animal or vegetable protein with fiber. Atherosclerosis 1977;26:397–403.


Reply to JS White

Dear Sir:

Studies using dietary conditions with exaggerated nutritional compositions are often used to define the potential for biological responses and to act as starting points for understanding nutritional effects on health. The monkeys in the study consumed 30% more fructose than the unhealthiest humans, with fructose approximating the 95th percentile of total carbohydrate consumption (1, 2). This intake was modeled on the clinical study by Stanhope et al (3), which showed changes in adiposity, plasma lipids, and insulin sensitivity. Monkeys supplemented with fructose at this same amount (4) similarly experienced detrimental changes in these variables. Thus, our goal was to evaluate the influence of an extreme change in fructose while holding adiposity constant by careful focus on caloric and body weight control. One advantage of animal models, especially Old World monkeys with gastrointestinal tracts more comparable to humans, is sufficient longer-term environmental control to unmask perturbations induced by manipulation of nutritional variables. A potential next step would be to narrow the intake range over which health effects may be seen, while matching all dietary ingredients including protein source. Casein is known to be proatherogenic in the context of cholesterol-loaded dietary experiments (5); however, we actually reported no elevations in plasma or liver cholesterol concentrations (free or esterified) with high-fructose/casein diets, endpoints that are known risk factors for atherosclerosis (6). We therefore believe that the lack of differences seen in these endpoints in our study suggests that protein source did not primarily influence metabolic health, microbial translocation, and liver injury outcomes. We conclude that consumption of very high amounts of simple carbohydrate is likely to impair intestinal integrity and initiate liver and metabolic changes. Human and rodent studies involving exaggerated diets support the greater potency of fructose compared with glucose in initiation of such changes (3, 7), and future experiments should refine these findings using relevant diets in relevant animal studies.

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