In a recent issue of the Journal, Duncan and Rees (1) conclude that “reexamination” of data from the National Longitudinal Study of Adolescent Health indicates that there is no association between smoking and depressive symptoms for US adolescents. Their study was based in large part on our prior work, which identified such an association (2). Although we are not challenging the validity of their study, we do strongly challenge the way in which the authors characterize our work and their assessment of the similarities and differences between them.

Duncan and Rees (1) state that it was “inappropriate” of us to include covariates such as grade point average and previous smoking in our analyses of depression’s effect on smoking because these covariates may “indicate depression.” This criticism is unjustified and incorrect. Furthermore, it suggests that the authors are unfamiliar with some of the basic teachings of adolescent psychology, especially those related to psychosocial problems (3). We believe that these are critical potential confounders, and we included them to reduce the influence of “unobservables.” Whether our analyses demonstrate confounding or mediation depends on underlying theory and is open to interpretation. In either case, there is nothing “inappropriate” about our carefully conceived analytic strategy, which enabled readers to see how the estimate for baseline high depressive symptoms changed when covariates were added.

Duncan and Rees (1) also suggest that the Center for Epidemiologic Studies Depression (CES-D) Scale cutpoints we used to define high depressive symptoms, which they repeatedly refer to as the cutpoints of “Goodman and Capitman,” were based on 90 percent of the distribution. This is not true. As we noted in our article (2), these cutpoints were chosen because Roberts et al. (4) showed them to be the most sensitive and specific for identifying major depressive disorder in adolescents. Major depressive disorder is a psychiatric illness with substantial associated morbidity. Because the National Longitudinal Study of Adolescent Health did not contain a diagnostic interview, we could not specifically identify individuals with major depressive disorder. We used the validated cutpoints as a proxy measure for this disorder, which is conceptually quite different from depressive symptoms, the outcome in the Duncan and Rees study.

Even if the same cutpoints are used, our findings vis-à-vis smoking’s effect on high depressive symptoms differ. Many readily identifiable factors may account for this difference. Operationalization of most of the variables, including smoking, differed. We used completely different analytic samples. Our analyses were restricted to 8,704 teens who did not have high depressive symptoms at baseline. Duncan and Rees (1) included 13,068 teens for whom wave 1 data were available, which leads to a qualitatively different analysis. There is also a major oversight in their models. The “comparable” logistic regression models do not adjust for baseline CES-D scores, which provide a measure of emerging or subclinical depression. We demonstrated significant effects for baseline CES-D scores and an interaction between depressive symptomatology and smoking at baseline. The analyses by Duncan and Rees do not account for these effects, which are not time invariant.

The crux of Duncan and Rees’ concern with prior research is that it did not account for “unobservables” such as “influences related to the home environment or an individual’s genetic predisposition” (1, p. 468). We agree, but we do not think that such factors would wholly explain the smoking–depression relation. In addition, the advantage of fixed-effects models, such as those used by Duncan and Rees, is that they account for time-invariant unobservables. Because it is likely that the behavioral and environmental factors that influence depression, and their interactions with genetic predispositions, vary over time, it is not clear to us how much of an improvement the fixed-effects models provide.

Blind allegiance to randomization and the statistical techniques that approximate it do not create better science. We stand firm by our work.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

REFERENCES


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DOI: 10.1093/aje/kwj105; Advance Access publication February 22, 2006