eat reasonable diets (that is, sufficient fruits and vegetables) to be concerned. It also questions whether all Americans will benefit from a low-fat diet, or whether for some, at least, there are risks involved as well, which is still an unanswered question. Indeed, three decades of low-fat diet recommendations has led many Americans to replace saturated fats with carbohydrates, not unsaturated fats. Certainly, the food industry has responded by creating low-fat and no-fat products that do just that. Moreover, there is suggestive but not definitive evidence both for and against the benefits of a low-fat diet, and clinical trials have both succeeded and failed in confirming the benefits.

Grundy discusses the trials that show a positive benefit, implying that these are the important ones, which makes for a compelling argument but does so at the expense of good science. Hegsted and Astrup *et al.* also pay attention only to those data that support the benefits of low-fat diets and ignore or reject as irrelevant or flawed all the copious evidence to the contrary. Astrup *et al.* for example cite a case-control study with 108 patients (and 142 controls) as unequivocal evidence that increased dietary fat intake is associated with increased heart disease rates, although such case-control studies are virtually meaningless. They say that meta-analyses demonstrate that low-fat diets are efficient weight loss diets, yet there are trials and even meta-analyses—a controversial tool, in any event—that suggest the opposite. They say that the obesity epidemic in America and elsewhere is "predominantly due to an inactive life-style," which is a reasonable hypothesis but unproven. There remains the complicated question of why individuals would continue to consume more calories than they expend, despite enormous social pressure against obesity.

Hegsted suggests that small individual risk reductions represent substantial gains for a population, but individuals are not populations. And deaths are not prevented, as I pointed out in the article, they are only delayed. Low-fat diets might indeed delay them, but if they do, the effect is marginal. The key point, as Grundy says, is that "all questions have not been answered,"and so we might not want to act as though they have been.

—GARY TAUBES

CORRECTIONS AND CLARIFICATION

Reports: "A β_2 adrenergic receptor signaling complex assembled with the Ca^{2+} channel $Ca_v1.2$" by M. A. Davare *et al.* (6 Jul., p. 98). First, in the list of authors, two present addresses were indicated for Johannes W. Hell, the correct one being the Department of Pharmacology, University of Iowa, Iowa City, IA 52242, USA. Second, the designations for the open and closed circles in figure 2J were reversed. The legend should have read as follows: "(**J**) Current amplitudes measured from ensemble averages with albuterol either applied to the bath (n = 10; closed circles) or by pipette backfilling (n = 10; open circles)...." And third, panel D of figure 2 was incorrectly reproduced. The correct panel D appears here.