included in the original validation study. Therefore, the validity of the calculated glycemic index values is essentially unknown. However, given the relatively low correlation coefficients for quantity of total carbohydrate, starches, and sucrose, it would seem unlikely that subtle differences in the quality (i.e., glycemic index) of that carbohydrate can be accurately assessed. The authors note that correlations for carbohydrate were higher in urban non-Hispanic white subjects (<20% of the sample) and that stratification for ethnicity did not alter their conclusions. However, due to the relatively small sample size, subanalysis may not have had sufficient statistical power to detect associations.

Interestingly, observational studies that found no association between glycemic index, glycemic load, and incidence of type 2 diabetes (8,9) also had validation correlation coefficients for total carbohydrate that were <0.5. In contrast, those studies finding glycemic index, glycemic load, or both to be predictive of diabetes (5,6,10) had *r* values >0.6.

Given the limitations of the IRAS study in relation to its ability to accurately assess total carbohydrate intake, starches, and sucrose, we believe that the conclusions drawn from the study of Liese et al., i.e., that glycemic index and glycemic load are not related to measures of insulin sensitivity, insulin secretion, and adiposity, should be interpreted with caution.

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A Single Factor Underlies the Metabolic Syndrome: A Confirmatory Factor Analysis

Response to Pladevall et al.

We were pleased to see the article by Pladevall et al. (1) in a recent issue of *Diabetes Care*, as it extends our prior results to Spanish and Mauritian samples. While the authors suggest that their findings differ from our results in the Normative Aging Study (2), we wish to clarify that we had previously demonstrated that a single factor underlies components of the metabolic syndrome using confirmatory factor analysis.

In our report, we showed that 10 risk factors associated with the metabolic syndrome were predicted by one primary factor, albeit through subfactors of insulin resistance, obesity, lipids, and blood pressure. The report of Pladevall et al. and the recently published review by Kahn et al. (3) refer to our model as a "correlated-factor model" or "four-factor model." However, our analyses went well beyond a model of correlated factors to establish that the four subfactors were not only correlated but indeed predicted by a single common factor (second-order factor) (Fig. 1 in ref. 2), which we labeled the metabolic syndrome. Furthermore, it should be noted that with a second-order factor, we have found that it is possible to include the full complement of components of the metabolic syndrome in confirmatory factor analysis. For example, it is possible to include both systolic and diastolic blood pressure, consistent with current metabolic syndrome criteria, rather than mean arterial pressure alone.

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Comments and Responses

A Single Factor Underlies the Metabolic Syndrome: A Confirmatory Factor Analysis

Response to McCaffery et al.

We thank McCaffery et al. (1) for their comments on our study (2). In their first report (3), they used confirmatory factor analysis to analyze the metabolic syndrome structure and to expose the limitations of exploratory factor analysis (EFA). We agree that their results support the concept of a common factor underlying the different components of the metabolic syndrome.

What, therefore, are the differences between our studies? The one-factor model in our study was based on a critical review of previous EFAs. In our estimation, those analyses have failed to identify a single factor because they included mostly redundant measures to represent the same component of the metabolic syndrome, insuring clustering within three or four factors. Therefore, our model included only one factor and only one measure for each of the metabolic syndrome traits. On the other hand, McCaffery et al. seemed to assume that the three- to four-factor solution, from previous EFAs, was correct and proposed a hierarchical four-factor model, where each factor included more than one measure per trait, with a second-order factor reflecting the metabolic syndrome.

McCaffery's group also tested a one-factor model (Fig. 2 in ref. 3) that performed poorly in their analysis. However, our analysis (Fig. 3 in ref. 2) of their data showed that a modified version of that one-factor model, allowing correlations between error terms (residuals) for measures of the same trait, had goodness-of-fit indexes comparable to the other two models they tested (four-factor hierarchical and four-factor correlated models).

Both study groups agree that their models support the notion of a single underlying factor. We view the one-factor model as statistically more parsimonious and its interpretation more straightforward than the hierarchical four-factor model. Unfortunately, most authors of the previous EFAs, and even the recent ADA Statement by Kahn et al. (4), interpreted the presence of more than one factor as evidence against a common pathophysiological process underlying the clinical expression of the syndrome. We trust that McCaffery et al. would agree that it could be misleading to interpret the goodness-of-fit indexes of four-factor confirmatory factor analysis models, hierarchical or not, as evidence against the existence of a single factor explaining the clustering of the metabolic syndrome components.

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