We appreciate the comments of Bates et al. (1) on our article (2), but unfortunately they misrepresented the focus of our argument. The claim that our hypothesis requires that blacks be more likely than whites to engage in poor health behaviors in response to experienced stress ignores the substantial evidence that blacks disproportionately live in areas that have severely limited availability of fresh fruits and vegetables (3) and easy access to highly processed, high-fat/carbohydrate foods (4). These areas also have greater availability of tobacco and alcohol products (5). In those instances in which blacks and whites do live in the same environment, not only are disparities in health and
associated behaviors smaller, but whites in these places also have much worse health than whites on average (6). This means that even if whites and blacks have similar propensities to engage in these behaviors in response to stress (which we argue they do), because of structural factors, including poor-quality education, residential segregation, high unemployment rates, and poverty (7), blacks simply have more varied sources of stressors, fewer opportunities to engage in healthy coping behaviors, and greater opportunities to engage in unhealthy ones.

This unmeasured heterogeneity and confounding with race, socioeconomic status, and place makes drawing conclusions about the relative prevalence of poor health behaviors (e.g., smoking) among whites and blacks difficult (8). Examining trends in smoking over the life course rather than using lifetime estimates reveals that whites have higher prevalence of smoking in adolescence, but blacks have higher prevalence in middle age (9, 10).

It is incorrect to say that the model assumes that the biologic effect of poor health behaviors differs by race; the model only posits that the environments in which whites and blacks live and grow up are systematically different. Blacks experience more varied and concentrated stressors have have available fewer positive (and an excess of negative) coping resources. There is a robust literature on health disparities that demonstrates that this is the case (5), and we feel that this ostensible race difference is best explained as the residual concatenation of experiences that captures systematic, pervasive, life-long, and intergenerational differences in the types of environments that blacks and whites experience on a daily basis in the United States. This model predicts, and we state explicitly in the article, that if whites lived and grew up in the same areas and experienced stressors and stress similar to those experienced by blacks in the United States, the effect we observed for blacks would also be similarly present among whites.

Finally, we did confirm that life stress was linearly associated with depression risk (Figure 1), and thus had no reason to believe that the interaction we observed was an artifact of model misspecification. The failure of Keyes et al. (11) to replicate our findings could be due to idiosyncrasies in their data, especially the measurement of stress; they reported that blacks with the highest levels of stress had the same risk of depression as those with the lowest levels, which is contrary to most epidemiologic data (12). In this instance, it would be difficult to estimate whether poor health behaviors moderate the risk of depression, because Bates et al. did not report finding a direct relation between stress and depression in the first place.

In conclusion, we feel our study is an example of “white box” epidemiology (13), an instance in which we have growing experimental human and animal data that indicate that poor health behaviors influence both the biologic and psychological responses to stressors (14–17). We aim to apply these findings to the established literature on health disparities and eventually to inform efforts to promote effective and sustainable interventions for lifestyle changes in highly stressful environments.

ACKNOWLEDGMENTS
Conflict of interest: none declared.

REFERENCES


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