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Reply to G Bahat and MA Karan

Dear Editor:

We thank Bahat and Karan for their thoughtful response to our recent publication (1). Our article reported 6 distinct dietary protein food clusters determined by novel food clustering methodology, previously validated and published with data from the Framingham Osteoporosis Study. Estimates of the associations between total dietary food clusters determined by novel food clustering methodology, pre-

Dear Editor:

find that although the model P value is more significant (P = 0.03, compared with other ALM models with P values of 0.11 and 0.20), the between-group differences were not significant (range: P = 0.13–0.97), which suggests that dietary protein pattern does not predict lean mass.

Bahat and Karan point out that there are multiple ways to adjust ALM for body size because of its correlation with functionality and muscle performance. We adjusted ALM for height squared, because this measurement is included in the current consensus definition of sarcopenia for the diagnosis of “low muscle mass” (2–7). All models in our published article were also adjusted for body size by including BMI (weight divided by squared height) as a covariate in the general linear models. Although we do not agree that dividing ALM by BMI is justified, we have since conducted this analysis. Overall, we found that although the model P value is more significant (P = 0.03, compared with other ALM models with P values of 0.11 and 0.20), the between-group differences were not significant (range: P = 0.13–0.97), which suggests that dietary protein pattern does not predict lean mass.

Bahat and Karan insightfully suggest that differences in BMI may be expected between the dietary protein patterns due to differing nutrient and energy composition of the protein food patterns. However, descriptive characteristics from our study show that individuals in the fast-food and full-fat dairy, fish, red meat, chicken, and low-fat milk clusters have very similar mean BMI (kg/m²), ranging from 26.5 to 27.4. Population studies, such as the Framingham Osteoporosis Study, purposely represent a wide range of independently living individuals to generalize results to the general population. Because of our large sample size (n = 2986), a range of BMIs was well represented within each of the dietary protein food patterns.

It is apparent that we share the same vision with Bahat and Karan in ensuring the deliberate use of appropriate sarcopenic guidelines in musculoskeletal research. It is important that researchers pay conscious attention to decisions encompassing variables related to sarcopenia and dynapenia, with the purpose of advancing research in this field collaboratively. With agreed-on definitions of sarcopenia and use of their variables in musculoskeletal research, results from studies can be compared effectively across study cohorts. We thank Bahat and Karan for their letter.

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