

Letters

Why Is There a Link Between Smoking and Suicide?

TO THE EDITOR: The association between smoking and suicide is controversial, because it is unclear whether smoking influences suicide through a biological effect of smoking itself or whether there is a form of reverse causation whereby depression both makes quitting smoking more difficult and increases suicide risk. In the February issue, Balbuena and Tempier (1) reported a case-control study in which suicide decedents were compared with a control group of persons who died from accidents or homicide. This is a powerful approach that may reduce confounding (by factors related to both suicide and to mortality from accidents or homicide). The influence of proxy reporting of behaviors should be similar for both the case and the control groups.

However, it has previously been shown that smoking is related in a similar way to the risk of homicide and to the risk of suicide (2), an observation that casts doubt on a biological effect of smoking on suicide itself. Balbuena and Tempier did not report the data in such a way that the differences in smoking between the homicide and accident decedents can be easily seen. In Table 1, it is stated that the zero category for duration of smoking abstinence denotes current smokers. However, it clearly does not do so from the numbers presented, and thus it is not possible to analyze the “ever smoker” versus “never smoker” associations. Furthermore, the age differences between the homicide and accident decedents could obscure findings. However, the presented data suggest that homicide decedents were more likely to be smokers than accident decedents. It would be useful if Balbuena and Tempier would report these analyses in an unambiguous way.

In their discussion, Balbuena and Tempier refer to an improvement of mood with abstinence from smoking and a difference in depression levels between successful quitters and those who are unsuccessful, and the authors suggest that this supports a causal interpretation. However, the evidence is not convincing in this regard. Application of the principle of Mendelian randomization (3) to studies has failed to provide evidence that smoking causes depression (4,5). It would be curious if smoking cessation led to a genuine reduction in depression levels unless smoking had itself contributed to increased depression levels. Given a lack of evidence for the latter, we are therefore left with the more parsimonious explanation that the relationship between smoking cessation and reduction in depression levels is the result of reverse causation. Together with confounding this could account for the smoking and suicide link, as previously discussed (2).

REFERENCES

1. Balbuena L, Tempier R: Independent association of chronic smoking and abstinence with suicide. *Psychiatric Services* 66:186–192, 2015
2. Davey Smith G, Phillips AN, Neaton JD: Smoking as “independent” risk factor for suicide: illustration of an artifact from observational epidemiology? *Lancet* 340:709–712, 1992
3. Davey Smith G, Ebrahim S: “Mendelian randomization”: can genetic epidemiology contribute to understanding environmental determinants of disease? *International Journal of Epidemiology* 32:1–22, 2003
4. Taylor AE, Fluharty ME, Bjørngaard JH, et al: Investigating the possible causal association of smoking with depression and anxiety using Mendelian randomization meta-analysis: the CARTA consortium. *British Medical Journal*, 2014 (doi 10.1136/bmjopen-2014-006141)
5. Bjørngaard JH, Gunnell D, Elvestad MB, et al: The causal role of smoking in anxiety and depression: a Mendelian randomization analysis of the HUNT study. *Psychological Medicine* 43:711–719, 2013

George Davey Smith, M.D., D.Sc.
Marcus Munafo, M.Sc., Ph.D.

The authors are with the MRC Integrative Epidemiology Unit, University of Bristol, Bristol, United Kingdom. Prof. Davey Smith is also with the School of Social and Community Medicine, and Prof. Munafo is also with the School of Experimental Psychology, both at the University of Bristol.

Psychiatric Services 2015; 66:331; doi: 10.1176/appi.ps.660301

Why Is There a Link Between Smoking and Suicide? In Reply

IN REPLY: We welcome the critique of our study by Dr. Davey Smith and Dr. Munafo and agree that our data do not demonstrate causation. We disagree with their conclusion that the smoking-suicide link is entirely accounted for by depression or other causes. The parallel association between homicide and smoking does not explain away the relationship between suicide and smoking. Homicide decedents and suicide decedents have similar neurobiological profiles (1), which justifies our using homicide decedents as a control group. Dr. Davey Smith and Dr. Munafo correctly observe that there was an age difference between the homicide and accident decedents—with the former group being older. To address this concern, we ran regression models limited to only suicide and accident decedents, whose ages were similar. The association of smoking with suicide persisted among males in both raw and fully adjusted analyses. [A table presenting these results is available in an online data supplement to this letter.] Of interest, significant associations were also found among females in unadjusted models—something that did not show up in the results of our analysis that included homicide decedents.

The omission of nonsmokers from our analysis was intentional. This group was not included because we wished to examine a dose-response relationship (in durations of smoking and of abstinence). Including nonsmokers in this case would bias the dose-response relationship (2) because of a different case-control ratio among lifetime nonsmokers [see Table 2 in the online supplement]. An interesting question is whether among people who die violently, lifetime smoking is associated with death from suicide (rather than from homicide or accident). We performed this analysis and entered lifetime smoking and depressive symptoms as independent variables. The analysis indicated that both variables significantly predicted suicide among males [see online Table 3]. Therefore, the possibility remains that smoking makes a residual contribution to suicide, as other recent studies have indicated (3,4). The absence of a causal association between smoking and depression in studies using Mendelian randomization (5) does not contradict our result. What is implied by our study is precisely that smoking has an association with suicide independent of depression. Collectively, our results do not prove causation, but ruling it out on the basis of parsimony seems, in our opinion, to be premature.

REFERENCES

1. Breslau N, Schultz LR, Johnson EO, et al: Smoking and the risk of suicidal behavior: a prospective study of a community sample. *Archives of General Psychiatry* 62:328–334, 2005
2. Greenland S, Poole C: Interpretation and analysis of differential exposure variability and zero-exposure categories for continuous exposures. *Epidemiology* 6:326–328, 1995
3. Lucas M, O'Reilly EJ, Mirzaei F, et al: Cigarette smoking and completed suicide: results from 3 prospective cohorts of American adults. *Journal of Affective Disorders* 151:1053–1058, 2013
4. Schneider B, Lukaschek K, Baumert J, et al: Living alone, obesity, and smoking increase risk for suicide independently of depressive mood findings from the population-based MONICA/KORA Augsburg cohort study. *Journal of Affective Disorders* 152:416–421, 2014
5. Bjørngaard JH, Gunnell D, Elvestad MB, et al: The causal role of smoking in anxiety and depression: a Mendelian randomization analysis of the HUNT study. *Psychological Medicine* 43:711–719, 2013

Lloyd Balbuena, M.S., Ph.D.

Raymond Tempier, M.D., F.R.C.P.C.

Psychiatric Services 2015; 66:331–332; doi: 10.1176/appi.ps.660304

Medications for Maltreated Children: Wrong Conclusions?

TO THE EDITOR: The authors of the article about Medicaid expenditures on psychotropic medication for maltreated children in the December issue drew the wrong conclusion (1). The finding that mood stabilizers and antidepressants were overprescribed for children and adolescents with a history of abuse is most likely a result of the unavailability and underprescribing of evidence-based psychotherapy. For this reason, any cost savings from a reduction in the use of antidepressants will result in less or no treatment for this population, which is anathema to health care goals.

Instead of wasting the time of patients' psychiatrists by requiring them to conduct drug reviews—a requirement that essentially treats them as medical students—we should transfer any Medicaid cost savings from reducing drug use to psychotherapy services, start prescribing and delivering such services, and monitoring their outcomes.

REFERENCE

1. Raghavan R, Brown DS, Allaire BT, et al: Medicaid expenditures on psychotropic medication for maltreated children: a study of 36 states. *Psychiatric Services* 65:1445–1451, 2014

Kim J. Masters, M.D.

Dr. Masters is a consultant to Three Rivers Midlands Campus Residential Treatment Center, West Columbia, South Carolina.

Psychiatric Services 2015; 66:332; doi: 10.1176/appi.ps.660302

Medications for Maltreated Children: Wrong Conclusions? In Reply

IN REPLY: We respectfully disagree with Dr. Masters' characterization of our study. In the article, we merely reported the pharmacological drivers of Medicaid expenditures in our sample, and we were careful to distance our results from any language that implies clinical appropriateness. "Overprescribing" is predicated on a clinical conclusion about the quality of care. As services researchers, we cannot discuss appropriateness because we lacked clinical information about our participants that would have permitted us to make such statements. The fact that there was increased spending on antidepressants and antimanic drugs is a pharmacoeconomic fact that is based on our data; it should not be interpreted as evidence of inappropriate use of these drugs.

The fact remains that Medicaid agencies experience considerable pressure to contain their spending, and drug spending has been a target of cost containment since the 1970s. Dr. Masters is right to emphasize the need for better forms of cost containment; many cost containment efforts have indeed produced mixed results (1,2). The statement in our article about "Focusing quality improvement and prior authorization programs . . .", in context, was merely meant to suggest targets for such efforts, given that cost containment programs exist in all Medicaid programs. It was not an endorsement of any approach to cost containment. That would be a separate study.

We also agree with Dr. Masters' call to enhance the full array of biobehavioral and psychosocial interventions for vulnerable children, and we have made this exact point in prior work (3). The last thing that child mental health services researchers would wish for is a reduction in resources to serve needy children. We fully endorse the need to devote greater resources to the care of such children, and we thank Dr. Masters for drawing our collective attention to this important issue.

REFERENCES

1. Cunningham PJ: Medicaid cost containment and access to prescription drugs. *Health Affairs* 24:780–789, 2005