

Duncan and Rees (1) recently reported on the association between smoking and depression among adolescents. The stated conclusion of their study is that, "for the average adolescent, the association between smoking and the symptoms of depression can in large part be attributed to the influence of unobservable factors" (1, p. 469). The authors' conclusion is untenable, given their data and statistical analysis performed on those data.

First, the fixed-effects regression analysis, designed by the authors to control for "unobservable factors," shows a strongly statistically significant effect of smoking on measures of depressive symptomatology (1). This finding suggests that there is a significant association between smoking and depression in the study sample, contrary to the authors' conclusion. Of the five regression analyses presented, the only one that shows a nonsignificant association between smoking and depression is the logistic model with fixed effects, in which depression was coded either present or absent based on a certain cutoff. Each of these models has its own drawbacks. The fixed-effects model can control for only those unobserved factors that are constant over time; therefore, changes in depressive symptomatology or smoking status that occur between the baseline and follow-up time points are not considered. Similarly, the logistic model observes depression as a yes or a no possibility only, rather than as gradient changes in depressive symptomatology.

Second, and more importantly, the data analysis is not designed to determine whether there is a causal association between smoking and depression. To determine cause, one variable must precede the other in time and show a dose-response relation. The data analysis presented by Duncan and Rees (1) assesses whether, relative to nonsmokers, smokers display increased depressive symptomatology. This design assesses individuals already smoking at the first contact relative to depression symptoms at a later time. If they are already smoking, and if smoking leads to increased depressive symptoms, then that increase in symptomatology could have conceivably already occurred. To assess a temporal relation in the data, the authors must assess differences in depressive symptomatology in individuals whose smoking status changes between the baseline and follow-up interviews. This assessment would include nonsmokers who began smoking, smokers who quit smoking, or smokers who increased the intensity of their smoking between time 1 and time 2. Even still, these analyses would show only an association between the two variables, and not necessarily cause, because no information is available to determine whether depression preceded smoking or smoking was subsequently followed by depressive symptoms.

Third, this study does not account for subjects who may be depressed, regardless of smoking behavior. Whether or not smoking leads to increased depressive symptoms, I think the authors (1) would agree that smoking is not the only possible cause of depression. Individuals with high levels of depression at baseline, whether they are smokers or nonsmokers, should be eliminated from the study sample.

The data used by the authors (1) are designed to determine whether teens who are smokers display higher depressive symptomatology. These data cannot answer the question of whether smoking causes depression, or even

whether underlying unobserved factors are responsible for the association between the two. It is possible that the authors' conclusions are correct, but these data are not able to provide that answer.

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REFERENCE

1. Duncan B, Rees DI. Effect of smoking on depressive symptomatology: a reexamination of data from the National Longitudinal Study of Adolescent Health. *Am J Epidemiol* 2005; 162:461–70.

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