JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
© 2015 BY THE AMERICAN COLLEGE OF CARDIOLOGY FOUNDATION
PUBLISHED BY ELSEVIER INC.

VOL. 66, NO. 14, 2015
ISSN 0735-1097/$36.00
http://dx.doi.org/10.1016/j.jacc.2015.07.050

THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Food Consumption and its Impact on Cardiovascular Disease: Importance of Solutions Focused on the Globalized Food System

A Report From the Workshop Convened by the World Heart Federation

Sonia S. Anand, MD, PhD,*† Corinna Hawkes, PhD,‡ Russell J. de Souza, ScD, RD,§ Andrew Mente, PhD,† Mahshid Dehghan, PhD,† Rachel Nugent, PhD,‖ Michael A. Zulyniak, PhD,* Tony Weis, PhD,¶ Adam M. Bernstein, MD,# Ronald M. Krauss, MD,** Daan Kromhout, MPH, PhD,†† David J.A. Jenkins, MD, PhD, DSc,‡‡§§ Vasanti Malik, ScD,‖‖ Miguel A. Martinez-Gonzalez, MPH, MD, PhD,¶¶ Dariush Mozaffarian, MD, DrPH,## Salim Yusuf, MD, DPhil,† Walter C. Willett, MD, DrPH,¶¶ Barry M. Popkin, PhD***

ABSTRACT

Major scholars in the field, on the basis of a 3-day consensus, created an in-depth review of current knowledge on the role of diet in cardiovascular disease (CVD), the changing global food system and global dietary patterns, and potential policy solutions. Evidence from different countries and age/race/ethnicity/socioeconomic groups suggesting the health effects studies of foods, macronutrients, and dietary patterns on CVD appear to be far more consistent though regional knowledge gaps are highlighted. Large gaps in knowledge about the association of macronutrients to CVD in low- and middle-income countries particularly linked with dietary patterns are reviewed. Our understanding of foods and macronutrients in relationship to CVD is broadly clear; however, major gaps exist both in dietary pattern research and ways to change diets and food systems. On the basis of the current evidence, the traditional Mediterranean-type diet, including plant foods and emphasis on plant protein sources provides a well-tested healthy dietary pattern to reduce CVD. (J Am Coll Cardiol 2015;66:1590–614) © 2015 by the American College of Cardiology Foundation.

From the *Department of Medicine, McMaster University, Hamilton, Ontario, Canada; †Population Health Research Institute, Hamilton Health Sciences and McMaster University, Hamilton, Ontario, Canada; ‡Centre for Food Policy, City University, London, United Kingdom; §Department of Clinical Epidemiology and Biostatistics, McMaster University, Hamilton, Ontario, Canada; ‖Department of Global Health, University of Washington, Seattle, Washington; ¶Department of Geography, University of Western Ontario, London, Ontario, Canada; #Center for Lifestyle Medicine, Cleveland Clinic, Lyndhurst, Ohio; **Children's Hospital Oakland Research Institute, Oakland, California; ††Division of Human Nutrition, Wageningen University, Wageningen, the Netherlands; ‡‡Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada; §§Clinical Nutrition & Risk Factor Modification Center, St. Michael's Hospital, Toronto, Ontario, Canada; ‖‖Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts; ¶¶Departamento de Medicina Preventiva y Salud Publica, Universidad de Navarra-CIBEROBN, Pamplona, Spain; ##Friedman School of Nutrition Science & Policy, Tufts University, Boston, Massachusetts; and the ***Department of Nutrition, School of Public Health, University of North Carolina, Chapel Hill, North Carolina. Dr. de Souza has served as an external resource person on trans and saturated fats to the World Health Organization's Nutrition Guidelines Advisory Group. Dr. Bernstein began working at Rally Health in April 2015. Dr. Krauss has received grant support from the U.S. National Dairy Council, the Dairy Research Institute, the Almond Board of California, and Quest Diagnostics; and has served as a consultant for Quest Diagnostics. Dr. Jenkins has served on the scientific advisory boards of Unilever, Sanitarium Company, California Strawberry Commission, Loblaw Supermarket, Herbal Life International, Nutritional Fundamental for Health, Pacific Health Laboratories, Metagenics, Bayer Consumer Care, Orafti, Dean Foods, Kellogg's, Quaker Oats, Procter & Gamble, Coca-Cola, NuVal Griffin Hospital, Abbott, Pulse Canada, Saskatchewan Pulse Growers, and the Canola Council of Canada; has received honoraria for scientific advice from the Almond Board of California, the International Tree Nut Council Nutrition Research and Education Foundation,

There is much controversy surrounding the optimal diet for cardiovascular health. Data relating diet to cardiovascular diseases (CVDs) has predominantly been generated from high-income countries (HIC), but >80% of CVD deaths occur in low- and middle-income countries (LMIC). Relatively sparse data on diet and CVD exist from these countries though new data sources are rapidly emerging (1,2). Noncommunicable diseases are forecasted to increase substantially in LMIC because of lifestyle transitions associated with increasing urbanization, economic development, and globalization. The Global Burden of Disease study cites diet as a major factor behind the rise in hypertension, diabetes, obesity, and other CVD components (3). There are an estimated >500 million obese (4,5) and close to 2 billion overweight or obese individuals worldwide (6). Furthermore, unhealthy dietary patterns have negative environmental impacts, notably on climate change.

Poor quality diets are high in refined grains and added sugars, salt, unhealthy fats, and animal-source foods; and low in whole grains, fruits, vegetables, legumes, fish, and nuts. They are often high in processed food products—typically packaged and often ready to consume—and light on whole foods and freshly prepared dishes. These unhealthy diets are facilitated by modern food environments, a problem that is likely to become more widespread as food environments in LMIC shift to resemble those of HIC (5,7,8).

In this paper, we summarize the evidence relating food to CVD, and the powerful forces that underpin the creation of modern food environments—what we call the global food system—to emphasize the importance of identifying systemic solutions to diet-related health outcomes. We do this in the context of increasing global attention to the importance of improving food systems by the international development and nutrition community (9–11). Although the "food system" may seem remote to a clinician sitting in an office seeing a patient, its impact on the individuals they are trying to treat are very real. This paper is on the basis of a World Heart Federation international workshop to review the state of knowledge on this topic. This review of diet, dietary patterns, and CVD is not on the basis of new systematic reviews or meta-analyses but represents a careful review of many published meta-analyses, seminal primary studies, and recent research by the scholars who participated in the Consensus conference.

This paper presents: 1) an overview of the development of the modern, globalized food system and its implications for the food supply; 2) a consensus on the evidence relating various macronutrients and foods to CVD and its related comorbidities; and 3) an outline of how changes to the global food system can address current diet-related public health problems, and simultaneously have beneficial impacts on climate change.

ABBREVIATIONS AND ACRONYMS

CHD = coronary heart disease
CI = confidence interval
CVD = cardiovascular disease
GI = glycemic index
GL = glycemic load
HDL-C = high-density lipoprotein cholesterol
HIC = high-income countries
LDL-C = low-density lipoprotein cholesterol
LMIC = low- and middle-income countries
MI = myocardial infarction
OR = odds ratio
RCT = randomized controlled trial
RR = relative risk
SSB = sugar-sweetened beverage
T2DM = type 2 diabetes mellitus

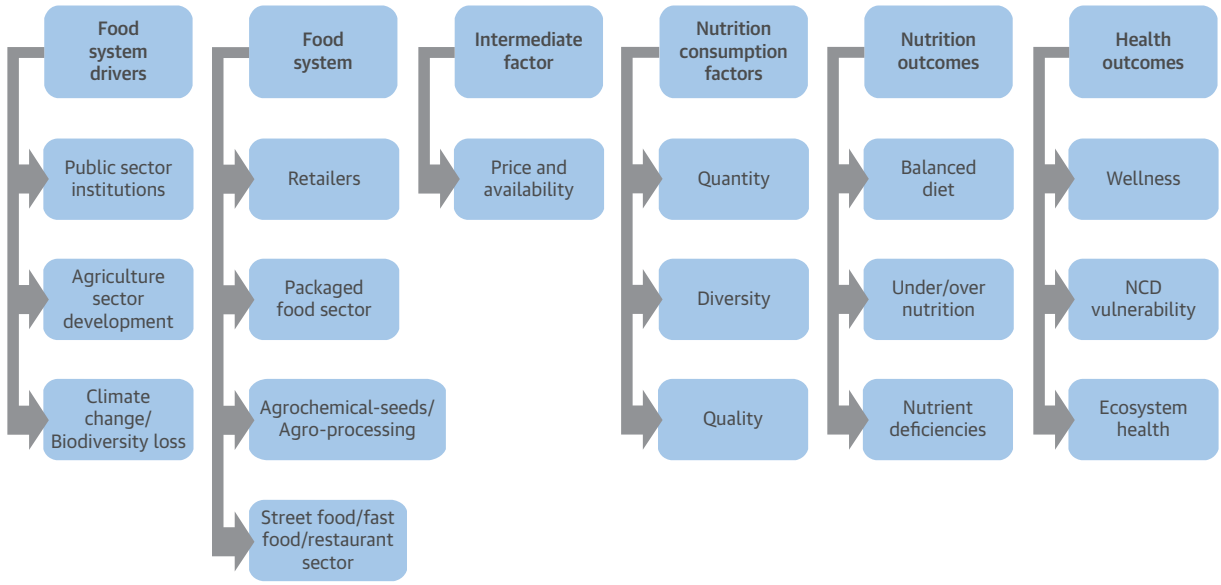
THE CHANGING FOOD SYSTEM AND FOOD SUPPLY AND IMPLICATIONS FOR DIETS AND THE ENVIRONMENT

THE DEVELOPMENT OF THE MODERN, GLOBALIZED FOOD SYSTEM. Food systems were once dominated by local production for local markets, with relatively little processing before foods reached the household (Online Appendix, Box 1) (12). In contrast, the modern

Barilla, Unilever Canada, Solae, Oldways, Kellogg's, Quaker Oats, Procter & Gamble, Coca-Cola, NuVal Griffin Hospital, Abbott, the Canola Council of Canada, Dean Foods, the California Strawberry Commission, Haine Celestial, and the Alpro Foundation; has served on the speakers panel for the Almond Board of California; has received research grant support from Loblaw Brands Ltd, Unilever, Barilla, the Almond Board of California, Solae, Haine Celestial, Sanitarium Company, Orafti, the International Tree Nut Council, and the Peanut Institute; has received travel support to attend meetings from the Almond Board of California, Unilever, the Alpro Foundation, the International Tree Nut Council, the Canadian Institutes for Health Research, the Canada Foundation for Innovation, and the Ontario Research Fund; has received salary support as a Canada Research Chair from the federal government of Canada; and discloses that his wife is a director of Glycemic Index Laboratories, Toronto, Ontario, Canada. Dr. Martinez-Gonzalez has had a research contract with Danone to support research on yogurt in the SUN cohort; and received a departmental grant from the International Nut Council. Dr. Mozaffarian has served on the scientific advisory board of Unilever North America; received ad hoc honoraria from Bunge and the Haas Avocado Board; received consulting fees from Nutrition Impact, Amarin, AstraZeneca, Life Sciences Research Organization, and Boston Heart Diagnostics; and receives royalties for an online chapter on fish oil entitled "Fish Oil and Marine Omega-3 Fatty Acids." Dr. Popkin has received funding to speak on sugar-sweetened beverages (SSB) behaviors globally from Danone water research center at 2 international conferences in the past 5 years; and was a coinvestigator to a water versus SSB randomized controlled trial funded by Danone to the Mexican National Institute of Public Health in Cuernavaca, Mexico. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. Drs. Anand and Hawkes contributed equally to this work.
Listen to this manuscript's audio summary by *JACC* Editor-in-Chief Dr. Valentin Fuster.

Manuscript received May 5, 2015; revised manuscript received July 16, 2015, accepted July 20, 2015.

FIGURE 1 Food System Impact on Nutrition-Related NCDs



Source: revised version of Nugent, 2011 "Bringing Agriculture to the Table" Chicago Council on Global Affairs. NCD = noncommunicable disease.

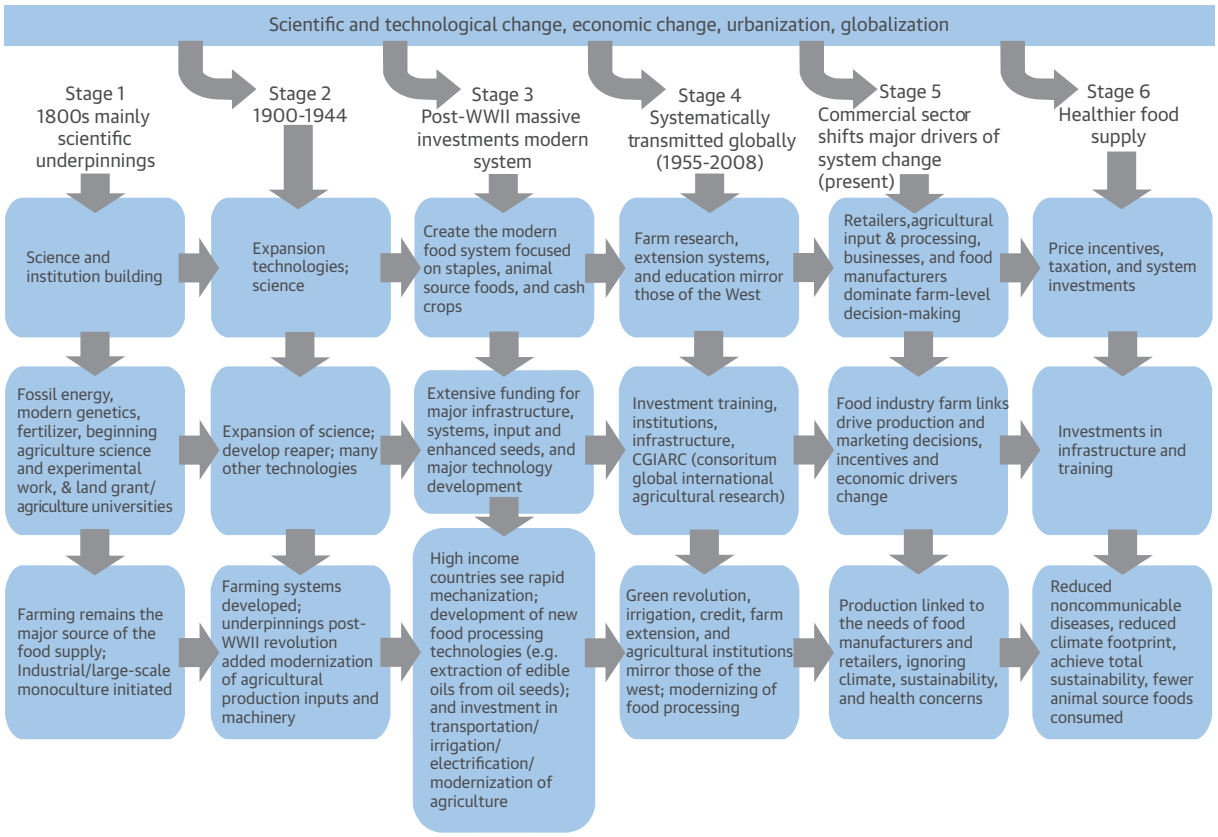
food system is characterized by a global web of interactions between multiple actors from farm to fork, geared toward maximizing efficiency to reduce costs and increase production (**Figure 1**). The major actors who control this system have changed dramatically in HIC and LMIC, as described subsequently (13).

The shift to a global food system started in the United States and other high-income industrialized countries, and was driven initially by government investment and intervention in markets, infrastructure and research intended to raise farm-sector productivity. Building on actions taken in the late 19th century (14), policies on agricultural research and supporting on-farm production introduced in the period from 1930 to 1960 in the United States (14) and Europe focused on few major crops, particularly grains (e.g., wheat, corn, rice), oilseeds (e.g., soybeans), livestock (e.g., pigs, poultry, cattle), and critical cash crops, especially sugar cane and other sources of sugar (15-18). State intervention in most LMIC took a different form, such as policies to subsidize food, taxes on agricultural products, and systems to control the supply and marketing of key commodities (19-22). The 1960s also saw the start of significant agricultural transformation in LMIC, with the "Green Revolution," which focused on increasing productivity of corn, rice, and wheat.

These investments and changes in production systems were designed to make calories from staples (e.g., wheat, corn, rice) cheaply available, in order to simultaneously address hunger in LMIC and national food insecurity in HIC (23). In addition to vastly increasing the calorie supply, the ensuing productivity boom also provided the basis of cheap feed for livestock and cheap inputs for processed foods, in turn creating incentives for the growth of manufacturers of processed foods (24). This coincided with huge technological innovations in food processing, (24-28), the rise of mass marketing to persuade consumers to eat more, supermarket retailing, and fast food (29,30). As a result of these changes, the transformation of raw commodities into food and the distribution of consumable food items beyond the farm gate has become far more important (31). Today, integration and control of our farm-to-fork food supply by major agribusinesses, food manufacturers, retailers, and food service companies is more the rule than the exception (13). Meanwhile, production of less processed foods such as coarse grains (e.g., millet, sorghum), roots, tubers, and legumes has declined (32,33) whereas animal source food production has grown dramatically (34).

Figure 2 sets out the stages of change involved in leading to this modern food system. This model has spread unevenly to most LMIC (35-37). Many countries retain various forms of state intervention in agriculture and food systems (18,38-41), but policies to liberalize trade and private sector investment have

FIGURE 2 Stages of Global Agricultural System Development



Source: © (copyright) Barry M. Popkin, 2015.

revolutionized the entire sector in many regions (13,42). Retailing has been transformed in LMIC through the growth of supermarkets (18,38-41). Although this process originated with companies in industrialized countries looking for growth in foreign markets, companies based in LMIC are now also investing back into HIC.

DIETARY IMPACTS. The way people eat has changed greatly across the globe; moreover, the pace of change in LMIC is quickening. Snacking and snack foods have grown in frequency and number (43-48); eating frequency has increased; away-from-home eating in restaurants, in fast food outlets, and from take-out meals is increasing dramatically in LMIC; both at home and away-from-home eating increasingly involves fried and processed food (47,49); and the overall proportion of highly processed food in diets has grown (50,51).

These changes in the global food system coupled with these food behavior shifts have enabled some critical changes to the global food supply, all with dietary implications. First is the shift to refined carbohydrates—refined grains and added sugars. Rapidly increasing production of starchy staples combined with processing technologies mean that refined flour is increasingly dominant in diets. White bread, for example, once rarely consumed in Latin America, became widespread after the introduction of high-yield wheat varieties. In Asia, white rice became dominant as a staple over legumes and coarse grains, with a more recent trend being rapidly rising consumption of instant noodles as a staple (52,53). Since 1964, average total carbohydrate intake in the United States has increased from about 375 g/day to 500 g/day (from 2 to 6 kg/year of ready-to-eat cereals), but the percent of carbohydrate that is fiber has not substantially changed over this time, reflecting increased refined carbohydrates and sugar-sweetened beverages (SSBs) is high in HIC (54). In the period from 1985 to 2005 extensive added sugar intake occurred across HIC (55) but more recently large increases have occurred in LMIC, particularly in consumption of SSBs and processed foods (56-59). Today in the

United States packaged and processed food supply, over 75% of foods, have some form of added sugar (60). With urbanization there is some evidence to show that refined carbohydrate consumption is increasing, whereas consumption of traditional grains (i.e., millet, maize) is decreasing in LMIC (61,62).

A second key change has been the increasing intake of vegetable oils, including processed vegetable oils, and a decline in consumption of animal fats (2,63). This was initially driven by rising production of soybeans in the United States, later in Argentina and Brazil, and then palm oil in East Asia. Oilseeds are now among the most widely traded crops, and are also processed to create margarines and vegetable shortenings and into partially hydrogenated fats and bleached deodorized oils for use in processed foods (Online Appendix, Box 2). Between 1958 and 1996, a major global shift occurred in the amount and types of available fats, with soybean, palm, and rapeseed/canola oils replacing butter, tallow, and lard. From 1958 to 1962, soybean, palm, and rapeseed/canola oils represented 20% of the 29 million metric tons of fat produced globally per year, whereas butter, lard, and tallow represented 37%. By 1996 to 2001, these oils accounted for 52% of the 103 million metric tons of fat produced globally per year, whereas butter, lard, and tallow contributed 20% (64). This has implications for consumption of fatty acids. Palm oil (deodorized) has become an increasing source of saturated fatty acids; and partially hydrogenated fats are the main source of trans fatty acids. Between 1990 and 2010, global saturated fat, dietary cholesterol, and trans fat intakes remained stable (trans fats going down in HIC and up in LMIC), whereas n-6, seafood n-3, and plant n-3 fat intakes each increased (65). Vegetable oil consumption remains 2 times higher in HIC than in LMIC. However, trans fat consumption is very high in many LMIC although decreasing markedly in HIC. In India, for example, vanaspati, a vegetable ghee used in bakery products, fried snacks, and foods sold by street vendors, is a leading source of trans fats, as is bakery shortening. In 75 countries representing 61.8% of the world's adult population, global saturated fat consumption was ~9% of energy; though considerable variation existed across countries (range 2% to 28%). Country-specific omega 6 consumption ranged from ~1% to 13% (global mean: 6%) of energy; trans fat consumption ranged from ~0.2% to 6.5% (global mean: 1%) of energy; dietary cholesterol consumption ranged from ~97 to 440 mg (global mean: 228 mg) per day; seafood n-3 ranged trans fat from ~35 to 3,886 mg (global mean: 163 mg) per day; and plant n-3 ranged from <~100 to 5,542 mg (global mean: 1,371 mg) per day (65).

A third key change has been the increasing global consumption of meat, which has been made economically feasible by subsidized production of crops for animal feed—most importantly corn and soybeans (soybean oil is a byproduct of soymeal production for animals) (66-69). At very low levels of intake animal food consumption may not induce harm, providing high-quality protein and iron, whereas excess animal food intake in HIC may be linked to adverse health outcomes, particularly from processed meats (70). Meat consumption has increased considerably worldwide, and there is substantially greater production of meat in HIC than in LMIC (71). North and South America, Europe, and Australia/New Zealand have the highest meat intake, whereas Asia and Africa have the lowest (72,73). Processed meats (which refers to post-butchering modifications of foods such as curing, smoking, or addition of sodium nitrate), account for 35.8% of all meat consumed in HIC (unpublished data from the PURE [Prospective Urban and Rural Epidemiological] study) (71).

Dietary consumption patterns of other protein sources have been mixed. Between 1973 and 1997, dairy consumption per capita (kilograms) increased in LMIC by ~48% and is projected to almost double (93% increase) by 2020. On average, fish consumption is 2 to 3 times higher in HIC than LMIC (74), although with marked heterogeneity within income categories. China has the highest per-capita consumption of fish in the world, followed by Oceania, North America, and Europe (74). Globally, although there has been little or no increase in sea fish consumption *per capita* since the 1960s, catches per year have risen exponentially (75) and freshwater fish intake has increased during this time (71). Eggs are similarly consumed in higher quantities (2 to 6 times per week) in HIC relative to LMIC, with a 14% decline in consumption in HIC observed between 1980 and 2000, and no change was observed in LMIC (76). The consumption of legumes declined in the United States from 1960 and into the 1980s, with reduced consumption patterns observed globally (8). Relatively, HIC such as Canada, the United States, and Western Europe, tend to consume the lowest quantities of legumes per capita in the world, whereas LMIC within Africa and India consume the greatest quantities of legumes, along with certain South American countries where beef is uncommon, such as Colombia and Peru (77-79). Globally, pulse consumption has decreased since 1961, from ~9.5 kg/person/year in 1961 to 6.5 kg/person/year in 2006. In LMIC countries pulses contributed ~4% of energy to the diets, and just 1% of energy to diets of HIC (80).

Total production of tree nuts in 2012 was 3.5 million metric tons, a 5.5% increase from 2011. World consumption of tree nuts in 2011 exceeded 3 million metric tons (81).

A fourth key change is the marked growth of purchases of all packaged foods and beverages (all categories of processing). This process is accelerating across all LMIC markets (13,82,83). For example, 58% of calories consumed by Mexicans come from packaged foods and beverages, which is similar throughout the Americas (83) and even within the United States (66%) (65,84). The proportion for China is 28.5% and rising rapidly (36,82,83). The component of snack foods that is "ultra-processed" (i.e., ready to eat) varies depending on the method of measurement but is increasing wherever it is studied at all income levels (50,85,86). The shift to ultra-processed foods has not just affected the food available for consumption but also the way food is consumed (87). The way people eat has changed greatly across the globe and the pace of change is quickening. Snacking and snack foods have grown in frequency and number (43–48); eating frequency has increased; away-from-home eating in restaurants, in fast food outlets, and from take-out meals is increasing dramatically in LMIC; both at home and away-from-home eating increasingly involve fried and processed food (47); and the overall proportion of highly processed food in diets has grown (50,51).

A fifth trend noted previously in relation to the added sugar change is the shift in the way LMIC are experiencing a marked increase in added sugar in beverages. In the 1985 to 2005 period extensive added sugar intake occurred across HIC (55) but more recently large increases have occurred in LMIC, particularly in consumption of SSBs and ultra-processed foods (56–59). Today in the U.S. packaged and processed food supply, >75% of foods have some form of added sugar (60).

In addition, fruit and vegetable intake has remained inadequate. Fruit and vegetable consumption is substantially higher in HIC compared with LMIC (88). Analysis of 52 LMIC countries taking part in the World Health Survey (2002 to 2003) (89) found that low fruit and vegetable consumption (i.e., <5 fruits and vegetables per day) prevalence ranged from 36.6% (Ghana) to 99.2% (Pakistan) for men and from 38.0% (Ghana) to 99.3% (Pakistan) for women. Overall, 77.6% of men and 78.4% of women consumed less than the minimum recommended 5 daily servings of fruits and vegetables. In the United States, 32.6% of adults consumed fruit 2 or more times per day and 27.2% ate vegetables 3 or more times per day (90). In 2012, 40.6% of Canadians 12 years of age and older, reported consuming fruits and vegetables 5 or more times per day (91).

Although all of these changes across LMIC display great heterogeneity (92), the global food system has clearly reached all corners of the LMIC urban and rural sector and major shifts in diets appear to be accelerating.

IMPLICATIONS FOR ENVIRONMENTAL IMPACTS. The modern food system is a major force in a range of serious environmental problems, including climate change (as a leading source of greenhouse gas emissions, including carbon dioxide, methane, and nitrous oxide), the loss of biodiversity, the strain on freshwater resources, and the release of persistent toxins, excess nitrates and phosphates (from fertilizer and concentrated livestock operations, causing widespread problems of eutrophication), and animal pharmaceutical residues into waterways (12,93–95).

The major causes are beef and other large animals for greenhouse gas and the metabolic losses associated with shifting the product of nearly one-third of the world's arable land to concentrated animals, which effectively magnifies the resource budgets and pollution loads of industrial monocultures (34). The expansion of low-input agriculture and extensive ranching are also major factors in deforestation, which bear heavily on both climate change (as carbon is released from vegetation and soils and sequestration capacity diminishes) and biodiversity loss.

In addition to being a major force in many environmental problems, world agriculture is also extremely vulnerable to climate change, biodiversity loss, declining freshwater availability, and the inevitable limits of nonrenewable resources (e.g., fossil energy, high-grade phosphorous) although vulnerability is highly uneven on a world scale (96). Many of the world's poorest-regions are poised to be most adversely affected by rising average temperatures, aridity, and water stress, as well as through increasingly severe extreme weather events such as drought or flooding; in fact some believe this is already occurring (97–99).

It is also noteworthy that the Food and Agriculture Organization of the United Nations estimates that one-third of all food produced for human consumption globally is wasted before it is consumed, which has both social and environmental costs that have been precisely measured in the United States (100–102). Waste affects the entire food system from production to post-harvest (including inside the home) (102). Hall et al.'s (101) estimate of home food waste of about 1,400 kcal/capita for the United States adds up to 25% to 45% waste of total food for some HIC.

MACRONUTRIENTS, FOODS, AND CVD RISK FACTORS

CARBOHYDRATES. Refined carbohydrates. *CVD Risk Factors.* U.S. ecological evidence demonstrates an association of refined carbohydrate (such as corn syrup) with type 2 diabetes mellitus (T2DM) and obesity (54). Robust data from systematic reviews and high-quality randomized controlled trials (RCTs) support a harmful effect of highly refined, high-glycemic load (GL) carbohydrates. A meta-analysis of observational studies indicated that high-glycemic index (GI) foods are associated with T2DM (103). Proof-of-concept studies have used alpha-glucosidase inhibitors, such as acarbose, to lower the GI of the foods consumed; for example, in the STOP-NIDDM (Study to Prevent Non-Insulin-Dependent Diabetes Mellitus) trial, acarbose reduced progression to T2DM by 25% compared with placebo (104). T2DM risk in individuals with the highest GL and lowest cereal fiber is 2.5-fold that of those with the lowest GL and highest cereal fiber diet (105). A large Danish prospective cohort study of the impact of replacing saturated fats with high-GI carbohydrates found that when high-GI carbohydrates replace saturated fat, myocardial infarction (MI) risk increases 33% (106). A meta-analysis of 10 prospective cohort studies (n = 296,849) (107) found increased GL associated with a 27% increased coronary heart disease (CHD) and MI risk (108). In controlled-feeding studies, replacing saturated fat with carbohydrates lowers low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) and increases triglycerides (109). Dietary interventions that raise HDL-C may not necessarily translate into CVD risk reduction, as serum HDL-C has recently been called into question regarding its role in the causal pathway of CVD (110). However, in a 5-week controlled-feeding trial of 163 generally healthy but overweight adults, low compared with high-GI diets did not improve insulin sensitivity, lipid levels, or systolic blood pressure in the context of a healthy, DASH (Dietary Approaches to Stop Hypertension)-like dietary pattern, and a low compared with high-GI diet increased LDL-C when the carbohydrate content of the diet was high (109). This study suggests that the adverse effect on CHD risk may not be mediated by short-term effects on classical risk factors, but that post-prandial hyperglycemia and hyperinsulinemia or hyperlipidemia may play a mediating role; further work is needed to clarify the effect of the GI on LDL-C and related lipid risk factors.

LMIC. Highly refined carbohydrates include polished white rice, cornstarch, and white wheat flour with reduced fiber content. Carbohydrate refinement is common in HIC and increasing in LMIC (111,112). Traditional diets in LMIC, once rich in whole grains and dietary fiber, now include highly refined carbohydrates, such as polished white rice and refined flours. In East Asian countries, white rice consumption is associated with a 55% higher T2DM risk (111,112). In the cross-sectional CURES 57 (Chennai Urban Rural Epidemiology Study 57) study, higher refined grain intake was associated with higher waist circumference, systolic and diastolic blood pressure, fasting glucose, triglycerides, and insulin resistance and lower HDL-C levels (61). Replacing white with brown rice (50 g/day) reduced T2DM risk by 16% (113), and substituting beans for white rice reduced the odds of metabolic syndrome by 35% (108). However, a 16-week randomized trial of replacing white with brown rice in middle-aged Chinese men and women with or at high risk for T2DM only improved HDL-C and reduced diastolic blood pressure in the brown rice group. Data from the international PURE study (138,926 individuals in 628 communities in 17 countries) suggest a need to consider the context and availability of specific foods before making food choice recommendations (114). Increasing whole grain and cereal fiber consumption whereas decreasing total and high-GI carbohydrate are helpful strategies to prevent T2DM and CVD in the general population (115). Furthermore, low-GI diets improve glycemic control and serum lipids in RCTs of participants with T2DM with major implications for CHD risk reduction in this vulnerable segment of the population whose numbers are increasing rapidly globally (116–119).

Sugar-sweetened beverages. *SSB and Cardiovascular Risk Factors.* SSB consumption accounts for up to 50% of added sugar in the American diet (120,121). The epidemiological relationships between SSB consumption, overweight, obesity, hypertension, and T2DM are strong (122). In a meta-analysis of prospective studies of SSB and hypertension, CHD, and stroke, the relative risk (RR) for a 1-serving increase in SSB/day was 1.17 (95% confidence interval [CI]: 1.10 to 1.24) for CHD and 1.08 (95% CI: 1.04 to 1.12) for incident hypertension, but no clear effect was seen for total stroke (RR: 1.06; 95% CI: 0.97 to 1.15) (123). A meta-analysis of 7 cohort studies and 5 RCTs found SSBs increased weight by 0.12 kg/serving/year (95% CI: 0.10 to 0.14) in adults (124). A World Health Organization systematic review reported similar positive associations (125). In a pooled analysis of NHS (Nurses' Health Study) and HPFS (Health Professionals Follow-up Study) data, a 1 SSB serving/day increment is associated with a 1 kg weight gain over a

4-year period (126), possibly due to incomplete compensation for this energy at other meals. After adjusting for important potential confounders, including body mass index, ≥1/day versus <1 SSB/month increased T2DM by 39% (127). A meta-analysis of 310,819 participants and 15,043 cases of T2DM reported a 26% increased T2DM risk among those consuming 1 to 2 SSB servings/day compared with nonconsumers (128). A meta-analysis of 4 cohort studies reported a linear association between SSB and hypertension risk (RR) of 1.08 (95% CI: 1.04 to 1.12) per serving per day (129). The Framingham Offspring Study reported a 22% higher incidence of hypertension among those consuming ≥1 SSB serving/day compared with nonconsumers (130). A potential explanation of the SSB-T2DM association is the high content of rapidly absorbed sugar from corn syrup, which increases blood glucose and insulin and de novo lipogenesis—which may contribute to pancreatic beta cell dysfunction and eventually T2DM (131). In the NHANES III (National Health and Nutrition Examination Survey III) study, with 831 CVD deaths during 163,039 person-years of follow-up, consumption of ≥7 SSB servings/week was associated with a 29% increased CVD mortality risk compared with <1 serving/week (120), with no increased risk up to 6 drinks/week. Few studies have investigated the association between SSBs and CVD events. Those that have, report increased CHD and stroke risk with SSB consumption. A 2015 meta-analysis reported an RR for incident CHD of 1.17 (95% CI: 1.10 to 1.24) per serving per day increase in SSB consumption (129). Both the HPFS and the NHS find an approximate 20% increased CHD risk in the highest category of SSB consumption compared with the lowest category (132,133); and, after adjusting for dietary and nondietary cardiovascular risk factors, a 16% increased stroke risk (134). Similarly, 2 Swedish prospective cohort studies in women and men reported a RR of 1.19 (95% CI: 1.04 to 1.36) for stroke among those consuming ≥2 SSB servings/day (135).

LMIC. Facing health concerns, the beverage industry is shifting from full-calorie carbonated soft drinks to lower-calorie carbonated soft drinks, coffees, and teas. According to the NHANES study, between 1999 and 2006 the average U.S. full-calorie carbonated soft drink intake decreased, whereas intake of diet carbonated soft drinks, low-calorie fruit drinks, and other sweetened beverages increased (122). These products will gradually enter the markets in China, Brazil, and other LMIC. However, there are practically no data on the effects of SSBs on health outcomes from LMIC (136,137).

FATS AND OILS. CVD risk factors. Vegetable oils that are primarily comprised of mono- (e.g., olive oil) and polyunsaturated fatty acids appear to reduce CHD risk, and sources of the n-3 polyunsaturated fat alpha-linolenic acid, such as rapeseed or canola oil, are cardioprotective. Replacing saturated fat with monounsaturated or polyunsaturated fat reduces LDL-C and preserves HDL-C (109). Further, canola oil, as part of a low-GI diet, improves glycemic control and blood lipids in T2DM (138). Trans fatty acids increase CHD risk compared with other macronutrients, with strong evidence of adverse effects of small amounts of trans fats on lipids (109,139) and CVD risk (140,141).

Though total fat (142), and specifically saturated fats, have generally been considered to be deleterious to insulin sensitivity (143), in large cohort studies, saturated fat is not associated with development of T2DM, after adjustment for body mass index, total dietary fiber, or magnesium intake (144–149). Macronutrient exchange generally does not influence markers of glucose homeostasis, though in 2 relatively large trials, replacing saturated fat with either monounsaturated fat or carbohydrate improved indices of glucose homeostasis (150,151). Associations have been seen between major food sources of saturated fat, such as red and processed meat, and development of T2DM (152,153), though dairy products, notably fermented dairy, may be protective (154,155).

CVD. Saturated fats have not been consistently associated with CVD in meta-analyses of cohort studies (odds ratio [OR]: 1.07; 95% CI: 0.96 to 1.19) of higher compared with lower intakes (156). However, in most of these studies the association of high saturated fat intake largely represents replacing highly refined carbohydrates. Replacing saturated fat with highly refined carbohydrate is not associated with lower CHD risk, whereas replacing saturated fat with polyunsaturated fat reduces CHD risk (157,158). This benefit of polyunsaturated fat includes the primary n-6 polyunsaturated fatty acid, linoleic acid (158,159). Replacing saturated fat with high-GI carbohydrate increases MI risk by 33%, whereas replacing with low- and medium-GI carbohydrates appears neutral (106). Emerging evidence suggests that the effect of a saturated fat on CHD may depend on the type of fatty acid and the specific food source (i.e., dairy vs. meat) (160,161). Consumption of nonhydrogenated vegetable oils appears to be superior to consumption of animal fats (162). Consumption of plant oils in a Mediterranean diet reduced CVD in 2 RCTs. The Lyon Diet Heart Study found regular consumption of alpha-linolenic acid (canola oil) significantly reduced

cardiac deaths and nonfatal CHD (163), whereas the PREDIMED (Prevención con Dieta Mediterránea) RCT found that a Mediterranean diet (50 g/day extra virgin olive oil) reduced CVD events by 30% over a 5-year period (164).

Palm oil is the dominant fat globally and is relatively high in saturated fat. On the basis of controlled-feeding studies examining changes in blood lipids, replacing palm oil with unsaturated fatty acids would be expected to lower CHD risk (109), but palm oil would be preferred to partially hydrogenated oils high in trans fatty acids. Few studies have directly compared palm oil with other oils for CHD risk. One large case-control study in Costa Rica found that soybean oil consumption was associated with lower acute MI risk compared with palm oil consumption (165).

Summary of fats and oils. Compared with saturated fat, vegetable oils rich in polyunsaturated fats reduce the TC:HDL-C (total cholesterol:HDL-C) ratio and CHD incidence; inclusion of n-3 fatty acids (alpha-linolenic acid) with the vegetable oils is important for CHD prevention. The effect of replacing saturated fat with carbohydrate on CHD risk appears to depend on the quality of the carbohydrate. Prospective studies consistently indicate adverse effects of trans fats on CHD. Effects of monounsaturated fat from plant sources require further study; extra virgin olive oil appears to reduce CVD.

PROTEIN SOURCES. Meats. *Nutrients and CVD Risk Factors.* Meat is rich in protein, iron, zinc, and B vitamins, but can also contain significant amounts of cholesterol and saturated fatty acids, which raise LDL-C and lower triglyceride (157). A high red meat intake (rich in heme iron), increases endogenous formation of *N-nitroso* compounds in the gastrointestinal tract that are associated with increased epithelial proliferation, oxidative stress, and iron-induced hypoxia signaling (166–168).

In a meta-analysis of 17 cohort studies (16 from Western countries), consumption of red and processed meat increased T2DM and CHD risk, but few included studies examined unprocessed red meat (169). Intake of red or processed meat was not associated with stroke, but only 3 studies evaluated these relationships (169). In more recent analyses, red meats, particularly processed red meats, were associated with increased CVD, CHD, stroke, and cancer mortality, whereas poultry was not (170–172). Potential mechanisms linking unprocessed red meat with CVD include saturated fat, cholesterol, iron, phosphatidylcholine, and carnitine; and cooking methods (e.g., barbecuing), that increase heterocyclic amine content and *N-nitroso* compounds, also implicated in colorectal cancer (167). The evidence suggests that processed meat consumption increases CHD risk, whereas unprocessed meat consumption has a small or no association with CHD, mainly when compared with refined starch and sugar. Both unprocessed and processed red meats are associated with greater CVD risk compared with poultry, fish, or vegetable protein sources. Both types of meat are associated with higher T2DM risk, although gram for gram the effect size is notably larger for processed meats.

LMIC. Data relating meat consumption to CVD risk in LMIC is limited. A recent pooled analysis of data from 296,721 individuals from Asian countries (i.e., Bangladesh, mainland China, Japan, Korea, and Taiwan) found no association between red meat and poultry consumption and CVD, cancer mortality, or all-cause mortality (173). Red meat intake is generally much lower in these areas than in HIC however, and current consumption does not likely reflect long-term patterns.

Dairy. *CVD Risk Factors.* The consumption of dairy products has been associated with weight loss in small studies (174), but the overall published data does not confirm an important effect on body weight. However, increased low-fat dairy consumption is associated with lower LDL-C, triglycerides, plasma insulin, insulin resistance, waist circumference, body mass index, possibly blood pressure; and reduced diabetes risk (174–182). In a large meta-analysis of cohort studies (13,000 incident cases), and the EPIC InterAct (European Prospective Investigation into Cancer and Nutrition) case-cohort study (12,000 incident cases), fermented dairy (i.e., yogurt, cheese, and thick fermented milk), but not total dairy, was inversely associated with T2DM (155,183). In a meta-analysis of prospective cohort studies, milk consumption was inversely associated with total CVD in a small subset of studies with few cases, but using a larger body of data with more specific endpoints, milk was not associated with CHD or stroke (173). In a prospective cohort study of 53,387 Japanese men and women, higher dairy calcium (173 mg/day vs. none) reduced risk for hemorrhagic stroke, ischemic stroke, and stroke mortality by ~50% over a 10-year follow-up (184). Collectively, these studies do not suggest a strong or consistent relationship between consumption of dairy products and T2DM and CVD risk.

LMIC. Data on dairy consumption and CVD in LMIC are limited. In a prospective cohort of 2,091 middle-aged Chinese men and women monitored for 6 years, individuals who reported consuming >1 dairy serving/day were 35% less likely to develop

T2DM (RR: 0.65; 95% CI: 0.49 to 0.85) than nonconsumers (185).

Egg. *CVD Risk Factors.* Eggs are a relatively inexpensive and low-calorie source of protein, folate, and B vitamins (186). Eggs are also a source of dietary cholesterol (a medium egg contains approximately 225 mg of cholesterol) (187). A meta-analysis showed that eggs increase TC, HDL-C, and TC:HDL-C (188), but 5 RCTs subsequently reported that egg consumption did not significantly alter these parameters (189–191) or endothelial function (192,193). No RCT has tested the effect of egg consumption on CVD events. In a meta-analysis (194) of 16 prospective cohort studies (90,735 participants) (191), egg consumption was not associated with overall CVD or CHD, stroke, or CHD or stroke mortality; but was associated with T2DM. Overall, consumption of eggs in moderation (1 egg/day) is likely neutral for CVD. However, relative to other protein-rich foods that lower LDL cholesterol, such as whole grains and nuts, eggs would likely increase CVD risk.

LMIC. Unpublished data from 3 large international studies, PURE (Prospective Urban and Rural Epidemiological Study), ONTARGET (Ongoing Telmisartan Alone and in Combination with Ramipril Global Endpoint Trial), and INTERHEART (a global study of risk factors for acute myocardial infarction), with collectively 200,000 individuals and 22,000 CVD events from regions including China, India, and Africa, show that moderate egg consumption appears to be neutral or protective against CVD. However, significant variations exist across regions, with a benefit of daily egg consumption in China but possible harm in South Asia.

Fish. *CVD Risk Factors.* Fish are a source of protein, vitamin D, multiple B vitamins, essential amino acids, and trace elements; and the long-chain omega-3 (n-3) fatty acids docosahexaenoic acid and eicosapentaenoic acid (195,196), though amounts vary over 10-fold across seafood species. Fatty fish, such as salmon, sardines, trout, white tuna, anchovies, and herring, have the highest concentrations. In clinical trials and meta-analyses of these trials the long-chain omega-3 polyunsaturated fatty acids in fish reduced multiple CVD risk factors, including vascular resistance, blood pressure, inflammation, serum lipids, and endothelial function (197).

Some prospective cohort studies find an inverse association between fish intake and CVD mortality, whereas others do not (198). Available evidence suggests cardiovascular benefits with fish consumption in secondary prevention, but the evidence is inconsistent regarding primary prevention. Possible explanations include differences in the amounts and types of fish consumed, cooking methods, and background fish consumption. Fifteen of the 16 cohort studies (with 1 exception) (199) were conducted in North America and European countries, where deep-frying fish is common. The DART-1 (Diet and Reinfarction) trials, a secondary prevention trial and DART-2, in men with stable angina, are the only randomized trial of fish intake and CVD outcomes. They arrive at opposite conclusions. In the DART-1 trial, fish lowered all-cause mortality and trended toward reducing CVD events after 2 years (194). In the DART-2 trial oily fish did not affect all-cause mortality or CVD events after 3 to 9 years, and increased sudden cardiac death, largely confined to the subgroup given fish oil capsules (200). Differential behavioral change or CVD stage may explain the discrepancy (201). Follow-up of the DART-1 trial at 5 years also showed increased rates of CVD in the fish/fish oil group that did not persist through the 10-year assessment (202). We know of no primary prevention trial on fish intake and CVD outcomes, but a meta-analysis of fish oil supplement RCTs is neutral (203).

Low-Income Countries and HIC. Most of the data indicating that fish is protective comes from studies in HIC (204–208). Unpublished data from 3 large international studies (PURE, ONTARGET, and INTERHEART) reflect considerable heterogeneity in the association between fish intake and CVD outcomes. In the PURE study fish intake was inversely associated with CVD outcomes in South America, China, North America, and Europe (RR: 0.76 to 0.84) but positively associated in South Asia (RR: 1.97). However, no associations between fish consumption and CVD outcomes were observed in a high-risk secondary population in the ONTARGET study. The INTERHEART study found fish intake beneficial in North America and Europe (RR: 0.73; 95% CI: 0.62 to 0.87) but harmful in the Middle East (RR: 1.59; 95% CI: 1.32 to 1.93). More work is needed in China and India to understand the effects of regional types and preparation methods of fish.

Nuts. *CVD Risk Factors.* In clinical trials nuts improve serum lipids (209–211). In observational studies, nuts lower CHD (196,212,213) and hypertension risk (213); but not stroke (207,213). In 1 meta-analysis of prospective cohorts, nuts were not associated with T2DM (214), but in another, they were protective (207). Despite their high energy density, nuts do not contribute to weight gain, changes in waist circumference, or obesity, perhaps due to their satiating effects and increased fecal energy losses (215,216).

In observational studies (133,217-220) and RCTs (164,221-223), a Mediterranean diet including nuts lowers CVD risk. However, no RCTs have assessed the effects of nut consumption alone on CVD events. Taken together, the published data from observational studies and clinical trials support nuts for lowering CVD risk.

LMIC. Unpublished data from the PURE cohort indicate that nut consumption is very low in LMIC (60% of individuals consume ≤1 nut serving/week). No associations between nut consumption and CVD outcomes are seen at this low level.

Legumes. *CVD Risk Factors.* In observational studies and RCTs consumption of legumes improves CVD risk factors, such as waist circumference, cholesterol, blood pressure, C-reactive protein, glucose; and is protective against T2DM (224-231). A meta-analysis of 26 RCTs (1,037 participants) found that 130 g legumes/day (~1 serving) reduced LDL-C by 0.17 mmol/l (95% CI: -0.25 to -0.09) (228). Legumes also reduce systolic blood pressure (by 2 mm Hg) and lowered mean arterial pressure (231) in a meta-analysis of 8 RCTs. A legume-rich diet reduced HbA1c (-0.3%; 95% CI: -1.4% to -0.1%) in an RCT in participants with diabetes (226).

CVD. In a meta-analysis of 5 observational studies, 100 g of legumes 4 times/week is inversely associated with CHD (RR: 0.86; 95% CI: 0.78 to 0.94) (232). However, that study and another meta-analysis of 8 prospective cohort studies found no association between legumes and diabetes (207) or stroke (232,233). A large prospective study of 2 cohorts of U.S. health professionals found a 45% increased risk of ischemic stroke per daily serving of legumes (RR: 1.45; 95% CI: 1.06 to 2.00) (234). This suggests that although legumes are valuable to reduce CHD risk, more research is required to understand their impact on total stroke risk.

LMIC. In a cohort of Chinese men and women, soy (the primary legume consumed in China) was negatively correlated with TC and LDL-C (235). A population-based cross-sectional study in India found that women consuming legumes once/day were less likely to develop T2DM (OR: 0.55; 95% CI: 0.34 to 0.88; n = 99,574). A similar but nonsignificant trend was observed in men (OR: 0.70; 95% CI: 0.39 to 1.26; n = 56,742) (236). In a prospective cohort study of middle-aged Shanghai women, a ~50 g/day increase in legume consumption over 4.6 years reduced T2DM risk by 38% (RR: 0.62; 95% CI: 0.51 to 0.74) (237). A prospective cohort study of 64,915 Chinese women 40 to 70 years of age (n = 62 cases; follow-up 2 years) found that individuals consuming ≥11.2 g/day of soy protein were less likely to develop CHD (RR: 0.25; 95% CI: 0.10 to 0.63) than were those consuming ≤4.5 g/day (238).

Summary of major protein sources. Reducing red meats, especially processed meats, and increasing fish, nuts, legumes, and possibly fermented dairy products are likely beneficial. Sustainability issues discussed in this paper must also be addressed specifically in relation to meat, fish, and dairy foods (12,34,239-241).

FRUITS AND VEGETABLES. CVD risk factors. In the DASH RCT, higher intake of fruits and vegetables, either as part of a typical Western diet or the DASH eating plan, reduced blood pressure, TC, LDL-C, and HDL-C without affecting triglycerides (242). A meta-analysis of 3 prospective cohorts (3,415 cases) found high adherence to the DASH eating reduced T2DM risk by 27% (243). A strong evidence base from observational studies indicates that high consumption of vegetables and fruits reduces CHD and stroke (196).

CVD in HIC and LMIC. Large global studies and systematic reviews of prospective cohorts generally support a protective role of fruits and vegetables against CVD (244-246). In the global INTERSTROKE study (3,000 stroke cases and 3,000 control cases), compared with fewer than 1 serving per day, 1 serving of fruit was protective against stroke (OR: 0.61; 95% CI: 0.50 to 0.73), but the benefits were not clear with higher consumption of up to 3 servings/day of vegetables was not (OR: 0.91; 95% CI: 0.75 to 1.10) (244). A meta-analysis of 20 prospective cohort studies (16,981 stroke events) found that fruit and vegetable consumption was associated with decreased stroke risk (RR: 0.79; 95% CI: 0.75 to 0.84 for highest vs. lowest categories), as were fruits (RR: 0.77; 95% CI: 0.71 to 0.84) and vegetables (RR: 0.86; 95% CI: 0.79 to 0.93) separately (245). In unpublished INTERHEART study data a 1-serving/day increase in fruit decreased MI risk by 12% (RR: 0.88; 95% CI: 0.84 to 0.92); a 1-serving/day increase in vegetables decreased MI risk by 5% (RR: 0.95; 95% CI: 0.92 to 0.97). Large meta-analyses of observational studies support a ~5% to 10% reduction in CVD mortality per serving per day (246), and a protective association of green leafy vegetables with T2DM (RR per 0.2 servings/day: 0.87; 95% CI: 0.81 to 0.93) (247). The beneficial effects of fruits and vegetables appear consistent across regions of the world. A major issue has been the lack of success in encouraging increased fruit and vegetable consumption by the citizens of western nations (248).

Overall summary. Increased refined grains, starches, and added sugar (i.e., carbohydrates of lower quality) have paralleled the rise in obesity and T2DM. Collectively, the data support emphasizing low-GI whole grains, legumes, and nuts, fruits, and vegetables and minimizing high-GI refined grains and foods with added sugars, including SSBs (**Table 1**).

DIETARY SODIUM. Sodium is an essential nutrient required for normal physiological function (249-251). Increasing sodium intake well beyond physiological requirements increases blood pressure (241-243) and CVD mortality (95-97). In LMIC sodium sources include table salt and salt additives and spices; whereas processed foods are the primary source in HIC. Sodium consumption has declined slightly in LMIC as refrigeration has replaced salted-preserved foods. Short-term RCTs have reported reductions in blood pressure with reduced sodium intake to <1.5 g/day. Population recommendations for low sodium intake (252-254) (<2.0 g/day) have been achieved in short-term feeding clinical trials (254), but not sustained in longer-term clinical trials (>6 months) (255-257). No RCTs have determined whether low sodium intake reduces CVD events or deaths compared with moderate intake (249). Meta-analyses of CVD event trials did not report a significant reduction in CVD with lower sodium intake, but the included trials were underpowered to detect moderate risk reductions (258). Prospective cohort studies suggest a *J*-shaped association between sodium intake and CVD events, consistent across methods of sodium estimation (259-265). In a recent Cochrane Review (266) of 23 epidemiological studies (n = 274,683), the lowest risk of CVD events and deaths occurs at an intake between 2.7 and 5.0 g/day. PURE study findings (263) are consistent with this evidence, with sodium excretion both >6 g/day and <3 g/day being associated with higher mortality and CVD events compared to 4.00 to 5.99 g/day, despite an overall positive association between sodium excretion and blood pressure (251,263). The increased risk of CVD events with higher sodium intake (>5 g/day) was most prominent in those with hypertension (263). However, methodological issues make studies of sodium intake particularly challenging (267). Additional large appropriately designed observational studies and RCTs are needed to provide further evidence on optimal sodium intake for preventing CVD events (249,268-270).

ALCOHOL. Consumption patterns. Greater amounts of alcohol are consumed *per capita* by adults in HIC (9.6 l/year) than in LMIC (4.1 l/year), or low-income countries (3.1 l/year), although this may be an underestimate in LMIC and low-income countries due to the use of homemade or unsafe alcohol products (e.g., industrial or medical) (271). In the PURE study, 31% of participants identified as current drinkers, with considerable variation across income regions (13% to 80%). In all regions, men were more likely to be current drinkers than were women (272).

CVD risk factors. In a meta-analysis of randomized controlled intervention studies of 2 to 8 weeks' duration (273), moderate alcohol consumption (up to 1 drink or 15 g/day in women; or up to 2 drinks or 30 g/day in men) compared with no alcohol: raised HDL-C, Apo-A1, and adiponectin, and reduced LDL-C and fibrinogen. Total cholesterol, triglycerides, and lipoprotein (a) were not significantly affected. Results stratified by beverage type (wine/beer/spirits) were similar to the pooled analyses. The changes in lipid and hemostatic risk factors align with lower cardiovascular risk.

CVD. The EPIC (European Prospective Investigation on Cancer and Nutrition) study monitored 380,395 participants for 13 years (4,187 CVD deaths) (274). Female never-drinkers were at increased risk of CVD death (RR: 1.31; 95% CI: 1.13 to 1.53) compared with moderate drinkers (0.1 to 4.9 g/day); but the association was not significant for male never-drinkers (RR: 1.20; 95% CI: 0.89 to 1.62). In a dose-response meta-analysis of 24 prospective cohort studies (275), the association of drinking with CHD was *J*-shaped: nondrinkers were at slightly increased risk; in men the greatest protection was at 31 g/day with a trend for increased risk beginning at 63 g/day; for women, the greatest protection was at 11 g/day with a trend for increased risk beginning at 14 g/day. In a meta-analysis of 27 prospective studies of alcohol and stroke, no association was found between light or moderate alcohol use and stroke, but heavy alcohol use (>45 g/day) was shown to increase total stroke risk (RR: 1.20; 95% CI: 1.01 to 1.43), particularly hemorrhagic stroke (RR: 1.29 ; 95% CI: 0.98 to 1.71) (275). Patterns of alcohol consumption may be a particularly important determinant of the magnitude of the association—in a meta-analysis of 10 prospective cohort and 4 case-control studies, irregular "binge" drinking (>60 g of pure alcohol or ≥5 drinks per occasion at least monthly) increased CHD risk by 45% (RR: 1.45; 95% CI: 1.24 to 1.70) (276). In summary, small and inconsistent cardiovascular benefit is seen in moderate drinkers, but a strong increase in CVD and stroke risk is seen with regular heavy alcohol consumption and binge drinking.

LMIC. In HIC/upper middle income countries in the PURE study (272), current drinking was protective against MI, but not in low-income countries/LMIC (p = 0.02 for interaction); a similar trend was seen for

TABLE 1 Summary of Evidence of Associations Between Major Foods and Cardiovascular Disease

	HIC			LMIC			
Food	Definitely Known	Suggested	Strength of Evidence	Definitely	Suggested	Strength of Evidence	Sustainability
Refined carbohydrates (solid foods)	Refined, high-glycemic carbohydrates are associated with T2DM and CHD.	Refined, high-glycemic carbohydrates adversely affect the lipid profile.	High (SRMA of both RCT and prospective cohorts).	None.	Refined, high-glycemic index carbohydrates are associated with T2DM, markers of central adiposity, blood pressure, insulin, and lipids.	Moderate (cross-sectional, RCT).	Moderate.
Sugar-sweetened beverages	Excessive sugar-sweetened beverage consumption is associated with CVD mortality, stroke, obesity, hypertension, and T2DM.	None.	High (SRMA of both RCT and prospective cohorts).	None.	None.	Absent.	Low.
Saturated fats	None.	Replacement of saturated fats with unsaturated plant fats or low-glycemic carbohydrates reduces the risk of cardiovascular disease, and reduces LDL-C, without adversely affecting HDL-C.	High (SRMA of both RCT and prospective cohorts).	None.	None.	None.	Low; higher saturated fat from palm oil is unsustainable.
Trans fatty acids	Replacement of trans fatty acids with unsaturated plant fats or low-glycemic carbohydrates reduces the risk of CVD, and reduces LDL-C, without adversely affecting HDL-C.	None.	High (SRMA of both RCT and prospective cohorts).	None.	None.	None.	Low.
Unsaturated fatty acids (MUFA, PUFA)	Diets higher in unsaturated plant fats (monounsaturated and polyunsaturated fats from plants and fish) reduce risk of CVD, and improve the lipid profile.	None.	High (SRMA of both RCT and prospective cohorts).	None.	None.	None.	Low.
Red and processed meat	Red and processed meats are associated with CHD and T2DM.	Red and processed meats are associated with stroke.	Moderate (SRMA of prospective cohorts).	None.	No association between red meat consumption and CVD in LMIC.	Low (1 pooled analysis).	Very high; meat production is harmful to the environment.
Dairy	None.	Higher low-fat and fermented dairy products are inversely associated with CVD, blood pressure, and improved serum lipids.	Moderate (SRMA of both RCT and prospective cohorts).	None.	Consuming dairy is associated with reduced risk of T2DM.	Low (single prospective cohort).	Very high; cow's milk production is harmful to the environment and resource-intensive.
Eggs	None.	Consuming <7 eggs per week does not increase risk of CVD, nor do they affect the lipid profile.	Low (SRMA of prospective cohorts, SRMA of RCTs on lipids).	None.	Moderate egg consumption appears protective against CVD.	Low (unpublished case-control data from 3 large international studies).	Moderate.

Continued on the next page

TABLE 1 Continued

Food	HIC			LMIC			Sustainability
	Definitely Known	Suggested	Strength of Evidence	Definitely	Suggested	Strength of Evidence	
Fish	None.	Long chain omega-3 fats from fish reduce multiple CVD risk factors.	Low (SRMA of prospective cohorts, large RCTs that conflict).	None.	There is considerable heterogeneity in the association between fish and CVD outcomes.	Low (unpublished case-control data from 3 large international studies).	Sustainability of fish is challenging.
Nuts	Many varieties of nuts show beneficial effects on LDL-C, the LDL-C:HDL-C ratio, TC, and triglycerides; and nuts reduce CHD risk and hypertension, but not stroke.	Nuts may reduce risk of T2DM.	High (SRMA of both RCT and prospective cohorts).	None.	Nuts are not associated with CVD outcomes.	Weak (single observational study).	Sustainable alternative protein source to meat.
Legumes	Legumes show beneficial effects on LDL-C, the LDL-C:HDL-C ratio, TC, and triglycerides; and legumes reduce CHD risk and hypertension, but not stroke.	Legumes reduce CHD risk, but not stroke risk.	High (SRMA of both RCT and prospective cohorts).	None.	Legume consumption reduces risk of CHD and T2DM.	Weak (small number of prospective cohort studies, cross-sectional association).	Sustainable alternative protein source to meat.
Fruits and vegetables	Diets high in fruits and vegetables reduce blood pressure, TC, LDL-C, and HDL-C and protect against CVD.	None.	High (SRMA of both RCT and prospective cohorts).	None.	Protective role of fruits and vegetables against CVD.	Weak (small number of prospective studies).	Sustainable source of carbohydrate energy and micronutrients.
Sodium	None.	Lower sodium diets reduce blood pressure, but the association between lower sodium and CVD is unclear.	Low (conflicting observational and RCT data).	None.	Excessive or low-sodium diets are associated with CVD.	Low (single multicountry study).	Low.
Alcohol	Compared with nonalcoholic beverages, alcoholic beverages raise HDL-C.	Compared with not drinking, moderate drinking (up to 5 g alcohol/day) may reduce risk of CVD; but higher intakes (>30 g/day and above) increase risk of hemorrhagic stroke and cancers.	Low (SRMA of both RCT and prospective cohorts)	None.	Alcohol does not reduce risk of MI in LMIC.	Low (single multicountry study).	Low.
DASH dietary pattern	Following a DASH dietary pattern lowers blood pressure and serum lipid risk factors for CVD.	Higher DASH diet scores are associated with reduced all-cause mortality, CVD, and cancer mortality.	Moderate (RCT data, prospective cohorts, but no long-term event trials).	None.	None.	None.	Low if only plant based; moderate when uses low-fat dairy as key aspect.
Dietary portfolio	Following the dietary portfolio approach lowers blood pressure and serum lipid risk factors for CVD.	None.	Moderate (strong RCT data, but no event trials).	None.	None.	None.	Focus on plant foods enhances sustainability.
Mediterranean diet	Following a Mediterranean diet is associated with reduced CVD, blood pressure, and serum lipid risk factors for CVD.	None.	High (SRMA of both RCT and prospective cohorts).	None.	None.	None.	Low.

CHD = coronary heart disease; CVD = cardiovascular disease; DASH = Dietary Approaches to Stop Hypertension; HDL-C = high-density lipoprotein cholesterol; HIC = high-income countries; MUFA = mono-unsaturated fats; LDL-C = low-density lipoprotein cholesterol; LMIC = low- and middle-income countries; MI = myocardial infarction; PUFA = polyunsaturated fats; RCT = randomized controlled trial; SRMA = systematic review/meta-analysis; T2DM = type 2 diabetes mellitus; TC = total cholesterol.

stroke, but the between-region difference was not significant ($p = 0.15$ for interaction). In low-income countries and LMIC, heavy episodic drinking pattern and higher intakes of alcohol were associated with a composite outcome of mortality, MI, stroke, cancer, injury, and hospitalization. Current drinking was associated with an increased risk of alcohol-related cancers.

Summary of alcohol. The CVD risk benefits of moderate alcohol consumption (up to 5 g/day) compared with abstinence are small but consistent within individual study populations. Higher levels of consumption increase CVD risk, beginning at 30 g/day in women and 45 g/day in men. Therefore, recommendations for moderate alcohol consumption for CVD prevention in people who already drink must consider individual preferences, population-specific responses, and risk of comorbid conditions, such as addiction and cancers. It is not recommended that nondrinkers take up drinking.

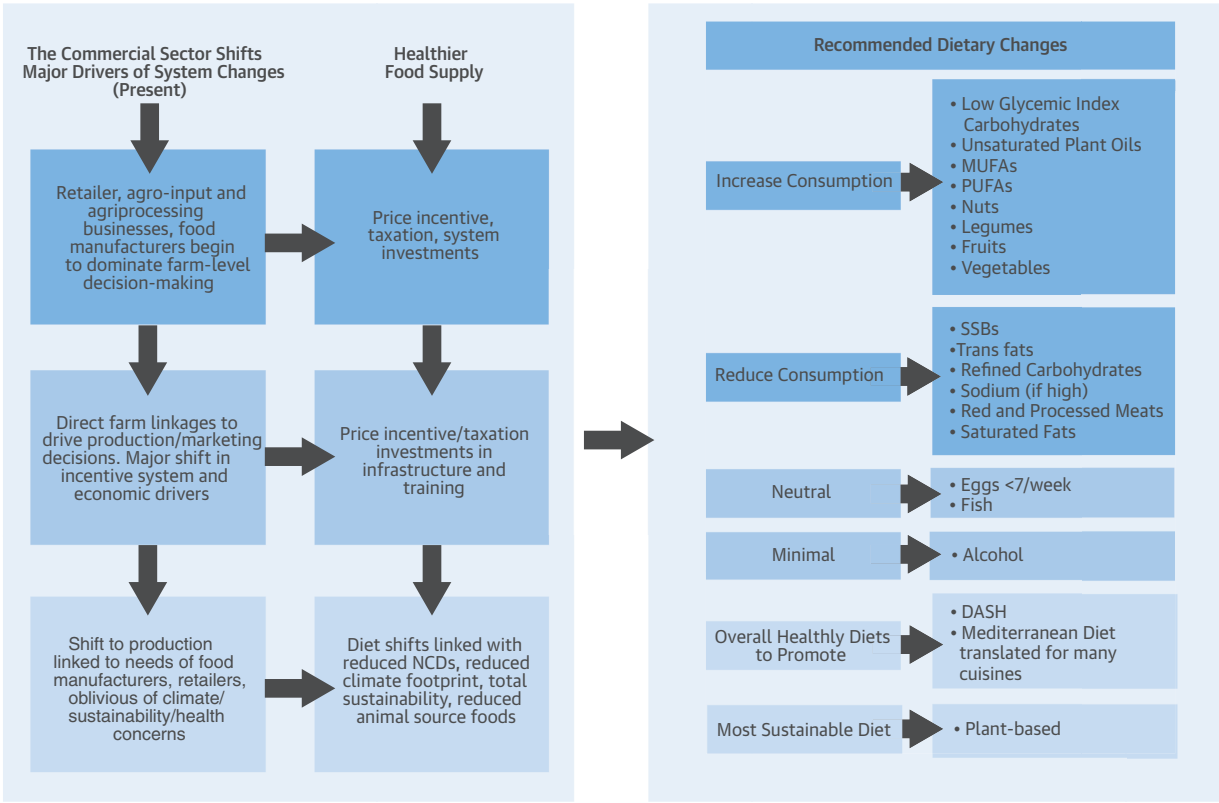
DIETARY PATTERNS. Dietary pattern analysis assesses overall diet quality and is easier to translate into dietary recommendations than recommendations for single foods or nutrients (277). The U.S. Dietary Guidelines Advisory Committee modeled 3 dietary patterns—the Healthy U.S.-Type Pattern, the Healthy Mediterranean-Style Pattern, and the Healthy Vegetarian Pattern—for healthfulness and nutritional adequacy. All 3 include components of a dietary pattern associated with health benefits (277). The DASH diet emphasizes fruits, vegetables, whole grains, low-fat dairy products, and discourages sugar-sweetened foods and beverages, red meat, and added fats (254). The DASH diet was associated with lower systolic blood pressure, diastolic blood pressure, and cholesterol. However, the DASH trials were short and not designed to evaluate the impact of this diet on CVD clinical events. Further improvements in cardiovascular risk factors were seen when part of the carbohydrate in DASH was replaced with either monounsaturated fat or protein (278). In prospective cohort studies, a higher adherence to a DASH dietary pattern is associated with reduced stroke, as well as all-cause, CVD, and cancer mortality (279,280). The Portfolio Diet (281), which includes 30 g/day of nuts, 20 g/day of viscous fiber (i.e., oats, barley, legumes), 80 g/day of vegetable protein (soy, beans, chickpeas, lentils), and 2 g/day of plant sterols (plant sterol margarine) from plant foods, lowers LDL-C by up to 30% within 4 weeks, similar to statins (281). Reductions of 13% to 23% are maintained when this diet was extended to 12 months (282,283). However, no RCT has evaluated the impact of this diet on CVD clinical events. Strong evidence from prospective cohort studies and randomized trials shows reduced CHD with the consumption of a higher-fat Mediterranean diet (196). In the PREDIMED randomized trial a plant-based diet high in nuts or extra virgin olive oil, vegetables, fruits, legumes, fish, and poultry but low in red meats, sweets, and whole-fat dairy was superior to the control group assigned to a low-fat Western diet in preventing CVD (164). It bears mention that low-fat diets have not been particularly effective for long-term CVD risk reduction, partly due to the difficulty sustaining such a diet in the long term (284,285). Online Table 1 translates the essence of the Mediterranean diet to other regions and cultures, suggesting replacement foods that maintain the major food groups in particular regions. Randomized trials of CVD outcomes are needed in different regions of the world.

The **Central Illustration** provides a summary of the key diet changes needed and some of the options for changing our food system to attain this goal is summarized in the next section.

HEALTHIER FOOD SYSTEMS

If more people made healthier choices, the food systems would have greater incentives to produce healthier items. At the same time, a healthier food supply enables individuals to make healthier choices. Developing food systems that underpin healthier dietary patterns on the basis of our consensus noted previously involves improving the food supply by producing more heart-healthy foods and fewer foods associated with CVD (**Central Illustration**). It extends beyond merely growing and raising these foods to ensuring they reach people everywhere in a still healthy form in a way that is accessible and acceptable, including groups of lower socioeconomic status. Changing food systems thus extends beyond agricultural production to what happens between farm and fork (31). This is challenging in the modern global food system given the often extensive distance between production and consumption; multiple and complex transformations in ingredients and foods between farm and fork; and a multitude of other forces discouraging diversity and freshness. Producing more poultry, for example, may be seen as a way to enhance the quality of the food supply because it is healthier relative to red meat, but much chicken is transformed into fast food and other calorie-rich, ultra-processed, heavily advertised presentations (286). Additional public health impacts of industrial poultry production are also well reported, such as food-borne disease (e.g., *E. coli*, *Salmonella*, *Listeria*), antimicrobial residues, and avian flu (287). Clearly families and individuals also play a major role in selecting the quality

CENTRAL ILLUSTRATION **Diet, Cardiovascular Disease, and the Food System: Recommendations for Global Food System Changes Needed to Create a Sustainable Healthy Diet**



Anand, S.S. et al. J Am Coll Cardiol. 2015; 66(14):1590–614.

This figure summarizes the relationship between the major components of diet as well as overall dietary patterns that will lead to reduced risk of cardiovascular disease. This summarizes how the global food system contributes to dietary patterns that greatly increase the risks for the population to experience ill health. Although many perceive food consumption as an individual choice driven by the desires of consumers for tasty food, linked with rising incomes and urbanization, we have shown that the food system and all the stakeholders within it should play a major role. DASH = Dietary Approaches to Stop Hypertension; MUFA = mono-unsaturated fats; NCD = noncommunicable disease; PUFA = polyunsaturated fats; SSB = sugar-sweetened beverage.

of their diet, particularly in HIC and among middle- and higher-income LMIC, but our focus herein is on the broader environmental factors affecting this system.

There are also huge political, economic, and environmental challenges to changing the food system, not the least of which is the capacity of governments to implement change (288). Yet change is possible. It can start with a wider understanding of how the modern food system shapes dietary patterns and eating behaviors, as supported by education that makes clear the interrelationship between food systems, food environments, diets and adverse public health and environmental outcomes, and the potential for better diets to lead to major improvements in public health and environmental sustainability. The pace and effectiveness of improvements will largely depend on political will, encouraged and reinforced by the work of civil society, with health-related professionals having important contributions to make and dietary guidelines sensitive to these issues having a major role to play.

IDENTIFYING FOOD SYSTEM SOLUTIONS. The task may be daunting, but there are many ways to move toward healthier food systems. There is no one solution but a multitude of both top-down and bottom-up starting points. Many changes need not be drastic and costly, and small modifications can improve nutritional intakes and overall diet quality (Online Appendix).

The first "top-down" step is to undertake rigorous analytical work at the global level in order to identify the main levers for change for each of the food groups and provide a framework more conducive to the success of bottom-up approaches (289). The bottom-up approach starts with engaging communities in finding their own solutions to food system–related problems with an emphasis on overall dietary patterns (277,290–292).

Experience indicates actions by the following key actors have potential to improve food systems, many of which can simultaneously have health and environmental benefits:

- **Governments** can review policies in various sectors which influence food systems for coherence with dietary objectives and realign policies so they "do no harm" (293–296); explore fiscal incentives from farm to fork to realign food and agricultural systems; and set a coherent framework of standards and regulations for large transnational food companies to disincentivize the production, sale, and marketing of foods high in refined carbohydrates and low in heart-healthy elements, including clear standards for promotional marketing (297,298). Continued efforts are needed to develop a systems-oriented, mutually reinforcing approach to reduce excessive consumption of SSBs (299).
- **Communities** can identify community-based solutions to poor food access, such as through engaging local/small/family farmers and/or innovative community-oriented production and marketing of whole grains, fruits, vegetables, legumes, and nuts in low-income communities, and developing local food policy councils (300).
- **Farmers and food producers** can leverage the potential of traditional/indigenous crops as part of biodiverse food systems, and support urban food production. Low-input, biodiverse farming systems are more sustainable than high-input monocultures and tend to be more productive per land area, making greater use of ecological space.
- **Agricultural and rural development agencies and programs** can, where whole grains, fruits, vegetables, legumes, and nuts are in short supply in LMIC, invest in transportation, distribution networks, procurement logistics, and price information systems to remove supply bottlenecks.
- **Investors in agriculture and food research and development** can shift investments and research toward the production, distribution, and consumption of healthier, more diverse sources of nutrients and agricultural programs; make national and international investments in food research aligned with a healthy food supply; and invest in pre- and post-harvest systems to reduce food waste and focusing on recycling in home food waste (101,301).
- **Food processors and manufacturers of all scales** can ensure food processing does not strip fiber and positive nutrients from foods; direct research and marketing to enhance the desirability of nutritious foods to consumers.
- **Educational institutions** can improve curricula and build capacity among nutrition and public health students and professionals to identify and implement multisectorial food systems solutions to unhealthy diets.

In addition, middle-income countries and HIC have introduced measures to improve food environments—mainly introduced in the context of obesity—including (302):

- Taxation of SSBs and unessential foods (junk foods in general) in Mexico. A number of other LMIC are considering these taxes, and several European countries have introduced taxes on different food groups.
- Front-of-pack labels on packaged food products considered healthy (303) or unhealthy (304). Several Latin American countries have regulated front-of-pack labels, including Ecuador's traffic light label (implemented in 2014), and Chile's warning label (being implemented over the next 4 years). Several Asian countries are developing a healthier choices option.
- Mandatory school food standards are the most widely implemented action, in around 11 middle-income countries and 15 HIC. However the direction of the mandate must be appropriate.
- Effective marketing controls of unhealthy foods and beverages have not been widely implemented. Chile's new program is the most extensive (implemented over the 2015 to 2019 period). Mexico, South Korea, the United Kingdom, and Ireland present rare exceptions, but the regulations have loopholes and do not comprehensively restrict marketing. Efforts to restrict marketing in other countries through regulation have been met with strong opposition from the food and advertising industries. A critical issue is to go beyond marketing focused at children's TV to all TV viewed during key hours or full time and also to consider all other media. A second issue is linking marketing controls with the front-of-pack labeling efforts so either only healthy foods are marketed or all unhealthy foods are not allowed to be marketed.

- Encouraging food processers to produce vegetable protein-rich foods as meat and dairy alternatives with subsidies/tax incentives.

Although the evidence base for these policy options is growing more and more rigorous, evaluation of these actions is needed, though econometric evaluations of the Mexican 10% SSB tax and 8% junk food taxes are underway using longitudinal nationally representative scanned food purchase data at the household level. A critical need is much more evaluation of all these options, including to assessments of how they feed back to affect the food system (302,305). Nutrition labels, for example, have been shown to have a clear impact on reformulation (303,306).

CONCLUSIONS

This paper has provided a state-of-the-art review of the link between specific macronutrients and foods and CVD and summarized how the global food system contributes to dietary patterns that greatly increase the risks for the population to experience ill health. Although many perceive food consumption as an individual choice driven by the desires of consumers for tasty food, linked with rising incomes and urbanization, we have shown that the food system and all the stakeholders within it should play a major role (307).

Short-term controlled-feeding studies with CVD risk factors as outcomes; long-term prospective cohort studies with CHD, stroke, and T2DM as outcomes; and a limited number of RCTs with CVD as the outcome collectively show that multiple aspects of diet substantially influence CVD risk. However, similar data are needed from LMIC as dietary patterns differ in various regions of the world and the context in which foods are accessed differs markedly.

On the basis of the current evidence, the optimal dietary pattern to reduce CVD is one that emphasizes whole grains, fruits and vegetables, legumes, nuts, fish, poultry, and moderate dairy and heart-healthy vegetable oil intake; this pattern will likely reduce the CVD risk by about one-third. This healthy dietary pattern needs also to be low in refined grains, added sugars, trans fats, SSBs, and red and processed meats. The traditional Mediterranean-type diet provides a well-tested prototype for this healthy dietary pattern. Given that we now understand the components of this diet sufficiently, it may be possible to translate this pattern to other regions, with appropriate similar food replacements on the basis of food availability and preferences (Online Table 1). Despite significant advances in our understanding of optimal dietary patterns to prevent CVD, additional research including large cohort studies and RCTs of dietary patterns are needed in different regions of the world to address existing knowledge gaps. This includes evaluation of the impact of specific fruits and vegetables, types of dairy foods, type and amount of carbohydrate, optimal cooking oils, region-specific dietary patterns, and cooking methods. Finally, we acknowledge that although this paper is focused on CVD, dietary choices and recommendations should also be made with consideration of the environmental implications of food choice (i.e., the environmental impact of poultry, livestock/cattle production, and diminishing wild fish stocks) and the role of diet in other disease processes. Human health must be linked to environmental health—the basis of the new Sustainable Development Goals (241). Additionally, modifications to our recommendations may provide improved protection against cancer or neurodegenerative or autoimmune diseases.

Policy actions and interventions that improve food supplies and dietary patterns have social, cultural, and environmental benefits. There are many opportunities to increase access to healthy food that are also likely to have significant environmental benefits. Health providers throughout the world can lead by advocating for action in the food system, as well as in food environments and behavior change communication. So too can other professionals, civil society and public interest organizations, influential writers and journalists, and organizations of chefs and gastronomes. A synergistic systems approach is essential. The challenge to create and sustain what is healthy and change what is unhealthy is compelling because improving the nourishment that goes into our bodies can have wide-ranging benefits in improving the health of societies and environments. A second major challenge is to identify and implement effective food systems and solutions and evaluate the national and local level policy actions underway to improve our diets.

ACKNOWLEDGMENTS This review comes out of a World Heart Federation consensus conference held May 14 to 16, 2014, at the Population Health Research Institute, Hamilton Health Sciences, and McMaster University in Hamilton, Ontario, Canada. The authors thank all participants and presenters for their thoughtful discussion.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Barry M. Popkin, School of Public Health, Carolina Population Center, University of North Carolina, 137 East Franklin Street, Chapel Hill, North Carolina 27516. E-mail: popkin@unc.edu.

REFERENCES

1. Imamura F, Micha R, Khatibzadeh S, et al. Dietary quality among men and women in 187 countries in 1990 and 2010: a systematic assessment. Lancet Global Health 2015;3:e132–42.

2. Micha R, Khatibzadeh S, Shi P, et al. Global, regional, and national consumption levels of dietary fats and oils in 1990 and 2010: a systematic analysis including 266 country-specific nutrition surveys. BMJ 2014;348:g2272.

3. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380:2224–60.

4. World Health Organization. Obesity and Overweight. Fact Sheet No. 311. Geneva, Switzerland: WHO, 2014.

5. WCRF-AICR. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. London, England: World Cancer Research Fund, 2007.

6. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 2014;384:766–81.

7. World Health Organization. Global Status Report On Noncommunicable Diseases 2010. Geneva, Switzerland: WHO, 2010.

8. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev 2012;70:3–21.

9. Food and Agricultural Organization of the United Nations. Second International Conference on Nutrition (ICN2). Rome, Italy: FAOUN, 2014.

10. Fan S, Brzeska J. Building a Resilient Global Food System by Lowering Food Price Spikes and Volatility. Washington DC: International Food Policy Research Institute, 2014.

11. Babu SC, Blom S. Strengthening Capacity for Resilient Food Systems. Resilience Conference Brief. Washington DC: International Food Policy Research Institute, 2014.

12. Weis T. The Global Food Economy: The Battle for the Future of Farming. London, England: Zed Books, 2007.

13. Reardon T, Chen KZ, Minten B, et al. The quiet revolution in Asia's rice value chains. Ann N Y Acad Sci 2014;1331:106–18.

14. Gardner BL. American Agriculture in the Twentieth Century: How It Flourished and What It Cost. Cambridge, MA: Harvard University Press, 2002.

15. Yudkin J. Sweet and Dangerous: The New Facts About the Sugar You Eat As a Cause of Heart Disease, Diabetes, and Other Killers. New York, NY: Bantam Books, 1972.

16. Mintz S. Sweetness and Power: The Place of Sugar in Modern History. New York, NY: Penguin, 1986.

17. Galloway J. Sugar. In: Kiple K, Ornelas K, editors. The Cambridge World History of Food 2000. New York, NY: Cambridge University Press, 2000: 437–49.

18. Popkin B. The World Is Fat: The Fads, Trends, Policies, and Products That Are Fattening the Human Race. New York, NY: Avery-Penguin Group, 2008.

19. Rashid S, Gulati A, Cummings R. From Parastatals to Private Trade: Lessons from Asian Agriculture. Baltimore, MD: Johns Hopkins University Press, 2008.

20. Anderson K. The Political Economy of Agricultural Price Distortions. Cambridge, England: Cambridge University Press, 2010.

21. World Bank. World Development Report 2008: Agriculture for Development. Washington, DC: World Bank, 2007.

22. Pinstrup-Andersen P. Food Subsidies in Developing Countries: Costs, Benefits, and Policy Options. Baltimore, MD: Johns Hopkins University Press, 1988.

23. Lang T, Heasman M. Food Wars: The Global Battle for Mouths Minds and Market. London, England: Routledge, 2003.

24. Connor J, Schiek W. Food Processing: An Industrial Powerhouse in Transition. New York, NY: Wiley, 1997.

25. Wilkinson J. The food processing industry, globalization and developing countries. Electron J Agr Dev Econ 2004;1:184–201.

26. Welch R, Mitchell P. Food processing: a century of change. Br Med Bull 2000;56:1–17.

27. Fellows P. Food Processing Technology: Principles and Practice. 3rd edition. Cambridge, England: Woodhead, 2009.

28. Moss M. Salt Sugar Fat: How the Food Giants Hooked Us. New York, NY: Random House, 2013.

29. Packard V. The Hidden Persuaders. New York, NY: David McKay Company, 1957:1251–62.

30. Burch D, Lawrence G. Supermarkets and Agri-Food Supply Chains: Transformations in the Production and Consumption of Foods. Cheltenham, England: Edward Elgar, 2007.

31. Hawkes C, Friel S, Lobstein T, Lang T. Linking agricultural policies with obesity and noncommunicable diseases: A new perspective for a globalising world. Food Policy 2012;37:343–53.

32. Starmer E, Witteman A, Wise TA. Feeding the Factory Farm: Implicit Subsidies to the Broiler Chicken Industry. GDAE Working Paper. Medford, MA: Tufts University, 2006.

33. Schaffer HD, Hunt DB, Ray DE. US Agricultural Commodity Policy and Its Relationship to Obesity. Knoxville, TN: Agricultural Policy Analysis Center University of Tennessee, 2007.

34. Weis T. The Ecological Hoofprint: The Global Burden of Industrial Livestock. London, England: Zed Books, 2013.

35. Neven D, Odera M, Reardon T, Wang H. Kenyan supermarkets, emerging middle-class horticultural farmers, and employment impacts on the rural poor. World Dev 2009;37:1802–11.

36. Reardon T, Timmer CP, Minten B. Supermarket revolution in Asia and emerging development strategies to include small farmers. Proceedings of the National Academy of Sciences 2012;109:12332–7.

37. Reardon T, Timmer CP. The economics of the food system revolution. Ann Rev Res Econ 2012;4:225–64.

38. Khandelwal S, Reddy K. Eliciting a policy response for the rising epidemic of overweight-obesity in India. Obes Rev 2013;14:114–25.

39. Wilde PE. Food Policy in the United States: An Introduction. New York, NY: Routledge/Earthscan, 2013.

40. Institute for Agriculture and Trade Policy. Food without Thought: How U.S. Farm Policy Contributes to Obesity. Minneapolis, MN: The Institute for Agriculture and Trade Policy, 2006.

41. Hawkes C, Murphy S. An overview of global food trade. In: Hawkes C, Blouin C, Henson S, Drager N, Dubé L, editors. Trade, Food, Diet and Health: Perspectives and Policy Options. London, England: Wiley-Blackwell, 2010:16–32.

42. Bruinsma J. World Agriculture: Toward 2015/2030. Summary Report. Rome, Italy: Food and Agriculture Organization of the United Nations, 2003.

43. Adair LS, Popkin BM. Are child eating patterns being transformed globally? Obes Res 2005;13:1281–99.

44. Duffey K, Pereira R, Popkin B. Prevalence and energy intake from snacking in Brazil: analysis of the first nationwide individual survey. Eur J Clin Nutr 2013;67:868–74.

45. Ng SW, Zaghloul S, Ali H, et al. Nutrition transition in the United Arab Emirates. Eur J Clin Nutr 2011;65:1328–37.

46. Popkin B, Duffey K. Does hunger and satiety drive eating anymore? Increasing eating occasions and decreasing time between eating occasions in the United States. Am J Clin Nutr 2010;91:1342–7.

47. Wang Z, Zhai F, Du S, Popkin B. Dynamic shifts in Chinese eating behaviors. Asia Pac J Clin Nutr 2008;17:123–30.

48. Duffey KJ, Rivera J, Popkin B. Snacking is prevalent in Mexico. J Nutr 2014;144:1843–9.

49. Monteiro C, Gomes F, Cannon G. The snack attack. Am J Public Health 2010;100:975–81.

50. Monteiro C, Moubarac J, Cannon G, et al. Ultra-processed products are becoming dominant in the global food system. Obes Rev 2013;14:21–8.

51. Poti JM, Mendez MA, Ng SW, Popkin BM. Is the degree of food processing and convenience linked with the nutritional quality of foods purchased by US households? Am J Clin Nutr 2015;99:162–71.

52. Reddy KS. Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action. Public Health Nutr 2002;5:231–7.

53. Reddy S, Katan M. Diet, nutrition and the prevention of hypertension and cardiovascular diseases. Public Health Nutr 2004;7:167–86.

54. Gross LS, Li L, Ford ES, Liu S. Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment. Am J Clin Nutr 2004;79: 774–9.

55. Duffey KJ, Popkin BM. High-fructose corn syrup: is this what's for dinner? Am J Clin Nutr 2008;88:1722S–32S.

56. Basu S, Yoffe P, Hills N, Lustig R. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. PLoS One 2013;8:e57873.

57. Kleiman S, Ng SW, Popkin B. Drinking to our health: can beverage companies cut calories while maintaining profits? Obes Rev 2012;13:258–74.

58. Monteiro C. The big issue is ultra-processing. Journal of the World Public Health Nutrition Association 2010. Available at: http://www.wphna.org/wn_commentary_ultraprocessing_nov2010.asp. Accessed August 7, 2015.

59. Monteiro CA, Levy RB, Claro RM, et al. Increasing consumption of ultra-processed foods and likely impact on human health: evidence from Brazil. Public Health Nutr 2011;14:5–13.

60. Ng SW, Slining MM, Popkin BM. Use of caloric and noncaloric sweeteners in US consumer packaged foods, 2005–2009. J Acad Nutr Diet 2012; 112:1828–34.

61. Radhika G, Van Dam RM, Sudha V, et al. Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57). Metabolism 2009; 58:675–81.

62. Wang L. Report of China Nationwide Nutrition and Health Survey 2002(1): Summary Report. Beijing, China: People's Medical Publishing House, 2005.

63. Drewnowski A, Popkin BM. The nutrition transition: new trends in the global diet. Nutr Rev 1997;55:31–43.

64. Aldersey C. Consumer trends and usage of fats and oils. Paper presented at: 14th Biennial Sunflower Conference; June 24-26, 2003; Australia.

65. Micha R, Khatibzadeh S, Shi P, et al. Global, regional, and national consumption levels of dietary fats and oils in 1990 and 2010: a systematic analysis including 266 country-specific nutrition surveys. Br Med J 2014;348:g2272.

66. Delgado CL, Rosegrant M, Steinfield H, et al. Livestock to 2020: The Next Food Revolution. Food, Agriculture, and the Environment. Washington, DC: International Food Policy Research Institute, 1999.

67. Delgado CL. Rising consumption of meat and milk in developing countries has created a new food revolution. J Nutr 2003;133 11 Suppl 2:3907S–10S.

68. Popkin BM, Du S. Dynamics of the nutrition transition toward the animal foods sector in China and its implications: a worried perspective. J Nutr 2003;133 11 Suppl 2:3898S–906S.

69. Food and Agricultural Organization of the United Nations. Livestock's Long Shadow: Environmental Issues and Options. Rome, Italy: Food and Agricultural Organization of the United Nations, 2007.

70. Sinha R, Cross AJ, Graubard BI, Leitzmann MF, Schatzkin A. Meat intake and mortality: A prospective study of over half a million people. Arch Intern Med 2009;169:562–71.

71. Kearney J. Food consumption trends and drivers. Philos Trans R Soc Lond B Biol Sci 2010; 365:2793–807.

72. Anand SS, Dehghan M, de Souza R, et al. Dietary Patterns and Macronutrients and CVD – A Summary Statement The World Heart Federation Nutrition Conference. Hamilton, Canada: World Heart Federation Nutrition Conference, 2014.

73. Food and Agriculture Organization of the United Nations. The State of Food and Agriculture, 2009. Rome, Italy: Food and Agriculture Organization of the United Nations, 2009.

74. Food and Agriculture Organization of the United Nations. The State of World Fisheries and Aquaculture. Rome, Italy: Food and Agricultural Organization United Nations, 2012.

75. Watson R, Pauly D. Systematic distortions in world fisheries catch trends. Nature 2001;414: 534–6.

76. Economic Research Service/USDA. International Agriculture and Trade Outlook. Washington, DC: Economic Research Service, 2003.

77. Schneider AVC. Overview of the market and consumption of puises in Europe. Br J Nutr 2002; 88:243–50.

78. Oniang O, Mutuku J, Malaba SJ. Contemporary African food habits and their nutritional and health implications. Asia Pac J Clin Nutr 2003;12:331–6.

79. Aykroyd WR, Doughty J, Walker A. Legumes in human nutrition. Food and Agriculture Organization of the United Nations. FAO Food Nutr Rep 1982;20:1–152.

80. Maredia M. Global pulse production and consumption trends: the potential of pulses to achieve 'feed the future' food and nutritional security goals. Paper presented at: Global Pulse Researchers Meeting; February 2012; Rwanda.

81. International Nutrition & Dried Fruit Council. Global Statistical Review 2007–2012. Reus, Spain: INC, 2012.

82. Zhou Y, Du S, Su C, Zhang B, Wang H, Popkin B. The food retail revolution in China and its association with diet and health. Food Policy 2015;55:92–100.

83. Popkin BM. Nutrition, agriculture and the global food system in low and middle income countries. Food Policy 2014;47:91–6.

84. Slining MM, Ng SW, Popkin BM. Food companies' calorie-reduction pledges to improve U.S. diet. Am J Prev Med 2013;44:174–84.

85. Monteiro CA, Cannon G. The impact of transnational "Big Food" companies on the south: A view from Brazil. PLoS Med 2012;9:e1001252.

86. Monteiro CA, Levy R, Claro R, de Castro I, Cannon G. Increasing consumption of ultra-processed foods and likely impact on human health: evidence from Brazil. Public Health Nutr 2011;14:5–13.

87. Ng SW, Popkin BM. The Healthy Weight Commitment Foundation Marketplace Commitment and Consumer Packaged Goods purchased by US households with children. Am J Prev Med 2014;47:508–19.

88. Blisard WN, Stewart H, Jolliffe D. Low-Income Households' Expenditures on Fruits and Vegetables. Washington, DC: Economic Research Service, 2004.

89. Hall KD, Guo J, Dore M, Chow C. The progressive increase of food waste in America and its environmental impact. PLoS One 2009;4:e7940.

90. Centers for Disease Control and Prevention. Fruit and vegetable consumption among adults–United States, 2005. Morb Mortal Wkly Rep 2007; 56:213–7.

91. Wang Y, Ge K, Popkin BM. Why do some overweight children remain overweight, whereas others do not? Public Health Nutr 2003;6:549–58.

92. Hall JN, Moore S, Harper SB, Lynch JW. Global variability in fruit and vegetable consumption. Am J Prev Med 2009;36:402–9.e5.

93. McMichael AJ, Powles JW, Butler CD, Uauy R. Food, livestock production, energy, climate change, and health. Lancet 2007;370:1253–63.

94. Hoekstra A, Chapagain A. Globalization of Water: Sharing the Planet's Freshwater Resources. New York, NY: Wiley-Blackwell, 2008.

95. Hoekstra AY, Chapagain AK. Water footprints of nations: Water use by people as a function of their consumption pattern. Water Resour Manage 2007;21:35–48.

96. McIntyre B, Herren H, Wakhungu J, Watson R. International Assessment of Agricultural Knowledge, Science and Technology for Development: Agriculture at a Crossroads. Washington DC: Island Press, 2009.

97. Hertel TW, Burke MB, Lobell DB. The poverty implications of climate-induced crop yield changes by 2030. Global Environ Change 2010;20:577–85.

98. Cline WR. Global Warming and Agriculture: Impact Estimates by Country. Washington, DC: Peter G. Peterson Institute for International Economics, 2007.

99. Intergovernmental Panel on Climate Change. Climate Change 2014: Synthesis Report. Geneva, Switzerland: IPCC, 2014.

100. Food and Agricultural Organization of the United Nations. Food Wastage Footprint: Impacts on Natural Resources: Summary Report. Rome, Italy: Food and Agricultural Organization of the United Nations, 2013.

101. Hall KD, Guo J, Dore M, Chow CC. The progressive increase of food waste in America and its environmental impact. PLoS One 2009;4:e7940.

102. Parfitt J, Barthel M, Macnaughton S. Food waste within food supply chains: quantification and potential for change to 2050. Phil Trans R Soc B: Bio Sci 2010;365:3065–81.

103. Bhupathiraju SN, Tobias DK, Malik VS, et al. Glycemic index, glycemic load, and risk of type 2 diabetes: results from 3 large US cohorts and an updated meta-analysis. Am J Clin Nutr 2014;100: 218–32.

104. Chiasson JL, Josse RG, Gomis R, et al. Acarbose for prevention of type 2 diabetes mellitus: the STOP-NIDDM randomised trial. Lancet 2002; 359:2072–7.

105. Salmeron J, Manson JE, Stampfer MJ, et al. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA 1997;277:472–7.

106. Jakobsen MU, Dethlefsen C, Joensen AM, et al. Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. Am J Clin Nutr 2010;91:1764–8.

107. Mirrahimi A, de Souza RJ, Chiavaroli L, et al. Associations of glycemic index and load with coronary heart disease events: a systematic review and meta-analysis of prospective cohorts. J Am Heart Assoc 2012;1:e000752.

108. Mattei J, Hu FB, Campos H. A higher ratio of beans to white rice is associated with lower cardiometabolic risk factors in Costa Rican adults. Am J Clin Nutr 2011;94:869–76.

109. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr 2003;77: 1146–55.

110. Voight B, Peloso G, Orho-Melander M, et al. Plasma HDL cholesterol and risk of myocardial infarction: a mendelian randomisation study. Lancet 2012;380:572–80.

111. Hu EA, Pan A, Malik V, Sun Q. White rice consumption and risk of type 2 diabetes: meta-analysis and systematic review. BMJ 2012;344: e1454.

112. Aune D, Norat T, Romundstad P, Vatten LJ. Whole grain and refined grain consumption and the risk of type 2 diabetes: a systematic review and dose–response meta-analysis of cohort studies. Eur J Epidemiol 2013;28:845–58.

113. Sun Q, Spiegelman D, van Dam RM, et al. White rice, brown rice, and risk of type 2 diabetes in US men and women. Arch Intern Med 2010;170: 961–9.

114. Yusuf S, Rangarajan S, Teo K, et al. Cardiovascular risk and events in 17 low-, middle-, and high-income countries. N Engl J Med 2014;371: 818–27.

115. Mattei J, Malik V, Wedick NM, et al. A symposium and workshop report from the Global Nutrition and Epidemiologic Transition Initiative: nutrition transition and the global burden of type 2 diabetes. Br J Nutr 2012;108: 1325–35.

116. Brand-Miller J, Hayne S, Petocz P, Colagiuri S. Low-Glycemic index diets in the management of diabetes: a meta-analysis of randomized controlled trials. Diabetes Care 2003;26:2261–7.

117. Livesey G, Taylor R, Hulshof T, Howlett J. Glycemic response and health—a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. Am J Clin Nutr 2008;87:258S–68S.

118. Jenkins DA, Kendall CC, McKeown-Eyssen G, et al. Effect of a low–glycemic index or a high–cereal fiber diet on type 2 diabetes: A randomized trial. JAMA 2008;300:2742–53.

119. Goff LM, Cowland DE, Hooper L, Frost GS. Low glycaemic index diets and blood lipids: A systematic review and meta-analysis of randomised controlled trials. Nutr Metab Cardiovasc Dis 2013;23:1–10.

120. Yang Q, Zhang Z, Gregg EW, et al. Added sugar intake and cardiovascular diseases mortality among US adults. JAMA Intern Med 2014;174: 516–24.

121. National Cancer Institute. Sources of Calories From Added Sugars Among the U.S. Population, 2005–06. Bethesda, MD: National Cancer Institute, 2014.

122. World Health Organization. Draft Guidelines on free sugars released for public consultation. 2014. Geneva, Switzerland.

123. Xi B, Huang Y, Reilly KH, et al. Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. Br J Nutr 2015;113:709–17.

124. Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. Am J Clin Nutr 2013;98:1084–102.

125. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. Br Med J 2013;346:e7492.

126. Mozaffarian D, Hao T, Rimm EB, et al. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med 2011;364: 2392–404.

127. Schulze MB, Manson JE, Ludwig DS, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA 2004;292:927–34.

128. Malik VS, Popkin BM, Bray GA, et al. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care 2010;33:2477–83.

129. Johnson RJ, Segal MS, Sautin Y, et al. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. Am J Clin Nutr 2007;86:899–906.

130. Dhingra R, Sullivan L, Jacques PF, et al. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation 2007;116:480–8.

131. Janssens JP, Shapira N, Debeuf P, et al. Effects of soft drink and table beer consumption on insulin response in normal teenagers and carbohydrate drink in youngsters. Eur J Cancer Prev 1999;8:289–96.

132. De Koning L, Malik VS, Kellogg MD, et al. Sweetened beverage consumption, incident coronary heart disease, and biomarkers of risk in men. Circulation 2012;125:1735–41.

133. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean diet and incidence of and mortality from coronary heart disease and stroke in women. Circulation 2009;119:1093–100.

134. Bernstein AM, de Koning L, Flint AJ, et al. Soda consumption and the risk of stroke in men and women. Am J Clin Nutr 2012;95:1190–9.

135. Larsson SC, Åkesson A, Wolk A. Sweetened beverage consumption is associated with increased risk of stroke in women and men. J Nutr 2014;144:856–60.

136. Odegaard AO, Koh W-P, Arakawa K, et al. Soft drink and juice consumption and risk of physician-diagnosed incident type 2 diabetes: the Singapore Chinese Health Study. Am J Epidemiol 2010;171: 701–8.

137. Hernández-Cordero S, Barquera S, Rodríguez-Ramírez S, et al. Substituting water for sugar-sweetened beverages reduces circulating triglycerides and the prevalence of metabolic syndrome in obese but not in overweight Mexican women in a randomized controlled trial. J Nutr 2014;144:1742–52.

138. Jenkins DJA, Kendall CWC, Vuksan V, et al. Effect of lowering the glycemic load with canola oil on glycemic control and cardiovascular risk factors: a randomized controlled trial. Diabetes Care 2014;37:1806–14.

139. Brouwer IA, Wanders AJ, Katan MB. Effect of animal and industrial trans fatty acids on HDL and LDL cholesterol levels in humans–a quantitative review. PLoS One 2010;5:e9434.

140. Bendsen NT, Christensen R, Bartels EM, Astrup A. Consumption of industrial and ruminant trans fatty acids and risk of coronary heart disease: a systematic review and meta-analysis of cohort studies. Eur J Clin Nutr 2011;65:773–83.

141. Mozaffarian D, Katan MB, Ascherio A, et al. Trans fatty acids and cardiovascular disease. N Engl J Med 2006;354:1601–13.

142. Marshall JA, Hamman RF, Baxter J. High-fat, low-carbohydrate diet and the etiology of non-insulin-dependent diabetes mellitus: the San Luis Valley Diabetes Study. Am J Epidemiol 1991;134: 590–603.

143. Riserus U. Fatty acids and insulin sensitivity. Curr Opin Clin Nutr Metab Care 2008;11:100–5.

144. Lindstrom J, Peltonen M, Eriksson JG, et al. High-fibre, low-fat diet predicts long-term weight loss and decreased type 2 diabetes risk: the Finnish Diabetes Prevention Study. Diabetologia 2006;49:912–20.

145. Meyer K, Kushi L, Jacobs D, Folsom A. Dietary fat and incidence of type 2 diabetes in older Iowa women. Diabetes Care 2001;24:1528–35.

146. Salmerón J, Hu FB, Manson JE, et al. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr 2001;73:1019–26.

147. Simila ME, Kontto JP, Valsta LM, et al. Carbohydrate substitution for fat or protein and risk of type 2 diabetes in male smokers. Eur J Clin Nutr 2012;66:716–21.

148. Song Y, Manson JE, Buring JE, Liu S. A prospective study of red meat consumption and type 2 diabetes in middle-aged and elderly women: The Women's Health study. Diabetes Care 2004;27:2108–15.

149. van Dam RM, Willett WC, Rimm EB, et al. Dietary fat and meat intake in relation to risk of

type 2 diabetes in men. Diabetes Care 2002;25:417–24.

150. Vessby B, Uusitupa M, Hermansen K, et al. Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: the KANWU Study. Diabetologia 2001;44:312–9.

151. Perez-Jimenez F, Lopez-Miranda J, Pinillos MD, et al. A Mediterranean and a high-carbohydrate diet improve glucose metabolism in healthy young persons. Diabetologia 2001;44:2038–43.

152. Pan A, Sun Q, Bernstein AM, Manson JE, Willett WC, Hu FB. Changes in red meat consumption and subsequent risk of type 2 diabetes mellitus: three cohorts of US men and women. JAMA Intern Med 2013;173:1328–35.

153. Micha R, Michas G, Mozaffarian D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes–an updated review of the evidence. Curr Atheroscler Rep 2012;14:515–24.

154. Gao D, Ning N, Wang C, et al. Dairy products consumption and risk of type 2 diabetes: systematic review and dose-response meta-analysis. PLoS One 2013;8:e73965.

155. Sluijs I, Forouhi NG, Beulens JW, et al. The amount and type of dairy product intake and incident type 2 diabetes: results from the EPIC-InterAct Study. Am J Clin Nutr 2012;96:382–90.

156. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr 2010;91:535–46.

157. Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. PLoS Med 2010;7:e1000252.

158. Jakobsen MU, O'Reilly EJ, Heitmann BL, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. Am J Clin Nutr 2009;89:1425–32.

159. Farvid MS, Ding M, Pan A, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. Circulation 2014;130:1568–78.

160. de Oliveira Otto MC, Mozaffarian D, Kromhout D, et al. Dietary intake of saturated fat by food source and incident cardiovascular disease: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr 2012;96:397–404.

161. Hu FB, Stampfer MJ, Manson JE, et al. Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. Am J Clin Nutr 1999;70:1001–8.

162. Halton TL, Willett WC, Liu S, et al. Low-carbohydrate diet score and the risk of coronary heart disease in women. N Engl J Med 2006;355:1991–2002.

163. de Lorgeril M, Renaud S, Salen P, et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. Lancet 1994;343:1454–9.

164. Estruch R, Ros E, Salas-Salvado J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med 2013;368:1279–90.

165. Kabagambe EK, Baylin A, Ascherio A, Campos H. The type of oil used for cooking is associated with the risk of nonfatal acute myocardial infarction in Costa Rica. J Nutr 2005;135:2674–9.

166. Bingham SA, Hughes R, Cross AJ. Effect of white versus red meat on endogenous N-nitrosation in the human colon and further evidence of a dose response. J Nutr 2002;132 11 Suppl:3522S–5S.

167. Bastide NM, Pierre FHF, Corpet DE. Heme iron from meat and risk of colorectal cancer: a meta-analysis and a review of the mechanisms involved. Cancer Prev Res 2011;4:177–84.

168. Joosen AMCP, Kuhnle GGC, Aspinall SM, et al. Effect of processed and red meat on endogenous nitrosation and DNA damage. Carcinogenesis 2009;30:1402–7.

169. Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus a systematic review and meta-analysis. Circulation 2010;121:2271–83.

170. Rohrmann S, Overvad K, Bueno-de-Mesquita HB, et al. Meat consumption and mortality–results from the European prospective investigation into cancer and nutrition. BMC Med 2013;11:63–74.

171. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and mortality: results from 2 prospective cohort studies. Arch Intern Med 2012;172:555–63.

172. Bernstein AM, Sun Q, Hu FB, et al. Major dietary protein sources and risk of coronary heart disease in women. Circulation 2010;122:876–83.

173. Lee JE, McLerran DF, Rolland B, et al. Meat intake and cause-specific mortality: a pooled analysis of Asian prospective cohort studies. Am J Clin Nutr 2013;98:1032–41.

174. Chen M, Pan A, Malik VS, Hu FB. Effects of dairy intake on body weight and fat: a meta-analysis of randomized controlled trials. Am J Clin Nutr 2012;96:735–47.

175. Kai SHY, Bongard V, Simon C, et al. Low-fat and high-fat dairy products are differently related to blood lipids and cardiovascular risk score. Eur J Prev Cardiol 2013;21:1557–67.

176. Pereira MA, Jacobs DR Jr., Van Horn L, et al. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. JAMA 2002;287:2081–9.

177. Fumeron F, Lamri A, Khalil CA, et al. Dairy consumption and the incidence of hyperglycemia and the metabolic syndrome results from a French prospective study, Data from the Epidemiological Study on the Insulin Resistance Syndrome (DESIR). Diabetes Care 2011;34:813–7.

178. Rideout TC, Marinangeli C, Martin H, et al. Consumption of low-fat dairy foods for 6 months improves insulin resistance without adversely affecting lipids or bodyweight in healthy adults: a randomized free-living cross-over study. Nutr J 2013;12:56–64.

179. Choi HK, Willett WC, Stampfer MJ, et al. Dairy consumption and risk of type 2 diabetes mellitus in men: a prospective study. Arch Internal Med 2005;165:997–1003.

180. Liu S, Choi HK, Ford E, et al. A prospective study of dairy intake and the risk of type 2 diabetes in women. Diabetes Care 2006;29:1579–84.

181. Elwood PC, Pickering JE, Givens DI, Gallacher JE. The consumption of milk and dairy foods and the incidence of vascular disease and diabetes: an overview of the evidence. Lipids 2010;45:925–39.

182. Tong X, Dong JY, Wu ZW, et al. Dairy consumption and risk of type 2 diabetes mellitus: a meta-analysis of cohort studies. Eur J Clin Nutr 2011;65:1027–31.

183. Chen M, Sun Q, Giovannucci E, et al. Dairy consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. BMC Med 2014;12:215.

184. Umesawa M, Iso H, Date C, et al. Dietary intake of calcium in relation to mortality from cardiovascular disease the JACC Study. Stroke 2006;37:20–6.

185. Zong G, Sun Q, Yu D, et al. Dairy consumption, type 2 diabetes and changes in cardiometabolic traits-a prospective cohort study of middle-aged and older Chinese in Beijing and Shanghai. Diabetes Care 2013;37:56–63.

186. Qureshi AI, Suri MFK, Ahmed S, et al. Regular egg consumption does not increase the risk of stroke and cardiovascular diseases. Med Sci Monit 2006;13:CR1–8.

187. McCance RA, Widdowson EM. The Composition of Foods. London, England: Medical Research Council, 1960.

188. Weggemans RM, Zock PL, Katan MB. Dietary cholesterol from eggs increases the ratio of total cholesterol to high-density lipoprotein cholesterol in humans: a meta-analysis. Am J Clin Nutr 2001;73:885–91.

189. Reaven GM, Abbasi F, Bernhart S, et al. Insulin resistance, dietary cholesterol, and cholesterol concentration in postmenopausal women. Metabolism 2001;50:594–7.

190. Goodrow EF, Wilson TA, Houde SC, et al. Consumption of one egg per day increases serum lutein and zeaxanthin concentrations in older adults without altering serum lipid and lipoprotein cholesterol concentrations. J Nutr 2006;136:2519–24.

191. Wenzel AJ, Gerweck C, Barbato D, et al. A 12-wk egg intervention increases serum zeaxanthin and macular pigment optical density in women. J Nutr 2006;136:2568–73.

192. Katz DL, Evans MA, Nawaz H, et al. Egg consumption and endothelial function: a randomized controlled crossover trial. Int J Cardiol 2005;99:65–70.

193. Njike V, Faridi Z, Dutta S, et al. Daily egg consumption in hyperlipidemic adults-Effects on endothelial function and cardiovascular risk. Nutr J 2010;9:28.

194. Shin JY, Xun P, Nakamura Y, He K. Egg consumption in relation to risk of cardiovascular

disease and diabetes: a systematic review and meta-analysis. Am J Clin Nutr 2013;98:146–59.

195. Zheng J, Huang T, Yu Y, et al. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. Public Health Nutr 2012;15:725–37.

196. Mente A, de Koning L, Shannon HS, Anand SS. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. Arch Intern Med 2009;169:659–69.

197. Mozaffarian D, Wu JHY. Omega-3 fatty acids and cardiovascular disease: Effects on risk factors, molecular pathways, and clinical events. J Am Coll Cardiol 2011;58:2047–67.

198. Mozaffarian D, Rimm EB. Fish intake, contaminants, and human health: evaluating the risks and the benefits. JAMA 2006;296:1885–99.

199. Yuan J-M, Ross RK, Gao Y-T, Mimi CY. Fish and shellfish consumption in relation to death from myocardial infarction among men in Shanghai, China. Am J Epidemiol 2001;154:809–16.

200. Ness AR, Gallacher JEJ, Bennett PD, et al. Advice to eat fish and mood: a randomised controlled trial in men with angina. Nutr Neurosci 2003;6:63–5.

201. Burr ML. Secondary prevention of CHD in UK men: the Diet and Reinfarction Trial and its sequel. Proceedings of the Nutrition Society 2007;66:9–15.

202. Ness AR, Hughes J, Elwood PC, et al. The long-term effect of dietary advice in men with coronary disease: follow-up of the Diet and Reinfarction trial (DART). Eur J Clin Nutr 2002;56:512–8.

203. Rizos EC, Ntzani EE, Bika E, et al. Association between omega-3 fatty acid supplementation and risk of major cardiovascular disease events: a systematic review and meta-analysis. JAMA 2012;308:1024–33.

204. Wallin A, Di Giuseppe D, Orsini N, et al. Fish consumption, dietary long-chain n-3 fatty acids, and risk of type 2 diabetes: systematic review and meta-analysis of prospective studies. Diabetes Care 2012;35:918–29.

205. Wu JH, Micha R, Imamura F, et al. Omega-3 fatty acids and incident type 2 diabetes: a systematic review and meta-analysis. Br J Nutr 2012;107 Suppl 2:S214–27.

206. Yamagishi K, Iso H, Date C, et al. Fish, ω-3 polyunsaturated fatty acids, and mortality from cardiovascular diseases in a nationwide community-based cohort of Japanese men and women: The JACC (Japan Collaborative Cohort Study for Evaluation of Cancer Risk) Study. J Am Coll Cardiol 2008;52:988–96.

207. Iso H, Kobayashi M, Ishihara J, et al. Intake of fish and n3 fatty acids and risk of coronary heart disease among Japanese: the Japan Public Health Center-Based (JPHC) Study Cohort I. Circulation 2006;113:195–202.

208. Nakamura Y, Ueshima H, Okamura T, et al. Association between fish consumption and all-cause and cause-specific mortality in Japan: NIPPON DATA80, 1980–99. Am J Med 2005;118:239–45.

209. Kris-Etherton PM, Hu FB, Ros E, Sabaté J. The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. J Nutr 2008;138:1746S–51S.

210. Sabaté J, Oda K, Ros E. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. Arch Intern Med 2010;170:821–7.

211. Banel DK, Hu FB. Effects of walnut consumption on blood lipids and other cardiovascular risk factors: a meta-analysis and systematic review. Am J Clin Nutr 2009;90:56–63.

212. Kris-Etherton PM, Zhao G, Binkoski AE, et al. The effects of nuts on coronary heart disease risk. Nutr Rev 2001;59:103–11.

213. Zhou D, Yu H, He F, et al. Nut consumption in relation to cardiovascular disease risk and type 2 diabetes: a systematic review and meta-analysis of prospective studies. Am J Clin Nutr 2014;100:270–7.

214. Guo K, Zhou Z, Jiang Y, Li W, Li Y. Meta-analysis of prospective studies on the effects of nut consumption on hypertension and type 2 diabetes mellitus. J Diabetes 2015;7:202–12.

215. Flores-Mateo G, Rojas-Rueda D, Basora J, et al. Nut intake and adiposity: meta-analysis of clinical trials. Am J Clin Nutr 2013;97:1346–55.

216. Smith JD, Hou T, Ludwig DS, et al. Changes in intake of protein foods, carbohydrate amount and quality, and long-term weight change: results from 3 prospective cohorts. Am J Clin Nutr 2015;101:1216–24.

217. 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.

218. Dilis V, Katsoulis M, Lagiou P, et al. Mediterranean diet and CHD: the Greek European prospective investigation into cancer and nutrition cohort. Br J Nutr 2012;108:699–709.

219. Mitrou PN, Kipnis V, Thiébaut ACM, et al. Mediterranean dietary pattern and prediction of all-cause mortality in a US population: results from the NIH-AARP Diet and Health Study. Arch Intern Med 2007;167:2461–8.

220. Buckland G, González CA, Agudo A, et al. Adherence to the Mediterranean diet and risk of coronary heart disease in the Spanish EPIC Cohort Study. Am J Epidemiol 2009;170:1518–29.

221. de Lorgeril M, Salen P, Martin J-L, 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;99:779–85.

222. Barzi F, Woodward M, Marfisi RM, Tavazzi L, Valagussa F, Marchioli R. Mediterranean diet and all-causes mortality after myocardial infarction: results from the GISSI-Prevenzione trial. Eur J Clin Nutr 2003;57:604–11.

223. Singh RB, Dubnov G, Niaz MA, et al. Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study): a randomised single-blind trial. Lancet 2002;360:1455–61.

224. Venn BJ, Perry T, Green TJ, et al. The effect of increasing consumption of pulses and wholegrains in obese people: a randomized controlled trial. J Am Coll Nutr 2010;29:365–72.

225. Hermsdorff HHM, MÁ Zulet, Abete I, Martínez JA. A legume-based hypocaloric diet reduces proinflammatory status and improves metabolic features in overweight/obese subjects. Eur J Nutr 2011;50:61–9.

226. Jenkins DJA, Kendall CWC, Augustin LSA, et al. Effect of legumes as part of a low glycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus: a randomized controlled trial. Arch Internal Med 2012;172:1653–60.

227. Mollard RC, Luhovyy BL, Panahi S, et al. Regular consumption of pulses for 8 weeks reduces metabolic syndrome risk factors in overweight and obese adults. Br J Nutr 2012;108 Suppl 1 :S111–22.

228. Liu J, Mazzone PJ, Cata JP, et al. Serum free fatty acid biomarkers of lung cancer. Chest 2014;146:670–9.

229. Anderson JW, Major AW. Pulses and lipaemia, short-and long-term effect: potential in the prevention of cardiovascular disease. Br J Nutr 2002;88:263–71.

230. Bazzano LA, Thompson AM, Tees MT, et al. Non-soy legume consumption lowers cholesterol levels: a meta-analysis of randomized controlled trials. Nutr Metab Cardiovasc Dis 2011;21:94–103.

231. Jayalath VH, de Souza RJ, Sievenpiper JL, et al. Effect of dietary pulses on blood pressure: a systematic review and meta-analysis of controlled feeding trials. Am J Hypertens 2013;27:56–64.

232. Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. Am J Clin Nutr 2014;100:278–88.

233. Shi ZQ, Tang JJ, Wu H, Xie CY, He ZZ. Consumption of nuts and legumes and risk of stroke: a meta-analysis of prospective cohort studies. Nutr Metab Cardiovas Dis 2014;24:1262–71.

234. Bernstein AM, Pan A, Rexrode KM, et al. Dietary protein sources and the risk of stroke in men and women. Stroke 2012;43:637–44.

235. Ho SC, Woo JLF, Leung SSF, et al. Intake of soy products is associated with better plasma lipid profiles in the Hong Kong Chinese population. J Nutr 2000;130:2590–3.

236. Agrawal S, Ebrahim S. Association between legume intake and self-reported diabetes among adult men and women in India. BMC Public Health 2013;13:706–19.

237. Villegas R, Gao Y-T, Yang G, et al. Legume and soy food intake and the incidence of type 2 diabetes in the Shanghai Women's Health Study. Am J Clin Nutr 2008;87:162–7.

238. Zhang X, Shu XO, Gao Y-T, et al. Soy food consumption is associated with lower risk of coronary heart disease in Chinese women. J Nutr 2003;133:2874–8.

239. Worm B, Barbier EB, Beaumont N, et al. Impacts of biodiversity loss on ocean ecosystem services. Science 2006;314:787–90.

240. Jenkins DJA, Sievenpiper JL, Pauly D, et al. Are dietary recommendations for the use of fish oils sustainable? CMAJ 2009;180:633–7.

241. Hawkes C, Popkin BM. Can the sustainable development goals reduce the burden of nutrition-related non-communicable diseases without truly addressing major food system reforms? BMC Med 2015;13:143–5.

242. 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.

243. Esposito K, Chiodini P, Maiorino MI, et al. Which diet for prevention of type 2 diabetes? A meta-analysis of prospective studies. Endocrine 2014;47:107–16.

244. O'Donnell MJ, Xavier D, Liu L, et al. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. Lancet 2010;376:112–23.

245. Hu D, Huang J, Wang Y, Zhang D, Qu Y. Fruits and vegetables consumption and risk of stroke a meta-analysis of prospective cohort studies. Stroke 2014;45:1613–9.

246. Wang X, Ouyang Y, Liu J, et al. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. Br Med J 2014;349:g4490.

247. Nerbass FB, Pecoits-Filho R, McIntyre NJ, et al. Demographic associations of high estimated sodium intake and frequency of consumption of high-sodium foods in people with chronic kidney disease stage 3 in England. J Renal Nutr 2014;24:236–42.

248. Krebs-Smith SM, Guenther PM, et al. Americans do not meet federal dietary recommendations. J Nutr 2010;140:1832–8.

249. Kotchen TA, Cowley AW Jr., Frohlich ED. Salt in health and disease–a delicate balance. N Engl J Med 2013;368:2531–2.

250. Powles J, Fahimi S, Micha R, et al. Global, regional and national sodium intakes in 1990 and 2010: a systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide. BMJ Open 2013;3:e003733.

251. Mente A, O'Donnell MJ, Rangarajan S, et al. Association of urinary sodium and potassium excretion with blood pressure. N Engl J Med 2014;371:601–11.

252. Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. Cochrane Database Syst Rev 2011:CD004022.

253. He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. Cochrane Database Syst Rev 2013;4:CD004937.

254. 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.

255. TOHP Investigators. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. The Trials of Hypertension Prevention Collaborative Research Group. Arch Intern Med 1997;157:657–67.

256. Bibbins-Domingo K, Chertow GM, Coxson PG, et al. Projected effect of dietary salt reductions on future cardiovascular disease. N Engl J Med 2010;362:590–9.

257. Coxson PG, Cook NR, Joffres M, et al. Mortality benefits from US population-wide reduction in sodium consumption: projections from 3 modeling approaches. Hypertension 2013;61:564–70.

258. Aburto NJ, Ziolkovska A, Hooper L, et al. Effect of lower sodium intake on health: systematic review and meta-analyses. BMJ 2013;346:f1326.

259. Ekinci EI, Clarke S, Thomas MC, et al. Dietary salt intake and mortality in patients with type 2 diabetes. Diabetes Care 2011;34:703–9.

260. O'Donnell MJ, Yusuf S, Mente A, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. JAMA 2011;306:2229–38.

261. Stolarz-Skrzypek K, Kuznetsova T, Thijs L, et al. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. JAMA 2011;305:1777–85.

262. Thomas MC, Moran J, Forsblom C, et al. The association between dietary sodium intake, ESRD, and all-cause mortality in patients with type 1 diabetes. Diabetes Care 2011;34:861–6.

263. O'Donnell M, Mente A, Rangarajan S, et al. Urinary sodium and potassium excretion, mortality, and cardiovascular events. N Engl J Med 2014;371:612–23.

264. Pfister R, Michels G, Sharp SJ, et al. Estimated urinary sodium excretion and risk of heart failure in men and women in the EPIC-Norfolk study. Eur J Heart Fail 2014;16:394–402.

265. Saulnier PJ, Gand E, Hadjadj S, Group SS. Sodium and cardiovascular disease. N Engl J Med 2014;371:2135–6.

266. Graudal N, Jurgens G, Baslund B, Alderman MH. Compared with usual sodium intake, low- and excessive-sodium diets are associated with increased mortality: a meta-analysis. Am J Hypertens 2014;27:1129–37.

267. Cobb LK, Anderson CA, Elliott P, et al. Methodological issues in cohort studies that relate sodium intake to cardiovascular disease outcomes: a science advisory from the American Heart Association. Circulation 2014;129:1173–86.

268. Mitka M. IOM report: Evidence fails to support guidelines for dietary salt reduction. JAMA 2013;309:2535–6.

269. O'Donnell M, Mente A, Yusuf S. Sodium intake and cardiovascular health. Circ Res 2015;116:1046–57.

270. Oparil S. Low sodium intake–cardiovascular health benefit or risk? N Engl J Med 2014;371:677–9.

271. World Health Organization. Global Status Report on Alcohol and Health 2014. Geneva, Switzerland: World Health Organization, 2014.

272. Smyth A, Teo K, Rangarajan S, et al., on behalf of the PURE Investigators. Alcohol consumption and cardiovascular disease, cancer, injury, admission to hospital, and mortality. Lancet 2015. In press.

273. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. BMJ 2011;342:d636.

274. Ferrari P, Licaj I, Muller DC, et al. Lifetime alcohol use and overall and cause-specific mortality in the European Prospective Investigation into Cancer and nutrition (EPIC) study. BMJ Open 2014;4:e005245.

275. Zhang C, Qin YY, Chen Q, et al. Alcohol intake and risk of stroke: a dose-response meta-analysis of prospective studies. Int J Cardiol 2014;174:669–77.

276. Roerecke M, Rehm J. Irregular heavy drinking occasions and risk of ischemic heart disease: a systematic review and meta-analysis. Am J Epidemiol 2010;171:633–44.

277. U.S. Department of Health and Human Services and the U.S. Department of Agriculture. Scientific Report of the 2015 Dietary Guidelines Advisory Committee. Washington, DC: Office of Disease Prevention and Health Promotion, 2015.

278. Appel LJ, Sacks FM, Carey VJ, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. JAMA 2005;294:2455–64.

279. Reedy J, Krebs-Smith SM, Miller PE, et al. Higher diet quality is associated with decreased risk of all-cause, cardiovascular disease, and cancer mortality among older adults. J Nutr 2014;144:881–9.

280. Struijk EA, May AM, Wezenbeek NL, et al. Adherence to dietary guidelines and cardiovascular disease risk in the EPIC-NL cohort. Int J Cardiol 2014;176:354–9.

281. Jenkins DJ, Kendall CW, Marchie A, et al. Effects of a dietary portfolio of cholesterol-lowering foods vs lovastatin on serum lipids and C-reactive protein. JAMA 2003;290:502–10.

282. Jenkins DJ, Jones PJ, Lamarche B, et al. Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. JAMA 2011;306:831–9.

283. Jenkins DJ, Kendall CW, Faulkner DA, et al. Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. Am J Clin Nutr 2006;83:582–91.

284. Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA 2006;295:655–66.

285. Look ARG, Wing RR, Bolin P, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med 2013;369:145–54.

286. Hawkes C. The influence of trade liberalization on global dietary change: the case of vegetable oils, meat and highly processed foods. In: Hawkes C, Blouin C, Henson S, Drager N, Dubé L, editors. Trade, Food, Diet and Health: Perspectives and Policy Options. New York, NY: Wiley-Blackwell, 2010:36–59.

287. Davis M. The Monster at Our Door: The Global Threat of Avian Flu. New York, NY: Holt Paperbacks, 2006.

288. Babu SC, Blom S. Building Resilience for Food and Nutrition Security. 2020 Conference Brief 6. Washington DC: International Food Policy Research Institute, 2014.

289. Hawkes C, Thow A, Downs S, et al. Identifying effective food systems solutions for nutrition and noncommunicable diseases: creating policy coherence in the fats supply chain. SCN News 2013;40:39–47.

290. Winne M. Closing the Food Gap: Resetting the Table in the Land of Plenty. New York, NY: Beacon Press, 2009.

291. Chavasit V, Kasemsup V, Tontisirin K. Thailand conquered under-nutrition very successfully but has not slowed obesity. Obes Rev 2013; 14:96–105.

292. Coitinho D, Monteiro C, Popkin B. What Brazil is doing to promote healthy diets and active lifestyles. Public Health Nutr 2002;5:263–7.

293. Unit for Policy Coherence for Development. Policy Framework for Policy Coherence for Development. Working Paper No 1. Paris: Organization for Economic Cooperation and Development, 2012.

294. Blouin C. Trade policy and health: from conflicting interests to policy coherence. Bull World Health Org 2007;85:169–73.

295. Blouin C, Chopra M, Hoeven R. Trade and health 3: trade and the social determinants of health. Lancet 2009;373:502–7.

296. Nilsson M, Zamparutti T, Petersen JE, Nykvist B, Rudberg P, McGuinn J. Understanding policy coherence: Analytical framework and examples of sector-environmental policy interactions in the EU. Environ Policy Govern 2012;22:395–423.

297. Igumbor EU, Sanders D, Puoane TR, et al. "Big Food," the consumer food environment, health, and the policy response in South Africa. PLoS Med 2012;9:e1001253.

298. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet 2011;378:804–14.

299. Thow AM, Hawkes C. Global sugar guidelines: an opportunity to strengthen nutrition policy. Public Health Nutr 2014;17:2151–5.

300. Burgan M, Winne M. Doing Food Policy Councils Eight: A Guide to Development and Action. Santa Fe, NM: Mark Winne Associates, 2012.

301. Godfray HCJ, Crute IR, Haddad L, et al. The future of the global food system. Philos Trans R Soc Lond B Biol Sci 2010;365:2769–77.

302. Popkin B, Monteiro C, Swinburn B. Overview: Bellagio conference on program and policy options for preventing obesity in the low- and middle-income countries. Obes Rev 2013;14 Suppl 2:1–8.

303. Roodenburg A, Popkin B, Seidell J. Development of international criteria for a front of package nutrient profiling system: international Choices Programme. Eur J Clin Nutr 2011;65: 1190–200.

304. Corvalán C, Reyes M, Garmendia ML, Uauy R. Structural responses to the obesity and noncommunicable diseases epidemic: The Chilean law of food labeling and advertising. Obes Rev 2013;14:79–87.

305. Hawkes C, Smith TG, Jewell J, et al. Smart food policies for obesity prevention. Lancet 2015; 385:2410–21.

306. Vyth EL, Steenhuis IH, Roodenburg AJ, et al. Front-of-pack nutrition label stimulates healthier product development: a quantitative analysis. Int J Behav Nutr Phys Act 2010;7:65.

307. Godfray H, Crute IR, Haddad L, et al. The future of the global food system. Phil Trans R Soc B: Bio Sci 2010;365:2769–77.

KEY WORDS **cardiovascular disease, climate change, diet, food consumption, food system, low- and middle-income countries**

APPENDIX **For expanded supporting information, a supplemental table, and additional references, please see the online version of this article.**

Supplementary Box 1: What do we mean by 'the food system'?

The "food system" can be defined as the activities, infrastructure, and people involved in feeding our global population (e.g., the growing, processing, distribution, consumption, and disposal of foods) (1). It includes the web of processes by which institutions, organizations, and individuals transform inputs (e.g., seed, fertilizer, chemicals, pharmaceuticals) into foods and ingredients (e.g., poultry) into the food we consume (e.g., chicken nuggets) (2). All food systems interact with their environmental, social, political and economic context (3). A core element of all food systems is the "food supply chain" or "food value chain" through which foods moves from farm to fork (4).

Around the globe there is a diversity of types of food systems. They can be very broadly characterized into those dominated by "short chains" – in which food is produced largely for local markets or household production and is typically consumed after relatively little transformation (e.g., processing); and those dominated by "long chains," with many steps between production and consumption beyond the farm gate. Greater complexity is most evident for processed, manufactured foods with numerous ingredients, and greater length is most evident for widely traded commodities like wheat and oilseeds (5).

Most high-income industrialized countries and an increasing number of LMICLMIC are now dominated by "long chain" industrialized food systems (often termed "conventional food systems"), in which agricultural production, food processing, distribution, and retailing are is geared towards maximizing efficiency to reduce costs and increase overall production .
Figure 1 offers a schematic view of this system, which begins with agriculture and aquaculture production, using expanded external inputs to farms and a research system geared towards raising yields. The main external influence on this system was once government policies, but

corporations involved in various sectors (e.g., machinery, fertilizers, seeds, chemicals, animal pharmaceuticals, food processers, manufacturers, retailers and caterers) have now become significantly more influential.

upplementary Box 2: Success in Reducing of *Trans*-fats

Actions to restrict industrial *trans*-fats in many countries are a clear example of success in shifting the food system away from the production, use and consumption of an unhealthy food ingredient.

Crisco, invented in the US a century ago, was the first commercially successful hydrogenated oil product (6-8). From the 1950s onwards, with the development of cheap oil production and more sophisticated hydrogenation technology, and spurred by public and private research that developed cheap ways of extracting oil from oilseeds, industrial use of *trans*-fats in margarine, baked goods and many other products exploded (9,10).

Prior to the 1980's, hydrogenated cooking fats and margarines were viewed as healthful while butter with its high saturated fat content was demonized. However, understanding emerged of the health dangers of hydrogenated oils, and nutrition scientists have had an emphatic role convincing governments and industry to remove transfats from ultra-processed food products in many high-income countries (11-15). Only in the past 15 years did governments begin to implement standards for information to be provided to consumers through labeling, as well as imposing restrictions on permissible levels of transfats in the food supply (16,17). A major combination of advocacy and scholarship created the policy changes that led to the food system alternatives, spurring agroindustry to invest in research into the

technology to provide alternatives while food manufacturer reformulated products.

Supplementary Table 1. Mediterranean Style Diet adapted to various regions of the world

Mediterranean style dietary pattern component	**Region-specific food choices**						
	East Asia	**South Asia**	**Middle East**	**Africa**	**South America**	**North America**	**Europe**
Fruits	Any type	Any type	Any type	Any type	Any type	Any type	Any type
Vegetables (non-starchy)[a]	Chard, kale, bok choi, pak choi,	Beetroots, spinach, cabbage, green leafy vegetables	Lettuce, spinach, carrots	Beetroot leaf, spinach, pumpkin leaf	Beetroots, spinach	Lettuce, kale, other dark leafy vegetables	Spinach, lettuce, cabbage, beets, chard
Legumes (beans, lentils)	Any type	Any type	Any type	Any type	Any type	Any type	Any type
Whole grain cereals (brown rice, whole grain breads, maize, whole-wheat pasta)	Sorghum, millet	Chapathi, paratha, roti from whole grains	Whole grain bread	Oats, *sadza*[b] from sorghum, whole grain bread	Brown rice, whole grain bread	Whole grain bread (e.g. rye bread)	Whole grain bread (e.g. rye bread)
Fatty fish[c]	Any type	Any type (e.g.	Any type (e.g.	Any type (e.g.	Any type (e.g.	Any type (e.g.	Any type (e.g.

	(e.g. tuna, sardine, mackerel)	sardines, queen fish, mackerel)	tuna, trout)	tuna)	tuna, salmon, sardine, trout)	salmon, tuna, trout, herring sardines, mackerel)	salmon, tuna, trout, herring, mackerel)
Reduce meats[d]*, increase meat alternatives*	Meats: Poultry, lean beef, pork without fat Alternatives: Tofu, legumes (e.g. mung beans, adzuki beans, broad beans) Seitan	Vegetarians should emphasize legumes and nuts	Meats: Any type of poultry, lamb or beef without fat Alternatives: Foul (fava beans), chickpeas (falafel), lentils, and pulses of all kinds	Meats: Poultry, lean beef, pork without fat Alternatives: legumes in general and nuts Soy beans, peanuts	Meats: Poultry, lean beef, pork without fat Alternatives: Burritos, kidney, Romano beans, and all dried pulses	Meats: Poultry, lean beef, pork without fat Alternatives: legumes in general and nuts Soy beans, peanuts	Meats: Poultry, lean beef, pork without fat Alternatives: legume (baked beans on toast) Bean stews Tofu and foods from other cultures
Low-fat dairy[e]	Plain milk	Plain milk, yoghurt	Plain milk, yoghurt	Plain milk, yoghurt	Plain milk, low-fat yoghurt	Plain milk, low-fat yoghurt	Plain milk, low-fat

							yoghurt
Cooking oil[f]	Grape seed oil, soybean oil	Sunflower oil	Mixed vegetable oils, olive oil	Sunflower oil	Canola oil, olive oil	Canola oil, olive oil	Soybean oil, sunflower oil
Nuts	Any type	Any type	Any type	Any type	Any type	Any type	Any type

For healthy eating, consumption of the above listed foods can be advised. Please note that this is not meant to be completely inclusive as many other locally available foods may be included.

[a]Following WHO classification starchy tubers such as potatoes are not included in the vegetable group

[b]Sadza is a staple food in Zimbabwe

[c]Fatty fish are good source of omega-3 fatty acids. Those populations which do not consume fish may obtain omega-3 fatty acids from plant sources, such as flaxseed, soybean, rapeseed, or walnuts.

[d]Meat is not an essential component of the healthy dietary pattern; population which consume meat are advised to do so sparingly, especially red meats; generally, plant-based protein sources (e.g. legumes, nuts, soy) are preferred over animal sources

[e]Those populations which do not consume dairy may obtain protein from other sources, such as nuts, legumes, whole grains, etc.; and calcium from fortified tofu, almonds, or kale.

[f]It is advised to avoid clarified butter, lard or tallow for food preparation.

References

1. Center for a Livable Future. The Food System, Environment & Public Health. 2014.
2. Babu SC, Blom S. Building resilience for food and nutrition security. 2020 Conference Brief 6. Washington DC: International Food Policy Research Institute, 2014:1-4.
3. Ericksen P, Stewart B, Dixon J et al. The value of a food system approach. In: Ingram J, Ericksen P, Liverman D, editors. Food Security and Global Environmental Change. London: Earthscan, 2014:25-45.
4. Hawkes C. Identifying inovative interventions to promote healthy eating using consumption-oriented food supply chain analysis. J Hunger Environ Nutr 2009;4:336-356.
5. Hawkes C, Thow A, Downs S et al. Identifying effective food systems solutions for nutrition and noncommunicable diseases: creating policy coherence in the fats supply chain. SCN News 2013;40:39-47.
6. Instituto Nacional de Estadística y Geografía. Indices de Precios. 2014.
7. Organization for Economic Co-opration and Development. Health at a Glance 2011: OEDC indicators. http://dxdoiorg/101787/health_glance-2011-en, 2011.
8. Ng S, Popkin B. Time use and physical activity: a shift away from movement across the globe. Obes Rev 2012;13:659-680.
9. Williams GW. Development and future direction of the world soybean market. Q J Int Agr 1984;23:319-337.
10. Popkin B, Drewnowski A. Dietary fats and the nutrition transition: New trends in the global diet. Nutr Rev 1997;55:31-43.

11. Willett WC, Stampfer MJ, Manson JE et al. Intake of trans fatty acids and risk of coronary heart disease among women. Lancet 1993;341:581-5.

12. Willett WC, Ascherio A. Trans fatty acids: are the effects only marginal? Am J Public Health 1994;84:722-4.

13. Katan MB. Regulation of trans fats: the gap, the Polder, and McDonald's French fries. Atheroscler Suppl 2006;7:63-6.

14. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans fatty acids and cardiovascular disease. New Eng J Med 2006;354:1601-13.

15. Walker RE, Keane CR, Burke JG. Disparities and access to healthy food in the United States: A review of food deserts literature. Health Place 2010;16:876-884.

16. Hawkes C, Jewell J, Allen K. A food policy package for healthy diets and the prevention of obesity and diet-related non-communicable diseases: The NOURISHING framework. Obes Rev 2013;14:159-168.

17. World Cancer Reserach Fund International. WCRF International Food Policy Framework for Healthy Diets: NOURISHING. London: World Cancer Reserach Fund International, 2014.