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THE AUTHORS REPLY

We thank Drs. Schonfeld and Bianchi for their comments (1) on our paper (2) and for opening a thoughtful discussion about ways to move the field forward. We appreciate the opportunity to continue the dialogue by responding to their comments.

We agree that it would be of interest to identify the specific association between job strain and the cortisol profile. However, the complex relationships among various stressors present practical challenges when considering them as confounders and consequently incorporating them in the models. Specifically, thinking about other stressors as confounders assumes that they cannot be affected by job-related stress—if they were, they would be mediators. Drs. Schonfeld and Biachi suggest controlling for family- and couple-related stressors, implying that family stress affects both job-related stress and cortisol. However, it is plausible that job-related stress could worsen family- and couple-related stressors (3). Hence, controlling for these sources of stress would bias the effect estimates, because family- and couple-related stressors could be mediators. We did consider financial sources of stress to be potential confounders; we assumed that financial strain could affect exposure to job strain but that it would be less likely for job strain to affect exposure to financial strain. In future research, investigators should carefully consider the role of other potential stressors, ideally in longitudinal studies that include repeated measures of stressors and therefore make it feasible to disentangle their mediating or confounding relationships.

Drs. Schonfeld and Bianchi raise another complex issue with regard to the consideration of depression in the context of our study. Again, considering depression as a confounder means that we must assume that depression does not lie on the causal pathway. However, sources of stress have been causally related to depression (4), and cortisol dysregulation could be a precipitating factor in the development or exacerbation of depression (5, 6). Controlling for depression if it is a mediator or a consequence of cortisol dysregulation would again introduce bias in estimated effects.

We agree with the authors' points about the dichotomization of job strain. Examining a continuous version of this exposure would be of interest in future research. We chose to dichotomize the variable for 2 main reasons. First, it allowed us to use well-established propensity score methods to control for a large number of confounders. Controlling for the majority of confounders in the propensity score model (rather than relying only on adjustment for them in the outcome model) was important because including a large number of confounders in our functional mixed models led to convergence problems. Second, it allowed us to limit the analysis to the area of common support, which was a major advantage because those with more job strain had a very different demographic profiles than did those with less job strain. This would be harder to do with a nonbinary treatment.

Lastly, the authors suggested that we examine job demands and job control separately. This sensitivity analysis, which showed that high job demands and low job control were each associated with lower cortisol levels, was discussed on page 502 of the original article (2). Again, we thank Drs. Schonfeld and Biachi for starting a thoughtful discussion of considerations and directions for future research in this area.

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