THE AUTHORS REPLY

In their letter to the editor, Goodman and Capitman (1) take issue with our characterization (2) of their research. Whenever we discuss the work of others, we hope to get it right. In this case, we believe that a dispassionate reader will find Goodman and Capitman’s complaints to be unjustified.

Goodman and Capitman (1) begin by taking issue with the word “inappropriate,” claiming that we used this word to describe their carefully conceived analytic strategy. What we wrote was, “if one is interested in ruling out the possibility that depression affects whether a teen takes up smoking, then it is inappropriate to control for potential indicators of depression” (2, p. 468). Obviously, this sentence hit a nerve, and therefore some elaboration is in order. We and Goodman and Capitman label a respondent as exhibiting high depressive symptomatology if he or she scores above a certain cutpoint on the Center for Epidemiologic Studies Depression (CES-D) Scale. A respondent’s CES-D score is based on answers to a series of questions. For example, respondents were asked whether “you felt that you were just as good as other people,” “you were happy,” “you enjoyed life,” and “you felt that people disliked you.” The control variables at issue are also based on self-reports, and they include “self-esteem,” “trouble relaxing,” “bad temper,” and parental perceptions of how “the teen’s life is going.” Clearly, these variables are correlated with the answers to the CES-D questions. Our concern is that this correlation occurs because both reflect the same underlying emotional state or condition. If so, then the Goodman and Capitman covariates are neither confounders nor mediators but are alternative indicators of this underlying condition. When multiple measures of the same condition are simultaneously included as explanatory variables in a regression, it should come as no surprise that the estimated effect of any single variable loses its statistical significance. Goodman and Capitman’s conclusion that “depression does not
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Dr. Aldridge’s letter to the editor (5) on our article (2) confuses the magnitude of an estimate with its precision, misrepresents our discussion of association versus causation, and contains factual errors regarding fixed-effects models. She is correct in one respect: we find that most of the association between smoking and depressive symptomatology (60–77 percent) can be explained by unobservable fixed effects (2). The remaining association is both precise (i.e., statistically significant) and small in magnitude. Moreover, we clearly state that there are reasons to believe that even this modest association may not be causal (2, p. 464). For example, if time-variant unobservables are positively correlated with both smoking behavior and depressive symptomatology, then the fixed-effects estimates will overstate the true effect of smoking on depressive symptoms. Longitudinal data contain variation both between individuals and within an individual over time. As we clearly state in our paper (2, p. 464), a fixed-effects regression identifies coefficients by using the within-individual variation, not the between-individual variation (6, p. 299). Thus, one individual is not compared with another individual, and Aldridge’s statement that the fixed-effects model assesses “whether, relative to nonsmokers, smokers display increased depressive symptomatology” (5, p. 780) is factually incorrect. Our fixed-effects estimates show the relation between within-individual changes in smoking status from baseline to follow-up and within-individual changes in CES-D score over the same period. Our sample includes nonsmokers who begin smoking, smokers who quit, and smokers who change their smoking intensity.

Including individuals with high depressive symptomatology at baseline does not bias the fixed-effects estimates. However, eliminating individuals based on the value of the dependent variable can lead to sample selection bias (6, p. 486).

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REFERENCES

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