Response to Invited Commentary

Schootman et al. Respond to “Diabetes Causality in African Americans”

Mario Schootman1, Elena M. Andresen2,3, Fredric D. Wolinsky4,5, Theodore K. Malmstrom6, J. Philip Miller7, Yan Yan8, and Douglas K. Miller9

1 Department of Medicine and Pediatrics, Washington University School of Medicine, St. Louis, MO.
2 North Florida/South Georgia Veterans Health System, Gainesville, FL.
3 Department of Epidemiology and Biostatistics, College of Public Health and Health Professions, University of Florida, Gainesville, FL.
4 Center for Research in the Implementation of Innovative Strategies in Practice, VA Iowa City Health Care System, Iowa City, IA.
5 Department of Health Management and Policy, College of Public Health, The University of Iowa, Iowa City, IA.
6 Department of Psychiatry, School of Medicine, St. Louis University, St. Louis, MO.
7 Division of Biostatistics, Washington University School of Medicine, St. Louis, MO.
8 Department of Surgery, Washington University School of Medicine, St. Louis, MO.
9 Indiana University Center for Aging Research and Regenstrief Institute, Inc., Indiana University School of Medicine, Indianapolis, IN.

Received for publication May 29, 2007; accepted for publication May 31, 2007.

We appreciate the thoughtful comments by Grant (1) on our paper (2). We agree that further exploration of the reasons for the observed association, using interdisciplinary research, is needed. We also agree that there are shortcomings of our pathways in that additional variables can be hypothesized to explain the association between housing conditions and diabetes development. Here, we briefly respond to the issues raised by Grant in his commentary.

Grant suggested that housing conditions may be a more sensitive measure of relative poverty. If this were true, the association between housing conditions and diabetes incidence would have been meaningfully altered by adjusting for perceived income adequacy in the logistic model, which it was not. Contrary to Grant’s assertions, exercise and medication adherence were included as potential mediators in our analysis; both did not explain our findings. Clearly, additional variables (e.g., eating patterns) might have better captured the given pathways, as Grant suggested.

We agree with Grant’s recommendation for greater interdisciplinary collaboration in an effort to better understand potentially mediating pathways. We further suggest not to limit this to quantitative methods but to include qualitative approaches as well. A triangulation approach, whereby quantitative and qualitative methods study the same problem, provides for a powerful means of data analysis and interpretation (3, 4).

As epidemiologists, we have often been trained to identify independent risk factors for the development of a particular disease, taking into account confounding variables. Our study used a different approach by exploring the mediating processes by which housing conditions affect diabetes development. Mediation and confounding are identical statistically and can be distinguished on only conceptual grounds (5). Moving the field of contextual effects forward will require more sophisticated approaches than controlling for confounders; it begs for examination of the causal processes by which such constructs affect disease occurrence (6). The standard analytical techniques traditionally used in observational epidemiology may not allow for full exploration of mediating pathways, especially when a more complex interplay between mediating pathways is hypothesized, as suggested by Grant.

According to Grant, one implication of our study is the potential presence of a family-level subculture. He further suggested that a reasonable next step would be to implement a home-based intervention addressing family-related attitudes...
in high-risk homes. Although ultimate certainty about the effect of family-related attitudes on development of diabetes can be obtained by a randomized intervention only, we think that this step would be premature since we did not measure this concept in our study. At this point, it is unclear whether this concept is the mediator that could explain the association observed.

Lastly, the conceptualization of neighborhood-based studies needs more attention. Developing ways to better investigate the adverse effects of neighborhood and housing conditions requires better understanding of confounders, mediators, and moderators/effect modification at the level of individual as well as at higher levels of aggregation.

ACKNOWLEDGMENTS

This research was supported by grants from the National Institutes of Health (AG10436, DK067172, DK20579).

Conflict of interest: none declared.

REFERENCES