ANNOTATION

Fatty Foods Make You Fat

Obesity is a major nutritional problem of affluent countries, and its prevalence shows no sign of decreasing. Being overweight, particularly for men with upper-body obesity (pot-belly), promotes hyperlipidaemia, diabetes, and hypertension [1]. In addition, obesity is a risk factor for coronary heart disease [2]. The condition is extremely resistant to treatment, and those patients who do manage to lose weight usually regain it.

The cause of obesity appears simple: people take in more dietary energy than they expend, and the excess is used for fat synthesis and stored in fat tissue. As for specific dietary culprits, both comparisons between populations and studies of time trends suggest that a diet rich in fatty foods promotes adiposity. However, this idea has been exceedingly difficult to substantiate. On the biochemical side, it has been argued that calorie for calorie, dietary fat is more fattening than an equivalent amount of carbohydrate. The reasoning here is that when carbohydrates are converted into fatty acids, about 20% of the energy is wasted in the conversion process and thus is no longer available for fat formation [3,4]. One can, however, envisage a biochemical scenario in which all of the energy in carbohydrates remains available for fat formation: an excess intake of dietary carbohydrates could allow the diversion of dietary fat from oxidation into storage, with carbohydrate taking its place as a fuel, and little carbohydrate being consumed in the biochemical conversion of glucose into fatty acid. Biochemical observations in man would seem to favour the latter mechanism: unlike rats, humans probably have only a very limited capacity to convert glucose into fatty acids [5].

On the behavioural side, the question of whether high-fat diets lead to caloric over-consumption remains a matter for debate. Duncan et al found that replacement of high-fat items by food rich in complex carbohydrates and fibre led to a spontaneous decrease in energy intake [6]. This is not unexpected. One gram of fat provides 9 kcal (37 kJ), while one gram of carbohydrate supplies only 4 kcal (17 kJ). Thus, for example, if 17 g of fat is left out of the diet one has to eat an extra 37 g of carbohydrate to maintain the same energy intake. When the energy density of the foods is kept constant it no longer appears to matter whether the energy is provided by fat or carbohydrate. This was neatly shown by van Stratum et al [7] in a well-designed and well-controlled study. They produced high-fat and high-carbohydrate liquid formulae, both of which provided 100 kcal per 100 g, with the same mouth feel, flavour, taste and colour. It turned out that subjects consumed as much energy from the high-carbohydrate as from the high-fat formulae when all other conditions were kept constant. Body weights did not change in this study.

In real life, fat-rich foods will usually pack more calories per gram than carbohydrate-rich foods. Lissner et al, in a simple but well-designed experiment, recently showed that the energy density of foods is indeed a major determinant of spontaneous energy intake [8]. Twenty-four women each participated in three randomly ordered dietary treatments, each of two weeks’ duration, in which 15–20%, 30–35%, or 45–50% of energy was derived from fat. These diets consisted of foods that were similar in appearance and palatability but differed in the amount of high-fat ingredients used. The subjects were healthy, non-smoking students and university staff, aged 22–41 years, with a range of weights-for-height. Breakfast and dinner were taken in the dining-room of the research unit; lunch and snacks were taken out. Subjects served themselves from large dishes and were free to eat as much or as little of any food they desired. The rotating menu consisted of 20 items, the fat content of which was manipulated by changing recipes and ingredients. Thus, muffins were prepared with different amounts of oil, pudding was made with low- or full-fat milk; for sandwiches or salads, mayonnaise provided a variable source of fat. As protein intake was fairly constant, the proportion of carbohydrates in the menus went up as the fat content went down.

The results of the study were clear-cut: mean caloric intake was 2714 kcal/day on the high-fat diet, 2352 on the medium-fat diet and 2087 on the low-fat diet. The lower energy intake was not caused by the low-fat foods being judged less palatable; the reverse was in fact the case. Evidently, subjects simply failed to increase their helpfuls of the low-fat carbohydrate-rich foods sufficiently to make up for the fat left out. Changes in body weight were consistent with variations in caloric intake: the average weight change during the two-week treatment periods were −0.40 kg on the low-fat diet, −0.03 on the medium-fat diet, and +0.32 kg on the high-fat diet. The differences in energy intake were, if anything, more pronounced for obese than for lean subjects. However, the number of obese subjects was relatively small, and the weight changes on the low- and high-fat diets were not significantly correlated with adiposity.

The authors suggest that habitual, unrestricted consumption of low-fat diets may be an effective approach to weight control. 'Unrestricted' may be giving patients a bit too much leeway — the use of items such as alcohol and soft drinks will usually need to be restricted if weight is to be controlled, even though such drinks do not contain fat. However, the conclusion that restricting the proportion of fat in the diet will lead to restriction
of total energy intake and weight loss appears to be well supported.

This conclusion has ramifications beyond the problem of obesity. The vigorously promoted use of low-fat diets for the control of hypercholesterolaemia has recently come under scrutiny because high-carbohydrate, low-fat diets tend not only to lower LDL-cholesterol but also to elevate VLDL-triglycerides and depress HDL-cholesterol. Experimental isocaloric diets high in monounsaturated safflower or olive oil and low in carbohydrates tended to produce a more specific lowering of LDL, without the adverse effect of high-carbohydrate diets on HDL and serum triglycerides [9,10]. Observations on the Cretan cohort of the Seven Countries Study appear to support the notion that the high-oil Cretan diet is associated with low coronary risk [11]. However, recent observations in Cretes suggest that, in spite of continued high olive oil intake, Cretan men and boys no longer have very favourable blood lipoprotein values [12]. One possible explanation for this is obesity, which has become very common in Crete in the past 25 years. Possibly this is caused by a decrease in hard physical labour combined with a continued high intake of fat in the form of olive oil. These developments in Crete, coupled with the observations of Lissner et al [8] should be taken into account when evaluating the nutritional value of monounsaturated oils. The diet that worked well for Cretan peasants in the 1950s could produce severe overweight in sedentary city-dwellers of the late 1980s, and thus obviate the favourable effects on lipoproteins which olive oil produces in controlled isocaloric experiments.

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References


Forthcoming Meetings

Aug 29–Sept 2, 1988; Brussels, Belgium

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III Inter-American Congress of Clinical and Therapeutic Pharmacology — Hypercholesterolemia: Clinical Implications and Therapeutic Advances (Dr. Manuel Velasco, c/o Mrs. M J. Osipina, MSD Venezuela, Apartado 70,320, Los Ruices, Caracas 1071A, Venezuela)