Since parents play such an integral role in the development and maintenance of their child's healthy eating behaviours, addressing misconceptions and unhealthy parental beliefs may be an important area for early intervention and prevention work in childhood obesity.

References

1. International Association for the Study of Obesity. Global childhood overweight. IASO Prevalence Data. http://www.iaso.org/publications/trackingobesity/. 2012.
2. He M, Evans A. Are parents aware that their children are overweight or obese? Do they care? Can Fam Physician. 2007;53:1493–9.
3. Martin HP. Nutrition: its relationship to children's physical, mental, and emotional development. Am J Clin Nutr. 1973;26:766–75.
4. Austin SB, Melly SJ, Sanchez BN, Patel A, Buka S, Gortmaker SL. Clustering of fast-food restaurants

around schools: a novel application of spatial statistics to the study of food environments. Am J Public Health. 2005;95:1575–81.
5. Popkin BM, Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. Int J Obes Relat Metab Disord. 2004;28 Suppl 3:S2–9.
6. Veugelers PJ, Fitzgerald AL, Johnston E. Dietary intake and risk factors for poor diet quality among children in Nova Scotia. Can J Public Health. 2005;96:212–6.
7. Keane A, Willetts A. Factors that affect food choice. Nutr Food Sci. 1994;94:15–7.
8. Seaman CEA. Healthy eating for children under two years—just what is appropriate? Nutr Food Sci. 1997;97:12–4.
9. Adamo KB, Papadakis S, Dojeiji L, et al. Using path analysis to understand parents' perceptions of their children's weight, physical activity and eating habits in the Champlain region of Ontario. Paediatr Child Health. 2010;15:e33–41.
10. Dammann KW, Smith C, Richards R. Low-income minority and homeless mothers' perceptions of their 9–13 year-old children's weight status, diet, and health. Matern Child Health J. 2011;15:106–14.
11. Roberts EM. Negotiating food choice: parents' perception of children's eating behaviour. Anthropol Noteb. 2006;12:63–77.
12. Kourlaba G, Kondaki K, Grammatikaki E, Roma-Giannikou E, Manios Y. Diet quality of preschool children and maternal perceptions/misperceptions: the GENESIS study. Public Health. 2009;123: 738–42.
13. Conner M, Armitage CJ. The social psychology of food. Buckingham: Open University Press; 2002.
14. Ventura AK, Birch LL. Does parenting affect children's eating and weight status? Int J Behav Nutr Phys Act. 2008;5:15.
15. Scaglioni S, Salvioni M, Galimberti C. Influence of parental attitudes in the development of children eating behaviour. Br J Nutr. 2008;99 Suppl 1:S22–5.
16. Wardle J, Carnell S, Cooke L. Parental control over feeding and children's fruit and vegetable intake: how are they related? J Am Diet Assoc. 2005;105:227–32.
17. Patrick H, Nicklas TA. A review of family and social determinants of children's eating patterns and diet quality. J Am Coll Nutr. 2005;24:83–92.
18. Brown K, McIlveen H, Strugnell C. Nutritional awareness and food preferences of young consumers. Nutr Food Sci. 2000;30:230–5.
19. Pocock M, Trivedi D, Wills W, Bunn F, Magnusson J. Parental perceptions regarding healthy behaviours for preventing overweight and obesity in young children: a systematic review of qualitative studies. Obes Rev. 2010;11:338–53.
20. Guidetti M, Cavazza N. Structure of the relationship between parents' and children's food preferences and avoidances: an explorative study. Appetite. 2008;50: 83–90.
21. Dennison BA, Erb TA, Jenkins PL. Predictors of dietary milk fat intake by preschool children. Prev Med. 2001;33:536–42.
22. Fisher JO, Birch LL. Restricting access to palatable foods affects children's behavioral response, food selection, and intake. Am J Clin Nutr. 1999;69:1264–72.
23. Kelly J, Tuner JJ, McKenna K. What parents think: children and healthy eating. Br Food J. 2006;108: 413–23.
24. Turner JJ, Kelly J, McKenna K. Food for thought: parents' perspectives of child influence. Br Food J. 2006;108:181–91.
25. Mrdjenovic G, Levitsky DA. Children eat what they are served: the imprecise regulation of energy intake. Appetite. 2005;44:273–82.
26. Variyam JN, Blaylock J, Lin B, Ralston K, Smallwood D. Mother's nutrition knowledge and children's dietary intakes. Am J Agric Econ. 1999;89:373–84.
27. Beydoun MA, Wang Y. Do nutrition knowledge and beliefs modify the association of socio-economic factors and diet quality among US adults? Prev Med. 2008;46:145–53.
28. Vauthier JM, Lluch A, Lecomte E, Artur Y, Herbeth B. Family resemblance in energy and macronutrient intakes: the Stanislas Family Study. Int J Epidemiol. 1996;25:1030–7.
29. Keane A. Too hard to swallow? The palatability of healthy eating advice. In: Caplan P, editor. Food, health and identity. London: Rutledge; 1997. p. 172–92.
30. Baumrind D. Rearing competent children. In: Damon W, editor. Child development today and tomorrow. San Francisco: Jossey-Bass; 1989. p. 349–78.
31. Berge JM, Wall M, Bauer KW, Neumark-Sztainer D. Parenting characteristics in the home environment and adolescent overweight: a latent class analysis. Obesity (Silver Spring). 2010;18:818–25.
32. Birch LL, Davison KK. Family environmental factors influencing the developing behavioral controls of food intake and childhood overweight. Pediatr Clin North Am. 2001;48:893–907.
33. Birch LL, Fisher JO. Mothers' child-feeding practices influence daughters' eating and weight. Am J Clin Nutr. 2000;71:1054–61.
34. Carper JL, Orlet FJ, Birch LL. Young girls' emerging dietary restraint and disinhibition are related to parental control in child feeding. Appetite. 2000;35:121–9.
35. Maccoby E, Martin J. Socialization in the context of the family: parent–child interaction. In: Hetherington EM, Mussen PH, editors. Socialization, personality, and social development. 4th ed. New York: Wiley; 1983. p. 1–101.
36. Kremers SP, Brug J, de Vries H, Engels RC. Parenting style and adolescent fruit consumption. Appetite. 2003;41:43–50.
37. Patrick H, Nicklas TA, Hughes SO, Morales M. The benefits of authoritative feeding style: caregiver feeding styles and children's food consumption patterns. Appetite. 2005;44:243–9.
38. Kim MJ, McIntosh WA, Anding J, Kubena KS, Reed DB, Moon GS. Perceived parenting behaviours pre-

dict young adolescents' nutritional intake and body fatness. Matern Child Nutr. 2008;4:287–303.
39. Berge JM, Wall M, Neumark-Sztainer D, Larson N, Story M. Parenting style and family meals: cross-sectional and 5-year longitudinal associations. J Am Diet Assoc. 2010;110:1036–42.
40. Neumark-Sztainer D, Hannan PJ, Story M, Croll J, Perry C. Family meal patterns: associations with sociodemographic characteristics and improved dietary intake among adolescents. J Am Diet Assoc. 2003;103:317–22.
41. McIntosh A, Kubena KS, Tolle G, et al. Determinants of children's use of and time spent in fast-food and full-service restaurants. J Nutr Educ Behav. 2011;43:142–9.
42. De Bourdeaudhuij I. Family food rules and healthy eating in adolescents. J Health Psychol. 1997;2:45–56.
43. De Bourdeaudhuij I, van Oost P. Family members' influence on decision making about food: differences in perception and relationship with healthy eating. Am J Health Promot. 1998;13:73–81.
44. Ofcom. Childhood obesity—food advertising in context. Children's food choices, parents' understanding and influence, and the role of food promotion. London: Office of Communications; 2004.
45. Wansink B. Nutritional gatekeepers and the 72 % solution. J Am Diet Assoc. 2006;106:1324–7.
46. Craig L. Does father care mean father share? A comparison of how mothers and fathers in intact families spend time with children. Gend Soc. 2006;20:259–81.
47. Baughcum AE, Chamberlin LA, Deeks CM, Powers SW, Whitaker RC. Maternal perceptions of overweight preschool children. Pediatrics. 2000;106:1380–6.
48. Cooke L, Wardle J, Gibson EL. Relationship between parental report of food neophobia and everyday food consumption in 2–6-year-old children. Appetite. 2003;41:205–6.
49. Glanz K, Basil M, Maibach E, Goldberg J, Snyder D. Why Americans eat what they do: taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. J Am Diet Assoc. 1998;98:1118–26.
50. Roos E, Lehto R, Ray C. Parental family food choice motives and children's food intake. Food Qual Prefer. 2012;24:85–91.
51. Drewnowski A. Obesity, diets, and social inequalities. Nutr Rev. 2009;67 Suppl 1:S36–9.
52. McDermott AJ, Stephens MB. Cost of eating: whole foods versus convenience foods in a low-income model. Fam Med. 2010;42:280–4.
53. Walker R, Dobson B, Middleton S, Beardsworth A, Keil T. Managing to eat on a low income. Nutr Food Sci. 1995;95:5–10.
54. Canadian Medical Association. National report card: general population. Ipsos Reid Corporation. Report No.: Final Summary Report; 2006.
55. Eckstein KC, Mikhail LM, Ariza AJ, Thomson JS, Millard SC, Binns HJ. Parents' perceptions of their child's weight and health. Pediatrics. 2006;117:681–90.
56. Garrett-Wright D. Parental perception of preschool child body weight. J Pediatr Nurs. 2011;26:435–45.
57. Dennison CM, Shepherd R. Adolescent food choice: an application of the theory of planned behaviour. J Hum Nutr Diet. 1995;8:9–23.
58. Baxter SD, Thompson WO, Davis HC. Trading of food during school lunch by first- and fourth-grade children. Nutr Res. 2001;21:499–503.
59. Hammons AJ, Fiese BH. Is frequency of shared family meals related to the nutritional health of children and adolescents? Pediatrics. 2011;127:e1565–74.
60. Rosenkranz RR, Dzewaltowski DA. Model of the home food environment pertaining to childhood obesity. Nutr Rev. 2008;66:123–40.
61. Garriguet D. Canadians' eating habits. Health Rep. 2007;18:17–32.
62. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. Pediatrics. 2004;113:112–8.
63. Stewart SD, Menning CL. Family structure, nonresident father involvement, and adolescent eating patterns. J Adolesc Health. 2009;45:193–201.
64. McNeal JU, Yeh CH. Consumer behavior of Chinese children: 1995–2002. J Consum Market. 2003;20: 542–4.
65. Jain A, Sherman SN, Chamberlin LA, Carter Y, Powers SW, Whitaker RC. Why don't low-income mothers worry about their preschoolers being overweight? Pediatrics. 2001;107:1138–46.
66. De Irala-Estevez J, Groth M, Johansson L, Oltersdorf U, Prattala R, Martinez-Gonzalez MA. A systematic review of socio-economic differences in food habits in Europe: consumption of fruit and vegetables. Eur J Clin Nutr. 2000;54:706–14.
67. Backett-Milburn KC, Wills WJ, Gregory S, Lawton J. Making sense of eating, weight and risk in the early teenage years: views and concerns of parents in poorer socio-economic circumstances. Soc Sci Med. 2006;63: 624–35.
68. World Health Organization. Health Behaviour in School-aged Children (HBSC) study: international report from the 2001/2002 survey, Eating habits; 2004.
69. Eaton DK, Kann L, Kinchen S, et al. Youth risk behavior surveillance—United States, 2009. MMWR Surveill Summ. 2010;59:1–142.
70. Harrison K, Marske AL. Nutritional content of foods advertised during the television programs children watch most. Am J Public Health. 2005;95:1568–74.
71. Adair LS, Popkin BM. Are child eating patterns being transformed globally? Obes Res. 2005;13:1281–99.
72. Dixon J, Banwell C. Heading the table: parenting and the junior consumer. Br Food J. 2004;105:182–93.
73. Schor JB, Ford M. From tastes great to cool: children's food marketing and the rise of the symbolic. J Law Med Ethics. 2007;35:10–21.
74. Gelperowic R, Beharrell B. Healthy food products for children. Br Food J. 1998;96:4–8.

15 Convenience Foods and Dietary Quality in Children

Ute Alexy

Key Points

- Studies showed the widespread use of convenience food in infants (i.e. commercial weaning food), children and adults.
- Composition of commercial weaning food is well-regulated by law; however, iodine content requires further alertness.
- Convenience food is a significant contributor of sodium and fat in the diet of children and adults.
- With increasing consumption of convenience food, values of a nutrient-based dietary quality score decline.
- Some studies indicate a positive association between convenience food consumption and body weight status.

Keywords

Convenience food • Dietary quality scores • Commercial weaning food • Body weight • Children

Abbreviations

CF	Convenience food
DONALD Study	Dortmund Nutritional Anthropometric Longitudinally Designed Study
ED	Energy density
HM	High meat
LM	Low meat
NQI	Nutrient Quality Index
VMP-Meal	Vegetable-meat-potato meal

Introduction

Pre-prepared food products, i.e. convenience foods (CF), are an important aspect of modern dietary habits. Consumer researchers reported an increasing trend towards the use of convenience food (CF) products during the last decades. Several studies showed their widespread use in children [1] and adults [2, 3]. In the UK, total sales of ready meals were valued at £1.78 billion

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in 2001, up 46 % on 1997. Those meals include: chilled (53 % of the value of retail sales); frozen (40 %); and room temperature (7 %)—including instant hot snacks [4].

This strong trend towards CF is attributed to several social changes, e.g., the increasing number of single or small family households, the increasing number of women in employment, a social trend towards more leisure time, or the break-up of the traditional family mealtimes, and an altogether increase in the demand for easily prepared and just-in-time meals [5, 6]. Simultaneously, the technical equipment of household kitchens (freezer, microwave) has increased [6]. A lack of cooking skills is predictive especially for the consumption of highly and moderately processed CF [7]. It is noticeable that this great demand for CF contravenes the negative attitudes towards CF and the negative image of these products regarding taste, nutritional value and healthiness [8].

The consumption of CF, especially of highly processed ready-to-eat meals, transfers responsibility for ingredients and nutrient content from the household to the food processor. Therefore, the regular CF consumption may affect overall dietary quality and health.

Definition

There is a lack of consensus about the definition of CF. Berghofer [6] cited the New Oxford Dictionary of English that stated: 'A CF is typically a complete meal that has been pre-prepared commercially and so requires minimum further preparation by the consumer'. This definition's limitation on complete meals does not reflect modern dietary habits and the actual food market, since also CF meal components, especially CF sauces, are very popular.

A more comprehensive definition of CF is available which defines it as products that transfer time and activities of preparation from the household manager to the food processor [9]. This definition would also include fast foods or takeaway food [3], but in the present text, we constrict on CF eaten at home.

Consumption

During life course, consumption of CF starts already during infancy. After 4–6 months of exclusive breastfeeding or formula feeding, the introduction of weaning food (beikost) starts. Weaning meals can be prepared at home by parents from fresh ingredients, but the food industry also offers a wide assortment of commercial products. Figure 15.1 shows consumption frequency of commercial weaning products by infants from the German DONALD (Dortmund Nutritional Anthropometric Longitudinally Designed) Study. Consumption frequency of commercial products depends on type of meal with highest frequencies in jarred vegetable-meat-meals, and decreased with age (Hilbig et al., unpublished data).

Due to the widespread use and the susceptibility of the young consumers, the composition of commercial weaning food is vitally important. A lot of aspects of composition are well-regulated by law, e.g. energy and fat content. However, some aspects, for example iodine content, require further alertness. Especially breastfed infants are in risk for inadequate iodine intakes without iodine-fortified weaning food [10, 11]. Iodine fortification is not required by law and for example in Germany, only half of products are fortified [10].

Another critical nutrient during infancy is iron. Infants in the second 6 months of life are at high risk for iron deficiency and iron deficiency anaemia, because of extraordinary requirements for growth. As human milk is a poor source of iron, complementary foods which are providing highly bioavailable iron should be introduced between 17 weeks of age at the earliest and 26 weeks of age at the latest [12]. In Germany, this iron-rich weaning food is traditionally a vegetable-meat-potato (VMP) meal. The meat content of commercial VMP was prescribed in 1996 to be 8–10 % of weight, whereas paediatric recommendations remained at about 12 %. A double-blinded randomised controlled trial examined the potential impact of the low meat content of commercial VMP meals on the iron status in a group of healthy well-nourished

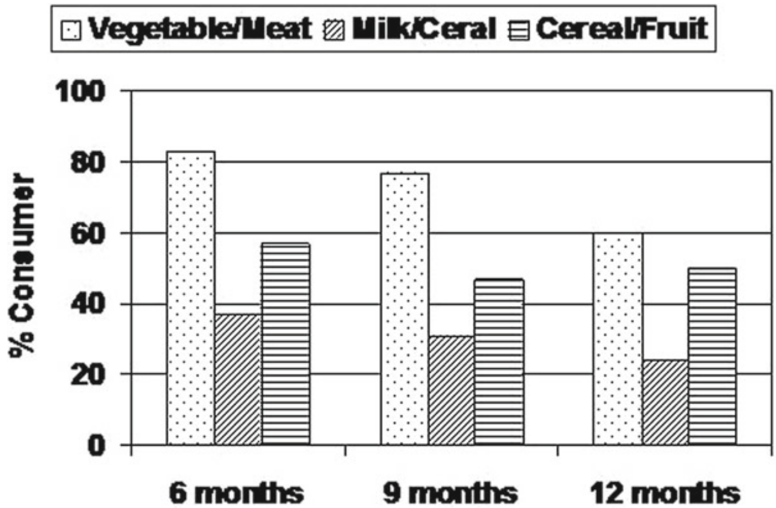

Fig. 15.1 Consumption of commercial weaning products during the second half year of life. Four hundred and ten infants from the DONALD Study, 1,167 3-day dietary records, 1989–1999 (Research Institute of Child Nutrition, unpublished data)

infants [12]. In the total sample ($N=97$), there were no differences in means of iron status biomarkers between infants of the low meat group (LM, receiving commercial VMP with 8 % meat reflecting the actual meat content in commercial baby jars) and the high meat group (HM, receiving commercial VMP with 12 % meat in accordance with paediatric recommendation). However, an analysis of the breastfed subgroup (fully breastfed 4–6 months, $N=53$) reveals a strong tendency towards an increased risk to develop a marginal iron status, since the mean haemoglobin concentration in the LM was lower than in the HM group, although the difference between the groups did not reach significance at the age of 10 months ($p=0.0563$).

Another aspect of diet during the first months of life is the development of food preferences. There is increasing evidence that early sensory experience shape and modify long-term food preferences. This begins during gestation and continues after birth. For example, preschool children, who were exclusively breastfed for 3 or more months had odds of 2.6 for consuming two or more servings of vegetables per day in comparison to children who were formula fed and/or partial breastfed [13]. There are clear sensory differences between commercial and homemade weaning food, due to the extensive heating of baby jars. Additionally, the variety of ingredients in commercial weaning food is only small. A recent market survey of German commercial baby menus showed that the predominant vegetable in baby jars is carrot (59 % of all jars), followed by tomato (15 %), parsnip (8 %), pumpkin (7 %) and spinach (3 %) (Fig. 15.2). Overall, only 12 sorts of vegetables were used by food industry [14]. Here, a higher diverseness would be desirable, since a higher variety early in weaning increases new food acceptance even long-term [15]. However, up to now, studies on the influence of commercial weaning food on later dietary habits are lacking.

After the age of 1 year, children can take part in the common family food. However, in Germany, an increasing number of special CF for toddlers are offered by the industry. Such toddler food not only includes special formula for children >1 year of age but also ready-to-eat meals with meat, vegetable and a starchy food in combination [16]. Data on the consumption frequency of such special toddler CF are lacking up to now.

During childhood, the consumption of CF increases with age. This is a result of an evaluation

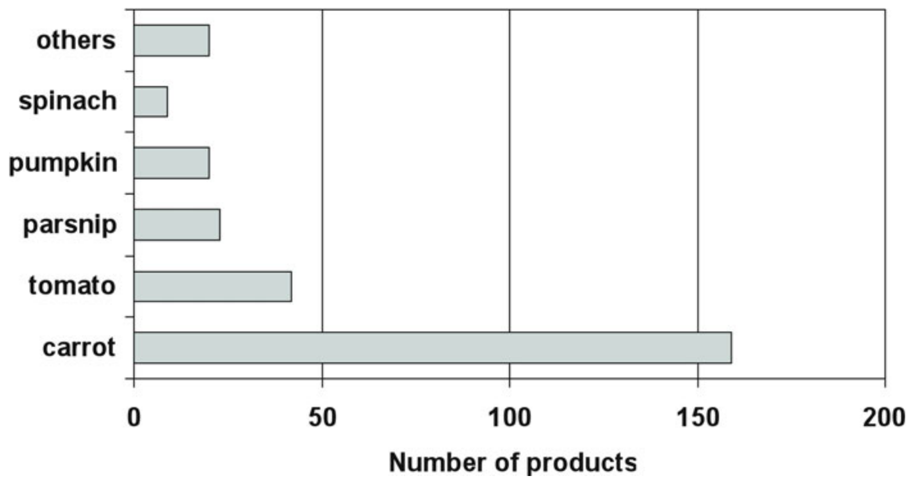

Fig. 15.2 Main vegetables in 273 commercial infant weaning products. Baby/junior menus, market survey, Germany, 2010, data from [14]

of the DONALD study on trends of CF intake in 3–18-year-old children and adolescents. In this analysis, CF has been defined as all pre-prepared savoury products. Sweet CF has been excluded, as well as CF eaten in communal feeding, e.g. day-care centres or schools. Using this definition, in 86 % of all 3-day dietary records, at least one CF product has been reported, in 21 % of the records on all three recorded days. CF consumption accounted for approximately 3 % in 3–8 year olds to 7 % in 14–18-year-old boys and 5 % in 14–18-year-old girls. Also the percentage of subjects who ate ≥20 % CF increased with age from 1 % in 3–8 year olds to 9 % in 14–18 year olds. During the analysed study period, 2003–2006, no time trend has been observed [1]. This is in accordance with another study in Germany [5], which reported that only 3 % of 2,200 families declared in a questionnaire never to use frozen CF, 16 % never used ready-to-eat meals and 14 % ready-to-eat soups. The widespread use of CF even in the diet of families is surprising, since another study showed that having children had a negative effect on CF consumption [7].

The most popular CF dishes in the DONALD Study were rice or pasta dishes and pizza, followed by soups (meals), sauces, vegetables and potato crisps (meal components) [17].

A recent study (2009) analysed the CF consumption of adults in Switzerland. Here, a frequency questionnaire composed of 17 common CF items was used, which was mailed to randomly selected households in Switzerland [7]. In this study, salad cut and/or washed in bags was the most consumed product with a mean 72.9 times per year, followed by frozen or canned vegetables (24.6 times per year), ready bought sandwiches (17.9 times), chilled fresh tortellini, pasta, gnocchi, etc., (15.5 times) and ready soups in bags or cans (13.9 times). Age was a negative predictor of consumption: The older the participant, the fewer convenience products he or she consumed [7], which was explained first by the availability of time for cooking after retirement, but also by a generation effect, since elderly are accustomed to conventional cooking. With respect to gender, men had a higher intake of ready meals than women [3].

Composition

It is widely assumed, that composition of CF is often not in agreement with current food-based dietary guidelines, because they are rich in energy, fat, salt, and sugar and lack the recommended servings of vegetables [3, 18]. However, only few surveys described the composition of a sample of CF products. An evaluation of the DONALD Study, the in-house food composition

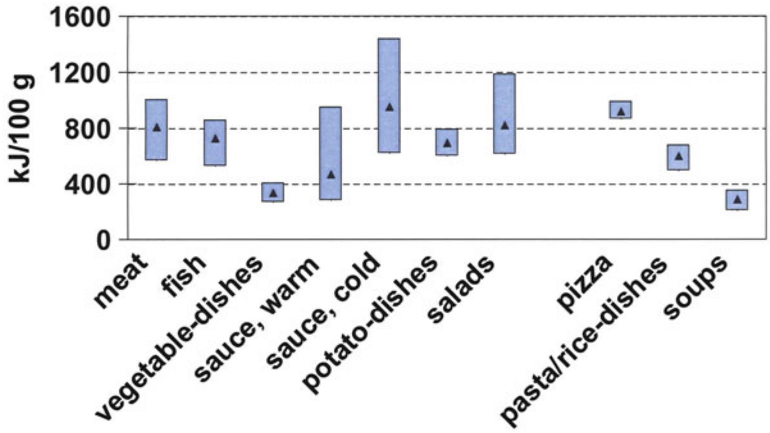

Fig. 15.3 Energy density (kJ/100 g) in 547 non-instant CF products consumed by 554 children and adolescents from the DONALD Study, 2003–2006. One thousand five hundred and fifty-eight 3-day dietary records, data from [1]

database LEBTAB was used to describe composition of CF eaten in the families of study participants. In LEBTAB, the energy and nutrient contents of composite foods are estimated by recipe simulation using labelled nutrient contents and ingredients. The ingredients of the simulated recipes are stored in LEBTAB [19]. From the 700 analysed products, 378 (54 %) products contained either flavourings and/or flavour enhancers [1]. This is not surprising, since the sensory quality of CF could be impaired by the warmed-over flavour and aroma losses from storage [6]. The use of high and standardised spicing in industrial food production results in a taste standardisation and a loss of the specific taste of 'mothers' cuisine'. In addition, the lower diversity in ingredients use can also contribute to taste standardisation. Studies demonstrating that sensory experiences in early childhood may have long lasting effects are increasing [20, 21]. Additionally, Berghofer [6] complained the agricultural disadvantage of the loss of biodiversity, if the number of plants used for human nutrition would further decrease.

Energy density (ED) of CF differed between dishes (Fig. 15.3): Highest median ED was found for pizza, which was the most popular complete CF meal in this study, salads (including salads on the basis of sausage or eggs) and cold sauces. Compared with the reference ED for warm lunch in schools, ED of most CF products was higher. The median % of energy from fat in all product groups was at least 35 %E [1].

Convenience Food and Dietary Quality

CF products are considered to be salty: In an Australian sample of 18 year olds, CF was the main contributor to sodium intake (22–27 % of total intake) [22]. In an evaluation of Finnish ready-to-eat foods from the leading brands, the reduction of salt content of almost all products has been recommended [18].

In German children and adolescents, CF had a higher impact on fat intake than on energy intake [1]. Compared with energy intake, percentage fat from CF was higher (mean: 1.3 %, $p<0.0001$), percentage carbohydrates from CF was lower (–0.95 %, $p<0.0001$). Also in an Australian survey with 18 year olds, CF was the major food group contributing to fat intake (32 % and 28 % in males and females, respectively) [1, 22].

To analyse the potential association between CF consumption and overall dietary quality, the Nutrient Quality Index (NQI) was calculated. This score determines the extent to which the nutritional recommendations for particular nutrients are met. In the present analysis, the intake of ten nutrients (vitamins E, A, C, B1 and folate, zinc, iron, magnesium, calcium and potassium;

individual mean of 3-day records) was compared with the recent age- and sex-specific dietary reference values issued by the German Nutrition Society. Percentages indicating the extent to which the recommendation was met were calculated. To avoid mathematical compensation of deficient intakes of one nutrient by exceeding intakes of another nutrient, percentage values were truncated at 100 if the intake of a nutrient exceeded the recommended level. For nutrients with a maximum desirable intake, percentage values >100 were reduced by the exceeding amount. The NQI was calculated as the individual harmonic mean of each subjects' nutrient components. This is more sensitive to imbalances in nutrient intake than the arithmetic mean. As a consequence, a low intake of one nutrient is not as easily compensated for by a high intake of another nutrient. The NQI score theoretically ranges from 0 to 100, with a higher score indicating better diet quality. Mean (SD) NQI in the sample was 75.7 (13.8), indicating an overall good dietary quality. With increasing intake of CF (% of total food intake), the values of NQI decreased (−0.1, $p = 0.0054$). Regarding the association between CF intake and food groups, the consumption of dairy decreased (−0.54 g/MJ, $p < 0.0001$) and of fat increased (+0.03 g/MJ, $p \leq 0.0001$) with increasing CF intake. No significant association was found for fruits and vegetables ($p = 0.09$) [23].

Convenience Food and Body Weight

There is some evidence of a positive association between the consumption of CF and body weight status. In a cross-sectional study, overweight respondents were more likely to consume ready meals (defined as meals that require few or no extra ingredients and are designed to replace the main course of a homemade meal) compared with normal-weight respondents. Also the attitudes of overweight subjects towards ready meals differ: Overweight persons perceived ready meals as containing more vitamins and nutrients compared with normal weight persons [3].

Two properties of CF are discussed to mediate the adipogenic effect of CF: the high ED of many products, or the high sodium content. ED is discussed as an important factor that determines total daily energy intake [24, 25]. In the DONALD Study, no association between long-term CF consumption and changes in parameters of body weight were found [23]. Only among boys, the baseline consumption of CF with a high ED significantly predicted change in % of body fat during the study period (β 0.104, $p = 0.0098$).

The high salt content of CF might indirectly act adipogenic due to an increasing consumption of sugar containing beverages. In an analysis from the National Diet and Nutrition Survey in Great Britain, the consumption of salt predicted the intake of sugar-sweetened soft drinks in children. Each additional gram of salt intake daily was associated with additional consumption of 27 g of soft drinks daily [26]. However, evidence from studies on the effect of soft drink consumption on body weight is controversial, but overall the literature on this topic suggests that the replacement of these beverages by non-caloric alternatives is efficient for the prevention of weight gain in children and adolescents [27–29]. In an analysis of the DONALD Study, urinary sodium was positively associated with BMI-SDS and % of body fat in boys and girls ($N = 364$). However, these associations remained significant after adjustment for sugar-sweetened beverage consumption and total energy intake. These results suggest that a high intake of processed salty foods could have a negative impact on body weight status in children and adolescents. However, this relationship seems to be independent from the consumption of sugar-sweetened beverages [30].

Conclusions

Studies on the effect of CF consumption on diet quality and indirectly on health are rare, although the relevance of these food groups on all-day dietary habits is beyond controversy. The few existing studies confirm the negative attitude of consumers towards CF, i.e. CF decreases diet quality and may contribute to the development of adiposity. However, it cannot be expected, that CF disappears from the food market or the kitchens. Therefore, the food industry is requested to offer healthy products with a composition of ingredients in accordance with the actual food-based dietary guidelines. The nutritional science should assist the food industry in this task.

Some studies showed the association between cooking skills and use of CF [3]. Probably, the loss of the ability to cook healthy meals from scratch will further increase, although the preparation of meals and the handling of foods can be seen as an important part of culture.

References

1. Alexy U, Sichert-Hellert W, Rode T, Kersting M. Convenience food in the diet of children and adolescents: consumption and composition. Br J Nutr. 2008;99:345–51.
2. Pryer JA, Nichols R, Elliott P, Thakrar B, Brunner E, Marmot M. Dietary patterns among a national random sample of British adults. J Epidemiol Community Health. 2001;55:29–37.
3. van der Horst K, Brunner TA, Siegrist M. Ready-meal consumption: associations with weight status and cooking skills. Public Health Nutr. 2010;14:239–45.
4. Food Standards Agency. The ready meals market. 2003 [cited November 2011]; Available from: www.food.gov.uk/news/newsarchive/2003/jun/readymealsmarket
5. Diehl J. Verbraucherverhalten bei Convenience-Food, Teil 1. Ernähr Fokus. 2001;1:91–4.
6. Berghofer E. Exploitation of convenience food in view of a diet diversification for better nutrition. Forum Nutr. 2005;57:112–23.
7. Brunner TA, van der Horst K, Siegrist M. Convenience food products. Drivers for consumption. Appetite. 2010;55:498–506.
8. Geeroms N, Verbeke W, Van Kenhove P. Consumers' health-related motive orientations and ready meal consumption behaviour. Appetite. 2008;51:704–12.
9. Buckley M, Cowan C, McCarthy M, O'Sullivan C. The convenience consumer and food related lifestyles in Great Britain. J Food Prod Market. 2005; 11:3–25.
10. Alexy U, Drossard C, Kersting M, Remer T. Iodine intake in the youngest: impact of commercial complementary food. Eur J Clin Nutr. 2009;63:1368–70.
11. Andersson M, Aeberli I, Wust N, Piacenza AM, Bucher T, Henschen I, et al. The Swiss iodized salt program provides adequate iodine for school children and pregnant women, but weaning infants not receiving iodine-containing complementary foods as well as their mothers are iodine deficient. J Clin Endocrinol Metab. 2010;95:5217–24.
12. Dube K, Schwartz J, Mueller MJ, Kalhoff H, Kersting M. Complementary food with low (8%) or high (12%) meat content as source of dietary iron: a double-blinded randomized controlled trial. Eur J Nutr. 2009; 49:11–8.
13. Burnier D, Dubois L, Girard M. Exclusive breastfeeding duration and later intake of vegetables in preschool children. Eur J Clin Nutr. 2011;65:196–202.
14. Mesch C, Stimming M, Kersting M, Libuda L. Lebensmittelvariation in kommerziellen Menüse für Säuglinge [Food variation in commercial baby infant menus]. Proc Germ Nutr Soc. 2011;15:80.
15. Maier AS, Chabanet C, Schaal B, Leathwood PD, Issanchou SN. Breastfeeding and experience with variety early in weaning increase infants' acceptance of new foods for up to two months. Clin Nutr. 2008;27: 849–57.
16. Düren M, Kersting M. Das Angebot an Kinderlebensmitteln in Deutschland. Ernähr Umschau. 2003;50:16–21.
17. Alexy U, Sichert-Hellert W, Rode T, Kersting M. Convenience Food in der Ernährung von Kindern und Jugendlichen. Ernährung Wissenschaft Praxis. 2007;1:396–401.
18. Outila TA, Simulainen H, Laukkanen TH, Maarit Kyyro A. A simple way of evaluating the healthiness of ready-to-eat foods and developing healthy foods in the food industry. Int J Food Sci Nutr. 2006;57: 137–42.
19. Sichert-Hellert W, Kersting M, Chahda C, Schaefer R, Kroke A. Commercial food products in a Food Composition Data Base for dietary evaluation in pediatric age groups. J Food Compos Anal. 2007;20: 63–70.
20. Mennella JA, Griffin CE, Beauchamp GK. Flavor programming during infancy. Pediatrics. 2004;113: 840–5.
21. Mennella JA, Beauchamp GK. Flavor experiences during formula feeding are related to preferences during childhood. Early Hum Dev. 2002;68:71–82.
22. Milligan RA, Burke V, Beilin LJ, Dunbar DL, Spencer MJ, Balde E, et al. Influence of gender and socio-economic status on dietary patterns and nutrient intakes in 18-year-old Australians. Aust N Z J Public Health. 1998;22:485–93.
23. Alexy U, Libuda L, Mersmann S, Kersting M. Convenience foods in children's diet and association with dietary quality and body weight status. Eur J Clin Nutr. 2011;65:160–6.
24. Ello-Martin JA, Ledikwe JH, Rolls BJ. The influence of food portion size and energy density on energy intake: implications for weight management. Am J Clin Nutr. 2005;82:236S–41.
25. Drewnowski A. The role of energy density. Lipids. 2003;38:109–15.
26. He FJ, Marrero NM, MacGregor GA. Salt intake is related to soft drink consumption in children and ado-

lescents: a link to obesity? Hypertension. 2008;51: 629–34.
27. Libuda L, Kersting M. Soft drinks and body weight development in childhood: is there a relationship? Curr Opin Clin Nutr Metab Care. 2009;12:596–600.
28. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. Am J Public Health. 2007;97:667–75.
29. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. Am J Clin Nutr. 2008;87:1662–71.
30. Libuda L, Kersting M, Alexy U. Consumption of dietary salt measured by urinary sodium excretion and its association with body weight status in healthy children and adolescents. Public Health Nutr. 2012;15(3):433–41.

16 Nutritional Education of Secondary Education Students and Diet Quality

María Isabel Martinez-Martinez
and Jose Martinez-Raga

Key Points

- Nutritional education is an effective tool to facilitate individuals to make adequate choices and decisions directed towards protecting, promoting or restoring health and well-being, as it helps them select and acquire food intelligently and, in the case of adults, to prepare, manipulate, combine and store foods.
- Nutritional education aims at helping individuals and their families to improve their health status and to help preventing nutrition-related health problems by establishing long-standing dietary habits and gaining a better understanding of the strong relationship between nutrition and health.
- There is also strong evidence of the beneficial effects of child overweight and obesity prevention programs, particularly for programs targeting children in primary and secondary education.
- Childhood obesity is associated with a large variety of long-term and in most cases severe adverse health consequences and comorbidities, including cardiovascular, respiratory gastrointestinal and metabolic disorders, orthopedic and rheumatologic complications, certain types of cancer and several psychosocial or psychiatric problems, as well as an increased and significant risk for adult obesity.
- Over the last 2 decades there has been a threefold increase in the number of children, adolescents and young adults with obesity in Spain mainly due to an increase in a sedentary lifestyle and changes in the dietary habits.

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- The available evidence shows that it is easier to promote healthy eating and lifestyles habits during childhood and early stages of adolescence than to modify or restructure them later in life.
- Solid evidence supports the beneficial effects of child obesity prevention programs and the specific importance of nutritional education. To maximize efficiency it is important to reinforce multidisciplinary prevention programs addressing both nutritional elements and lifestyles changes, including physical and leisure activities, both at home and in the school.
- Childhood and adolescence are key priority educational moments to generate long-term beliefs, attitudes and values related to overall health and particularly towards nutrition.

Keywords
Nutritional education • Adolescent obesity • Overweight • Mediterranean diet • Secondary education

Abbreviations

%BF	Percentage of body fat
BMI	Body mass index
NAOS strategy	Strategy for nutrition, physical activity and the prevention of obesity (Estrategia Nacional de Nutrición, Actividad Física y Prevención de la Obesidad)
SD	Standard deviation
WC	Waist circumference
WHO	World Health Organization

Introduction

Over the recent 3 decades changes in food and dietary habits have lead to an increase in obesity, diabetes and chronic disorders such as cardiovascular disease (CVD) worldwide [1]. As a consequence, there has been a greater recognition and awareness of the importance of nutritional education as an essential element in the prevention and control of food intake and diet-related problems [2]. Nutritional education has been defined as any combination of learning strategies designed to facilitate voluntary adoption of food choices and other food- and nutrition-related behaviors directed to health and well-being [3]. The goals of nutritional education are to help individuals and their families to establish long-standing dietary habits to gain a better understanding of the strong relationship between nutrition and health, to improve their health status and to help preventing nutrition-related health problems.

Nutritional education is an effective tool to facilitate individuals to make adequate choices and decisions directed towards protecting, promoting or restoring health and well-being, as it helps them select and acquire food intelligently and, in the case of adults, to prepare, manipulate, combine and store foods. A good knowledge of foods and appropriate healthy eating practices are required for improving access and consumption of nutritionally balanced diets, which in turn is essential for achieving a nutritional well-being [4].

It is estimated that by 2020 two-thirds of the global burden of disease will be attributable to chronic non-communicable diseases, therefore becoming the principal global causes of morbidity and mortality, most of them strongly associated with diet [5]. Worldwide there are two major problems associated with diet and nutrition:

1. Overnutrition, which causes overweight and obesity, is the primary etiological factor in a number of disease processes, including

metabolic/endocrine disease, cancer, osteoarthritis, CVD, or obstructive lung disease [6, 7].
2. Under-nutrition, which is directly related with hunger and is an underlying cause of chronic malnutrition resulting in stunting and wasting, together with micronutrient deficiencies [8].

Both when there is a lack or an excess of food supplies, it is essential that individuals become aware of implementing the resources that ensure their nutritional well-being and consequently enhances their health status. To reach this well-being, it is necessary that individuals gain access to sufficient foods and of good quality and that they understand what is implied in an adequate and healthy diet. They also need to be aware of the skills, behaviors and motivations leading to the correct food choices. Failure to provide information may result from several problems, such as:

- Lack of awareness on nutrition education by the general population and failure to pay adequate attention to the relationship between health status and food intake.
- Inadequate or lack of nutrition education at school and in the school curriculum, with a much more limited access to information, available resources and teaching materials, as well as a consequence the absence of an effective and pragmatic learning model on nutritional education focused more on action and attitudes than on mere information [4].
- Underestimation and limited financial support by governments and policy makers.
- Insufficient funding for disseminating nutrition information. Overall, communication of nutritional facts is deficient in most countries and usually the language used is very technical and advice given is often perceived as not very practical.

In order to overcome these problems, the Global Forum on Food Security and Nutrition (FSN Forum) have suggested [2, 9] a series of measures, including:

- To adopt a multidisciplinary approach and reinforce coordination at national level. Nutrition education ought to be included at all levels of education, from primary school to university. Nutrition education in schools is important for the development of the individual skills and motivation needed to adopt healthy food practices. Therefore, primary and secondary schools can contribute decisively to the overall efforts of the community to reach food safety and nutritional well-being.
- Children and adolescents should be considered as tomorrow's adult consumers. Food habits are learned at an early age and schools may play an important role in promoting adequate criteria for food selection, and healthy and long-term dietary patterns [9].

In the present global scenario of limited resources and raising healthcare expenditure the development of preventive measures and strategies directed towards addressing and avoiding the adverse health and societal consequences of overnutrition and poor dietary habits are becoming increasingly important. There is a growing recognition that prevention demands public health actions at both the national and global levels [1, 2, 4]. There is also strong evidence of the beneficial effects of child overweight and obesity prevention programs, particularly for programs targeting children in primary and secondary education [10]. Within this context nutritional education is clearly a very important prevention approach.

The first part of the chapter provides an overview of childhood and adolescent overweight and obesity and the problems associate with both, particularly in the Mediterranean countries and specifically in Spain. In the second part, an outline is provided of a nutritional educational program developed with secondary education students in Valencia (Spain), as well as the main outcomes of the program, aiming at healthier eating and lifestyle habits and focusing at the Mediterranean Diet.

Overnutrition, Overweight and Obesity

Overnutrition is directly associated with obesity, the most prevalent nutritional and metabolic disorder not only in industrialized, but in developing countries with important socioeconomic disparities, and even in countries where hunger is endemic, as well [5, 11]. The epidemic of overweight and obesity is one of the leading causes of morbidity and mortality and is associated with raising direct and indirect societal and economic costs, worldwide, therefore becoming a serious public health concern [6, 8, 12–14]. Specifically, childhood obesity is associated with a large variety of long-term and in most cases severe adverse health consequences and comorbidities, including cardiovascular, respiratory gastrointestinal and metabolic disorders, orthopedic and rheumatologic complications, certain types of cancer and several psychosocial or psychiatric problems, as well as an increased and significant risk for adult obesity (Table 16.1). A good knowledge of the causes and the multiple and complex interrelationships between childhood obesity and these disorders is essential for changing or modifying unhealthy dietary and lifestyle habits.

Obesity is defined as the excessive accumulation of body fat or adipose tissue. Whilst environmental factors, lifestyle preferences, and cultural environment are key factors to understand the rising prevalence of obesity worldwide, this chronic, multifactorial disorder is the result of the complex interactions between genetic, neuroendocrine, metabolic, psychological, environmental, and sociocultural factors [11, 12]. These are particularly evident in childhood and adolescent onset obesity. Childhood and adolescent obesity dramatically increases the risk of adult obesity, and has therefore been identified as a valid predictive factor of adult obesity [15]. Assessment of overweight in children and adolescents is more difficult than in adults due to a number of factors such as the changes in body fat during development. The fast growth rate and the developmental processes during adolescence affect body size, structure and composition. Nutrition therefore plays a key role during this period [16, 17].

Body weight tends to be associated with adiposity; however weight alone is an insufficient measure of obesity, because it is correlated with height [18]. A number of anthropometric methods, through direct measurements of various body parameters, are most commonly used to measure body fat in human subjects [18]. By calculating body fat from skinfold measurements and indicators

Table 16.1 Adverse long-term health consequences and comorbidities of childhood obesity [5, 6, 11–15]

Cardiovascular	Atherosclerosis
	Coronary heart disease (CHD)
	Congestive cardiac failure
	Cerebrovascular disease (hemorrhagic and non-hemorrhagic)
	Hypertension
	Deep venous thrombosis
Gastrointestinal	Gallstones
	Nonalcoholic fatty liver disease steatohepatitis
	Liver fibrosis
Metabolic/endocrine disorders	Insulin resistance
	Type 2 diabetes mellitus (NIDDM)
	Dyslipidemia
	Gout
	Menstrual abnormalities
	Polycystic ovary syndrome
	Obesity in adulthood
Orthopedic/rheumatologic	Osteoarthritis
	Hip fracture
	Extremity fractures
Psychological	Low self-esteem
	Depression
	Eating disorders
	Obsessive concern about body image
	Sleep disorders
Respiratory	Asthma
	Increased bronchial hyperactivity
	Obstructive sleep apnea
Other	Cancer (breast, ovaries, uterus and prostate cancer, colorectal cancer, …)
	Increased healthcare costs and disability
	Premature mortality

of biomedical, waist circumference or waist-hip ratio it is possible to assess the nutritional status of the adolescent and whether overweight or obesity is present [15]. The measurement of weight in relation to height, via body mass index (BMI), is a valuable indirect and the most common measure of body fat in clinical practice and in research [10, 19, 20].

Overweight and obesity in children and adolescents are generally defined using age- and sex-specific normograms for BMI [11]. Children with BMI equal to or exceeding the age-gender-specific 85th percentile (P_{85}) are considered overweight. Although some authors have suggested that only those children with BMI equal to or above the P_{98} should be defined obese [21, 22], in clinical practice, as well as the majority of reports and studies rely on the P_{95} for defining child or adolescent obesity [11, 18].

Obesity in Spain and Other Mediterranean Countries

The *enKid* study, a cross-sectional study conducted on a national random sample of the Spanish population aged 2–24 years revealed the magnitude and increasing trends of obesity among Spanish children and youth [23]. Over the last 2 decades there has been a threefold increase in the number of children, adolescents and young adults with obesity in Spain mainly due to an increase in a sedentary lifestyle and changes in the dietary habits [24].

The prevalence of childhood obesity in the *enKid* study was amongst the highest in Europe, with 12.4 % of children and youngsters estimated to have overweight and a further 13.9 % having obesity, based on BMI and considering the P_{85} and the P_{97} as cutoffs for overweight and obesity of the Spanish reference distribution and growth charts, respectively [23, 25]. Based on the P_{95} cutoff, the prevalence of obesity in the *enKid* study in adolescents aged 12–18 years was approximately 9 % in male children and 7 % in girls. In addition, obesity was significantly associated with low education level of the mother and low socioeconomic [23, 26].

Dietary habits are acquired during early childhood, at 3 or 4 years of age, tend to consolidate long-term during adolescence. The latter is a critical period for acquiring healthy dietary practices and lifestyles that will influence eating behaviors during adulthood [24, 25]. The available evidence shows that it is easier to promote healthy eating and lifestyles habits during childhood and early stages of adolescence than to modify or restructure them later in life [25, 27]. Therefore, although adequate nutrition and food intake is important across the lifespan, this is particularly so during childhood, school age, and adolescence, which play a very important part in health prevention and promotion [10, 13, 27].

The vast majority of cases of obesity are due to unhealthy dietary habits and lack of regular physical activity. High consumption of cakes and buns, soft-drinks, and other products with a high fat content and low physical activity levels, including spending 3 or more hours watching TV daily are well-recognized lifestyle factors associated with an increased likelihood of being obese [4, 25, 28]. For instance, the prevalence of obesity is higher in individuals that have no or very little breakfast [23, 26], and in Spain an estimated 8 % of Spanish children go to school without having had breakfast and only 26 % after having had a full breakfast [28]. In addition, technological and transport progress have favored a more sedentary lifestyle that often neglects daily significant physical activity. Childhood leisure activities have also become increasingly sedentary. Available data indicate that Spanish children and adolescents spend an average of two and half hours watching television and a further half an hour playing videogames [26]. These data further highlight that the best treatment to tackle overweight and obesity is prevention, particularly when initiated in childhood [3, 5].

Nutritional education is a basic element in the prevention of childhood and adolescent overweight and obesity. Within this context, the Spanish Ministry of Health set up the National Strategy for Nutrition, Physical Activity and the Prevention of Obesity (*Estrategia Nacional de Nutrición*, *Actividad Física y Prevención de la Obesidad or* NAOS strategy), aiming at changing the trend of the growing prevalence of overweight and obesity, raising public awareness of the problems of obesity as a public health priority, encouraging citizens, particularly children and young people, to adopt healthy lifestyles, primarily through healthy diets and regular physical activity, and establishing a working relationship between the Public Administrations, experts in the field, the private sector and the general population [25, 29, 30]. Furthermore, the *NAOS strategy* recommends implementing classroom and extra curricula activities at school to promote healthy eating habits and regular physical activity and sport, considering the school environment as one of the most efficient places for altering the lifestyles of children and adolescents [29, 30].

Development of a Nutritional Education Program with Secondary Education Students

We conducted an intervention study to better characterize the nutritional and lifestyle habits of secondary education students in our setting, as well as to assess the feasibility and effectiveness of a nutritional education program [31]. The program included both theoretical and practical activities. A total of 372 adolescents aged 12–16 years (181 females and 191 males) from a secondary school (Instituto de Enseñanza Secundaria or IES) in Valencia (Spain) participated in this project aimed at promoting healthy dietary and lifestyle habits and implementing a nutritional intervention for those with overweight or obesity. Specifically, the nutritional and lifestyle habits, the rates of overweight and obesity and the effects of a nutritional education program were evaluated in a sample of students in mandatory secondary education (Enseñanza Secundaria Obligatoria or ESO).

Phase 1: Assessment of Dietary Habits and Nutritional Status

Prior to starting the program, each student enrolled in the study was thoroughly assessed. Baseline assessments with complete anthropometric measurements, including weight, height, waist circumference (WC) and percentage of body fat (%BF), were conducted on all subjects using standardized methods. These helped to calculate the daily energetic needs of participating students and distribute a diet with hypocaloric balance adapted to age. Overweight and obesity in the sample were defined based on age- and sex-specific BMI, so that a BMI equal to or above the P_{85} and the P_{95} was used as cutoff for overweight and obesity, respectively. Table 16.2 shows the mean BMI values with standard deviation (SD), the lowest value (zero percentile), the maximum value (percentile 100), the median (50th percentile), as well as the cutoff points of the P_{85} (used as an index of overweight) and of the P_{95} (used as an index of obesity) of the BMI. All these BMI data are grouped by gender and by age.

Based on the BMI, 36 adolescents (18 males and 18 females) in the sample had overweight and 28 (12 males and 16 females) had obesity. From this group of 64 students with overweight

Table 16.2 BMI values of adolescent boys and girls aged 12–16 years included in the study [31]

	12 years	13 years	14 years	15 years	16 years
Females	*n* = 36	*n* = 47	*n* = 36	*n* = 35	*n* = 27
Mean (SD)	20.77 (4.79)	22.09 (4.46)	22.84 (3.52)	22.24 (2.93)	23.14 (3.88)
P_0	14.52	14.39	16.31	13.93	17.75
P_{50}	20.60	21.46	22.15	21.85	22.43
P_{85}	24.10	27.07	26.00	24.64	27.31
P_{95}	29.79	30.26	30.45	27.35	31.02
P_{100}	34.38	33.56	30.86	27.59	31.61
Males	*n* = 36	*n* = 41	*n* = 35	*n* = 42	*n* = 37
Mean (SD)	21.59 (3.87)	21.01 (3.94)	22.36 (4.23)	22.17 (4.10)	21.58 (2.96)
P_0	14.18	15.04	15.48	16.71	16.67
P_{50}	21.04	20.68	21.74	21.82	21.33
P_{85}	26.28	25.92	26.38	24.17	24.17
P_{95}	28.48	28.16	30.74	29.75	25.23
P_{100}	28.92	30.23	33.30	37.08	32.66

SD standard deviation

or obesity, six changed or left school and one was expelled prior to finishing the study, 14 refused to participate in the study, and six additional students abandoned the study prematurely. Therefore, 38 students (19 girls and 19 boys) aged 12–16 years completed the nutrition intervention for overweight or obesity, which included individualized Nutritional Intervention.

Baseline socio-demographic characteristics, family history data, lifestyle, and nutritional habits were evaluated with a self-report questionnaire. The instrument provided information on the average consumption of different foods at individual and group level and their distribution during the day. To assess the educational needs in the study sample and conduct a more effective nutritional education we also explored the knowledge and attitudes towards the Mediterranean diet with the Mediterranean Diet Quality Index for children and adolescents (KIDMED) [32]. The KIDMED was administered again at the end of the study to assess the behavioral changes following the nutritional intervention. As seen Table 16.3, the KIDMED index is a 16-item instrument specifically devised for children, adolescents and young adults that can either be used as a self-report measure or administered by a trained interviewer. The index ranges from 0 to 12 and was originally developed based on principles sustaining Mediterranean dietary patterns as well as those undermining it [32]. Items with a negative view towards the Mediterranean diet were valued as −1, while those with a positive connotation +1. As a result, the total sum of the values on the different items was classified into three levels:

- ≤3: low quality diet
- 4–7: improvement in the food pattern is needed to adapt it to the Mediterranean diet
- ≥8: good Mediterranean quality diet

At follow-up assessments, students were regularly asked to provide details about their daily diet during the previous week, which was then used to provide feedback. This was used to assess the dietary habits of students throughout the program as well as their daily or weekly exercise pattern and their psychological state.

Table 16.3 KIDMED test to assess the Mediterranean diet quality during childhood and adolescence [40]

Diet quality	Scoring
Takes a fruit or fruit juice every day	+1
Has a second fruit every day	+1
Has fresh or cooked vegetables regularly once a day	+1
Has fresh or cooked vegetables more than once a day	+1
Consumes fish regularly (at least 2–3 times per week)	+1
Goes more than once a week to a fast-food (hamburger) restaurant	−1
Likes pulses and eats them more than once a week	+1
Consumes pasta or rice almost every day (5 or more times per week)	+1
Has cereals or grains (bread, etc.) for breakfast	+1
Consumes nuts regularly (at least 2–3 times per week)	+1
Uses olive oil at home	+1
Skips breakfast	−1
Has a dairy product for breakfast (yoghurt, milk, etc.)	+1
Has commercially baked goods or pastries for breakfast	−1
Takes two yoghurts and/or some cheese (40 g) daily	+1
Takes sweets and candy several times every day	−1

KIDMED Mediterranean Diet Quality Index in children and adolescents

Phase 2: Nutrition Education Program in the Classroom

The program was particularly directed towards secondary education students and their teachers. Initially the work was conducted with the students tutoring hours in theoretical and practical workshops with the following topics:

- Healthy breakfast
- The nutritional pyramid: balanced food
- Comparison of two dishes with practically the same ingredients, but prepared and cooked differently, thus with different caloric content

As evidenced in the *NAOS* strategy, 38 % of young Spaniards identify themselves as being sedentary [25, 30]. When considering the average global daily time spent in sedentary activities (studying, TV, computer, videogames, …), prevalence of obesity is higher in adolescents that dedicate more time in these activities [33]. Watching TV has become the second most common activity in young individuals, only after sleeping, and the main leisure activity and free-time activity, with a lot of time spent as well on videogames and new technologies [6, 23]. In our sample there were substantial differences between boys and girls, independently of age, on these behaviors, as girls spent less time watching TV and more time reading than boys. Prevalence of obesity is much higher among adolescent who do not practice any sports, compared to those who practice sports in a regular basis. It is therefore important to promote both activities of low energetic balance such as routine home chores, as well as those with high-energy consumption, such as structured exercise or thrice weekly sport practice [34, 35].

Breakfast is a very important meal, particularly in school-age children. It should contain 20–25 % of total daily calories intake. A balanced meal during the first hours of the day reduces compulsive eating and consumption of inadequate foods, improves intellectual abilities, and physical as well as cognitive and academic performance [36, 37]. Breakfast should contain dairy products, cereals, and some fruit or fresh fruit juice. As indicated by the *enKid* study, the BMI decreases in both genders with the increase of calories in breakfast. Prevalence of obesity is higher among those children and adolescents who take very little (1–15 % of daily caloric intake) or no breakfast compared to those who take a large breakfast and this association is stronger for males than for females [23]. Indeed, it has been suggested that omitting breakfast may induce metabolic changes responsible for future development of obesity [38].

Phase 3: Nutritional Intervention in Students with Overweight and Obesity

A nutritional program cannot solely include the list of concepts and notions associated with healthy eating. The goals have to be realistic and adapted to the targeted population. It is also important to provide the child or adolescent with the necessary skills to promote permanent lifestyle changes, partly by helping them to develop self-efficacy and self-control strategies and reinforce healthy behaviors. To facilitate such a change it is essential to restructure certain circumstances in the immediate environment of the individual, in addition to influence on the dietary habits and the physical activity environment. Any intervention has to address the cues and craving associated with eating certain foods, while replacing them with others that facilitate a healthy and balanced food intake and which will serve for future illness prevention [39]. The different activities should ultimately aim at health promotion, particularly for adolescents with overweight or obesity [40].

It is also important to implement strategies directed towards controlling and modifying negative emotions inherent to obesity [13]. Similarly, it is also necessary to enhance motivation changing lifestyle and nutritional habits. Therapeutic dietary modifications should allow for satisfying the nutritional needs of the individual without interfering the growth rate and the adequate development. The main goal of nutritional programs is weight stabilization not simply weight loss. Dietary changes have to be planned in the context of promoting a realistic body weight, potentiating self-esteem, and acceptance of the body image [39].

Individual follow-up meetings were held with students with baseline overweight or obesity to discuss their daily food intake, going over each meal and each group of foods on a daily basis with the help of self-report diaries. Rather than quantifying the different foods a color scheme was used for the different groups of foods. Physical exercise was also emphasized at the individual level, and depending on the preferences and capacities to avoid a sedentary lifestyle. Therefore, it was aimed that with short-term and realistic goals the targeted students gradually implemented the nutritional knowledge and lifestyle changes addressed during the program.

The family plays a key role in modeling and consolidating the child's dietary habits. The participation of parents can therefore be very important, particularly in the preparation of daily menus. This is a critical point that may create some difficulties, as no all parents may be willing to participate. To facilitate this we conducted a series of meetings with the Association of Mothers and Fathers of Students to present and address the specific topics and subsequently reach a greater adherence to the program. However, different studies that incorporated the parents to the treatment of their adolescent sons and daughters failed to find any effectiveness in inducing any behavioral changes, or even had a detrimental effect [26, 41]. Even in studies with very positive results, only one-third of children and adolescents reached and maintained a very optimal weight [41].

Practical Outcome Results of the Program

Changes in the Anthropometric Measures Following the Nutritional Intervention

Regular measurements of BMI and a variety of other anthropometric parameters, including WC, %BF, arm circumference, triceps skinfolds, and subscapular skinfolds were conducted during follow-up (Table 16.4). There were statistically significant differences in the BMI, WC, %BF and arm circumference following the nutritional intervention.

Assessment of the Changes in the Dietary Habits After the Nutritional Education

A total of 217 study participants (103 females and 114 males) aged 12–16 years completed the KIDMED prior and after receiving nutritional education. Changes in the KIDMED index scores and in the percentage of food intake prior and after the program were analyzed to assess the efficacy of the nutritional intervention (Table 16.5).

As shown in Table 16.5, the percentage of students having a daily piece of fruit or a fruit juice increased by 17.6 %, whilst an additional 18.4 % were having a second piece of fruit every day after the nutritional program. Similarly, 14.8 % more students and 18.4 % more students were eating fresh or cooked vegetables once a day and more once a day, respectively. While intake of nuts, olive oil, and pulses practically remained unchanged after the nutritional unchanged, 9.8 % more study participants were eating fish regularly, and 9.2 % more of the sample were eating pasta or rice, carbohydrates, a central element of the Mediterranean diet, almost every day almost every day after the intervention. In contrast, 12 % less participants were going to fast-food restaurants every week, and 11.1 % less of them were having sweets or candies several times every day, after completing the program. The latter two items reflect the significant improvements in dietary and nutritional habits, which were particularly evident in those students with overweight or obesity, so that 89.5 % of kids with overweight

Table 16.4 Anthropometric parameters at baseline and following the nutritional intervention

	Baseline	After 1 month	After 2 months	
BMI (kg/m^2)	28.55 (2.85)	27.72 (3.05)	27.50 (3.18)	$p<0.001$
Waist circumference (cm)	88.64 (8.86)	87.71 (9.33)	85.63 (12.92)	$p<0.05$
Percentage of body fat	34.99 (4.42)	33.94 (4.24)	33.13 (4.34)	$p<0.001$
Arm circumference (cm)	30.76 (2.83)	30.43 (2.75)	29.61 (3.53)	$p<0.001$
Subscapular skinfolds (mm)	16.41 (6.94)	17.71 (6.35)	17.48 (6.28)	n.s.
Triceps skinfolds (mm)	19.84 (3.93)	19.79 (4.41)	19.74 (4.05)	n.s.

Results are expressed as mean values (±SD) [40]
n.s. not significant

Table 16.5 KIDMED test to assess the Mediterranean diet quality during childhood and adolescence

	Nutritional education	
Items on the KIDMED test	Baseline students (%)	Post-intervention students (%)
Takes a fruit or fruit juice every day	70.0	87.6
Has a second fruit every day	33.2	51.6
Has fresh or cooked vegetables regularly once a day	60.8	75.6
Has fresh or cooked vegetables more than once a day	12.9	31.3
Consumes fish regularly (≥2–3 times per week)	59.9	69.1
Goes at least once a week to a fast-food restaurant	25.4	13.4
Likes pulses and eats them more than once a week	71.4	76.0
Consumes pasta or rice almost every day (≥5 times/week)	77.9	87.1
Has cereals or grains (bread, etc.) for breakfast	52.5	67.3
Consumes nuts regularly (at least 2–3 times/week)	45.2	45.2
Uses olive oil at home	92.6	94.0
Skips breakfast	20.7	9.2
Has a dairy product for breakfast (yoghurt, milk, etc.)	73.3	84.8
Has commercially baked goods or pastries for breakfast	25.3	15.7
Takes two yoghurts and/or some cheese (40 g) daily	40.6	56.2
Takes sweets and candy several times every day	25.8	14.7

Results at baseline and following the nutritional intervention [40]
KIDMED Mediterranean Diet Quality Index in children and adolescents

Table 16.6 KIDMED test to assess the Mediterranean diet quality during childhood and adolescence

	Nutritional education	
Items on the KIDMED test	Baseline students (%)	Post-intervention students (%)
Takes a fruit or fruit juice every day	65.8	94.7
Has a second fruit every day	34.2	68.4
Has fresh or cooked vegetables regularly once a day	63.2	92.1
Has fresh or cooked vegetables more than once a day	13.2	42.1
Consumes fish regularly (≥2–3 times/week)	63.2	76.3
Goes at least once a week to a fast-food restaurant	28.9	10.5
Likes pulses and eats them more than once a week	73.7	78.9
Consumes pasta or rice almost every day (≥5 times/week)	84.2	92.1
Has cereals or grains (bread, etc.) for breakfast	31.6	65.8
Consumes nuts regularly (at least 2–3 times/week)	26.3	26.3
Uses olive oil at home	89.5	89.5
Skips breakfast	34.2	2.6
Has a dairy product for breakfast (yoghurt, milk, etc.)	60.5	94.7
Has commercially baked goods or pastries for breakfast	15.8	5.3
Takes two yoghurts and/or some cheese (40 g) daily	34.2	47.4
Takes sweets and candy several times every day	21.1	2.6

Results at baseline and following the nutritional intervention in children with overweight or obesity [40]
KIDMED Mediterranean Diet Quality Index in children and adolescents

or obesity were not going regularly to fast-food restaurants after the program (Table 16.6).

As previously mentioned, breakfast is the most important meal of the day, particularly in children and adolescents. At the end of the study, 90.8 % of the total sample of adolescents that completed the questionnaires had breakfast daily, and only one student of the overweight or obesity subsample continued without having breakfast (2.6 %). Prior to the nutritional educational program, less than half of the sample (Table 16.5) and only one-third of overweight or obese students (Table 16.6) were taking any type of cereal-based foods for breakfast. After the intervention

14.8 % more of the overall sample and 34.2 % more (more than double) of overweight or obese students had incorporated these foods to their breakfasts. Similarly, the percentage of overall students and of students with overweight and obesity taking dairy products for breakfast increased by 11.5 % and 34.2 %, respectively, while there was a marked decrease in the intake of commercially baked goods or pastries for breakfast as observed in Tables 16.5 and 16.6.

Following the intervention, 47.4 % of students with overweight or obesity participating in the intensive nutritional program showed an improvement in the quality of the diet and none of them worsened ($p < 0.001$) it, as shown with the KIDMED index.

Conclusions

Albeit the limited sample size, the fact the nutritional intervention was limited to a single secondary school and the limited follow-up period the results in the present study show a significant change in the quality of the diet following the nutritional education. Indeed, these positive results, both in the overall sample and in the subsample of students with overweight or obesity, indicate that nutritional education aiming at acquiring healthy dietary habits typical of the Mediterranean diet can be effectively applied to adolescent populations. Despite its limitations, the study supports the feasibility of these programs targeting adolescent populations aiming to adopt healthier nutritional and lifestyles. However, it appears necessary that such prevention and educational programs have to be much longer in duration, so that they would have to cover primary and secondary education, to ensure solid, firm results [4], particularly considering the increasing rates of childhood and adolescent obesity in most countries [6, 14, 28, 40]. Among the students in our sample the intake of fruits and vegetables prior to initiating the program was not good, only 5.09 % combined carbohydrates, with a proteic food, a vegetable and a fruit at lunch, and 3 % at dinner. After the program, there was a significant increase in regular use of fruits, vegetables, and other healthy foods. Importantly, active participation of the students was promoted throughout the study, partly to facilitate resolving any doubts that may arise.

Solid evidence supports the beneficial effects of child obesity prevention programs and the specific importance of nutritional education [2, 6, 40]. To maximize efficiency it is important to reinforce multidisciplinary prevention programs addressing both nutritional elements and lifestyles changes, including physical and leisure activities, both at home and in the school [3, 25, 42]. These prevention initiatives need to be appropriately planned and developed to the needs of the targeted population. It is equally important to find the adequate motivations for behavior and attitude changes, as motivation is one of the key elements in learning [43]. Childhood and adolescence are key priority educational moments to generate long-term beliefs, attitudes and values related to overall health and particularly towards nutrition [22].

References

1. Contento IR. Nutrition education: linking research, theory, and practice. Asia Pac J Clin Nutr. 2008;17 Suppl 1:176–9.
2. Olivares S, Snel J, McGrann M, Glasauer P. Nutrition education in primary schools. Food Nutr Agric. 1998. http://www.fao.org/docrep/X0051T/X0051t00.htm#TopOfPage. Accessed 1 Dec 2011.
3. Contento IR. Nutrition education: linking research, theory, and practice. Sudbury, MA: Jones & Bartlett; 2007.
4. Perez-Rodrigo C, Aranceta J. School-based nutrition education: lessons learned and new perspectives. Public Health Nutr. 2001;4:131–9.
5. Chopra M, Sarah Galbraith S, Darnton-Hill I. A global response to a global problem: the epidemic of overnutrition. Bull World Health Organ. 2002;80:952–8.
6. Avenell A, Broom J, Brown TJ, et al. Systematic review of the long-term effects and economic consequences of treatments for obesity and implications for health improvement. Health Technol Assess. 2004;8:1–182.
7. Birketvedt GS, Thom E, Bernersen B, Florhomen J. Combination of diet, exercise and intermittent treatment of cimetidine on body weight and maintenance of weight loss. A 42 months follow-up study. Med Sci Monit. 2000;6:699–703.
8. World Health Organization. The global burden of disease: 2004 update. Geneva: World Health Organization; 2008.

9. Burgess A, Glasauer P. Family nutrition guide. Rome: Food and agriculture Organization of the United Nations; 2004.
10. Waters E, de Silva-Sanigorski A, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev. 2011;12:CD001871.
11. Raj M, Kumar RK. Obesity in children & adolescents. Indian J Med Res. 2010;132:598–607.
12. Kosti RI, Panagiotakos DB. The epidemic of obesity in children and adolescents in the world. Cent Eur J Public Health. 2006;14:151–9.
13. Cornette R. The emotional impact of obesity on children. Worldviews Evid Based Nurs. 2008;5:136–41.
14. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. Int J Obes (Lond). 2011;35:891–8.
15. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med. 1997;337:869–73.
16. Fonseca V, Sichieri R, Da Veiga GV. Factors associated with obesity among adolescents. Rev Saude Publica. 1998;32:541–9.
17. Moreno LA, González-Gross M, Kersting M, et al. Assessing, understanding and modifying nutritional status, eating habits and physical activity in European adolescents: the HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence) Study. Public Health Nutr. 2008;11:288–99.
18. Sweeting HN. Measurement and definitions of obesity in childhood and adolescence: a field guide for the uninitiated. Nutr J. 2007;6:32.
19. Lehingue Y. The European Childhood Obesity Group (ECOG) Project: the European collaborative study on the prevalence of obesity in children. Am J Clin Nutr. 1999;70(1):166–8.
20. Poskitt EME. Body mass index and child obesity: are we nearing a definition? Acta Paediatr. 2000;89:507–9.
21. Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. Arch Dis Child. 1995;73:25–9.
22. Zoppi G, Bressan F. [Obesity in paediatrics: analysis of some definitions and determination of their limits on Italian standards]. Riv Ital Pediatr. 1990;16:139–43.
23. Serra-Majem LL, Aranceta-Bartrina J, Perez-Rodrigo C, Ribas Barba L, Delgado A. Prevalence and determinants of obesity in Spanish children and young people. Br J Nutr. 2006;96 Suppl 1:S67–72.
24. Aranceta Bartrina J, Serra-Majem LL, Foz Sala M, Moreno Esteban B, Grupo Colaborativo SEEDO. Prevalencia de la obesidad en España. Med Clin (Barc). 2005;125:460–6.
25. Aranceta J, Perez-Rodrigo C, Serra-Majem LL, et al. Prevention of overweight and obesity: a Spanish approach. Public Health Nutr. 2007;10:1187–93.
26. Serra-Majem LL, Ribas Barba L, Aranceta Bartrina J, Pérez Rodrigo C, Saavedra Santana P, Peña Quintana L. [Childhood and adolescents obesity in Spain. Results of the enKid study (1998–2000)]. Med Clin. 2003;121:725–32.
27. Darnton-Hill I, Nishida C, James WPT. A life course approach to diet, nutrition and the prevention of chronic diseases. Public Health Nutr. 2004;7(1A):101.
28. Moreno LA, Rodriguez G. Dietary risk factors for development of childhood obesity. Curr Opin Clin Nutr Metab Care. 2007;10:336–41.
29. http://www.naos.aesan.msps.es/naos/ficheros/estrategia/NAOS_Strategy.pdf. Accessed 1 Dec 2011.
30. Moreno B, Charro A, editors. Nutrición, Actividad Física y Prevención de la Obesidad. Estrategia NAOS. Ministerio de Sanidad y Consumo. Madrid: Editorial Médica Panamericana; 2007.
31. Martínez MI, Hernández MD, Ojeda M, Mena R, Alegre A, Alfonso JL. Desarrollo de un programa de educación nutricional y valoración del cambio de hábitos alimentarios saludables en una población de estudiantes de Enseñnza Secundaria Obligatoria. Nutr Hosp. 2009;24:504–10.
32. Serra-Majem L, Ribas L, Ngo J, et al. Food, youth and the Mediterranean diet in Spain. Development of KIDMED, Mediterranean Diet Quality Index in children and adolescents. Public Health Nutr. 2004;7:931–5.
33. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ. 2000;320:1240–3.
34. Maffeis C. Aetiology of overweight and obesity in children and adolescents. Eur J Pediatr. 2000;159 Suppl 1:S35–44.
35. Levine JA, Eberhardt NL, Jensen MD. Role of non-exercise activity thermogenesis in resistance to fat gain in humans. Science. 1999;283:212–4.
36. Giovannini M, Agostoni C, Shamir R. Symposium overview: do we all eat breakfast and is it important? Crit Rev Food Sci Nutr. 2010;50:97–9.
37. Hoyland A, Dye L, Lawton CL. A systematic review of the effect of breakfast on the cognitive performance of children and adolescents. Nutr Res Rev. 2009;22:220–43.
38. Schlundt DG, Hill JO, Sbrocco T, Pope Cordle J, Sharp T. The role of breakfast in the treatment of obesity: a randomized clinical trial. Am J Clin Nutr. 1992;55:645–51.
39. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. Am J Clin Nutr. 1994;59:307–16.
40. Ottevaere C, Huybrechts I, Benser J, et al. Clustering patterns of physical activity, sedentary and dietary behavior among European adolescents: the HELENA study. BMC Public Health. 2011;11:328.
41. World Health Organization. [Organización Mundial de la salud]. Salud para todos en el siglo XXI. Madrid: Ministerio de Salud y Consumo; 1999.
42. Serra Majem L, Ribas Barba L, Pérez Rodrigo C, et al. Methodological limitations in measuring childhood adolescent in epidemiological studies: does overweight fare better than obesity? Public Health Nutr. 2007;10:1112–20.
43. Maslow AH. Motivación y personalidad. Madrid: Ed Díaz de Santos; 1991.

17 Diet Quality and Older Adults: Special Considerations

Alice H. Lichtenstein

Key Points

- Dietary guidance for older adults should be aimed at maintaining optimal health and forestalling the onset of chronic diseases.
- Specific dietary recommendations, for the most part, are consistent with those associated with optimal health outcomes throughout adulthood.
- Evidence suggests that in older adults there is a direct relationship between the diet quality and health outcomes.
- With advancing age there is a decrease in energy requirements to maintain a healthy body weight, yet nutrient requirements remain either unchanged or increase.
- Emphasis should be placed on choosing nutrient dense foods within each food category.
- Current data suggests that older adults who report using nutrient supplements have diet and lifestyle characteristics that are more closely associated with lower rather than higher risk for nutrient insufficiency.
- Special attention should be given to adapting living environments for older adults to enable them to retain the ability to acquire and prepare familiar foods that are appealing.
- Changes in social situations that could impact on food intake should be monitored on a regular basis and adjustments made, when necessary.

Keywords

Diet quality • Physical activity • MyPlate for Older Adults • MyPyramid for Older Adults • Nutrient density

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Abbreviation

RDA Recommended Dietary Allowances

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Introduction

The demographic shift occurring in both developed and developing countries is towards an older population. For example, the US population over the age of 65 years grew from 3.1 million in 1900 to 35.0 million in 2000. During that same time period individuals aged 85 years and older increased by 34-fold. In 2010, approximately 14 % of the US population was over 65 years of age, with the first individuals of the Baby Booth generation having turned 65 years old in 2012. The US Census Bureau has estimated that the current percentage of individuals 65 years of age and older will double between 2000 and 2030. At that rate, by 2030, 70 million Americans will be over the age of 65 years. This is equivalent to one out of every five Americans. Similar trends are seen in other countries. As an increasing proportion of the world population enters the older age categories, more emphasis needs to be placed on optimal diet guidance so as to enable these older individuals to stay healthy and active. This emphasis will need to happen within the context of the biological and psychological changes known to occur with advancing years.

Nutrient Recommendations for Older Adults

Evidence suggests that within a population, older adults who score in the higher categories for diet quality [1–4] and physical activity [5, 6] measures have the highest survival rates. In recognition of these and other findings, the Food and Nutrition Board of the Institute of Medicine placed renewed emphasis on the unique needs of older adults when the Recommended Dietary Allowances (RDA) was revamped in 1990s. A major change from prior RDA's was an expansion of the age categories for which individual recommendations were made. This approach was a joint effort between the United States and Canada. Prior iterations of the RDA's included a single category for individuals above the age of 50 years [7]. Current guidelines include recommendations for adults aged 51–70 years and greater than 70 years (Table 17.1) [8–15]. Of interest is whether a distinction will be made among adults greater than 70 years of age as new data emerge in the future. Nevertheless, general recommendations can be made for older adults that will likely benefit them as they advance in years.

Energy requirements decline with advancing age which makes it increasingly challenging to achieve optimal nutrient intakes while maintaining a healthy body weight. The decline in the total energy requirements is attributed to multiple factors, including a gradual reduction in physical activity and a shift in body composition from lean to fat even in the absence of a change in body weight. This latter factor, in turn, leads to a reduction in basal metabolic rate [16]. Within the context of reduced energy needs nutrient requirements either remain the same or increase. For example, the RDA for vitamin D has recently been increased for all individuals and is higher for individuals >70 years than those below 70 years [17, 18]. Likewise, the RDA for vitamin B_6 and calcium is higher for individuals >70 years than those below 70 years [9]. In contrast, the RDA for sodium is lower for older adults, making it more challenging to comply within our current food environment.

Unique Diet-Related Considerations for Older Adults

Gradual physical and psychosocial changes occur with advancing years, but the rate at which these occur for each individual is highly variable. In addition, the onset of chronic disease can limit food choices. Older adults can also become increasingly susceptible to the lures of food fads or nutrient supplement claims, particularly those that promise the fountain of youth. These claims can drain scarce resources from food budgets and potentially lead to the overconsumption of individual nutrients. Nutrient overconsumption may interfere with the actions or utilization of prescription and nonprescription drugs, and the utilization of essential nutrients.

Table 17.1 Recommended Dietary Allowances for older adults age 31–50 years, 51–70 years, and greater than 70 years

Nutrient	Females (years)			Males (years)		
	31–50	51–70	>70	31–50	51–70	>70
Vitamin A (μg/day)[a]	700	700	700	900	900	900
Vitamin C (mg/day)	75	75	75	90	90	90
Vitamin D (μg/day)	15	15	20	15	15	20
Vitamin E (mg/day)	15	15	15	15	15	15
Vitamin K (μg/day)[b]	*90*	*90*	*90*	*120*	*120*	*120*
Thiamin (mg/day)	1.1	1.1	1.1	1.2	1.2	1.2
Riboflavin (mg/day)	1.1	1.1	1.1	1.3	1.3	1.3
Niacin (mg/day)	14	14	14	16	16	16
Vitamin B_6 (mg/day)	1.3	1.5	1.5	1.3	1.7	1.7
Folate (μg/day)	400	400	400	400	400	400
Vitamin B_{12} (μg/day)	2.4	2.4	2.4	2.4	2.4	2.4
Pantothenic acid (mg/day)	*5*	*5*	*5*	*5*	*5*	*5*
Biotin (μg/day)	*30*	*30*	*30*	*30*	*30*	*30*
Choline (mg/day)	*425*	*425*	*425*	*550*	*550*	*550*
Calcium (mg/day)	1,000	1,200	1,200	1,000	1,000	1,200
Chromium (μg/day)	*25*	*20*	*20*	*35*	*30*	*30*
Copper (μg/day)	900	900	900	900	900	900
Fluoride (mg/day)	*3*	*3*	*3*	*4*	*4*	*4*
Iodine (μg/day)	150	150	150	150	150	150
Iron (mg/day)	18	8	8	8	8	8
Magnesium (mg/day)	320	320	320	420	420	420
Manganese (mg/day)	*1.8*	*1.8*	*1.8*	*2.3*	*2.3*	*2.3*
Molybdenum (μg/day)	45	45	45	45	45	45
Phosphorus (mg/day)	700	700	700	700	700	700
Selenium (μg/day)	55	55	55	55	55	55
Zinc (mg/day)	8	8	8	11	11	11
Potassium (g/day)	*4.7*	*4.7*	*4.7*	*4.7*	*4.7*	*4.7*
Sodium (g/day)	*1.5*	*1.3*	*1.2*	*1.5*	*1.3*	*1.2*
Chloride (g/day)	*2.3*	*2.0*	*1.8*	*2.3*	*2.0*	*1.8*

[a]Standard font—Recommended Dietary Allowance values
[b]Italic font—adequate intake values

Energy Requirements

As noted previously, although the energy levels required to maintain a healthy body weight decrease with advancing years, nutrient requirements either remain constant or increase (Table 17.2). Successfully fulfilling nutrient requirements within the context of reduced energy intakes can be challenging, and can only be accomplished by ensuring that the nutrient density of the diet (nutrients per calorie) be maintained at a relatively high level. For some individuals, this may be easy to accomplish, for example, by switching from iceberg lettuce to romaine lettuce, or increasing the relative proportion of vegetables and fruits in the diet compared to traditional snack foods. For other individuals, necessary changes may mean reversing prior lifelong trends, for example, by seeking out low lactose forms of dairy products to replace regular forms or boasting fiber intake by shifting from refined to whole grain breads and cereals. Any change should be accomplished within the context of established eating patterns and food availability so that the enjoyment of food and promotion of long term changes is maintained.

Table 17.2 Factors that can contribute to compromised nutrient intake in older adults

Factor	Change
Energy needs	↓ Energy requirements due to ↓ physical activity and ↑ fat to lean muscle mass
Dexterity and strength	↑ Arthritic involvement finger and hand joints
	↑ Tremor
	↓ Manual dexterity
Senses	↓ Acuity (taste, smell)
Mobility and vision	↓ Physical activity
	↓ Respiratory capacity
	↓ Lean muscle mass (strength, physical disability)
	↑ Physical isolation
Oral cavity	↑ Peritoneal disease
	↑ Ill-fitting dentures
	↓ Salivary gland function

Dexterity and Strength

Diminished dexterity can make food preparation difficult, which in turn can have a negative impact on the variety, quality, and enjoyment of the resulting products. Challenging tasks during food preparation include difficulty opening jars, cans, or other types of food and beverage packaging, and pealing and cutting foods due to arthritis or diminished strength. Small accommodations such as ergonomically designed kitchen aides (e.g., can openers and scissors) and kitchen reorganization (e.g., eliminating clutter and reorganizing draws and cabinets to increase accessibility of frequently used items) can minimize the impact of these changes. Also helpful can be the introduction of newer forms of familiar foods, such as partially processed or prepared foods, for example, precut frozen vegetables and fruits, boneless chicken breasts, and prewashed salad greens or grated carrots. Older adults may not automatically take advantage of these newer forms of common food items and require some regular guidance in this area to take advantage of good options.

Taste and Smell

Retaining the desire to eat a variety of foods is integral to ensuring optimal nutrition for older adults. Changes that can occur with aging which can lead to poor appetite include diminished taste acuity primarily resulting from loss of taste buds, particularly those sensing salt and sweet. The impact of these changes on food intake can be exacerbated by recommendations to restrict sodium intake. In addition, these changes can result in greater sensitivity to acid and bitter tastes [19]. Another change that is frequently observed in older adults includes a diminished sense of smell. Older adults with poor odor perception have been reported to have lower nutrient intakes than those with good odor perception [20–22]. Encouraging the liberal use of herbs and spices may enhance the continued enjoyment of favorite items.

Mobility and Vision

Diminished mobility and vision can make it difficult to leave the home, particularly during times of inclement weather. This can limit social interaction and exposure to sunlight, as well as the ability to purchase food due to limited transportation options and types of stores accessible. Potential solutions include identifying alternative forms of transportation, such as taxi-pooling to the market once a week with neighbors, taking advantage of senior shuttles/discounts, and asking for help. Finding solutions to limited mobility and vision is critical to minimizing their potential negative impact on both psychological well-being and food intake.

In addition to addressing transportation-related issues, there are a number of additional strategies than can be gradually adapted to further minimize the effect that diminished mobility and vision has on food intake. These include, for example, shifting the type of foods purchased to those that can be stored for longer periods. Resealable bags of pre-cleaned and cut frozen vegetables and fruits are particularly good choices for older adults because they allow for small quantities to be removed at any one time, eliminate the need to peel and cut, minimize waste due to spoilage of excess quantities, and provide variety during winter months. Newer varieties of low sodium and low sugar canned soups, vegetables and fruits, and shelf-stable milk can minimize the need to go shopping during short periods of inclement weather.

Psychosocial Factors

In addition to dealing with declines in physical capacity associated with the aging process, there are also changes in the social environment of older adults that can have an impact on nutritional status (Table 17.3). With advancing years the loss of a spouse or other family members with whom an individual shared and prepared meals with is common. These changes can lead to social isolation, especially during mealtime, and diminished desire to prepare well-balanced and varied meals. Due to deterioration in mental or economic status older adults are frequently faced with having to adapt to a new living environment, resulting in dramatic changes in meal times, opportunities for food preparation and types of food available. Depression frequently accompanies the aging process in individuals without adequate support and can make the necessary adaptations even more challenging. Older adults are also at increased risk of alcohol abuse. All of these factors have been associated with a deterioration of dietary habits and nutrient intakes [23].

Table 17.3 Psychosocial factors that can contribute to compromised nutrient intake in older adults

Factor	Potential changes
Companionship	↑ Loss of spouse and contemporaries
	↑ Social isolation secondary to ↓ mobility and ↑ loss contemporaries
Mental state	↑ Depression
	↑ Mental deterioration (dementia)
	↑ Alcoholism
Economic	↑ Fixed income
	↓ Choice, variety, and availability secondary to ↓ mobility
Nutrition knowledge	↑ Susceptibility to food fads
Housing	↑ Change in status (loss of home)

Nutrient Supplements and Older Adults

Nutrient supplement use is more common in older than younger adults [24–28]. The primary reasons cited by older adults for taking nutrient supplements are to improve health and delay the onset of chronic disease [29, 30]. This issue is of particular concern because in general, older adults who choose to use nutrient supplements are least likely to have biomarkers of nutrient inadequacy or diets rated as a poor [28, 31]. In light of the widespread availability of fortified foods this group may be particularly vulnerable to excess nutrient intakes and drug-nutrient interactions [25, 27, 32–34]. The latter issue is of particular concern given the limited amount of information available on the topic [35]. General characteristics of individuals using supplements, in addition to being older [24–28], including being female [26–28], non-Hispanic white [27, 28], college educated or beyond [24, 26–28], and affluent [28]. In addition, nutrient supplement users are more likely to have body mass indices within the normal range [24, 27], engage in regular physical activity [24, 27], have optimal chronic disease biomarkers [28], have low rates of smoking [24], have better nutrient intakes, and hold strong attitudes about the importance of a good diet [30, 31, 34].

Unique Nutrition-Related Issues in Older Adults

Nutrient-related chronic diseases particularly prevalent in middle and later years include cardiovascular disease, cancer, Type 2 diabetes, hypertension, osteoporosis, and most recently overweight and obesity. Also of concern are disorders of dentition and associated senses. In some cases, the goal of unique nutrient recommendations for older adults is to accommodate physiological changes associated with aging. In other cases, the goal is to delay the onset of, accommodate, or treat chronic diseases.

Vitamin B_{12}

Many individuals experience a decline in gastric hydrochloric acid secretion (atrophic gastritis) as they increase in age [36]. The resulting hypochlorhydria results in a decrease in the bioavailability of vitamin B_{12}. After age 60, it has been estimated that 10–30 % of individuals will have some degree of atrophic gastritis [37]. The resulting higher gastric pH leads to a decrease in vitamin B_{12} bioavailability. This necessitates higher intakes of vitamin B_{12} to achieve nutrient adequacy. In addition, atrophic gastritis can result in abnormal bacterial colonization of the upper gastrointestinal tract. These bacteria utilize vitamin B_{12} for growth, further compromising vitamin B_{12} status [38].

Vitamin D and Calcium

The requirement for vitamin D and calcium increases with advancing years. This is due to a number of factors. The efficiency of the skin to synthesize vitamin D from cholesterol declines with age [39]. Residing in northern latitudes, reducing outdoor activities and intentionally limiting sun exposure due to skin cancers concerns will compromise endogenous vitamin D synthesis [40]. More recently, overweight and obesity has been linked to lower circulating vitamin D concentrations, likely attributable to a sequestration of the vitamin in adipose tissue [41]. Accompanying these trends, an increase in the incidence of lactose intolerance can result in a decrease in the intake of milk and other dairy products, rich sources of calcium and potentially vitamin D [42–44]. Important to consider is the dependency of calcium absorption on adequate vitamin D status.

Dentition and Associated Senses

Salivary secretions decrease with increasing age. Changes in bite pattern resulting from partial or complete tooth extraction/loss is common [45, 46]. For those who have lost teeth, poorly fitted dentures may result in an avoidance of certain types of foods and limit the variety consumed. For example, chewing and swallowing fibrous foods may become difficult, resulting in a shift towards highly processed foods or juices that are low in fiber [47]. It is critical when evaluating dietary intakes of older adults to consider possible concerns regarding food textures and preparation methods. Extended cooking times and small shifts in preparation techniques, for example, grating rather than slicing vegetables, can remedy some of the problems.

Osteoporosis

Age-related or type II osteoporosis (bone loss) is a concern with advancing years. It has been estimated that osteoporotic fractures affect 50% of females and 30% of males over the age of 50 years [48]. There are a number of factors that are associated with accelerated bone loss in older adults. These include diminished estrogen production, decreased intake and absorption of calcium, decreased renal calcium resorption, diminished rates of physical activity, compromised vitamin D status, and decreased calcitriol production secondary to hyperparathyroidism [48, 49]. In general, calcium balance is favorably affected by vitamin D intake, and negatively affected by high sodium, protein, alcohol, and caffeine intakes [49]. Supplemental calcium and vitamin D in postmenopausal women living in northern latitudes may minimize bone loss [50].

Cardiovascular Disease

The rate of cardiovascular disease increases with age, particularly after menopause in women [51]. The American Heart Association [52], the National Cholesterol Education Program [53], and the 2010 Dietary Guidelines for Americans [54] recommend the lifelong consumption of diets that are rich in vegetables and fruits, whole grains, legumes (beans), fat-free and low-fat dairy products, fish, lean meat, and vegetable oils. This ensures that the diets are low in saturated and *trans* fat, rich in unsaturated fat and nutrient dense. No specific modifications are recommended for older adults. The response to these recommendations in terms of plasma lipids appears consistent across different age groups and both sexes [55].

Hypertension

The incidence of hypertension, particularly isolated systolic hypertension, increases with age [51]. This increase is associated with changes in the vasculature and kidneys, and when present is exacerbated by weight gain. A number of clinical trials have demonstrated clear benefits of dietary modification to treat hypertension in older adults. The Dietary Approaches to Stop Hypertension (DASH) type dietary pattern, rich in vegetables, fruits, and fat-free and low-fat dairy products, decreases blood pressure in a wide range of individuals [56]. Further coupling this dietary pattern with sodium restriction has been shown to further decrease blood pressure [57].

Glucose Intolerance/Type 2 Diabetes

The incidence of glucose intolerance and Type 2 diabetes mellitus increases with advancing age [58, 59]. This trend frequently is associated with an increase in body weight. Lifestyle interventions in most age groups, including older adults, have been shown to be efficacious in preventing or delaying the onset of Type 2 diabetes mellitus [60–62]. These include achieving and maintaining a healthy body weight, engaging in regular physical activity, and consuming a diet with moderate rather than low level of fat.

Cancer

The incidence of cancer shows tremendous variability on the basis of worldwide distribution, type, and site in the body. The incidence of all types of cancer increases with age. Support for a diet/cancer incidence link comes from data suggesting associations between markedly divergent food consumption patterns and incidence rates of cancer among different population groups [63]. Some data has been reported for associations involving the following dietary components: alcohol intake (laryngeal), calcium and vitamin D intake (stomach, colon, breast), fat intake (breast, colon, prostate), fiber intake (breast), antioxidant vitamin and/or orange and dark green vegetable intake—vitamin A and beta-carotene, vitamin C, vitamin E and trace elements (wide range of sites) [63–67]. However, results from randomized controlled trials in the area of cancer and diet are limited [68]. Frequently the data between observational and interventional studies are inconsistent and few recommendations are made for diet and cancer risk reduction with the exception of achieving and maintaining a healthy body weight, engaging in regular physical activity, and eating a diet rich in vegetables and fruits [69]. Vitamin A supplementation in individuals at high risk for lung cancer has been associated with increased incidence [70, 71]. Concerns have also been raised regarding high intake levels of folate and vitamin B_{12}, and increased colon and prostate cancer risk [72–74]. At this time the general dietary guidance to reduce cancer risk is consistent with the dietary guidance to prevent the onset of chronic diseases of concern in the twenty-first century [69].

Dietary Guidance for Older Adults

Food-Based Dietary Recommendations

From an applied perspective, nutrient-based guidelines for older adults can be viewed on the basis of food-based recommendations. The nutrient goals remain unchanged; however, the approach towards making the recommendations has shifted from individual nutrients to foods. In general, the unique considerations of food-based dietary recommendations for older adults include issues related to diminishing total energy needs, maintaining nutrient density, increasing incidence of lactose intolerance, ensuring adequacy of fluid intake, and reducing chronic disease risk.

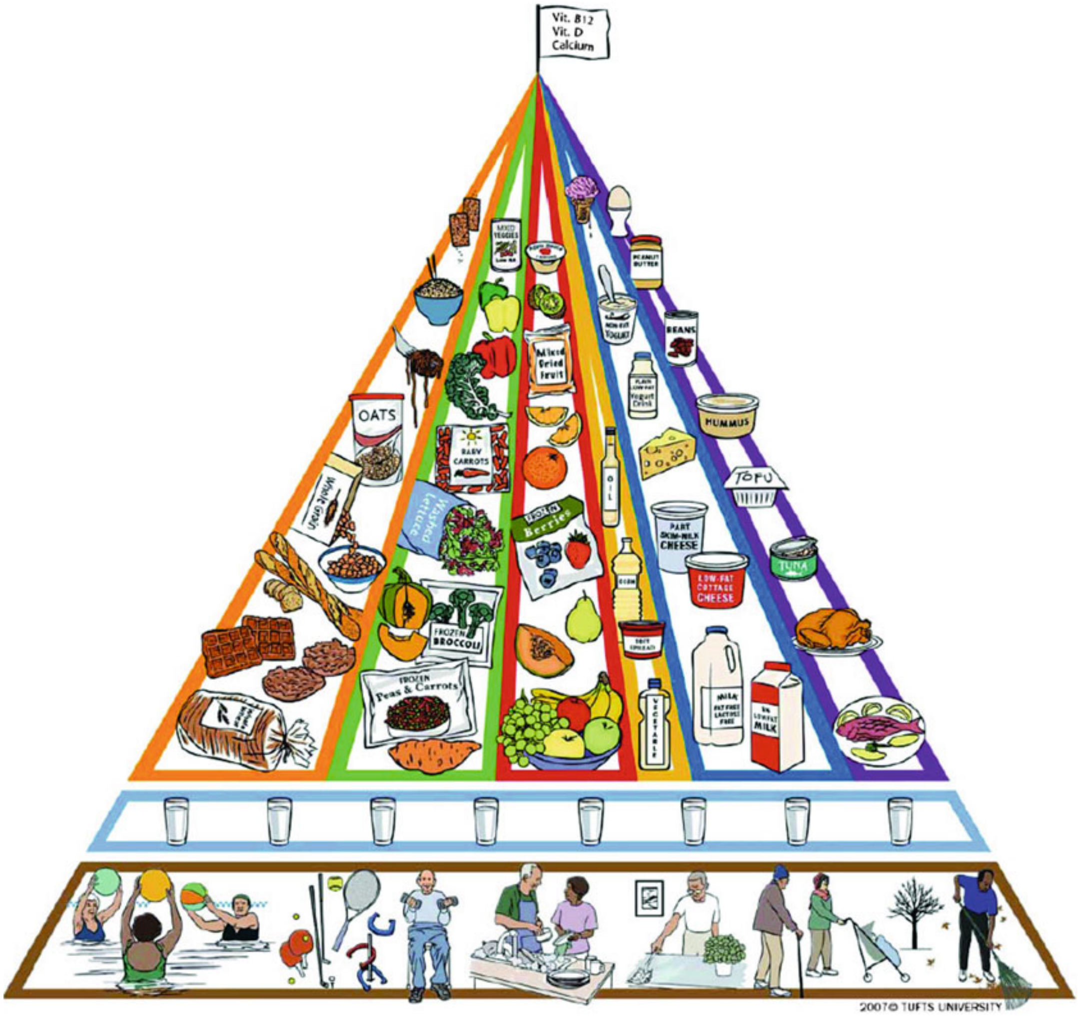

Fig. 17.1 *MyPyramid* for Older Adults, http://nutrition.tufts.edu/docs/pdf/releases/ModifiedMyPyramid.pdf

Two tools are available to guide older adults in adapting their food intake to match the changing nutrient requirements associated with advancing years. Important to note, food-based recommendations intended for older adults are, for the most part, likewise appropriate for younger adults as well. One graphic representation of food-based recommendations for older adults is the familiar Food Guide Pyramid as adapted for older adults, *MyPyramid for Older Adults* (Fig. 17.1) [75]. The second is MyPlate, also adapted for older adults, *MyPlate for Older Adults* (Fig. 17.2) [76]. Differences from the original icons incorporated into the versions developed specifically for older adults are aimed at addressing their unique needs and are specific to the two different icons.

MyPyramid for Older Adults

Modifications made to the original version of the Food Guide Pyramid specifically for older adults include the following: narrowing the pyramid base to reflect diminished energy requirements; inserting icons to represent illustrative examples of nutrient dense food choices within each food grouping; illustrating forms of foods particularly useful when adapting to the changing needs and abilities of older adults such as bags of frozen vegetables and fruits, low lactose dairy products

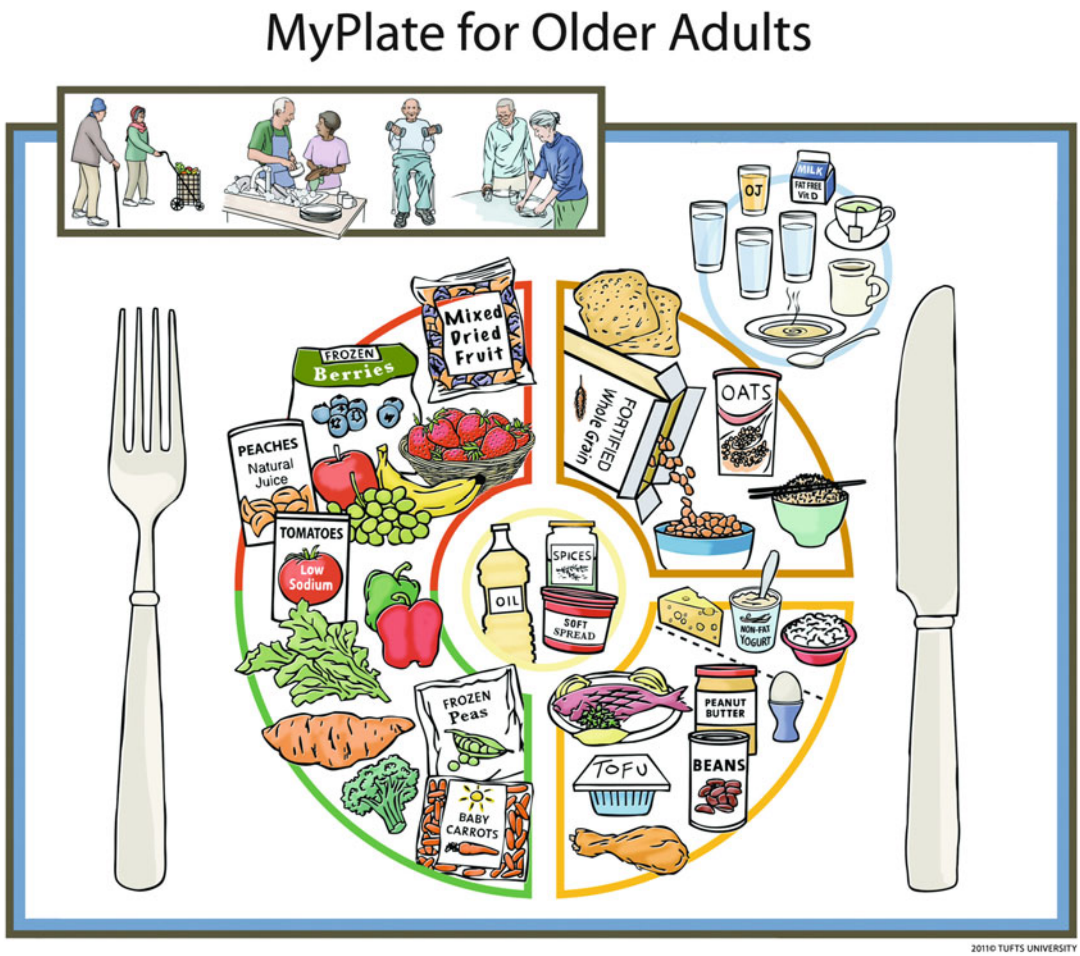

Fig. 17.2 *MyPlate* for Older Adults, http://nutrition.tufts.edu/docs/pdf/releases/ModifiedMyPyramid.pdf

and canned low sodium vegetables and low sugar fruits; and including a band at the base of the pyramid with icons of drinking glasses to emphasize the importance of adequate fluid intakes (Fig. 17.1). Due to the potential disassociation between the sensation of thirst and state of hydration in older adults [77–79], meeting fluid needs particularly for individuals who spend prolonged periods in hot environments requires increased attention. Also depicted at the base of the pyramid is a band with icons of individuals engaging in different forms of activities to illustrate the importance of engaging in daily physical activity regularly. Regarding the food icons, specific emphasis is given to vegetables and fruits with deep-colored interiors that tend to be nutrient dense; whole vegetables and fruits rather than juice and whole grains and cereals products to ensure adequate fiber intakes; low-fat and nonfat dairy products as well as low lactose dairy products to minimize saturated fat intake and promote continued intake of high calcium foods, respectively; sources of protein (fish, legumes, poultry) to minimize saturated fat intake; and liquid vegetable oils and soft spreads that are rich in unsaturated fat.

MyPlate for Older Adults

Modifications made to the original MyPlate specifically for older adults include the addition of food icons to the different sectors of the plate to provide illustrative examples of nutrient dense choices such as deeply colored vegetables and fruits along with whole grain alternatives; shift of the dairy sector into the protein sector; fusion of the vegetable and fruit sectors; creation of a fluid sector on the top right of the plate to emphasize the importance of this category; construction of a physical activity panel in the top left of the plate to likewise emphasize the importance of engaging in regular physical activity; insertion of a sector in the center of the plate containing vegetable oils and herbs-spices to emphasize the benefit across the diet of using vegetable oils for food preparation in place of animal fats and flavorings as an alternative to salt; addition of a knife with the fork to emphasize the importance to focusing on and enjoying food while eating meals rather than using electronic gadgets; and depiction of a broad range of different forms of foods particularly useful to older adults such as bags of frozen fruits, precut and prewashed vegetables, and canned low sodium foods (Fig. 17.2).

Conclusions

The aim of dietary guidance specifically targeted for older adults is to maintain optimal health and forestall the onset of chronic diseases. The actual dietary recommendations, for the most part, are consistent with those associated with optimal health outcomes throughout adulthood. Evidence suggests that there is a direct relationship between the nutrient quality of the diet and survival rates. Due to decreased levels of physical activity, decreased metabolic rates, and increased proportions of fat to lean muscle mass, energy requirements decline with advancing years yet nutrient requirements remain either unchanged or increase. This situation requires a greater emphasis on choosing nutrient dense foods within each food category. Attention also needs to be placed on monitoring nutrient supplement use in older adults to avoid overconsumption. Current data suggest those who are more likely to report using nutrient supplements have dietary and lifestyle characteristics that are more closely associated with lower rather than higher risk for nutrient insufficiency. Special attention needs to be given to adapting living environments for older adults to enable them to retain the ability to acquire and prepare familiar foods that are appealing. Changes in social situations that could impact on food intake should be monitored on a regular basis. There is no data to suggest a person is too old to benefit from improvements in diet quality and physical activity. The definitions for old age and expectations for the period of time individuals can remain active, productive, and independent is expanding. Efforts towards maintaining and when necessary improving diet quality and physical activity patterns with advancing years should keep up with this trend.

References

1. de Groot LC, van Staveren WA, Burema J. Survival beyond age 70 in relation to diet. Nutr Rev. 1996;54: 211–2.
2. Sahyoun NR, Jacques PF, Russell RM. Carotenoids, vitamins C and E, and mortality in an elderly population. Am J Epidemiol. 1996;144:501–11.
3. Houston DK, Johnson MA, Daniel TD, Poon LW. Health and dietary characteristics of supplement users in an elderly population. Int J Vitam Nutr Res. 1997; 67:183–91.
4. Anderson AL, Harris TB, Tylavsky FA, et al. Dietary patterns and survival of older adults. J Am Diet Assoc. 2010;111:84–91.
5. Blain H, Carriere I, Sourial N, et al. Balance and walking speed predict subsequent 8-year mortality independently of current and intermediate events in well-functioning women aged 75 years and older. J Nutr Health Aging. 2010;14:595–600.
6. Studenski S, Perera S, Patel K, et al. Gait speed and survival in older adults. JAMA. 2010;305:50–8.
7. IOM. How should the recommended dietary allowances be revised? Washington, DC: National Academy of Sciences; 1994.
8. IOM. Dietary reference intakes. Calcium, phosphorus, magnesium, vitamin D and fluoride. Washington, DC: National Academy of Sciences; 1997.
9. IOM. Dietary reference intakes. Thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin and choline. Washington, DC: National Academy of Sciences; 1998.
10. IOM. Dietary reference intakes. Vitamin C, vitamin E, selenium and carotenoids. Washington, DC: National Academy of Sciences; 2000.
11. IOM. Dietary reference intakes. Vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium and zinc. Washington, DC: National Academy of Sciences; 2001.

12. IOM. Dietary supplements: a framework for evaluating safety. Committee on the framework for evaluating the safety of dietary supplements. Washington, DC: National Academy of Sciences; 2004.
13. IOM. Dietary reference intakes. Energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. Washington, DC: National Academy of Sciences; 2005.
14. IOM. Dietary reference intakes. Water, potassium, sodium, chloride and sulfate. Washington, DC: National Academy of Sciences; 2005.
15. IOM. Dietary reference intakes. The essential guide to nutrient requirements. Washington, DC: National Academy of Sciences; 2006.
16. Williamson DF. Descriptive epidemiology of body weight and weight change in U.S. adults. Ann Intern Med. 1993;119:646–9.
17. IOM. Dietary reference intakes for calcium and vitamin D. 2010. http://www.iom.edu/Reports/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D.aspx
18. Wolfe RR, Miller SL, Miller KB. Optimal protein intake in the elderly. Clin Nutr. 2008;27:675–84.
19. Lipson LG, Bray GA. Energy intake and utilization in aging man. In: Chen LH, editor. Nutritional aspects of aging, vol. I. Boca Raton, FL: CRC Press; 1986.
20. Griep MI, Collys K, Mets TF, Slop D, Laska M, Massart DL. Sensory detection of food odour in relation to dental status, gender and age. Gerodontology. 1996;13:56–62.
21. Griep MI, Verleye G, Franck AH, Collys K, Mets TF, Massart DL. Variation in nutrient intake with dental status, age and odour perception. Eur J Clin Nutr. 1996;50:816–25.
22. Griep MI, Mets TF, Collys K, Ponjaert-Kristoffersen I, Massart DL. Risk of malnutrition in retirement homes elderly persons measured by the "mini-nutritional assessment". J Gerontol A Biol Sci Med Sci. 2000;55:M57–63.
23. James WP, Nelson M, Ralph A, Leather S. Socioeconomic determinants of health. The contribution of nutrition to inequalities in health. BMJ. 1997;314:1545–9.
24. Foote JA, Murphy SP, Wilkens LR, Hankin JH, Henderson BE, Kolonel LN. Factors associated with dietary supplement use among healthy adults of five ethnicities: the Multiethnic Cohort Study. Am J Epidemiol. 2003;157:888–97.
25. Yoon SL, Schaffer SD. Herbal, prescribed, and over-the-counter drug use in older women: prevalence of drug interactions. Geriatr Nurs. 2006;27:118–29.
26. Gardiner P, Graham R, Legedza ATR, Ahn AC, Eisenberg DM, Phillips RS. Factors associated with herbal therapy use by adults in the United States. Altern Ther Health Med. 2007;13:22–9.
27. Rock CL. Multivitamin-multimineral supplements: who uses them? Am J Clin Nutr. 2007;85:277S–9.
28. Block G, Jensen CD, Norkus EP, et al. Usage patterns, health, and nutritional status of long-term multiple dietary supplement users: a cross-sectional study. Nutr J. 2007;6:30.
29. Buhr G, Bales CW. Nutritional supplements for older adults: review and recommendations—part I. J Nutr Elder. 2009;28:5–29.
30. Buhr G, Bales CW. Nutritional supplements for older adults: review and recommendations—part II. J Nutr Elder. 2010;29:42–71.
31. Sebastian RS, Cleveland LE, Goldman JD, Moshfegh AJ. Older adults who use vitamin/mineral supplements differ from nonusers in nutrient intake adequacy and dietary attitudes. J Am Diet Assoc. 2007;107:1322–32.
32. Radimer K, Bindewald B, Hughes J, Ervin B, Swanson C, Picciano MF. Dietary supplement use by US adults: data from the National Health and Nutrition Examination Survey, 1999–2000. Am J Epidemiol. 2004;160:339–49.
33. Kishiyama SS, Leahy MJ, Zitzelberger TA, et al. Patterns of dietary supplement usage in demographically diverse older people. Altern Ther Health Med. 2006;11:48–53.
34. Murphy SP, White KK, Park SY, Sharma S. Multivitamin-multimineral supplements' effect on total nutrient intake. Am J Clin Nutr. 2007;85:280S–4.
35. Yetley EA. Multivitamin and multimineral dietary supplements: definitions, characterization, bioavailability, and drug interactions. Am J Clin Nutr. 2007;85:269S–76.
36. Byrd D, Russell RM. Malabsorption in an elderly patient. Gastroenterologist. 1993;1:287–90.
37. Hurwitz A, Brady DA, Schaal SE, Samloff IM, Dedon J, Ruhl CE. Gastric acidity in older adults. JAMA. 1997;278:659–62.
38. Suter PM, Golner BB, Goldin BR, Morrow FD, Russell RM. Reversal of protein-bound vitamin B12 malabsorption with antibiotics in atrophic gastritis. Gastroenterology. 1991;101:1039–45.
39. MacLaughlin J, Holick MF. Aging decreases the capacity of human skin to produce vitamin D3. J Clin Invest. 1985;76:1536–8.
40. Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr. 2004;80:1678S–88.
41. Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF. Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr. 2000;72:690–3.
42. Goulding A, Taylor RW, Keil D, Gold E, Lewis-Barned NJ, Williams SM. Lactose malabsorption and rate of bone loss in older women. Age Ageing. 1999;28:175–80.
43. Di Stefano M, Veneto G, Malservisi S, Strocchi A, Corazza GR. Lactose malabsorption and intolerance in the elderly. Scand J Gastroenterol. 2001;36:1274–8.
44. Lovelace HY, Barr SI. Diagnosis, symptoms, and calcium intakes of individuals with self-reported lactose intolerance. J Am Coll Nutr. 2005;24:51–7.

45. Papas AS, Joshi A, Giunta JL, Palmer CA. Relationships among education, dentate status, and diet in adults. Spec Care Dentist. 1998;18:26–32.
46. Papas AS, Palmer CA, Rounds MC, Russell RM. The effects of denture status on nutrition. Spec Care Dentist. 1998;18:17–25.
47. NIDDK. NIH Publication No. 07-2754 July 2007 National digestive diseases information clearinghouse. 2007. http://digestive.niddk.nih.gov/ddiseases/pubs/constipation/
48. Prince RL. Diet and the prevention of osteoporotic fractures. N Engl J Med. 1997;337:701–2.
49. Faine MP. Dietary factors related to preservation of oral and skeletal bone mass in women. J Prosthet Dent. 1995;73:65–72.
50. Chung M, Balk E, Bendel M, et al. The relationships of vitamin D and calcium intakes to nutrient status indicators and health outcomes. Evidence report AHRQ Publication No. Rockville, MD: Agency for Healthcare Research and Quality; 2009.
51. AHA website. www.americanheart.org/presenter.jhtml?identifier=200011
52. Lichtenstein AH, Appel LJ, Brands M, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. Circulation. 2006;114:82–96.
53. Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive summary of the third report of the National Cholesterol Education Program (NCEP). JAMA. 2001;285:2486–97.
54. Dietary Guidelines for Americans. 2010. http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm
55. Lapointe A, Balk EM, Lichtenstein AH. Gender differences in plasma lipid response to dietary fat. Nutr Rev. 2006;64:234–49.
56. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336:1117–24.
57. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med. 2001;344:3–10.
58. Ford ES, Ajani UA, Croft JB, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. N Engl J Med. 2007;356:2388–98.
59. Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH. Prevalence of the metabolic syndrome among U.S. adolescents using the definition from the International Diabetes Federation. Diabetes Care. 2008;31:587–9.
60. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med. 2001;344:1343–50.
61. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002;346:393–403.
62. Sakane N, Sato J, Tsushita K, et al. Prevention of type 2 diabetes in a primary healthcare setting: three-year results of lifestyle intervention in Japanese subjects with impaired glucose tolerance. BMC Public Health. 2011;11:40–9.
63. Serra-Majem L, La Vecchia C, Ribas-Barba L, et al. Changes in diet and mortality from selected cancers in southern Mediterranean countries, 1960–1989. Eur J Clin Nutr. 1993;47 Suppl 1:S25–34.
64. Johnson K, Kligman EW. Preventive nutrition: disease-specific dietary interventions for older adults. Geriatrics. 1992;47:39–40.
65. Bostick RM, Potter JD, McKenzie DR, et al. Reduced risk of colon cancer with high intake of vitamin E: the Iowa Women's Health Study. Cancer Res. 1993;53:4230–7.
66. Bostick RM, Potter JD, Sellers TA, McKenzie DR, Kushi LH, Folsom AR. Relation of calcium, vitamin D, and dairy food intake to incidence of colon cancer among older women. The Iowa Women's Health Study. Am J Epidemiol. 1993;137:1302–17.
67. Jevtic M, Velicki R, Popovic M, Cemerlic-Adjic N, Babovic SS, Velicki L. Dietary influence on breast cancer. J BUON. 2010;15:455–61.
68. Kushi LH, Byers T, Doyle C, et al. American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. CA Cancer J Clin. 2006;56:254–81.
69. Kushi LH, Doyle C, McCullough ML, et al. American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. CA Cancer J Clin. 2012;62:30–67.
70. Albanes D, Heinonen OP, Taylor PR, et al. Alpha-tocopherol and beta-carotene supplements and lung cancer incidence in the alpha-tocopherol, beta-carotene cancer prevention study: effects of base-line characteristics and study compliance. J Natl Cancer Inst. 1996;88:1560–70.
71. Omenn GS, Goodman GE, Thornquist MD, et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. N Engl J Med. 1996;334:1150–5.
72. Lucock M, Yates Z. Folic acid fortification: a double-edged sword. Curr Opin Clin Nutr Metab Care. 2009;12:555–64.
73. Collin SM, Metcalfe C, Refsum H, et al. Associations of folate, vitamin B12, homocysteine, and folate-pathway polymorphisms with prostate-specific antigen velocity in men with localized prostate cancer. Cancer Epidemiol Biomarkers Prev. 2010;19:2833–8.
74. Kennedy DA, Stern SJ, Moretti M, et al. Folate intake and the risk of colorectal cancer: a systematic review and meta-analysis. Cancer Epidemiol. 2011;35:2–10.
75. Lichtenstein AH, Rasmussen H, Yu WW, Epstein SR, Russell RM. Modified MyPyramid for older adults. [Erratum appears in J Nutr. 2008 Jul;138(7):1400]. J Nutr. 2008;138:5–11.

76. MyPlate. http://www.nutrition.tufts.edu/research/myplate-older-adults
77. Phillips PA, Johnston CI, Gray L. Disturbed fluid and electrolyte homoeostasis following dehydration in elderly people. Age Ageing. 1993;22:S26–33.
78. Phillips PA, Rolls BJ, Ledingham JG, et al. Reduced thirst after water deprivation in healthy elderly men. N Engl J Med. 1984;311:753–9.
79. Ferry M. Strategies for ensuring good hydration in the elderly. Nutr Rev. 2005;63:S22–9.

Part IV

Foods and Dietary Components

18 Mediterranean Diet and Dietary Sodium Intake

Paul Farajian and Antonis Zampelas

Key Points

- Limiting sodium intake is crucial for the treatment of high blood pressure in children and adults.
- The average estimated salt intake in most countries around the world is higher than the recommended, a trend also seen in childhood populations.
- Sodium is a mineral found naturally in foods but is also used greatly in processed foods. In westernized diets, an estimated 80 % of total sodium intake comes from processed or restaurant-prepared foods.
- Adherence to the Mediterranean diet besides providing many undoubted health benefits is also associated with a lower risk of elevated blood pressure levels.
- Recent studies reveal that children and adult populations of Mediterranean countries have elevated sodium intake from "hidden" sources which are mainly processed foods.
- Public health initiatives, in association with the food industry, are urgently needed to lower salt consumption, by decreasing sodium concentration of foods and especially those that are consumed regularly.

Keywords

Mediterranean diet • Salt • Food processing • Hypertension • Dietary indices • Mediterranean Diet Quality Index • Food industry

Abbreviations

BMI	Body mass index
BP	Blood pressure
CVD	Cardiovascular disease
DASH	Dietary approaches to stop hypertension
DBP	Diastolic blood pressure

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GRECO study	Greek Childhood Obesity study
KIDMED	Mediterranean Diet Quality Index for children and adolescents
MD	Mediterranean diet
NHANES	National Health and Nutrition Examination Surveys
SBP	Systolic blood pressure
WHO	World Health Organization

Table 18.1 Definition and classification of hypertension in children and adolescents

Class	SBP and/or DBP percentile
Normal	<90th
High-normal	≥90th to <95th ≥120/80 even if below 90th percentile in adolescents
Stage 1 hypertension	95th percentile to the 99th percentile plus 5 mmHg
Stage 2 hypertension	>99th percentile plus 5 mmHg

Adapted from ref. [11]

Introduction

Hypertension is a global burden which has been associated with renal and cardiovascular disease (CVD), the latter being the leading cause of death in developed countries. Worldwide, it is estimated that nearly one billion adults have hypertension which is a major cause of CVD, responsible for 62 % of stroke and 49 % of coronary heart disease [1–3]. While few would dispute the importance of taking effective steps to assess and manage this condition in adults, relatively little attention has been paid to the problem of high blood pressure (BP) in children. It is now established, that high BP is detectable in children and adolescents, but is relatively common, and is increasing in prevalence [4, 5]. It has been demonstrated that childhood hypertension tracks into adulthood, and children with systolic blood pressure (SBP) that meets the definition of high BP at any age have higher the odds of developing hypertension in adulthood by 3–4-fold compared with children whose SBP is normal [4]. Furthermore, the negative and potentially severe consequences of high BP are not only limited to adulthood, but also during the early ages [4]. Evidence of end-organ damage, such as left ventricular hypertrophy, pathological vascular changes, and possible renal dysfunctions have been found even in young children with high BP [5]. Even central nervous system end-organ damage, which manifest as reduced cognitive function and poorer performance on selected tests of cognition, have also been detected among children with high BP, compared with normotensive children [6, 7]. However, what is not known is whether interventions to reduce high BP during childhood would prevent or delay the progression to hypertension in adulthood [8]. Estimates for the prevalence of high BP among children and adolescents are in the range of 3–5 %, with much higher rates reported in certain populations and subgroups [4]. Nevertheless, despite the variations in age, sample size, and definition across the different studies, the reported prevalence of high BP among children and adolescents describes an important health issue. In the United States, mean BP values in children have increased in recent years. Data from the National Health and Nutrition Examination Surveys (NHANES) show that mean systolic BP (SBP) and diastolic BP (DBP) increased by 1.4/3.3 mmHg from 1988–1994 to 1999–2000 [9].

According to the position paper of the American Society of Hypertension regarding the dietary approaches to lower BP [10], the report from the National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents for the diagnosis, evaluation, and treatment of high BP in children and adolescents [8], and the recommendations of the European Society of Hypertension for the management of high BP in children and adolescents [11], therapeutic lifestyle changes are recommended as an initial treatment strategy for adults with abnormal BP values, and children or adolescents with BP greater than the 90th percentile for age, sex, and height (Table 18.1). These lifestyle modifications include regular physical activity, avoiding excess weight gain, limiting dietary sodium intake, and increasing the consumption of fresh fruits, fresh vegetables, fiber, and low-fat dairy products.

Dietary Sodium Intake and High Blood Pressure in Adults and Children

Sodium is a mineral found naturally in many foods but is also used greatly in processed foods. It is the mineral that has been, mainly, linked to hypertension and CVD, and most often studied and assessed in relation to BP. Besides to its effects on BP, excess dietary sodium consumption has been associated directly with coronary heart disease, stroke, and stomach cancer [12– 14]. Animal experiments, epidemiological studies, and clinical trials provide compelling evidence for a detrimental effect of sodium intake on BP among both hypertensive and normotensive individuals. The INTERSALT study demonstrated a significant positive relationship between salt intake and BP, while there was also a positive relationship between salt intake and the increase in BP with age [15]. Moreover, the INTERMAP (the international study of macro- and micronutrients and BP) epidemiological study, and the Norfolk Cohort of the European Prospective Investigation into Cancer, have provided further support for the important role of salt intake in determining the levels of population BP [16, 17]. A meta-analysis of randomized trials of 1 month or longer, demonstrated that a modest reduction in salt intake caused significant and important falls in BP in both hypertensive and normotensive individuals, while there was a dose response to salt reduction [18]. Finally, the Dietary Approaches to Stop Hypertension (DASH)-Sodium Trial was undertaken to study the effect of the DASH diet (diet high in fruits, vegetables, low-fat dairy products, and low in red meat and refined carbohydrates) on the BP among normotensive and hypertensive individuals who consumed varying levels of sodium. It was found that in a short period of time the DASH diet lowered BP at all levels of sodium consumption, although these effects were further enhanced with sodium restriction [19]. The effect of multiple dietary factors that have been suggested in the literature to lower or increase BP, besides sodium, are summarized in Table 18.2. Some of the evidence regarding the effects of the dietary factors is strong, while other is less certain.

Table 18.2 A summary of the evidence on the effects of dietary factors and dietary patterns on blood pressure

	Hypothesized effect	Evidence
Weight	Direct	++
Sodium chloride (salt)	Direct	++
Potassium	Inverse	++
Magnesium	Inverse	±
Calcium	Inverse	±
Alcohol	Direct	++
Fat		
Saturated fat	Direct	±
ω-3 Polyunsaturated fat	Inverse	++
ω-6 Polyunsaturated fat	Inverse	±
Monounsaturated fat	Inverse	+
Protein		
Total protein	Uncertain	+
Vegetable protein	Inverse	+
Animal protein	Uncertain	±
Carbohydrate	Uncertain	+
Fiber	Inverse	+
Cholesterol	Direct	±
Dietary patterns		
Vegetarian diets	Inverse	++
DASH-type dietary patterns	Inverse	++

DASH dietary approaches to stop hypertension
Key to evidence: ±, limited or equivocal evidence; +, suggestive evidence typically from observational studies and some clinical trials; ++, persuasive evidence typically from clinical trials (adapted from ref. [10] with permission of Elsevier)

The importance of sodium intake in determining the BP levels in children and adolescents is also shown in many observational studies. Although there are studies that did not show a significant association [20] the observational studies that were methodologically stronger (i.e., multiple measurements of salt intake were made, urinary sodium was measured, and confounding factors were controlled), showed in most cases a significant positive association between salt intake and BP [21]. In the meta-analysis of He and MacGregor [21] of ten salt reduction trials with 966 participants for an average duration of 4 weeks, it was demonstrated that a modest reduction in salt intake had a significant effect on BP in children and adolescents. A 42 % reduction in salt intake reduced systolic BP by 1.2 mmHg and diastolic by 1.3 mmHg. In three trials with infants (median duration 20 weeks) it was also shown that a salt reduction by 54 % reduced the systolic pressure by 2.5 mmHg. According to the authors, these results in conjunction with other evidence provide strong support for the necessity of reduction of salt (and subsequently sodium) in children [21].

Sodium Intakes and Main Food Sources

In the light of the previous data many countries have developed their own guidelines on dietary salt intake. The United Kingdom and US guidelines recommend salt intake of less than 6 g/day (2.4 g/day of sodium) for adults. The World Health Organization (WHO) set a worldwide target of a maximum intake of 5 g/day (2.0 g/day of sodium). However, the average estimated salt intake in most countries around the world is approximately 9–12 g/day, with many Asian countries having mean intakes more than 12 g/day. In children older than 5 years, salt intake is commonly more than 6 g/day, and increases with age [22]. In European and Northern American diets, an estimated 75–80 % of sodium intake comes from processed or restaurant-prepared foods, 10–12 % occurs naturally in foods; and a similar proportion is from optional use of table salt [22]. According to the UK National Food Survey data collected in 2000, cereal products (including bread, other baked goods, and breakfast cereals) accounted for the greatest proportion (38 %) of household sodium intake. The second largest source (21 %) was meat products (including processed meats such as ham, bacon, etc.) [23]. Data from the USA show a similar pattern: cereals and baked goods providing >16 % sodium and meat products >13 %. A different picture with regard to dietary sources of sodium is noticeable for some Asian countries. In China and Japan, the greatest proportion of dietary sodium comes from sodium added during cooking and sauces (e.g., soy sauce) [22]. In children and young people fewer data are available on sodium intake than in adults, and these are mainly limited to the developed nations of Europe and North America. The National Diet and Nutrition Survey in young people in Great Britain which was carried out in 1997 and measured salt intake using a 7-day dietary record in 856 boys and 845 girls, demonstrated that the average salt intake, at the age of 4–6 years, was 5.2 g/day for boys and 4.6 g/day for girls. With increasing age, there was an increase in salt intake, and by the age of 15–18 years, salt intake was 8.2 and 5.7 g/day for boys and girls, respectively [24]. In France, the main contributors to sodium and potassium intakes were essentially the same in adults and children. Breads, soups, cooked pork meats, convenience foods, pastries, and sugary products were the major sources of sodium whereas vegetables, dairy products, and fruits were the major providers of potassium, similarly to what has been described in other industrialized populations [25]. Detailed presentation of the available data concerning the sodium intakes of children and adolescents are presented in the review of Brown et al. [22].

According to estimations, salt intake in children in developed countries has increased even more than the data presented in the aforementioned review, due to the increasing consumption of processed foods. Surveys in the United States showed that the proportion of foods that children consumed from restaurants and fast-food restaurants increased by nearly 300 % between 1977 and 1996, and it is very likely to have increased even further in more recent years [21]. Snack food consumption showed a similar trend. The processed, restaurant, fast foods, and snacks are generally very high in salt, fat, and sugar, therefore it is possible that children nowadays consume as much salt as adults [26].

Hidden Sources of Sodium in the Modern Diets

In a recent study of Magriplis et al. [27], under the context of the GRECO (Greek Childhood Obesity) study, the daily dietary sodium intake (excluding table salt and salt added during cooking) of 10–12 years old Greek children, was studied, within the context of the Mediterranean

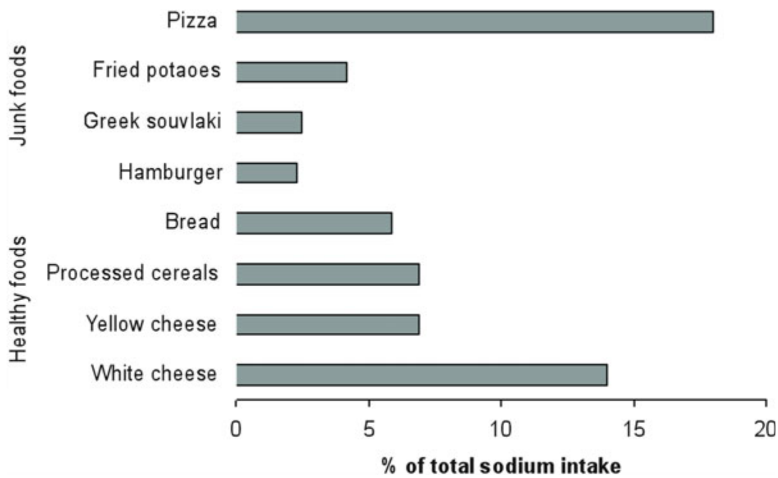

Fig. 18.1 Sodium intake from healthy or "junk" foods as percent of total consumption other than table salt and salt added during cooking, from the GRECO study in children. Adapted from ref. [27] with permission of Wolters Kluwer Health

diet (MD) pattern. Four thousand five hundred and eighty children aged 10–12 years were enrolled, in a cross-sectional, population-based survey. Among other measurements, dietary data were obtained by a semiquantitative food frequency questionnaire, and sodium intake was calculated. High sodium consumption was considered an intake over 2,200 mg/day. Adherence to the Mediterranean dietary pattern was evaluated using the Mediterranean Diet Quality Index for children and adolescent score (KIDMED score) [28]. The KIDMED index was initially developed in an attempt to combine the MD guidelines (e.g., daily fruit and vegetable consumption, weekly fish and legumes intake, olive oil is recommended for culinary use, daily consumption of grains and cereals is recommended during breakfast, pasta and rice should be consumed at least 5 times per week) for adults, as well as the general dietary guidelines for children (such as breakfast skipping) in a single index. It was based on the principles sustaining the Mediterranean dietary pattern as well as on those that undermine it (such as frequent consumption of fast-food and increased intake of sweets). The index comprises 16 yes or no questions. Questions denoting a negative connotation with respect to the MD were assigned a value of −1 and those with a positive aspect +1. The total score ranged from −4 to 12 and was classified into three levels: >8, optimal MD; 4–7, improvement needed to adjust intake to the MD; and <3, very low diet quality.

The results of the study showed that 23 % of the total sample had high sodium intake (>2,200 mg/day), without taking into account added salt at the table or while cooking. In addition, a further 20.9 % of the children had a moderate sodium intake (between 1,500 and 2,200 mg/day), but only via foods, again without taking into account the salt added at the table and the salt added during cooking, implying that a significant and alarming proportion of the children consumes sodium above the guidelines, which makes it an important public health issue in Greece. The major sodium sources were pizza, hamburgers, fried potatoes, souvlaki (traditional meat product), white and yellow cheese, processed cereals, and bread (Fig. 18.1). Another important finding was that children closer to the MD reported a higher dietary sodium intake. In particular, children that reported moderate and high adherence to the MD (i.e., KIDMED score >4) had higher sodium intake from the majority of the food groups ($p<0.001$), by the exception of pizza, hamburgers, souvlaki (a type of meat), saltines (including crisps, crackers, cheese sticks), and cakes ($p>0.05$). In addition, the level of adherence to the MD was associated with sodium intake from various food sources. Analyses revealed that 1 U increase in the KIDMED score was associated with 4–50 % (i.e., ORs varied from 1.04 to 1.50, all *p*-values <0.05) increased likelihood of consuming sodium intake above the median value for the majority of foods. These results were confirmed even after adjusting for age, sex, body mass index (BMI), and physical activity. Moreover, 1 U increase in KIDMED score was associated with 10 % increase in likelihood of consuming total sodium >1,500 mg/day (which is the EU upper level).

The strong association between the level of adherence to MD and sodium intake may seem controversial after taking into consideration that adherence to a Mediterranean food pattern has been shown to be associated with substantial reductions in total mortality and CVD mortality in adults [29–31]. In addition, studies have also found an inverse association between hypertension incidence, as well as BP levels in individuals following the MD. A possible explanation of the high sodium intake of children with moderate and high MD adherence is that 34 % of total sodium intake was found to be consumed by those foods known as "healthy" (i.e., bread, processed cereal, and white cheese), compared with 18 % that was observed from pizza. These foods which are recommended to be consumed on a daily basis according to the MD scheme are the everyday hidden sources of sodium, due to sodium addition during manufacturing, and seem to add substantially to the total dietary sodium of an otherwise healthy dietary pattern.

The Association of the Mediterranean Diet with Blood Pressure Levels in Adults and Children

Since the first results of the Seven Countries Study establishing that the traditional Mediterranean dietary pattern, as followed in Crete, was associated with lower ischemic heart disease [32], the term Mediterranean diet (MD) has been widely used to describe the traditional dietary habits followed by the populations in Crete, Southern Italy, Spain, and other countries of the Mediterranean region, during the decade of the 1960s [33, 34]. The MD has been associated with longevity [35] and has been promoted for its numerous health benefits, since there are many intervention and epidemiological studies suggesting that MD is protective against several morbid conditions including atherosclerosis, coronary heart disease, diabetes, metabolic syndrome, and inflammation [31, 36–38].

The traditional MD is characterized by abundant plant foods: fruits, vegetables, bread, and other forms of cereals (pasta, brown rice, bulgur, etc.), legumes, nuts, and seeds. It also includes olive oil as the principal source of added lipid (probably the most characteristic component of the MD is olive oil), moderate amounts of dairy products (mainly cheese and yogurt), low to moderate amounts of fish and poultry, low amounts of red meat, and wine consumed in low to moderate quantities (Fig. 18.2). The Mediterranean dietary pattern is characterized as rich in monounsaturated fatty acids, high monounsaturated to saturated fat ratio, balanced ratio of (n-6):(n-3) essential fatty acids, and high amounts of fiber as well as antioxidants, such as vitamins E and C, carotenoids, polyphenols (coming mainly from olive oil), and selenium [33, 39].

Although information regarding the sodium intake of Mediterranean populations (both adults and children) is very limited, the inverse relation of MD adherence and arterial BP levels has been shown in recent epidemiological studies. In a cross-sectional study in Greece, it was reported that adherence to a MD increases the likelihood of having the arterial BP controlled. Specifically, consumption of a MD was associated with a 26 % lower risk of being hypertensive, and with a 36 % greater probability of having the BP controlled in adults [40]. According to the authors, the protective influence is not caused by single nutrients, such as dietary fatty acids, potassium, or dietary fiber, but can be

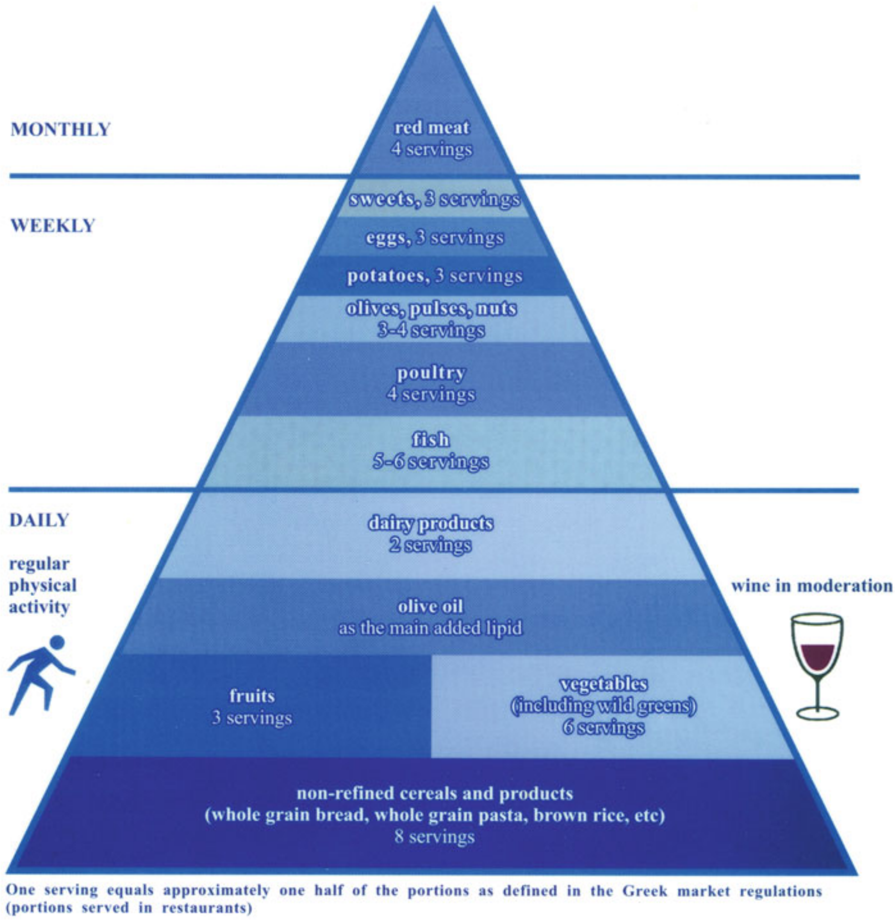

Fig. 18.2 The Mediterranean diet pyramid. *Source*: Higher supreme scientific health council, Ministry of Health and Welfare, Greece

attributed to the MD as a whole. Furthermore, in the Greek arm of the European Prospective Investigation into Cancer and Nutrition (EPIC) study, it was investigated whether the MD, as an entity, and olive oil in particular, can reduce BP [38]. According to the results of the study, the MD score was inversely associated with both systolic and DBP, after controlling for socio-demographic and anthropometric variables as well as for energy intake and energy expenditure. Additionally, it was shown that olive oil, vegetables, and fruits are the principal factors that explain the overall effect of the MD on arterial BP. Nevertheless, it was noted that cereals (including bread), a nutritional factor generally considered to be beneficial to health, were positively associated with both systolic and DBP, as was the high intake of ethanol, and the consumption of meat and meat products. The consumption of fish and seafood was inversely associated with systolic BP. Although sodium intake was not assessed in this study, the fact that one of the beneficial components of the MD, cereal consumption, was actually positively associated with both systolic and DBP was attributed to the salt that is frequently added to cereal products, particularly bread, which is a widely consumed in Greece.

Concerning the association between the adherence to the MD and BP levels in children, in the study of Lazarou et al. [41] the relationship between diet quality, as assessed by the Foods E-KINDEX, and BP levels was assessed. The index includes 13 components that assess consumption frequency of 11 major food groups or foods (i.e., bread, cereals and grains, fruit and fruit juices, vegetables, legumes, milk, fish and seafood, meat [excluding delicatessen and processed meat], salted and smoked meat food, sweets and snacks, and soft drinks), as well as two cooking techniques (fried and grilled foods). Its score ranges between 0 and 37. A subsample of 622 Cypriot children from the CYKIDS national cross-sectional study was used. The Foods E-KINDEX score was found to be independently associated with lower BP among healthy children. Compared with children with a low diet score, those with at least an average foods E-KINDEX score were 57 % less likely to have elevated systolic BP levels, regardless of various potential confounders, and children who scored below mean in the Foods E-KINDEX (i.e., poor diet quality) were 2.3 times more likely to have elevated SBP. However, as the authors pointed out, when the foods E-KINDEX was replaced by its individual components, no significant associations were observed between any of the individual foods and BP levels, suggesting that dietary patterns represent a stronger regulating factor of BP levels than individual foods and nutrients.

These results are in line with previous evidence from a study using a sample of children from the Framingham prospective study, where investigators found that children who consumed four or more servings of fruit and vegetables per day plus two or more servings of dairy products had smaller yearly gains in systolic and DBP throughout childhood, even after accounting for differences in BMI. Those with higher intakes of fruits and vegetables alone or dairy alone had intermediate levels of adolescent systolic BP [42].

The mechanism by which the MD favors the reduction of BP levels is basically unclear. It could be however postulated that of all the components of the MD, vegetables, fruits, and olive oil (high ratio of monounsaturated to saturated lipids), are mainly responsible for the apparent protection against hypertension conveyed by the adherence to the MD. The high content of plant foods and dairy products in minerals such as potassium, calcium, and magnesium, which have been referred in the literature to reduce arterial BP, may represent the mediating mechanisms of the apparent protective effects of these foods. Furthermore, the high content of the dietary pattern in fruits and vegetables and olive oil, together with the moderate consumption of red wine (resveratrol as the main antioxidant) result to a daily high antioxidants intake which may also contribute to the health of the vascular system [38]. In particular, the naturally occurring antioxidants present in olive oil such as tocopherols, and phenolic compounds have been suggested to inactivate the effects of free radicals and lipid peroxidation, which could affect arterial stiffness [43, 44]. Moreover, the recommended low intake of red meat and meat products, and the relatively high intake of fish and seafood seems to enhance the BP lowering effect of the MD [45–47] (Table 18.2).

The fact that healthy dietary patterns can represent a stronger predictive factor of BP levels among adults and children than the intake of individual foods and nutrients have also been reported from the DASH study, demonstrating the BP lowering benefits in adults with high BP of a diet that was high in fresh fruits, vegetables, whole grains, and low-fat dairy products [19]. The study of Couch et al. [48] provided data to support that children with high BP may benefit from diets that emphasize fruits, vegetables, fiber, and dairy, plus a reduction in sodium. The investigators compared a DASH diet for children vs. standard nutrition counseling in children with hypertension or prehypertension. There was a significantly greater reduction in SBP in children assigned to the DASH diet compared with standard diet counseling, and BP was normalized in 50 % of children in the DASH group compared with 36 % in the control group. The similarities of the DASH diet and the MD have been pointed out since they both represent healthy dietary patterns that could be used to tackle the increasing prevalence of adult and childhood hypertension. The main difference between the two diets is that the MD is high in olive oil [38].

Regulating Factors of BP Levels Besides Sodium Intake: The Role of Healthy Dietary Patterns

Improving diet quality and increasing physical activity and weight loss (if overweight and obesity are present) are cornerstones for both the prevention and the non-pharmacologic treatment of hypertension in adults as well as children. Regarding the effects of the dietary habits and intakes on the BP levels, many epidemiological studies that focus on the relationship between diet and risk of chronic disease, examined the intake of a single nutrient, food, or food group. However, this approach does not consider the complexity of dietary behaviors, as food and nutrients are not eaten in isolation. To address this issue, investigators are now including indexes of dietary quality, patterns, and variety in their research [49]. Indices assessing the adherence of adults and children to the MD, have demonstrated the inverse relationship shown between the adherence to the MD dietary pattern and BP [38, 40, 41]. Nevertheless, the study of Magriplis et al. [27], under the context of the GRECO study, revealed that 23 % of the total sample of children had high sodium intake (>2,200 mg/day), without taking into account added salt at the table or while cooking, and showed a strong association between the level of adherence to the MD and sodium intake. A possible explanation of the high sodium intake of children with moderate and high MD adherence was that 34 % of total sodium intake was found to be consumed by those foods known as "healthy" (i.e., bread, processed cereal, and white cheese). However, although it would then be expected that high adherent to the MD children would also have higher BP levels, this was not the case in the representative sample of Greek children since there were no differences in the KIDMED score (index used to evaluated the adherence to the MD for children and adolescent) between the prehypertensive or the hypertensive children compared with the normotensive children (unpublished data from the GRECO study). The former data imply that although the MD adherence is shown to be accompanied by high intakes of sodium, still provides protection against high BP levels probably through the high intakes of potassium mainly from the increased consumption of fruits and vegetables.

Conclusions

The health benefit of decreasing dietary sodium intake in adults and children is beyond question, since it has been demonstrated that a reduction in salt intake has a significant lowering effect on BP. If we take into account that dietary habits adopted during childhood track into adulthood, and the hypothesis that if highly salted foods are consumed on a regular basis, the salt taste receptors are suppressed and habituation to salty foods occurs, with greater demand for highly salted foods [12], the need to reduce sodium intake in children becomes even more critical. Given that most of the sodium in westernized diets is hidden in processed foods or restaurant-prepared foods [22], individuals are often unaware of the amount of sodium they consume. In European and Northern American diets, it is estimated that 75–80 % of sodium intake comes from processed or restaurant-prepared foods [22], therefore a significant decrease in sodium intake cannot be made without avoiding or reducing the amount consumed of these foods, or without a reduction of the amount of salt added to these foods by the food manufacturers, especially in regularly consumed foods like breads and cereals. In addition, implementing effective food labeling practices would also facilitate consumers exercise informed choice with respect to sodium, and even track their everyday intake [50]. Public health initiatives, in association with efforts by the food industry, are urgently needed to lower salt consumption and consequently lower CVD burden. Such public health approaches can be relatively simple, low cost and effective [12].

References

1. Geleijnse JM, Grobbee DE, Kok FJ. Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. J Hum Hypertens. 2005;19: S1–4.
2. Kearney PM, Whelton M, Reynolds K, Muntener P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. Lancet. 2005;365: 217–23.
3. Lewington S, Clarke R, Qizilbash N, et al. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet. 2002;360:1903–13.
4. Falkner B, Lurbe E, Schaefer F. High blood pressure in children: clinical and health policy implications. J Clin Hypertens. 2010;12:261–7.
5. Feber J, Ahmed M. Hypertension in children: new trends and challenges. Clin Sci. 2010;119:151–61.
6. Drukteinis JS, Roman MJ, Fabsitz RR, et al. Cardiac and systemic hemodynamic characteristics of hypertension and prehypertension in adolescents and young adults: the Strong Heart Study. Circulation. 2007;115: 221–7.
7. Lande MB, Carson NL, Roy J, et al. Effects of childhood primary hypertension on carotid intima media thickness: a matched controlled study. Hypertension. 2006;48:40–4.
8. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004;114: 555–76.
9. Muntner P, He J, Cutler JA, et al. Trends in blood pressure among children and adolescents. JAMA. 2004;291:2107–13.
10. Appel LJ. ASH position paper: dietary approaches to lower blood pressure. J Am Soc Hypertens. 2009;3:321–31.
11. Lurbe E, Cifkova R, Cruickshank JK, Dillon MJ, Ferreira I, Invitti C, et al. Management of high blood pressure in children and adolescents: recommendations of the European Society of Hypertension. J Hypertens. 2009;27:1719–42.
12. He FJ, MacGregor GA. Reducing sodium intake worldwide: from evidence to implementation. Prog Cardiovasc Dis. 2010;52:363–82.
13. Nagata C, Takatsuka N, Shimizu N, et al. Sodium intake and risk of death from stroke in Japanese men and women. Stroke. 2004;35:1543–7.
14. Perry IJ, Beevers DG. Salt intake and stroke: a possible direct effect. J Hum Hypertens. 1992;6:23–5.
15. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. BMJ. 1988;297:319–28.
16. Zhou BF, Stamler J, Dennis B, et al. Nutrient intakes of middleaged men and women in China, Japan, United Kingdom, and United States in the late 1990s: the INTERMAP study. J Hum Hypertens. 2003;17: 623–30.
17. Khaw KT, Bingham S, Welch A, et al. Blood pressure and urinary sodium in men and women: the Norfolk Cohort of the European Prospective Investigation into Cancer (EPIC-Norfolk). Am J Clin Nutr. 2004;80: 1397–403.
18. He FJ, MacGregor GA. Effect of modest salt reduction on blood pressure: a meta-analysis of randomized trials. Implications for public health. J Hum Hypertens. 2002;16:761–70.
19. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med. 2001;344:3–10.
20. Simons-Morton DG, Obarzanek E. Diet and blood pressure in children and adolescents. Pediatr Nephrol. 1997;11:244–9.
21. He FJ, MacGregor GA. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. Hypertension. 2006;48:861–9.
22. Brown IJ, Tzoulaki I, Candeias V, et al. Salt intakes around the world: implications for public health. Int J Epidemiol. 2009;38:791–813.
23. Scientific Advisory Committee on Nutrition. Salt and health. London: HMSO; 2003.
24. Gregory J, Lowe S, Bates CJ, Prentice A, Jackson LV, Smithers G, et al. National diet and nutrition survey: young people aged 4–18 years. vol. 1. Report of the Diet and Nutrition Survey. London: TSO; 2000.
25. Meneton P, Lafay L, Tard A, Dufour A, Ireland J, Menard J, et al. Dietary sources and correlates of sodium and potassium intakes in the French general population. Eur J Clin Nutr. 2009;63:1169–75.
26. St-Onge MP, Keller KL, Heymsfield SB. Changes in childhood food consumption patterns: a cause for concern in light of increasing body weights. Am J Clin Nutr. 2003;78:1068–107.
27. Magriplis E, Farajian P, Pounis GD, Risvas G, Panagiotakos DB, Zampelas A. High sodium intake of children through 'hidden' food sources and its association with the Mediterranean diet: the GRECO study. J Hypertens. 2011;29:1069–76.
28. Serra-Majem L, Ribas L, Ngo J, Ortega R, Garcia A, Perez C, et al. Food, youth and the Mediterranean Diet in Spain. Development of KIDMED, Mediterranean Diet Quality Index in children and adolescents. Public Health Nutr. 2004;7:931–5.
29. Sánchez-Taínta A, Estruch R, Bulló M, Corella D, Gómez-Gracia E, Fiol M, et al. Adherence to a Mediterranean-type diet and reduced prevalence of clustered cardiovascular risk factors in a cohort of 3,204 high-risk patients. Eur J Cardiovasc Prev Rehabil. 2008;15:589–93.
30. Pérez-López FR, Chedraui P, Haya J, Cuadros JL. Effects of the Mediterranean diet on longevity and age-related morbid conditions. Maturitas. 2009;64: 67–79.

31. Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, Ruiz-Gutiérrez V, Covas MI, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. Ann Intern Med. 2006;145:1–11.
32. Keys A. Coronary heart disease in seven countries. Circulation. 1970;41–42:1–211.
33. Kafatos A, Verhagen H, Moschandreas J, Apostolaki I, VanWesterop JJ. Mediterranean diet of Crete: foods and nutrient content. J Am Diet Assoc. 2000;100:1487–93.
34. Simopoulos AP. The Mediterranean diets: what is so special about the diet of Greece? The scientific evidence. J Nutr. 2001;131 Suppl 11:S3065–73.
35. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348: 2599–608.
36. Esposito K, Ciotola M, Giugliano D. Mediterranean diet, endothelial function and vascular inflammatory markers. Public Health Nutr. 2006;9:1073–6.
37. Panagiotakos DB, Tzima N, Pitsavos C, Chrysohoou C, Zampelas A, Toussoulis D, et al. The association between adherence to the Mediterranean diet and fasting indices of glucose homoeostasis: the ATTICA study. J Am Coll Nutr. 2007;26:32–8.
38. Psaltopoulou T, Naska A, Orfanos P, Trichopoulos D, Mountokalakis T, Trichopoulou A. Olive oil, the Mediterranean diet, and arterial blood pressure: the Greek European Prospective Investigation into Cancer and Nutrition (EPIC) study. Am J Clin Nutr. 2004;80:1012–8.
39. Willett WC, Sacks F, Trichopoulou A, Drescher G, Ferro-Luzzi A, Helsing E, et al. Mediterranean diet pyramid: a cultural model for healthy eating. Am J Clin Nutr. 1995;61:S1402–6.
40. Panagiotakos DB, Pitsavos CH, Chrysohoou C, et al. Status and management of hypertension in Greece: role of the adoption of a Mediterranean diet: the Attica study. J Hypertens. 2003;21:1483–9.
41. Lazarou C, Panagiotakos DB, Matalas A. Foods E-KINDEX: a dietary index associated with reduced blood pressure levels among young children: the CYKIDS study. J Am Diet Assoc. 2009;109:1070–5.
42. Moore LL, Singer MR, Bradlee ML, Djoussé L, Proctor MH, Cupples LA, et al. Intake of fruits, vegetables, and dairy products in early childhood and subsequent blood pressure change. Epidemiology. 2005;16:4–11.
43. Chernichow S, Blacher J, Hercberg S. Antioxidants vitamins and blood pressure. Curr Hypertens Rep. 2004;6:27–30.
44. Visioli F, Galli C. Biological properties of olive oil phytochemicals. Crit Rev Food Sci Nutr. 2002;42: 209–21.
45. Thadhani R, Camargo Jr CA, Stampfer MJ, Curhan GC, Willett WC, Rimm EB. Prospective study of moderate alcohol consumption and risk of hypertension in young women. Arch Intern Med. 2002;162:569–74.
46. Appleby P, Davey G, Key T. Hypertension and blood pressure among meat eaters, fish eaters, vegetarians and vegans in EPIC-Oxford. Public Health Nutr. 2002;5:645–54.
47. Engler MM, Engler MB, Pierson DM, Molteni LB, Molteni A. Effects of docosahexaenoic acid on vascular pathology and reactivity in hypertension. Exp Biol Med. 2003;228:299–307.
48. Couch SC, Saelens BE, Levin L, et al. The efficacy of a clinic-based behavioral nutrition intervention emphasizing a DASH-type diet for adolescents with elevated blood pressure. J Pediatr. 2008;152:494–501.
49. Wirt A, Collins CA. Diet quality-what is it and does it matter. Public Health Nutr. 2009;12:2473–92.
50. Reddy KS, Katan MB. Diet, nutrition and the prevention of hypertension and cardiovascular diseases. Public Health Nutr. 2004;7:167–86.

Nutritional Quality of Foods: Sweet Potato

19

Hoang Van Chuyen and Jong-Bang Eun

Key Points

- The sweet potato (*Ipomoea batatas*) has been known for thousands of years as one of the most common carbohydrate foods.
- Many studies have reported that sweet potato is not only a rich source of starch but some varieties also contain remarkable amounts of dietary fiber, phenolic compounds, high quality proteins, and other bioactive nutrients.
- Among various types of sweet potato, orange-fleshed and purple-fleshed ones have been proven as the richest sources of β-carotene and anthocyanins, respectively.
- The nutrients in sweet potato have been demonstrated to have many important biological functions such as provitamin A, antioxidant, anti-mutagenicity, antidiabetic activities, and antihypertensive effect.
- The results suggest that sweet potato might bring many benefits to human health other than as a carbohydrate food.

Keywords

Anthocyanins • β-Carotene • Carbohydrate • Phenolic compounds • Sporamins • Sweet potato

Introduction

The sweet potato (*Ipomoea batatas*) belongs to the family Convolvulaceae, which is grown to obtain starchy tuberous root as a carbohydrate supply. Although the name "sweet potato" makes us look back on the common potato (*Solanum tuberosum*), it is not closely related to this plant.

Sweet potato is thought to be originated and domesticated in Central America and South America at least 5,000 years ago [1]. Gradually, it

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Table 19.1 Production of sweet potato of major producers in 2009 (in million tons)

China	Nigeria	Uganda	Indonesia	Vietnam	Tanzania	India	Japan	World
80.5	3.3	2.7	1.88	1.32	1.31	1.1	1.0	106.5

Source: FAO statistics 2009 [3]

had spread widely to the tropical, subtropical, and even temperate areas all over the world. Sweet potato can grow well in many farming conditions and variety of soils (even in poor soil) with low requirements of fertilizer and pesticide. By this reason, it becomes a staple food crop in some tropical regions in Africa and Asia, where productions of wheat, rice, or other crops are often disadvantaged due to climatic restraints. It was reported that sweet potatoes are the seventh most commonly consumed carbohydrate-rich food source, and they are one of the most important food crops in developing countries among rice, wheat, maize, and cassava [2].

According to the Food and Agriculture Organization (FAO) statistics, annual world production of sweet potato is more than 100 million tons. China is the largest grower of sweet potatoes with about 80 % of the world's supply. Some other Asian countries, such as Indonesia, Vietnam, and India, also produce considerable yield of sweet potato. In Uganda and Rwanda, sweet potato plays an important role in the diet of population, and they are also the biggest producers of this crop in Africa. The contributions of some major countries in world production of sweet potato in 2009 are provided in Table 19.1 [3].

Sweet potato is commonly used as food in the form of fresh root, which is boiled or steamed before consuming. Nevertheless, the fresh root is highly perishable so it is processed into dried slices and flour for extensive storage and application in food products. Recently, sweet potato flour is more considered because it can be applied in many kinds of foods as a thickener (in soup, gravy, fabricated snack, and bakery products), an enhancer of color and flavor, and a supplement of nutrients [4].

Not only as a rich source of carbohydrate, sweet potato also contains considerable amounts of functional nutrients including dietary fiber, β-carotene, phenolic compounds, anthocyanins, vitamin C, and high quality proteins (sporamins) [5]. Among various types of sweet potato with skin color ranging between purple, red, brown, and beige and flesh ranging from beige through yellow, orange, and purple, the orange- and purple-fleshed ones are the most considered for their high content of functional nutrients. While the orange-fleshed sweet potato exhibits as a potential source of β-carotene (the precursor of vitamin A) [6], the purple-fleshed one was reported to be rich in anthocyanins and phenolic compounds [7]. Due to this special nutrient composition, sweet potato has shown many important biological functions such as provitamin A, antioxidative, antimutagenicity activities, and antihypertensive effect [8, 9].

Consequently, the discussion about diet quality of sweet potato in this chapter mainly focuses on the functional nutrients with their biological activities and benefits for human being other than on the major component, carbohydrate.

Diet Quality of Sweet Potato

Sweet Potato: A Carbohydrate-Rich Source

As mentioned in the previous part, sweet potato ranks as the seventh most commonly consumed carbohydrate-rich food source, and plays an important role in carbohydrate supply in some areas having climatic restraints. It was reported that carbohydrates constitute up to 90 % of dry matter of sweet potato [10]. The major carbohydrate in sweet potato is starch, which is composed of 60–70 % amylopectin and 30–40 % amylose. Besides starch, free sugars also present in fresh roots with the concentration ranging between 4.3 and 14.9 % dry weight depending on varieties. The major sugar in raw roots is sucrose while maltose, the product of starch conversion, is the dominant one in cooked roots [11].

Sweet Potato and Beneficial Effects on Blood Sugar

Although sweet potato is classified as a carbohydrate-supplied food, several studies have demonstrated that this tuber root has ability to improve blood sugar regulation and the related disease, type 2 diabetes.

Remarkable antidiabetic effect of white-skinned sweet potato (*Ipomoea batatas* L.) was demonstrated in a study by Kusano and Abe in obese Zucker fatty rats [12]. The oral administration of sweet potato for 6 weeks reduced 60 % of hyperinsulinemia, a symptom of type 2 diabetes mellitus, in these rats. Decreases in blood triacylglyceride and free fatty acid lactate levels were also observed in sweet potato-administered rats compared to the control group. Increase in blood glucose levels after glucose loading was also inhibited with 7-week administration of sweet potato. In addition, the administration of sweet potato showed even higher antidiabetic effect than an antidiabetic drug, troglitazone, in terms of body weight gain when increase in body weight gain appeared in troglitazone-treated group but not in sweet potato-treated group. A study by Ludvik et al. [13] on male type 2 diabetic patients for 6 weeks showed positive effects of caiapo, the extract of white-skinned sweet potato (*Ipomoea batatas*), containing powdered white-skinned sweet potato via reducing fasting plasma glucose and low-density lipoprotein cholesterol. The ingestion at high dose (4 g caiapo/day) also resulted in an increase in insulin sensitivity of the patients without side effects. In a later study, they observed an improvement of insulin sensitivity even at the low dose of caiapo by frequently sampled intravenous glucose tolerance test (from $2.02 \pm 0.7 \times 10^4$ to 2.76 ± 0.89/min/(μU/mL)). These results suggested that the extract of white-skinned sweet potato might be a potential application in the treatment of type 2 diabetes [14].

Recently, a parameter related to carbohydrate food, glycemic index has been considered in many studies because of its positive correlation with the risk increase in chronic diseases such as type 2 diabetes, cancer, and cardiovascular diseases [15, 16]. The glycemic index is a measure of how quickly a carbohydrate food affects the blood glucose levels. Fifty grams of glucose is the standard reference food on the glycemic index, and it is ranked as value of 100 because pure glucose raises blood sugar very quickly. The effect of other carbohydrate-containing foods on blood sugar level can then be compared with the effect of glucose to ascertain a particular food glycemic ranking [17]. Glycemic index of sweet potato and some other common carbohydrate foods is shown in Table 19.2 [18].

Table 19.2 Glycemic index (GI) of some common carbohydrate foods (glucose GI = 100)

Potato (baked)	98	Wheat cereal	67
Potato (boiled)	70	Table sugar	65
Pumpkin	75	*Sweet potato*	*61*
Millet	71	Oatmeal	58
Rice (white)	70	Taro	55
Rice (brown)	55	Corn	52
Tapioca (steamed)	70	Barley	25

Source: Foster-Powell et al. [18]

The low glycemic index of sweet potato in comparison with other rich-carbohydrate foods is related to low digestibility of starch and flour in this tuber root. A study on flour from fresh roots of the six sweet potato genotypes in China showed that the mean digestibility of those was only 54.0 (47.7–59.2 %) [19]. Astawana and Widowati [20] also investigated hypoglycemic responses of eight Indonesian sweet potato varieties. The highest hypoglycemic activity was observed in the variety having lowest starch digestibility (51.4 %). In addition, they reported that processing methods significantly influenced glycemic index of sweet potato. Glycemic index of fried, boiled, and baked sweet potatoes was 47, 62, and 80, respectively. Recently, remarkable effects of traditional cooking methods of ten sweet potato cultivars in Jamaica on glycemic index of nondiabetic people were reported [21]. Matured tubers were cooked by roasting, baking, frying, or boiling and consumed immediately by the ten nondiabetic test subjects (5 males and 5 females; mean age of 27 ± 2 years). Sweet potatoes processed by boiling had the lowest glycemic index (41–50), followed by fried (63–77), roasted (79–93), and baked ones (82–94).

The hypoglycemic effect of sweet potato suggested that this fresh root and its products are potential candidates in the use of selected diets as one of the ways for treatment of diabetes.

Sweet Potatoes with High β-Carotene Content, Potential Candidates for Solving Vitamin A Deficiency

Literature indicated that sweet potato is one of the best natural sources of β-carotene. However, β-carotene level in sweet potato varies in a wide range depending on many factors including genotype, age, farming area, and farming conditions. It was reported that β-carotene is not detected in some cultivars while some others contain extremely high content of this compound, even significantly higher than those of the common sources of β-carotene such as carrots and mangoes. Among variety of sweet potato cultivars with different flesh colors, orange-fleshed one was demonstrated to have highest β-carotene [5, 6]. By these reasons, several studies were established to evaluate influences of rich-β-carotene sweet potatoes on the vitamin A situation in developing countries.

As one of the major crops in developing countries, rich-β-carotene sweet potatoes were demonstrated to play an important role in solving the deficiency of vitamin A, one of the most prevalent problems in Sub-Saharan Africa and South Asia. While the supplementation from rich-vitamin A animal foods, such as fish oils, liver, milk, eggs, and butter, is becoming costly, sweet potato immerges as a potential supplementation of vitamin A in these countries. It was reported that 100 g of boiled roots of sweet potato can satisfy 50 % daily vitamin A requirement of a young child. In comparison with Golden Rice®, which is considered as a natural source of β-carotene, orange-fleshed sweet potatoes might contain this nutrient more than 20–30 times [8]. A study on children 3–6 years old in Sumatra, Indonesia, for 3 weeks showed that incorporation of β-carotene sources (mainly in the form of red sweet potato) into the meals and snacks significantly increased serum retinol concentrations of this group compared to the control group. In addition, the observation of an increase in serum retinol with the addition of extra fat in the meals suggested that fat might support the absorption of β-carotene in these objects. Changes in serum retinol of the children supplemented with red sweet potato and dietary fat are given in Fig. 19.1 [22].

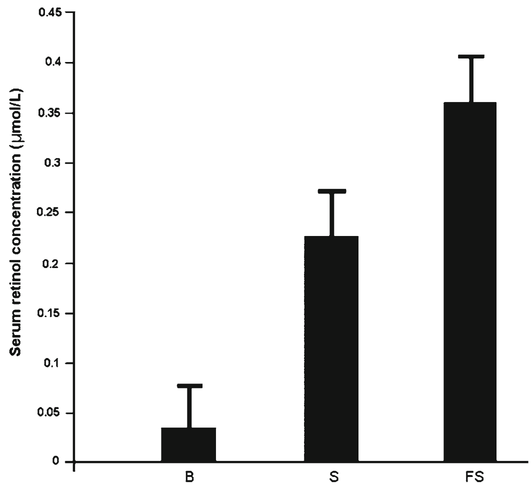

Fig. 19.1 Mean (±SEM) changes in serum retinol of children after supplementation. B: basic meal; S: basic meal + red sweet potato; FS: basic meal + red sweet potato + added dietary fat. *Source*: Jalal et al. [22]

In Sub-Saharan Africa, a project was carried out to investigate influences of replacement of white-fleshed sweet potatoes by orange-fleshed ones, which contain higher β-carotene, on vitamin A deficiency of population in some countries. It was concluded that orange-fleshed sweet potatoes are the promising food-based approach for controlling vitamin A deficiency problem in Sub-Saharan Africa [23]. This potential of sweet potato was asserted in a later study in South Africa by Jaarsveld et al. [24] which showed a greater improvement of vitamin A liver store in the children (5–10 years old) who consumed 125 g boiled and mashed orange-fleshed sweet potato than in the control group.

Anthocyanins: Biofunctional Pigment in Purple-Fleshed Sweet Potatoes

Besides orange-fleshed sweet potato, purple-fleshed one is also an important variety due

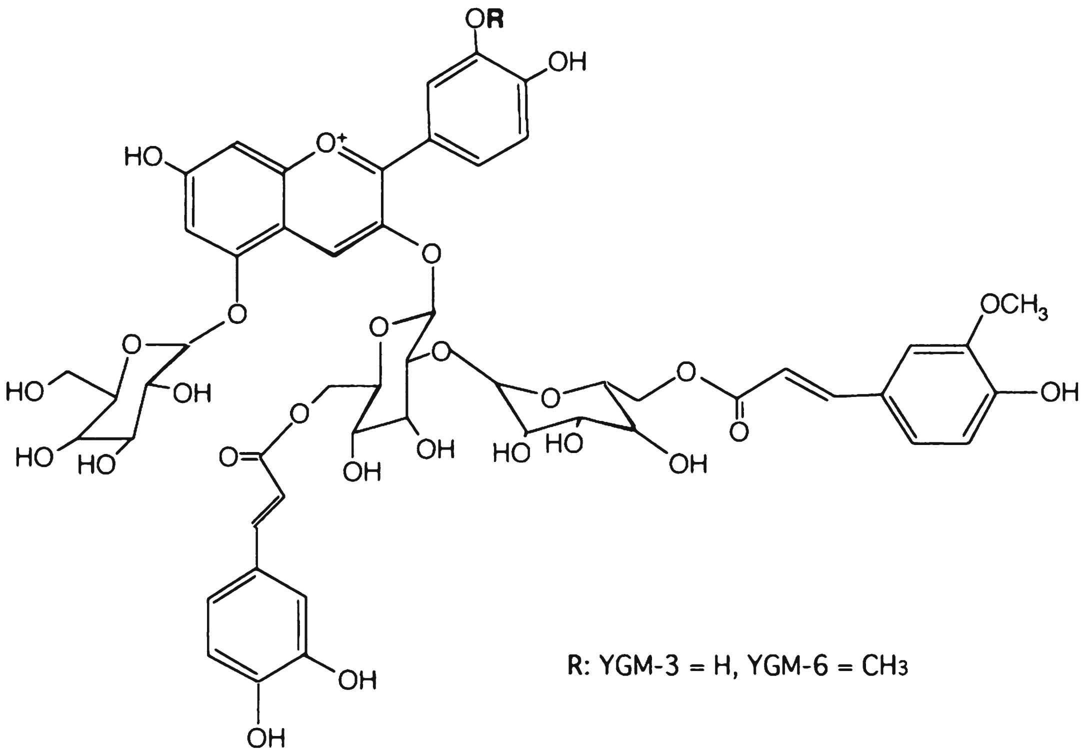

Fig. 19.2 Chemical structures of YGM-3 and YGM-6

to considerable content of anthocyanins in its root. Several anthocyanin pigments were found in purple-fleshed roots, and mono- or diacylated forms of cyanidin and peonidin were identified as predominant anthocyanins. The chemical structures of two most abundant anthocyanins in purple-fleshed sweet potato, 3-caffeylferulysophoroside-5-glucosides of cyanidin (YGM-3) and peonidin (YGM-6), are shown in Fig. 19.2 [25].

Anthocyanins in plants have been proven as powerful antioxidants for long time and administration of natural anthocyanins has been reported to be beneficial for treating diabetes, ulcers, and some other diseases [26]. Recently, a series of studies have suggested that purple-fleshed sweet potato might become a functional food due to strong antioxidative activity of its pigments, anthocyanins.

In a comparison of antioxidant property between 21 sweet potato cultivars with several flesh colors, ethanol extracts of sweet potato with purple flesh showed very high *tert*-butylperoxyl radical scavenging activity and antioxidant activity against lipid peroxidation induced by autooxidation of linoleic acid; whereas, these activities of cultivars with white, white-yellow, yellow, and orange flesh were low in almost all samples [27]. Anthocyanins were identified as the dominant DPPH radical-scavengers besides phenolic compounds in ethanol extracts of five purple-fleshed sweet potato cultivars in a study on the contribution of these compounds to radical scavenging activity of sweet potato [9]. Steed and Truong [7] reported that anthocyanin contents in sweet potatoes with purple skin and flesh in USA were in the range between 51.5 and 174.7 mg/100 g fresh weight. These sweet potatoes also exhibited comparable antioxidant capacity with various purple-colored fruits and vegetables, which was indicated by their DPPH radical scavenging activities (from 47.0 to 87.4 μmol trolox equivalent (TE)/g fresh weight) [7]. In vivo antioxidant activity of purple-fleshed

Table 19.3 Biological activities of anthocyanins in purple-fleshed sweet potato

Activity	Model	Effect	References
Anticancer	Rat liver microsomal activation systems	Inhibition of the reverse mutation induced by Trp-P-1, Trp-P-2, and IQ	[29]
	Salmonella typhimurium TA98	Inhibition of Trp-P-1-induced reverse mutation	[30]
	Human promyelocytic leukemia cells (HL-60)	Inhibition of cell proliferation	[30]
Hepatoprotective	Rats	Ameliorative effect against carbon tetrachloride-induced liver injury	[31]
	Impaired hepatic function volunteers	Reduction to normal level of serum γ-glutamyl transpeptidase, glutamic oxaloacetic transaminase, and glutamic pyruvic transaminase	[32]
	Healthy men	Decrease in levels of serum γ-glutamyl transferase, aspartate aminotransferase, and alanine aminotransferase	[33]
Anti-atherosclerotic	Human macrophage cells	Inhibition of fructose-mediated protein glycation and oxidation of low-density lipoprotein	[34]
Blood circulation improving	Rats	Enhancement of brain blood flow increase in ATP level in the stratum corneum	[35]
Memory enhancing	Rats and mice	Inhibition of lipid peroxidation in brain homogenates of rats	[36]
		Enhancement of cognitive performance in mice	

sweet potato anthocyanin concentrate was also exhibited in rats. The ingestion of this concentration resulted in a significant elevation in plasma antioxidant capacity of rat. The major acylated anthocyanin (Pn 3-Caf sop-5-glc) in this concentration was confirmed to be absorbed directly into rats in an intact form [28].

Anthocyanins from sweet potato have been demonstrated to have antimutagenic, vasoprotective, anti-inflammatory, and hepatoprotective activities in vitro and in vivo, as summarized in Table 19.3. Yoshimoto et al. [29] showed that extract from the whole roots of a purple-fleshed variety (Ayamurasaki) containing 3-(6,6′-caffeylferulylsophoroside)-5-glucoside of cyanidin (YGM-3) and peonidin (YGM-6) significantly inhibited the reverse mutation induced by Trp-P-1, Trp-P-2, and IQ in rat liver microsomal activation systems. They also suggested that the caffeyl or ferulyl group in the chemical structure of these anthocyanins may be responsible for their strong inhibition of reverse mutation. The potent antimutagenic activity of anthocyanin-rich aqueous extracts from this variety was then clarified by remarkable inhibition of Trp-P-1-induced reverse mutation of *Salmonella typhimurium* TA98 and the proliferation of human promyelocytic leukemia cells (HL-60) with treatments for 24 h. A dose of 1 mg/plate of extract from storage root resulted in 36 % and 25 % inhibition against the growth of Trp-P-1 and HL-60 cells, respectively [30].

Besides antimutagenic activity, the purple-fleshed sweet potato (Ayamurasaki) also exhibited positive effects on hepatic function. In an in vivo study by Suda et al. [31], administration with juice of this sweet potato showed ameliorative effect against carbon tetrachloride-induced liver injury of treated rats compared to the control. They also reported about hepatic restorative capacity of this juice by a clinical trial, in which, serum γ-glutamyl transpeptidase, glutamic oxaloacetic transaminase, and glutamic pyruvic transaminase of human volunteers with impaired hepatic function were lowered to normal levels after administration of this sweet potato juice [32]. A reduction in levels of serum hepatic biomarkers (γ-glutamyl transferase, aspartate aminotransferase, and alanine aminotransferase) was also observed by these authors in healthy Japanese men using purple sweet potato beverage rich in acylated anthocyanins [33].

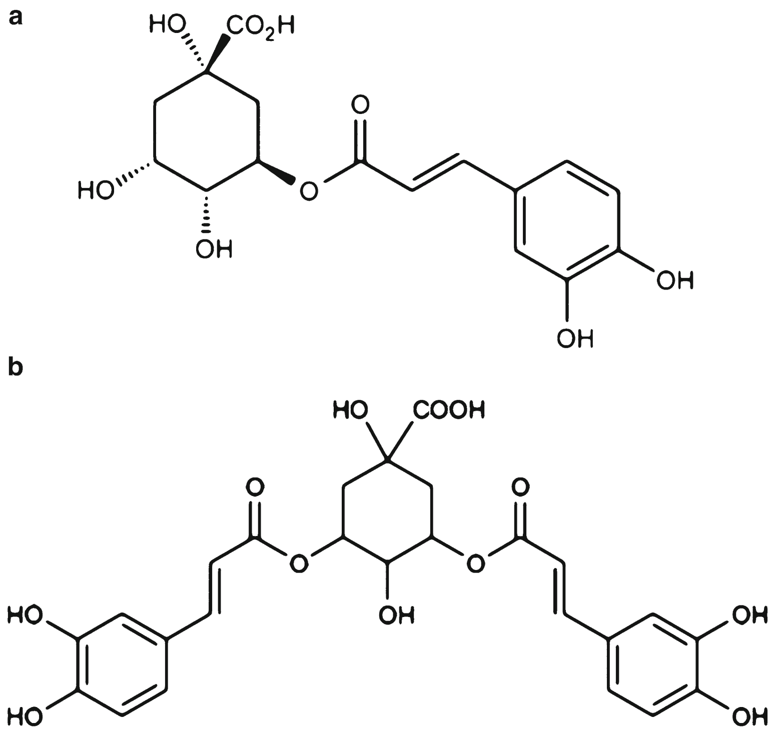

Fig. 19.3 Chemical structures of chlorogenic acid (**a**) and isochlorogenic acid A (**b**)

Anti-atherosclerotic activity of purple sweet potato was reported recently in a study by Park et al. [34]. The ethanol extract of purple sweet potato resulted in strong inhibitions to risk factors for atherosclerosis, fructose-mediated protein glycation, and oxidation of low-density lipoprotein. The inhibitory effect of this extract to cupric ion-mediated low-density lipoprotein oxidation was even comparable with that of vitamin C treatment [34]. The ability of anthocyanins in sweet potato to improve the blood circulation was demonstrated by an enhancement of brain blood flow and an increase in ATP level in the stratum corneum in the rats treated with anthocyanin fraction of sweet potato [35]. In addition, anthocyanin prepared from purple sweet potato was claimed to exhibit memory enhancing effects, which were higher than effects of Cordyceps mushroom, an extremely expensive Chinese medicine. The effective inhibition to lipid peroxidation initiated by Fe^{2+} and ascorbic acid in brain homogenates was only observed in rats treated with purple sweet potato extract but not in the group treated with the mushroom extract [36].

Phenolic Compounds and Antioxidant Activity of Sweet Potato

Phenolic content in hand-peeled storage roots sweet potato cultivars in the USA was reported in the range between 0.14 and 0.51 mg of chlorogenic acid equivalent/g fresh weight. It varied widely among cultivars, and chlorogenic acid and isochlorogenic acids were identified as the major phenolic compounds in these cultivars. Their chemical structures are shown in Fig. 19.3 [37].

As mentioned in the previous part, phenolic compounds and anthocyanins are dominant groups contributing to the antioxidant activity of sweet potato. Consequently, several studies have focused on the relation between antioxidant capacity and content of these compounds in sweet potato. Furuta et al. [27] showed a positive correlation between increase in the total phenolic content and radical scavenging activities of ethanol extracts of 21 sweet potato cultivars with several flesh colors. This observation suggested that phenolic compounds are important candidates for the radical scavenging activity of various genotypes of sweet potato [27]. Nineteen sweet potato genotypes in the USA with various flesh colors from white, cream, yellow, orange, and purple were also evaluated for phenolic content and antioxidant activity. The purple-fleshed cultivars showed the highest total phenolic content, followed by orange, yellow, and white-fleshed ones. The antioxidant activities of these genotypes were found to be highly correlated with their phenolic content [38]. In a later study by Steed and Truong [7], purple sweet potato showed very high phenolic content (313.6–1483.7 mg chlorogenic acid equivalent/100 g fresh weight) with correlative DPPH radical scavenging activities (47.0–87.4 μmol trolox equivalent (TE)/g fresh weight). These indices of purple sweet potato were comparable with various purple-colored fruits and vegetables [7]. In the Philippines, total phenolic content and antioxidant activities of five sweet potato varieties were evaluated by Rumbaoa et al. [39]. They reported that phenolic content of these varieties ranged from 192.7 to 1159.0 mg gallic acid equivalent (GAE)/100 g dry sample; antioxidant capacities of methanol extracts from these sweet potatoes were even higher than those of EDTA. Besides, positive correlations were also observed between phenolic content and DPPH radical scavenging activity and reducing power and iron-chelating capacity of these extracts [39]. Zhu et al. [40] recently identified mono- and dicaffeoylquinic acids and caffeoyl-hexoside as the main phenolic compounds in the crude extracts of roots of ten Chinese purple-fleshed sweet potato genotypes. These phenolic compounds were demonstrated to be responsible for antioxidant capacity of the tested sweet potato samples. In addition, great differences between genotypes and parts of the roots in phenolic content and antioxidant activity were also reported [40]. In South America, a red-fleshed sweet potato in the Andean region exhibited even higher phenolic content and antioxidant capacity than a certain cultivar of blueberry, a fruit known as a rich source of antioxidants. This result suggests that some sweet potato cultivars can be considered as novel sources of natural antioxidants for dietary supplementations and functional foods [41].

High Quality Storage Proteins in Sweet Potato

Sporamins were identified as the major storage proteins, which account for about 80 % of total protein in root of sweet potato [42]. In sweet potato, proteins not only act as a nutrient component for energy supplying but also exhibit some other important biological functions. Lin [43] found that water-soluble proteins are the trypsin inhibitors in sweet potato storage roots. Among the trypsin inhibitors purified from a Chinese sweet potato (cv. Tainong 57), a 33 kDa trypsin inhibitor showed scavenging activity against DPPH radical in a dose-dependent manner. This evidence suggested a certain contribution of storage proteins to antioxidant capacity of sweet potato [44]. The sporamin B protein from sweet potato also exhibited significant antioxidant activity through total antioxidant status test by Huang et al. [45]. Concentration of 100 μg/mL of sporamin B expressed 4.21 ± 0.0078 mM trolox equivalent antioxidative value [45]. They also reported the ability of trypsin inhibitor isolated from sweet potato storage roots to scavenge $^{\bullet}$ON and $ONOO^{-}$, the factors related to several human diseases such as Alzheimer's disease, atherosclerosis, and stroke [46]. Results showed that trypsin inhibitor suppressed the production of nitrite and superoxide radicals in a dose-dependent manner. Trypsin inhibitor also showed a significant ability to inhibit nitration of bovine serum albumin and inhibition to lipopolysaccharide-induced nitrite production in macrophages. This effect of trypsin inhibitor on the reactive nitrogen species suggested that it is useful for the prevention of NO^{-}- and ONOO-involved diseases. This suggestion was partially confirmed in their later study, which found that trypsin inhibitor might be good for control of hypertension and other diseases. In this study, trypsin inhibitor from sweet potato inhibits angiotensin converting enzyme, a factor related to hypertension and other diseases, in a dose-dependent manner. Treatments with concentration from 50 to 200 μg/mL of this compound resulted in inhibitions of this enzyme in a range from 31.9 to 53.2 % [47].

Dietary Fiber of Sweet Potato

Fiber in sweet potato is also an important nutrient in the diets. It was reported that sweet potatoes generally contain 2–4 % dietary fiber, which vary with cultivars, ages, and farming conditions [4]. Dietary fiber contributes potential benefits for human health including reduction of bowel transit time, decrease in the risk of colorectal cancer, lowering of serum blood cholesterol, and promotion of the growth of gut microflora [48].

Conclusions

Sweet potato, the seventh most commonly consumed carbohydrate-rich food source, has emerged recently among other food crops due to its special nutrient composition. Phenolic compounds, β-carotene, anthocyanins, and sporamins were demonstrated to be responsible for various biological functions of sweet potato. The benefits in treating diabetes, cancers, and hypertension, solving vitamin A deficiency, improving hepatic functions, and enhancing memory of this tuber root suggested that it should be considered as a high quality food. However, the underlying mechanisms of these health benefits are waiting for being clarified by further studies. The future work should also be done to find out other nutritional values of sweet potato, improve nutritional value of the existing varieties, and produce new varieties with high yield of biofunctional compounds.

References

1. O'Brien PJ. The sweet potato: its origin and dispersal. Am Anthropol. 1972;74:342–65.
2. Scott GJ. Transforming traditional food crops: product development for roots and tubers. In: Scott GJ, Wiersema S, Ferguson PI, editors. Product development for roots and tuber crops, vol. 1. Lima, Peru: International Potato Center (CIP); 1992. p. 3–20.
3. Food and Agricultural Organization (FAO) Web site. Available at: http://faostat.fao.org/site/567/default.aspx#ancor. Accessed on October 20, 2011.
4. Van Hal M. Quality of sweet potato flour during processing and storage. Food Rev Int. 2000;16:1–37.
5. Kays SJ. The chemical composition of the sweet potato. In: Hill WA, Bonsi CK, Loretan PA, editors. Sweet potato technology for the 21st century. Tuskegee, AL: Tuskegee University; 1992. p. 201–62.
6. K'osambo LM, Carey EE, Misra AK, Wilkes J, Hagenimana V. Influence of age, farming site, and boiling on pro-vitamin A content in sweet potato (Ipomoeu batatus (L.) Lam.) storage roots. J Food Compos Anal. 1998;11:305–21.
7. Steed LE, Truong VD. Anthocyanin content, antioxidant activity, and selected physical properties of flowable purple-fleshed sweetpotato purees. J Food Sci. 2008;73:215–21.
8. Ye XS, Al-Babili S, Kloti A, Zhang J, Lucca P, Beyer P, et al. Engineering the provitamin A (β-carotene) biosynthetic pathway into (carotenoid-free) rice endosperm. Science. 2000;287:303–5.
9. Oki T, Masuda M, Furuta S, Nishiba Y, Terahara N, Suda I. Involvement of anthocyanins and other phenolic compounds in radical-scavenging activity of purple-fleshed sweet potato cultivars. J Food Sci. 2002;67:1752–6.
10. Hoover R. Composition, molecular structure, and physicochemical properties of tuber and root starches: a review. Carbohydr Polym. 2001;45:253–67.
11. Babu L. Changes in carbohydrate fractions of sweet potato tubers on processing. Trop Agric. 1994;71: 71–3.
12. Kusano S, Abe H. Antidiabetic activity of white skinned sweet potato (Ipomoea batatas L.) in obese Zucker fatty rats. Biol Pharm Bull. 2000;23:23–6.
13. Ludvik B, Mahdjoobian K, Waldha W. The effect of Ipomoea batatas (caiapo) on glucose metabolism and serum cholesterol in patients with type 2 diabetes: a randomized study. Diabetes Care. 2002;25:239–40.
14. Ludvik B, Waldhausl W, Prager R, Kautzky-Willer A, Pacini G. Mode of action of Ipomoea batatas (caiapo) in type 2 diabetic patients. Metabolism. 2003;52: 875–80.
15. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. Pediatrics. 1999;103:E26.
16. Higginbotham S, Zhang ZF, Lee IM. Dietary glycemic load and risk of colorectal cancer in the women's health study. J Natl Cancer Inst. 2004;96:229–33.
17. Willett W, Manson J, Liu S. Glycemic index, glycemic load and risk of type 2 diabetes. Am J Clin Nutr. 2002;76:274–80.
18. Foster-Powell KF, Holt SHA, Miller JCB. International table of glycemic index and glycemic load values. Am J Clin Nutr. 2002;76:5–56.
19. Zhang Z, Wheatley CC, Corke H. Biochemical changes during storage of sweet potato roots differing in dry matter content. Postharvest Biol Technol. 2002;24:317–25.
20. Astawana M, Widowati S. Evaluation of nutrition and glycemic index of sweet potatoes and its appropriate processing to hypoglycemic foods. Indonesian J Agric Sci. 2011;12:40–6.
21. Bahado-Singh PS, Riley CK, Wheatley AO, Lowe HIC. Relationship between processing method and the glycemic indices of ten sweet potato (Ipomoea batatas) cultivars commonly consumed in Jamaica. J Nutr Metab. 2011; doi: 10.1155/2011/584832.

22. Jalal F, Nesheim MC, Agus Z, Sanjur D, Habicht JP. Serum retinol concentrations in children are affected by food sources of beta-carotene, fat intake, and anthelmintic drug treatment. Am J Clin Nutr. 1998;68: 623–9.
23. Low J, Walker T, Hijmans R. The potential impact of orange-fleshed sweet potatoes on vitamin A intake in Sub-Saharan Africa. A paper presented at a regional workshop on food based approaches to human nutritional deficiencies. The VITAA project, vitamin A and orange-fleshed sweet potatoes in sub-Saharan Africa, 9–11 May 2001, Nairobi, Kenya.
24. Jaarsveld PJV, Faber M, Tanumihardjo SA, Nestel P, Lombard CJ, Benadé AJS. Beta-carotene-rich orange-fleshed sweet potato improves the vitamin A status of primary school children assessed with the modified-relative-dose–response test. Am J Clin Nutr. 2005;81:1080–7.
25. Odake K, Terahara N, Saito N, Toki K, Honda T. Chemical structures of two anthocyanins from purple sweet potato, Ipomoea batatas. Phytochemistry. 1992; 31:2127–30.
26. Amouretti M. Therapeutic value of Vaccinium myrtillus anthocyanosides in an internal medicine department. Therapeutique. 1972;48:579–81.
27. Furuta S, Suda I, Nishiba Y, Yamakawa O. High tert-butylperoxyl radical scavenging activities of sweet potato cultivars with purple flesh. Food Sci Technol Int Tokyo. 1998;4:33–5.
28. Suda I, Oki T, Masuda M, Nishiba Y, Furuta S, Matsugano K, et al. Direct absorption of acylated anthocyanin in purple-fleshed sweet potato into rats. J Agric Food Chem. 2002;50:1672–6.
29. Yoshimoto M, Okuno S, Yoshinaga M, Yamakawa O, Yamaguchi M, Yamada J. Antimutagenicity of sweet-potato (Ipomoea batatas) roots. Biosci Biotechnol Biochem. 1999;63:537–41.
30. Konczak-Islam I, Yoshimoto Y, Hou D, Terahara N, Yamakawa O. Potential chemopreventive properties of anthocyanin-rich aqueous extracts from in vitro produced tissue of sweetpotato (Ipomoea batatas L.). J Agric Food Chem. 2003;51:5916–22.
31. Suda I, Furuta S, Nishiba Y, Yamakawa O, Matsugano K, Sugita K. Reduction of liver injury induced by carbon tetrachloride in rats administered purple sweetpotato juice. J Jpn Soc Food Sci Technol. 1997;44:315–8.
32. Suda I, Yamakawa O, Matsugano K, Sugita K, Takeguma Y, Irisa K, et al. Changes of serum γ-GTP, GOT, and GPT levels in hepatic function-weakling subjects by ingestion of high anthocyanin sweetpotato juice. J Jpn Soc Food Sci Technol. 1998;45:611–7.
33. Suda I, Ishikawa F, Hatakeyama M, Miyawaki M, Kudo T, Hirano K, et al. Intake of purple sweet potato beverage affects on serum hepatic biomarker levels of healthy adult men with borderline hepatitis. Eur J Clin Nutr. 2008;62:60–7.
34. Park KH, Kim JR, Lee JS, Lee HH, Cho KH. Ethanol and water extract of purple sweet potato exhibits anti-atherosclerotic activity and inhibits protein glycation. J Med Food. 2010;13:91–8.
35. Jo A, Sawai Y, Kakuo H. Food ingredients from purple sweet potato improving the blood circulation. Japanese patent, 2001-145471; 2001.
36. Cho J, Kang JS, Long PH, Jing J, Back Y, Chung KS. Antioxidant and memory enhancing effects of purple sweet potato anthocyanin and cordyceps mushroom extract. Arch Pharm Res. 2003;26:821–5.
37. Walter Jr WM, Purcell AE, McCollum GK. Use of high-pressure liquid chromatography for analysis of sweetpotato phenolics. J Agric Food Chem. 1979;27: 942–6.
38. Teow CC, Truong VD, McFeeters RF, Thompson RL, Pecota KV, Yencho GC. Antioxidant activities, phenolic and β-carotene contents of sweet potato genotypes with varying flesh colours. Food Chem. 2007;10:3829–38.
39. Rumbaoa RGO, Cornago DF, Geronimo IM. Phenolic content and antioxidant capacity of Philippine sweet potato (Ipomoea batatas) varieties. Food Chem. 2009;113:1133–8.
40. Zhu F, Cai YZ, Yang X, Ke J, Corke H. Anthocyanins, hydroxycinnamic acid derivatives, and antioxidant activity in roots of different Chinese purple-fleshed sweetpotato genotypes. J Agric Food Chem. 2010;58: 7588–96.
41. Cevallos-Casals BA, Cisneros-Zevallos LA. Stoichiometric and kinetic studies of phenolic antioxidants from Andean purple corn and red-fleshed sweetpotato. J Agric Food Chem. 2003;51:3313–9.
42. Matsuoka K, Matsumoto S, Hattori T, Machida Y, Nakamura K, Maeshima M. Vacuolar targeting and posttranslational processing of the precursor to the sweet potato tuberous root storage protein in heterologous plant cells. J Biol Chem. 1990;265: 19750–957.
43. Lin YH. Relationship between trypsin-inhibitor activity and water-soluble protein and cumulative rainfall in sweet potato. J Am Soc Hortic Sci. 1989;114: 814–8.
44. Hou WC, Chen YC, Chen HJ, Lin YH, Yang LL, Lee MH. Antioxidant activities of trypsin inhibitor, a 33 KDa root storage protein of sweet potato (Ipomoea batatas (L.) Lam cv. Tainong 57). J Agric Food Chem. 2001;49:2978–81.
45. Huang GJ, Chen HJ, Chang YS, Sheu MJ, Lin YH. Recombinant sporamin and its synthesized peptides with antioxidant activities in vitro. Bot Stud. 2007;48:133–40.
46. Huang GJ, Sheu MJ, Chen HJ, Chang YS, Lin YH. Inhibition of reactive nitrogen species in vitro and ex vivo by trypsin inhibitor from sweet potato 'Tainong 57' storage roots. J Agric Food Chem. 2007;55: 6000–6.
47. Huang GJ, Ho YL, Chen HJ, Chang YS, Huang SS, Hung HJ, et al. Sweet potato storage root trypsin inhibitor and their peptic hydrolysates exhibited angiotensin converting enzyme inhibitory activity in vitro. Bot Stud. 2008;49:101–8.
48. Brennan CS. Dietary fibre, glycaemic response, and diabetes. Mol Nutr Food Res. 2005;49:560–70.

Cooking and Diet Quality: A Focus on Meat

20

Cristina M. Alfaia, Anabela F. Lopes, and José A.M. Prates

Key Points

- This chapter deals with the influence of cooking on meat composition, nutritional value, safety, and sensory and functional properties.
- Detailed nutrient composition of the most eaten meats (beef, pork, and poultry) reviewed from the literature was complemented with original data from the authors.
- A broad number of food cooking techniques are considered ranging from traditional to emerging thermal technologies.
- Cooking may have either beneficial or harmful effects on the physicochemical properties of meat, including lipid and protein oxidation.
- The suitable cooking time and temperature parameters must represent a compromise between nutritional and sensory quality.

Keywords
Cooking • Meat • Diet • Quality • Nutrient losses • Oxidative processes

Abbreviations

CLA	Conjugated linoleic acid
COPs	Cholesterol oxidation products
dl-PCBs	Dioxin-like polychlorinated biphenyls
HAAs	Heterocyclic aromatic amines
MUFA	Monounsaturated fatty acids
PAHs	Polycyclic aromatic hydrocarbons
PUFA	Polyunsaturated fatty acids
SFA	Saturated fatty acids
TFA	*Trans* fatty acids
TR	True retention

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Introduction

Diet quality involves food composition, consumer appreciation, and food safety. Food quality has been related to safety, sensory characteristics, and shelf life of food products, but, nowadays, it is more associated with nutrition, well-being, and health [1]. Remarkable changes in consumer's attitude towards food have been occurring during the last few decades increasing the consumer's demand for a high quality diet (variety, adequacy, moderation, and overall balance) covering the nutritional concerns. The purpose of an equilibrated diet is to provide the nutrients required for normal growth, development, and maintenance of a healthy life. It is well established that the accurate balance of micro- and macronutrient intake has been associated with optimal health, but it can also play an important role in reducing the risk of lifestyle-related diseases [2]. Nevertheless, increases only in the quantity of foods will not address diet quality, which cover supply adequate amounts of particular micronutrients, namely vitamins, minerals, and trace metals. Animal food products, particularly meat and milk, hold great promise for improving diets of poor quality in developing countries [3]. In addition, the dietary patterns in Mediterranean countries are changing with increased consumption of animal products and saturated fat to the detriment of vegetable foodstuffs. Some reasons for this nutrient transition could be highly associated to the modernization of society, which implies socio-economic and cultural changes, and technological innovations that affect feeding preferences and dietary habits, including food components and cooking processes. Indeed, the majority of foods become more edible and palatable when submitted to cooking. However, considerable changes occur during processing, and these modifications are strongly dependent upon the specific food (Fig. 20.1).

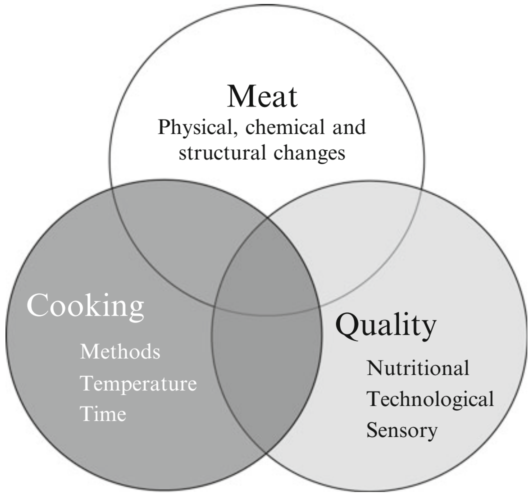

Fig. 20.1 Schematic representation of the relationship between meat quality characteristics and main cooking variables

This chapter provides an overview on the cooking techniques behind the main methods used in today's food industry and households (section "An Overview of the Cooking Methods"). The methodologies available range from the conventional methods to the recent developments of minimal processing technologies, which are designed to limit the impact of heating on the nutritional quality of foods. In section "Meat Changes Induced by Cooking," the most relevant meat changes induced by cooking will be discussed. Heat treatments can either improve the nutritional value of meat by making nutrients more available or lead to leaching of essential micronutrients. The effect of cooking methods on meat composition of beef, pork, and poultry is reviewed from the literature and complemented with original data (unpublished), obtained by our research group (section "Nutritional Changes"). In addition, meat composition undergoes significant biochemical changes, including lipid and protein oxidation (section "Oxidative Changes"), which may in turn affect the sensory characteristics, and its acceptability by consumers (section "Sensory Properties and Eating Quality"). Finally, section "Safety Concerns of Meat Cooking" is focused on the major concerns associated with the formation of potentially harmful compounds in cooked meats and, therefore, the information related with food quality and food safety are of utmost importance with regard to public health.

An Overview of the Cooking Methods

Cooking can be described as the practice of preparing food for eating by the application of heat. Although it has long been argued that cooking improves the quality of diet while maintaining product safety, the nature of the improvement has not yet been well defined [4].

The majority of foods are submitted to thermal processing like cooking, pasteurization, or sterilization. Cooking helps to achieve the desirable sensory characteristics while pasteurization or sterilization aims to assure microbiological safety, and to eliminate some enzymatic activities that reduce food preservation [5]. The heating processes applied in food industry are based on the same principles as those prevailing in the households. Most of the traditional cooking methods rely on conductive and/or convective mechanisms from the heating medium (air, water, oil) to the food product. Depending on the product geometry, it may take considerable time to conduct sufficient heat into the product core to reach a safe end-point temperature. This may cause some parts of the product to be overcooked or undercooked, adversely causing quality deterioration [6]. The two general heating strategies differentiated by heat transfer include dry heat cooking and moist heat cooking. Dry cooking uses hot air to conduct the heat and food cooks in its own juice. Such methods of cooking include baking, roasting, and grilling. Cooking by moist heat consists in heating food by immersion in boiling water, or cooking in the steam, which comes out from the boiling water. In oven cooking, the forced air convection method is often coupled with steam injection to reduce the cooking time and prevent surface dehydration. Traditional wet cooking methods include boiling, stewing or braising, steaming, and frying. Fry-cooking methods comprise deep-frying, pan-frying, and stir-frying/wok. Deep-frying is a high temperature cooking process in which the oil provides an effective medium of heat transfer. However, a huge number of traditional cooking methods cause inevitable nutrient losses, which can adversely affect the nature and quality of foods. These nutrient losses may vary according to the food type, size and shape, and cooking technique.

The novel thermal processes often referred to as "minimal processes" aim to produce safer foods with high nutritional quality. These minimal processing technologies have gained importance as potential alternative methods because conventional heating requires longer cooking time and nonuniform temperature distribution. The emerging technologies include microwaving, radio frequency, and ohmic heating. Electromagnetic heating methods transfer energy from its source directly into the food without heating up the heat transfer surface of the processing equipment. Industrial microwave food applications use the two frequencies of 915 and 2,450 MHz, while domestic ovens use 2,450 MHz. Radio frequency applications are restricted to 13.56, 27.12, and 40.68 MHz for domestic, industrial, and scientific and medical purposes, respectively. Two mechanisms (dielectric and ionic) are involved in microwaving and radio frequency heating, with water in food serving as the foremost component for heating. The water molecules respond readily to the oscillating electromagnetic field, resulting in frictional interactions that generate heat. The other mechanism of heating is the oscillatory migration of ions present in food [7]. Microwave cooking has been applied successfully to the tempering or partial defrosting of foods. Although microwave heating can have beneficial effects on chemical reactions, some publications have claimed that microwaves do not show any improvement in efficiency over conventional methods [8]. Thus, the effect of microwaves and its mechanism are still under debate. An extension of microwaves is the application of radio frequency heating in the food industry. With radio frequency cooking, the product is heated internally, not through the surfaces, offering a more controlled heating of foods [7]. In addition, ohmic heating, also called electrical resistance heating or direct resistance heating, is one of the promising techniques to heat products where the energy is directly dissipated into the product. Ohmic heating becomes a very effective high temperature technology with the potential to shorten heat processing times. One of the major advantages of ohmic heating compared to the conventional cooking, despite a 15-fold reduction in cooking time, is the reduced cooking loss [9].

Meat Changes Induced by Cooking

Meat is known for its high nutritive composition, which could explain why it is consumed worldwide. Beef, lamb, pork, and chicken are the foremost meats consumed in Western societies. Despite a shift towards higher poultry consumption, red meat still represents the largest proportion of meat consumed (58 %) in the USA [10]. In contrast, meat consumption in the UK has remained steady since the 1980s. While consumption of chicken, pork, and meat products has increased, beef and lamb has declined. In the European Community, the outlook is characterized by a further contraction in total meat consumption, which is expected to decline by 0.7 % in 2012 and 0.4 % in 2013. Only poultry meat has proven to be resilient, with consumption estimated to have grown slightly in 2011 [11].

Meat constitutes an important source of high biological value protein, providing all the essential amino acids, fundamental for growth and development to enhance well-being and health. Proteins from animal sources contain the essential amino acids required by the organism since they cannot be synthesized de novo, and, therefore, must be available through the diet. Meat is also an excellent source of micronutrients (vitamins, minerals, and trace elements), which are often limiting in the diet, such as vitamin A, iron, zinc, and other bioactive compounds [12]. Moreover, ruminant meat represents a major source of conjugated linoleic acid (CLA) isomers in the human diet. CLA is a group of fatty acids composed of positional and geometric isomers of linoleic acid, containing a conjugated double-bond system. Twenty-four different CLA isomers occur naturally in ruminant-derived foods, namely meat, milk, and dairy products. Some CLA isomers, at least the *cis*9,*trans*11 and *trans*10,*cis*12, have been shown to have an impressive range of promising health benefits, including prevention of heart diseases and atherosclerosis, inhibition of tumors growth, and modulation of body fat composition [13].

Meat is commonly cooked prior to consumption. Depending on the consumers' meat preferences, a wide range of traditional cooking methods have been employed at home to cook meat, such as dry heat methods, moist heating methods, and microwave cooking. Sometimes, a combination of dry and moist heat methods is used to cook meat. The selection of the cooking treatment must take into account the meat type, the amount of connective tissue, and the shape and size of meat. For experimental purposes, the heating parameters must be standardized, controlled, and not overshadow the effects of the treatment. Cooking temperature is one of the well-known altering factors that can influence the characteristics of foods. The temperature on the surface of meat, the temperature profile through the meat, and the method of heat transfer are the most important differences across the cooking methods. Perhaps, the major difficulty during the cooking process is the low rate of heat conduction to the thermal center. Consumers base empirically the evaluation of cooked meat by their experience and judgment (e.g., change of color, juice) or, alternatively, by cooking times reported on labels, but these methods are generally misleading them [14]. Hence, consumers are being advised on appropriate temperatures to which meat products should be cooked. Since 2001, the Department of Agriculture launched campaigns to encourage consumers to cook food to a safe internal temperature providing a detailed list of end-point temperatures [15]. Likewise, the Food and Drug Administration has distributed a summary chart with minimum cooking food temperatures and holding times [16].

Nutritional Changes

The nutritional quality of meat is influenced by numerous factors, including cooking (temperature, rate, time, moist or dry heat). Heating can lead to undesirable changes such as a decrease in the nutritional value of meat mainly due to nutrient losses. Table 20.1 summarizes the effect of cooking processes on proximate composition, energy content,

Table 20.1 Effect of cooking methods on proximate composition, energy content and cooking loss of meat (g/100 g, except where noted)

Meat type/ nutrient	Raw	Conventional oven/roasting	Boiling	Microwave oven	Grilling	Frying	Ohmic heating	Heat processing parameters	Reference
Beef									
Moisture	74.8		61.2 (49.2)	54.5 (41.8)	62.3 (56.1)			BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	74.2	63.8 (66.4)		63.9 (61.1)	67.3 (68.1)	65.9 (72.3)		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
	72.3	68.8					68.3	Steam 80 °C; ohmic heating 3.5 kW (15 A, 50 Hz, 0–250 V)	[9]
Protein	20.8	27.2 (101)		30.6 (104)	28.7 (103)	25.6 (100)		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
	19.6		30.9 (106)	28.2 (92.5)	29.4 (95.8)			BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
	23.2	27.0					28.3	Steam 80 °C; ohmic heating 3.5 kW (15 A, 50 Hz, 0–250 V)	[9]
Fat	1.25		2.17 (107)	2.61 (122)	2.2 1 (121)			BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	1.97	1.79 (70.7)		1.73 (62.0)	1.69 (64.3)	3.71 (153)		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
	2.80	2.87					2.66	Steam 80 °C; ohmic heating 3.5 kW (15 A, 50 Hz, 0–250 V)	[9]

(continued)

Table 20.1 (continued)

Meat type/ nutrient	Raw	Conventional oven/roasting	Boiling	Microwave oven	Grilling	Frying	Ohmic heating	Heat processing parameters	Reference
Ash	3.38	3.50 (83.7)		4.01 (73.0)	3.71 (82.3)	3.67 (88.1)		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
	0.98		0.77 (46.5)	1.40 (89.9)	1.76 (104)			BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
	1.22	1.34					1.37	Steam 80 °C; ohmic heating 3.5 kW (15 A, 50 Hz, 0–250 V)	[9]
Energy*	103	96.3		97.6	97.4	111		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
	123		183	171	189				†
Cooking loss		22.8		29.3	25.0	18.6		RO, 170 °C/15 min; MW, 700 W/2.5 min and 300 W/2.5 min; GR, 210 °C/3 min; FR, 170 °C/5 min	[30]
			39.9	42.7	32.6			BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
		33.8					36.3	Steam 80 °C; ohmic heating 3.5 kW (15 A, 50 Hz, 0–250 V)	[9]
	123		183	171	189			BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†

Pork									
Moisture	54.1	40.5		46.9	43.3	49.2		GR, 190 °C/4 min; FR, 170 °C/4 min; MW, 450 W/90 s; RO, 150 °C/20 min	[26]
	74.3[a]; 75.5[b]	61.4[a]				67.7[b]		RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Protein	25.5	31.8		28.8	29.0	27.7		GR, 190 °C/4 min; FR, 170 °C/4 min; MW, 450 W/90 s; RO, 150 °C/20 min	[26]
	23.3[a]; 24.1[b]	31.9[a]				29.5[b]		RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Fat	2.55	3.18		2.88	2.90	2.77		GR, 190 °C/4 min; FR, 170 °C/4 min; MW, 450 W/90 s; RO, 150 °C/20 min	[26]
	2.2[a]; 1.6[b]	8.6[a]				3.5[b]		RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Ash	1.2[a]; 1.2[b]	1.2[a]				1.3[b]		RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Energy*	112[a]; 114[b]	206[a]				151[b]		RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Cooking loss		14.4		48.6		28.0		Steam, 190 °C/17 min; MW, 700 W/8 min and 1 min grill; FR, 180 °C/10 min	[25]

(continued)

Table 20.1 (continued)

Meat type/ nutrient	Raw	Conventional oven/roasting	Boiling	Microwave oven	Grilling	Frying	Ohmic heating	Heat processing parameters	Reference
Poultry									
Moisture	76.6	70.3; 47.3						Dry air, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	76.6	65.6; 56.5						Air–steam, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	74.4[a]; 76.5[b]	65.8[a]; 65.3[b]			65.2[a]; 65.2[b]	69.5[a]; 69.4[b]		RO, 200 °C/20 min; GR, 200 °C/20 min; FR, 180 °C/5 min	[37]
	66.2			61.6		61.4		MW, 900 W/3 min; FR, 180 °C/3 min	[28]
Protein	21.5	27.2; 47.5						Dry air, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	21.5	32.1; 38.9						Air–steam, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	23.7[a]; 21.1[b]	31.4[a]; 34.0[b]			32.8[a]; 31.3[b]	28.1[a]; 27.6[b]		RO, 200 °C/20 min; GR, 200 °C/20 min; FR, 180 °C/5 min	[37]
Fat	0.64	1.04; 3.04						Dry air, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	0.64	0.93; 2.11						Air–steam, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	3.07[a]; 3.11[b]	1.65[a]; 1.81[b]			1.54[a]; 1.81[b]	1.37[a]; 1.23[b]		RO, 200 °C/20 min; GR, 200 °C/20 min; FR, 180 °C/5 min	[37]
	10.0			10.7		11.5		MW, 900 W/3 min; FR, 180 °C/3 min	[28]

Ash	1.29	1.37; 2.18		Dry air, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	1.29	1.41; 2.43		Air–steam, 130 °C/4 min and 170 °C/12 min, respectively	[38]
	0.80	0.90 (95)	0.90 (81)	RO, 100 min; MW core temperature, 88 and 93 °C, respectively	[31]

BO boiling, *MW* microwaving, *GR* grilling, *FR* frying, *RO* roasting
Data in the same row indicate contents and retention values (%) in parenthesis. The same letters (abcd) correspond to the contents of the same meat in raw and cooked state
†Our values (unpublished results)
*expressed as kcal

and cooking loss of some of the most eaten meats (beef, pork, and poultry). The data reviewed from literature was complemented with original data obtained by our research group.

Water is the major constituent of meat accounting for approximately 75 % of its weight. Most of this water is physically retained by capillarity within myofibrillar proteins, while a small amount is bound by the sarcoplasmic proteins and the connective tissue [17]. During cooking, meat proteins contract as a result of thermal denaturation leading to a substantial weight loss, which can be up to 20–40 % of the initial weight, mainly in the form of water and fat [18]. Internal transport of liquid water, due to protein denaturation, causes the shrinkage of the meat fibers network resulting in a mechanical force that expels the excess interstitial water towards the surface [19]. Depending on heat and mass transfer conditions at surface, the water can be lost by evaporation through the crust or as drip. Goñi and Salvadori [20] reported that oven cooking (or roasting) involves heat transfer from the surrounding ambient to the food surface and, consequently, induces a temperature gradient inside the product. An increase of the internal temperature results in huge changes of water distribution and structure of meat [18, 21]. Normally, the water content of meat decreases while fat and protein contents increase, indicating that the main fraction of the cooking loss is water [22, 23]. Alfaia et al. [24] reported significant losses of moisture in cooked beef, which were more evident in microwaves. In microwave-heated foods, the water migration towards its surface is fast and, thus, facilitates water evaporation from food [25]. Regarding pork meat, cooking causes a great reduction of moisture, except in fried samples, and suggests a water loss due to the high temperatures during treatments [26]. Pork cooked in conventional oven shows the lowest amounts of moisture [26, 27]. The moisture value (54 %) reported for raw pork meat is lower to the ones obtained by other authors, because the samples came from Iberian pigs, a special breed characterized by high levels of total lipids and, thus, lower moisture content [26]. In chicken patties, microwave heating and frying decreased the moisture contents [28].

Meat proteins are subject to various changes during heating involving the molecule itself or resulting from interactions with other meat constituents. These modifications mostly comprise denaturation (loss of native tertiary structure) and hydrolytic degradation (proteolysis) of proteins by endogenous and/or exogenous enzymes [29]. The nature and extent of these modifications are temperature- and time-dependent, although other factors, like amino acid sequence and protein conformation, pH, and water activity (a_w), may play a decisive role. Table 20.1 shows the effect of heating on the protein content of meat samples. Herein, the data support the fact generally accepted that total protein increases as total moisture content decreases [30, 31]. The true retention (TR) values of proteins around 100 % suggest that proteins are not susceptible to migration due to coagulation or denaturation. Theoretically, the consumption of 100 g of raw bovine meat would be enough to supply the daily adult's requirement of amino acids. During cooking, aromatic amino acids (tryptophan, phenylalanine, and tyrosine) have been described to be particularly sensitive to oxidative processes causing a net loss of these amino acids [32]. Gatellier et al. [33] observed that heating at 60 °C had little influence on the aromatic amino acid levels while higher temperatures (100 and 140 °C) had a remarkable effect on the amino acid stability. Table 20.2 shows the effect of cooking on the amino acid contents of beef samples analyzed by our group. The contents of amino acids increase during cooking, except for histidine. This increase in amino acid contents may be due to the cooking loss. The TR values of amino acids tended to be lower in boiling and microwaving than in grilling. Since grilling proceeds at higher temperature and in the absence of water, it probably allowed for a high retention of amino acids.

Fat content and fatty acid composition of meat are of major importance for consumers due to its implications for human health. The specific guidelines for fat (20–35 % of caloric intake) and fatty acids in human nutrition were reviewed recently by the Food and Agriculture Organization of United Nations [34]. Among the recommendations, reducing the intake of saturated fatty acids

Table 20.2 Effect of cooking methods on amino acid content of beef (g/100 g)

Amino acid	Raw	Boiling	Microwaving	Grilling
Alanine	1.41	2.61 (125)	2.59 (117)	3.03 (137)
Arginine	0.86	1.49 (117)	1.65 (123)	1.66 (123)
Aspartic acid	1.81	3.47 (129)	3.10 (110)	4.02 (143)
Glutamic acid	3.22	5.46 (115)	5.07 (102)	6.50 (130)
Glycine	0.36	0.72 (134)	0.88 (145)	1.02 (180)
Histidine	0.65	ND	ND	0.21 (22.0)
Isoleucine	1.27	2.41 (128)	2.29 (116)	2.83 (142)
Leucine	2.06	3.75 (123)	3.75 (117)	4.49 (177)
Lysine	0.38	0.72 (125)	1.13 (189)	0.75 (127)
Methionine	0.25	0.51 (137)	0.53 (135)	0.44 (112)
Norleucine	0.33	0.56 (115)	0.51 (98.2)	0.79 (154)
Phenylalanine	0.80	1.30 (110)	1.57 (127)	1.67 (133)
Proline	0.44	0.78 (119)	0.87 (126)	1.00 (144)
Serine	1.50	2.32 (104)	2.58 (111)	2.64 (113)
Threonine	1.45	2.59 (120)	2.45 (109)	3.08 (136)
Tyrosine	0.30	0.57 (126)	0.61 (129)	0.63 (135)
Valine	1.50	2.51 (113)	2.44 (105)	2.94 (125)

Our values (unpublished results) with the following cooking conditions: boiling, 81 °C, 40 min; microwave, 750 W, 90 s; grilling, 225 °C, 30 min

*Data in the same row indicate contents and retention values (%) in parenthesis

ND not detected

(SFA; <10 % of caloric intake), cholesterol (<300 mg/day), and *trans* fatty acids (TFA; <1 %), as well as increasing the intake of *n*-3 polyunsaturated fatty acids (*n*-3 PUFA; 0.5–2 %) are particularly encouraged. The fatty acid content and composition of cooked meat, in contrast to those of raw meat, have been less studied. During cooking, fat content and composition of meat might change by different mechanisms such as oxidation, *cis–trans* isomerization, or hydrogenation, affecting the nutritional value of meat lipids. Post-slaughtering activities such as the trimming of visible fat can also greatly influence the amount of fat in meat. The effect of cooking on total fat in the selected meat species is shown in Table 20.1. Regarding beef samples, the values of total fat, in general, increase in cooked samples compared with raw beef control because of moisture loss through cooking [23, 24]. The TR values for total fat vary widely being higher for both microwave and grilled beef compared to boiling. This variability has been explained by the presence of unpredictable levels of subcutaneous and intermuscular fats, which liquefy during cooking, and absorption by the lean tissue leads to TR values higher than 100 % [35]. If only intramuscular fat is present, TR values of 100 % are expected, unless fat is partially degraded or lost to the cooking medium. In pork samples, Broncano et al. [26] reported that cooking does not change significantly the total fat content in meat samples. Similar results were obtained by others authors [30], excepting for the fried meat samples where they found an increase of the total lipid content although it is known that the water loss during frying produces an incorporation of cooking oil. Gerber et al. [36] found a relative increase in total fat of pork cuts (neck steak and belly) after cooking and trimming of visible fat, but when compared to the original fresh weight an absolute fat loss was observed. In addition, fat contents in raw chicken and duck breast were higher than those in cooked samples, except in poultry cooked by deep-frying, which can be explained by the dripping of fat during heating [37]. Great differences in total lipid contents were also detected in chicken breast cooked in oven by dry air and by air–steam [38].

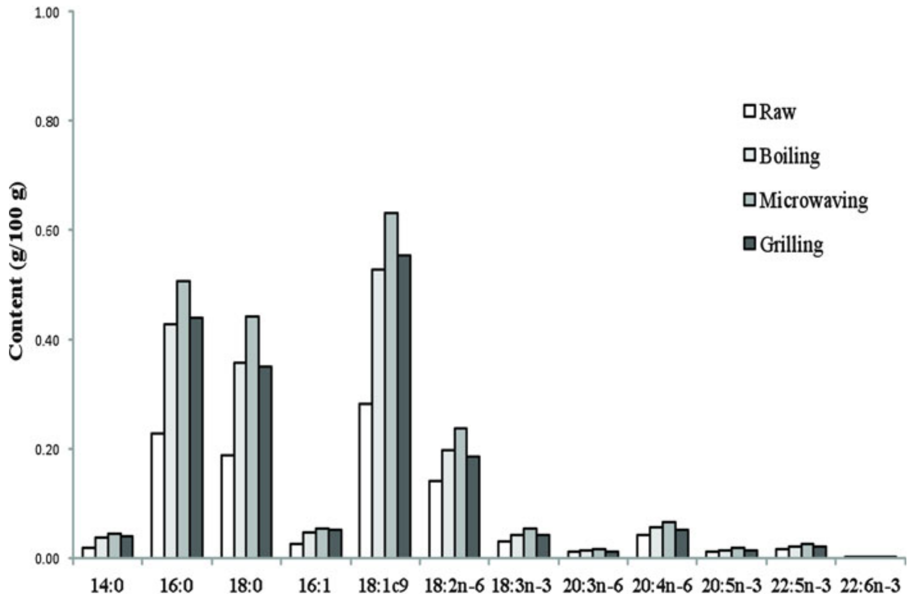

Fig. 20.2 Effect of cooking on the major beef fatty acids. Data are expressed as mean contents in raw and cooked state as reported by Alfaia et al. [24]

The effect of cooking on the fatty acid composition of meat varies among studies involving different animal species, which make it difficult to draw general conclusions [23, 24]. Increases in fatty acid concentrations in response to different cooking methods have been reported in trimmed beef and in restructured beef steaks [24, 30]. Figure 20.2 shows the effect of cooking methods on the major fatty acids of beef samples. Raw and cooked beef showed a clear predominance of oleic (18:1*c*9), followed, in decreasing order, by palmitic (16:0), stearic (18:0), and linoleic acids (18:2*n*-6). The cooking process increases the content, expressed as mg per 100 g of meat, of the individual beef fatty acids. Alfaia et al. [24] reported that the relative amounts (g/100 g total fatty acids) of 14:0, 16:0, 17:0, 18:0, and 18:1*c*9 were significantly higher in cooked beef samples than in the uncooked meat control while the percentages of 18:2*n*-6 and almost all *n*-6 PUFA decreased in cooked beef compared to raw meat.

By contrary, in Fig. 20.3, the content of the major fatty acids in grilled pork compared to raw samples decreases, probably due to lipid losses containing mainly triacylglycerols of adipose tissue [36].

Poultry raw and cooked samples show a similar predominance of the individual fatty acids as for beef and pork, being linoleic acid the third most predominant fatty acid instead of stearic acid. Both microwave heating and frying improve the amounts of the major fatty acids of chicken patties (Fig. 20.4) [28].

The partial sums of fatty acids in raw and cooked meat samples are listed in Table 20.3. The pattern of the sums of fatty acids reflects the values described for the major individual fatty acids of each group. The TR of *n*-6 and *n*-3 PUFA below or around 100 % suggests a little loss or oxidative degradation of these fatty acids during cooking. Concerning the different fatty acids groups, cooked beef samples show lower PUFA/SFA ratios close to the lowest nutritional recommended limit (0.45) [39]. Contrarily, Ono et al. [40] found higher PUFA/SFA ratio in cooked beef than in raw samples. According to the former authors, PUFA are less affected by heat since they are part of the membrane structure. The PUFA/SFA ratios in fried chicken patties increase, which may be considered as beneficial from the nutritional point of view. In addition, cooking not changes the values of the

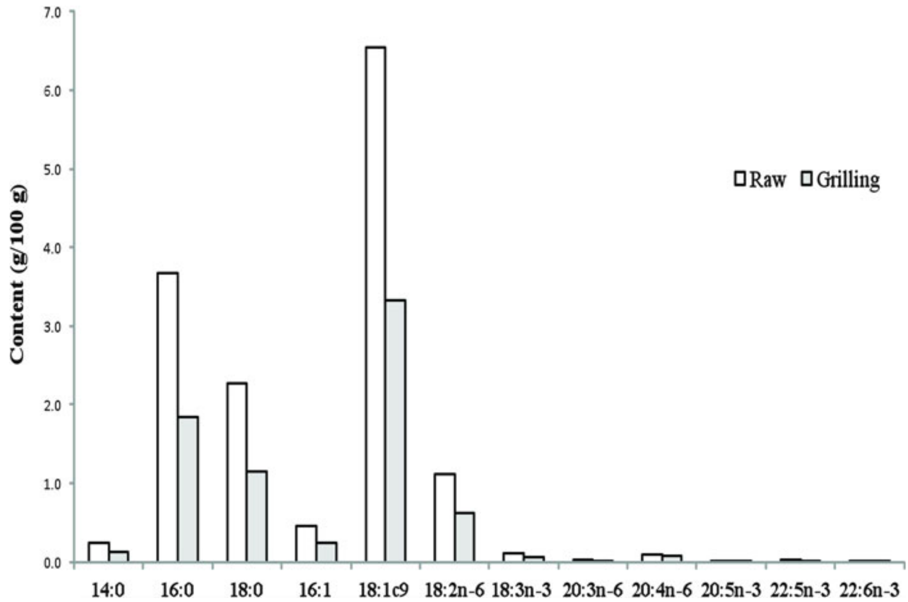

Fig. 20.3 Effect of cooking on the major pork fatty acids. Data are expressed as mean contents in raw and cooked state as reported by Gerber et al. [36]

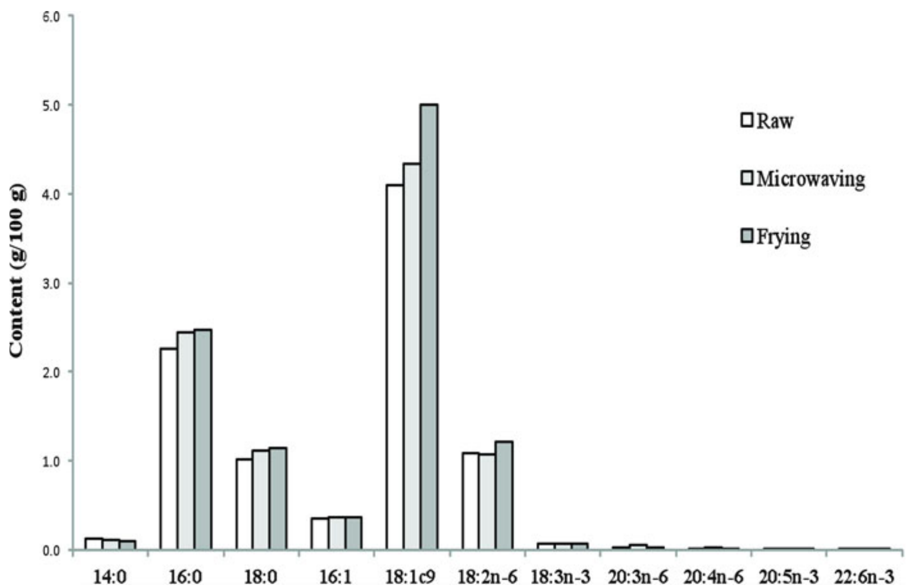

Fig. 20.4 Effect of cooking on the major fatty acids of chicken patties. Data are expressed as mean contents in raw and cooked state as reported by Echarte et al. [28]

n-6/*n*-3 ratio in beef samples. Echarte et al. [28] found that microwave heating did not modify the *n*-6/*n*-3 ratio but decreased the PUFA/SFA ratio in beef patties, whereas no change was observed in chicken patties. Cooking also induce a remarkable increase in cholesterol content of meat. Fat and cholesterol contents have been positively correlated in both raw and cooked meat [23]. In addition, the data shows higher total CLA content in cooked beef than in raw samples probably because of moisture loss [24]. The cooking methods with higher internal temperatures lead to

Table 20.3 Effect of cooking methods on partial sums of fatty acids and cholesterol contents of meat (g/100 g, except where noted) and their nutritional fatty acid ratios

Meat type/ nutrient	Raw	Roasting	Boiling	Microwaving	Grilling	Frying	Heat processing parameters	Reference
Beef								
$\sum$ SFA	0.456		0.854 (118)	1.04 (138)	0.866 (134)		BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	0.989^a; 2.79^b; 1.10^c; 1.44^d	1.62^a (103)	4.88^b (102)	1.78^c (104)	1.83^d (105)		RO, 180 °C; BO, 100 °C; MW, 700 W/10 min, 350 W/15 min + 350 W/5 min; GR, 220 °C/12 min	[23]
$\sum$ MUFA	0.351		0.646 (119)	0.772 (137)	0.681 (140)		BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	0.957^a; 2.71^b; 1.05^c; 1.39^d	1.56^a (102)	4.84^b (102)	1.69^c (104)	1.76^d (101)		RO, 180 °C; BO, 100 °C; MW, 700 W/10 min, 350 W/15 min + 350 W/5 min; GR, 220 °C/12 min	[23]
$\sum$ TFA*	34.0		61.6 (118)	79.1 (141)	66.4 (143)		BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
$\sum$ PUFA	0.254		0.352 (86.0)	0.427 (99.7)	0.333 (90.6)		BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	0.153^a; 0.281^b; 0.155^c; 0.158^d	0.240^a (91.1)	0.472^b (95.5)	0.248^c (99.4)	0.213^d (92.3)		RO, 180 °C; BO, 100 °C; MW, 700 W/10 min, 350 W/15 min + 350 W/5 min; GR, 220 °C/12 min	[23]
$\sum$ *n*-6 PUFA	0.196		0.275 (85.3)	0.328 (95.2)	0.255 (89.6)		BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	0.380^a; 0.240^b		0.210^a		0.140^b		BO, 1 h; GR, internal temperature 72 °C	[36]

∑ n-3 PUFA*	58.2		77.5 (86.8)	98.4 (102)	77.7 (95.9)	BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	200^{a}; 150^{b}		110^{a}		80^{b}	BO, 1 h; GR, internal temperature 72 °C	[36]
Total CLA*	4.5		8.6 (117)	8.7 (112)	7.8 (117)	BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
Cholesterol*	49^{a}; 55^{b}; 49^{c}; 48^{d}	80^{a} (99.4)	96^{b} (105)	72^{c} (94.8)	67^{d} (107)	RO, 180 °C; BO, 100 °C; MW, 700 W/10 min, 350 W/15 min + 350 W/5 min; GR, 220 °C/12 min	[23]
Ratios							
PUFA/SFA	0.65		0.46	0.45	0.42	BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	0.16^{a}; 0.13^{b}		0.21^{a}		0.18^{b}	BO, 1 h; GR, internal temperature 72 °C	[36]
n-6/n-3	6.41		6.47	6.14	5.91	BO, 80 °C/60 min; MW, 750 W/210 s; GR, 225 °C/30 min	[24]
	1.88^{a}; 1.63^{b}		1.97^{a}		1.72^{b}	BO, 1 h; GR, internal temperature 72 °C	[36]
Pork							
∑ SFA	6.39^{a}; 7.27^{b}				3.23^{a}; 4.27^{b}	GR, internal temperature 72 °C	[36]
∑ MUFA	7.21^{a}; 8.70^{b}				3.68^{a}; 4.14^{b}	GR, internal temperature 72 °C	[36]
∑ PUFA	1.63^{a}; 2.07^{b}				0.930^{a}; 0.780^{b}	GR, internal temperature 72 °C	[36]
∑ n-6 PUFA	1.28^{a}; 1.57^{b}				0.760^{a}; 0.600^{b}	GR, internal temperature 72 °C	[36]
∑ n-3 PUFA*	180^{a}; 270^{b}				100^{a}; 90.0^{b}	GR, internal temperature 72 °C	[36]
Ratios							
PUFA/SFA	0.27^{a}; 0.29^{b}				0.34^{a}; 0.20^{b}	GR, internal temperature 72 °C	[36]
n-6/n-3	7.02^{a}; 5.89^{b}				7.89^{a}; 6.96^{b}	GR, internal temperature 72 °C	[36]

(continued)

Table 20.3 (continued)

Meat type/ nutrient	Raw	Roasting	Boiling	Microwaving	Grilling	Frying	Heat processing parameters	Reference
Poultry								
∑ SFA	3.76			3.81		3.44	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
∑ MUFA	4.86			5.17		5.84	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
∑ PUFA	1.29			1.29		1.40	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
∑ *n*-6 PUFA	1.20			1.20		1.31	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
∑ *n*-3 PUFA*	80.0			80.0		90.0	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
Cholesterol*	60.9			73.3		75.5	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
Ratios								
PUFA/SFA	1.63			1.69		2.09	MW, 900 W/3 min; FR, 180 °C/3 min	[28]
n-6/*n*-3	15.0			15.0		14.6	MW, 900 W/3 min; FR, 180 °C/3 min	[28]

BO boiling, *MW* microwaving, *GR* grilling, *FR* frying, *RO* roasting, *CLA* conjugated linoleic acid, *MUFA* monounsaturated fatty acids, *PUFA* polyunsaturated fatty acids, *SFA* saturated fatty acids, *TFA trans* fatty acids

Data in the same row indicate contents and retention values (%) in parenthesis. The same letters (abcd) correspond to the contents of the same meat in raw and cooked state

†Our data (unpublished results)

*expressed as mg/100 g

the highest CLA concentrations, probably owing to the higher cooking losses. The TR values for total CLA content are in line with the TR values obtained for total lipids. Although CLA has been described to be more sensitive than linoleic acid to oxidation, and even to isomerization, minor changes in the CLA isomeric profile of beef have been reported throughout cooking [24].

The energy value of foods is one of the most relevant nutritional factors for consumers. Reductions in gross caloric value due to cooking have been calculated by comparing the protein, lipid, and carbohydrate contents of raw and cooked samples, and multiplying these values by the typically caloric conversion factors of 4, 9, and 4 kcal/g, respectively [41]. However, it is not well documented whether the negative effects of cooking on the gross caloric value of meat due to fat loss are outweighed by potential positive effects of cooking on the net energy values of the residual fat and protein [4]. Some mechanisms have been suggested by which cooking might increase the energy available from meat, including increasing food intake through positive effects on palatability, rendering proteins more digestible through denaturation, lowering the cost of digestion through food softening, and reducing immune up-regulation by eliminating foodborne pathogens [7]. Therefore, more controlled studies of energy balance on cooked and raw meat-rich diets will be required in order to address the potential energetic effects of cooking meat [7]. The energy values of raw and cooked samples in different meat species are shown in Table 20.1. In general, the energy content in beef and pork increases from raw to cooked state. In restructured cooked beef, the energy values were slightly lowest compared to raw samples, except in pan-frying, probably because of the highest fat content [30].

Meat is also an excellent source of vitamins and trace elements in human nutrition, particularly of vitamins and minerals, such as iron, selenium, vitamins A, B_{12} (cyanocobalamin), and folic acid. Water- (B complex and vitamin C) and fat-soluble vitamins (A, D, E, and K) are a broad group of essential components required for the normal growth, self-maintenance, and functioning of human and animal bodies. No single food contains all the vitamins, so, a balanced and varied diet is necessary for an adequate intake of these compounds. Vitamin E is a potent fat-soluble antioxidant. The only form of the tocopherols and tocotrienols that has vitamin E activity is α-tocopherol. The requirements for vitamin E, in beef cattle, are dependent on the quantities of dietary PUFA, other antioxidants, S-containing amino acids, and selenium. Dietary vitamin E supplementation of beef cattle has been reported to increase the skeletal muscle concentrations of α-tocopherol [42]. In addition, red meat provides the essential minerals to human nutrition as well as trace inorganic elements, which our body needs in small amounts, usually obtained by consuming a conventional mixed diet. A daily intake of 100 g of meat can supply 100 % of vitamin A and up to 50 % of the recommended daily allowance for vitamins B_1 (thiamin), B_2 (riboflavin), B_6 (pyridoxine), B_{12}, iron, zinc, and selenium [42]. However, the vitamin and mineral compositions of meat change with breed, age of animals, muscle type, rearing, diet, and processing.

Vitamins are the first and foremost sensitive food component influenced by heating. The main factors responsible for vitamins losses during cooking include cooking time, temperature, and cooking methods. In addition, vitamin degradation also depends on pH, metals, other vitamins and enzymes, oxygen, light, and water solubility. The most heat-sensitive vitamins are A, D, E, and β-carotene within fat-soluble vitamins, and C, B_1 and B_2, biotin, nicotinic and pantothenic acids among the water-soluble vitamins. A great loss of B_1, B_2, and B_3 vitamins has been reported in beef roasts cooked by microwave heating than by conventional roasting [43]. In contrast, chicken breasts cooked by microwaves have greater retention of vitamin B_6 compared with meat cooked by roasting in a conventional oven [44]. Vitamin concentrations in raw and cooked meats are shown in Table 20.4. The inconsistency on vitamin contents mainly reflected the water loss (and possibly the lipid loss) of meat during cooking [45]. B_1 vitamin is undoubtedly the most susceptible to thermal degradation while B_2 and B_3 vitamins being relatively stable to heat. The contents of retinol and α-tocopherol in raw and cooked meats reinforce that fat-soluble vitamins

Table 20.4 Effect of cooking methods on vitamin contents of meat (μg/100 g, except where noted)

Meat type/nutrient	Raw	Roasting	Boiling	Microwaving	Grilling	Frying	Heat processing parameters	Reference
Beef								
Cyanocobalamin (B_{12})	0.90^{a}; 1.17^{b}; 2.04^{c}	0.41^{a}			0.21^{b}	0.24^{c}	RO, 240 °C/50 min; FR, fire 4 min; GR, 380 °C/90 s	[45]
Thiamin (B_1)*	81.1^{a}; 84.0^{b}		ND^{a}		21.6^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
	10.0; 20.0; 50.0; 80.0; 80.0					ND	Iron-free pan with medium heat until red color disappeared	[12]
Riboflavin (B_2)	182^{a}; 163^{b}		30.2^{a}		82.5^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
	90.0^{a}; 100^{b}; 120^{c}; 120^{d}; 170^{e}					50.0^{a}; 100^{b}; 40.0^{c}; 70.0^{d}; 70.0^{e}	Iron-free pan with medium heat until red color disappeared	[12]
Niacin (B_3)*	4.6^{a}; 4.5^{b}		1.61^{a}		2.7^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
	5.00^{a}; 5.00^{b}; 5.50^{c}; 5.70^{d}; 6.50^{e}					3.00^{a}; 3.20^{b}; 3.30^{c}; 3.30^{d}; 4.24^{e}	Iron-free pan with medium heat until red color disappeared	[12]
α-Tocopherol (E)	342^{a}; 458^{b}		275^{a}		392^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
Retinol (A)	2.6^{a}; 10.9^{b}		1.5^{a}		7.1^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
Pork								
Cyanocobalamin (B_{12})	0.47^{a}; 0.25^{b}	0.41^{a}				0.30^{b}	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]

Thiamin (B_1)	849[a]; 879[b]		206[a]; 229[b]		GR, internal temperature 72 °C	[36]
	600[a]; 900[b]; 900[c]			150[a]; ND[b]; 210[c]	Iron-free pan with medium heat until red color disappeared	[12]
	730[a]; 840[b]	720[a]		820[b]	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Riboflavin (B_2)	163[a]; 222[b]		83.4[a]; 103[b]		GR, internal temperature 72 °C	[36]
	100[a]; 130[b]; 150[c]			60.0[a]; 90.0[b]; 70.0[c]	Iron-free pan with medium heat until red color disappeared	[12]
	130[a]; <50[b]	150[a]		<50[b]	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
Niacin (B_3)*	4.1[a]; 4.5[b]		2.1[a]; 2.21[b]		GR, internal temperature 72 °C	[36]
	4.20[a]; 5.20[b]; 5.70[c]			3.20[a]; 3.30[b]; 3.30[c]	Iron-free pan with medium heat until red color disappeared	[12]
	8.8[a]; 9.4[b]	10[a]		6.3[b]	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]
α-Tocopherol (E)	467[a]; 457[b]		389[a]; 407[b]		GR, internal temperature 72 °C	[36]
	180[a]; 210[b]	100[a]		260[b]	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[27]

(continued)

Table 20.4 (continued)

Meat type/nutrient	Raw	Roasting	Boiling	Microwaving	Grilling	Frying	Heat processing parameters	Reference
Retinol (A)	6.5[a]; 9.5[b]				5.5[a]; 7.0[b]		GR, internal temperature 72 °C	[36]
	<5	<5				<5	RO, 180 °C (loin roast); FR, medium-high (loin steak)	[12]
Poultry								
Thiamin (B_1)	20.0; 40.0					ND	Iron-free pan with medium heat until red color disappeared	[12]
	90.0	ND		80.0			RO and MW to core temperature of 88 and 93 °C, respectively	[31]
Riboflavin (B_2)	30.0[a]; 60.0[b]					10.0[a]; 30.0[b]	Iron-free pan with medium heat until red color disappeared	[12]
	160	30.0		100			RO and MW to core temperature of 88 and 93 °C, respectively	[31]
Niacin (B_3)*	7.20[a]; 8.00[b]					5.30[a]; 5.00[b]	Iron-free pan with medium heat until red color disappeared	[12]
	5.90	5.40		6.50			RO and MW to core temperature of 88 and 93 °C, respectively	[31]
Retinol (A)	<5	<5		<5			RO and MW to core temperature of 88 and 93 °C, respectively	[31]

The same letters (abcde) correspond to the contents of the same meat in raw and cooked state
BO boiling, *MW* microwaving, *GR* grilling, *FR* frying, *RO* roasting
*expressed as mg/100 g

are less heat-labile than water-soluble vitamins. Nevertheless, fat-soluble vitamins are also susceptible to degradation in the presence of oxygen and high temperatures. Concerning cooking methods, the culinary practices that make no use of water (e.g., grilling and microwave) allow for a greater retention of vitamins in meat.

Heat treatment also leads to a decrease in the nutritional value of foods mainly due to mineral losses. Cooking methods involving water, such as steaming and boiling, seem to affect deeply the mineral content of meat. However, the magnitude of the mineral loss is largely dependent on the cooking medium and use of drip [42]. The effect of cooking on the contents of trace elements in beef, pork, and poultry meats is presented in Table 20.5. Cooked meat compared to raw meat shows higher iron concentration due to the moisture losses during heating [46]. The contents of potassium, sodium, magnesium, and phosphorus decrease in cooked beef and pork. Few studies have been performed on mineral retention of cooked meats, but all agree that zinc, copper, and iron are the most stable minerals during cooking [42].

Cooking loss includes a combination of liquid and soluble matters lost from meat being water the main component [22]. Generally, the cooking loss starts to develop around 40 °C. Variations in cooking loss can be associated to changes in meat structure caused by increasing temperature during cooking. The sarcoplasmic and myofibrillar proteins begin to denature at 40–50 °C. Further protein denaturation and protein coagulation occur, which lead to myofilament shrinkage and textural toughening at 45 °C. The cooking losses of beef at low cooking temperatures (50–58 °C) are linked to the denaturation of α-actinin and myosin while losses observed in the range of 58–65 °C are mainly attributed to collagen contraction [21]. As shown by Lawrence et al. [47] beef samples cooked under the same conditions to the same final internal temperature may differ in cooking loss due to moisture, fat, and collagen contents of raw material. In addition, different heating methods could also result in different cooking losses. Table 20.1 shows the cooking losses in meat samples after cooking. Alfaia et al. [24] reported higher cooking losses in beef cooked by microwaving followed by boiling and grilling. Beef and pork patties cooked by microwave oven exhibit the highest cooking loss [25, 30].

Oxidative Changes

Meat composition, and its physicochemical properties, undergoes significant changes such as oxidative reactions through cooking. However, these changes differ with muscle location, animal species, and cooking time and temperature. The time/temperature cycle during cooking deeply affects free fatty acid content and enzyme activity. The lipolytic processes liberate free fatty acids, which are the substrates for subsequent oxidative reactions and, therefore, leading to the development of rancidity and loss of shelf life, palatability, functionality, and nutritional quality of foods.

Lipid oxidation is the most important reaction responsible for deterioration of food quality, especially meats, and for the development of oxidized flavors, generally, called warmed-over flavor. It is widely acknowledged that a higher unsaturation index in meat may affect its oxidative stability, since unsaturated fatty acids are more prone to oxidation. Numerous analytical methods have been developed for assessing lipid oxidation in foods. Among them, the peroxide value and conjugated dienes analysis are used to measure primary oxidative changes whereas thiobarbituric acid reactive substances and volatile compound determinations to evaluate secondary lipid oxidation changes. Lipid oxidation of muscle foods occurs in the following order, poultry (chicken and turkey) > pork > beef > lamb [48]. It has been shown that roasting, which uses high temperatures during a long time, produces an increase of lipid oxidation compared to other heating methods [49]. However, microwave treatment using short time also enhances lipid oxidation. This high oxidation after microwave cooking was explained by a decrease of PUFA from phospholipids suggesting an increase of secondary oxidation products originated from these fatty acids [49]. In addition, an increase in

Table 20.5 Effect of cooking methods on trace element contents of meat (mg/100 g)

Meat type/ nutrient	Raw	Roasting	Boiling	Microwave	Grilling	Frying	Heat processing parameters	Reference
Beef								
Iron	1.42[a]; 1.61[b]		1.74[a]		1.81[b]		BO, 1 h; GR, internal temperature 72 °C	[36]
	1.80[a]; 1.91[b]; 1.93[c]; 1.95[d]; 2.37[e]					3.50[a]; 2.86[b]; 3.11[c]; 3.46[d]; 3.38[e]	Iron-free pan with medium heat until red color disappeared	[12]
	1.69		2.49 (87.8)	2.13 (78.9)	3.14 (105)		BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
Zinc	4.65[a]; 4.72[b]		5.37[a]		4.83[b]		BO, 1 h; GR, internal temperature 72 °C	[36]
	3.94[a]; 4.01[b]; 4.09[c]; 4.29[d]; 4.75[e]					5.54[a]; 5.62[b]; 5.74[c]; 7.23[d]; 9.44[e]	Iron-free pan with medium heat until red color disappeared	[12]
	3.44		5.39 (93.2)	4.89 (88.9)	5.79 (97.0)		BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
Magnesium	18		13		16		BO, 1 h; GR, internal temperature 72 °C	[36]
	12.9		20.4 (93.4)	28.5 (138)	30.1 (135)		BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
Potassium	309[a]; 337[b]		137[a]		253[b]		BO, 1 h; GR, internal temperature 72 °C	[36]
	289		129 (26.5)	316 (68.4)	157 (31.6)		BO, 81 °C/40 min; MW, 750 W/90 s; GR, 225 °C/30 min	†
Copper	0.04[a]; 0.05[b]; 0.05[c]; 0.07[d]; 0.09[e]					0.07[a]; 0.10[b]; 0.08[c]; 0.08[d]; 0.11[e]	Iron-free pan with medium heat until red color disappeared	[12]
Calcium	7.0[a]; 5.4[b]		9.0[a]		3.7[b]		BO, 1 h; GR, internal temperature 72 °C	[36]
Sodium	65[a]; 45[b]		36[a]		33[b]		BO, 1 h; GR, internal temperature 72 °C	[36]

Phosphorus	162^{a}; 168^{b}		107^{a}	136^{b}		BO, 1 h; GR, internal temperature 72 °C	[36]
Pork							
Iron	0.83^{a}; 0.98^{b}			0.91^{a}; 1.07^{b}		GR, internal temperature 72 °C	[36]
	0.42^{a}; 0.49^{b}; 0.70^{c}				0.68^{a}; 0.79^{b}; 1.09^{c}	Iron-free pan with medium heat until red color disappeared	[12]
Zinc	2.89^{a}; 2.90^{b}			2.14^{a}; 2.72^{b}		GR, internal temperature 72 °C	[36]
	0.98^{a}; 1.55^{b}; 2.28^{c}				1.79^{a}; 2.54^{b}; 4.17^{c}	Iron-free pan with medium heat until red color disappeared	[12]
Magnesium	20^{a}; 17^{b}			15^{a}; 15^{b}		GR, internal temperature 72 °C	[36]
Potassium	314^{a}; 335^{b}			224^{a}; 230^{b}		GR, internal temperature 72 °C	[36]
Copper	0.04^{a}; 0.05^{b}; 0.07^{c}				0.06^{a}; 0.07^{b}; 0.12^{c}	Iron-free pan with medium heat until red color disappeared	[12]
Calcium	8.0^{a}; 4.6^{b}			6.8^{a}; 4.0^{b}		GR, internal temperature 72 °C	[36]
Sodium	66^{a}; 51^{b}			48^{a}; 35^{b}		GR, internal temperature 72 °C	[36]
Phosphorus	171^{a}; 174^{b}			129^{a}; 132^{b}		GR, internal temperature 72 °C	[36]
Poultry							
Iron	0.40^{a}; 0.50^{b}	0.58^{a}; 0.79^{b}				RO, 180 °C/50 min	[12]
Zinc	0.65^{a}; 1.08^{b}	0.90^{a}; 1.24^{b}				RO, 180 °C/50 min	[12]
Copper	0.05^{a}; 0.06^{b}	0.06^{a}; 0.09^{b}				RO, 180 °C/50 min	[12]

Data in the same row indicate contents and retention values (%) in parenthesis. The same letters (abcde) correspond to the contents of the same meat in raw and cooked state
BO boiling, *MW* microwaving, *GR* grilling, *FR* frying, *RO* roasting
†Our values (unpublished results)

lipid oxidation could be explained by the loss of antioxidant activity, which could drop by up to 80 % after heat treatment [50]. Nevertheless, lipid oxidation can be minimized either by dietary supplementation with antioxidants or by adding directly antioxidants to meat during processing. Therefore, the oxidative stability of meats depends upon the balance and interaction between endogenous anti- and pro-oxidant substances. Cholesterol may also undergo autoxidation during cooking of meat and meat products. Cholesterol oxidation products (COPs) in food exhibiting mutagenic, carcinogenic, and cytotoxic properties are strongly dependent on cholesterol concentrations.

The susceptibility of meat to oxidative degradation has been traditionally focused on lipids, mainly PUFA and cholesterol, whereas the impact of protein oxidation on meat quality is an issue of recent interest [29, 51]. Cooking plays a key role in protein oxidation because it affects the physicochemical state of proteins and the bioavailability of their amino acids [21, 52]. The main changes in proteins include denaturation (loss of their native tertiary structure) and hydrolytic degradation (proteolysis) by endogenous and/or exogenous enzymes. Gatellier et al. [51] investigated the oxidation of three classes of amino acids (basic amino acids, aromatic amino acids, and cysteine), which can negatively affect the nutritional quality of meat by inducing cross-linking and aggregation of proteins. The formation of carbonyl groups from specific amino acids, thiol oxidation, and aromatic hydroxylation have been reported as the most remarkable chemical modifications in oxidized proteins, being used as protein oxidation indicators in both biological and food systems [29]. The oxidation of amino acid residues generates oxidation products, which can induce structural and physical changes such as protein aggregation, hydrophobicity changes, and a decrease in protein solubility [52]. Santé-Lhoutellier et al. [52] showed that the aggregation of meat proteins during cooking is associated to the increase of protein surface hydrophobicity. Even if some of these aggregates could be broken down by enzymatic reaction, it has been demonstrated that heating could reduce meat protein susceptibility to digestive proteases, which can negatively influence the nutritional value of meat [33].

Sensory Properties and Eating Quality

Cooking influences several attributes of eating quality through multiple physicochemical changes. It is well known that cooking time, temperature, and cooking method have a great effect on eating quality indicators including tenderness, flavor, color, and overall acceptability by the onset of lipid and protein oxidation [22]. Consequently, maintaining a balance of multiple quality attributes is a requisite in the processing of cooked meat [53].

Tenderness is the most important meat quality characteristic that affects ultimate palatability and, thus, dictates consumer acceptance. During meat cooking, the time–temperature relationship influences the meat texture parameters (cohesiveness, springiness, chewiness, hardness, elasticity). Heat-induced alterations of primary structure components in the muscle tissue, mainly collagen and myofibrillar proteins, are associated with changes in meat tenderness. Heating can strengthen myofibrillar protein networks, which leads to toughening, but at the same time may solubilize connective tissue leading to tenderization [53]. Therefore, cooking can be responsible for either tenderization or toughening of meat with the net effect being dependent on the composition of muscle, and the time/temperature combinations employed. In addition, cooking method is also an important factor affecting the tenderness of meat. Cooking in water seems to enhance meat tenderness than that of microwave heating due to the higher loss of water from muscle during microwave heating [30].

Flavor represents a very complex sensory attribute of meat palatability that influences the overall eating quality. Cooking enhances the flavor of foods. Meat flavor is primarily generated through the cooking process being the Maillard reaction and lipid oxidation the major pathways to produce flavor components. The food industry makes extensive use of heat treatment to induce the Maillard reaction to generate hundreds of

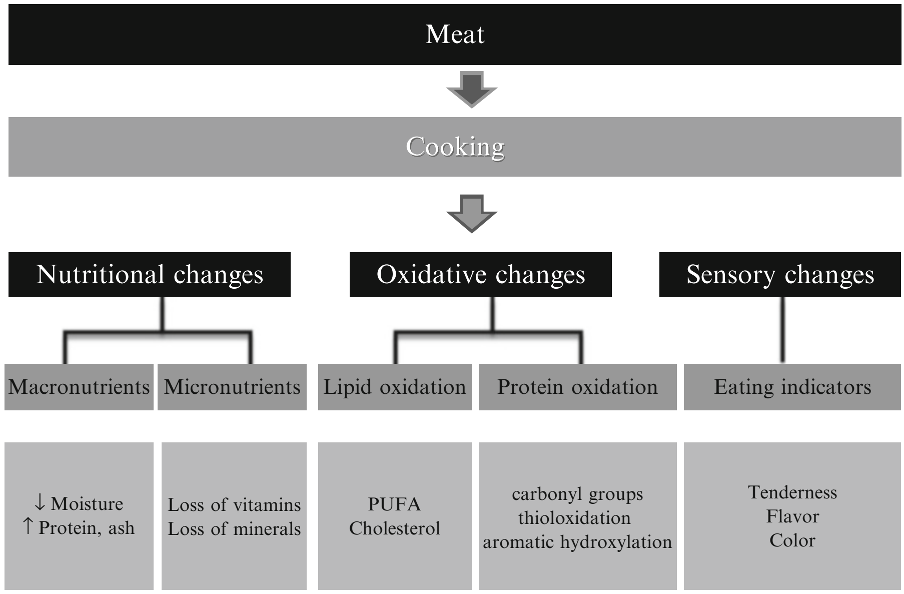

Fig. 20.5 Main changes induced by cooking on meat traits

different flavor compounds and aromas. The Maillard reaction, which is known to be highly pH-, temperature-, moisture-, and reactant-dependent, at high temperatures produces brown pigments, called melanoidins, and aromatic compounds, as sulfur-containing amino acids and reducing sugars recombine. Even though, this reaction may be desirable in generating characteristic flavors in cooked meat, the degradation of proteins into smaller peptides and free amino acids may contribute to sour, bitter, and umami taste of meat products. Furthermore, mild conditions of cooking meats (at temperatures between 70 and 80 °C) lead to the disruption of muscle membrane structure and facilitate the interaction of lipid oxidation catalysts with unsaturated fatty acids, resulting in the production of free radicals and propagation of rancid off-flavors [54].

Color is also an important quality attribute for meat industry and consumers. Meat color is largely determined at the meat surface by the relative amounts of the three forms of myoglobin, specifically deoxymyoglobin, oxymyoglobin, and metmyoglobin. Among pH, fat content, and heating, the chemical state of myoglobin is the key factor for meat color changes. During cooking, these forms of myoglobin interconvert, and meat becomes progressively browner affecting the product's final appearance [55].

Figure 20.5 summarizes the main changes induced by thermal processing in meat quality described in this chapter.

Safety Concerns of Meat Cooking

Cooking is a critical process to assure microbiological and chemical safety of foods. The main concerns associated with cooking involve the formation of potential carcinogens in food such as heterocyclic aromatic amines (HAAs), polycyclic aromatic hydrocarbons (PAHs), dioxin-like polychlorinated biphenyls (dl-PCBs) compounds, *N*-nitrosamines, and COPs. HAAs, a potent class of carcinogens, are common pyrolysis products formed when muscle meat is cooked using high temperature methods. To date, more than 25 highly mutagenic HAAs have been found in cooked meats, including beef, poultry, fish, and pork [56]. Cooking methods such as frying, roasting, grilling and boiling, and other high temperature cooking are known to yield high concentrations of HAAs. The effect of microwave on HAAs formation in meat is controversial and remains unclear. Some studies have suggested that microwave pretreatment before frying may be a way to minimize the formation of HAAs due to the loss of precursors by leakage [56]. Likewise, the effect of cooking on the levels of some PAHs and dl-PCBs in foodstuffs (meat, fish, and dairy products) has also been investigated [57]. Cooking can also generate PAHs through grilling and smoking of food. The presence of these chemical contaminants in cooked foods is still a matter of concern with negative health outcomes. A possible role of HAAs plus PAHs and other harmful compounds (e.g., acrylamide, high fat, salt intake) in human cancer etiology has been documented [58]. High intake of red and processed meat has been associated with increased risk of colorectal cancer. The evidence that red and processed meat influences colorectal carcinogenesis was judged convincing in the 2007 World Cancer Research Fund report. Ongoing studies are investigating the associations between meat intake, cooking methods, and cancer risk [59].

Conclusions and Future Perspectives

This chapter highlights the effect of cooking on meat composition, which is quite complex, depending not only on the different cooking methods but also on the product type and its fatness and degree of doneness. In general, cooking promotes remarkable increases in the concentration of fat, protein, and ash as moisture content decreases. In addition, the data herein reported shows distinct fatty acid composition related to different meat species, cooking techniques, and heating parameters. Finally, vitamins and trace elements are the most heat-sensitive nutrients, affecting mainly B vitamins. The cooking practices involving higher temperatures and longer times allowed for low retention values of these essential compounds reflecting the water loss of meat during heating. Moreover, heat treatment induces protein and lipid degradation that have a strong impact on meat quality. However, the heterogeneity of studies on meat makes hard to draw general conclusions.

Forthcoming research regarding the role of cooking methods and control of variables should be focused on the proper time–temperature parameters relative to a specific food, minimizing nutritional losses and reducing the potential carcinogenic exposure to chemical contaminants. In summary, cooking time and temperature combinations must represent an acceptable compromise between sensory and nutritional quality, safety, and energy use. The maintenance of meat quality throughout cooking is of great interest as meat represents an important part of a balanced and varied diet.

References

1. Troy DJ, Kerry JP. Consumer perception and the role of science in the meat industry: review. Meat Sci. 2010;86:214–26.
2. Perelló G, Martí-Cid R, Castell V, et al. Influence of various cooking processes on the concentrations of PCDD/PCDFs, PCBs and PCDEs in foods. Food Control. 2010;21:178–85.
3. Serra-Majem L, Ribas L, Ngo J, et al. Food, youth and the Mediterranean diet in Spain. Development of KIDMED, Mediterranean Diet Quality Index in children and adolescents. Public Health Nutr. 2004;7:931–5.
4. Carmody RN, Wrangham RW. The energetic significance of cooking. J Hum Evol. 2009;57:379–91.
5. Arnoldi A. Thermal processing and food quality: analysis and control. In: Richardson P, editor. Thermal technologies in food processing. Cambridge: Woodhead CRC; 2001. p. 138–54.
6. Wills CTM, de Witt AM, Sigfusson H, et al. Effect of cooking method and ethanolic tocopherol on oxidative stability and quality of beef patties during refrigerated storage. J Food Sci. 2006;71:109–14.
7. Zhao Y, Flugstad B, Kolbe E, et al. Using capacitative (radio frequency) dielectric heating in food processing and preservation: a review. J Food Process Eng. 2000;23:25–55.
8. Raner KD, Strauss CR, Vyskoc F, et al. A comparison of reaction kinetics observed under microwave irradiation and conventional heating. J Org Chem. 1993;58:950–3.
9. Zell M, Lyng JG, Cronin DA, et al. Ohmic cooking of whole beef muscle—evaluation of the impact of a novel rapid ohmic cooking method on product quality. Meat Sci. 2010;86:258–63.
10. Daniel CR, Cross AJ, Koebnick C, et al. Trends in meat consumption in the USA. Public Health Nutr. 2011;14:575–83.

11. AGRI. DG AGRI short term outlook, February 2012; 2012. http://epp.eurostat.ec.europa.eu
12. Lombardi-Boccia G, Lanzi S, Aguzzi A. Aspects of meat quality: trace elements and B vitamins in raw and cooked meats. J Food Compos Anal. 2005;18: 39–46.
13. Prates JAM, Bessa RJB. *Trans* and *n*-3 fatty acids. In: Nollet LM, Toldrá F, editors. Handbook of muscle foods analysis. New York: CRC Press; 2009. p. 399–417.
14. Rinaldi M, Chiavaro E, Massini R. Real-time estimation of slowest heating point temperature and residual cooking time by coupling multipoint temperature measurement and mathematical modelling: application to meat cooking automation. Food Control. 2012;23:412–8.
15. US United States Department of Agriculture Food Safety and Inspection Service. USDA table of nutrient retention factors. Release 6. Beltsville, MD: U.S. Nutrient Data Laboratory, Department of Agriculture; 2007.
16. FDA Food Code. Recommendations of the United States Public Health Service, Food and Drug Administration. Alexandria, VA: National Technical Information Service; 2009.
17. Toldrá F. Biochemistry of meat and fat. In: Toldrá F, editor. Handbook of fermented meat and poultry. 1st ed. Oxford: Blackwell; 2007. p. 51–8.
18. Palka K, Daun H. Changes in texture, cooking losses, and myofibrillar structure of bovine M. semitendinosus during heating. Meat Sci. 1999;51:237–43.
19. Davey CL, Gilbert KV. Temperature-dependent cooking toughness in beef. J Sci Food Agric. 1974;25: 931–8.
20. Goñi SM, Salvadori VO. Prediction of cooking times and weight losses during meat roasting. J Food Eng. 2010;100:1–11.
21. Tornberg E. Effects of heat on meat proteins—implications on structure and quality of meat products. Review. Meat Sci. 2005;70:493–508.
22. Heymann H, Hedrick HB, Karrasch MA. Sensory and chemical characteristics of fresh pork roasts cooked to different centre temperatures. J Food Sci. 1990;55: 613–7.
23. Badiani A, Stipa S, Bitossi F, et al. Lipid composition, retention and oxidation in fresh and completely trimmed beef muscles as affected by common culinary practices. Meat Sci. 2002;60:169–86.
24. Alfaia CMM, Alves SP, Lopes AF, et al. Effect of cooking methods on fatty acids, conjugated isomers of linoleic acid and nutritional quality of beef intramuscular fat. Meat Sci. 2010;84:769–77.
25. Danowska-Oziewicz M. The influence of cooking methods on the quality of pork patties. J Food Process Preserv. 2009;33:473–85.
26. Broncano JM, Petrón MJ, Parra V, et al. Effect of different cooking methods on lipid oxidation and formation of free cholesterol oxidation products (COPs) in *Latissimus dorsi* muscle of Iberian pigs. Meat Sci. 2009;83:431–7.
27. Greenfield H, Arco J, Barnes JA, et al. Nutrient composition of Australian retail pork cuts 2005/2006. Food Chem. 2009;117:721–30.
28. Echarte M, Ansorena D, Astiasarán I. Consequences of microwave heating and frying on the lipid fraction of chicken and beef patties. J Agric Food Chem. 2003;51:5941–5.
29. Estévez M. Protein carbonyls in meat systems: a review. Meat Sci. 2011;89:259–79.
30. Serrano A, Librelotto J, Cofrades S, et al. Composition and physicochemical characteristics of restructured beef steaks containing walnuts as affected by cooking method. Meat Sci. 2007;77:304–13.
31. Kumar S, Aalbersberg B. Nutrient retention in foods after earth-oven cooking compared to other forms of domestic cooking. J Food Compos Anal. 2006;19: 302–10.
32. Davies KJA, Delsignore ME, Lin SW. Protein damage and degradation by oxygen radicals. II. Modification of amino acids. J Biol Chem. 1987;262:9902–7.
33. Gatellier P, Kondjoyan A, Portanguen S, et al. Determination of aromatic amino acid content in cooked meat by derivative spectrophotometry: implication in nutritional quality of meat. Food Chem. 2009;114:1074–8.
34. Burlingame B, Nishida C, Uauy R, et al. Fats and fatty acids in human nutrition. Report of a Joint FAO/WHO Expert Consultation, November 2008. Ann Nutr Metab. 2009;55:1–308.
35. Bragagnolo N, Rodriguez-Amaya DB. New data on the total lipid, cholesterol, and fatty acid composition of raw and grilled beef *longissimus dorsi*. Arch Latinoam Nutr. 2003;53:312–9.
36. Gerber N, Scheeder MRL, Wenk C. The influence of cooking and fat trimming on the actual nutrient intake from meat. Meat Sci. 2009;81:148–54.
37. Liao GZ, Wang GY, Xu XL, et al. Effect of cooking methods on the formation of heterocyclic aromatic amines in chicken and duck breast. Meat Sci. 2010;85: 149–54.
38. Barbanti D, Pasquini M. Influence of cooking conditions on cooking loss and tenderness of raw and marinated chicken breast meat. LWT. 2005;38: 895–901.
39. British Department of Health. Nutritional aspects of cardiovascular disease. Report on health and social subjects No. 46. London: HMSO; 1994.
40. Ono K, Berry BW, Paroczay E. Contents and retentions of nutrients in extra lean, lean and regular ground beef. J Food Sci. 1985;50:701–6.
41. Merrill AL, Watt BK. Energy value of foods, basis and derivation (revision), Agriculture handbook, vol. 74. Washington, DC: US Department of Agriculture; 1973.
42. Liu Q, Lanari MC, Schaefer DM. A review of dietary vitamin E supplementation for improvement of beef quality. J Anim Sci. 1995;73:3131–40.
43. Thomas MH, Brenner S, Eaton A, et al. Effect of electronic cooking on nutritive value of foods. J Am Diet Assoc. 1949;25:39–45.

44. Wing RW, Alexander JC. Effect of microwave heating on vitamin B, retention in chicken. J Am Diet Assoc. 1972;61:661.
45. Ortigues-Marty I, Thomas E, Prévéraud DP, et al. Influence of maturation and cooking treatments on the nutritional value of bovine meats: water losses and vitamin B12. Meat Sci. 2006;73:451–8.
46. Purchas RW, Rutherfurd SM, Pearce PD, et al. Cooking temperature effects on the forms of iron and levels of several other compounds in beef semitendinosus muscle. Meat Sci. 2004;68:201–7.
47. Lawrence TE, King DA, Obuz E, et al. Evaluation of electric-grill, forced-air convection oven and electric broiler cookery methods for beef tenderness research. Meat Sci. 2001;58:239–46.
48. Rhee KS, Anderson LM, Sams AR. Lipid oxidation potential of beef, chicken, and pork. J Food Sci. 1996;61:8–12.
49. Hernández P, Navarro JL, Toldrá F. Lipids of pork meat as affected by various cooking techniques. Food Sci Technol Int. 1999;5:501–8.
50. Hoac T, Daun C, Trafikowska U, et al. Influence of heat treatment on lipid oxidation and glutathione peroxidase activity in chicken and duck meat. Innov Food Sci Emerg Technol. 2006;7:88–93.
51. Gatellier P, Kondjoyan A, Portanguen S, et al. Effect of cooking on protein oxidation in *n*-3 polyunsaturated fatty acids enriched beef. Implication on nutritional quality. Meat Sci. 2010;85:645–50.
52. Santé-Lhoutellier V, Astruc T, Marinova P, et al. Effect of meat cooking on physicochemical state and in vitro digestibility of myofibrillar proteins. J Agric Food Chem. 2008;56:1488–94.
53. Huang F, Huang M, Xu X, et al. Influence of heat on protein degradation, ultrastructure and eating quality indicators of pork. J Sci Food Agric. 2011;91: 443–8.
54. Pearson AM, Love JD, Shorland FB. Warmed-over flavor in meat, poultry and fish. Adv Food Res. 1977;23:1–74.
55. Liu Y, Chen YR. Analysis of visible reflectance spectra of stored, cooked and diseased chicken meats. Meat Sci. 2001;4:395–401.
56. Oz F, Kaya M. Heterocyclic aromatic amines in meat. J Food Process Preserv. 2011;35:739–56.
57. Felton JS, Fultz E, Dolbeare FA, et al. Reduction of heterocyclic aromatic amine mutagens/carcinogens in fried beef patties by microwave pretreatment. Food Chem Toxicol. 1994;32:897–903.

The Quality of Orange Juice

21

Luciano Cinquanta and Marisa Di Matteo

Key Points

- Orange juice (OJ) is 100 % fruit juice available for consumer year-round, after the pasteurization or the process of concentration/freezing/pasteurization.
- Thermal pasteurization is used to prolong OJ shelf life, through inactivation of pectin-methylesterase, enzyme that causes loss of fresh juice cloudiness, and the suppression of spoilage microorganisms. Generally, the shelf life of OJ is estimated as the time period in which there is a 50 % of vitamin C (AA) loss.
- Mild processing technologies, such as high hydrostatic pressures, allow to obtain OJ with characteristics closer to those of fresh orange, namely for its content in substances exhibiting free radical scavenging and antioxidant activities. Other alternative stabilization techniques (microwave, ohmic heating, pulsed electric field), are under study to improve OJ quality.
- OJ contains an array of antioxidants including AA, flavonoids, and carotenoids, which exhibit different absorption kinetics, bioavailability, and antiradical mechanism.
- In the human organism AA may play a critical role in reducing the formation of compounds produced by the random oxidation of phospholipids by oxygen radicals, synergistically with carotenoids, that are part of the antioxidant defense system in membranes and other lipophilic compartments.

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- OJ contains several possible anticancer agents such as limonoids and flavonoids, such properties are attributed to their ability to inhibit cell proliferation, promote differentiation, to act as antioxidants and to modulate tyrosine kinases.
- Beyond the nutritional and healthy values, sensorial quality of OJ is also important, in fact, because of its pleasant taste, OJ often represents the major vitamin C intake source in children's dietary.

Keywords
Antioxidant • Ascorbic acid • Bioavailability • Carotenoids • Flavanones • Orange juice • Pasteurization • Shelf life

Abbreviations

AA	Ascorbic acid or vitamin C
C	AA concentration at time *t*
C_0	AA concentration at time 0
DPPH	Diphenyl-1-picrylhydrazyl
Ea	Activation energy (J/mol)
FCOJ	Frozen concentrate orange juice
FRAP	Ferring reducing antioxidant capacity
HHPs	High hydrostatic pressures
K	AA loss rate (days^{-1})
k_{ref}	AA acid loss rate at a reference temperature T_{ref}
k_T	AA loss rate at a storage temperature *T*
NFC	Not from concentrate
OJ	Orange juice
ORAC	Oxygen absorbing capacity
PET	Polyethylene terephthalate
R	Gas constant (8.314 J/(mol K))
R^2	Coefficient of determination
RDA	Recommended daily acceptance
RECON	Reconstituted
ROS	Reactive oxygen species
RTD	Ready to drink
T	Storage time (days)
TEAC	Trolox equivalent antioxidant capacity
UPOJ	Unpasteurized orange juice

Introduction

Orange juice (OJ) is defined in the *European Union*: "the fermentable but unfermented product obtained from *Citrus sinensis* (L.) *Osbeck*, which is sound and ripe, fresh or preserved by chilling, having the characteristic color, flavor, and taste typical of the juice of the fruit from which it comes. Flavor, pulp, and cells from the juice which are separated during processing may be restored to the same juice. The fruit juice must come from the endocarp with minimum 11.2°Brix." In the United States OJ is also obtained by the citrus hybrid commonly called "Ambersweet." OJ is prepared by mechanically squeezing of fruits: it is always 100 % fruit juice. Nectar is also made from citrus fruit but presents at least 50 % juice content and usually has added sugar. Still drinks contain some juice and a lot of sugar.

Orange juice is commonly marketed in four forms:

– Unpasteurized (UPOJ): squeezed from fresh fruit and packaged in cartons, glass, or plastic containers, without being pasteurized. The product is clearly labeled, with a shelf life of only a few days.
– Ready to drink (RTD), or not from concentrate (NFC): immediately processed and pasteurized after squeezing the fruit. The product can be stored frozen or chilled for at least a year.
– Frozen concentrate (FCOJ): obtained by removing, through evaporation, the water from the OJ. It can be stored for several years at −6.7 °C or lower.
– Reconstituted (RECON): processed to obtain the frozen concentrate and then reconstituted by adding back the water originally removed.

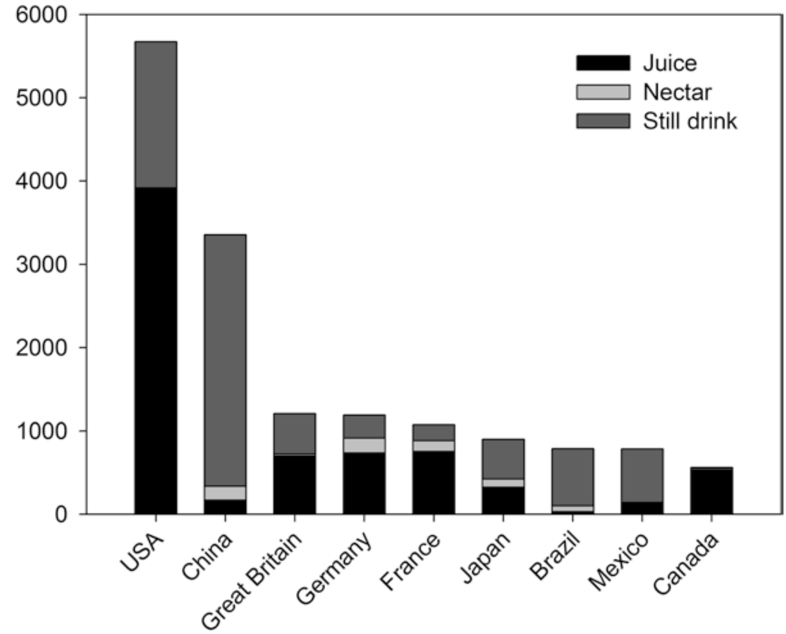

Fig. 21.1 Consumption (10^6 L) of beverage made from oranges in main countries during 2009. Juice: 100 % orange juice content; nectar: at least 50 % juice content; still drink: some juice content (*Source*: various reports)

Reconstituted single strength juice is sold as a ready-to-serve (RTS) product either in chilled form or in aseptic form, in bottles or cartons, with a shelf life of 6–8 months at room temperature.

Brazil is the country's largest producer of oranges and orange derivatives, production is located primarily in the state of São Paulo (70 % of Brazil's production) and the four major orange varieties used for processing OJ are the *Hamlin*, *Pera Rio*, *Natal*, and *Valencia*. The second largest producer is the United States, followed by China, Costa Rica, Mexico, and the Mediterranean region. The two major orange varieties under cultivation in United States (Florida, California) are the *Navel* and *Valencia*. A blend of different types of oranges is generally used to provide a specific flavor. Europe in 2009 has consumed almost 30 % of the world's beverages with orange flavor, of which 56 % was in the form of juice. The total consumption of OJ in Europe amounts to approximately 10 L per head and year. Consumers with elevated per capita income tend to consume 100 % premium OJ; so, a supply shift had the largest percentage impact on frozen concentrate orange juice (FCOJ) and RECON OJ and the smallest impact on not from concentrate (NFC) [1]. On the other hand, a consumer with lower per capita income tends to consume larger quantities of nectars and still drinks (Fig. 21.1). Total emission of greenhouse gas for 1 L of OJ produced in United States was estimated ranging between 0.22 and 0.38 kg carbon equivalent [2]. In the estimate data is not taken into account the carbon emission during shipping, for instance in transoceanic transportation of OJ in Europe.

A Brief History of Orange Juice

Orange trees, originated in Southeast Asia and cultivated in China by 2500 BC, were introduced to Brazil in the 1500s. The observations of great navigators from the fifteenth centuries, when scurvy plagued ships' crews, played an important role in clarifying scurvy's etiology. *Vasco da Gama*, during his circumnavigation of Africa noticed that many men fell sick showing swelling in the legs, arms, and gums, and he also noticed that eating oranges had a beneficial effect. Among

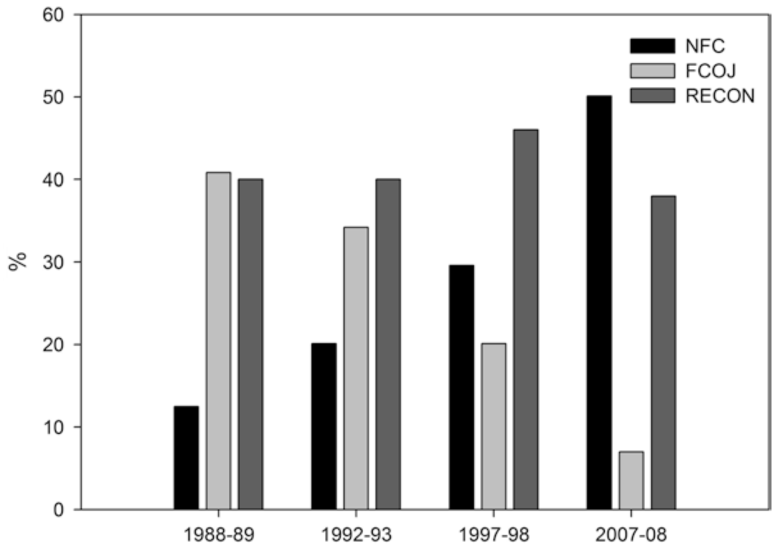

Fig. 21.2 Trends in category shares of the US orange juice market during last decades. *NFC* not from concentrate, *FCOJ* from concentrate orange juice, *RECON* reconstituted orange juice (*Source*: various reports)

the personalities in the history of the disease, *James Lind* and Nobel Prize-winning *Albert Szent-Györgyi* are the most noteworthy, the first, an officer of the British Royal Navy, in 1753 reported the first clinical trial on the treatment of scurvy with OJ. *Szent-Györgyi* is famous for discovering and identifying vitamin C in 1930s [3], while the advances in pasteurization techniques improved juice quality and the industry expanded significantly. Prior to the late 1940s, consumers obtained OJ from either squeezing fresh fruit at home or from canned juice, until scientists found a way to concentrate fruit juice in a vacuum and freeze it without destroying the flavor or vitamin content. FCOJ were first sold in the United States in 1945, and they became widely available and popular. In the same decade, hot packed orange concentrate in small flat glass bottles, was made available in the United Kingdom to supplement vitamin C intake for children and pregnant women. For many years, FCOJ was the most popular form of processed OJ sold at retail: in the 1950s it accounted for about 70 % of the OJ market. Afterwards, few things have stirred the hopes of the public in matters of nutrition as much as a double Nobel Prize-winning scientist *Linus Pauling*'s 1970 book, *Vitamin C and the Common Cold*. The book's main claim was that taking 1 g of AA daily would reduce the incidence of colds by 45 % for most people. Nowadays vitamin C does not seem to prevent the onset of the common cold, but in some studies it has been reported to reduce the length and severity of the symptoms. Furthermore, *Pauling* popularized use of AA for cancer therapy. In 1970s, a shift from glass to paper packaging facilitated reprocessing bulk FCOJ into RTS reconstituted OJ. By 1990 FCOJ popularity had declined: it accounted for less than 50 % of the OJ market, and RECON and NFC juices first outsold the frozen type (Fig. 21.2). The NFC juice requires special extraction and storage conditions to ensure high quality, for instance oil content <0.035 %, chilling and storing aseptically or in frozen blocks to provide a year-round supply to packers. Anyway, frozen technology was responsible for most of the early growth of the citrus industry, as shown by the development of "futures contract" as the world benchmark contract in the global FCOJ market. Shelf-stable OJ hot-filled into heatset polyethylene terephthalate (PET) containers have steadily grown in popularity since they first started appearing in 1995, made from concentrate. Today commercial aseptic packaging allows ready to drink (RTD) to be marketed without refrigerated storage. The recent trends show the increase of consumer preference in the United States and Europe towards unpasteurized orange juice (UPOJ), due to its superior taste, aroma, healthy and nutritive values. However, the juice is less stable during storage.

Nutritional and Sensory Quality of Orange Juice

The main energy-yielding nutrients in OJ are simple carbohydrates: fructose, glucose, and sucrose, as well as citric and malic acids, which can also provide a small amount of energy. OJ is also considered a good source of important essential nutrients such as AA, flavonoids, carotenoids, folate, and potassium (Table 21.1).

Vitamin C

The AA content of OJ is highly variable, depending on variety, fruit maturity, orange handling, processing parameters, packaging and storage conditions. The physiological functions of AA are attributed to the capacity to provide reducing equivalents for biochemical reactions. AA, an essential water-soluble vitamin, plays a key role in the formation of collagen, a primary component of much of the connective tissue in the body, is an essential cofactor in neurotransmitter synthesis and is important in vascular function [4]. AA is also an important aid in the absorption of inorganic iron; it has also been shown to aid in the treatment of anaemia and stress. AA is considered as a most important water-soluble antioxidant. It protects compounds in extracellular and intracellular spaces in most biological systems and reduces tocopherol radicals back to their active form at the cellular membranes [5]. It can directly scavenge superoxide radical, singlet oxygen, hydrogen peroxide, and hydroxyl radical. The nutritional quality of OJ is related primarily to its content of L-ascorbic acid, and dehydroascorbic acid, an oxidized form of AA. However, several studies revealed that the latter accounts for a small percentage (around 5 %) of the AA activity of the samples [6, 7]. Dehydroascorbic acid is not readily absorbed across the intestinal mucosa [8] and supplemental dehydroascorbic acid has little antiscorbutic activity. Thus, the oxidized AA content of OJ can be used an indicator of oxidative stress but cannot be considered a dietary source of vitamin C [9]. Based on available biochemical, clinical, and epidemiological studies, the current recommended daily acceptance (RDA) for AA is suggested to be 100–120 mg/day to achieve cellular saturation and optimum risk reduction of heart diseases, stroke, and cancer in healthy individuals [4, p. 9]. The AA content in OJ range from 150 to 550 mg/L; one glass of OJ (200 mL), can deliver about 30–90 % of recommended daily intake of AA. The main factors that can affect AA loss in OJ include temperature, salt and sugar concentration, pH, oxygen, enzymes, light, metal catalysts, initial concentration of AA, microbial load and protection provided by the container [10]. The decrease of AA concentration toward levels unacceptable by legislation or industrial practice, often defines the OJ shelf life. Generally, shelf life of OJ was estimated as the time period in which there is a 50 % AA loss. AA loss was found to follow apparent first order kinetics during storage (I):

$$C = C_0 e^{(-k \cdot t)} \qquad \text{(I)}$$

The effect of storage temperature on AA degradation rate was described adequately by *Arrhenius* kinetics (II):

$$k_T = k_{ref} e^{\left[-\frac{Ea}{R}\left(\frac{1}{T} - \frac{1}{T_{ref}}\right)\right]} \qquad \text{(II)}$$

Table 21.1 Average composition of commercial fresh squeezed orange juice (100 mL)

Energy (kJ)	170	Vitamin C (mg)	50
pH	4	Flavanones (mg)	55
Acidity (meq)	11	Limonoids (mg)	40
Carbohydrates (g)	10	Folate (μg)	30
Protein (g)	<1	Carotenoids(μg)	400
Phenolic acids (mg)	7	Potassium (mg)	190

J joule, *meq* milliequivalents, *μg* 10^{-6} g (*Source*: various reports)

Table 21.2 Estimated shelf life (days) based on 50 % ascorbic acid reduction at different temperatures (°C) of various form of orange juices

Storage temperature	Thermally pasteurized	HHPs treated	Concentrate
0	88	187	–
5	58	109	–
10	39	64	–
15	26	39	–
28	–	–	173
30	9	9	–
37	–	–	26
45	–	–	19

Adapted from refs. [11, 12]
HHPs high hydrostatic pressures

The activation energy (*Ea*) was determined to be 68.5 kJ/mol ($R^2=0.97$) and 53.1 kJ/mol ($R^2=0.97$) for high pressurized and thermally pasteurized OJ, respectively, suggesting greater temperature dependence of the AA degradation rate for high pressurized OJ [11]. In another trial with concentrate OJ, the activation energy was determined to be 105.4 kJ/mol [12]. The shelf life (t) of high pressure, heat pasteurized juice, and concentrate OJ at different storage temperatures can therefore be calculated through (I), substituting C with $0.5C_0$ and k with the predicted value from *Arrhenius* equation (II) for each storage temperature. The shelf life increase of HPPs treated OJ, compared to thermal pasteurized juice, ranged from 13 days (49 % increase), for storage at 15 °C, to 99 days (112 % increase), for storage at 0 °C (Table 21.2) [11]. In concentrate OJ, shelf life ranged from 173 days at 28 °C to 19 days at 45 °C [12]. It was then demonstrated that RECON OJ from frozen concentrate had the lowest content of oxidized AA (30 % of AA at preparation), whereas the NFC had higher concentration of oxidized AA (51 % of AA at 4 weeks from expiration). These data demonstrate a substantial loss of active vitamin C available to the consumer in NFC juice (Table 21.3). Pasteurized, NFC OJ typically contains 25 % less vitamin C per serving (80 mg), compared to frozen concentrate (100 mg), a result, in part, of heat-induced destruction of AA [9, p. 10].

Flavanones

Oranges are a rich source of health-promoting flavonoids, especially flavanones and hydroxycinnamic acids, or esters of ferulic, *p*-coumaric, sinapic, and caffeic acids. In contrast to other flavonoids like flavonols, which are found in a wide range of foods, flavanones are present in our diet almost exclusively in citrus fruits. Prominent flavanone glucosides in citrus are hesperidin (hesperetin-7-*O*-rutinoside) and naringin (naringenin-7-*O*-rutinoside), followed by narirutin and poncirin. The content of hesperidin and narirutin in OJ ranges from 188 to 590 mg/L and from 16 to 96 mg/L, respectively [13]. One glass of OJ (200 mL) of OJ provides approximately 83.3 mg of flavanones [14]. It was reported that concentration technique doubled the total flavanones content in the cloud portion (pulp), due to the precipitation from the soluble fraction [15]. Hesperidin content variation appears to be greater in NFC juice than that of FCOJ. In general, hesperidin, narirutin, and total flavonoids content of NFC juices were lower compared to FCOJ [16].

Carotenoids

Characteristic color of both the peel and the pulp of most varieties of ripe oranges are mainly due to carotenoid pigments. The carotenoids profile can vary markedly within the same variety, in function of the climatic features, of the geographical area of the cultivar and of industrial processing and storage conditions. Carotenoids possess structural diversity and are important from a nutritional point of view and for their potential health benefits, because some of them have provitamin A activity. Total carotenoid content in fresh OJ varied from 26 to 81 mg/100 mL, originated by more than 14 different carotenoid compounds, the main of them is antheraxanthin (44 %), followed by β-cryptoxanthin (10 %), zeaxanthin, and lutein (8 %) [17, 18]. Pasteurization leads to decreases in the levels of some carotenoids, such as violaxanthin, antheraxanthin [19] and β-cryptoxanthin, considered the main source of provitamin A in OJ.

Table 21.3 Loss of active vitamin C (reduced AA) in different form of orange juice at 4, 1, and 0 expiration weeks

Expiration date	Total AA (mg/100 mL)	Reduced AA (%)	Oxidized AA (%)	Label claim (mg/100 mL)	Label claim (%)
RECON Freshly prepared	122.7	70	30	96	90
NFC[a]					
4	133.0	49	51	60	108
0	83.0	28	72		39
NFC[b]					
4	118.5	46	54	72	75
1	98.1	46	54		63

Adapted from ref. [9]
AA ascorbic acid, *NFC* not from concentrate, *RECON* reconstituted orange juice
[a]Waxed carton container
[b]Plastic container

Folate

Folate content in OJ ranged from 13 to 30 mg/100 mL, with wide variation between juices from different brands, freshly pressed juice and juice made from concentrate [20, 21]. OJ may be considered a good source of natural folate also in respect to stability during storage and simulated digestion. Folate is a B-vitamin essential for cellular replication. The RDA for folate for adults aged 51 and greater is 400 μg/day. Adequate folate status has been defined as a serum or plasma folate concentration above 7 nmol/L (3 ng/mL) and a red blood cell concentration above 305 nmol/L (140 ng/mL). Plasma homocysteine has been used as a folate status functional indicator, with 14 μmol/L more often used as a cutoff to define elevated concentrations [22].

Limonoids

Limonoids are unique highly oxygenated triterpenoid compounds present in Rutaceae long recognized as significant biologically active natural compounds. Citrus limonoids appear in large amounts in citrus juice as water-soluble limonoid glucosides, their concentrations can reach levels of 350–400 mg/L in OJ. Pharmacological investigations have shown that certain citrus limonoids have potential health benefits, owing to their anti-inflammatory and anticancer properties. Limonoid aglycones are converted to non-bitter 17β-D-glucopyranoside derivatives such as limonin 17β-D-glucopyranoside (LG) during maturation. This natural debittering process is catalyzed by the enzyme UDPD-glucose (limonoid glucosyltransferase). Bitterness is an important factor for determining the quality of citrus fruits and their processed products. Particularly, the reduction of the bitter taste due to limonin is still a major problem in the citrus industry worldwide. An ongoing molecular biology study aimed at the creation of transgenic citrus fruit could provide a way to eliminate the bitterness problems [23].

Volatile Compounds

Another important attribute for OJ acceptance by consumers is its aroma: during the industrial processing of OJ the biggest losses in the concentration of volatile components occurs during deaeration, applied before pasteurization. Afterwards, to reduce the costs for transportation and storage, OJ undergoes a concentration process, yielding three important fractions: the concentrate, the aqueous phase, and the essential oil. In the distributing countries, the concentrate is diluted with water and, after addition of the two aroma fractions the RECON juice is pasteurized before bottling. In comparison to a hand squeezed OJ, compounds with higher odor activity values (OAVs), in the freshly reconstituted OJ were: linalool, octanal, decanal, (*R*)-limonene, (*R*)-α-pinene and myrcene. On the other hand, acetaldehyde, (*S*)-ethyl 2-methylbutanoate, ethyl butanoate, and (*Z*)-3-hexenal were significantly reduced in RECON OJ, as compared to the hand squeezed juice. Moreover, (*E*)-β-damascenone, dimethyl sulfide and carvone, probably formed by the thermal treatment and/or oxidation, were found in the RECON juice, but not in the hand squeezed juice [24].

Advanced Technologies to Improve Orange Juice Quality

Nowadays there is a strong consumer demand for premium OJ that maintains a nutrient profile as close as possible to that of fresh fruits. As the processing techniques (pasteurization, freezing, and concentration), could affect OJ compounds, manufacturers and researchers have a common aim to find the best conditions and processing techniques able to improve the health benefits and taste from OJ.

Extraction and Juice Concentration

In the extraction of OJ the fluid is moved to a finisher, to separate the solids from the stream of liquid. A process for debittering OJ involves separating the pulp by centrifugation or ultrafiltration and recombining the pulp with the debittered juice prior to concentration. OJ is then moved to an evaporator, commonly a seven-effect short-time evaporator (TASTE), where about 2.8 kg of water are removed for each kilogram of steam used, followed by pasteurization and chilling prior to retail packaging.

Advanced Technologies for Pasteurization

Thermal pasteurization is used to inactivate heat-stable pectin-methylesterase enzyme (PME EC 3.1.1.11), which is more thermal resistant than spoilage microorganisms (yeasts and lactic acid bacteria), to prolong OJ shelf life. PME activity is important because this enzyme is responsible for the hydrolysis of the pectin present in citrus fruit juices that causes loss of fresh juice cloudiness and gelation of pectin in concentrate juice. Due to the low pH (generally ≤4.0) of OJ, growth of pathogenic microorganisms is normally suppressed, anyway, the FDA recommends a 5-log reduction of the "pertinent microorganism," which is the most resistant microorganism, in OJ: *E. coli O157*:*H7*. Typically, for shelf-stable juices, processing times for thermal pasteurization are equivalent to 90 °C for 1 min. Thermal processing has a negative impact on the quality of OJ such as loss of fresh flavor, degradation of AA, and discoloration. A relatively large loss of provitamin A (β-carotene, α-carotene, and β-cryptoxanthin), and carotenoids occurred after thermal processing in OJ. Alternative stabilization techniques are under study to improve OJ quality. Microwaves' heating seems a useful technique, owing to its characteristic volumetric heating without intermediate fluid, thus allowing for faster and more effective treatment. Microwave processing, with the support of an effective temperature control (i.e., infrared thermography), resulted effective to stabilize fresh OJ, minimizing thermal damage [25]. Another method proposed for OJ pasteurization was ohmic heating, which advantages, instead, include uniform heating of food. The results obtained with a thermal treatment by continuous ohmic heating suggested that it can be used to extend the sensorial shelf life of pasteurized freshly squeezed OJ [26]. Pulsed electric field (PEF) processing is a nonthermal preservation process: OJ is treated in a chamber with two electrodes giving short electric pulses to inactivate microorganisms, in order to increase the shelf life of OJ with a minimal effect on its quality.

High Hydrostatic Pressures

High hydrostatic pressures (HHPs) represent an industrial method for the inactivation of microbial activity through the induction of membrane damage under reduced heating regimes, providing better retention of many antioxidant compounds. HHPs have been used to inactivate PME enzymes in OJ, thereby preventing the hydrolysis of pectin and maintaining the desirable cloud [11, p. 14]. HHPs treated OJ is available in Europe, United States, and Japan, thus evidencing the utility and cost effectiveness of the process. HHPs have been demonstrated to be effective against *E. coli* O157, inactivated in OJ following treatment at 250 MPa for 5 min at 40 °C. HHPs treatment, compared with thermally processed juices, maintained higher or equivalent levels of AA in OJ, increased the bioavailability of folate, and improved the recovery of the major flavonones, naringenin, and hesperetin. Regarding lipophilic antioxidants, HPPs treatment improved the recovery of carotenoids in OJ up to 54 %, probably as a result of enhanced carotenoid release from denatured proteins and membranes [7, p. 14].

Packaging and Shelf Life of Orange Juice

The adverse effects of dissolved oxygen on OJ quality include degradation of AA, increased browning and growth of aerobic bacteria and moulds. The packaging prevents microorganisms from entering and proliferating inside and reduces the exposure of OJ to oxygen through the use of high barrier materials such glass or foil laminates in brick packs, nitrogen flushing or improving gas barrier of PET by blending with aromatic polyamides [27]. The reasons for AA loss in fully pasteurized, aerobically stored OJ are not enzymatic, because oxidative enzymes such as cytochrome oxidase, ascorbic acid oxidase, and peroxidase, responsible for the loss of AA in freshly squeezed OJ, are destroyed during pasteurization. Chilled OJ contains significantly less AA (–40 %), than freshly prepared OJ from frozen concentrate [9, p. 15]. Most chilled OJ is packaged in polyethylene or polystyrene containers, which are permeable to oxygen, causing AA destruction (20 % at 3–4 weeks postproduction). FCOJ retain more than 90 % of its AA content after 12 months at –20 °C. UPOJ showed higher ascorbic acid retention respect to pasteurized juice during storage, owing to: oxygen consumption by natural flora in single strength juice, the thermal oxidative destruction of several protectors of AA such as flavonoids, and the production of oxidative molecules during the first steps of the *Maillard* reaction. The shelf life of the natural fresh OJ stored at 25 °C and 4 °C could be extended only 1 and 6 days, respectively, because beyond these periods, their total colony counts, odor, and chemical changes were unacceptable [28].

Health Benefits of Orange Juice

Antioxidant Capacity of Orange Juice

OJ contains an array of potent antioxidants including flavonoids (hesperetin and naringenin, predominantly as glycosides), carotenoids (xanthophylls, cryptoxanthins, carotenes), and AA. The term "antioxidant" has been defined: "a substance in foods that significantly decreases the adverse effects of reactive species, such as reactive oxygen species (ROS) and nitrogen species, on normal physiologic function in humans." Oxidative stress has been associated with the development of many chronic and degenerative diseases as well as being involved in the process of aging. ROS can damage biological molecules such as proteins, lipids, and DNA. ROS are generated as byproducts of normal cell aerobic respiration that is essential to life. Antioxidants can reduce radicals by single electron transfer and hydrogen atom transfer mechanisms, thus its activity can been determined in food using several assays. AA scavenges superoxide anions and plays an important role in the control of the intracellular redox state [29]. Diphenyl-1-picrylhydrazyl (DPPH) and FRAP measure the single electron transfer, and oxygen absorbing capacity (ORAC) and TRAP represent the hydrogen atom transfer mechanism (Table 21.4) [30, 31]. The positive and very strong correlation between AA content and total antioxidant activity of NFC suggested that the AA is the main responsible for the antioxidant activity [32]. In OJ the AA content explained 50 % of the antioxidant capacity measured by the DPPH assay. AA protects endothelial cells from either intra- or extracellular oxidant stress and also may reduce the risk of atherosclerosis. High intakes of AA (500 mg/day, corresponding to five glasses of

Table 21.4 Antioxidant potency in orange juice

Antioxidant assay	Content	Reference
DPPH index	25.3	[30]
DPPH (% inhibited)	12.7	[30]
ORAC index	23.3	[30]
ORAC (μmol of TE/mL)	7.4	[30]
H-ORAC[a] (μmol of TE/day)	454.0	[31]
FRAP index	18.5	[30]
FRAP (μmol of FE/mL)	1.5	[30]
TEAC index	10.1	[30]
TEAC (μmol/mL)	1.5	[32]
VCEAC (mg/100 mL)	19.4	[32]
Peroxides[b]	12.9	[30]
Malondialdehyde[b]	7.7	[30]
Total phenols (mg gallic acid/L)	3.3	[32]
GAEs (mg gallic acid/L)	0.7	[30]

Adapted from refs. [30–32]
TEAC trolox equivalent antioxidant capacity, *ORAC* oxygen absorbing capacity, *FRAP* ferring reducing antioxidant capacity, *DPPH* free radical scavenging properties by duphenyl-1-picrylhydrazil radical, *VCEAC* vitamin C, equivalent antioxidant capacity, *GAE* gallic acid equivalent
[a]*H* hydrophilic
[b]Inhibition of LDL oxidation

OJ), obtained from OJ, prevented a rise in the levels of oxidized LDL (low density lipoproteins), even in the presence of a high-saturated fat diet. Drinking OJ increased AA concentrations and reduced oxidative stress in vivo, by lowering the concentration of F_2-isoprostanes. Moreover, two glasses of OJ reduced the concentrations of 8-e*pi*-prostaglandina (8-*epi* $PGF_{2\alpha}$), marker of oxidative stress, inversely correlated with total AA content [33]. Such data suggested that AA may play a critical role in reducing the formation of compounds produced by the random oxidation of phospholipids by oxygen radicals. Moreover, it was ascertained that low blood levels of AA are detrimental to health of older persons [34]. In the past it was assumed that AA was the only chemo preventive agent in citrus, whilst recent accumulative evidences suggest that citrus contains several possible anticancer agents such as limonoids and flavonoids [35]. Physiological properties of these flavonoids are attributed to their ability to inhibit cell proliferation, promote differentiation and function as antioxidants and to modulate tyrosine kinases. Thus, in OJ coexist components exhibiting fast antiradical kinetics (mainly AA and minor amounts of some polyphenolic derivatives), and components exhibiting slow antiradical kinetics (mainly citrus flavanone-7-*O*-glycosides and minor amounts of some polyphenolic derivatives). Drinking OJ with meals can counter the pro inflammatory effects of high-fat, high-carbohydrate meal that induce a protein, suppressor of cytokine signaling 3 (SOCS-3), which interferes with the action of insulin, promoting insulin resistance. These adverse changes can be avoided when OJ is consumed with a meal. This implies that drinking OJ could potentially help prevent insulin resistance, diabetes, and coronary heart disease. Such benefit may be attributable to the OJ flavonoids, which suppress ROS. The intake of glucose or a high-fat, high-carbohydrate meal, but not OJ, induces an increase in inflammation and oxidative stress in circulating mononuclear cells (MNCs) of normal-weight subjects. These observations may partly explain the mechanisms underlying postprandial oxidative stress and inflammation, pathogenesis of insulin resistance, and atherosclerosis. In contrast, the intake of OJ containing fructose, glucose, and sucrose does not cause an increase in ROS generation. In addition, hesperetin and naringenin, but not AA, are able to suppress ROS generation by MNCs in vitro by 50 % [36]. Carotenoids may be scavengers of reactive species, showing antioxidant activity, and they may protect humans from serious disorders such as skin disorders, cardiovascular disease, age-related diseases of the eye, such as macular degeneration, or cataracts [17, p. 17]. The lipophilicity of the carotenoids determines their subcellular distribution in membranes and other lipophilic compartments. In the human organism, carotenoids are part of the antioxidant defense system, since are most likely involved in the scavenging of two of the ROS, singlet molecular oxygen and peroxyl radicals. Singlet oxygen quenching by carotenoids occurs via physical or chemical quenching. Mixtures of carotenoids are more effective than single compounds and they interact synergistically with other antioxidants. Anyway, under specific conditions AA, flavonoids and carotenoids may also act as pro-oxidants, as were discussed in context of adverse effects observed upon supplementation at high levels. Thus, it is important to get antioxidant intake directly by vegetables or minimally processed fruits (i.e., OJ), and to consider that antioxidants health-promoting properties depending on specific people baseline levels of ROS. ROS, in fact, play either harmful or beneficial role in biological systems, the last including physiological roles in cellular responses against infectious agents and in several cellular signaling pathways [37].

Bioavailability and Benefits of Orange Juice Micronutrients in Humans

At low concentrations, the absorption of AA occurs through an active transport process, whereas at high concentrations, absorption is mediated by a combination of both active and passive diffusion in the gastrointestinal tract. Thus, consumption of foods rich in AA may favor absorption by slowing the interaction of the juice with the gastric wall. Consumption of 250 mg AA, contained in two and a half glasses of OJ (500 mL), significantly increased plasma AA from 30–50 to 60–90 mmol/L in just 3 h [36, p. 18]. The increased concentration was maintained as long as the subjects were drinking the OJ, which suggests that this is an efficient means of increasing AA concentrations in the body. For a long time, absorption of flavonoids from dietary sources was considered to be negligible. Furthermore, recent reports suggest that humans absorb appreciable amounts of flavonoids in the small intestine and citrus flavonoids could reach relatively high concentrations in human plasma after ingestion of orange [38]. Flavanones absorption takes place in the colon, a concentration of 0.5–1.5 mmol/L of flavanone glucuronides was found in plasma only for a short period. In fact, 24 h after the ingestion of orange juice, the urinary elimination of flavanones was nearly complete (98 %), and the metabolites were no longer present in plasma [39]. It has been suggested that flavonoids from OJ affect cholesterol metabolism in the liver, because they inhibit the production of endogenous lipoproteins. Hesperidin and naringin have been reported to reduce plasma cholesterol and to protect animals against chemically induced cancers of colon, bladder, skin, and oesophagus [40]. Adequate folate intake and status has been also associated with reduced risk for chronic conditions that may particularly affect the elderly, including hyperhomocysteinemia, a risk factor for vascular disease, cancer, and cognitive dysfunction. The primary chemical form of folate occurring in nature is a pteroylglutamate compound; folate must be cleaved to the monoglutamate form by an intestinal conjugase enzyme before being absorbed in the intestinal tract. The bioavailability of carotenoids is governed by characteristics of the food matrix, which affect the efficiency of physical, enzymatic, and chemical digestion. Following release from the food matrix, the major limiting factor is solubility of carotenoids in digesta. There is a large body of literature that indicates that dietary carotenoids are cancer preventative: α-carotene has been found to be a stronger protective agent than its well-known isomer β-carotene [41]. To support healthy benefits of OJ, some results of the association of 100 % OJ consumption by children 2–18 years of age ($n=7{,}250$), participating in the 2003–2006 National Health and Nutrition Examination Survey with intakes of select nutrients, were reported. Compared with non consumers, OJ consumers had a higher percentage of the population meeting the Estimated Average Requirement for vitamin A (19.6 % vs. 30.2 %), vitamin C (0.0 % vs. 29.2 %), folate (1.3 % vs. 5.1 %), and magnesium (25.5 % vs. 39.0 %). The Healthy Eating Index-2005 was significantly ($P<0.01$) higher in consumers (52.4 ± 0.4 vs. 48.5 ± 0.3). So, a moderate consumption of OJ should be encouraged in children as a component of a healthy diet [42].

Conclusions

Mild processing technologies allow to obtain OJ available year-round, with characteristics closer to those of fresh orange, namely for its content in substances exhibiting free radical scavenging and antioxidant activities. These healthy components, mainly vitamin C, carotenoids, and flavonoids, exhibit different absorption kinetics, bioavailability, and antiradical mechanisms. Anyway, there is greater absorption of these nutrients when taken not as singly as supplements, but when consumed in minimally processed fruit, such as OJ, in which they naturally appear along with all the other biologically active phytonutrients that citrus fruits contain. Type of processing, packaging and storage conditions affect the OJ quality; generally, the shelf life of OJ is estimated as the time period in which there is a 50 % of vitamin C loss. Beyond the nutritional and healthy values, sensorial quality of OJ is also important, in fact, because of its pleasant taste, the orange juice often represents the major vitamin C intake source in children's dietary.

References

1. Brown MG. Demand for OJ, by product form, in a conditional demand system for beverage, and sensitivity of product forms to supply. Florida Department of Citrus. Research paper 2010-1.
2. Spreen T, Dwivedi P, Goodrich-Schneider R. Estimating the carbon footprint of Florida orange juice. In: Proceedings in food system dynamics. http://centmapress.ilb.unibonn.de/ojs/index.php/proceeding/article/view/42. Accessed 3 May 2013.
3. Magiorkinis E, Beloukas A, Diamantis A. Review article. Scurvy: past, present and future. Eur J Int Med. 2011;22:147–52.
4. Naidu KA. Vitamin C, in human health and disease is still a mystery? An overview. Nutr J. 2003;2:1–10.
5. Kaur C, Kapoor CH. Antioxidants in fruits and vegetables—the millennium's health. Int J Food Sci Technol. 2001;36:703–25.
6. Behrens WA, Madere RA. Procedure for the separation and quantitative analysis of ascorbic acid, dehydroascorbic acid, isoascorbic acid, and dehydroisoascorbic acid in food and animal tissue. J Liq Chromatogr. 1994; 17:2445–55.
7. Sánchez-Moreno C, Plaza L, de Ancos B, Cano MP. Effect of high-pressure processing on the health promoting attributes of freshly squeezed orange juice (Citrus sinensis L.) during chilled storage. Eur Food Res Technol. 2003;216:18–22.
8. Malo C, Wilson JX. Glucose modulates vitamin C transport in adult human small intestinal brush border membrane vesicles. J Nutr. 2000;130:63–9.
9. Johnston CS, Bowling DL. Stability of ascorbic acid in commercially available orange juices. J Am Diet Assoc. 2002;102:525–9.
10. Tannenbaum SR, Archer MC, Young VR. Vitamins and minerals. In: Fennema OR, editor. Food chemistry. New York: Marcel Dekker; 1985. p. 488–93.
11. Polydera AC, Stoforos NG, Taoukis PS. Quality degradation kinetics of pasteurised and high pressure processed fresh Navel orange juice: nutritional parameters and shelf life. Innov Food Sci Emerg Technol. 2005;6:1–9.
12. Burdurlu HS, Koca N, Karadeniz F. Degradation of vitamin C in citrus juice concentrates during storage. J Food Eng. 2006;74:211–6.
13. Tomás-Barberán FA, Clifford MN. Flavanones, chalcones and dihydrochalcones—nature, occurrence and dietary burden. J Sci Food Agric. 2000;80:1073–80.
14. Bronner WE, Beecher GR. Extraction and measurement of prominent flavonoids in orange and grapefruit juice concentrates. J Chromatogr A. 1995;705:247–56.
15. Gil-Izquierdo A, Gil MI, Ferreres F. Effect of processing techniques at industrial scale on orange juice antioxidant and beneficial health compounds. J Agric Food Chem. 2002;50:5107–14.
16. Vanamala J, Reddivari L, Yoo KS, Pike LM, Patil BS. Variation in the content of bioactive flavonoids in different brands of orange and grapefruit juices. J Food Compos Anal. 2006;19:157–66.
17. Melèndez-Martìnez AJ, Vicario IM, Heredia FJ. Review: analysis of carotenoids in orange juice. J Food Compos Anal. 2007;20:638–49.
18. Fratianni A, Cinquanta L, Panfili G. Degradation of carotenoids in orange juice during microwave heating. Food Sci Technol Leb. 2010;43:867–71.
19. Lee HS, Coates GA. Effect of thermal pasteurization on Valencia orange juice color and pigments. Food Sci Technol Leb. 2003;36:153–6.
20. Öhrvik V, Witthöft C. Orange juice is a good folate source in respect to folate content and stability during storage and simulated digestion. Eur J Nutr. 2008;47:92–8.
21. Matella NJ, Braddock RJ, Gregory JF, Goodrich RM. Capillary electrophoresis and high-performance liquid chromatography determination of polyglutamyl 5-methyltetrahydrofolate forms in citrus products. J Agric Food Chem. 2005;53:2268–74.
22. Franke AA, Cooney RV, Henning SM, Custer LJ. Bioavailability and antioxidant effects of orange juice components in humans. J Agric Food Chem. 2005;53:5170–8.
23. Moriguchi T, Kita M, Hasegawa S, Omura M. Molecular approach to citrus flavonoid and limonoid biosynthesis. J Food Agric Environ. 2003;1:22–5.
24. Averbeck M, Schieberle PH. Characterisation of the key aroma compounds in a freshly reconstituted orange juice from concentrate. Eur Food Res Technol. 2009;229:611–22.

25. Cinquanta L, Albanese D, Cuccurullo G, Di Matteo M. Effect on orange juice of batch pasteurization in an innovative pilot-scale microwave oven. J Food Sci. 2010;75:46–50.
26. Leizerson S, Shimoni ES. Stability and sensory shelf life of orange juice pasteurized by continuous ohmic heating. J Agric Food Chem. 2005;53:4012–8.
27. Ros-Chumillas M, Belissario Y, Iguaz A, López A. Quality and shelf life of orange juice aseptically packaged in PET bottles. J Food Eng. 2007;79: 234–42.
28. Supraditareporn W, Pinnthong R. Physical, chemical and microbiological changes during storage of orange juices cv. Sai Nam Pung and cv. Khieo Waan in Northern Thailand. Int J Agric Biol. 2007;9:726–30.
29. Wu X, Beecher GR, Holden JM, Haytowitz DB, Gebhart SE, Prior RL. Lipophilic and hydrophilic antioxidant capacities of common foods in the United States. J Agric Food Chem. 2004;52:4026–37.
30. Seeram NP, Aviram M, Zhang Y, et al. Comparison of antioxidant potency of commonly consumed polyphenol-rich beverages in the United States. J Agric Food Chem. 2008;56(4):1415–22.
31. Tabart J, Kevers C, Pincemail J, Defraigne JO, Dommes J. Evaluation of spectrophotometric methods for antioxidant compound measurement in relation to total antioxidant capacity in beverages. Food Chem. 2010;120:607–14.
32. Stella SP, Ferrarezi AC, dos Santos KO, Monteiro M. Antioxidant activity of commercial ready-to-drink orange juice and nectar. J Food Sci. 2011;76:392–7.
33. Sánchez-Moreno C, Cano MP, de Ancos B, et al. Effect of orange juice intake on vitamin C concentrations and biomarkers of antioxidant status in humans. Am J Clin Nutr. 2003;78:454–60.
34. Fletcher AE, Breeze E, Shetty PS. Antioxidant vitamins and mortality in older persons: findings from the nutrition add-on study to the Medical Research Council Trial of assessment and management of older people in the community. Am J Clin Nutr. 2003;78: 999–1010.
35. Poulose SM, Harris ED, Patil BS. Antiproliferative effects of citrus limonoids against human neuroblastoma and colonic adenocarcinoma cells. Nutr Cancer. 2006;56:103–12.
36. Ghanim H, Sia CL, Upadhyay M, et al. Orange juice neutralizes the proinflammatory effect of a high-fat, high-carbohydrate meal and prevents endotoxin increase and Toll-like receptor expression. Am J Clin Nutr. 2010;91:940–9.
37. Balsano C, Alisi A. Antioxidant effects of natural bioactive compounds. Curr Pharm Des. 2009;15:3063–73.
38. Erlund I, Meririnn E, Alfthan G, Aro A. Plasma kinetics and urinary excretion of the flavanones naringenin and hesperetin in humans after ingestion of orange juice and grapefruit juice. J Nutr. 2001;131:235–41.
39. Manach C, Morand C, Gil-Izquierdo A, Bouteloup-Demange C, Rémésy C. Bioavailability in humans of the flavanones hesperidin and narirutin after the ingestion of two doses of orange juice. Eur J Clin Nutr. 2003;57:235–42.
40. Tanaka T, Makita H, Kawabata K, et al. Chemoprevention of azoxymethane-induced rat colon carcinogenesis by the naturally occurring flavonoids, diosmin and hesperidin. Carcinogenesis. 1997;18:957–65.
41. Männistö S, Smith-Warner SA, Spiegelman D, et al. Dietary carotenoids and risk of lung cancer in a pooled analysis of seven cohort studies. Cancer Epidemiol Biomarkers Prev. 2004;13:40–8.
42. O'Neil CE, Nicklas TA, Rampersaud GC, Fulgoni VL. One hundred percent orange juice consumption is associated with better diet quality, improved nutrient adequacy, and no increased risk for overweight/obesity in children. Nutr Res. 2011;31:673–82.

22 Whole Grains and Diet

Satya S. Jonnalagadda

Key Points

- Whole grains contain significant amounts of functional, biologically active nutrients and phytonutrients.
- Global dietary guidelines recommend incorporation of whole grain foods into a healthy diet and lifestyle program.
- Global whole grain intake continues to be well below dietary recommendations.
- Regular consumption of whole grains contributes to improved nutrient intakes.
- Whole grains play a significant role in lowering the risk of chronic diseases, such as coronary heart disease, diabetes, and cancer, and also contribute to body weight management and gastrointestinal health.
- The essential macro- and micronutrients, along with the phytonutrients present in whole grains, synergistically contribute to beneficial health effects of whole grains.
- Ways to encourage whole grain consumption include: Increasing consumer awareness of the health benefits of whole grains, increasing availability of whole grain foods, enabling easy identification of whole grain foods in the marketplace and changing perceptions of taste and palatability of whole grain foods.
- It's important for health professionals, industry, government, and public health campaigns to more vigorously promote consumption of whole grains.

Keywords

Whole grains • Whole grain foods • Nutrients • Phytonutrients • Diet quality

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Abbreviations

CSFII	Continuing Survey of Food Intake
EAT	Eating attitudes of teenagers
HEI	Healthy Eating Index
NHANES	National Health and Nutrition Examination Survey
RTEC	Ready-to-eat-cereals
TE	Trolox equivalents

Introduction

Whole grains and whole grain foods are an important component of nutrient-dense diets and play a significant role in maintaining health and preventing chronic disease. This chapter briefly summarizes the role of whole grains in health and nutrition through discussion of: definition of whole grains, whole grain composition, whole dietary recommendations and consumption patterns, dietary sources of whole grains, whole grains impact on diet quality and whole grain foods in the market place. This chapter is not meant to be a comprehensive review of these topics but rather provides an overview of the importance of whole grains.

Whole Grains Definition

Whole grains are defined by the American Association of Cereal Chemists International and the United States Food and Drug Administration as consisting of the "intact, ground, cracked or flaked fruit of the grain whose principal components, the starchy endosperm, germ and bran, are present in the same relative proportions as they exist in the intact grain" [1, 2]. This definition was adapted by other regulatory agencies and groups, such as the United Kingdom's Food Standards Agency [3], and Health Grain Forum [4]. In order to be considered whole grain, whole grains foods that undergo processing and reconstitution must deliver the same proportion of bran, germ, and endosperm as that of the original grain [1, 2]. During the refining of whole grains into white flour, the outer bran and inner germ layers are removed and the remaining endosperm is processed into flour. Consequently, whole grains are inherently richer in dietary fiber compared to refined grains, containing approximately 80 % more dietary fiber [5]. Furthermore, the refining process results in a substantial loss of essential minerals, vitamins, and phytonutrients [5]. Table 22.1 provides examples of common whole grains and whole grain foods.

Table 22.1 Common whole grains and examples of whole grains food products

True cereals (common names)	Pseudo cereals
Wheat	Amaranth
Oat	Buckwheat
Barley	Quinoa
Brown rice	
Maize (corn)	
Rye	
Millets	
Sorghum	
Teff	
Triticale	
Examples of common whole grains food products	
Whole wheat flour	Whole grain (wheat) pasta
Whole wheat bread	Whole grain (e.g., wheat, corn) tortilla
Rolled oats, oatmeal	Brown rice
Popcorn	Baked goods (e.g., whole wheat blueberry muffin, whole wheat bread)
Rye bread	Whole grain ready-to-eat breakfast cereal
	Whole grain snacks (e.g., crackers, bars)

Whole Grains Composition

Table 22.2 shows the macronutrient composition of the common whole grains [6]. In addition to fiber, whole grains are rich sources of vitamins (B-complex, E), minerals (Iron, Magnesium, Potassium, Zinc, Selenium), dietary fiber, lignins, cellulose, beta-glucan, inulin, numerous phytochemicals, phytosterols, phytin, and sphingolipids [5, 7]. It is suggested that these compounds exert an additive, synergistic effect on health when consumed together [5, 7].

Table 22.2 Macronutrient composition of whole grains [6]

Grain	Calories (kJ)	Total carbohydrate (g)	Protein (g)	Total fat (g)	Total dietary fiber (g)
	Per 100 g				
Wheat, soft white	1,421	75.4	10.7	1.99	12.7
Oat	1,626	66.3	16.89	6.90	10.6
Barley, hulled	1,480	73.5	12.48	2.3	17.3
Rice, brown, long-grain	1,547	77.2	7.9	2.9	3.5
Corn, yellow	1,526	74.3	9.4	4.7	7.3
Rye	1,413	75.9	10.3	1.6	15.1
Millets	1,580	72.8	11.0	4.2	8.5
Sorghum	1,413	74.6	11.3	3.3	6.3
Teff	1,534	73.1	13.3	2.4	8.0
Triticale	1,404	72.1	13.0	2.1	Not available

Individual Whole Grain Components

The outer bran layer of whole grains is the multi-layered outer skin of the grain that protects the germ and endosperm from damaging sunlight, pests, water, and disease. The bran is composed of non-digestible, mainly insoluble, poorly fermentable carbohydrates (e.g., cellulose, hemicellulose), phenolic compounds, vitamins, minerals, and fiber. The inner germ and starchy endosperm contain viscous soluble fibers, fermentable oligosaccharides, resistant starch, lignins, vitamins, minerals, polyphenols, unsaturated fatty acids, and other phytonutrients [5]. The endosperm is the largest component of the whole grain and contains carbohydrates, protein, vitamins, and minerals. It serves as the food supply for the germ and provides energy for the rest of the plant. The germ refers to the embryo, the part that forms the new plant, and contains vitamins, some protein, minerals, and fat.

Whole Grain Phytonutrients

Dietary phytochemicals are defined as bioactive, non-nutrient plant compounds that are associated with a reduced risk of chronic diseases. Adom et al. [8] reported that the majority of the beneficial phytochemicals are present in the bran and germ fractions of whole grains. The additive and synergistic effects of these bioactive phytochemicals may be responsible for the health benefits associated with whole grains. Additionally, these phytochemicals can potentially complement those found in fruits and vegetables when consumed together [5, 8].

Whole Grain Phenolics

Plant phenolics provide chemical defense against pathogens, parasites, and predators. There are various classes of phenolic compounds in whole grains, including derivatives of benzoic and cinnamic acids, namely ferulic acid, vanillic acid, caffeic acid, syringic acid, *p*-coumaric acid, anthocyanidins, quinines, flavonols, chalones, flavones, flavanones, and amino phenolic compounds [5, 8]. These phenolics are present in water-soluble conjugated and free-form in whole grains [9]. Ferulic acid is one of the most studied whole grains phenolics [5, 8, 9]. Food processing (e.g., thermal processing and milling) helps to release these phytochemicals, making them more bioaccessible. Additionally, colonic digestion results in release of the bound phenolic compounds, thus enabling them to exert their health effects both locally and systemically upon absorption. Andreasen et al. [10] showed that human gastrointestinal esterase, from the intestinal mucosa and intestinal microbiota, can release ferulic acid and diferulics from cereal bran, thereby potentially contributing to the lower risk of certain cancers (e.g., colon cancer) associated with consumption of whole grains [11].

In wheat kernels, ferulic acid and other phenolic acids provide protection through physical and chemical barriers such as: cross-linking with carbohydrates, antioxidant activities to combat destructive radicals and astringency that deters consumption by insects and animals [5, 8, 9]. The whole grains phenolic compounds function in the body as antioxidants by donating hydrogen atoms to free radicals [5, 9]. The total phenolic content of whole grains corresponds to their total antioxidant capacities. Miller et al. [12] reported the antioxidant capacity of whole grains breakfast cereals to range from 2,200 to 3,500 Trolox Equivalents (TE). In comparison, antioxidant capacity of fruits generally ranged from 600 to 1,700 TE, and vegetables averaged 450 TE. The average antioxidant capacity of a 41 g serving of ready-to-eat whole grains breakfast cereal was 1,120 TE, compared to 380 and 1,020 TE in an average 85 g serving of vegetables and fruits, respectively [12].

A few other phytonutrient and nutrients found in whole grains will be discussed below.

Plant Sterols

Plant sterols and stanols found in whole grains vary in type and amount based on variety of whole grains and the whole grains component. These substances are known to inhibit absorption and increase excretion of cholesterol, thereby regulating blood cholesterol levels. Whole grains consumption may be associated with increased phytosterol intake, therefore increasing whole grains consumption may contribute to cholesterol reduction and cardioprotection [5].

Carotenoids

Carotenoids are another group of compounds found in whole grains [5, 8]. The most common carotenoids, lutein, zeaxanthin, β-cryptoxanthin, β-carotene, and α-carotene, are generally concentrated in the bran or germ portion of whole grains [5, 8]. In addition to providing pigmentation, they play an important role in reproduction and protection of the whole grains, and provide antioxidant and provitamin A activity (β-cryptoxanthin, β-carotene, and α-carotene) in the body.

Vitamin E and Unsaturated Fatty Acids

Vitamin E in the form of tocopherols and tocotrienols is generally concentrated in the germ fraction of the grain [5], and may play an important role in the antioxidant activity of whole grains. β-tocotrienol is the predominant form of vitamin E in whole wheat grain [5]. Whole grains also contain unsaturated fatty acids, mainly oleic and linoleic acid, which are known for their protective role in cardiovascular and other chronic diseases.

Dietary Fiber

Whole grains contain dietary fiber, resistant starch, oligosaccharides, arabinoxylans, and lignans, which have important biological activities and functions [5]. For example, dietary fiber, resistant starch, and oligosaccharides in whole grains may contribute to cholesterol reduction, maintenance of healthy blood glucose and insulin concentrations, improved digestive health, and a lower risk of certain gastrointestinal cancers [5]. Lignans found in whole grains have strong antioxidant and phytoesterogenic effects. Intestinal microflora play a role in converting these plant lignans into the mammalian lignans, enterolactone and enterodiole, which may provide protection against hormone-related cancers, diabetes, and heart disease [5].

Anti-nutrients

Anti-nutrients, i.e., compounds that interfere with nutrient absorption, such as phytic acid, tannins and enzyme inhibitors, are also present in whole grains and may contribute to their overall protective effects [5]. Phytic acid chelates with various metals suppressing redox reactions and associated oxidative damage. Likewise, phytic acid may protect the intestinal epithelium by suppressing the oxidant damage associated with the oxygen radicals produced by the colonic bacteria. Protease inhibitors, phytic acid, phenolics, and saponins present in whole grains have also been suggested to lower risk of certain cancers (e.g., colon and breast cancer). Additionally, phytic acid, lectins, phenolics, amylase inhibitors, and saponins have also been shown to lower plasma glucose, insulin and/or plasma cholesterol and triacylglycerol levels, which could potentially also contribute to the lower risk of chronic diseases associated with whole grains consumption [5].

Whole grains have higher phytonutrient content and antioxidant activity than refined grains. Refining of whole grains may result in losses in vitamins and minerals, total phenolics, flavonoids, ferulic acid, zeaxanthin, lutein, and β-cryptoxanthin [8]. Further research on the health benefits of whole grains phytochemicals, their bioaccessibility, effects of processing on their physiological effects and amounts in whole grains products is warranted.

Whole Grains Dietary Recommendations

The majority of global dietary guidelines recommend the consumption of whole grains; Table 22.3 provides a summary of existing global dietary whole grain guidelines. The 2010 Dietary Guidelines for Americans recommend that individuals should "consume three or more ounce-equivalents of whole grains products per day, with the rest of the recommended grains coming from enriched or whole-grain products. In general, at least half of the grains should come from whole grains" [13]. Similar country-specific guidelines are listed in Table 22.3.

Despite these dietary guidelines, dietary intake data in the United States from the Continuing Survey of Food Intakes (CSFII) and the National Health and Nutrition Examination Survey (NHANES) indicate that the average whole grains intake is 1 serving/d, with 95 % of Americans not meeting their whole grains daily intake recommendations [13–15]. Since the 2005 Dietary Guidelines for Americans, some data show that consumers increased purchases of whole grain foods, particularly ready-to-eat breakfast cereals, breads, and pasta, which maybe in part due to more whole grain products on supermarket shelves [16].

Whole Grains Consumption Patterns

Estimation of an individual's whole grains intake can be obtained in a variety of ways, such as 24-h dietary intake recalls, food records and food frequency questionnaires, with the latter being the most common method used in epidemiological studies. For each of these methods it is important to clearly understand the definition of whole grain foods. Definitions vary between studies and nutrient intake databases. Early studies included whole grain foods such as: whole grains breakfast cereals (containing ≥25 % of whole grains content), dark bread, brown rice, oatmeal, and other specific individual foods such as wild rice, whole wheat crackers, and bran muffins. In addition, added bran and added germ were included in the whole grains category. Based on the definition of a "whole grain food" estimation of whole grains intake maybe easily under- or overestimated. There is a need for standardization of dietary intake assessment methods and nutrient databases to better quantify intake of whole grains.

Whole Grain Intake: Adults

Data from the 1994 to 1996 CSFII, show that US adults consumed 1 serving of whole grains/d (16 g/d) [14]. In this survey, 36 % of the population averaged <1 whole grain serving/d and only 8 % met the recommendation to eat at least 3 servings/d. Similarly, in the Baltimore Longitudinal Study of Aging, Maras et al. [17] observed low whole grains intake in older participants, but women consumed more whole grains per day than men (25 vs. 20 g/d). Data from the 1999 to 2004 NHANES show that adults aged

Table 22.3 Global dietary whole grain guidelines

Country/ organization	Specific recommendation
Australia	The Australian Dietary Guidelines and Guide to Healthy Eating recommend 3–12 servings (dependent upon age, sex, or caloric requirements) of grain (cereal) foods, mostly wholegrain, such as breads cereals, rice, pasta, noodles, polenta, couscous, oats, quinoa, and barley
Austria	The Austrian Food Pyramid ("Die österreichische Ernährungspyramide") recommends consuming 4 servings daily of cereals, bread, pasta, rice, or potatoes (5 servings for active adults and children), preferably whole grain
Canada	The Canadian Food Guide recommends 3–8 servings (age and sex dependent) of grain products per day and advises making at least half of the grain product choices whole grain each day. Further recommendations state to eat a variety of whole grains such as barley, brown rice, oats, quinoa, and wild rice
China	The Chinese Dietary Guidelines and the Diet Pagoda recommend adults to consume 300–500 g (dependent upon energy requirements) of total grains, cereals and legumes daily, among them—at least 50 g/d of coarse grains, including whole grains
Denmark	Denmark's Food Administration uses the Diet Compass and the Dietary 8 (Danish: "Kostkompasset" and "8 kostråd") to recommend consuming 75 g of whole grain a day (for energy requirements of 10 MJ/d). Bread, grains, rice, and pasta should be an essential part of the diet and for older children and adults and 500 g is recommended per day
France	France's Guide of the National Health and Nutrition Program (French: "Guides alimentaires du programme national nutrition-santé") recommends consumption of breads, cereals, and starchy foods at each meal, especially whole grain foods that provide significant amounts of fiber
Germany	The German Nutrition Society's ten guidelines for wholesome diet and food pyramid (German: "Ernährungspyramide"), recommends consuming ample bread, pasta, rice, grain flakes, preferably from whole grain. At least 30 g of daily dietary fiber is recommended, especially from whole-grain products. A high intake of these foods lowers the risk of various nutrition-related diseases
Greece	The Dietary Guidelines for adults in Greece suggest consuming 8 servings of non-refined cereals and products, preferably whole-grain varieties (whole-grain bread, whole-grain pasta, brown rice, etc.)
Iceland	Iceland's Food Circle (Icelandic: Fæðuhringurinn) recommends eating whole grain products and fiber-rich food. Recommendations state to choose more whole grain products rather than refined products because fiber and other beneficial substances for your health remain intact
Ireland	The Irish Heart Foundation's Nutrition Guidelines for Heart Health state that 6 or more servings of starch and complex carbohydrates should be achieved by consuming more wholemeal bread, other cereals, and potatoes. Individuals should eat wholegrain cereal products or potatoes with every meal
India	The Dietary Guidelines for Indians recommends increasing consumption of whole grains, legumes, and nuts to maintain body weight and body composition
Latvia	The Latvian Health Ministry recommends consumption of 4–6 servings of cereals per day especially whole grains such as fiber-rich whole-grain products (bread, pasta, oatmeal porridge) to reduce the risk of diseases
Mexico	Mexico's Department of Nutrition and Health Promotion uses the Plate of Good Eating (Spanish: "El Plato del Bien Comer") to recommend. Consumption of cereals should be recommended, preferably whole grains without added sugar. Their fiber and nutrients should be highlighted. Whole grains should be eaten with every meal, with legume seeds
Norway	The Health Directorate of Norway's Key Advice for a Healthy Diet (Norwegian: "Nøkkelråd for et sunt kosthold") suggests increasing intake of whole grain products and cereals each day. The whole-grain products should together provide 70–90 g of wholemeal flour or whole grain per day
Oman	The Omani Guide to Healthy Eating recommends choosing whole grains and cereals, and consuming potatoes, with their skin. For an average diet of 2,000 kcal, 2–3 servings of whole grains daily is advised
Singapore	The Dietary Guidelines for Adult Singaporeans and Healthy Diet Pyramid recommend eating sufficient amounts of grains especially whole grains. Out of the 5–7 servings of rice and alternatives, adults should consume 2–3 servings of whole-grain food daily
Sweden	Sweden's Food Administration recommends selecting primarily whole grains when eating bread, cereals, grains, pasta, and rice. The recommended amount of whole grains is approximately 70–90 g/d

(continued)

Table 22.3 (continued)

Country/ organization	Specific recommendation
Switzerland	The Swiss Society for Nutrition recommends states that each main meal should be served with 1 starch-rich side dish (i.e., 3 portions a day, 1 portion = 75–125 g of bread or 60–100 g of pulses [raw weight] for instance lentils/chick peas or 180–300 g of potatoes or 45–75 g of pasta/rice/flakes/corn/other grains [raw weight]), including at least two portions of whole grain products
United Kingdom	The National Health Service's Eatwell Plate recommends to eat plenty of bread, rice, potatoes, pasta, and other starchy foods (shown as one-third of a plate) and choose whole grain varieties whenever you can
United States of America	The 2010 Dietary Guidelines for Americans states to consume 3 or more ounce-equivalents of whole grain products per day, with the rest of the recommended grains coming from enriched or whole grain products (at the 2,000 kcal intake level). Consume at least half of all grains as whole grains. Increase whole-grain intake by replacing refined grains with whole grains
World Health Organization	The World Health Organization and the Food and Agriculture Organization of the United Nation recommend increasing consumption of whole grains as a strategy to prevent diet-related chronic diseases. The WHO/FAO rate the strength of evidence for whole grain consumption and decreased risk of cardiovascular disease and diabetes is rated as "probable"

Note: References to the individual country guidelines are available upon request

19–50 and 51+ y consumed a mean of 0.63 and 0.77 servings of whole grains/d, respectively [18]. More than 70 % of US adults in the 1999–2004 NHANES failed to consume the mean number of servings of whole grains. Only 4.8 % and 6.6 % of individuals in these age groups consumed the recommend 3 or more daily servings of whole grains (48 g/d) [18].

Similar to the intake patterns observed among the adult US population, Thane et al. [19] observed that whole grain intake of British adults was low in 1986–1987, which became even lower over the subsequent decades. The median whole grain intake of British adults in 1986–1987 was 16 vs. 14 g/d in 2000–2001. In 1986–1987, 25 % of adults reported no whole grain intake and 77 % had less than three 16 g amounts of whole grain intake/d [19]. The improvements in 2000–2001 were minimal, with 29 % consuming no whole grains and 84 % eating less than the recommended levels. Foods with less than 51 % whole grain content provided 18 % of whole grain intake in 1986–1987 and 27 % in 2000–2001. Whole grain intake was observed to increase with age during both survey years, but intakes were higher in 1986–1987 vs. 2000–2001 and whole grain intake was significantly lower among adults with manual occupations and smokers, indicative of low socioeconomic status. In 1986–1987, whole grain intake was lowest among adults living in Scotland, followed by those living in Wales; these differences were not observed in 2000–2001 due to decline in intakes in regions other than Scotland and Wales [19].

Unlike the adults in the UK, Egeberg et al. [20] in the Diet, Cancer and Health Study among a cohort of adults in Denmark, observed higher whole grain intakes; the median consumption of whole grain foods was 140 g/d among men and 119 g/d among women, with rye bread contributing to 63 % and 55 % of the total whole grain food consumption. A similar trend was observed among a Scandinavian cohort [21]. Kyrø et al. [21] recently reported the whole grain intake in Norway, Sweden, and Denmark based on 1995–2000 subset of the adult (30–65 y) Scandinavian cohort "HELGA." The mean whole grain intake among this cohort was well below the recommended level, with only 16 % of Danish men, 35 % of Norwegian women consuming at least the recommended intake of whole grains. Among women, the median intake of whole grain products was 114 g/d in Norway and 108 g/d in Denmark, whereas the intake was much lower in Sweden at 64 g/d. For women, the median intake of whole grain in absolute amount (i.e., grams of calculated whole grain content) was highest in Norway (44 g/d), and lower in Sweden (35 g/d) and Denmark (31 g/d). Among men, the intake of whole grain products was higher in Denmark (138 g/d) compared to lower intake in Sweden (79 g/d), while the intake of whole grain in absolute amount was highest in Sweden (49 g/d) compared to Denmark (41 g/d).

Whole Grain Intake: Children and Adolescents

Similar to the adult UK population, using the 1997 National Diet and Nutrition Survey, Thane et al. [22] examined the whole grain intake of 4–18 y olds in Great Britain. Median whole grain intake was 7 g/d with a mean intake of 13 g/d among this young population, with 27 % of this population not consuming any whole grains. Although no differences in intake were observed by age, gender, region, or season, whole grain intake was significantly lower among young people whose head of household had a manual occupation. A similar lower intake pattern was observed among German children [23]. In the Dortmund Nutritional and Anthropometric Longitudinally Designed Study, mean whole grain intake of German children and adolescents was between 20 and 33 g/d, which highest intake among 13–18 y olds, with the percentage of whole grain consumers decreasing with age [23]. In this study, 19 % of the total cohort recorded no whole grain intake, 16 % among the 2–5 y olds, 17 % in 6–12 y olds and 24 % in 13–18 y olds [23].

Among American adolescents in project EAT (Eating Attitudes of Teenagers), Larson et al. [24] observed mean whole grains intake of 0.59 servings/d among males and 0.61 servings/d among females, with young adults consuming 0.68 servings/d (males) and 0.58 servings/d (females), which were all below the whole grain dietary recommendations. In 1999, only 11 % of adolescent males and 13 % of adolescent females in this cohort reported consuming more than 1 serving/d of whole grains [25]. Whole grain intake was observed to be lowest among Native American and white youth and youth of high socioeconomic status. Asian male and female adolescents were most likely to report consumption of more than 1 serving of whole grain/d. During the transition from middle to late adolescents, an increase by 0.14 servings of whole grains/d was observed among males and by 0.09 servings/d was observed among females. No significant changes were observed in whole grain intakes during the transition from early to middle adolescence. Availability of whole grain bread at home was observed to be positively associated with whole grain intake among both age cohorts. Frequency of breakfast consumption, greater involvement in food preparation and shopping, greater preferences for taste of whole grain bread and self-efficacy to consume greater than 3 daily whole grain servings were also positively associated with whole grain intake. Additionally, among male adolescents, family meal frequency and peer support for healthful eating were observed to be positively associated with whole grain intake. Youth who consumed fast food more frequently consumed fewer servings of whole grains.

Nationally representative US data from 1994 to 1996 show that the average whole grain intake ranged from 0.8 servings/d for preschool-aged children to 1.0 servings/d for adolescents [26]. Preschool-aged and school-aged children in households with an income ≤150 % of federal poverty threshold were more likely to consume ≤1 serving of whole grains/d. Adolescent females were 1.37 times more likely than males to consume ≤1 serving of whole grains/d. Likewise, based on the NHANES 1999–2004 data, among children 2–5 and 6–12 y, O'Neil et al. [27] observed that the whole grain intake was 0.45, 0.59, and 0.63 servings/d for children and adolescents at the age of 2–5, 6–12, and 13–18 y, respectively.

Whole Grain Intake: Overall

The 2010 US Dietary Guidelines [13] noted that Americans of all ages consume fewer whole grains than recommended. Currently, Americans consume less than 20 % of the recommended intakes for whole grains. More than 95 % of all age-sex groups fail to consume the minimum recommended amounts of whole grains. Median whole grain intakes for adult US men and women are 0.50 and 0.47 ounce-equivalents (oz eq)/d, respectively, compared to the recommended minimum of 3 oz eq/d (one-half of total grains). Children, aged 1–3 and 4–8 y, also have low intakes of whole grains, with median intakes of 0.37 and 0.41 oz eq/d, respectively, less than the recommended 1.5 or 2 oz eq/d, respectively. Median whole grain intakes are 0.48 and 0.34 oz eq/d for boys and girls, aged 9–13 y, respectively, compared to recommended levels of 3 and 2.5 oz eq/d, respectively; and 0.26 and 0.33 oz eq/d, respectively, for adolescent boys and girls, aged 14–18 y, compared to the recommended level of 3.5 and 3 oz eq/d, respectively [13].

Approaches to Increase Whole Grain Intake

Recently, Keast et al. [28] investigated the influence of substituting whole grain for refined grain ingredients on whole grain consumption in children. Refined wheat flour contained in foods such as pizza crust, pasta, breads, and other baked goods was replaced with whole wheat flour and white rice was replaced with brown rice at levels that were acceptable by school age children (15–50 % replacement). This modeling showed that whole grain intake can be increased by 1.7 oz eq/d (range 0.5–2.2 oz eq/d), contributing to 28 % of total grain intake. The major sources of whole grain intakes were breads/rolls (28 %), pizza (14.2 %), breakfast cereals (11 %), rice/pasta (10.6 %), quick breads such as tortillas, muffins, and waffles (10.8 %), other baked goods (9.9 %) and grain-based savory snacks other than popcorn (7.3 %). These data show that substitution of refined grain ingredients for whole grains may enable reduction in disparities between demographic subgroups of children and teens. Furthermore, Sadeghi and Marquart [29] showed that graham crackers that were partially whole grains (5 g/serving) had a higher rate of acceptance by elementary school children compared to 100 % whole grains graham crackers (26 g/serving). These studies identify ways to incorporate whole grains into commonly consumed foods and ultimately increase whole grain consumption in the general population.

There are a number of methodological challenges to developing a standardized approach for accurately quantifying consumption of whole grain foods. The greatest challenge may be the handling of foods that contain a combination of whole and non-whole grains ingredients. A growing number of partial whole grain foods appear to be entering the marketplace and therefore this methodological issue must be addressed to more accurately assess dietary intake of whole grain foods. Low consumption of whole grain is attributed to multiple factors, including lack of understanding of the health benefits, inability to identify or purchase whole grains, inability to incorporate whole grains in the lifestyle, lack of familiarity with preparation methods, higher price of whole grain products and perceived poor taste and texture of whole grain products. Ongoing efforts are necessary to more accurately estimate whole grains intake to gain a better representation of consumption patterns at individual and population level and to find ways to promote regular daily consumption of whole grains.

Dietary Sources of Whole Grains

Table 22.4 provides a brief overview of common food sources of whole grains in the United States and Europe. In the United States, major contributors to whole grain dietary intakes are grain-based foods, providing almost one-third of whole grains servings [14, 25, 30]. In the United States, the 1994–1996 CSFII data show that yeast breads and breakfast cereals each contributed one-third of the whole grain servings, grain-based snacks provided about one-fifth and less than one-tenth came from quick breads, pasta, rice, cakes, cookies, pies, pastries and miscellaneous grains [14]. Whole grains made up more than half of the grain servings from breakfast cereals, while they made up a relatively small percentage of the total yeast bread servings. In this study, whole grain consumers were more likely to meet the food guide pyramid recommendations for grain, fruit, and

Table 22.4 Examples of common food sources of whole grains in the global diet

Country	Common food sources of whole grains
United States	Breakfast cereals (ready-to-eat and hot)
	Yeast breads
	Grain-based snacks (e.g., crackers, pretzels, popcorn, corn chips)
	Common grains consumed: wheat, corn, oats, barley, rice
United Kingdom	Breakfast cereals
	Wholemeal bread
	Common grains consumed: wheat
Germany	Muesli
	Bread
	Grain (e.g., flour, cakes, popcorn)
Scandinavia	Breakfast cereals
	Whole grain bread
	Whole grain crisp bread
	Common grains consumed: rye, wheat, oats

dairy food groups. Albertson and Tobelman [31] observed that the average number of eating occasions of all grain products in the diets of Americans was <3/d for all age groups, with 40 % of individuals consuming grain products 2–2.99 times/d.

Data from 1994 to 1996 CSFII show that only ten foods accounted for most (94.7 %) of the whole grain intake among those aged 2–18 y [26]. Ready-to-eat cereals (RTEC), corn and other chips, and yeast breads were found to be the major food sources of whole grains accounting for 30.9 %, 21.7 % and 18.1 % of whole grain intake respectively among those aged 2–18 y.

Major sources of whole grain foods for adolescents in Project EAT were yeast breads, popcorn, and RTEC [25]. Five year secular trends showed that the mean percentage of daily whole grain servings from yeast breads increased significantly, by 12.5 % among males and by 11.1 % among female adolescents. A decrease in daily whole grain servings from hot breakfast cereals (−6.4 %) among male adolescents, and a decrease in daily whole grain servings from cooked grains (−3 %) among female adolescents was observed. These data suggest that more targeted approaches may be required to increase the whole grain consumption among adolescents.

In 2001–2002, the leading food sources of whole grains in the American diet were RTEC (28.7 %), yeast breads (25.3 %), hot cereals (13.7 %), popcorn (12.4 %), and crackers (6.4 %), with these food categories accounting for close to 90 % of all whole grains consumed [30]. Likewise, among older participants in the Baltimore Longitudinal Study of Aging, RTEC, hot breakfast cereals, multigrain bread, and whole wheat bread were the main contributors to the whole grain intakes [17]. The most common types of whole grains in the American diet are wheat, corn, oats, barley, and rice, with wheat being the most prominent grain consumed on a daily basis [13].

Among adult British population, whole grain breakfast cereals and wholemeal bread contributed to 28 % and 48 %, respectively, to the whole grain intake in 1986–1987 and 45 % and 31 % in 2000–2001, respectively [19]. Among younger individuals in Great Britain, the main sources of whole grains in the diet of these young individuals were breakfast cereals (56 %) and breads (25 %) [22]. The majority of the whole grains intake was in the form of wheat.

In the adult, 30–65 y, Scandinavian cohort "HELGA," main source of whole grains, among the Norwegian and Danish participants, was whole grain bread (>80 %), whereas breakfast cereals, whole grain bread, whole grain crisp bread were all major sources of whole grains intake among the Swedes [21]. Rye was by far the most important source of whole grains in Denmark (>70 %), whereas whole grain wheat was the most important source among Norwegian women (72 %). In Sweden, both rye and wheat were important sources of whole grains. Additionally, in Denmark and Sweden, oats also contributed substantially to the whole grains intake (15 %), while they only contributed to 6 % of whole grains intake among Norwegian women.

In the Dortmund Nutritional and Anthropometric Longitudinally Designed Study, among German children and adolescents, of all the foods consumed by this cohort, Muesli had the highest whole grain density, followed by bread and grain (flour, cakes, popcorn, etc.); a trend towards increasing whole grain RTEC at the expense of bread consumption was also observed [23].

The current available data suggests that despite a wide range of whole grain products in the market place, there a few top categories, such as breakfast cereals and breads, that continue to be the main sources of whole grains in the diets of the global population. The type of whole grains consumed varies by region; with a mix of grains being most commonly consumed. Given the low daily intake of whole grains observed in different population groups, there is a need to increase the availability of whole grain food options in the marketplace.

Whole Grains Impact on Diet Quality

Whole grains are one of the major food sources of carbohydrates; whole grains are also good sources of protein, fiber, unsaturated fatty acids, vitamins, minerals, and numerous phytonutrients. Inadequate intakes of whole grains contribute to the lack of adequate intakes of certain nutrients like magnesium and fiber across all age groups. Data from the 1994 to 1996 CSFII survey in the United States show that individuals who consume whole grain foods have significantly better intakes of nutrients and other dietary components than nonconsumers [14]. Compared to nonconsumers, whole grain consumers had significantly higher intakes of carbohydrate and protein (as percent of food energy), vitamins and minerals, and significantly lower intakes of total fat, saturated fat, and added sugars [14]. Intakes of vitamin A, vitamin E, vitamin C, thiamin, riboflavin, niacin, vitamin B6, folate, calcium, magnesium, iron, zinc, and dietary fiber per 1,000 kcal were significantly higher than non-whole grain consumers. Whole grain consumers were more likely to be male, older, white, nonsmokers, to use dietary supplements and to have higher household income, education, and physical activity [14].

Data from the 1999 to 2004 NHANES show that adults aged 19–50 and 51+ y who consumed the most servings of whole grains had higher diet quality and intake of energy, fiber, polyunsaturated fatty acids and most micronutrients, including potassium, a common micronutrient deficient in most diets, while intakes of total sugars, added sugars, saturated fatty acids, monounsaturated fatty acids, cholesterol, sodium, and vitamin B12 were significantly lower [18]. Mean Healthy Eating Index (HEI) scores were significantly higher in those with higher consumption of whole grains than in those consuming fewer servings. Increasing consumption of whole grains was associated with improved diet quality and nutrient intake in children and adolescents. Among United States children 2–5 and 6–12 y, the HEI and intakes of energy, fiber, vitamin B6, folate, magnesium, phosphorus, and iron were significantly higher in those consuming greater than 3 servings of whole grains/d, while intakes of protein, total fat, saturated fat, monounsaturated fat, and cholesterol levels were lower [27].

Compared to standard hypocaloric diets, those diets with whole grains cereals contributed to higher intakes of total fiber (21.2 vs. 17.5 g/d), insoluble fiber (14.9 vs. 11.3 g/d), magnesium (301.1 vs. 247.6 mg/d), vitamin B6 (2.12 vs. 1.91 mg/d) and lower intakes of saturated fat (13.7 vs. 14.34 g/d) [32]. Magnesium intakes were closer to recommendations and vitamin B6 were adequate (>1.3 mg/d); in RTEC, magnesium is from the whole grain ingredients and vitamin B6 is from the whole grain ingredients and fortification. Additionally, most studies also show that individuals who consume a diet high in whole grains tend to have healthier lifestyles, including increased physical activity, reduced smoking, and consumption of foods generally considered to be healthy [32]. The Diet, Cancer and Health Study [20] observed that consumption of whole grain foods was positively associated with cycling, taking dietary supplements, high school education and negatively associated with intake of alcohol, BMI and smoking, suggesting that whole grain intake may be indicative of healthy lifestyle practices. Among this cohort whole grain consumption was positively associated with other dietary factors, mainly intake of processed meat and vegetables and negatively associated with intake of red meat and refined grain products [20].

The 2010 US dietary guidelines technical advisory committee chose to use food pattern modeling to determine the impact on intake of folate and other nutrients if all recommended grains were selected as whole grains rather than half whole and half enriched refined grains [13]. In this exercise consumption of all grains as whole grains, without including any fortified whole grain products, was observed to contribute to lower dietary folate and iron intake to levels less than adequate amounts for individuals in population groups who may be at high risk for inadequate intakes of these nutrients (e.g., children 2–8 y, girls 14–18 y, women with low to moderate energy needs, men >50 y). Thus individuals are encouraged to consume most of their grains as whole grains, and when doing so, they are encouraged to select some whole grains products that have been fortified with folic acid and other key micronutrients.

Whole Grain Foods in the Market Place

Hectic, unhealthy lifestyles and poor dietary habits have heightened concerns about obesity, cardiovascular and other chronic diseases, leading to changes in food choices. Rising consumer awareness about the dietary significance of whole grain foods is one of the major factors contributing to higher consumption of such foods. The shift in consumer perception and trends could also be attributed to greater investments towards consumer education and availability of products with improved tastes and flavors.

Global trends indicate that the number of whole grain products in the marketplace have increased dramatically. As increasing numbers of consumers become more health and fitness conscious, the global market for whole grain foods is forecast to reach over US$24 billion by the year 2015 [33, 34]. Claims relating to the benefits of whole grains were the most sought after health claims on food packages, followed by claims about dietary fiber. The number of new whole grain products increased nearly 20-fold between 2000 and 2010; in 2010, 3,272 new products formulated with whole grains were introduced [34]. There is an increasing trend in the restaurant industry featuring exotic rice, spelt, faro, amaranth and other ancient grains in everything from salads, pancakes, pizza crusts, soups, cocktails, and desserts [34]. A 2009 survey found that 36 % of adults were eating more whole grains mainly because they enjoyed the taste, which is a dramatic increase since 2006 (13 %), potentially due to the broader availability of whole grain products, improvements in technologies to help deliver tasty and healthy whole grain products and the greater acceptance of the health and nutrition benefits of whole grains [34].

There was marginal impact of the global economic slowdown on whole grain foods market [34]. Bread, a baked food considered an essential staple in most of the economies, withstood the recessionary impact. In 2009, global bread products sales (at retail level) increased by over 5 % over 2008. Breakfast cereals also fared well during the crisis period, and were preferred over other morning foods owing to the nutritional content and affordable pricing. Usage of whole grains and reduction of fat and sugar levels were some of the most popular claims in the new products rolled out during the year. Blended flours are gaining popularity among consumers wishing to consume whole grains but averse to the taste, flavor, and texture of whole grain flour or products.

The US constitutes the largest regional market for whole grain foods [34]. The dominance of the US market is likely to continue, backed by increased adoption of rice, wheat, barley as well as various specialty grains among consumers and by health professionals. Europe and Asia-Pacific follow the United States as the next major markets. Demand for whole grain and high fiber foods in Asia-Pacific is projected to grow at a rate of 6 % through 2015. The increasing health orientation among upper and middle class consumers in developing regions may drive the demand for nutritious, value-added products, such as whole grain foods.

Conclusions

Existing evidence indicates that whole grains have a beneficial health effect. A recent evidence review by Gil et al. [7] observed that individuals who consume 3 or more servings of whole grain foods/d have a 20–30 % lower risk of cardiovascular disease than individuals who ingest lower amounts of whole grain foods; this level of protection was not observed with ingestion of refined grains and intake of fruits and vegetables. Likewise, higher intake of whole grain foods was associated with a 20–30 % reduction in risk of type 2 diabetes. Additionally, regular consumption of whole grain foods was associated with protection against risk of colorectal cancer and polyps, other cancers of the digestive tract, and other hormone-related and pancreatic cancers [7]. Gil et al. [7] conclude that regular consumption of whole grains may contribute to reduction of risk factors related to noncommunicable chronic diseases. The essential macro and micronutrients, along with the phytonutrients present in whole grains, synergistically contribute to their beneficial effects. Current evidence lends credence to the recommendations to incorporate whole grains foods into a healthy diet and lifestyle program and they need to become more common place in the diets of all individuals.

References

1. American Association of Cereal Chemists International. AACC members agree on definition of whole grain. Available at: http://www.aaccnet.org/initiatives/definitions/Pages/WholeGrain.aspx. Accessed 17 Dec 2011.
2. United States Food and Drug Administration. FDA provides guidance on "whole grain" for manufacturers. Available at: http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/2006/ucm108598.htm. Accessed 17 Dec 2011.
3. United Kingdom Food Standards Agency, National Health Services Eatwell Plate. Available at: http://www.nhs.uk/Livewell/Goodfood/Pages/starchy-foods.aspx. Accessed 17 Dec 2011.
4. Health Grain Forum. Whole grain definition. Available at: http://www.healthgrain.org/webfm_send/44. Accessed 17 Dec 2011.
5. Okarter N, Liu RH. Health benefits of whole grain phytochemicals. Crit Rev Food Sci Nutr. 2010;50:193–208.
6. U.S. Department of Agriculture, Agricultural Research Service. 2009. USDA national nutrient database for standard reference, Release 22. Nutrient Data Laboratory home page. http://www.ars.usda.gov/ba/bhnrc/ndl. Accessed 17 Dec 2011.
7. Gil A, Ortega RM, Maldonado J. Wholegrain cereals and bread: a duet of the Mediterranean diet for the prevention of chronic diseases. Public Health Nutr. 2011;14:2316–22.
8. Adom KK, Sorrells ME, Liu RH. Phytochemicals and antioxidant activity of milled fractions of different wheat varieties. J Agric Food Chem. 2005;53: 2297–306.
9. Adom KK, Liu RH. Antioxidant activity of grains. J Agric Food Chem. 2002;50:6182–7.
10. Andreasen MF, Kroon PA, Williamson G, et al. Intestinal release and uptake of phenolic antioxidant diferulic acids. Free Radic Biol Med. 2001;31(3):304–14.
11. Chatenoud L, Tavni A, La Vecchia C, et al. Whole grain food intake and cancer risk. Int J Cancer. 1998;77:24–8.
12. Miller HE, Rigelhof F, Marquart L, et al. Antioxidant content of whole grain breakfast cereals, fruits and vegetables. J Am Coll Nutr. 2000;19:312S–9.
13. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans. 2010. Available at: http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm. Accessed 17 Dec 2011.
14. Cleveland LE, Moshfegh AJ, Albertson AM, et al. Dietary intake of whole grains. J Am Coll Nutr. 2000;19(3 Suppl):331S–8.
15. Good CK, Holschuh NM, Albertson AM, et al. Whole grain consumption and body mass index in adult women: an analysis of NHANES 1999–2000 and the USDA pyramid serving database. J Am Coll Nutr. 2008;27:80–7.
16. Mancino L, Kuchler F, Leibtag E. Getting consumers to eat more whole-grains: the role of policy, information, and food manufacturers. Food Policy. 2008;33: 489–96.
17. Maras JE, Newby PK, Bakun PJ, et al. Whole grain intake: the Baltimore longitudinal study of aging. J Food Compost Anal. 2009;22:53–8.
18. O'Neil CE, Nicklas TA, Zanovec M, et al. Whole-grain consumption is associated with diet quality and nutrient intake in adults: the National Health and Nutrition Examination Survey, 1999–2004. J Am Diet Assoc. 2010;110:1461–8.
19. Thane CW, Jones AR, Stephen AM, et al. Comparative whole-grain intake of British adults in 1986–7 and 2000–1. Br J Nutr. 2007;97:987–92.
20. Egeberg R, Frederiksen K, Olsen A, et al. Intake of wholegrain products is associated with dietary, lifestyle, anthropometric and socio-economic factors in Denmark. Public Health Nutr. 2009;12:1519–30.
21. Kyrø C, Skeie G, Dragsted LO, et al. Intake of whole grain in Scandinavia: intake, sources and

compliance with new national recommendations. Scand J Public Health. 2012;40(1):76–84. doi: 10.1177/1403494811421057.
22. Thane CW, Jones AR, Stephen AM, et al. Whole-grain intake of British young people aged 4–18 years. Br J Nutr. 2005;94:825–31.
23. Alexy U, Zorn C, Kersting M. Whole grain in children's diet: intake, food sources and trends. Eur J Clin Nutr. 2010;64:745–51.
24. Larson NI, Neumark-Sztainer D, Story M, Burgess-Champoux T. Whole-grain intake correlates among adolescents and young adults: findings from Project EAT. J Am Diet Assoc. 2010;110:230–7.
25. Burgess-Champoux TL, Larson NI, Neumark-Sztainer DR, et al. Longitudinal and secular trends in adolescent whole-grain consumption, 1999–2004. Am J Clin Nutr. 2010;91:154–9.
26. Harnack L, Walters SA, Jacobs Jr DR. Dietary intake and food sources of whole grains among US children and adolescents: data from the 1994–1996 continuing survey of food intakes by individuals. J Am Diet Assoc. 2003;103:1015–9.
27. O'Neil CE, Nicklas TA, Zanovec M, et al. Consumption of whole grains is associated with improved diet quality and nutrient intake in children and adolescents: the National Health and Nutrition Examination Survey 1999–2004. Public Health Nutr. 2011;14:347–55.
28. Keast DR, Rosen RA, Arndt EA, Marquart LF. Dietary modeling shows that substitution of whole-grain for refined-grain ingredients of foods commonly consumed by US children and teens can increase intake of whole grains. J Am Diet Assoc. 2011;111:1322–8.
29. Sadeghi L, Marquart L. Whole grain snack intake in an after-school snack program: a pilot study. J Food Serv. 2009;20:71–80.
30. Bachman JL, Reedy J, Subar AF, Krebs-Smith SM. Sources of food group intakes among the US population, 2001–2002. J Am Diet Assoc. 2008;108:804–14.
31. Albertson AM, Tobelmann RC. Consumption of grain and whole-grain foods by an American population during the years 1990 to 1992. J Am Diet Assoc. 1995;95:703–4.
32. Melanson KJ, Angelopoulos TJ, Nguyen VT, et al. Consumption of whole-grain cereals during weight loss: effects on dietary quality, dietary fiber, magnesium, vitamin B-6, and obesity. J Am Diet Assoc. 2006;106:1380–8.
33. PRWeb. Global whole grain and high fiber foods market to reach US $24 billion by 2015. http://www.prweb.com/printer/8041782.htm. Accessed 17 Dec 2011.
34. Sloan E. Consumers go with the grain. Food Technol. 2011;65:18.

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