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Dietary fat and obesity: an unconvincing relation[1,2]

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Bray and Popkin (1) attempted to refute my earlier conclusion that diets high in fat do not appear to be the primary cause of excess body fat in our society and that a reduction in dietary fat is not the solution (2). The topic is important because replacement of dietary fat with carbohydrate has been the predominant nutritional advice in the past decade (3). In a sedentary population with widespread insulin resistance, this change in diet induces hyperinsulinemia, hypertriglyceridemia, and low HDL-cholesterol concentrations (4, 5). Thus, if weight is not substantially reduced, rates of coronary artery disease may not be reduced and could even increase.

Bray and Popkin criticize the epidemiologic data that I cited on time trends and on geographic comparisons relating dietary fat to body fat (2). Although these data provide some useful evidence, I concluded that the relation between dietary fat and body fat is particularly difficult to evaluate in nonexperimental studies and that the best evidence should come from long-term randomized trials. Bray and Popkin devote considerable attention to nonexperimental data, which they deem to be superior. Their alternative geographic analysis, which they characterize as more "representative" of world populations, compares poor developing countries such as India, Mali, and China with the United States and other affluent countries. The differences in wealth and lifestyle among these countries are so extreme that their analysis hardly constitutes evidence for a causal relation between dietary fat and body fat. However, some of the data for individual countries depicted in their Figure 1 are informative. The fact that nearly 60% of the South African population is overweight, with an intake of ≈22% of energy from fat, indicates that a massive obesity problem can occur even with fat intakes that are generally considered to be low. The same applies to Saudi Arabia. Bray and Popkin specifically criticize the ecologic study of 65 Chinese counties because there was little variation in body fat. However, these data are actually more informative because confounding by extreme variations in affluence and physical activity is less problematic among these counties than among countries worldwide. The lack of variation in body weight despite substantial variation in dietary fat intakes (range: 7–22% of energy from fat) is just the point.

Bray and Popkin's longitudinal analysis of dietary fat intake and weight gain in China during its period of transition from poverty to a modern society is treacherous. Dietary fat has increased concurrently with increases in wealth and food availability, reductions in infectious disease, and declines in physical activity (in part related to the acquisition of television by most households). In this extreme context, where many diets were monotonous and very low in fat, more varied and palatable higher-fat diets could plausibly contribute to weight gain; however, it is implausible that many Chinese would voluntarily resume the diets they consumed during times of poverty. Despite the likelihood of overstating the relation between dietary fat and weight gain because of serious confounding, Bray and Popkin's Table 2 indicates only a trivial association. The coefficients for percentage of energy from fat were not significant by conventional criteria and they predict that an increase of 10% of energy from fat would increase the body mass index by only 0.1 in adolescents and by 0.03 in adults! Also, a formal test of the difference in coefficients for energy from fat and from nonfat sources would clearly be nonsignificant.

Bray and Popkin's meta-analysis of dietary trials concluded that a 10% reduction in the percentage of energy from fat would reduce body weight by 16 g/d. However, the association is only marginally significant ($P = 0.05$) and a visual inspection of Figure 2 is unimpressive and shows marked heterogeneity. The temporal issue is key in their conclusion because if the effect is sustained over only a few weeks, the effect is trivial, but if sustained for ≥1 y it would be important. As acknowledged by Bray and Popkin, the duration of most of the studies they analyzed was only a few weeks and the longer-term trials did not show a continued decline in body weight beyond 6 mo; some showed a regain of weight. For this reason, I included in my overview only studies lasting ≥1 y; these studies consistently showed little if any effect of dietary fat reduction on weight.

Bray and Popkin omitted the recent 1-y study by Knopp et al (6) from their analysis—the largest study to date of the relation between dietary fat and weight change. In this study, subjects were randomly assigned to 1 of 4 levels of fat intake; at 1 y there were no differences in weight change among the groups. Compliance was documented by a large (39%) increase in plasma triacylglycerol in the group with the lowest fat intake. On the basis of Bray and Popkin's analysis, there should have been a 3-kg difference in weight change between the groups with the lowest (22% of energy from fat) and highest (27% of energy from fat) fat intakes in Knopp et al's study; however, there was no such difference. These data add further evidence that Bray and Pop-

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kin's results, based mainly on short-term studies, are irrelevant to long-term weight control.

In summary, the data that Bray and Popkin interpreted to support a relation between dietary fat and overweight are flawed and indicate at most only a weak association. Whether the effect of dietary fat intake on body fat is small or nonexistent remains to be determined by long-term trials. Unfortunately, a focus on fat intake alone distracts from the more appropriate focus on total energy intake and physical activity levels. Bray and Popkin do agree that total energy intake, not fat per se, in relation to energy expenditure determines body fat accumulation. If, as they argue, dietary fat increases body fat because the fat itself increases the energy density of the diet, then weight-reducing dietary interventions should focus on reductions in energy density rather than on the replacement of fat with carbohydrate. The latter strategy has spurred the development and production of hundreds of new low-fat foods that have the same energy density as the original product. Moreover, the notion that energy density has an important effect on long-term weight control remains an unproven hypothesis. Rather than relying on theoretical arguments, this approach must be thoroughly evaluated in long-term studies before admonishing the public to adopt yet another ineffective weight-reducing strategy. In the meantime, advice regarding dietary fat intake should emphasize replacing saturated and *trans* fats with nonhydrogenated, unsaturated oils, and balancing energy intake from both carbohydrates and fats with regular physical activity. This strategy, which is supported by a wealth of empirical data from metabolic studies of blood lipids and from epidemiologic and randomized trials of coronary artery disease (7), will reduce the incidence of coronary artery disease, the leading cause of death in the United States and worldwide.

REFERENCES

1. Bray GA, Popkin BM. Dietary fat intake does affect obesity! Am J Clin Nutr 1998;68:1157–73.
2. Willett WC. Is dietary fat a major determinant of body fat? Am J Clin Nutr 1998;67(suppl):556S–62S.
3. US Department of Agriculture, US Department of Health and Human Services. Nutrition and your health: dietary guidelines for Americans. Washington, DC: US Government Printing Office, 1995. (Homes and Garden Bulletin no. 232.)
4. Jeppesen J, Schaaf P, Jones G, Zhou MY, Chen YD, Reaven GM. Effects of low-fat, high-carbohydrate diets on risk factors for ischemic heart disease in postmenopausal women. Am J Clin Nutr 1997;65:1027–33.
5. Katan MB, Grundy SM, Willett WC. Should a low-fat, high-carbohydrate diet be recommended for everyone? Beyond low-fat diets. N Engl J Med 1997;337:563–6.
6. Knopp RH, Walden CE, Retzlaff BM, et al. Long-term cholesterol-lowering effects of 4 fat-restricted diets in hypercholesterolemic and combined hyperlipidemic men: The Dietary Alternatives Study. JAMA 1997;278:1509–15.
7. Willett WC. Nutritional epidemiology. 2nd ed. New York: Oxford University Press, 1998.

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