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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Pergamon

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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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cereal bran, that the authors do not specifically recommend an increase in whole grain and cereal consumption. They acknowledge, however, that to concur with other guidelines, e.g. in regard to decreasing fat consumption, will require more calories to be obtained from this source.

Cereals have been inversely related to large bowel cancer risk in ecological studies yet several case–control studies have found an increased risk with the consumption of rice and pasta [4–7]. A possible explanation for this anomaly might be that at the population level cereals are inversely related to energy availability, but within a population the consumption of cereal staples is positively related to energy intake. Energy intake has generally not been measured in the case–control studies mentioned above so it has not been possible to control for its effect. The case–control study of Iscovich and colleagues [8] is particularly noteworthy in this regard because of the finding that carbohydrate was the most important factor driving the adverse effect of total energy intake. There is a view that the macronutrient composition of the diet should be modified to increase complex carbohydrates at the expense of fat. Mindful of the ethical considerations that are so well expressed in the manuscript, it would seem that we are not quite yet in a position to offer this advice to the public. Bingham [9] has shown that the effect of starch has seldom been reported in studies of colorectal cancer in humans. Future studies should report the effects of oligosaccharides, starch, non-starch polysaccharides and energy. If dietary "fibre" is to be reported at all, the source of the fibre needs to be stated clearly, as does the method of fibre analysis. Our ignorance is profound, and each incremental gain in knowledge forces us to revise previously held beliefs. The point is well made about the changing behaviour of potato starch which acts physiologically like "fibre" when eaten cold. If this is the case, diet diaries and questionnaires will need to specify this in future studies, particularly where potatoes are a staple food.

There are obviously many difficulties in researching the effects of dietary "fibre" on cancer risk and, therefore, in knowing what to recommend in dietary guidelines. There is little to suggest, however, that people pay much attention to official guidelines, relying more for information on the food industry and other media propaganda. It would be a shame to see the widespread commercial fortification of food supplies with today's popular dietary icons. Ultimately, people eat foods not "fibre", and dietary guidelines should reflect and promote cuisine rather than quasi-pharmaceutical nutrients and non-nutritive substances. To this end, the authors have achieved a reasonable compromise. We join in their plea for better databases, better methodology and better studies.

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THE STATEMENT of Doll and Peto 1981 [1], according to which "diet is a chronic source of both frustration and excitement to epidemiologists" [1], still holds true, although data accrual and methodological advancements in nutritional epidemiology in the last decade are probably unrivalled by any other field [2]. The article by Miller and co-authors in the present issue of *European Journal of Cancer* (pp. 207–220) is a vast, though obviously not complete, overview of the present state of the art. Some issues (e.g. cancer of the breast and colon-rectum) and some approaches (e.g. case–control studies) have received more attention than others.

The tendency, for instance, to discuss separately macronutrients and a few micronutrients is very strong, and reflects the way nutritional data are routinely collected (generally by means of frequency questionnaires), analysed and published. This somewhat fragmentary approach has been partly attenuated by the present awareness of the implications of total energy intake [2], but can make us miss some potentially important aspects of eating patterns (e.g. the number and timing of meals during the day).

The variety of our daily sources of calories may also be an important aspect of a healthy eating pattern which has been generally overlooked. From an evolutionary view point, pre-neolithic hunter-gatherers were surprisingly good at choosing a balanced diet where no single food (mostly of vegetarian origin) provided more than a few point per cent of daily energy

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