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...
 seem to be an antecedent to heavy cigarette use among teens” (3, p. 748) is based solely on their preferred specification, which contains all of the covariates in question. Nowhere do they discuss the possibility that these covariates could themselves indicate high depressive symptomatology. Although we will leave it to the reader to decide the appropriateness of any particular covariate, we believe that the discussion of this issue is perfectly appropriate.

Next, Goodman and Capitman (1) claim that we mistakenly refer to the CES-D cutpoints as being based on the 90th percentile rather than on the work of Roberts et al. (4). In fact, we cite Roberts et al. when we write that “previous work has shown that a dichotomized version of the CES-D Scale can be used as a screening instrument for depression” (2, p. 462). Later, we note that “Goodman and Capitman investigated the impact of smoking on ‘high depressive symptomatology’ with cutpoints set at approximately the 90th percentile” (2, p. 469). We fail to see why Goodman and Capitman would take issue with a simple statement of fact. We do not dispute the validity of the cutpoints proposed by Roberts et al., and in fact we use them ourselves. If future authors wish to refer to these cutpoints as “the cutpoints used by Duncan and Rees,” we would not object.

Finally, Goodman and Capitman (1) criticize us for not including the baseline CES-D score in one of our logistic regressions, and they question what can be learned from a fixed-effects regression. These issues are related. Our interest is in understanding the role played by unobservables in the association between smoking and depression. We do not include the baseline CES-D score in the first logistic regression we present because its purpose is to estimate this association—without controlling for unobservables. The next logistic regression includes fixed effects, which control for the baseline CES-D score as well as all other baseline characteristics, unobservable and otherwise. What we learn from this exercise is that the association between smoking and depressive symptomatology is reduced by 60–77 percent after controlling for time-invariant factors. What would happen to this association if we were to control for time-variant unobservables is a question we will leave for future research.

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 non-smokers, 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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