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Invited Viewpoints

Petr Skrabanek

DIET AS the cause of cancer has exercised the minds of preventionists at least since the time of Hippocrates. A 1903 editorial on cancer noted that "there is hardly an article of food which has not at one time or another fallen under the ban of some more or less acute theoriser" [1]. Yet cancer mortality in afffluent countries, despite changes in dietary patterns, has remained unaffected.

Marked differences in the incidence of individual cancers in different countries have led risk factor epidemiologists to argue that most cancers are preventable. However, overall cancer mortality rates are very similar in countries with most unsimilar diets. Thus, even if diet were to be implicated, the same diet associated with a low rate of some cancer or disease could be linked to an increase in other causes of death, as if following the principle of communicating vessels. Therefore, before issuing recommendations for a change in the national diet, with all its cultural and economic consequences, the proponents of such a change should present convincing evidence for a beneficial effect on overall morbidity and mortality. It is not enough to assume such benefit, as witnessed by counter-intuitive results from cholesterol-lowering trials.

Miller and his colleagues provide no such evidence, and their recommendations do not follow from their literature review. While the bulk of their review deals with fat, the authors also state that sugars "may increase [cause?] the incidence of colon and other cancers". They do not mention protein, for which there is even more "evidence" for an association with cancer. If fat, sugar and protein are associated with cancer, a sceptic may be forgiven for drawing the conclusion that people who eat, die. However, it may be more true to say that cancer is as much "caused" by diet as tuberculosis was "caused" by diet before the real cause was found in the laboratory by Robert Koch.

Risk factor epidemiology is unlikely to advance our understanding of cancer, beyond the identification of "risk factors". It is a logical nonsequitur to assume that the removal of such markers of risk would remove the risk itself. Thus, for example, cutting off ears with the ear-lobe crease (a well-known risk factor for coronary heart disease) will do nothing for the risk of heart disease. Similarly, it does not follow that lowering (or increasing) the consumption of a particular dietary item will result in increased life expectancy.

The misuse of language betrays the authors' uncritical bias. Thus, when they describe dietary fat as a "determinant" of breast cancer, exerting a "significant effect", they imply causation. This is not science. An editorial in *Nature* [2] comments that "despite abundant evidence that dietary fat bears no relation to development of cancer of breast, the NIH intends (under the fashionable umbrella of "women's health") to initiate a study of 40 000 women. . . to try again to prove a link that is probably not there. Is it only because of the faddish infatuation with fat as the root of all dietary evil?"

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The preoccupation with fat and slimming, especially in the U.S.A, has reached a stage of lipophobia. Yet, serum cholesterol is inversely associated with cancer. Serum lipids are not positively associated with cancer. Controlled trials of fat and cholesterol reduction failed to reduce cancer incidence, or even increase cancer mortality. So why do epidemiologists still allude to international comparisons of fat consumption, when inferences drawn from such studies are textbook examples of the ecological fallacy? And why are case–control studies, with relative risks of less than 2, used as "evidence", when such small elevations are accountable for by methodological biases?

There is no scientific justification for making specific recommendations for the whole population, such as, do not consume more than 30% of total calories as fat. No evidence is provided to show that people with a fat consumption of, say, 40% have shorter life expectancy (other things being equal) than people who consume only 25%. And why should 6–8% of the total energy intake be in the form of polyunsaturated fats? Which fats, *cis* or *trans*? In what foods? Is 10%, until very recently recommended by other committees as part of the "prudent diet", now wrong? On what evidence? Would 5% or 9% be harmful? It is disappointing that Miller and his colleagues support their quantitative recommendations only by reference to other consensus committees.

It is irrelevant to use dietary data from Uruguay, Japan or China for designing "optimal" European diet. Furthermore, there is something absurd in making blanket recommendations for hundreds of millions of people. For the young and the old, for the sedentary and the manual workers, for fat men and for pregnant women, for the healthy and for the sick, for those who live in hot climates and for those who live in warm climates. Food is not just a source of calories or of omega-3 fatty acids; eating is a social affair, a pleasure, a tradition of recipes, a culture of cuisine, a regional speciality appreciated by travellers. Should a fisherman in Iceland, eating smoked or salted guillemots, as generations of his ancestors did, now switch to a Mediterranean diet of pasta, garlic and wine? The authors are in two minds about the "Mediterranean" diet, since they don't like its main component– plenty of wine. They fear that this would lead to a "major increase in cancer in Central and Northern Europe". Conversely, it could lead to a dramatic decline in heart disease.

"Speculation as to the proportion of total cancer attributable to diet is so tenuous as to be almost frivolous" [3]. Yet Miller and his colleagues indicate that 66–98% of cancers are "potentially preventable", though these estimates are deemed "conservative"! Thus, the gap between unwarranted assumptions and foregone conclusions is finally bridged.

The authors recommend six servings of vegetable and fruit, and five servings of whole grain and cereal products a day. Is Europe to adopt the lifestyle of Seventh Day Adventists? From the age of 2? Why do the authors think that governments play into the hands of the industry if they provide full strength milk and cream "even to schools"?

There is an ethical dimension to the authors' proposals, which they do not discuss. Imputing causality without proof leads to victim-blaming among cancer sufferers who did not follow the "recommendations". The authors state that "the final evidence of disease causality will only come from a reduction in disease incidence following relevant action". In other words, a population experiment is required, yet the population is promised 66–98% of cancer reduction. If a healthy volunteer, or a patient, has a right to be fully informed about the risks and benefits of the trial in which he takes part, even more meticulous attention should be paid to the rights of a whole population of healthy people who are subjected to mass prevention programmes and intervention, however well meant [4].

Risks are not as far-fetched as they may seem. In many randomised controlled trials of multifactorial risk reduction, an increased mortality was observed, especially in the early phases of such trials, perhaps due to a sudden change in the body's homoeostasis. Abrupt changes in diet may result in mood changes, depression, violent behaviour or suicide. The change from eating as pleasure to eating as "healthy behaviour" has the potential to induce obsessive behaviour, hypochondriasis and, in young girls, anorexia.

I am reminded of Sancho Panza's opinion of Doctor Pedro Recio who boasted that he did not cure existing maladies but prevented them from arising. "And the remedies he uses," says Sancho Panza, "are diet, diet and still more diet. . . in short he is killing me".

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A.J. McMichael

EPIDEMIOLOGISTS HAVE spent much time over the past three decades studying dietary factors in human cancer causation. The welter of results is becoming increasingly hard to digest — particularly since there have been rather few striking and consistent findings. Perhaps it is time to ask ourselves some more basic questions. Blow-by-blow reviews of this complex topic, such as is contained in the first half of the paper by Miller and colleagues, are increasingly unsatisfying, particularly if they lack contextual comment about the nature of the low-yield struggle between epidemiologists and diet-and-cancer research.

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