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ORIGINAL ARTICLE

The association between smoking and subsequent suicide-related outcomes in the National Comorbidity Survey panel sample

RC Kessler[1], G Borges[2,3], N Sampson[1], M Miller[4] and MK Nock[5]

[1]*Department of Health Care Policy, Harvard Medical School, Boston, MA, USA;* [2]*National Institute of Psychiatry, Mexico City, Mexico;* [3]*Universidad Autonoma Metropolitana, Mexico City, Mexico;* [4]*Harvard Injury Control Research Center, Harvard School of Public Health, Boston, MA, USA and* [5]*Department of Psychology, Harvard University, Cambridge, MA, USA*

Controversy exists about whether the repeatedly documented associations between smoking and subsequent suicide-related outcomes (SROs; ideation, plans, gestures and attempts) are due to unmeasured common causes or to causal effects of smoking on SROs. We address this issue by examining associations of smoking with subsequent SROs with and without controls for potential explanatory variables in the National Comorbidity Survey (NCS) panel. The latter consists of 5001 people who participated in both the 1990–2002 NCS and the 2001–2003 NCS follow-up survey. Explanatory variables include sociodemographics, potential common causes (parental history of mental–substance disorders; other respondent childhood adversities) and potential mediators (respondent history of *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn, revised mental–substance disorders). Small gross (that is, without controls) prospective associations are found between history of early-onset nicotine dependence and both subsequent suicide ideation and, among ideators, subsequent suicide plans. None of the baseline smoking measures, though, predicts subsequent suicide gestures or attempts among ideators. The smoking-ideation association largely disappear, but the association of early-onset nicotine dependence with subsequent suicide plans persists (odds ratio = 3.0), after adjustment for control variables. However, the latter association is as strong with remitted as active nicotine dependence, arguing against a direct causal effect of nicotine dependence on suicide plans. Decomposition of the control variable effects, furthermore, suggests that these effects are due to common causes more than to mediators. These results refine our understanding of the ways in which smoking is associated with later SROs and for the most part argue against the view that these associations are due to causal effects of smoking.
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Introduction

Several large prospective epidemiological studies have documented significant positive associations between smoking and subsequent suicide deaths.[1–7] Dose–response relationships have been found in some of these studies, with suicide risk higher among current than past[5] and heavy than light[2,4,5,7] smokers. These studies also have generally shown that the smoking–suicide relationship persists even after controlling for sociodemographics and alcohol consumption.[1,4–7] However, the one study that introduced more comprehensive controls for indicators of baseline psychological functioning (for example, psychiatric diagnosis, medication for emotional problems and drug use) found that the smoking–suicide relationship disappeared.[2] Although this result might be interpreted as showing that psychological distress explains the association between smoking and suicide, another possibility is that smoking is a risk factor for psychological distress, that distress mediates the effects of smoking on suicide, and that smoking does, in fact, have a causal effect on suicide. There is no way to distinguish these two interpretations with available published data.

Some insights into the predictive effect of smoking on suicide can be obtained from studies of smoking and nonfatal suicide-related outcomes (SROs), such as suicide ideation, suicide plans and nonfatal suicide attempts, although it also important to recognize that only a very small proportion of people with ideation, plans or even attempts ever die from

Correspondence: Dr RC Kessler, Department of Health Care Policy, Harvard Medical School, 180 Longwood Avenue, Boston, MA 02115, USA.
E-mail: kessler@hcp.med.harvard.edu
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suicide. Research on smoking and suicide requires very large samples that sometime have to be followed many years. It is much easier to study the relationship of smoking with more commonly occurring SROs. The latter are relevant outcomes because SROs are the immediate precursors to suicide death and are important in their own right because they are associated with substantial distress and injury.

Statistically significant associations similar to those between smoking and suicide deaths have been documented between smoking and SROs in cross-sectional[8–10] and prospective[9,11] studies in both general population samples[8–10,12] and clinical samples.[13,14] These associations have held up after controlling for sociodemographics and limited measures of psychological functioning (including measures of depression and drug use disorders). A number of other studies, though,[15–18] have found that smoking is not significantly related to SROs after controlling more comprehensively for mental disorders. However, as the temporal priorities between smoking and mental disorders were not established in these studies, it remains unclear whether this finding should be interpreted as due to confounding (that is, mental disorders causing both smoking and SROs) or mediation (that is, smoking causing mental disorders and mental disorders, in turn, causing SROs).

It is difficult to make sense of available epidemiological data on this question because smoking is a complex addictive behavior that clusters with other behavioral risk factors for suicide and SROs.[19–22] An additional complication is that basic research on smoking suggests that smoking might plausibly be both a cause and a consequence of psychopathology.[23–27] Chronic smoking, for example, has been found to reduce the sensitivity of serotonin receptors in the frontal cortex,[24] possibly promoting depression. Acute exposure to smoking, on the other hand, may have the opposite physiologic effect by raising serotonin levels in the frontal cortex,[26] providing a physiologic explanation for the hypothesis that some people with affective disorders self-medicate their dysphoria by smoking.

The current study was designed to provide new information about the epidemiology of smoking and SROs by analyzing data from a nationally representative two-wave panel survey of the US population that was interviewed originally in 1990–1992 and then again a decade later in 2001–2003. Unlike previous studies, which merely attempted to document and then explain away the prospective association between smoking and SROs by introducing controls for baseline variables, we set out to distinguish the effects of confounding and mediating variables in explaining this association. The analysis is based on a dataset that has more comprehensive measures of smoking, SROs and mental disorders than previous studies. In addition, the sample is more broadly representative and the follow-up period is longer than in most previous studies.

Materials and methods

Sample

Data come from the 5001 respondents who participated in the 1990–1992 National Comorbidity Survey (NCS)[28] and the 2001–2003 NCS follow-up survey (NCS-2).[29] The NCS[28] was a nationally representative US survey of 8098 respondents aged 15–54 years with a response rate of 82.4%. Interviews were conducted by professional survey interviewers and administered in two parts. Part I, which included the core diagnostic interview, was administered to all respondents. Part II, which included additional disorders and risk factors, was administered to a probability subsample of 5877 respondents including all respondents aged 15–24 years, all others with any lifetime *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn, revised (DSM-III-R) disorder assessed in Part I, and a random subsample of remaining Part I respondents. The Part II sample was weighted to adjust for differential probabilities of selection and for nonresponse bias. The nonresponse adjustment weight was based on the results of a brief screening survey carried out in a representative subsample of initial survey nonrespondents. Further details about the NCS design and weighting are reported elsewhere.[28]

The NCS-2 sought to trace and reinterview all Part II NCS respondents a decade after the NCS. Of the 5463 respondents successfully traced, 166 were deceased and 5001 reinterviewed, for a conditional response rate of 87.6%. The unconditional response rate was 72.2% (0.876×0.824). NCS-2 respondents were assessed using an expanded version of the baseline interview that assessed onset and course of disorders between the two surveys. Relative to other baseline NCS respondents, NCS-2 respondents were significantly more likely to be female, well educated and residents of rural areas. A propensity score adjustment weight[30] corrected for these discrepancies. Importantly, there was no difference between NCS-2 respondents and nonrespondents in their reports of SROs at the baseline assessment.[31]

Suicide-related outcomes

SROs were assessed using parallel questions in the NCS and NCS-2. At the baseline assessment, respondents were asked about lifetime experiences of suicide ideation ('Have you ever seriously thought about committing suicide?'), suicide plans ('Have you ever made a plan for committing suicide?') and suicide attempts ('Have you ever attempted suicide?'). Because self-administered surveys have been shown to yield higher rates of reporting embarrassing behaviors than interviewer-administered surveys,[32] SROs were assessed in a self-administered booklet. Respondents who reported an SRO were asked questions about age of onset and recency. Those who reported a suicide attempt were also presented with three statements and asked to identify the one that best described their experience: '(1) I made a

serious attempt to kill myself and it was only luck that I did not succeed; (2) I tried to kill myself but I knew the method was not foolproof; (3) My attempt was a cry for help, I did not want to die.' In line with contemporary distinctions between suicidal and nonsuicidal self-injury,[33,34] respondents endorsing statements 1 or 2 were considered to have made a suicide attempt, whereas respondents endorsing statement 3 were considered to have made a suicide 'gesture' (that is, potentially injurious behavior in which the intent was not to die, but to signal distress and/or to seek help from others). At the NCS-2 follow-up assessment, respondents were asked a similar series of self-administered SRO questions as at baseline, but this time focusing on the interval between the two surveys rather than over their lifetime. In analyzing these data, we distinguished between new onsets (that is, SROs at follow-up among those who denied ever having such an experience at baseline) and persistence (that is, SROs that occurred between the two surveys among respondents who also reported a lifetime history of the same experiences at baseline).

Smoking

All baseline NCS respondents were asked if they ever smoked even occasionally. Those who responded affirmatively were asked retrospectively to report the age when they had their first smoke. Recency of smoking was also assessed, with respondents who reported smoking at all in the past year being defined 'current' smokers and those with less recent smoking defined 'past' smokers. In addition, a Tobacco Supplement, in which more detailed information about history of smoking was obtained, was administered to a representative subsample of 4414 Part II NCS respondents out of the total Part II sample of 5877. The Tobacco Supplement began with a screening question that asked respondent if they ever in their life smoked daily for a month or more. Lifetime daily smokers were also assessed for DSM-III-R nicotine dependence. Age of onset and recency of daily smoking and nicotine dependence were assessed using the same approach as in the assessment of lifetime use. The NCS-2 assessment repeated these questions for respondents who were not in the baseline Tobacco Supplement.

Other risk factors for SROs

We also considered the effects of additional baseline risk factors for SROs, focusing on risk factors that have been documented in the literature as potential determinants of the relationship between smoking and SROs.[2,17,18] These included sociodemographic controls, baseline measures that might most reasonably be conceptualized as common causes of respondent smoking and SROs (parental history of common mental–substance disorders and other respondent childhood adversities), and baseline measures that might possibly be mediators of the effects of smoking on SROs (respondent lifetime of DSM-III-R mental disorders, history of substance use and history of treatment for mental–substance problems). We also controlled for baseline history of SROs to distinguish first onsets from recurrences in the intervening decade. As the results of analyses using these controls to predict the outcomes considered here have been reported previously,[31] they are not discussed in the current report.

Baseline sociodemographic controls included age, sex, race-ethnicity, marital status, employment status, religious affiliation, education, family income and having/not having a young child. Parental mental disorders were assessed with the Family History Research Diagnostic Criteria interview[35] and its extension.[36] The parental disorders included were major depression, generalized anxiety disorder, panic disorder, antisocial personality disorder and alcohol–drug dependence. Other respondent childhood adversities assessed included neglect, physical abuse, sexual abuse and childhood financial adversity. These measures were developed specifically for the baseline NCS.[37]

Baseline respondent DSM-III-R disorders included lifetime and recent (in the year before the baseline interview) mood disorders (major depressive, bipolar I and dysthymic disorders), anxiety disorders (panic disorder, agoraphobia without panic, social phobia, simple phobia, generalized anxiety disorder and posttraumatic stress disorder), substance use disorders (alcohol or other drug abuse or dependence) and antisocial spectrum disorders (conduct disorder and adult antisocial behavior). These disorders were assessed at baseline with the World Health Organization Composite International Diagnostic Interview (CIDI).[38–40] Quantity–frequency of alcohol use were assessed with both lifetime (the one year in the respondent's life when he drank the most) and 12-month questions about how many days in a typical month the respondent drank and number of drinks on days drinking. Frequency of illicit drug use was assessed with both lifetime and 12-month questions about how many days in a typical month the respondent used each of a wide variety of illicit drugs. Baseline history of treatment for emotional–substance problems was defined dichotomously in response to a series of questions about treatment for specific disorders in the CIDI.

Statistical analyses

Cross-tabulations were used to estimate the prevalence of SROs that occurred between the two surveys. Multivariate logistic regression analysis[41] was used to estimate associations of baseline smoking with the subsequent onset and persistence of SROs, with and without controls for other baseline risk factors. In order to distinguish effects in predicting suicide ideation from effects on rarer SROs that are contingent on ideation, separate equations were used to predict ideation in the total sample and then, within the subsample of ideators, to predict conditional risk of plans, gestures and attempts. A Tobacco

Supplement dummy variable (DTS) was included in the prediction equations to distinguish panel survey respondents who were in the Tobacco Supplement from other respondents. Both types of respondents were included in the initial analyses, where the baseline smoking data were treated as equivalent in both subsamples even though these data were obtained retrospectively from panel sample respondents who did not participate in the NCS Tobacco Supplement. Once final models were developed, these models were expanded to include interactions between the smoking predictors and the DTS dummy variable in order to determine if the magnitude of the associations of baseline smoking and nicotine dependence with the later SROs differs significantly in the subsamples where smoking was assessed prospectively vs retrospectively.

Logistic regression coefficients and their 95% confidence intervals (95% CIs) were exponentiated to create odds ratios (ORs) and their 95% CIs. The latter are presented here because they are easier to interpret than logistic regression coefficients. The term 'gross effects' of baseline smoking and nicotine dependence is used to refer to associations based on prediction equations that controlled only for baseline sociodemographics and baseline history of SROs. The term 'net effects,' in comparison, is used to refer to associations based on prediction equations that additionally controlled for the full set of baseline risk factors enumerated above. Continuous predictors were divided into categories to minimize the effects of extreme values, whereas some categories of predictors were combined to stabilize associations when the odds ratio (OR) did not differ meaningfully across contiguous categories. Standard errors and significance tests were estimated using the Taylor series method[42] implemented in the SUDAAN software system[43] to adjust for NCS and NCS-2 design effects. Multivariate significance was evaluated using Wald χ^2 tests based on design-corrected coefficient variance-covariance matrices. Statistical significance was evaluated using two-tailed 0.05 level tests.

Results

Onset and persistence of SROs

As reported elsewhere,[31] 13.3% of the 5001 respondents who participated in both waves of the survey reported a lifetime history of suicide ideation at baseline, whereas 4.0% reported a baseline history of a suicide plan, 2.2% a suicide gesture and 2.2% a suicide attempt (Table 1). Substantial proportions of the respondents with a baseline SRO history reported recurrences a decade later: 35.0% of those with a baseline history of suicide ideation, 21.2% of those with a history of plans, 10.8% of those with a history of gestures and 15.4% of those with a baseline history of attempts. An additional 6.2% of panel respondents reported the first onset of suicide ideation in the decade between the surveys, whereas 2.3% reported the first onset of a suicide plan, 0.7% a suicide gesture and 0.9% a suicide attempt. Combining recurrences and first onsets, a full 10.0% of respondents reported having suicide ideation in the decade between the surveys, whereas 3.0% had a suicide plan, 0.9% made a suicide gesture and 1.3% made a suicide attempt. The majority of these cases (53.7% of ideation and 71.8–73.5% of the other SROs) represent first onsets in the decade between the two surveys.

Gross associations of baseline smoking and nicotine dependence with subsequent SROs

The gross associations (odds-ratios) of baseline smoking-related predictors with subsequent SROs were found generally not to differ in predicting SRO onset and persistence (detailed results available on request). As a result, we focus here on pooled associations that combine the prediction of onset with the prediction of persistence. A total of eight separate baseline smoking-related variables were considered one at a time in models to predict subsequent SROs: lifetime smoking, age of first use, lifetime daily smoking, age of first daily use, frequency-recency of lifetime use (non-daily lifetime use, past daily use, current daily use), lifetime nicotine dependence, age of first dependence and recency of dependence. A total of 17 regression coefficients were estimated to examine the associations of these eight variables with each of the subsequent SROs (Table 2). All of these coefficients are elevated, 70% of them (12/17) significantly so at the 0.05 level, in predicting suicide ideation. A somewhat smaller proportion (88%) are elevated, 17% of them (3/17) significantly so, in predicting suicide plans. Only slightly more than half of the coefficients (58%), in comparison, are elevated in predicting suicide gestures and only a minority (29%) are elevated in predicting suicide attempts. None of the coefficients is significant in predicting either gestures or attempts.

Interactions of the DTS dummy variable with the 17 smoking-related predictors in predicting the four outcomes are significant for only 1 of the 68 coefficients, which is no more than we would expect by chance. Consistent with this result, replication of these analyses exclusively in the subsample of respondents who participated in the baseline Tobacco Supplement found the same results as in Table 2 (detailed results available on request). These findings argue against the possibility that retrospective recall bias in the subsample of respondents who did not participate in the baseline Tobacco Supplement accounts for the findings in Table 2. On the basis of these findings, we retained respondents who did not participate in the Tobacco Supplement in subsequent analyses.

A modest dose–response pattern can be seen in the aggregate predictions in two places in Table 2. First, the OR associated with lifetime dependence is somewhat higher than the OR associated with daily use, whereas the latter is somewhat higher than the OR associated with use. Second, early ages of use, daily

Table 1 The distribution of suicide-related outcomes in the NCS (T1)–NCS-2 (T2) panel sample ($n = 5001$)

	T1 lifetime prevalence[a] *(% (s.e.))*	*T1–T2 recurrence*[b] *(% (s.e.))*	*T1–T2 first onset*[c] *(% (s.e.))*	*T1–T2 period prevalence*[d] *(% (s.e.))*	*T1–T2 first onset/period prevalence*[e] *(% (s.e.))*
Ideation	13.3 (0.7)	35.0 (1.8)	6.2 (0.5)	10.0 (0.5)	53.7 (3.0)
Plan	4.0 (0.4)	21.2 (3.2)	2.3 (0.2)	3.0 (0.2)	71.8 (4.7)
Gesture	2.2 (0.3)	10.8 (0.9)	0.7 (0.2)	0.9 (0.2)	73.5 (8.1)
Attempt	2.2 (0.2)	15.4 (3.4)	0.9 (0.1)	1.3 (0.1)	72.6 (6.7)

Abbreviations: SRO, suicide-related outcome; NCS, National Comorbidity Survey.
[a]The lifetime prevalence of each SRO at baseline.
[b]The proportion of respondents with a T1 lifetime history of the SRO who reported at T2 that the same SRO occurred in the decade between the two surveys.
[c]The proportion of respondents with no T1 lifetime history of the SRO who reported at T2 that they had a first onset of the SRO in the decade between the surveys.
[d]The proportion of T2 respondents who reported having the SRO in the decade between the two surveys, whether or not they had a T1 lifetime history of the SRO.
[e]The proportion of respondents reporting SROs in the decade between the surveys that were first onsets.

use and dependence are associated with somewhat higher risks than later onsets. However, neither of these patterns is statistically significant. Furthermore, contrary to expectation, current (at the time of the baseline interview) daily use and dependence are not associated with higher risk than past daily use and dependence. Similarly modest evidence for a dose–response pattern can be seen in the prediction of suicide plans. Even this modest pattern breaks down, though, in predicting gestures and attempts.

Net associations of baseline smoking and nicotine dependence with subsequent SROs

The introduction of additional controls in the net models generally leads to a decrease in the already fairly modest associations of baseline smoking and nicotine dependence with subsequent suicide ideation (Table 3). It should be noted that even though some of the control variables are highly intercorrelated, no evidence of multicollinearity can be found using standard tests (that is, examination of the condition number of the predictor variable covariance, inspection for high negative correlations in the coefficient variance–covariance matrix and comparison of increases in the 95% CIs in the net models vs the gross models). Although the sign pattern in the net models holds up with the introduction of the additional controls (88% of the ORs remain elevated), none of these ORs is significant at the 0.05 level. Furthermore, the dose–response pattern found in the gross coefficients virtually disappears in the net models. In the case of suicide plans, in comparison, the sign pattern remains (88% of the ORs remain elevated) and the proportion of ORs that are statistically significant (17%, 3/17) is the same as in the gross models. Furthermore, some aspects of the dose–response pattern continue to hold in the net models, most notably the higher ORs associated with early than later onsets of use, daily use and dependence. Interestingly, the net coefficients are more consistently elevated than the gross coefficients in predicting suicide gestures, with 88% of the ORs elevated, although none is statistically significant and there is no evidence of a dose–response pattern in the coefficients. Only a minority of the ORs in the net models to predict suicide attempts, finally, is elevated (11%, 2/17) and none is statistically significant.

Elaboration of the classification of smoking involvement

We next created a more elaborate ordinal measure designed to describe baseline smoking history in a way that takes into consideration information contained in all eight of the measures considered above. This results in a six-category classification scheme in which the lowest category consists of respondents who never smoked and the highest of respondents with a history of early-onset use, early-onset daily use and early-onset dependence (Table 4). The two highest categories in this scheme are associated with significantly elevated ORs of ideation in the gross model. The highest category is associated with a significantly elevated OR of plans in the gross model. None of the categories, though, is significantly associated with either gestures or attempts in the gross models.

The situation is different in the net models. In the case of ideation, the significant ORs disappear entirely in the net model and no dose–response relationship can be seen in the set of ORs as a whole. In the case of plans, the significant OR associated with the highest category of involvement (3.0) is unchanged from the gross effects model. In the case of gestures, each OR associated with any of the smoking categories becomes elevated compared to the never-smoked comparison group in the net model. These ORs are in the range 1.7–2.0, but there is no dose–response relationship and none of these ORs is statistically significant. In light of this consistently elevated set of ORs, we might suspect that the association is genuine rather than due to random error. However, the pooled OR (95% CI in parentheses)

Table 2 Gross effects[a] of tobacco use and tobacco dependence at baseline NCS as predictors of suicide ideation, plans, gestures and attempts in the NCS-2

	In the total sample		*In the subsample of respondents with ideation*			
	Ideation		*Plan*		*Gesture*	*Attempt*
	%[b] *(s.e.)*	*OR*[c] *(95% CI)*	*%*[b] *(s.e.)*	*OR*[c] *(95% CI)*	*OR*[c] *(95% CI)*	*OR*[c] *(95% CI)*
Ever smoked (Y/N)	72.4 (0.02)	1.4* (1.0–1.9)	79.2 (0.02)	1.3 (0.7–2.4)	1.1 (0.4–2.8)	0.9 (0.4–1.8)
Age at first use						
Less than 12	21.4 (0.01)	1.6* (1.1–2.4)	29.9 (0.02)	1.7 (0.9–3.2)	1.2 (0.5–3.0)	1.3 (0.6–2.7)
13–15	22.3 (0.01)	1.3 (0.9–2.0)	25.2 (0.02)	1.2 (0.6–2.5)	1.4 (0.5–4.0)	0.5 (0.2–1.3)
16+	28.7 (0.01)	1.2 (0.8–1.9)	24.1 (0.02)	0.9 (0.4–1.9)	0.5 (0.1–2.1)	0.8 (0.3–2.2)
χ^{2d}_3		8.1*		6.7	4.4	6.1
Lifetime daily use (Y/N)	47.4 (0.01)	1.5* (1.1–2.0)	58.0 (0.02)	1.7* (1.0–2.7)	0.8 (0.3–2.0)	1.1 (0.5–2.3)
Age at first daily use						
Less than 16	19.3 (0.01)	1.9* (1.3–2.6)	31.7 (0.02)	2.0* (1.2–3.3)	1.1 (0.4–2.8)	1.4 (0.6–3.6)
17–19	12.0 (0.02)	1.1 (0.8–1.5)	12.8 (0.02)	1.2 (0.6–2.2)	0.6 (0.1–2.8)	0.5 (0.2–1.4)
20+	16.2 (0.02)	1.5* (1.0–2.1)	13.5 (0.02)	1.1 (0.6–2.1)	0.9 (0.3–2.5)	1.5 (0.6–3.5)
χ^{2d}_3		14.3*		8.7*	1.3	6.0
Lifetime nondaily use (Y/N)	25.8 (0.01)	1.2 (0.8–1.8)	22.9 (0.02)	0.9 (0.4–1.8)	1.4 (0.4–4.9)	0.8 (0.4–1.8)
Past daily use (Y/N)	31.3 (0.01)	1.6* (1.1–2.4)	35.2 (0.02)	1.7 (0.9–3.2)	0.9 (0.3–2.4)	0.8 (0.3–1.9)
Current daily use (Y/N)	15.3 (0.01)	1.3 (0.9–2.0)	21.1 (0.02)	1.5 (0.8–3.0)	1.1 (0.3–3.2)	1.1 (0.4–2.6)
χ^{2d}_2		7.1		6.0	0.7	1.0
Lifetime dependence (Y/N)	23.1 (0.01)	1.6* (1.1–2.2)	35.3 (0.02)	1.6 (1.0–2.5)	1.2 (0.5–2.9)	0.8 (0.4–1.7)
Age at first dependence						
Less than 20	9.3 (0.02)	1.8* (1.1–2.8)	16.7 (0.04)	2.0* (1.2–3.6)	1.9 (0.5–7.2)	0.7 (0.3–1.5)
21–29	6.2 (0.02)	1.5* (1.0–2.3)	8.4 (0.03)	1.1 (0.7–2.0)	1.6 (0.6–4.5)	0.8 (0.3–1.9)
30+	7.6 (0.02)	1.7* (1.1–2.5)	10.2 (0.02)	1.5 (0.9–2.7)	0.9 (0.4–2.0)	0.7 (0.3–2.0)
χ^{2d}_3		12.6*		7.3	2.2	0.9
Past dependence (Y/N)	9.8 (0.01)	1.7* (1.1–2.7)	12.3 (0.02)	1.3 (0.7–2.3)	0.5 (0.2–1.3)	0.8 (0.3–2.1)
Current dependence (Y/N)	13.4 (0.01)	1.5* (1.0–2.2)	23.0 (0.01)	1.7 (1.0–3.0)	1.6 (0.6–4.2)	0.8 (0.4–1.9)
χ^{2d}_2		8.8*		3.7	4.7	0.3
(n_1/n_2)[e]	(5001)	(646/5001)	(1221)	(197/1221)	(55/1221)	(84/1221)

Abbreviations: CI, confidence interval; NCS, National Comorbidity Survey; OR, odds ratio.
*Significant at the 0.05 level, two-sided test.
[a]Gross effects are based on a single multivariate logistic regression equation for each tobacco variable predicting each SRO, controlling for baseline demographics and the baseline SROs only.
[b]The percentages represent the distribution of the categories of the baseline smoking-dependence gradient in the total sample ($n = 5001$) and in the subsample of respondents who either reported suicide ideation at T1 or in the decade between the two surveys ($n = 1221$).
[c]Odds ratios from logistic regression coefficients. The 95% confidence intervals were obtained using the Taylor series linearization method.
[d]The χ^2-test evaluates the significance of each set of smoking variables predicting the outcome.
[e]n_1 is the unweighted number of respondents who experienced the SRO in the decade between the two surveys. n_2 is the unweighted number of respondents in the analysis. In the case of ideation, $n_2 = 5001$ is the number of respondents in the total sample. In the case of the other outcomes, $n_2 = 1221$ is the number of respondents who either reported having suicide ideation at T1 or in the decade between the two surveys.

of 1.8 (0.7–4.9) is not statistically significant in a model that makes a global contrast between respondents who ever smoked (that is, pooling all the smoking categories) vs those who never smoked. In the case of attempts, finally, none of the ORs in the net effects model is statistically significant and there is no dose–response pattern across the coefficients to suggest that the odds ratio of an attempt increase with higher smoking involvement.

In an effort to interpret the effects of the controls in explaining the association between early-onset dependence and subsequent ideation, we estimated two revised versions of the net effects model to predict suicide ideation. Both versions include controls for sociodemographics. One of the two also includes controls for common causes (that is, parental history of psychopathology and other respondent childhood adversities), but not for

Table 3 Net effects[a] of smoking and nicotine dependence at baseline NCS as predictors of suicide ideation, plans, gestures and attempts in the NCS-2

	In the total sample		*In the subsample of respondents with ideation*			
	Ideation		*Plan*		*Gesture*	*Attempt*
	%[b] (s.e.)	*OR[c] (95% CI)*	*%[b] (s.e.)*	*OR[c] (95% CI)*	*OR[c] (95% CI)*	*OR[c] (95% CI)*
Ever smoked (Y/N)	72.4 (0.02)	1.1 (0.8–1.6)	79.2 (0.02)	1.4 (0.7–2.6)	1.8 (0.7–4.9)	0.7 (0.3–1.4)
Age at first use						
Less than 12	21.4 (0.01)	1.2 (0.8–1.6)	29.9 (0.02)	1.7 (0.9–3.2)	2.2 (0.8–6.0)	0.8 (0.4–1.7)
13–15	22.3 (0.01)	1.1 (0.7–1.5)	25.2 (0.02)	1.4 (0.7–3.0)	2.8 (0.9–8.4)	0.6 (0.2–1.3)
16+	28.7 (0.01)	1.2 (0.8–1.8)	24.1 (0.02)	1.0 (0.5–2.3)	0.7 (0.1–3.3)	0.7 (0.2–2.2)
χ^{2d}_3		0.9		5.0	11.8*	1.9
Lifetime daily use (Y/N)	47.4 (0.01)	1.3 (0.9–1.8)	58.0 (0.02)	1.8* (1.0–2.9)	1.3 (0.4–3.6)	0.9 (0.4–2.0)
Age at first daily use						
Less than 16	19.3 (0.01)	1.4 (0.9–2.2)	31.7 (0.02)	2.0* (1.1–3.6)	1.9 (0.5–6.5)	1.2 (0.5–2.9)
17–19	12.0 (0.02)	0.9 (0.6–1.4)	12.8 (0.02)	1.3 (0.7–2.5)	1.1 (0.2–5.6)	0.4 (0.1–1.5)
20+	16.2 (0.02)	1.4 (1.0–2.1)	13.5 (0.02)	1.2 (0.6–2.5)	1.3 (0.3–5.4)	1.7 (0.7–3.8)
χ^{2d}_3		5.7		6.6	2.5	5.0
Lifetime nondaily use (Y/N)	25.8 (0.01)	1.1 (0.7–1.6)	22.9 (0.02)	1.0 (0.5–2.1)	2.1 (0.6–7.2)	0.8 (0.4–1.7)
Past daily use (Y/N)	31.3 (0.01)	1.3 (0.9–1.9)	35.2 (0.02)	1.9 (0.9–3.7)	1.5 (0.5–4.8)	0.6 (0.3–1.6)
Current daily use (Y/N)	15.3 (0.01)	1.0 (0.6–1.5)	21.1 (0.02)	1.5 (0.7–3.2)	2.1 (0.7–6.4)	0.7 (0.3–1.6)
χ^{2d}_2		2.5		5.9	3.4	1.2
Lifetime dependence (Y/N)	23.1 (0.01)	1.2 (0.9–1.7)	35.3 (0.02)	1.4 (0.9–2.4)	1.6 (0.6–4.2)	0.6 (0.3–1.2)
Age at first dependence						
Less than 20	9.3 (0.02)	1.4 (0.9–2.3)	16.7 (0.04)	2.0* (1.1–3.5)	2.5 (0.6–10.4)	0.5 (0.2–1.3)
21–29	6.2 (0.02)	1.2 (0.8–1.9)	8.4 (0.03)	1.1 (0.6–2.1)	2.9 (0.9–9.5)	0.7 (0.3–1.8)
30+	7.6 (0.02)	1.4 (0.9–2.3)	10.2 (0.02)	1.6 (0.9–2.9)	1.5 (0.4–5.3)	0.4 (0.2–1.2)
χ^{2d}_3		3.4		6.2	4.3	3.6
Past dependence (Y/N)	9.8 (0.(0.01)	1.4 (0.8–2.3)	12.3 (0.02)	1.2 (0.6–2.4)	0.6 (0.1–2.3)	0.5 (0.2–1.1)
Current dependence (Y/N)	13.4 (0.01)	1.2 (0.8–1.7)	23.0 (0.01)	1.5 (0.9–2.8)	2.4 (0.9–6.4)	0.7 (0.3–1.4)
χ^{2d}_2		1.7		2.2	5.3	3.1
$(n_1/n_2)^e$	(5001)	(646/5001)	(1221)	(197/1221)	(55/1221)	(84/1221)

Abbreviations: CI, confidence interval; OR, odds ratio.
*Significant at the 0.05 level, two-sided test.
[a]Net effects are based on a single logistic regression equation for each tobacco variable predicting each SRO, controlling for baseline demographics and the baseline SROs and number of psychiatric disorders, father and mother history of suicide or suicide attempt, father and mother history of ASP, depression, GAD, parental alcohol or drug use, childhood neglect, abuse, adverse physical, adverse finance, parental death, separation or divorce, number of drinks, number times used nine substances and having any professional treatment (lifetime).
[b]The percentages represent the distribution of the categories of the baseline smoking-dependence gradient in the total sample ($n = 5001$) and in the subsample of respondents who either reported suicide ideation at T1 or in the decade between the two surveys ($n = 1221$).
[c]Odds ratios from logistic regression coefficients. The 95% confidence intervals were obtained using the Taylor series linearization method.
[d]The χ^2-test evaluates the significance of each set of smoking variables predicting the outcome.
[e]n_1 is the unweighted number of respondents who experienced the SRO in the decade between the two surveys. n_2 is the unweighted number of respondents in the analysis. In the case of ideation, $n_2 = 5001$ is the number of respondents in the total sample. In the case of the other outcomes, $n_2 = 1221$ is the number of respondents who either reported having suicide ideation at T1 or in the decade between the two surveys.

potential mediators (that is, respondent mental–substance disorders, quantity-frequency of alcohol and drug use). The other of the two models includes controls for potential mediators, but not for common causes. The OR in the model that controls for common causes (1.8 (0.9–3.8)) is smaller than the OR in the model that controls for potential mediators (2.0 (1.1–3.6)), suggesting that common causes play a somewhat more important role than mediators in accounting for the gross association between early-onset dependence and subsequent suicide ideation.

Table 4 Gross and net effects[a] of a composite smoking-dependence gradient at baseline NCS as a predictor of suicide ideation, plans, gestures and attempts in the NCS-2

	In the total sample		*In the subsample of respondents with ideation*			
	Ideation		*Plan*		*Gesture*	*Attempt*
	%[b] *(s.e.)*	*OR*[c] *(95% CI)*	*%*[b] *(s.e.)*	*OR*[c] *(95% CI)*	*OR*[c] *(95% CI)*	*OR*[c] *(95% CI)*
I. Gross effects						
Never smoked	27.6 (0.02)	1.0 (—)	20.8 (0.02)	1.0 (—)	1.0 (—)	1.0 (—)
Use: never dependence, no early ages	30.7 (0.01)	1.1 (0.8–1.6)	22.9 (0.02)	1.1 (0.6–2.3)	1.2 (0.3–4.5)	1.1 (0.5–2.6)
Use: never dependence, early ages of use or daily use	19.0 (0.01)	1.3 (0.9–2.0)	21.5 (0.02)	1.2 (0.6–2.5)	1.1 (0.4–3.0)	0.8 (0.4–1.7)
Dependence: no early ages	6.4 (0.01)	1.4 (0.8–2.4)	6.3 (0.01)	0.5 (0.2–1.3)	0.8 (0.2–2.7)	0.5 (0.3–2.2)
Dependence: early ages of use or daily use or early age of dependence	10.7 (0.01)	1.7* (1.0–2.8)	18.0 (0.02)	1.3 (0.6–2.7)	1.1[d] (0.4–2.8)	0.8[d] (0.5–2.6)
Dependence: early ages of use and daily use and dependence	5.6 (0.00)	2.4* (1.3–4.3)	10.5 (0.01)	3.0* (1.5–5.8)*	1.1[d] (0.4–2.8)	0.8[d] (0.5–2.6)
χ^2_{4-5}[e]		10.3		25.4*	0.5	3.0
II. Net effects						
Never smoked	27.6 (0.02)	1.0 (—)	20.8 (0.02)	1.0 (—)	1.0 (—)	1.0 (—)
Use: never dependence, no early ages	30.7 (0.01)	1.1 (0.7–1.6)	22.9 (0.02)	1.3 (0.6–2.8)	1.8 (0.4–7.5)	1.2 (0.5–2.5)
Use: never dependence, early ages of use or daily use	19.0 (0.01)	1.1 (0.7–1.5)	21.5 (0.02)	1.2 (0.6–2.7)	1.7 (0.5–5.8)	0.5 (0.2–1.2)
Dependence: no early ages	6.4 (0.01)	1.1 (0.6–2.0)	6.3 (0.01)	0.5 (0.2–1.6)	1.9 (0.5–6.3)	0.5 (0.1–3.4)
Dependence: early ages of use or daily use or early age of dependence	10.7 (0.01)	1.2 (0.7–2.0)	18.0 (0.02)	1.6 (0.7–3.6)	2.0[d] (0.8–4.9)	0.5[d] (0.2–1.3)
Dependence: early ages of use and daily use and dependence	5.6 (0.00)	1.6 (0.9–3.1)	10.5 (0.01)	3.0* (1.4–6.2)	2.0[d] (0.8–4.9)	0.5[d] (0.2–1.3)
χ^2_{4-5}[e]		2.7		21.7*	2.3	6.1
(n_1/n_2)[f]	(5001)	(646/5001)	(1221)	(197/1221)	(55/1221)	(84/1221)

Abbreviations: CI, confidence interval; OR, odds ratio.
*Significant at the 0.05 level, two-sided test.
[a]Gross effects are based on a single multivariate logistic regression equation, controlling for baseline demographics and the baseline SROs. Net effects are based on a single multivariate logistic regression equation, controlling for baseline demographics and the baseline SROs and number of psychiatric disorders, father and mother history of suicide or suicide attempt, father and mother history of ASP, depression, GAD, parental alcohol or drug use, childhood neglect, abuse, adverse physical, adverse finance, parental death, separation or divorce, number of drinks, number times used nine substances and having any professional treatment (lifetime) as of the baseline interview.
[b]The percentages represent the distribution of the categories of the baseline smoking-dependence gradient in the total sample ($n = 5001$) and in the subsample of respondents who either reported suicide ideation at T1 or in the decade between the two surveys ($n = 1221$).
[c]Odds ratios from logistic regression coefficients. The 95% confidence intervals were obtained using the Taylor series linearization method.
[d]The last two categories of the predictor set were collapsed because of sparse data.
[e]The χ^2-test evaluates the significance of the full set of smoking variables predicting the outcome.
[f]n_1 is the unweighted number of respondents who experienced the SRO in the decade between the two surveys. n_2 is the unweighted number of respondents in the analysis. In the case of ideation, $n_2 = 5001$ is the number of respondents in the total sample. In the case of the other outcomes, $n_2 = 1221$ is the number of respondents who either reported having suicide ideation at T1 or in the decade between the two surveys.

In an effort to confirm the robustness of the continued significance of the association between early-onset dependence and subsequent suicide plans in the net effects model that includes all controls, this model was reestimated separately among women and men and separately in the younger and older halves of the sample. The OR of dependence predicting plans remains elevated in all these subsamples (2.3–8.3). We also reestimated the net effects model to predict plans in such a way as to distinguish early-onset

dependence that was active vs remitted at the time of the baseline interview. Both ORs are significant, but the OR for remitted dependence (3.9 (1.2–12.9)) is higher than the OR for active dependence (2.6 (1.1–6.0)), suggesting that the causal pathways linking dependence with subsequent suicide plans are for the most part not mediated by current tobacco involvement.

Discussion

Four methodological limitations need to be noted in evaluating the substantive meaning our results. First, our predictors are limited by the fact that no information was collected about smokeless tobacco use or about quantity–frequency of smoking (for example, number of cigarettes, cigars or pipes smoked per day; number of years smoked). The prospective associations found here might have been stronger if the predictors had included these refinements. Second, we did not consider the possibility that some people who make suicide plans and attempts might deny ever having suicide ideation, as the skip logic used in our surveys only assessed plans and attempts among respondents who reported a history of suicide ideation. Third, we did not control for all DSM-IV disorders. Nonaffective psychosis (NAP), for example, was not included in the core NCS-R assessment. Yet we know that both smoking[44] and suicidality[45] are comparatively common among people with NAP. Exclusion of NAP, then, presumably led to an overestimation of the net effects of smoking. Fourth, although control data on history of preexisting mental disorders and suicidality were gathered prospectively in our two-wave panel survey, the measures in each survey were based on retrospective reports. For example, the aspect of baseline tobacco use most strongly predictive of later SROs was early-onset nicotine dependence, a measure that required respondents to provide retrospective reports in the baseline survey about their age when they first experienced symptoms of dependence. Similarly, the outcome measures required respondents to make retrospective reports about the occurrence of SROs in the decade since the baseline survey. Systematic recall errors in these reports could have introduced bias into our estimates of predictive associations. Some indication that retrospective recall of smoking history is likely to be unbiased comes from our investigation of the fact that the detailed baseline assessment of smoking history was obtained only from the respondents in the Tobacco Supplement. Our finding that the associations of baseline smoking with subsequent SROs were equivalent in magnitude whether smoking was assessed prospectively or retrospectively argues against the existence of retrospective recall bias at least for this part of the assessment of smoking history.

In the context of these limitations, we were able to reproduce the same basic data pattern that has been documented in previous prospective studies of smoking and SROs: a significant time-lagged dose–response association between smoking-related variables and subsequent SROs. The associations involving suicide ideation disappeared when we introduced controls for baseline risk factors. The conditional association of the highest level of smoking involvement (lifetime nicotine dependence with early ages of onset of use, daily use and dependence) with suicide plans among ideators, in comparison, remained statistically significant even in a model that included a comprehensive set of controls.

Several previous prospective epidemiological studies found, unlike us, that significant gross prospective associations between smoking and SROs disappeared entirely after controlling for a series of risk factors.[15,17,18] The fact that we found at least one of these associations to remain, between the highest level of smoking involvement and subsequent suicide plans, might be due to the fact that the smoking variables in these earlier studies all measured use rather than nicotine dependence, whereas the significant net association in our study involved early-onset nicotine dependence. Another possible explanation for the difference between our result and the results of these earlier studies is that our finding of a significant net association is a chance finding due to the fact that we examined associations between many different measures of smoking and four different measures of SROs. Replication of our finding that early-onset nicotine dependence predicts suicide plans among ideators is needed before we can reject the hypothesis of chance association. It is also noteworthy that one other previous prospective epidemiological study found more consistent evidence than we did of statistically significant effects of smoking on SROs after controlling for risk factors.[11] This difference might be due to the fact that the controls used in that earlier study were much less complete than those used here.

By using more refined measures of both smoking and SROs than previous studies, we were able to expand our understanding of these associations by discovering that early-onset nicotine dependence is the aspect of smoking most strongly predictive of subsequent SROs and that suicide ideation and, among ideators, suicide plans, are the only SROs predicted. Importantly, neither suicide gestures among ideators nor suicide attempts among ideators were significantly predicted by any of the smoking-related variables we considered. Significant unconditional time-lagged associations of smoking-related variables with subsequent suicide gestures and suicide attempts are, in fact, present in our data due to the people who make suicide gestures and attempts being a subset of the people who have suicide ideation, but our decomposition of the SROs shows that the significant associations are actually only with ideation and plans. Previous research has shown that other predictors of ideation differ from the conditional predictors of attempts among ideators.[46] The predictive effects of smoking now have to be

conceptualized in terms of this emerging evidence of differential effects.

Although, as noted above, some previous prospective studies[15,17,18] showed, like us, that the association between smoking and subsequent suicide ideation can largely be explained by baseline risk factors, those previous studies made no attempt to distinguish the explanatory effects of common causes from the mediating effects of controls that might have been both consequences of smoking and determinants of subsequent suicide ideation. Our decomposition of these different types of effects is consequently especially useful in showing that the subset of these variables most reasonably conceptualized as common causes account for more of the explanatory effect than the variables that might, at least in part, be mediators. This finding argues against the otherwise plausible possibility that the significant gross effects documented here are due to causal associations of smoking-related predictors that are mediated by intervening mental disorders.

We found, in comparison, that our control variables did not explain the statistically significant association between early-onset nicotine dependence and subsequent suicide plans among ideators. Although this failure might be seen as indirectly arguing that nicotine dependence might have a causal effect on suicide plans, the finding that remitted nicotine dependence was as strong a predictor as active nicotine dependence is inconsistent with this interpretation. A more plausible interpretation in light of this specification is that the determinants of nicotine dependence, which are presumably indicated by respondents having either active dependence or a history of remitted dependence, rather than dependence itself are the true causal factors. Our failure to explain the association between baseline history of early-onset dependence and subsequent suicide plans, under this interpretation, might be seen as due to the fact that we did not measure the actual common causes of the two variables. It remains for future research to determine what those common causes might be, but it seems likely based on the current results that smoking is not itself of causal importance in this regard, at least in predicting SROs. It is important to reiterate the caution in the introduction, though, that the same results might not hold in predicting suicide deaths, as only a small fraction of the people who have SROs go on to complete suicides and the predictive associations of smoking with suicide deaths might go through different causal pathways than those involving SROs.

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