Reply to Dr. Dickson et al.

Dear Editor,

Dickson et al. have written in response to our recent report (1) that a high-fat meal augments blood pressure and peripheral vascular reactivity to stress when compared with a meal that is lower in fat (and protein) and higher in carbohydrates. They argued that these effects were likely due to the saturated fat content of the meals because of the large body of literature linking saturated fat to postprandial endothelial dysfunction (2,3). We also hypothesized that the low-fat high-carbohydrate meal resulted in greater vasodilation because it produced a larger insulin response, although blood samples were not collected in our study. Ongoing studies will examine this hypothesis directly by matching macronutrient content of the meals more closely and assessing insulin, glucose, and lipid and/or lipoprotein response to the meals.

Dickson et al. correctly note that our test meals were not matched for protein intake and that the high-fat meal contained twice as much protein as the low-fat meal. They outline potential mechanisms through which protein might be expected to augment blood pressure reactivity. On the surface, this is a reasonable hypothesis that merits further exploration. In contrast to the large body of literature on the acute effects of saturated fat on endothelial function (2,3), very few studies have examined the acute effects of protein on systemic hemodynamics, either at rest or during stress. In response to their Letter, we found 2 previous studies (4,5) that compared the hemodynamic effects of meals containing high concentrations of protein or fat in isolation. Both showed that resting cardiac output increased postprandially and the meals containing fat vs. protein did not differ. Most relevant to this question, Uijtdehaage et al. (6) examined cardiovascular reactivity to stress after meals containing fat or protein. They also measured stress reactivity in the same subjects in the absence of meals. They found that the high-protein meal was not associated with elevated blood pressure reactivity to stress, whereas the meal containing carbohydrates significantly increased cardiac output and heart rate.

Although there are few studies testing acute effects of protein, there are several well-controlled studies showing that high-protein diets actually reduce resting blood pressure, e.g., the OmniHeart trial (7). Dickson et al. hypothesize that increased protein intake would increase blood pressure acutely, and that these changes would be mediated by increases in cardiac output via reductions in glomerular filtration rate and increases in blood viscosity. However, we found that cardiac output and stroke volume did not differ after the 2 test meals (1). Most importantly, there is evidence that peptides resulting from the degradation of dietary protein may act as inhibitors of the renin-angiotensin II system (8). This is in contrast to the proposal put forward by Dickson et al., who suggest that protein would be expected to inhibit renal function and increase production of angiotensin II. Furthermore, the higher protein content of the high-fat meal may have attenuated hyperinsulinemia, and this may explain why the high-carbohydrate low-fat meal was associated with greater vasodilation.

We agree with Dickson and colleagues that testing cardiovascular response following mixed meals does not allow for tidy conclusions about specific mechanisms of action. In fact, it is impossible to do a study in which energy is held constant while one nutrient is decreased and all others remain unchanged. However, there is clearly a need for more careful control of nutrients in postprandial studies, and the literature would benefit from direct tests of potential mechanisms of action.

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Literature Cited


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