

Major diet-related chronic diseases

Cardiovascular disease (CVD)

CVD is the term used by the scientific community to embrace not just conditions of the heart [ischemic heart disease (IHD), valvular, muscular, and congenital heart disease] but also hypertension and conditions involving the cerebral, carotid, and peripheral circulation. Diabetes is usually placed in a separate category from CVD. It is well recognized that the risk of CVD is related to diet, physical activity, and body [38, 39]. The patterns of food supplies and of food and nutrition that modify the risk of CVD are also well known.

The governments of many developed and many developing countries have issued dietary and other recommendations designed to control the incidence of CVD [41, 43]. The basic agreed finding is that varied diets that are high in vegetables, fruits, and starchy staple foods (preferably in minimally processed form) and that are relatively low in energy density, fats, saturated fats, sugar, and salt, are most protective against CVD. Such diets approximate those that have been traditional in many countries in Asia and Pacific SIDS, where public health problems have been food insecurity, undernutrition, and monotony of diets. However, when people have enough to eat and when diets are varied, diseases of undernutrition are not major public health problems. Table 3 summarizes these general relationships.

Additionally, there is conclusive evidence that regular physical activity maintained throughout life, together with nutrient-dense diets, protects not only against obesity and therefore indirectly against CVD. This finding has very important implications for developing countries in Asia and the Pacific, where protective diets and lifestyles are not as established as they are in developed countries. Programs and policies designed to control obesity and CVD in Asia and the Pacific should emphasize what is valuable in traditional and existing agriculture and food systems, food supplies, diets, and activity patterns.

Inappropriate diets are a major determinant of the risk of cerebrovascular diseases including hyperten-

sion and stroke [43]. Dietary recommendations for preventing these pathologies are the same as those for obesity and CVD except for an added focus on diets with less salt.

Cancers

There is also now a consensus that, in broad terms, the same diets and associated lifestyles that protect against obesity, diabetes, cerebrovascular diseases, and CVD, also protect against certain cancers, i.e., major cancers of epithelia as well as hormone-related cancers that become or remain epidemic as a consequence of demographic, nutrition transitions [40]. These cancers include those of the lung, breast, endometrium, colon, and rectum, and also cancers that may be associated with traditional diets, such as those of the mouth, throat, esophagus, and stomach (table 5).

It has been estimated [40] that cancer incidence might be reduced by 30% to 40% through appropriate diets and associated lifestyle. At 1995 levels, such a reduction would correspond to three to four million cases per year worldwide. This report is remarkable for its global perspective and for reconciling its recommendations with those for other major chronic diseases, including obesity and CVD, adult-onset diabetes and osteoporosis [40]. Its findings are consistent with those of this review. Diets and physical activity that protect against cancer also protect against other chronic diseases, and have no deleterious effect on other diseases.

Current diet recommendations, following food science, emphasize food and dietary patterns more than individual macro- and microconstituents. Food-based dietary guidelines are now generally accepted [73]. The diets that are most protective against chronic diseases, especially in sedentary populations, comprise mainly foods of plant origin. These diets are varied and high in nutrients, but relatively low in energy. They include substantial amounts of legumes and other vegetables, fruit, and minimally processed grains and other starchy

staples. They are also relatively low in fat, especially saturated fat, and in sugar, salt, and alcohol. In general they contain only modest amounts of foods of animal origin. The World Cancer Research Fund (WCRF) emphasized the importance of year-round variety in diets, and also the value of small amounts of meat, fish, poultry, and dairy foods, especially when diets might otherwise be monotonous [40].

Health costs

Epidemic, diet-related chronic diseases, entail important human and economic costs. The human costs have been quantified in terms of disability and death [47]. To date, however, there are almost no estimates of the economic costs in developing countries, although a study is in process for the Pacific islands [74]. There have also been studies for the United States, for example [75].

Using cautious assumptions, estimates are presented here for the human and economic costs of such diseases in the PRC and Sri Lanka. Estimates are presented for losses in 1995 and for projected losses in 2025, to give a sense of how these costs might evolve, together with nutrition and epidemiological transitions. These estimates should be treated cautiously, as ballpark estimates, rather than as definitive. They are intended to provide policy guidance on targeting interventions, as well as to show how the relative costs of undernutrition (including nutrient deficiencies) and overnutrition might change over time. The numbers of deaths, economic costs of hospital resources, and economic costs of premature mortality are included. No data were available from which to estimate the costs of lost work outputs due to morbidity, whether from lost workdays, or from lowered productivity by those with chronic diseases continuing to work. Therefore, the total costs presented here are almost certainly underestimates.

The five diet-related chronic diseases considered here are CVD (especially IHD), diabetes, hypertension, stroke, and cancers. Among these, CVD, stroke and cancers are major causes of premature death. Hypertension and diabetes, themselves life-threatening diseases at late stages of pathology, also increase the risk of stroke and CVD.

Figure 25 depicts the major causal pathways included in the model and some other important pathways which could not be modeled because epidemiological data were too limited (for example, the effects of *trans* fatty acid intake on IHD) or because of overlapping effects. For example, the link between overweight and hypertension, although well established, was not modeled. The effects of overweight on IHD directly were modeled. It is not valid to double count and to add the indirect effects of overweight, via hypertension, on IHD. The focus here is on diet and does not include

the additional effects of physical activity, except in so far as activity has indirect effects, via energy imbalance and overweight status. However, physical activity is known to have direct effects on hypertension, diabetes, and CVD. Therefore, again, the estimates here are cautious.

The effect of inappropriate diets on disease risk is multidimensional. There are potentially overlapping effects, e.g., LBW, via obesity, on CVD; LBW, via hypertension, on CVD; and LBW, via diabetes, on CVD. Therefore, care was taken, when estimating overall effects, not to double count such effects. The separate effects of different pathways can be added only if the clinical studies, on which the RR estimates are based, also control for confounding effects. For example, in order to be able to add up the effects of LBW, via overweight and via hypertension, on CVD, it is necessary to control for current weight in the link between LBW and hypertension and in the link between hypertension and CVD. Because studies do not always adjust for confounding factors, caution was exercised here and, when adding up effects via different pathways, the maximum amount of overlap was assumed. In the literature on cardiovascular and cancer epidemiology, there is an attempt to control for key biological confounding factors other than nutrition. However, studies on fetal and infant undernutrition are very new and only a few have controls for subsequent confounding factors [43]. For example, to analyze the effects of LBW or LBW-low PI on adult-onset diabetes requires controls for obesity, which is another direct determinant of diabetes. Such studies to date have lacked adequate controls for current weight.

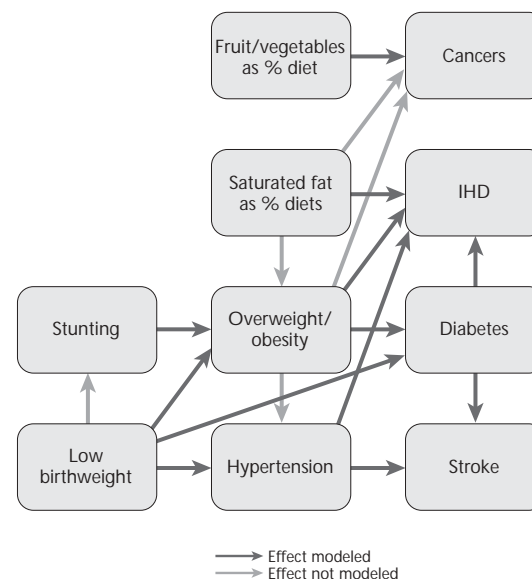


FIG. 25. Important pathways for diet-related chronic diseases. Notes: IHD = ischemic heart disease.

Figures 26 to 28 illustrate RRs for IHD, stroke, and diabetes, respectively. These RRs are taken from existing literature, using meta-analyses wherever possible. Much of this literature is for populations in rich countries, often the United States, and the majority of the studies are for men. The applicability of these RRs to Asian and Pacific island populations, and to women or men and women combined, is not clear. If

anything, studies suggest that RRs for some of these chronic conditions are even higher in Asian populations [76, 77]. It is not possible to give separate RRs for morbidity and mortality. Such RRs are not generally available, except for CVD, and even for CVD it is not yet possible to separate out all the pathways for mortality and morbidity.

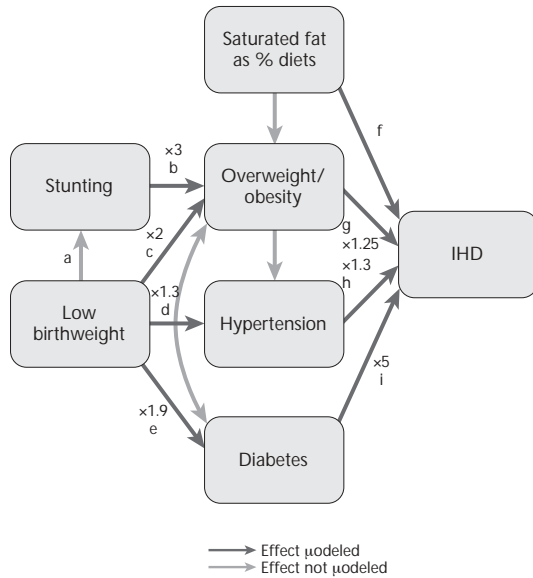


FIG. 26. Relative risks (RR) for ischemic heart disease (IHD), expressed as multiples (x).

Sources and notes:

- a Assumes that all low birthweight babies and some normal birthweight babies become stunted. Data on overlap between stunting and low birthweight are limited.
- b see text.
- c see text.
- d [54] citing [78]; that the relative risk (RR) of hypertension is 1.6 for birthweights from <5 lbs as compared to birthweight >10 lb (>2.27 to >4.54 kg) is 1.6. The RR of hypertension for birthweights <5 lbs as compared to those >5 lbs (<2.27 kg compared to >2.27 kg) is assumed to be 1.3, using linear interpolation and controls for current body mass index (BMI).
- e [13] using a Health Professional Study of the USA for men, with controls for current body mass index (BMI).
- f [43] citing [79]; data are cross-country regression averages for men, for ten-year incidence of IHD, which increases by 78 per 100,000 per 1% increase in saturated fat as % of diet calories.
- g [80] using a study from Framingham, MA for 12-year, age-adjusted IHD rates in which RR is averaged across three tertiles of subscapular skinfold thickness.
- h [43] citing [81], for effects on IHD from treatment trials of hypertension, associated with long-term decline of diastolic blood pressure of 5–6 mm of Hg.
- i [43] citing [82] for death rates for IHD averaged across men and women.

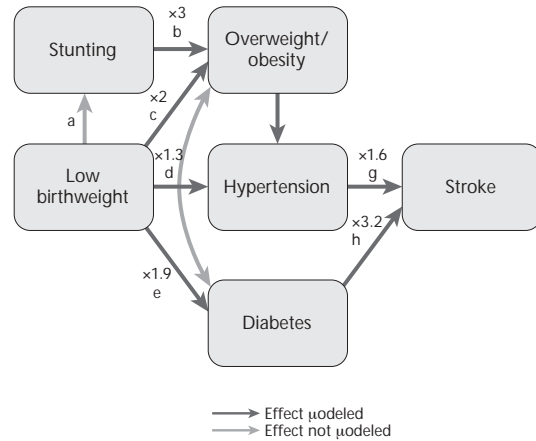


FIG. 27. Relative risks (RR) for stroke, expressed as multiples (x)

Sources and notes:

- a–e as for fig. 26.
- f [83]; for US adults.
- g [43] citing [81], for effects on IHD from treatment trials of hypertension, associated with long term decline of diastolic blood pressure of 5–6 mm of Hg.
- h [43]; average for men and women, from a heart study based in Framingham, MA, USA.

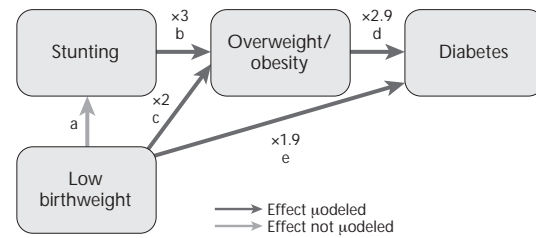


FIG. 28. Relative risks (RR) for diabetes, expressed as multiples (x)

Sources and notes:

- a,b,c,e as for fig. 26.
- d RR for BMI>25 as compared to BMI<25; estimated from [83]; using National Health and Nutrition Survey data (NHANES II).