Depression and smoking

In their study of a birth cohort (n = 1265) in Christchurch, New Zealand, Boden et al found that cigarette smoking increased the risk of depression. The cohort was 'studied' at birth, 4 months, 1 year, then annually to age 16 years, and at age 18, 21 and 25 years. At the last three assessments, the study participants were interviewed and data on depression and smoking were collected. The Composite International Diagnostic Interview (CIDI) was used to ascertain the symptoms of major depression and data on the number of cigarettes smoked and the symptoms of nicotine dependence were recorded. The authors used a variety of regression analyses to determine the causal relationship between depression and smoking, adjusted for covariates.

No matter how sophisticated the analyses are, the results of the study reflect the quality of data. The data for this study were incomplete and flawed. The data on depression and smoking were for three 12-month periods and three 1-month periods prior to the interviews. Consequently, the data on the prevalence of depression before age 17 and from age 18 to 20 and age 21 to 24 were missing. Except for three 1-month periods between age 18 and 25, all data on smoking and nicotine dependence were also missing. It is possible that some teenagers experienced depression and smoked cigarettes before age 17. It is also possible that the study participants started and quit smoking or recovered from depression between age 18 and 20, and between age 21 and 24, periods for which data were not collected. In effect, the data collected at age 18, 21 and 25 were almost cross-sectional, which cannot provide evidence for the direction of the association. If a study participant reported smoking at the age 18 interview and gave history of depression prevalent in the year prior to age 21, the authors would conclude that smoking caused depression because, according to their data, smoking preceded depression. But the authors did not know that this participant had quit smoking before the onset of depression at age 19 because they did not obtain the data for the 2 years prior to age 20. In fact, this participant's depression had been caused by smoking cessation, not by smoking.

As Munafó & Araya remarked in their editorial, the CIDI uses symptoms to determine the diagnosis of depression, not its severity. The number of cigarettes smoked is an appropriate measure of exposure to tobacco smoke, not the number of symptoms of nicotine dependence. Consequently, an association between the number of symptoms of depression and those of nicotine dependence is meaningless.

Given that tobacco smoke has anti-anxiety and antidepressant properties and that attempted or successful smoking cessation results in depression regardless of prophylactic nicotine replacement or antidepressant therapy, smoking cannot cause depression. If smoking causes depression, smoking cessation would relieve depression. The authors neglected to describe data on smokers developing depression when they quit smoking and data on antidepressant therapy during the observation period. Any study that does not use data on depression following reduction in or cessation, even transient, of tobacco smoking and data on pharmacotherapy cannot reliably determine the direction of the cause–effect relationship between smoking and depression.


Authors' reply: Dr Sheikh notes that 'it is possible that some teenagers experienced depression and smoked cigarettes before age 17'. In response we would point out that the purpose of the study was not to measure or compare the onset or first cause of either depression or cigarette smoking, but rather to examine the dynamic interplay between cigarette smoking and symptoms of depression during early adulthood, and the extent to which either cigarette smoking or depression played a causal role in the maintenance of this association across time.

He also asserts that 'the data collected... were almost cross-sectional'. This is not true. The data were discrete longitudinal data, in which both smoking and depression were assessed over several time periods. The separation of these assessments by unobserved periods was not sufficient to render the data cross-sectional.

It is also not strictly true to suggest that data observed at the same time periods could not be used to model causality. Given the availability of data observed at multiple points in time, it proves possible to fit structural equation models of the time-dynamic associations between two variables (such as cigarette smoking and depression) across time, comparing the relative fit of models that posit: (a) a reciprocal causal effect between smoking and depression; (b) a unidirectional causal effect from smoking to depression; and (c) a unidirectional causal effect from depression to smoking. Our data clearly show that the most parsimonious model is one in which there is a unidirectional causal effect from smoking to depression. This same approach has been used to examine the causal associations between numerous variables using the Christchurch Health and Development Study (CHDS) data.

Dr Sheikh argues that measures other than nicotine dependence might have led to differing results. We have in fact conducted several additional analyses using a range of measures of both cigarette smoking and depression, including: measures of smoking frequency; measures of the number of cigarettes smoked; and whether participants met criteria for DSM-IV nicotine dependence and major depression. In all cases the analyses were consistent with those reported in the original study; measures of smoking and measures of depression demonstrated significant (P < 0.05) associations using fixed-effects regression models; and the results of structural equation modelling showed...