Diet composition and obesity

Boyd Swinburn and colleagues (Aug 27, p 804) report that, over the first half of the 20th century, increased mechanisation and motorisation were accompanied by a declining food intake and that this kept the prevalence of obesity low. They attribute the epidemic of obesity since the 1960s largely to excessive food production.

The evidence, however, is that it is the excessive consumption of specific elements of the modern diet that is propelling this epidemic. When the diet and weight of 120 000 individuals was monitored over 4 years, consumption of specific items such as French fries and sugar-sweetened beverages (soft drinks) was associated with weight gain whereas consumption of other items such as vegetables and whole-grain foods was associated with relative weight loss. Per capita consumption of French fries and soft drinks increased by about 400% after 1960 in the USA.

Although the use of sugars as palatability enhancers in manufactured foods in general is potentially obesogenic, the evidence linking soft drink consumption to obesity and obesity-related illness is the most comprehensive.

A 10% increase in the price of soft drinks has been shown to decrease consumption by 10%. A 50% sales tax on soft drinks alone could raise as much as US$50 billion in the USA per year. This is a third of the annual medical costs of overweight and obesity to that country—ie, $147 billion. More importantly, such a tax would diminish one important propellant of this epidemic. Thus a tax on the nutritionally valueless soft drinks should be a priority of the war against obesity.

I declare that I have no conflicts of interest.

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We thank Boyd Swinburn and colleagues for their informative articles on obesity. However, we challenge their conclusion that diet composition does not matter. If one accepts that “junk” food, characterised by high energy density and refined carbohydrates, is obesity-promoting, one must also accept that diet composition has a role.

Kevin Hall and colleagues acknowledge that some diets can lead to reduced hunger and improved satiety, but also state that the reported energy and macronutrient intakes in diet trials are “almost certainly erroneous” and make their interpretation difficult. Hall and colleagues overlook large-scale studies that have used biological markers to track compliance to predefined intakes of specific nutrients. The Diogenes trial clearly showed that a diet with modestly higher protein content and lower glycaemic index (GI) prevents weight regain after a major weight loss in adults, with lower dropout rate and spontaneous fall of 18% in the prevalence of overweight in their children, without any restrictions on energy intake. This finding shows that a small change in diet composition can reduce spontaneous energy intake and hence be important for obesity.

Swinburn and colleagues state that high protein or low GI diets are not suitable because of their “detrimental effect on the environment”. However, the 23% energy from protein in the Diogenes trial can be achieved by increasing plant proteins such as nuts and legumes. Low GI varieties of rice are eaten throughout south Asia, and the low GI of sushi might help explain the lower obesity incidence in Japan.

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Authors’ reply

Both letters raise important questions about the types of foods implicated in global obesity. In relation to determinants, we agree with Patrick Bradley that higher intakes of high-fat, high-sugar foods and beverages...
have been the main drivers of higher energy intakes. We highlighted that excess energy consumption has been driven over decades by an increasingly globalised food industry producing low-cost, highly palatable, heavily marketed processed foods and beverages that displace healthier foods from the diet.

In terms of potential solutions, the cost-effectiveness of taxes on obesogenic foods is impressive but, although increased revenue might be a real political incentive for such taxes, the substantial revenue raised is not included in cost-effectiveness analyses. Several countries have instituted taxes on obesogenic foods and, despite fierce opposition from the food industry, fiscal interventions are highly likely to increase as governments get serious about reducing obesity.

In our papers, we did not conclude that diet composition doesn’t matter. Rather, we enumerated the ways that diet composition could affect bodyweight and that the notion of “a calorie is a calorie” is too simplistic. On the basis of Diogenes data, Arne Astrup and Jennie Brand-Miller assert that “a diet with modestly higher protein content and lower glycaemic index (GI)...can reduce spontaneous energy intake”. However, their data do not support this conclusion since all diets in the Diogenes study led to similar reported decreases in energy intake by more than 2000 kJ per day. This reduction should have led to continued weight loss in all groups rather than weight regain, and suggests that the measurements of food intake were inaccurate. Future assessment of diet adherence might be improved by use of serial bodyweight measurements, mathematical models, and biomarkers such as urinary nitrogen.

Although the prescription of higher protein and lower GI diets might show some benefits in clinical weight-loss trials, we questioned the value of extrapolating these to population diets. Theoretically, vegetable protein could increase but, in reality, it is animal protein (and its greenhouse gas emissions) that has been increasing. Western countries need to reduce the high animal content of their diets if they are to achieve a sustainable carbon footprint, and so the sending of “high protein” messages to developing countries seems particularly inappropriate. A shifting of the traditional staple foods in countries such as China from polished white rice to brown rice or lower GI varieties would require far more evidence on the sheer feasibility and effectiveness of this approach than currently exists.

We declare that we have no conflicts of interest.

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Exclusion of Indigenous youth from important parts of the Australian Health Survey

The poor health of Australia’s Indigenous people is well recognised, and the recent campaign to “Close the gap” (or more realistically to minimise the gap) has been welcomed. Sound strategies depend on knowledge of determinants and markers of conditions that drive health disparities, and their distribution by region, remoteness, and socioeconomic status.

The unfolding Australian Health Survey, designed by the Australian Bureau of Statistics (ABS), is the first study of the nation’s health to include some direct physical measures and blood and urine tests for markers of nutritional status and of the risk, and presence, of common disorders. The non-Indigenous component of the survey is already underway, and the Indigenous wave will begin in April, 2012.

However, advised by several external bodies, the ABS will exclude Indigenous (but not non-Indigenous) people younger than 18 years from the “measures” or sample collection components of the survey. This omission directly contravenes an evidence-based approach to health and our knowledge of the importance of early determinants of life-long health and the efficacy of early interventions.

Does this decision stem from the costs and logistics of screening in remote areas? Other minority and disadvantaged individuals might also need interpreters, extended explanations, and decision supports, but are not excluded from the opportunity to participate. Do we assume that Indigenous children are less likely to comply? In our Aboriginal research projects and those of our colleagues, refusal rates of children have been negligible. Are we reluctant to expose the scope of the problems, or their potential effect on health services? Do we doubt that Indigenous parents or guardians can give informed consent on behalf of their children? Do we fear misuse of data and of biological samples? Are we caught in a medieval conflict between social and clinical approaches to health improvement in Indigenous people? What is the role of bodies with vested interests in current processes and advisory structures or in the myth of homogeneous Indigenous health profiles and health service needs? Does not this exclusion from the right to participate in national campaigns for the common good constitute a