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**Cigarette smoking and suicidal behavior: Results from a 25-year longitudinal study**

Joseph M. Boden, PhD

David M. Fergusson, PhD

L. John Horwood, MSc

Christchurch Health and Development Study

Christchurch School of Medicine and Health Sciences

*Corresponding author:* Dr. Joseph M. Boden, Christchurch Health and Development Study,  
Christchurch School of Medicine and Health Sciences, PO Box 4345, Christchurch, New Zealand  
Phone: +64 3 372 0406 Fax: +64 3 372 0407 Email: [joseph.boden@chmeds.ac.nz](mailto:joseph.boden@chmeds.ac.nz)

## Abstract

**Background:** This study examined the associations between the frequency of cigarette smoking and both suicidal ideation and suicide attempts, both before and after control for potentially confounding factors using fixed effects regression models.

**Methods:** Data were gathered during the course of the Christchurch Health and Development Study, a 25-year longitudinal study of a birth cohort of New Zealand children (635 males, 630 females). The analysis was based on a sample of 1041 participants with available data on cigarette smoking and suicidal behaviour from ages 16 to 25 years. The main outcome measures were suicidal ideation and suicide attempts, ages 16-18, 18-21, and 21-25.

**Results:** There were significant bivariate associations between the frequency of cigarette smoking and both suicidal ideation ( $B = .24$ ,  $SE = .05$ ,  $p < .0001$ ) and suicide attempts ( $B = .30$ ,  $SE = .07$ ,  $p < .0001$ ). Control for non-observed fixed confounding factors reduced the association between cigarette smoking and suicidal ideation ( $B = -.0004$ ,  $SE = .08$ ,  $p > .90$ ) and suicide attempts ( $B = .12$ ,  $SE = .08$ ,  $p > .10$ ) to statistical non-significance.

**Conclusions:** The findings suggest that the associations between frequency of cigarette smoking and suicidal behaviour may largely be explained by the background factors and life circumstances that are associated with cigarette smoking.

**Keywords:** Cigarette smoking, suicidal behaviour, longitudinal study

In recent years, a large number of research studies have found links between cigarette smoking and suicidal behaviour, with findings suggesting that smokers are at greater risk of suicidal ideation, suicide attempt, and completed suicide (Beratis *et al.*, 1997, Breslau *et al.*, 2005, Hemmingsson and Kriebel, 2003, Iancu *et al.*, 2006, Iwasaki *et al.*, 2005, Makikyro *et al.*, 2004, Malone *et al.*, 2003, McGee *et al.*, 2005, Miller *et al.*, 2000a, Miller *et al.*, 2000b, Moriya and Hashimoto, 2005, Moriya *et al.*, 2006, Patten *et al.*, 2003, Schneider *et al.*, 2005, Shaffer, 1993, Smith *et al.*, 1992, Tanskanen *et al.*, 2000, Tanskanen *et al.*, 1998, Wu *et al.*, 2004). However, there have been ongoing debates as to whether cigarette smoking plays a causal role in suicidal behaviour (Breslau *et al.*, 2005, McGee *et al.*, 2005, Smith *et al.*, 1992). In particular, while some studies have concluded that cigarette smoking is an independent risk factor for suicidal behaviour (Breslau *et al.*, 2005, Iwasaki *et al.*, 2005, Makikyro *et al.*, 2004, Miller *et al.*, 2000a, Miller *et al.*, 2000b, Schneider *et al.*, 2005), other studies have concluded that the effects of cigarette smoking on suicidal behaviour are non-causal, and instead reflect the influence of antecedent factors associated with cigarette smoking (Hemmingsson and Kriebel, 2003, McGee *et al.*, 2005, Patten *et al.*, 2003, Smith *et al.*, 1992, Wu *et al.*, 2004).

Several epidemiological studies have recently examined the association between cigarette smoking and suicidal behaviour using both prospective designs and control for potentially confounding factors. A prospective study of 50,000 US men by Miller and colleagues (Miller *et al.*, 2000b) found a positive dose-response relationship between cigarette smoking and completed suicide, after adjustment for a number of potentially confounding factors. A further prospective study of over 300,000 US military personnel by Miller and colleagues (Miller *et al.*, 2000a) also found a persistent association between cigarette smoking and completed suicide. Similarly, Breslau *et al.* (Breslau *et al.*, 2005), in a prospective longitudinal study of young adults, reported that current daily smoking predicted both suicidal ideation and suicide attempt, even after control for confounding factors, including concurrent depression.

Other epidemiological studies, however, have failed to find support for a causal link between cigarette smoking and suicidal behaviour. For example, McGee and colleagues (McGee et al., 2005), using data from a prospective birth cohort, found that the associations between cigarette smoking and later suicidal ideation could be explained by the influence of confounding factors. Similarly, Hemmingsson et al. (Hemmingsson and Kriebel, 2003), in a study of over 49,000 Swedish men, found that the association between cigarette smoking and completed suicide was explained by the effects of both alcohol consumption and mental illness.

The results of these epidemiological studies suggest that the major issue in examining the association between cigarette smoking and suicidal behaviour is control for potentially confounding factors. In particular, it may be suggested that the apparent associations between smoking and suicidality reflect the effects of non-observed factors that are: (a) associated with the development of cigarette smoking; and (b) related to the development of suicidal behaviours. One solution to this issue is to employ methods that account for the effects of non-observed confounding factors; one such approach is using fixed effects regression models, which can be employed in longitudinal studies (Cameron and Trivedi, 1998, Greene, 1990), and have been used increasingly within psychiatric epidemiology to examine linkages between risk factors and outcomes net of common fixed sources of confounding (Fergusson *et al.*, 2006, Fergusson *et al.*, 2002).

Fixed effects models make it possible to take into account non-observed genetic and environmental factors that have a fixed effect on the association between cigarette smoking and suicidal behavior. The logic of the fixed effects model can be illustrated by the following example in which interest centers on the relationship between an exposure variable  $X$  and a continuous and normally distributed outcome  $Y$ , which are both observed at repeated time periods. Let  $X_{it}$ ,  $Y_{it}$  denote the scores on  $x$  and  $y$  of the  $i$ th individual at time  $t$ . Assume that the model linking  $Y_{it}$  and  $X_{it}$  is given by

$$Y_{it} = B_0 + B_1 X_{it} + U_i + E_{it} \quad (1)$$

where  $U_i$  represents nonobserved systematic factors that influence the outcome  $Y$  for subject  $i$  and  $E_{it}$  is a random error term. The non-observed variable  $U_i$  represents all fixed (i.e. time invariant) factors that influence the score  $Y_{it}$ . This variable thus represents all potential fixed sources of confounding that could influence the relationship between  $X_{it}$  and  $Y_{it}$ . If  $U_i$  were an observed variable it would be a straightforward matter to fit the model in Equation 1 taking into account the sources of confounding represented by  $U_i$ . Subject to the model in Equation 1 being correctly specified, this would lead to the causal effect  $B_1$  of  $X_{it}$  on  $Y_{it}$  being correctly estimated. The difficulty that arises is that  $U_i$  is not observed.

However, it proves possible to estimate the parameter  $B_1$  in the following way. First sum Equation 1 over the time periods  $t$  ( $t = 1 \dots n$ ) and divide both sides of the equation by  $n$ . This operation yields:

$$y_i = B_0 + B_1 x_i + U_i + e_i \quad (2)$$

where  $y_i$  is the mean value of  $Y_{it}$  over the  $n$  time periods,  $x_i$  is the corresponding mean value of  $X_{it}$ , and  $e_i$  is the mean value of the disturbance  $E_{it}$ . Subtracting Equation 2 from Equation 1 gives

$$(Y_{it} - y_i) = B_1(X_{it} - x_i) + (E_{it} - e_i) \quad (3)$$

The important feature of Equation 3 is that it provides a means of estimating the parameter of interest  $B_1$  in a way that takes into account the non-observed fixed sources of confounding represented by the variable  $U_i$  (this follows since Equation 3 gives an estimate of  $B_1$  but the solution of this equation does not depend on knowledge of the value of  $U_i$ ).

At this point it is useful to clarify the meaning of the fixed effect factor  $U_i$  and to consider the types of non-observed variables that might contribute to the fixed effects term. Such factors will be characterized by two general features: a) the factor will be a fixed feature of the individual or his/her social context; and b) the factor will be related to later outcomes  $Y_{it}$  by a main effects model in which the effects of the factor are constant across all observation periods.

In the context of research into cigarette smoking and later suicidal behavior, factors that may potentially be included in the fixed effects term are all individual, family, social, and related factors

that are fixed at the point of adolescence and which have a fixed effect on later cannabis use and illicit drug use. It is clear from the above that control for fixed effects does not control all confounding processes that may affect the association between  $Y_{it}$  and  $X_{it}$ . Specifically, the model does not control for: (a) fixed confounding factors whose effects on cannabis use and other illicit drug use vary with age; (b) fixed confounding factors that combine interactively with  $X_{it}$  to influence  $Y_{it}$ ; and (c) time dynamic covariate factors.

Thus while the fixed effects model succeeds in controlling some aspects of confounding, it fails to provide a comprehensive method of control. Nonetheless, it may be argued that the method represents a substantial advance over conventional covariate control using observed confounders since it does attempt to address the issue of omitted confounders.

Although the fixed effects model described in Equation 3 has been used widely in economic applications (e.g. Judge et al., 1980, chap. 8), it has been less often applied in epidemiological and public health research. One possible reason for this is that often such research uses either dichotomous outcome measures or rate information that is non-normally distributed and ill-suited to ordinary least-squares regression. However, in recent years the fixed effects model has been generalized to provide estimates for both dichotomous outcome measures and count data through the use of conditional fixed effects logistic and Poisson regression models (Hamerle and Ronning, 1995, Hausman *et al.*, 1984). Although the methods of estimation for these models differ from the methods of the linear regression model they produce estimates of the effects of an exposure variable  $X_{it}$  on an outcome variable  $Y_{it}$  adjusted for non-observed sources of confounding  $U_i$ .

Against this background, the present study reports the results of a 25-year longitudinal study of the relationships between cigarette smoking and suicidal behaviours in a birth cohort of young people studied to age 25. The aims of this analysis are to examine the associations between cigarette smoking and suicidal ideation and suicide attempt, and to adjust any associations between cigarette smoking in late adolescence and suicidal behaviour for the effects of non-observed fixed confounding factors.

## **Method**

### *Participants*

The data were gathered during the course of the Christchurch Health and Development Study (CHDS). In this study a birth cohort of 1265 children (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977 has been studied at birth, 4 months, 1 year and annually to age 16 years, and again at ages 18, 21 and 25 years (Fergusson and Horwood, 2001, Fergusson *et al.*, 1989). All study information was collected on the basis of signed and informed consent from study participants. The study had ethical approval from the Canterbury Ethics Committee.

### *Measures*

*Frequency of cigarette smoking, ages 16, 18, and 21.* At each assessment, cohort members were queried as to whether they had smoked cigarettes during the previous month, and were asked to estimate the average number of cigarettes they had smoked per day. A measure of cigarette smoking frequency at ages 16, 18, and 21 was obtained by classifying the responses according to the mean number of cigarettes smoked per day: none, less than 1 per day, 1-4 per day, 5-9 per day, 10-20 per day, and 21+ per day.

*Suicidal behaviour outcomes, ages 16-18, 18-21, and 21-25 years.* Suicidal behavior during the period 16-18 years, 18-21 years, and 21-25 years was assessed by asking sample members whether they had ever thought about killing themselves or had attempted suicide during the assessment period, and the frequency of such thoughts or attempts. Those individuals who reported having any suicidal thoughts, or who reported having attempted suicide at least once in the assessment interval were classified as having suicidal ideation (20.2% of the sample at ages 16-18; 14.2% of the

sample at ages 18-21; 12.5% of the sample at ages 21-25) or having attempted suicide (3.6% of the sample at ages 16-18; 3.7% of the sample at ages 18-21; 2.1% of the sample at ages 21-25).

### *Statistical analysis*

The analysis was conducted in three stages. In the first stage the bivariate associations between the frequency of cigarette smoking at ages 16, 18, and 21 (classified into six levels – none, < 1/day, 1-4/day, 5-9/day, 10-20/day, and 21+/day) and suicidal ideation and suicide attempt during the assessment period following the measure of smoking (e.g. the association between smoking at age 16 and suicidal behaviour at ages 16-18) were tested for significance using the Mantel-Haenszel chi squared test of linearity for suicidal ideation and Poisson regression for suicide attempts.

The second stage of the analysis involved fitting regression models to the three waves of assessment data to estimate the joint associations between frequency of cigarette smoking and rates of subsequent suicidal behaviour pooled over the assessment periods. These associations were modeled by fitting random effects models (Gibbons et al., 1988) to the three waves of assessment data for the period from 16-25 years. For suicidal ideation logistic regression models were fitted, whereas for number of suicide attempts Poisson regression models were used. These models were of the general form:

$$G(Y_{it}) = B_{0t} + B_1 X_{it} + v_i + e_{it} \quad (\text{EQ1})$$

where  $Y_{it}$  was the outcome (ideation or number of attempts) for participant  $i$  in the  $t$ -th age interval (16-18, 18-21, 21-25 years); the function  $G(Y_{it})$  represented either the log odds of suicidal ideation or the log rate of suicide attempt respectively;  $X_{it}$  was the frequency of smoking for participant  $i$  immediately prior to interval  $t$ ;  $v_i$  was an individual specific random effect that was uncorrelated with smoking ( $X_{it}$ ); and  $e_{it}$  was a random error term. In these models the coefficient  $B_1$  represents the effect of smoking on suicidal behaviour pooled over the three observation periods. The intercept term  $B_{0t}$  was permitted to vary with age  $t$  to take into account changes in the base rate of suicidal behaviour over time. From the coefficients of these models, estimates of the effect size for

the associations between cigarette smoking and suicidal behaviour were calculated for varying levels of smoking behaviour. For suicidal ideation, these estimates were given by the odds ratio (OR), whereas for the count measure of suicide attempts, these estimates were given by the incidence rate ratio (IRR).

The third stage of the analysis, adjustment for non-observed fixed factors, involved fitting conditional fixed effects logistic and Poisson regression models (Cameron and Trivedi, 1998, Greene, 1990) respectively to the observed rate data for suicidal ideation and suicide attempt. As described in the Introduction, fixed effects models permit control for the effects of all non-observed fixed confounding factors that influence the outcome variable (suicidal behaviour) and may be confounded with the exposure variable (cigarette smoking). In this stage, the fixed effects models fitted were of the form:

$$G(Y_{it}) = B_{0t} + B_1 X_{it} + u_i + e_{it} \quad (\text{EQ2})$$

This model is of similar form to that in EQ1 above. However, in this instance the term  $u_i$  represents the effects of (non-observed) fixed confounding factors specific to the  $i$ -th individual that are correlated with smoking ( $X_{it}$ ) and that may also influence suicidal behaviour. The coefficient  $B_1$  represents the effects of smoking ( $X_{it}$ ) on the outcome ( $Y_{it}$ ) net of non-observed fixed confounding factors. In addition, the adjusted model parameters were used to calculate adjusted estimates of effect size for varying levels of smoking behaviour.

### *Sample Size and Sample Bias*

The present analysis is based upon a sample of 1041 participants for whom data were available on cigarette smoking and subsequent suicidal behaviours for at least one of the observation periods 16-18, 18-21 or 21-25 years. However, since not all participants were observed on all assessment occasions, the observed samples with complete data for each assessment interval ranged from 935-983. These samples represented between 74% to 78% of the initial cohort of 1265 children. To examine the effects of sample losses on the representativeness of the sample, the obtained sample

was compared with the remaining sample members on a series of socio-demographic measures collected at birth. This analysis suggested that there were statistically significant ( $p < .01$ ) tendencies for the obtained sample to under-represent individuals from disadvantaged backgrounds characterized by low parental education, low socioeconomic status and single parenthood. To address this issue, the data weighting methods described by Carlin et al. (1999) were used to examine the possible implications of selection effects arising from the pattern of missing data. These analyses produced essentially the same pattern of results to those reported here, suggesting that the conclusions of this study were unlikely to have been influenced by selection bias.

## **Results**

### *Associations between frequency of cigarette smoking, ages 16, 18, and 21, and suicidal behaviour ages 16-18, 18-21, and 21-25*

Table 1 shows the associations between the frequency of cigarette smoking at ages 16, 18 and 21 years and rates of subsequent suicidal behaviours in the intervals 16-18, 18-21 and 21-25 years respectively. In each case the frequency of cigarette smoking is classified into six groups ranging from non-smoker to those who smoked 21+ cigarettes/day. For each group the Table reports the rate of suicidal ideation and suicide attempts during the subsequent assessment period. The individual associations have been tested for linearity using the Mantel-Haenszel chi squared test for measures of suicidal ideation and Poisson regression for rates of suicide attempt. In all cases, the Table shows evidence of clear and significant tendencies for increasing frequency of smoking to be associated with increasing levels of suicidal ideation ( $p < .05$ ) and increasing rates of suicide attempt ( $p < .0001$ ).

To examine the joint associations between frequency of cigarette smoking and rates of suicidal behaviour pooled over the three assessment periods the data in Table 1 were reanalyzed by fitting random effects regression models to the three waves of data (see Methods). For suicidal ideation a random effects logistic regression model was fitted and for suicide attempt a random effects Poisson regression model was used. Consistent with the findings in Table 1 these analyses

produced evidence of significant associations between frequency of smoking and both suicidal ideation ( $B = .24$ ,  $SE = .05$ ,  $p < .0001$ ) and suicide attempt ( $B = .30$ ,  $SE = .07$ ,  $p < .0001$ ). From the parameters of the fitted models, effect size estimates were obtained describing the relative rate of suicidal behaviours for varying levels of smoking compared to the rate for non-smokers, pooled over the three observation periods. These estimates are given at the foot of Table 1. For suicidal ideation effect sizes were based on the odds ratio (OR), for suicide attempt the effect size measure was the incidence rate ratio (IRR). The pooled effect size estimates suggest evidence of moderate to strong associations between smoking and suicidal behaviours, with individuals who smoked more than a pack of cigarettes per day having odds of suicidal ideation that were 3.39 (95%CI: 2.06 to 5.59) times higher and rates of suicide attempt that were 4.39 (95%CI: 2.18 to 8.85) times higher than those who did not smoke.

INSERT TABLE 1 HERE

#### *Adjustment for fixed factors*

A possible limitation to the results presented in Table 1 is the possibility that the associations between frequency of cigarette smoking and suicidal behaviour could be due to confounding factors that were related to both cigarette smoking and suicidal behaviour. To address this issue, the associations presented in Table 1 were adjusted using fixed effects regression models (see Methods). These analyses revealed that the associations between cigarette smoking and both suicidal ideation and suicide attempts had been greatly reduced in magnitude, to the point of statistical non-significance (suicidal ideation,  $B = -.0004$ ,  $SE = .08$ ,  $p > .90$ ; suicide attempts,  $B = .12$ ,  $SE = .08$ ,  $p > .10$ ). Table 2 shows the OR for suicidal ideation and the IRR for suicide attempts, for varying levels of smoking behaviour, after adjustment for fixed confounding factors. The Table shows that, after controlling for fixed effects, those individuals who smoked more than 20 cigarettes per day had odds of suicidal ideation that were 1.00 (95% CI: 0.46 to 2.18) times that

of those who did not smoke cigarettes, and rates of suicide attempt that were 1.84 (95% CI: 0.81 to 4.18) times higher than those who did not smoke cigarettes.

INSERT TABLE 2 HERE

The results of these analyses show that the association between cigarette smoking and suicidal ideation can be explained by controlling for fixed confounding factors. In addition, after controlling for fixed confounding factors, there remained a modest, but statistically non-significant association between cigarette smoking and suicide attempt. Collectively, the results of these analyses suggest that the associations between cigarette smoking and subsequent suicidal behaviour were largely, if not wholly, explained by the influence of fixed confounding factors that are correlated with both cigarette smoking and suicidal behaviour.

## **Discussion**

In this paper we have used data gathered over the course of a 25-year longitudinal study to examine the linkages between frequency of cigarette smoking and suicidal behaviour (ages 16-25). The principal focus of this analysis was on ascertaining the extent to which cigarette smoking increased the risks of both suicidal ideation and suicide attempt. The findings of this analysis and their implications are outlined below.

In agreement with a broad range of research examining links between cigarette smoking and suicidal behaviour (Beratis *et al.*, 1997, Breslau *et al.*, 2005, Hemmingsson and Kriebel, 2003, Iancu *et al.*, 2006, Iwasaki *et al.*, 2005, Makikyro *et al.*, 2004, Malone *et al.*, 2003, McGee *et al.*, 2005, Miller *et al.*, 2000a, Miller *et al.*, 2000b, Moriya and Hashimoto, 2005, Moriya *et al.*, 2006, Patten *et al.*, 2003, Schneider *et al.*, 2005, Shaffer, 1993, Smith *et al.*, 1992, Tanskanen *et al.*, 2000, Tanskanen *et al.*, 1998, Wu *et al.*, 2004), we found a consistent bivariate association between increasing frequency of cigarette smoking and increasing rates of suicidal behaviour, including both

suicidal ideation and suicide attempts. Individuals who smoked 20 or more cigarettes per day had odds of suicidal ideation that were more than three times greater than those who were non-smokers, and had rates of suicide attempt that were more than four times greater than those of non-smokers.

Further analyses suggested that the linkages between cigarette smoking and suicidal behaviour were likely due to the effects of confounding factors that were associated with cigarette smoking. The use of fixed effects regression models reduced substantially the magnitude of the observed associations. After adjustment for fixed effects, individuals who smoked 20 or more cigarettes per day had odds of suicidal ideation that were no greater than those of non-smokers, and had rates of suicide attempt that were 1.8 times those of non-smokers. In both cases the adjusted associations were statistically non-significant. These results suggest that the association between cigarette smoking and suicidal behaviour was largely if not wholly explained by non-observed, fixed confounding factors that were correlated with cigarette smoking, and both suicidal ideation and suicide attempt.

As noted earlier, there have been ongoing debates in the literature as to whether cigarette smoking plays a causal role in increasing risks of suicidal behaviour (Breslau *et al.*, 2005, McGee *et al.*, 2005, Smith *et al.*, 1992). The results of the present study would suggest that cigarette smoking does not play a causal role *per se* in increasing risks of suicidal behaviour. The weight of the evidence would suggest that, for this cohort, cigarette smoking served as a marker for future suicidal ideation and suicide attempts, and that when unobserved confounding factors were taken into account, cigarette smoking no longer predicted suicidal behaviour. This evidence is congruent with a range of research findings that suggest that the risk of suicidal behaviour is increased by a constellation of background factors that are also related to increasing frequency of cigarette smoking (Beautrais *et al.*, 1996, Heikkinen *et al.*, 1995, Vilhjalmsson *et al.*, 1998).

The clinical implications of the current study are that, while cigarette smoking may not be a discrete cause of suicidal behaviour, the bivariate associations between smoking and suicidal behaviour suggest that the risk of suicidal behaviour is elevated in individuals who smoke. The

results of the current study suggest that clinicians should interpret cigarette smoking in patients as an indicator of increased risk of suicidal behaviour.

These conclusions need to be considered in the light of possible limitations of the study. These limitations include the fact that the study was based on a specific cohort studied in a specific social context; and that the measures of cigarette smoking frequency and suicidal behaviours were obtained via self-report. As such these variables may be subject to errors of measurement that may compromise the estimation of model parameters. In addition, the results of the current study are applicable only to suicidal ideation and suicide attempt, but not to completed suicide. A number of previous studies have found large and persistent associations between cigarette smoking and completed suicide (e.g. Miller *et al.*, 2000a, Miller *et al.*, 2000b); however, given the small number of completed suicides in the present cohort (5), the current study was unable to address this issue.

Notwithstanding these potential limitations, the results of the present study suggest that, while cigarette smoking may serve as a marker for increased risk of suicidal ideation and suicide attempt, these associations largely reflected background factors and current life circumstances that were associated with increases in cigarette smoking, rather than the direct effects of smoking on suicidal behaviour.

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Table 1. Association between frequency of cigarette smoking, ages 16, 18, and 21 years, and suicidal behaviour, ages 16-18, 18-21, and 21-25 years.

	Frequency of Cigarette Smoking at Given Age						p <sup>1</sup>
	None	<1/day	1-4/day	5-9/day	10-20/day	21+/day	
<u>Age 16</u>							
<u>n</u>	634	150	92	36	21	2	
% Suicidal ideation ages 16-18	15.3	20.7	32.6	41.7	66.7	50.0	<.0001
Mean number of suicide attempts (per 100) ages 16-18	2.1	10.7	18.5	36.1	33.3	50.0	<.0001
<u>Age 18</u>							
<u>n</u>	603	98	79	88	96	19	
% Suicidal ideation ages 18-21	12.4	13.3	11.4	20.5	12.5	47.4	<.05
Mean number of suicide attempts (per 100) ages 18-21	3.5	8.2	5.1	14.8	17.7	10.5	<.001
<u>Age 21</u>							
<u>n</u>	589	35	74	108	151	21	
% Suicidal ideation ages 21-25	9.0	8.6	16.2	13.9	19.9	28.6	<.0001
Mean number of suicide attempts (per 100) ages 21-25	1.7	0.0	0.0	2.8	12.6	28.6	<.0001

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Effect Size Estimates

OR (95% CI) for suicidal ideation	1	1.28 (1.16-1.41)	1.63 (1.34-1.99)	2.08 (1.54-2.81)	2.17 (1.78-3.96)	3.39 (2.06-5.59)
IRR (95% CI) for suicide attempts	1	1.34 (1.17-1.55)	1.81 (1.37-2.39)	2.43 (1.60-3.70)	3.26 (1.86-5.72)	4.39 (2.18-8.85)

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<sup>1</sup> Mantel-Haenszel  $\chi^2$  test of linearity for suicidal ideation: Poisson regression for suicide attempts.

Table 2. Effects size estimates for the associations between frequency of cigarette smoking and suicidal behaviour, after adjustment for fixed effects.

	Frequency of Cigarette Smoking at Given Age					
	None	<1/day	1-4/day	5-9/day	10-20/day	21+/day
OR (95% CI) for suicidal ideation	1	1.00 (0.85-1.17)	1.00 (0.73-1.37)	1.00 (0.62-1.60)	1.00 (0.53-1.87)	1.00 (0.46-2.18)
IRR (95% CI) for suicide attempts	1	1.13 (0.96-1.33)	1.28 (0.92-1.77)	1.44 (0.88-2.36)	1.63 (0.84-3.15)	1.84 (0.81-4.18)