A targeted goal for energy-restricted diets in the management of coronary risk?1,2

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Over the past decade, the independent contribution of elevated body weight to the development of coronary artery disease (CAD) has been elucidated (1). Treatment of blood lipids in overweight individuals (and the insulin resistance that frequently accompanies it) raises the question of how body weight reduction fits into treatment strategies for CAD. A central question addressed by Raeini-Sarjaz et al (2) in this issue of the Journal focuses on whether a diet restricted in energy to induce weight loss (yet containing a high percentage of energy from fat) might have a more beneficial effect on blood lipids than does the currently advocated American Heart Association Step I diet, which focuses primarily on a reduction in dietary saturated fat while energy intakes are maintained. Given the abundance in the literature of studies of the effects of low-fat and low-saturated-fat diets on blood lipids and CAD risk (3), it is somewhat surprising that the differential effects of a low-energy compared with a low-fat diet have not been directly addressed. Equally surprising is the paucity of research investigating the effects of energy restriction compared with the effects of low-fat diets on cholesterol synthesis, a secondary objective of this study. By contrast, the effect of these diets on blood cholesterol concentrations has been studied, as reviewed by Hegsted and Kritchevsky (3).

In Raeini-Sarjaz et al’s study, 4 dietary phases—each lasting 6 wk—were used in a repeated-measures design. One diet was euenergetic (providing sufficient energy to meet needs) and low in fat and a second diet was restricted in energy and high in fat. Two other dietary phases were included to serve as controls: a euenergetic, high-fat diet and an energy-restricted, low-fat diet. The authors’ concluded that the energy-restricted, high-fat diet reduced the risk of CAD more by lowering blood triacylglycerol concentrations while maintaining HDL-cholesterol concentrations (with a more modest reduction in LDL-cholesterol concentrations). By contrast, the euenergetic, low-fat, high-carbohydrate diet significantly lowered LDL cholesterol but also lowered HDL-cholesterol concentrations.

The authors’ interpretation of beneficial lipid changes resulting from the consumption of a low-energy diet is influenced by many issues that are extremely important to clinical nutrition therapy today. First, the low-energy diet was deemed superior because it did not result in a reduction in HDL cholesterol, as did the low-fat diet. Because isolated low HDL-cholesterol concentrations that are genetically controlled have been associated with increased CAD risk, many scientists believe that a dietary-induced reduction in HDL cholesterol is equally atherogenic (4). However, this has not been proven, especially when the reduction in HDL cholesterol occurs simultaneously with a significant reduction in LDL cholesterol. The same is true for the low-fat diet—induced elevations in triacylglycerol concentrations (5). Although there are epidemiologic data that suggest that carbohydrate-induced hypertriacylglycerolemia may confer a risk similar to that of endogenous hypertriacylglycerolemia (4, 6), this issue is still controversial.

A second area of active discussion concerns whether LDL cholesterol alone or the ratio of total to HDL cholesterol is a better indicator of risk. Use of the ratio as an indicator of change in risk is problematic because the ratio is based on a relatively small number in the denominator (ie, HDL cholesterol), which must be measured accurately (7). Because the variability in HDL-cholesterol concentrations can range up to 20%, accurate quantitation of HDL cholesterol in general practice may be difficult; therefore, the ratio of total to HDL cholesterol can be a less reliable measure. However, one advantage of the ratio is that the measurement of total cholesterol in the numerator somewhat reflects concentrations of remnant particles (eg, VLDL cholesterol) that can also contribute to CAD risk. Thus, in some patients, LDL cholesterol alone will be the primary target for treatment, whereas in other patients, treatment should be balanced to address a cluster of risk factors (small, dense LDL cholesterol; elevated triacylglycerols; and low HDL cholesterol). As the understanding of secondary risk factors advances, treatment guidelines will expand to better target treatment for the spectrum of risk factors in patients (8).

The third controversy that influences the interpretation of the present study’s findings is the effectiveness of euenergetic, low-fat diets to promote weight loss (9). Numerous studies have shown an average loss of 2–3 kg within the first 6 wk of initiating dietary fat reduction and such weight loss occurs even when patients are not specifically advised to reduce their energy intakes (reviewed in reference 5). This latter observation suggests that weight loss is stimulated in this circumstance, even when the subjects are not trying to lose weight, a goal that would be clearly appropriate for obese individuals who have extreme

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challenges in adopting dietary changes targeted at weight loss. It is also a matter of controversy whether such modest weight loss is beneficial in obese individuals. In diabetes, small reductions in weight can have dramatic effects on hepatic glucose production (10), although whether the same effects are beneficial in all patients is unknown. One caveat of Raeini-Sarjaz et al’s study was that no women were included; therefore, whether similar results would have been observed in women is also unknown. This point is of interest because elevated blood triacylglycerol concentrations and low HDL-cholesterol concentrations may be more significant risk factors for the development of CAD in women (11). Additionally, it would be important to confirm the results of this study in weight-stable subjects because active weight loss is associated with transient changes in blood lipids and high fatty acid availability (12).

One last, and key, issue raised in the article by Raeini-Sarjaz et al is how energy restriction will fit into treatment strategies, given the growing importance of obesity as a CAD risk factor (2). The healthy, hyperlipidemic men studied were not obese and therefore represented a group that would not necessarily be counseled to lose weight. Indeed, not all individuals with risk factors for CAD are overweight, and thus it would not be prudent to prescribe weight loss across the board. Nonetheless, if the incidence of obesity continues to increase at its current rate, so too will its contribution to the development of chronic disease. As a result, more and more patients who are overweight will attend lipid clinics seeking better weight-loss strategies for primary CAD prevention. The opportunity is great for nutrition research and food intake strategies to play key roles in formulating these solutions.

Over the past decade, the metabolic basis for the atherogenicity of elevated body weight, with accompanying insulin resistance, has been recognized (2). The article by Raeini-Sarjaz et al highlights current relations between body weight and lipid concentrations in the study of nutrition. Future modifications of guidelines for CAD treatment await the slow progress of careful research designed to clarify these controversies and to better target therapeutic strategies in individuals.

REFERENCES