THE AUTHORS REPLY

We are reassured that correspondents’ views (1–3)—that exploratory factor analysis should be treated with caution—coincide with our position (4). We do not say that factor analysis can be used to determine whether a syndrome exists, as Hanley et al. (1) suggest, although it might be implied by the poorly worded section subtitle in our paper. Our starting point was to consider each of the important questions to be answered when a new syndrome is proposed (refer to table 1 in our paper (4)). We then assessed whether or how factor analysis could be used to tackle any of these questions. It is a limitation of work in this area that so many of the published studies do not clearly state how the analysis furthers understanding of the syndrome.

Hanley et al. take our comments regarding the use of confirmatory factor analysis based on sound biologic theory out of context, because we clearly acknowledged that current pathophysiologic understanding may be insufficient to drive confirmatory factor analysis and suggested that an advance would be to confirm earlier results of exploratory analyses in new independent data sets. Although Hanley et al. believe that there is “remarkable uniformity of the more than 20 exploratory factor analysis studies on the MetS [metabolic syndrome] published thus far” (1, p. 1183), they were not able to consider the table summarizing all studies that we had identified in our literature search. We submitted this table for publication with our original paper, but it was removed during the review process. Because evidence on the consistency of findings is central to this debate, this table (Web table 1) is now posted on the Journal’s website (http://oupjournals.org). It shows that the number of factors extracted in the different studies varies from two to five, the variables included in each factor from different studies, and that the methods used in each study vary, which we believe stretches the concept of “confirmatory” rather too far.

Hanley et al. (1) are correct to point out that other highly correlated pairs of risk factors (in addition to systolic and diastolic blood pressures), such as fasting and 2-hour post-load glucose concentrations and fasting triglyceride and high density lipoprotein cholesterol levels, are frequently included in factor analyses. We agree and believe that a carefully thought out and explicit rationale for the inclusion of risk factors in any factor analysis is required. The outcomes from exploratory factor analyses reflect grouping of variables that correlate more strongly with each other than they do with other variables included in the analysis. Thus, the finding of “hypertension,” “dyslipidemia,” “insulin/glucose,” and “body size” factors in several analyses (refer to Web table 1) is not surprising, since one would predict a priori that triglycerides and high density lipoprotein cholesterol, for example, would correlate more strongly with each other than with other risk factors, and similarly with the other associations. The question then is how do such analyses help us understand the insulin resistance syndrome?

We agree with Tang et al. (3) that factor analysis might be used to produce a quantitative trait representing the syndrome that provides more power than a dichotomous trait for genetic association analyses (3). However, it should be noted that, to our knowledge, there are no published replications of such genetic associations studies, and it is possible that the potential for arbitrary changes in definitions used in factor analyses, together with strong positive associations for first genetic study publications (often with failure to replicate), will enhance publication bias and confusion in this area. The cautions we discuss in undertaking and interpreting factor analyses should be acknowledged in these genetic association studies. We thank Novak et al. (2) for identifying two further studies that were published after our paper was submitted (although before its final publication).

We agree with all correspondents (1–3) that exploratory factor analyses might be useful in generating new hypotheses, particularly with regard to the role of emerging risk factors that might be related to the syndrome. It would be useful to see new studies building on previous exploratory analyses and undertaking formal planned confirmatory analyses and, at the same time, attempting to relate these to specific biologic hypotheses. The methods suggested by Novak et al. (2) might be useful in this respect.

ACKNOWLEDGMENTS

D. A. Lawlor is funded by a United Kingdom Department of Health Career Scientist Award, and M. May is funded by the British Heart Foundation.

Conflict of interest: none declared.

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DOI: 10.1093/aje/kwi304; Advance Access publication September 8, 2005