on the perspectives of further epidemiological research to offer more reliable and accurate assessment of risk for general dietary patterns and specific nutrients or micronutrients.

Over the last decade, epidemiological research on diet and cancer has appreciably increased in quantity and quality, and has provided a number of relevant new evidences. It is, however, difficult to evaluate how much new information can be provided in the near future by further improvements in dietary assessment, and in the methodology and design of case–control and cohort studies. In any case, problems of collinearity between several components of diet, and inherent limitations in any assessment of dietary history, will almost certainly continue to pose serious difficulties for quantifying the role of specific nutrients and micronutrients. Some of these difficulties, at least for micronutrients, will possibly be solved only through randomised intervention trials. In the absence of evidence from controlled intervention studies, we are now not yet in the position of recommending dietary supplements, but also a general recommendations to avoid use of dietary supplements can be open to criticism, and may (hopefully) be changed in the near future.

Finally, descriptive epidemiology should remain a basic framework for monitoring the impact of dietary changes. Attention should be paid both to absolute values of current rates and to their trends over time. Miller and colleagues, for instance, indicate that "incidence of breast cancer in eastern Europe is approximately half that of high risk countries of western Europe". However, in 1985–1989, overall age-standardised (world standard) breast cancer mortality was 22.35/100 000 women in the 12 countries of the EEC, 20.44/100 000 in other western European countries, and 16.57/100 000 in eastern European countries (i.e. a difference in mortality of only 20 to 25% between western and eastern Europe) [8]. Trends in mortality over time, moreover, have been more unfavourable in eastern than western Europe since, in 1960–1964, rates were 9.67/100 000 women in eastern Europe, 17.54/100 000 women in the 12 EEC countries, and 17.96/100 000 women in other western European countries. In eastern Europe, over recent calendar periods, mortality trends have been comparatively even more unfavourable at younger ages. It is, therefore, difficult to make any quantitative inference on a more favourable dietary pattern for breast cancer in eastern as compared to western Europe, in terms of fats or other nutrients. Likewise, any quantitative estimate of potential incidence reduction only on the basis of some selected overall age-adjusted rates may be severely misleading.

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D. Forman

MILLER AND COLLEAGUES have provided the scientific community with an admirable and succinct review of the epidemiological evidence relating diet to the aetiology of cancer. Building from this, they have ventured into the contentious area of making dietary recommendations for the general public and provided, in Table 1, quantitative estimates for the impact of their recommendations on the reduction in cancer incidence. An exercise such as this inevitably has shortcomings, and it would be all too easy to lose sight of the major contribution made in this review by criticising specific points of detail. It is, however, only by considering the detail that gaps in our present understanding will be highlighted, and future research issues prioritised.

The point of detail which I want to discuss concerns the recommendations about stomach cancer. It may be argued that dietary advice to reduce the risk of a cancer which is the second most common in the world, and which is the only specific cancer site to have a recommendation exclusively devoted to it (number 5, limit the use of salt and the consumption of salty, salt preserved foods and nitrites) is no mere point of detail. Also, examination of the figures in Table 1 shows that the incidence of stomach cancer could potentially be reduced by 68% from dietary changes alone. This is greater than the equivalent incidence reduction for any of the other 12 sites (or groups of sites) listed — the figures of 80% for lung cancer, and 90% for cancers of the upper gastrointestinal and respiratory tracts including major effects from eliminating smoking.

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There is a highly consistent body of epidemiological evidence associating low fruit and vegetable intake and high salt intake with an increased risk of stomach cancer. Changes in dietary behaviour to increase consumption of fruit and vegetables, and reduce consumption of salt would result in several other benefits to health, apart from an effect on stomach cancer. I have, therefore, no disagreement with the recommended actions for these foods.

My concern is with the advice to reduce the intake of cured meat and salt-preserved foods (the latter not necessarily being the same as salty foods) in which the ingredient of concern is nitrite, added to inhibit bacterial growth. Although a few epidemiological studies have shown that high consumption of these foods is a risk factor for stomach cancer, it is also the case that foods preserved by other means, e.g. smoked meat, pickled foods, have been identified as risk factors in other studies. Populations consuming a lot of preserved food (whatever the method of preservation) are likely to be deficient in their intake of fresh food, especially fruit and vegetables. It is unclear whether preserved foods really do cause cancer *per se* or, perhaps, are markers for overall dietary patterns that are unhealthy. Until this confounding can be adequately untangled, it is my belief that it would not be appropriate to make general health recommendations about these dietary items. It should also be noted that in many populations such foods are consumed in relatively small quantities, and that the level of nitrite added to meat products has declined in recent decades.

The idea that nitrite might be involved in gastric carcinogenesis owes much to the hypothesis of Correa and colleagues [1], suggesting that endogenous formation of *N*-nitroso compounds in the stomach (from the reaction of nitrite with protein products) is an important aetiological factor leading to disease. However, the small amounts of nitrite ingested directly from cured meat are generally considered to be insignificant in comparison with the amounts that are formed either from the reduction of nitrate or from nitrogen oxides produced by stimulated macrophages.

In the context of the very sensible set of recommendations listed in the review, I think it is unnecessarily alarmist to convey the impression that cured meats (and the nitrite within) are causing cancer. The level of evidence relating to this relationship is substantially less than that for any of the others listed. It is essential that well founded advice is not diminished by confusion with ill-founded scare stories. I am going to adopt the other recommendations, but will continue to eat my bacon and salami.

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G.G. Giles and P. Ireland

OUTSTANDING ASPECTS of this review include its recognition of the lack of epidemiological evidence upon which to base dietary guidelines for the prevention of cancer, and a thoughtful discussion of how such guidelines might be used to achieve desirable changes, placing particular emphasis on the formation of dietary patterns in childhood.

One specific item which deserves additional comment, as it highlights some of the problems in devising dietary policy, is the question concerning a possible protective effect of "fibre" on the risk of cancer (largely of the colon and rectum). The authors draw attention to the historical difficulties in defining "fibre" and the ensuing inconsistencies in the literature. To this must be added the confusion concerning "fibre" in the minds of the general population, a situation that has been exacerbated by the food industry. To members of the general public "fibre" is now commonly synonymous with cereal bran, and there is a danger that people may increase consumption of bran rather than vegetables in response to advice to increase "fibre" intake. Nowadays, "fibre" is a collective rubric for a heterogeneous group of non-starch polysaccharides (cellulose, hemicellulose, pectin) and other components of plant cell walls that are resistant to digestion in the human gastrointestinal tract (lignin, suberin, cutin).

Apart from problems with its definition, research to isolate the effects of "fibre" is frustrated by the collinearity of "fibre" with other dietary components. "Fibre" is obtained from vegetable and other plant sources, and it is difficult to separate the effects of "fibre" from other plant substances such as antioxidant vitamins, carotenoids and possibly anti-carcinogenic phytochemicals that are yet to be discovered. The review does not cover a recent overview of vegetables and the effects of their nutrient and non-nutrient components on cancer risk [1, 2] which addresses these issues in some detail.

The evidence for a protective effect of "fibre" is weak, and there is a growing view that "fibre" intake is possibly only a marker for vegetable and fruit consumption [3]. Much of the evidence for a protective effect of fibre has been obtained from studies which have based measures of intake on the consumption of a few fibre-rich foods. Nevertheless, more than one authority has chosen to support policies aimed to increase "fibre" consumption in the absence of any definitive studies, but making inferences from meta-analyses of several poor studies (and the lack of evidence of any harm). It is reassuring, in view of the minimal evidence for a protective effect of "fibre" in the form of

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