COMMENTS AND RESPONSES

Monounsaturated Fat–Rich Diet Prevents Central Body Fat Distribution and Decreases Postprandial Adiponectin Expression Induced by a Carbohydrate-Rich Diet in Insulin-Resistant Subjects

Response to Walker and O'Dea

We thank Walker and O'Dea (1) for their interest in our study, wherein we have shown that when centrally overweight insulin-resistant subjects (offspring of obese and type 2 diabetic patients) are subjected to a short-term, weight-maintenance, carbohydrate-enriched diet, they redistribute their fat mass toward the abdominal fat depot (2). Walker and O'Dea (1) raised the concern that in our study, no significant differences in body weight and body fat distribution were observed when subjects were fed isocaloric diets enriched either with monounsaturated (MUFA) or high-saturated (SAT) diets. This contrasted with the observation by Walker and colleagues (3) that an isocaloric SAT-rich diet promotes more body weight gain that is predominantly deposited in the trunk than an isocaloric MUFA-rich diet.

In our opinion, this difference may be related to differences in the design of both studies. For instance, two of the eight subjects included in the Walker and colleagues (3) study had a waist circumference <0.99 cm. Also, the content of polyunsaturated fat (PUFA) present in the SAT (3%)– and MUFA (7%)–rich diets differed from our study. The changes in body weight observed by Walker et al. may also be related to the fact that the individuals following an SAT-rich diet had expended significantly more energy than those individuals following the MUFA-rich diet, probably due to differences in physical activity. In our opinion, factors such as the degree of central obesity, insulin-resistant state, level of physical activity, and PUFA content of diet could affect the body fat distribution response to the diet. Thus, we attempted to the best of our capabilities to control for these factors. Previous research by Walker et al. (4) observed this effect in type 2 diabetic patients. However, a similar effect was not observed by Clifton et al. (5) in nondiabetic patients subjected to a hypocaloric diet. Consequently, the initial phenotype of the patients and differences in the study design could be determinant to observe this phenomenon. The mechanisms explaining how specific macronutrient compositions may induce these topographic changes in fat deposition are unclear. Walker and colleagues (3) have hypothesized that MUFA and/or PUFA could be more effective at activating peroxisome proliferator–activated receptor (PPAR)-α and promoting fat oxidation. We have also shown that the rate of fat oxidation was higher with a MUFA-rich diet than that with SAT- and carbohydrate-rich diets. Finally, we speculate that preferential activation of PPAR-γ signaling could also explain at least in part the differential result in carbohydrate metabolism observed when these subjects were fed an isocaloric MUFA-rich diet (2).

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