The relationship of tobacco smoking with depressive symptomatology in the Third Mexican National Addictions Survey

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ABSTRACT

Background. While an association between cigarette smoking and depression has been established in Anglo populations, replication of tobacco–depression associations in countries where smoking is growing may provide important new insights. The objectives of this study were to estimate the association of depressive symptomatology with tobacco smoking, number of cigarettes smoked daily, and smoking cessation in a representative sample of the Mexican population.

Method. The data come from the Third National Addictions Survey (1998) conducted by the Mexican Ministry of Health, representative of Mexico's civilian population residing in cities and towns with 2500+ inhabitants, aged 18–64. Part of a multi-stage, stratified, probability sample, 1935 men and women answered a version of the survey that also included the CES-D depression scale. Analyses addressed the survey's complex design and controlled for income and educational level.

Results. Among women only, current smokers had twice the odds of elevated depressive symptomatology than never smokers (OR 2·1, 95% CI 1·3–3·5, $p = 0·002$). For men, only those smoking a pack or more a day had greater odds of depressive symptomatology (OR 5·9, 95% CI 1·6–21·9, $p = 0·008$). Overall, former smokers who ceased smoking within 6 months had lower odds of depressive symptomatology than current smokers (OR 0·4, 95% CI 0·1–1·0, $p = 0·042$).

Conclusions. These findings add to the accumulating evidence for the association between smoking and depression in different cultures and populations.

INTRODUCTION

Both cigarette smoking and depression are prevalent and costly public health problems. Worldwide, four million deaths were attributed to tobacco use in 1998 and the number is estimated to rise up to 10 million a year by 2030, over 70% of those in the developing world (WHO, 1999). In Mexico, there were an estimated 13 million smokers as of 1998 (Tapia *et al.* 2000*b*) with 1 in 10 deaths attributable to tobacco use (Kuri *et al.* 2000). Depression, though not as great a cause of mortality as smoking, is costly in terms of suffering, morbidity, social impact and the global burden of disease (Wagner *et al.* 1999). In fact, the World Health Organization and the World Bank calculate that unipolar depression is the fourth ranking factor in disease burden and projected to rise to second place by 2020, first place in developing nations (Murray & Lopez, 1997). In Mexico City, the prevalence of any lifetime affective disorder is estimated to be 9·2% (WHO International Consortium in Psychiatric Epidemiology, 2000).

An association between these two important health problems, tobacco smoking and depression, has been well-established in Anglo

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populations (Anda *et al.* 1990; Glassman *et al.* 1990; Kendler *et al.* 1993; Covey *et al.* 1998). Among samples in the USA, there is good evidence that those who smoke are more likely to develop depression (Breslau *et al.* 1993; Wu & Anthony, 1999), that those who are depressed (or experience depressive symptomatology) are more likely to initiate smoking (Escobedo *et al.* 1998; Patton *et al.* 1998), progress to daily smoking (Breslau *et al.* 1998) or nicotine dependence (Breslau *et al.* 1993), have less success at smoking cessation (Anda *et al.* 1990; Glassman *et al.* 1990) and experience more severe withdraw symptoms when having stopped smoking (Covey *et al.* 1990). Additionally, young people who quit smoking appear to experience less depressive severity (Martini *et al.* 2002). There is also evidence for US Latinos (e.g. Pérez-Stable *et al.* 1990*a*), however, these associations have not yet been studied in the Mexican population.

Understanding the relationship between tobacco and depression is important because of the many implications for prevention and treatment. While analyzes to address these issues have been conducted elsewhere, replication of tobacco-depression associations in countries where smoking is growing and where there is less legislation restricting smoking in certain places or banning tobacco advertisements, may provide important new insights regarding this relationship (Martini *et al.* 2002). In addition, characterizing these relationships for Mexican samples is a first step in elucidating these processes for a deeper insight in that country. In this study, our purpose is to describe this relationship between smoking tobacco and depressive symptomatology in a representative sample of the Mexican adult population. Specifically, to estimate the odds of depressive symptomatology associated with smoking status, number of cigarettes smoked daily, and among former smokers, the time lapsed since smoking cessation.

METHOD

The data for this study came from the Third National Addictions Survey (ENA for its initials in Spanish) conducted in 1998 by the Health Ministry to update information on the prevalence of substance use and abuse. This survey was part of a series of National Household Surveys performed in Mexico first in 1988 and again in 1993 and 1998. More detailed descriptions of these surveys have been published elsewhere (Rosovsky *et al.* 1999; Kuri *et al.* 2000; Tapia *et al.* 2000*a*).

For the ENA, three versions of the questionnaire forms were used, each containing a large common core group of questions (including demographics and substance, tobacco and alcohol use/abuse/dependency) and one of three small sets of specific questions limited to that form. The core group of questions had been extensively tested in Mexico since 1974 and were included in the previous two national surveys. The 'A' form, given to every third participant included additional scales of associated problems, such as depressive symptomatology. Respondents were unaware of which version was applied. The following description of participants and measures is restricted solely to this sub-sample.

Population and participants

The estimated population of Mexico in 1998 was about 94 938 220. The survey was designed to yield valid data on national and regional drug use prevalence estimates and covariates of the civilian population aged 18–64 years, residing in cities and towns with 2500 or more inhabitants (so-called urban population). It is estimated that about 75% of the Mexican population lived in urban areas. Part of a multistage, stratified probability sample, 1935 men and women answered the 'A' version of the ENA survey.

Measures
Tobacco smoking

Items assessed smoking status, number of cigarettes smoked per day and smoking cessation. We defined smoking status as never-smoker (has never smoked or has smoked less than 100 cigarettes in their lifetime), former smoker (has smoked more than 100 cigarettes, but not in the previous 30 days), and current smoker (has smoked in the previous 30 days).

Depressive symptomatology

A Spanish-language version of the Center for Epidemiological Studies Depression Scale

(CES-D; Radloff, 1977) measured depressive symptomatology. The Spanish-language version of the scale has been evaluated and utilized in various studies with the Mexican adult population (Mondragón *et al.* 2001). Twenty Likert-type items assessed the frequency of depressive symptomatology in the previous week, including depressed affect, feelings of guilt and worthlessness, psychomotor retardation, loss of appetite and sleep difficulties. The continuous score from the scale was converted to a dichotomous measurement of low or elevated depressive symptomatology. Participants were classified as experiencing elevated depressive symptomatology if their score fell one standard deviation or more above the mean for this sample (mean $= 14.9$, s.d. $= 6.8$).

Procedures

Four stages of selection were employed. Primary selection units were census count areas (or groups of them) with an average size of 3000–4000 persons of all ages, secondary selection units were city blocks (or groups of them), tertiary selection units were households. Once a household was selected, all eligible household members were listed and a systematic sampling scheme applied to produce a self-weighting sample of respondents. All adults (aged 18–64 years) were listed in age order and selected according to a weighted random selection procedure using Kish (1965) tables. Informed consent was obtained from each participant. The data were collected through confidential, face-to-face interviews, conducted in privacy at the participant's home by a trained, lay interviewer. The interviewer read each question to the participant and recorded his or her responses. There were 13 288 households visited, from which first contact was obtained for 6523 adults (contact rate $= 90.4\%$). Non-response at the household level included 'no one at home' (3.4% of the total visits, $n = 13\,288$), and 'refusal to answer' (3.3%). From the targeted 6523 adults (aged 18–65), there were 5712 completed individual interviews and 811 non-response (individual participation response rate of 87.5%). Reasons for individual non-response was 'refusal to answer' (7.2% of the total targeted adults, $n = 6523$), 'temporarily absent from home' (2.7%), 'inadequate informant' (1.0%), 'no one home' (0.5%), 'incomplete interview'

(0.1%) and 'others' (1.0%). Approximately equal numbers of each of the three versions of the instrument were distributed and returned.

Analyses

We first computed the demographic and tobacco use characteristics of the sample. The strength of the association between depressive symptomatology and the three smoking characteristics was estimated using logistic regression (Hosmer & Lemeshow, 2000). More specifically, a logistic regression model was used to estimate the association between depressive symptomatology and smoking status (never, current or former smoker) controlling for sex, income and educational level. Subsequently, this analysis was performed for men and women separately to evaluate the possible modification of effect. In order to estimate the odds of depression associated with number of cigarettes smoked daily, another logistic regression was performed to compare the odds ratios (OR) of those who smoked a pack or more a day, those smoking half a pack, those smoking less than half a pack, and former smokers to never smokers. Logistic regression models also estimated the association of time since smoking cessation with depressive symptomatology, holding constant sex, income and education. This analysis could not be performed separately for men and women due to the small number of participants in certain groups. Finally, to accommodate the unequal probability of selection for the survey across respondents, as well as the clustering by sampling units and the stratification design of the survey, the STATA 7.0 *svy* procedure, which is a procedure to adjust for design effects, was used in our analyzes. This procedure generates standard errors of proportions and standard errors of odds ratios estimated using the Taylor series linearization method that takes into account the weights of the observations, the stratification of the country and the clustering of the survey (StataCorp, 2001).

RESULTS

Table 1 presents the weighted sample characteristics for those classified with low depressive symptomatology, elevated depressive symptomatology and the sample as a whole. The sample comprised 58.5% women and 41.5%

Table 1. *Weighted sample characteristics by low and elevated depressive symptomatology*

| | Depressive symptomatology | | | | | | | | | |
| | Low | | | Elevated | | | Total | | | |
	n*	%	S.E.	n	%	S.E.	n	%	χ^2	p
Sex									49·6	0·001
Male	761	92·3	1·2	54	7·7	1·2	815	100		
Female	922	80·1	1·4	198	19·1	1·4	1120	100		
Age (years)									2·9	0·569
18–29	710	84·7	1·5	113	15·3	1·5	823	100		
30–39	411	84·7	2·0	68	15·3	2·0	479	100		
40–49	316	86·9	2·2	41	13·1	2·2	357	100		
50–64	246	88·2	2·3	30	11·8	2·3	276	100		
Education									8·8	0·257
No formal	85	81·8	4·9	17	18·2	4·9	102	100		
Primary	576	83·7	1·6	111	16·3	1·6	687	100		
Middle	477	85·6	1·9	60	14·4	1·9	537	100		
High/vocational	324	89·5	1·7	40	10·5	1·7	364	100		
University	221	86·1	2·9	24	14·0	2·9	245	100		
Income (in increments of minimum wage)									21·3	0·005
<min. wage	84	75·3	4·7	26	24·7	4·7	110	100		
min. wage	133	84·1	3·3	26	15·9	3·3	159	100		
<2 min. wages	536	84·1	1·7	94	15·9	1·7	630	100		
<4 min. wages	552	86·3	1·6	77	13·7	1·6	629	100		
5+ min. wages	316	91·9	1·9	21	8·1	1·9	337	100		
Smoking status									0·1	0·964
Non-smoker	896	85·8	1·3	128	14·2	1·3	1024	100		
Ex-smoker	369	85·1	2·1	50	14·9	2·1	419	100		
Active smoker	418	85·6	1·9	74	14·4	1·9	492	100		
No. of cigarettes per day									0·2	0·906
1–5 cigarettes	306	86·0	2·2	55	14·0	2·2	361	100		
½ pack: 6–15 cigs	74	86·0	4·4	12	14·0	4·4	86	100		
1+ packs: 16+ cigs	36	83·0	6·6	6	17·0	6·6	42	100		
Time since smoking cessation									9·2	0·249
Non-smoker	896	85·8	1·3	128	14·2	1·3	1024	100		
Active smoker	418	85·6	1·9	74	14·4	1·9	492	100		
>1 month <6 months	78	93·0	3·0	5	7·0	3·0	83	100		
>6 months <1 yr	28	92·7	5·0	2	7·3	5·0	30	100		
>1 yr <3 yr	76	85·4	4·6	10	14·6	4·6	86	100		
3 years or more	187	80·9	3·2	33	19·1	3·2	220	100		
Total	1683	87·0		252	13·0		1935	100		

* While the presented percentages (%) and standard errors (S.E.) are weighted, the sample sizes (n) are actual sample sizes.

men. The age, educational level and income of the sample is representative of the Mexican population. More participants were in the youngest ages groups: 43% (S.E. = 1·2) were 18–29 years of age, 25% (S.E. = 1·1) were 30–39 years of age, 18% (S.E. = 1·0) were 40–49 years of age and 15% (S.E. = 0·9) were 50–64 years of age. Educational attainment was predominantly at the primary and middle school level. Thus, 6% (S.E. = 0·6) had no formal education, 35% (S.E. = 1·3) primary school, 28% (S.E. = 1·2) middle school, 19% high school (S.E. = 1·0), 1% (S.E. = 0·2) vocational training and 12%

(S.E. = 1·0) university. As regards income, 6% (S.E. = 0·6) had an income less than the minimum wage in Mexico at the time of the study, 9% (S.E. = 0·8) had an income reflecting the Mexican minimum wage, 36% (S.E. = 1·3) had an income representing up to twice minimum wage, 33% (S.E. = 1·3) between two and four times the minimum wage and 16% (S.E. = 1·0) five or more times the minimum wage.

Roughly half the sample (51·6%, S.E. = 1·4) were never smokers; 22% were former smokers (S.E. = 1·1) and 26% current smokers (S.E. = 1·2). However, a greater proportion of smokers and

former smokers were men. Indeed, 40 % of men were current smokers and 30 % former smokers as compared to 15 % of women who were current smokers and 15 % former smokers ($\chi^2 = 310$, $p = 0.000$). Inversely, a greater proportion of women were classified in the elevated depressive symptoms group (19 % of women as compared to 8 % of men, $\chi^2 = 50$, $p = 0.000$).

Overall, controlling for sex, income and educational level, current smokers and former smokers had 80 % greater odds of elevated depressive symptomatology than never smokers (OR 1·8, 95 % CI 1·2–2·7, $p = 0.007$). Current smokers who smoke a pack or more a day had three times the odds of elevated depressive symptomatology than never smokers (OR 3·2, 95 % CI 1·2–8·5, $p = 0.017$), while those who smoke half a pack per day and those who smoke less than half a pack a day had 70–80 % greater odds of elevated depression (OR 1·8, 95 % CI 0·8–3·9, $p = 0.132$ and OR 1·7, 95 % CI 1·0–2·7, $p = 0.032$ respectively).

However, these results suggest that sex modified the association between smoking and depressive symptomatology. We, thus, present these analyses separately for women and men in Table 2. When analyzed separately for women, female smokers had twice the odds of elevated depressive symptomatology than female never smokers while there was only a non significant trend for men. Furthermore, female current smokers were about five times more likely to be high in depressive symptomatology than male current smokers (OR 5·3, 95 % CI 2·9–9·9, $p < 0.001$). The greater odds for former smokers become non-significant when analyzed separately by sex.

For women (refer to Table 2), there was no progressive relationship between quantity of cigarettes smoked daily and depressive symptomatology (probably because so few women, only 12 reported smoking a pack or more per day and only 29 half a pack). For men (refer to Table 2), however, those who smoked a pack or more daily had more than five times the odds of elevated depressive symptomatology than never smokers (OR 5·9, 95 % CI 1·6–21·9, $p = 0.008$). Only those men at the highest level of consumption had significant odds of elevated depression. Because the confidence intervals are large, these findings should be interpreted cautiously.

Table 2. *Estimated associations for women and men between smoking and elevated depressive symptomatology controlling for income and educational level**

| | Elevated depression† | | | | | |
| | Women | | | Men | | |
	OR	95% CI	*p*	OR	95% CI	*p*
Smoking status						
Current smoker	2·1	1·3–3·5	0·002	1·4	1·1–2·5	0·096
Former smoker	1·5	1·0–2·5	0·079	2·1	0·6–3·2	0·374
Non-smoker	1·0	—	—	1·0	—	—
Number of cigarettes daily						
A pack or more	1·8	0·5–7·0	0·391	5·9	1·6–21·9	0·008
½ pack	2·5	0·9–7·4	0·090	1·3	0·4–4·6	0·655
Less than half a pack	2·1	1·2–3·6	0·006	1·2	0·5–2·9	0·679
Former smokers	1·6	1·0–2·5	0·078	2·1	0·9–5·2	0·094
Non-smokers	1·0	—	—	1·0	—	—
Income	0·9	0·7–1·0	0·098	0·6	0·4–0·8	0·001
	1·0	—	—	1·0	—	—
Education	0·9	0·8–1·1	0·332	1·2	0·9–1·5	0·14
	1·0	—	—	1·0	—	—

* Odds ratios (OR), 95 % confidence intervals (95 % CI) and probability of significance (*p*) from logistic regressions.

† Elevated depression is defined as one standard deviation or more above the mean of CESD scores. Those not in the elevated depression group, while low in depressive symptomatology may have some symptoms.

Due to very few observations in some cells, the association of depressive symptomatology with time since smoking cessation controlling for income and education could not be analyzed separately by sex. Those who recently ceased smoking (more than one but less than 6 months prior) had lower odds of elevated depressive symptomatology than current smokers (OR 0·4, 95 % CI 0·1–1·0, $p = 0.042$), while those ceasing to smoke 6–12 months prior, more than 1 year but less than 3 years, and 3 or more years prior did not differ significantly from current smokers (OR 0·3, 95 % CI 0·1–1·7, $p = 0.131$; OR 1·0, 95 % CI 0·4–2·2, $p = 0.703$; OR 1·5, 95 % CI 0·8–2·6, $p = 0.765$ respectively). Because of the reduced observations, the confidence intervals are large and limit the conclusiveness of these findings.

DISCUSSION

The main finding of this study is that tobacco use is associated with elevated depressive symptomatology for Mexican adult women and for

Mexican adult men only at the highest levels of tobacco consumption (a pack or more daily). We were able to present evidence that current smokers and former smokers have higher odds of depression than never smokers, that higher levels of tobacco use were more strongly associated with depression for men, and that recent tobacco cessation (within 6 months) was associated with less depressive symptomatology. While income was also associated with depressive symptomatology, its effect did not account for the tobacco depression association. Before we discuss these findings in detail, it is necessary to acknowledge several limitations of studies of this type. First, the cross-sectional nature of the design does not permit an evaluation of the direction of causality. Second, tobacco involvement was assessed via self-report only, and as such the data are subject to recall bias and willingness to report completely and truthfully. Participants may have underreported cigarette consumption, as is suggested by studies with US Hispanics which have compared self-report measures and serum or salivary cotinine concentrations (Coutlas *et al.* 1988; Pérez-Stable *et al.* 1990*b*). For example, it was estimated that 20·4 % of Mexican–American men and 24·7 % of Mexican–American women who reported smoking less than 10 cigarettes per day may be underreporting the number of cigarettes smoked.

On the other hand, although depressive symptomatology was assessed using a screening instrument that has been useful in prior studies, it is important to acknowledge that the data correspond to the 7 days prior to the survey, with several disparities with respect to the window of observation for several measures of tobacco smoking (e.g. current, past month, past year and so forth). Given the cross-sectional nature of the data and the disparate windows of observation, it is impossible to analyze complex patterns in which tobacco smoking and depression might influence each other reciprocally. Finally, a word on statistical power is needed. Even with the relatively large epidemiologic sample of the ENA, the low proportion of reported smoking in women and depressive symptomatology in men precluded the possibility of analyzing sex-related differences more in depth and resulted in large confidence intervals, such that these findings should be interpreted with caution.

Notwithstanding these limitations, this study represents the first attempt to shed light on the association between tobacco smoking and depressive symptomatology with a representative sample of adults in Mexico. The overall, sex-adjusted odds of elevated depressive symptomatology for this Mexican sample is similar to those found in US Latinos (Pérez-Stable *et al.* 1990*a*), though the differences in strength of association between men and women were more pronounced. Replications of the tobacco depression relationship in studies from diverse countries will allow us to make inferences regarding the universality of this relationship and comparing countries where smoking is and is not socially acceptable will allow us to make better inferences about the role social acceptability plays in the tobacco depression relation. These data suggest that smoking is related to depression in the Mexican context as it is in the US context, regardless of fewer social control measures for tobacco consumption in Mexico.

These findings point to moderate associations of smoking status and smoking levels with depressive symptomatology. However, the mechanisms of this association are not well understood. Unidirectional, bi-directional and non-causal pathways have been proposed with some empirical support. Among unidirectional explanations, depression is proposed to lead to smoking either because of 'self-medication' wherein the depressed person smokes to relieve depressive symptomatology (Glass, 1990; Lerman *et al.* 1996) or because of the opposing explanation that exposure to nicotine or withdraw from nicotine leads to depression via neurochemical pathways (Pomerleau & Pomerleau, 1984; Xu *et al.* 2001). It is also possible that the social undesirability associated with smoking may contribute to depressive symptomatology (Link *et al.* 1989). A bi-directional explanation proposes that while depressed persons may smoke to relieve depression, the alleviation of depressed mood by nicotine positively reinforces smoking (Wang *et al.* 1996). Finally, it is proposed that smoking and depression are only related because of shared environmental and biological risk factors, but there is no causal relationship (Kendler *et al.* 1993).

One finding, which is not consistent with the international literature, is the possible effect

modification of sex in which smoking status is associated with depressive symptomatology for Mexican women at all levels of consumption and for men only at the highest levels of consumption. One explanation may have to do with differences in the social acceptability of smoking between the sexes. Because of less social acceptance of female smoking than male smoking in Mexico (Berenzon *et al.* 2000), Mexican women who smoke may have greater co-morbid problems overall than men who smoke (i.e. are more deviant than their non-smoking peers). Another explanation might be that lesser social acceptability of smoking in women contributes directly to greater depressive symptomatology. Furthermore, it may be that women and men smoke for different reasons or their smoking behavior is differentially reinforced. While data suggest that nicotine isolated from tobacco smoke is often reinforcing in men but not women, smoking in women may be primarily reinforced by other sensory or contextual factors (Perkins, 1999). For example, women may be more likely to smoke as a coping aid or to regulate affect, thus explaining a stronger relationship between depression and smoking in women. There may also be biological mechanisms which are not yet understood that contribute to a stronger association between depression and smoking in women, as there are gender differences in nicotine metabolism, nicotine discrimination, and cessation rates. However, for these last explanations to be viable, one would expect the greater association of depression and smoking in women to be found in varying countries and cultures which up till now is not the case. Of course we cannot rule out that this gender difference is a spurious finding, perhaps related to a greater measurement inaccuracy of depression in Mexican men or their greater reluctance to report depressive symptomatology.

The results of this study also suggest tentatively that the odds of depression are reduced the first 6 months after smoking cessation. There are several ways to interpret this finding. One interpretation is that tobacco consumption causes depression and thus depressive symptomatology is reduced immediately following cessation of tobacco consumption. Another possible interpretation is that those smokers who are less depressed are more able to cease smoking than more depressed smokers and thus the risk of depression is lower among the recent former smokers. This finding does not lend support to the self-medication hypothesis (though it cannot rule it out) for which one would expect increased depression following smoking cessation.

Should support for the tobacco depression association continue to accumulate among varying cultures and populations, research focusing on the question of causality and mechanisms of causality could have important implications for preventive and treatment interventions. Some studies have already begun to address questions regarding the impact of combining interventions targeting both depression and smoking cessation. For example, a controlled clinical trial of a self-administered smoking cessation program for Spanish speaking Latinos in the USA, found greater abstinence rates for a combined treatment which included a mood management intervention and a smoking cessation guide than for the use of the smoking cessation guide alone (Muñoz *et al.* 1997). Additionally the abstinence rates were greatest for those with a history of major depressive episodes, than for those without such a history or for those with a current depressive episode. Paradoxically, while smoking cessation was greatest in the combined mood management condition, CES-D scores were not reduced more in this condition than the smoking cessation without mood management condition. Thus, the mechanisms of change are unclear and apparently not related to decreasing depressive symptomatology. Research into the mechanisms of causality between smoking and depression should shed more light onto how interventions aimed at one of these disorders affects the other and how best to design interventions that increase the likelihood of targeting both problems.

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