REVIEW ARTICLE

GLOBAL HEALTH

Behavioral and Dietary Risk Factors for Noncommunicable Diseases

Majid Ezzati, Ph.D., and Elio Riboli, M.D.

From the School of Public Health, Imperial College London, London. Address reprint requests to Dr. Ezzati or Dr. Riboli at the School of Public Health, Imperial College London, Norfolk Pl., London W2 1PG, United Kingdom, or at majid.ezzati@imperial.ac.uk or e.riboli@imperial.ac.uk.

EXCEPT IN EASTERN EUROPE AND PARTS OF AFRICA, MORTALITY AMONG adults has declined in most countries for decades.[1] Lower rates of death from infectious diseases were the early driver of this improvement, but there have been subsequent declines in mortality from cardiovascular disease and some cancers.[2,3] There have also been important trends in various cancers[2] — for example, the rise and subsequent decline in lung-cancer incidence and mortality among men in many high-income countries, a decline in stomach-cancer incidence and mortality as economies develop, and the worldwide increase in breast-cancer incidence.
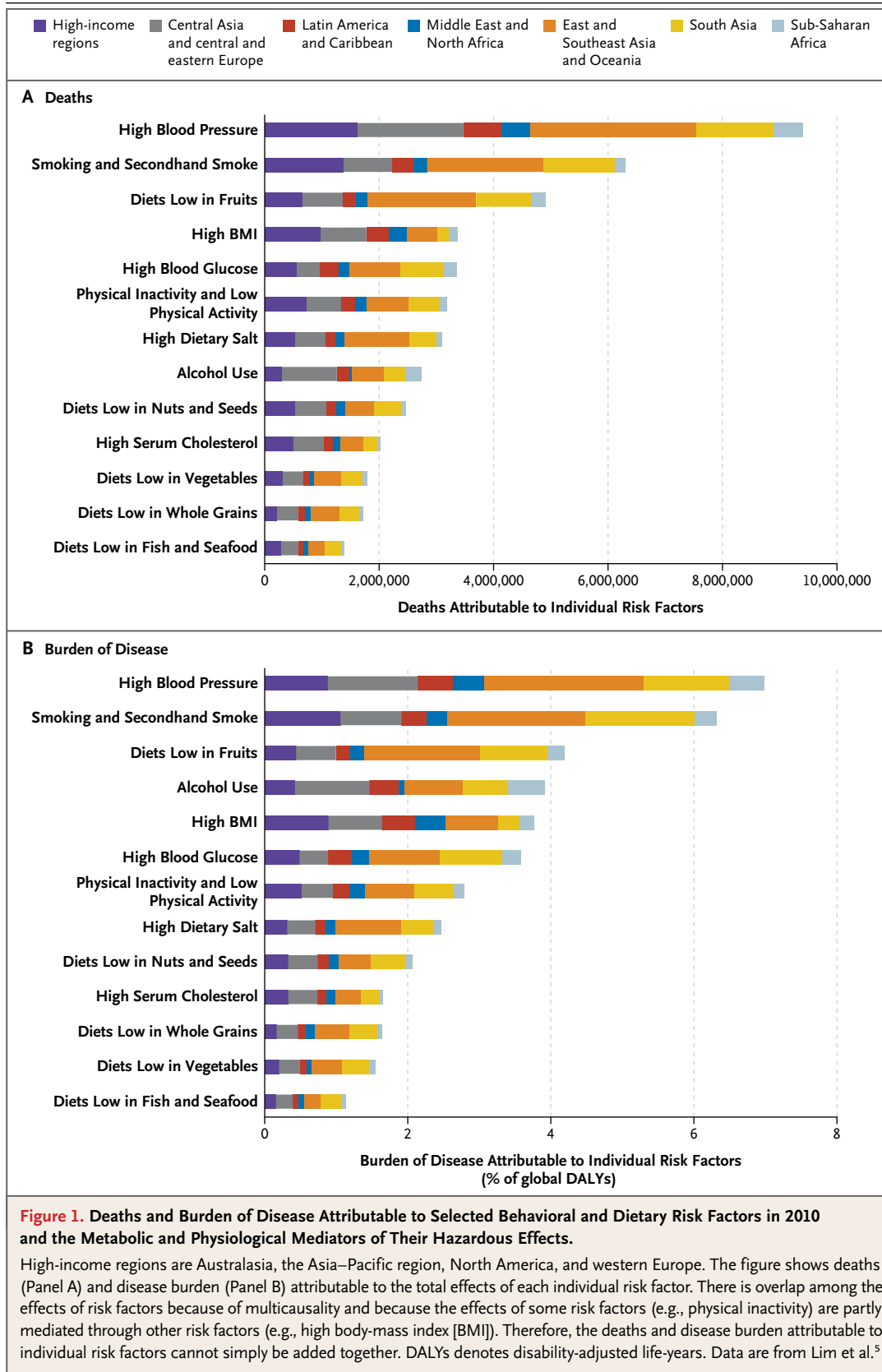
The hazardous effects of behavioral and dietary risk factors on noncommunicable diseases, and the metabolic and physiological conditions that mediate their effects, have been established in prospective cohort studies and randomized trials. This knowledge, together with data from risk-factor surveillance, has helped to establish the mortality and disease burden attributable to risk factors, globally and by region and country.[4-7] There is less information on risk-factor trends, which makes it difficult to assess how they have affected population health in the past or how they may do so in the future.

In this article, we summarize the available data on trends in selected behavioral and dietary risk factors for noncommunicable diseases and examine the effects they have had, or may have in the future, on the health of populations around the world. Risk factors such as smoking, alcohol consumption, excess weight, and dietary factors are responsible for a large share of the global disease burden, directly or through conditions such as high blood pressure and elevated blood glucose and cholesterol levels (Fig. 1).[4,5]

SMOKING

The hazardous effects of smoking on mortality from cancers and cardiovascular and respiratory diseases have been known for decades. Effects on other globally important diseases such as diabetes[8] and tuberculosis[9] have also been shown. In parallel, evidence of the hazards of smoking in Asian countries has established that it is a global problem.[10-12] Moreover, exposure of pregnant women, children, and nonpregnant adults to secondhand smoke at home and in public places is associated with adverse birth outcomes, childhood respiratory diseases, and many of the same diseases that are associated with active smoking.[13]

In most Western high-income countries, the prevalence of smoking increased among men in the first half of the 20th century and peaked in the post–World War II decades, with 80% of men having smoked at some point in their adult lives. Smoking among men subsequently declined in English-speaking countries and in northern Europe, followed by more than two decades of decline in age-standardized mortality from lung cancer (Fig. 2A) and in deaths from other diseases attributable
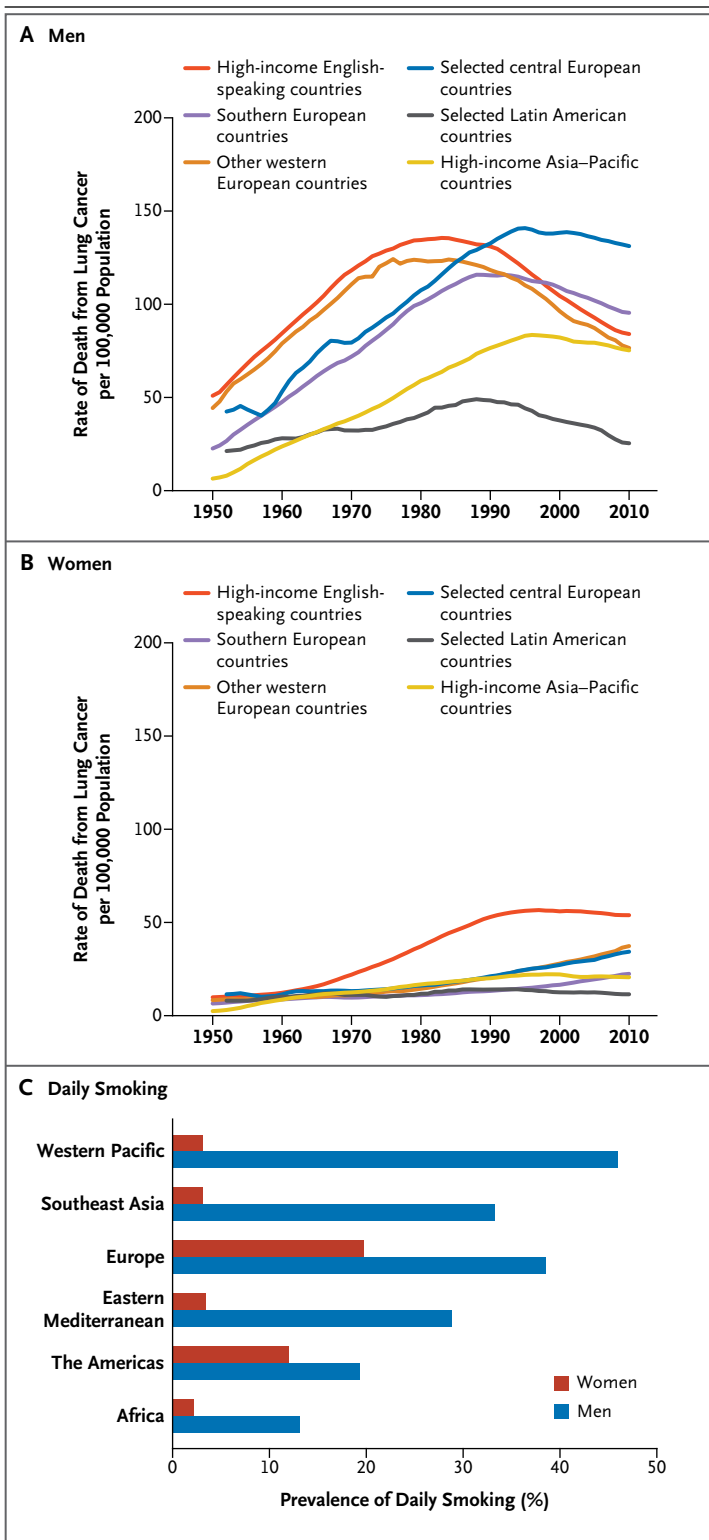
A Deaths

Legend: High-income regions | Central Asia and central and eastern Europe | Latin America and Caribbean | Middle East and North Africa | East and Southeast Asia and Oceania | South Asia | Sub-Saharan Africa

A Deaths

- High Blood Pressure
- Smoking and Secondhand Smoke
- Diets Low in Fruits
- High BMI
- High Blood Glucose
- Physical Inactivity and Low Physical Activity
- High Dietary Salt
- Alcohol Use
- Diets Low in Nuts and Seeds
- High Serum Cholesterol
- Diets Low in Vegetables
- Diets Low in Whole Grains
- Diets Low in Fish and Seafood

Deaths Attributable to Individual Risk Factors

B Burden of Disease

- High Blood Pressure
- Smoking and Secondhand Smoke
- Diets Low in Fruits
- Alcohol Use
- High BMI
- High Blood Glucose
- Physical Inactivity and Low Physical Activity
- High Dietary Salt
- Diets Low in Nuts and Seeds
- High Serum Cholesterol
- Diets Low in Whole Grains
- Diets Low in Vegetables
- Diets Low in Fish and Seafood

Burden of Disease Attributable to Individual Risk Factors
(% of global DALYs)

Figure 1. Deaths and Burden of Disease Attributable to Selected Behavioral and Dietary Risk Factors in 2010 and the Metabolic and Physiological Mediators of Their Hazardous Effects.

High-income regions are Australasia, the Asia–Pacific region, North America, and western Europe. The figure shows deaths (Panel A) and disease burden (Panel B) attributable to the total effects of each individual risk factor. There is overlap among the effects of risk factors because of multicausality and because the effects of some risk factors (e.g., physical inactivity) are partly mediated through other risk factors (e.g., high body-mass index [BMI]). Therefore, the deaths and disease burden attributable to individual risk factors cannot simply be added together. DALYs denotes disability-adjusted life-years. Data are from Lim et al.[5]

The New England Journal of Medicine

A Men

B Women

C Daily Smoking

Figure 2. Trends in Lung-Cancer Mortality and Prevalence of Daily Tobacco Smoking in 2008.

Panels A and B show trends in age-standardized mortality from lung cancer among men and women, respectively, 30 years of age or older. Death rates are age-standardized to the World Health Organization (WHO) standard population. Data are from the WHO database of vital statistics, with adjustment for completeness of death registration and for validity and comparability of cause-of-death assignment. Panel C shows the age-standardized prevalence of daily tobacco smoking among adults in 2008, according to WHO region. Data are from the WHO.[14]

and countries in Latin America and central and southern Europe. The more recent declines in the United Kingdom, North America, and Australia are beginning to translate into a plateau or decline in lung-cancer mortality (Fig. 2B) and in smoking-attributable deaths from other diseases among women.[15] However, lung-cancer mortality continues to rise among continental European women (Fig. 2B).

The majority of the more than 1 billion smokers worldwide now live in low- and middle-income countries. The prevalence of smoking has fallen below 20% in Australia and Canada but has plateaued at high levels among men and women in central and eastern Europe, among women in some western and southern European countries, and among men in East Asia (Fig. 2C).[14] An estimated 60% of men in some countries in eastern Europe and East Asia smoke. The prevalence of smoking among women is still highest in Western societies, with a prevalence of about 40% in some European countries.[14] The prevalence of smoking remains relatively low in sub-Saharan Africa (Fig. 2C), and smokers there tend to smoke fewer cigarettes than do their Western and Asian counterparts.

In addition to shifting patterns of smoking prevalence, there have been changes in the type of cigarettes available, including the introduction of "low-tar" and "light" cigarettes. A recent review concluded that "five decades of evolving cigarette design had not reduced overall disease risk among smokers."[16] Prevention and cessation remain the only effective public health measures to reduce the harmful effects of smoking.

Tobacco smoking and exposure to secondhand smoke together are responsible for about 6.3 million annual deaths worldwide and 6.3% of the global burden of disease, mostly in low- and middle-income countries (Fig. 1).[5] The death toll from smoking is especially large in eastern

to smoking.[15] The prevalence of smoking among women rose throughout the second half of the 20th century, first in English-speaking countries and northern European countries, then in Japan

Europe, where the prevalence of smoking and the prevalence of other cardiovascular risk factors are concurrently high; this death toll is increasing in the large populations of Asia and slowly declining in Western countries. In addition to smoking, oral tobacco use and betel-nut chewing are highly prevalent in South Asia and are responsible for a large number of cases of oral cancer and deaths from this disease.[17]

ALCOHOL CONSUMPTION

Alcohol consumption is associated with numerous diseases and injuries. Moderate alcohol consumption has been inversely associated with the risk of cardiovascular diseases and diabetes, although the benefits may be greater for persons with existing cardiovascular risk factors than for those without such risk factors.[18] Epidemiologic studies that have measured both the amount and patterns of alcohol consumption have shown that heavy episodic (or binge) drinking not only substantially raises the risk of injuries but can also increase the risk of or exacerbate cardiovascular disease and liver disease.[19-21]

Although cultural factors are important determinants of alcohol consumption, including harmful drinking, social change and policy interventions have modified alcohol-drinking behaviors in some countries.[22,23] For example, per capita alcohol consumption has decreased by about one half in traditional wine-producing and wine-drinking countries such as Italy and France during the past few decades[22,23]; during the same period, it has doubled in the United Kingdom and Denmark, and levels of consumption in the two groups of countries have converged (Fig. 3). Alcohol consumption has increased steadily in Japan, China, and many other countries in Asia, where it was previously low.
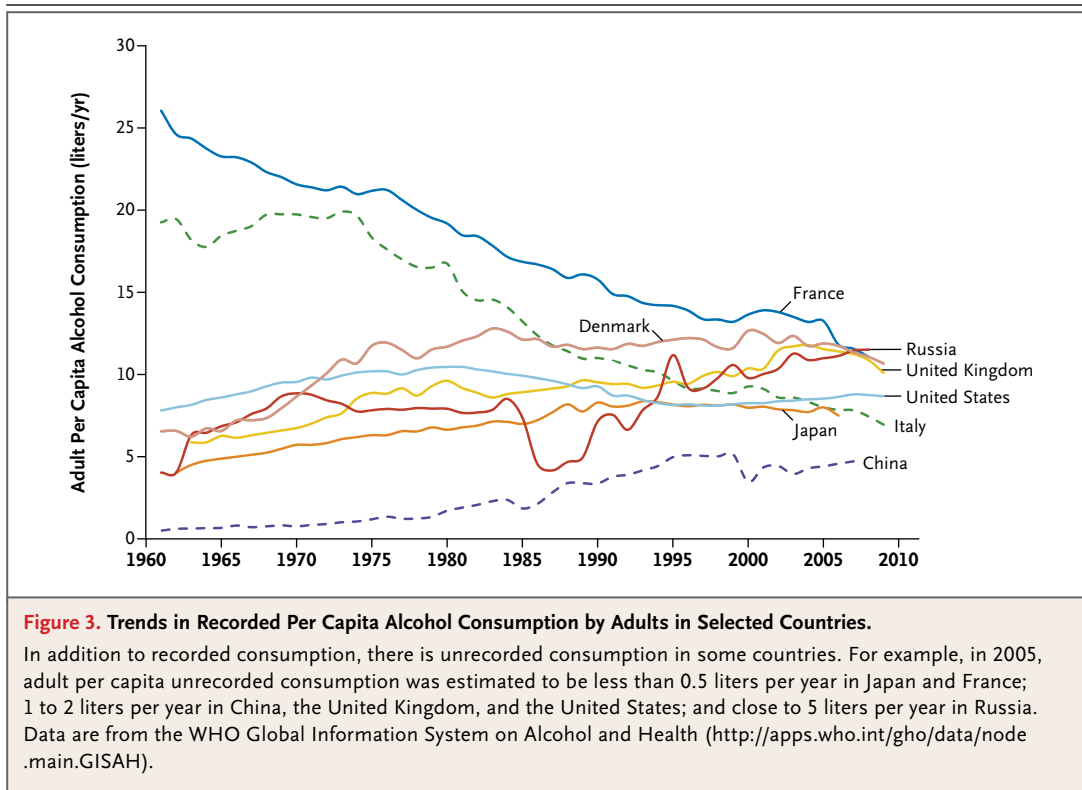
Alcohol consumption is responsible for about 2.7 million annual deaths and 3.9% of the global burden of disease (Fig. 1).[5] The major contributors to the alcohol-attributable disease burden are cancers, chronic liver disease, unintentional injuries, alcohol-related violence, neuropsychiatric conditions, and, in some regions (especially eastern Europe) that have a high prevalence of binge and harmful drinking, a large death toll from cardiovascular diseases.[4,5,24,25] The role of alcohol consumption in injuries and violence among young adults and in nonfatal neuropsychiatric conditions makes its contribution to the disease burden larger than its contribution to mortality, relative to other risk factors for noncommunicable diseases (Fig. 1).

Alcohol consumption is the leading single cause of the disease burden in eastern Europe and is one of the top three risk factors, along with high blood pressure and overweight or obesity, in much of Latin America, where it ranks ahead of smoking.[4,5,7] The effects of alcohol on population health are greatest in Russia and some other former Soviet republics. Though recorded per capita alcohol consumption in Russia is the same as or only slightly higher than consumption in western European countries, the health effects are substantially larger. In traditional wine-producing countries, most alcohol is consumed as wine during meals, in relatively modest daily amounts, by a large proportion of the population. In contrast, in Russia and neighboring countries, men (especially those of low socioeconomic status) consume very large amounts of spirits, either as a regular daily habit or by binge drinking. A substantial proportion of consumed alcohol is from unrecorded and nonbeverage sources such as medicinal and industrial ethanol. Alcohol consumption may be responsible for one third to one half of deaths among young and middle-aged men in Russia.[24,26] In contrast to the current, enormous death toll, mortality declined temporarily in the 1980s, when policies introduced under Mikhail Gorbachev reduced alcohol consumption by about one half (Fig. 3).[25]

EXCESS WEIGHT AND OBESITY

Numerous observational studies in Western and Asian populations have associated different measures of adiposity and excess body weight with increased total mortality and increased risks of disease or death from diabetes, ischemic heart disease and ischemic stroke, cancers, chronic kidney disease, and osteoarthritis.[27-32] The risks of diabetes and ischemic heart disease increase monotonically with an increase in the body-mass index (BMI, the weight in kilograms divided by the square of the height in meters), starting at a BMI in the low 20s. In contrast, the association with hemorrhagic stroke, which is more common in Asian populations than in other populations, has been observed only at a BMI of 25 or higher.[27,31] Currently, excess weight is responsible for about 3.4 million annual deaths and 3.8%
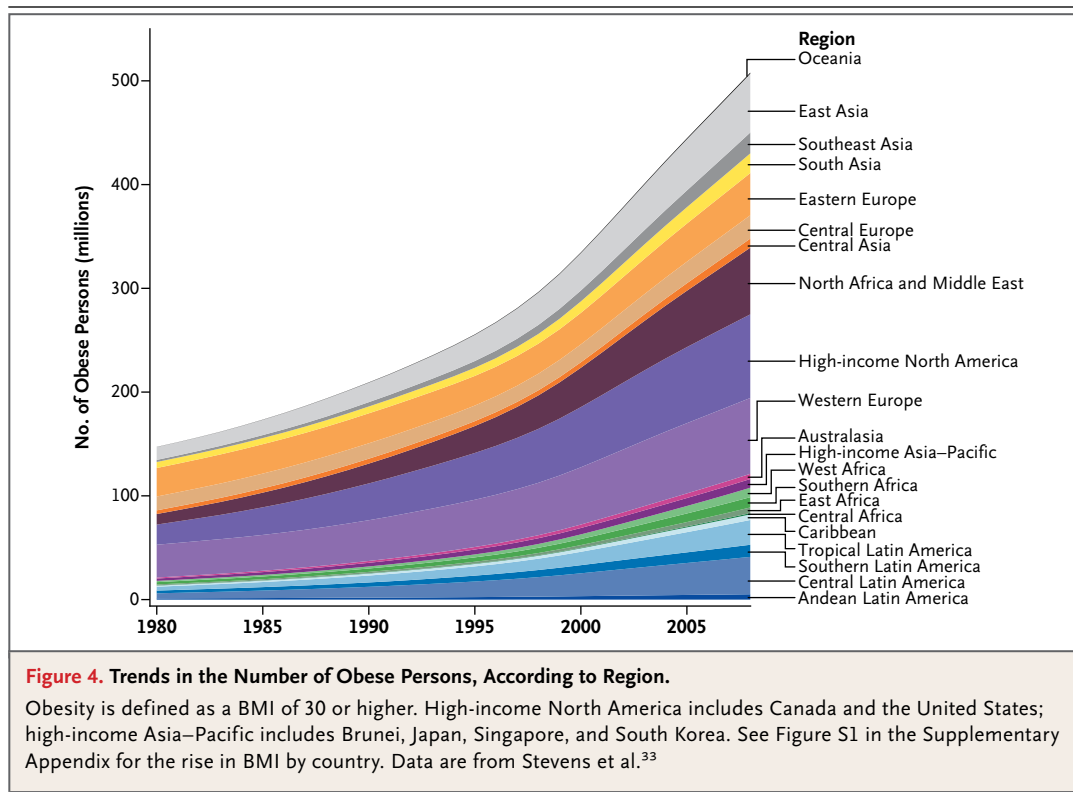
Figure 3. Trends in Recorded Per Capita Alcohol Consumption by Adults in Selected Countries.

In addition to recorded consumption, there is unrecorded consumption in some countries. For example, in 2005, adult per capita unrecorded consumption was estimated to be less than 0.5 liters per year in Japan and France; 1 to 2 liters per year in China, the United Kingdom, and the United States; and close to 5 liters per year in Russia. Data are from the WHO Global Information System on Alcohol and Health (http://apps.who.int/gho/data/node .main.GISAH).

of the global burden of disease, with diseases that have low mortality and long periods of disability, such as diabetes and musculoskeletal diseases, accounting for a proportion of this burden (Fig. 1).[5]

In recent decades, men and women in all but a few countries have gained weight, with the age-standardized mean BMI increasing by more than 2 units per decade in some Pacific islands (Fig. S1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). In high-income regions, the BMI is higher in English-speaking countries than in continental Europe and the Asia–Pacific region, especially for women. The global prevalence of obesity (defined as a BMI ≥30) doubled between 1980 and 2008, to 9.8% among men and 13.8% among women — equivalent to more than half a billion obese people worldwide (205 million men and 297 million women) (Fig. 4).[33,34] An additional 950 million adults have a BMI of 25 to less than 30. The United States has had the largest absolute increase in the number of obese people since 1980, followed by China, Brazil, and Mexico.[33] Currently, the age-standardized mean BMI ranges from

less than 22 in parts of sub-Saharan Africa and Asia to 30 to 35 in some Pacific islands and countries in the Middle East and North Africa.[34] The prevalence of obesity ranges from less than 2% in Bangladesh to more than 60% in some Pacific islands.[33]

DIET AND NUTRITION

Centuries after the effects of specific dietary intakes on conditions such as scurvy were discovered, nutritional epidemiology has established the associations of specific foods and nutrients or overall dietary patterns with cancers, cardiovascular diseases, and diabetes[35,36] and with intermediate outcomes such as weight gain, increased blood pressure, and insulin resistance and hyperglycemia.[37-39] The large body of observational studies is increasingly complemented by well-designed randomized trials that have, for example, shown the benefits of lower salt intake, the replacement of saturated fats with polyunsaturated fats, and healthy dietary patterns.[37,38,40,41] Low dietary intakes of fruits, vegetables, whole grains, or nuts and seeds or a high dietary intake of salt are

Figure 4. Trends in the Number of Obese Persons, According to Region.

Obesity is defined as a BMI of 30 or higher. High-income North America includes Canada and the United States; high-income Asia–Pacific includes Brunei, Japan, Singapore, and South Korea. See Figure S1 in the Supplementary Appendix for the rise in BMI by country. Data are from Stevens et al.[33]

individually responsible for 1.5% to more than 4% of the global disease burden (Fig. 1).[5]

There have so far been few population-based analyses of trends in specific dietary risk factors. Administrative data, such as the United Nations Food and Agriculture Organization (FAO) food balance sheets, provide a broad picture of dietary patterns and trends based on the availability of different food types for human consumption. FAO data show that consumption of animal fats and high-calorie foods is increasing in Mediterranean countries, such as Greece, but declining slightly in Nordic countries and New Zealand,[42] with consumption in these countries converging at similar levels (Fig. 5A). These changes may also partly explain trends in serum cholesterol levels, which have declined more rapidly in Nordic countries and New Zealand than in southern Europe, with cholesterol levels now lower in Sweden and Finland than in Italy.[45] Dietary change has been even more drastic in parts of Asia, with China rapidly adopting a Western, animal-based diet (Fig. 5A) and having one of the largest worldwide increases in serum cholesterol levels.[45]

Parallel to this westernization trend, fruits, vegetables, nuts, and cereals have become more available in Nordic and English-speaking Western countries and in Asia, partly because technological and economic developments have increased year-around availability through expanded production, imports, and storage capacity (Fig. 5B). Similarly, there has been a modest increase in the availability of fish and other marine products in some Western and Asian countries (Fig. 5C).

The FAO data, which are based on agricultural production and trade statistics, do not capture food waste or subsistence production, nor do they account for food processing. For example, these data do not include specific information about consumption of refined flour versus whole grains, sugar-sweetened beverages, and partially hydrogenated vegetables oils (and hence trans-fat consumption), all of which are important dietary risk factors. The FAO databases also do not record consumption of salt, which is common in the diets of countries at all stages of economic development.[46] A high intake of salt is a risk factor for stomach cancer
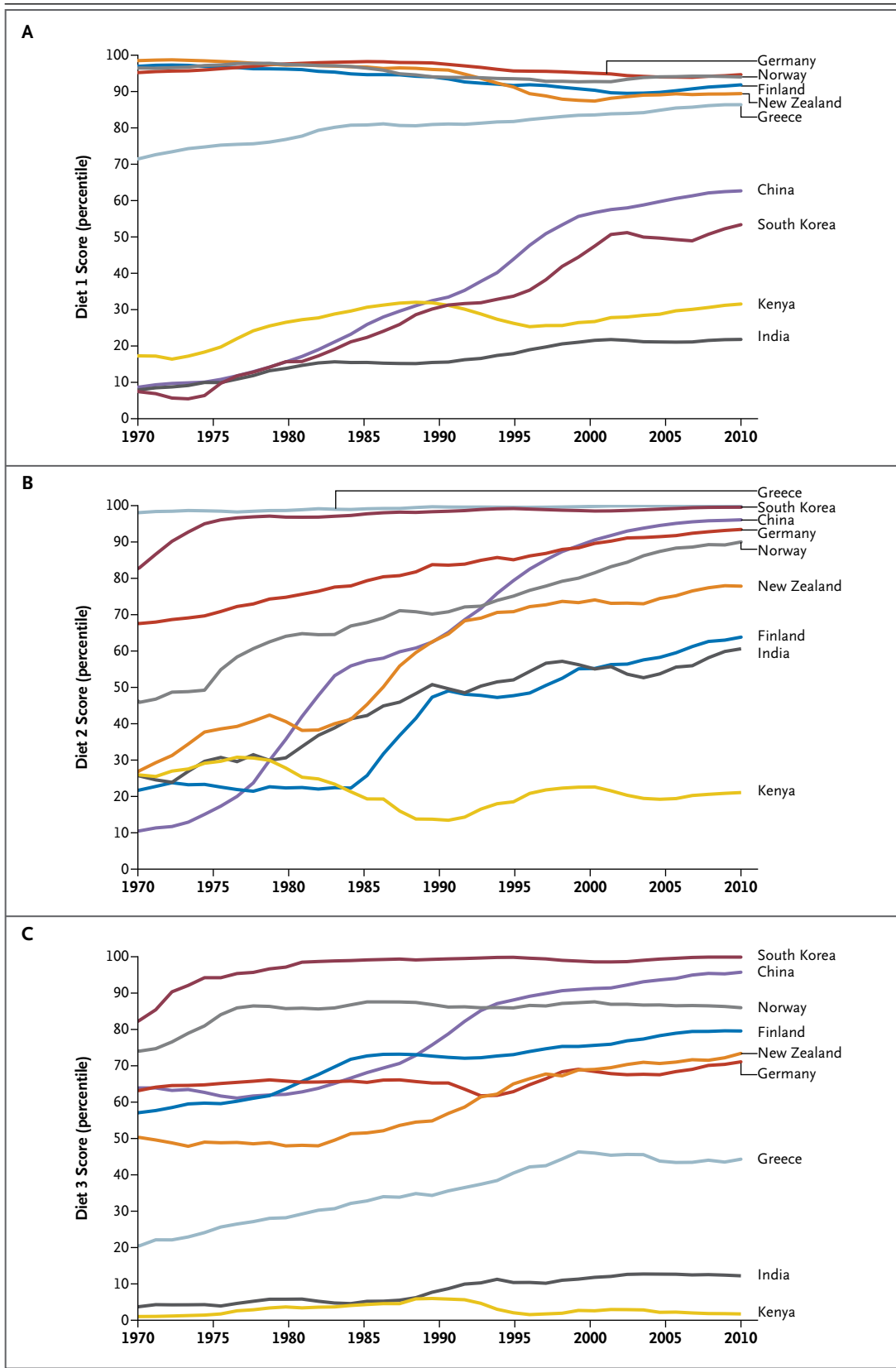
A — Diet 1 Score (percentile): Germany, Norway, Finland, New Zealand, Greece, China, South Korea, Kenya, India (1970–2010)

B — Diet 2 Score (percentile): Greece, South Korea, China, Germany, Norway, New Zealand, Finland, India, Kenya (1970–2010)

C — Diet 3 Score (percentile): South Korea, China, Norway, Finland, New Zealand, Germany, Greece, India, Kenya (1970–2010)

Each dietary score is a composite indicator that captures the availability of 21 food types in the United Nations Food and Agriculture Organization food balance sheets. A high Diet 1 score (Panel A) indicates a high availability of sugars; meat, animal products, animal fats, milk, and eggs; and total calories, as well as a low availability of pulses (legumes) and cereals. A high Diet 2 score (Panel B) indicates a high availability of nuts; fruits, vegetables, and vegetable oils; cereals; and total calories. A high Diet 3 score (Panel C) indicates a high availability of fish, aquatic products, and oil crops (e.g., soybeans, coconuts, and olives) and a relatively low availability of milk and pulses. Scores were calculated with the use of principal-component analysis, an approach that aggregates food types on the basis of the degree of correlation to each other within the data set.[43,44] Because principal components do not have a directly interpretable scale, we used percentiles of values (smoothed using a 5-year moving average) for presentation. The re-scaled values therefore range from 0% (lowest value observed in any country in the world between 1970 and 2009) to 100% (highest value observed in any country in the world between 1970 and 2009).

and also for elevated blood pressure, which in turn increases the risk of stroke, other cardiovascular diseases, chronic kidney disease, and kidney cancer. A decrease in dietary salt in Japan and Finland, countries in which salt intake was previously more than 20 g per day in some areas, has been associated with a decline in rates of stomach cancer and hemorrhagic stroke.[47,48]

PHYSICAL ACTIVITY

Studies of the beneficial health effects of physical activity date back to the 1950s[49] and have been replicated in large cohorts.[50] Physical activity at work, walking, and, in some populations, bicycling used to be major contributors to total energy expenditure but have declined dramatically in industrial and urban societies. Paralleling this shift, more recent epidemiologic studies in high-income countries have focused on leisure-time activity, with less emphasis on work and methods of local transportation, which are important in developing countries.[51] Only recently has attention been given to population-based measurement of physical activity in countries at all stages of urbanization and economic development. The limited available global data nonetheless show low levels of activity and long periods in sedentary conditions in high-income and ur-

banized countries and higher activity levels in rural populations that engage in agricultural activity and walk or bicycle long distances for daily activities.

THE GLOBAL RISK-FACTOR TRANSITION

Thus far, the epidemiologic transition has been viewed as a process through which the share of noncommunicable diseases as causes of death increases with declining mortality and rising longevity. As population-based data on medical causes of death and, more recently, on risk factors have become available, a more complete picture of the epidemiologic transition is emerging — one in which the interplay among risk factors and medical care leads to distinct disease patterns in different populations, with variations even among noncommunicable diseases.[2] Despite this diversity, an increasingly salient feature of risk-factor transitions is that many behavioral and dietary risks, and their metabolic and physiological mediators, that have been prominent in high-income countries are now at the same or higher levels in low- and middle-income countries.[44] This pattern parallels the higher prevalence of most risk factors and higher mortality from noncommunicable diseases in lower socioeconomic groups than in higher socioeconomic groups within high-income countries.[52]

Knowledge of risk-factor trends provides a more complete picture of the epidemiologic transition as well as lessons for how the risk factors can be reduced and managed in countries at all levels of economic development, with the use of various preventive strategies.[2] From a public health perspective, smoking is currently the most policy-responsive behavioral risk factor, with major successes in tobacco control in a number of high- and middle-income countries but with a shifting burden to low- and middle-income nations. Harmful alcohol consumption has been curbed in some Western countries but remains a major public health burden or is even worsening in others, especially in eastern Europe and Latin America.[25] Curbing its current harms and preventing its rise in Asia and other developing regions with the use of interventions known to be effective[2] should be a priority.

Although dietary patterns are shaped by cultural, environmental, technological, and eco-

nomic factors, they can also be modified through mechanisms that range from broad food and agricultural policies to targeted pricing and regulatory interventions related to specific harmful or beneficial dietary components. Such mechanisms are reviewed elsewhere.[2,53]

The availability of population-based and personal interventions for tobacco smoking, excessive alcohol consumption, and elevated blood pressure or lipid levels has made overweight, obesity, and high blood glucose levels the wild cards of noncommunicable-disease risks globally. Some have argued that the obesity epidemic may reverse life-expectancy gains in high-income nations.[54] At the same time, blood pressure and cholesterol levels, which partially mediate the hazardous effects of excess weight on cardiovascular diseases, have declined in most high-income countries and in parts of Latin America.[43,45] This has probably helped dampen or delay the effects of weight gain on cardiovascular diseases, which have declined impressively in industrialized countries.[55] However, there are currently few effective measures against the harms of overweight and obesity with respect to hyperglycemia, diabetes, and cancers, making the concurrent epidemic of diabetes a global health challenge.[56] Randomized trials of dietary changes (in some cases combined with exercise) have shown moderate weight-loss benefits for up to 2 years,[57] but the

long-term and large-scale community effectiveness of such interventions has not been established.[58] Similarly, studies have modeled or qualitatively assessed the potential benefits of physical-activity interventions,[59] but the empirical evidence of their effectiveness at the population level remains limited. As a result, policy options and recommendations for weight control[60] and increased physical activity remain broad and untested but are needed to avoid a slowdown or even reversal of the progress in mortality reduction.[2]

Although the behaviors of individuals are important factors in the patterns of risk factors for noncommunicable diseases, successful efforts to reduce smoking, alcohol consumption, and, more recently, trans-fat and salt consumption show that there is great scope for collective action through policy formulation and implementation.[2] Successful policies, such as tobacco and alcohol taxes and restrictions, should be replicated in all populations. There is also a need for bold and creative policies that address harmful alcohol consumption, improve diet, and increase physical activity.

REFERENCES

1. Wang H, Dwyer-Lindgren L, Lofgren KT, et al. Age-specific and sex-specific mortality in 187 countries, 1970-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380: 2071-94.
2. Ezzati M, Riboli E. Can noncommunicable diseases be prevented? Lessons from studies of populations and individuals. Science 2012;337:1482-7.
3. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380:2095-128.
4. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Selected major risk factors and global and regional burden of disease. Lancet 2002;360:1347-60.
5. 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. [Erratum, Lancet 2013;381:1276.]
6. Danaei G, Ding EL, Mozaffarian D, et al. The preventable causes of death in the United States: comparative risk assessment of dietary, lifestyle, and metabolic risk factors. PLoS Med 2009;6(4): e1000058.
7. Stevens G, Dias RH, Thomas KJ, et al. Characterizing the epidemiological transition in Mexico: national and subnational burden of diseases, injuries, and risk factors. PLoS Med 2008;5(6):e125.
8. Willi C, Bodenmann P, Ghali WA, Faris PD, Cornuz J. Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis. JAMA 2007; 298:2654-64.
9. Lin HH, Ezzati M, Murray M. Tobacco

smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. PLoS Med 2007;4(1):e20.
10. Gu D, Kelly TN, Wu X, et al. Mortality attributable to smoking in China. N Engl J Med 2009;360:150-9. [Erratum, N Engl J Med 2010;363:2272.]
11. Nakamura K, Huxley R, Ansary-Moghaddam A, Woodward M. The hazards and benefits associated with smoking and smoking cessation in Asia: a meta-analysis of prospective studies. Tob Control 2009;18:345-53.
12. Jha P, Jacob B, Gajalakshmi V, et al. A nationally representative case–control study of smoking and death in India. N Engl J Med 2008;358:1137-47.
13. Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Atlanta: National Center for Chronic Disease Prevention and Health Promotion, Office on

Smoking and Health, 2006. (DHHS publication no. [CDC] 89-8411.)

14. Global status report on noncommunicable diseases 2010. Geneva: World Health Organization, 2011.

15. Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. Tob Control 2012;21:96-101.

16. Department of Health and Human Services. A report of the Surgeon General: how tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease. Atlanta: National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2010. (DHHS publication no. [CDC] 89-8411.)

17. Secretan B, Straif K, Baan R, et al. A review of human carcinogens — Part E: tobacco, areca nut, alcohol, coal smoke, and salted fish. Lancet Oncol 2009;10: 1033-4.

18. Roerecke M, Rehm J. The cardioprotective association of average alcohol consumption and ischaemic heart disease: a systematic review and meta-analysis. Addiction 2012;107:1246-60.

19. Mathurin P, Deltenre P. Effect of binge drinking on the liver: an alarming public health issue? Gut 2009;58:613-7.

20. Rehm J, Baliunas D, Borges GL, et al. The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction 2010;105:817-43.

21. 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.

22. Allamani A, Prina F. Why the decrease in consumption of alcoholic beverages in Italy between the 1970s and the 2000s? Shedding light on an Italian mystery. Contemp Drug Probl 2007;34:187-97.

23. Cipriani F, Prina F. The research outcome: summary and conclusions on the reduction in wine consumption in Italy. Contemp Drug Probl 2007;34:361-78.

24. Zaridze D, Brennan P, Boreham J, et al. Alcohol and cause-specific mortality in Russia: a retrospective case-control study of 48,557 adult deaths. Lancet 2009; 373:2201-14.

25. Leon DA, Chenet L, Shkolnikov VM, et al. Huge variation in Russian mortality rates 1984-94: artefact, alcohol, or what? Lancet 1997;350:383-8.

26. Leon DA, Shkolnikov VM, McKee M. Alcohol and Russian mortality: a continuing crisis. Addiction 2009;104:1630-6.

27. Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. Lancet 2009;373:1083-96.

28. Wormser D, Kaptoge S, Di Angelanto-nio E, et al. Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. Lancet 2011;377:1085-95.

29. Renehan AG, Tyson M, Egger M, Heller RF, Zwahlen M. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. Lancet 2008;371:569-78.

30. Ni Mhurchu C, Parag V, Nakamura M, Patel A, Rodgers A, Lam TH. Body mass index and risk of diabetes mellitus in the Asia-Pacific region. Asia Pac J Clin Nutr 2006;15:127-33.

31. Ni Mhurchu C, Rodgers A, Pan WH, Gu DF, Woodward M. Body mass index and cardiovascular disease in the Asia-Pacific Region: an overview of 33 cohorts involving 310 000 participants. Int J Epidemiol 2004;33:751-8.

32. Pischon T, Boeing H, Hoffmann K, et al. General and abdominal adiposity and risk of death in Europe. N Engl J Med 2008;359:2105-20. [Erratum, N Engl J Med 2010;362:2433.]

33. Stevens GA, Singh GM, Lu Y, et al. National, regional, and global trends in adult overweight and obesity prevalences. Popul Health Metr 2012;10:22.

34. Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet 2011;377:557-67.

35. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research, 2007.

36. Mozaffarian D, Appel LJ, Van Horn L. Components of a cardioprotective diet: new insights. Circulation 2011;123:2870-91.

37. He FJ, Li J, Macgregor GA. Effect of longer term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomised trials. BMJ 2013;346:f1325.

38. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. N Engl J Med 2009;360:859-73.

39. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med 2011;364:2392-404.

40. 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;(3)7:e1000252.

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

42. Puska P, Stahl T. Health in All Policies — the Finnish initiative: background, principles, and current issues. Annu Rev Public Health 2010;31:315-28.

43. Danaei G, Finucane MM, Lin JK, et al. National, regional, and global trends in systolic blood pressure since 1980: systematic analysis of health examination surveys and epidemiological studies with 786 country-years and 5.4 million participants. Lancet 2011;377:568-77.

44. Danaei G, Singh GM, Paciorek CJ, et al. The global cardiovascular risk transition: associations of four metabolic risk factors with national income, urbanization, and Western diet in 1980 and 2008. Circulation 2013;127:1493-502.

45. Farzadfar F, Finucane MM, Danaei G, et al. National, regional, and global trends in serum total cholesterol since 1980: systematic analysis of health examination surveys and epidemiological studies with 321 country-years and 3.0 million participants. Lancet 2011;377:578-86.

46. Asaria P, Chisholm D, Mathers C, Ezzati M, Beaglehole R. Chronic disease prevention: health effects and financial costs of strategies to reduce salt intake and control tobacco use. Lancet 2007;370: 2044-53. [Erratum, Lancet 2007;370:2004.]

47. Hirayama T. Epidemiology of stomach cancer in Japan: with special reference to the strategy for the primary prevention. Jpn J Clin Oncol 1984;14:159-68.

48. Tuomilehto J, Geboers J, Joossens JV, Salonen JT, Tanskanen A. Trends in stomach cancer and stroke in Finland: comparison to northwest Europe and USA. Stroke 1984;15:823-8.

49. Morris JN, Heady JA, Raffle PA, Roberts CG, Parks JW. Coronary heart-disease and physical activity of work. Lancet 1953; 265:1053-7.

50. Sattelmair J, Pertman J, Ding EL, Kohl HW III, Haskell W, Lee IM. Dose response between physical activity and risk of coronary heart disease: a meta-analysis. Circulation 2011;124:789-95.

51. Levine JA, Weisell R, Chevassus S, Martinez CD, Burlingame B, Coward WA. The work burden of women. Science 2001;294:812.

52. Di Cesare M, Khang YH, Asaria P, et al. Inequalities in non-communicable diseases and effective responses. Lancet 2013; 381:585-97.

53. Mozaffarian D, Afshin A, Benowitz NL, et al. Population approaches to improve diet, physical activity, and smoking habits: a scientific statement from the American Heart Association. Circulation 2012;126:1514-63.

54. Olshansky SJ, Passaro DJ, Hershow RC, et al. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med 2005;352:1138-45.

55. Di Cesare M, Bennett JE, Best N, Stevens GA, Danaei G, Ezzati M. The contributions of risk factor trends to cardiometabolic mortality decline in 26 industrialized countries. Int J Epidemiol 2013; 42:838-48.

56. Danaei G, Finucane MM, Lu Y, et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. Lancet 2011; 378:31-40.

57. Nordmann AJ, Nordmann A, Briel M, et al. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. Arch Intern Med 2006;166:285-93. [Erratum, Arch Intern Med 2006;166:932.]

58. Douketis JD, Macie C, Thabane L, Williamson DF. Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. Int J Obes (Lond) 2005;29:1153-67.

59. Cobiac LJ, Vos T, Barendregt JJ. Cost-effectiveness of interventions to promote physical activity: a modelling study. PLoS Med 2009;6(7):e1000110.

60. Gortmaker SL, Swinburn BA, Levy D, et al. Changing the future of obesity: science, policy, and action. Lancet 2011; 378:838-47.

Copyright © 2013 Massachusetts Medical Society.