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Persistent heavy smoking as risk factor for major depression (MD) incidence – Evidence from a longitudinal Canadian cohort of the National Population Health Survey

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ABSTRACT

Background: Reports of bidirectional associations between smoking and major depression (MD) have been interpreted as providing evidence for confounding by shared-vulnerability factors (SV) that predispose individuals to both conditions. If this is true, then smoking cessation may not reduce the risk of MD. From clinical practice and public health perspectives, the long-term outcomes associated with smoking persistence and cessation are potentially important and deserve exploration. To this end, the 12-year risk of MD in persistent heavy smokers and abstainers who were former-heavy smokers with and without adjustment for potential confounders were compared.

Methods: Follow-up data from the National Population Health Survey (NPHS) was used. Multinomial logistic (ML) models were fit to identify potential confounders. Using proportional hazard (PH) models, unadjusted and adjusted hazard ratios (HRs) for MD outcome were estimated for different smoking patterns.

Results: The unadjusted HR relating the risk of MD among current-heavy versus former-heavy smokers was 4.3 (95% CI: 2.6–6.9, $p < 0.001$). Current-heavy smoking predicted onset of MD (HR = 3.1, 95% CI: 1.9–5.2, $p < 0.001$) even after adjustment for age, sex and stress – the main confounders. However, this was not the case for the never, former-light, and current-light categories. Evidence of decreased risk of MD among former-heavy relative to current-heavy smokers as function of smoking cessation maintenance time was also found.

Conclusions: Contrary to common beliefs about the benefits of smoking for mental health, our results suggest that current-heavy rather than ever-heavy smoking is a major determinant of MD risk and point towards the benefits of smoking cessation maintenance.

1. Introduction

The association between smoking and depression is a well-documented phenomenon that is consistently reported among studies with different populations, study designs, analytical methods, definitions of smoking, and definitions of depression (Anda et al., 1990; Breslau et al., 1998; Brown et al., 1996; Dierker et al., 2002; Fergusson et al., 2003; Fergusson et al., 1996; Glassman et al., 2001; Glassman et al., 1990; Goodman and Capitman, 2000; Hall et al., 1991; Johnson et al., 2004; Kendler et al., 1993; Windle and Windle, 2001a; Wu and Anthony, 1999). Several cause-and-effect hypotheses have been proposed in response to these findings: a) unidirectional self-medication effects of pre-morbid depression on the risk of smoking and nicotine dependence (Breslau et al., 1991; Breslau et al., 1993; Carmody, 1989; Pomerleau and Pomerleau, 1984); b) unidirectional effects of chronic exposure to psychoactive substances in tobacco smoke including the proposed pharmacological effects of nicotine and

Abbreviations: MD, major depression; MDE, Major Depressive Episode; CPD, cigarettes smoked per day; SV, shared-vulnerability; HR, hazard ratio; CI, Confidence Interval; NPHS, National Population Health Survey; OR, odds ratio; CIDI-SF, Composite International Diagnostic Interview Short Form.

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monoamine-oxidase (MAO) inhibitors on the neurobiological processes that are implicated in the etiology of a Major Depressive Episode (MDE) (Balfour and Ridley, 2000; Benwell et al., 1990; Breese et al., 1997; Fowler et al., 2003; Korhonen et al., 2011; Pomerleau and Pomerleau, 1984; Schildkraut and Kety, 1967); and c) reciprocal or bidirectional effects with MDE increasing the risk for smoking and vice versa (Breslau et al., 1998; Mueser et al., 1998).

Alternatively, a non-causal explanation (Fergusson et al., 2003), that the association results from shared genetic, behavioral, and environmental factors that predispose individuals to both conditions (Hughes, 1988), has also been proposed. This is often referred to as the shared-vulnerability (SV) hypothesis. A number of mechanisms have been proposed under this hypothesis. An example is a bio-psycho-social model in which smoking and depression share certain personality phenotypes (neuroticism, extraversion, impulsive-unsocialized, and sensation-seeking) (Gilbert and Gilbert, 1995). Another possibility is common neurobiology that may influence the probability of both conditions (Greene, 2006; Levinson, 2006; Munafo et al., 2007).

The main approach in epidemiology has been to determine whether the effects are unidirectional or bidirectional, under the assumption that common etiologic factors as outlined by the SV hypothesis would likely to manifest as bidirectional associations. Although many prospective epidemiological studies have reported bidirectional associations between smoking and depression (Audrain-McGovern et al., 2009; Breslau et al., 1993, 1998; Brown et al., 1996; Windle and Windle, 2001b), only a few of these studies have actually assessed specific comorbidity hypotheses beyond the directionality of effects (Audrain-McGovern et al., 2009; Windle and Windle, 2001b). Furthermore, other epidemiological studies have also reported a unidirectional association; the majority of which reported smoking as predictor of depression onset (Boden et al., 2010; Flensborg-Madsen et al., 2011; Goodman and Capitman, 2000; Klungsoyr et al., 2006; Wu and Anthony, 1999).

Several twin and family studies have evaluated the possibility of shared genetic vulnerability between smoking and depression. Conflicting results have emerged with some studies supporting shared genetic liability (Johnson et al., 2004; Kendler et al., 1993; Lyons et al., 2008), while others have failed to replicate these findings (Dierker et al., 2002; Korhonen et al., 2007; McCaffery et al., 2003). This discrepancy may be due to different assessment methods of depression and smoking transitions as well as different analytical procedures (Johnson et al., 2004).

Based on findings from the abovementioned literature, it is very likely that SV factors account for some of the observed association between antecedent smoking and consequent MD. Whether these factors account for all of this association remains controversial. In particular, if the latter proposition is true, then smoking cessation may not reduce the risk of MDE. By explicating the long-term consequences of persistent heavy smoking as opposed to smoking cessation maintenance, the current study aims to clarify the clinical and public health implications, if any, of the smoking-to-MD association.

The current analysis has the following specific objectives: (1) to estimate the effect of antecedent heavy smoking persistence on the risk of MDE while adjusting for potential confounders of this association (2) to assess any dose–response effects of heavy smoking persistence on risk of MDE, and (3) to assess the potential impact of heavy smoking cessation maintenance on risk of MDE.

2. Materials and methods

2.1. Study design

The current analyses are based on data from the National Population Health Survey (NPHS), a population-based cohort study of a representative community sample of the Canadian population. The initial interviews were conducted in 1994–1995. The respondents have been prospectively followed and re-interviewed every second year in subsequent cycles for up to 7 cycles to date (2006–2007). Detailed information on the characteristics and sampling methods of the NPHS are described elsewhere (Swain, Catlin, and Beaudet, 1999; Tambay and Catlin, 1995).

2.2. Study sample

In light of previous findings implicating short-term quitting and relapse in MDE etiology (Covey et al., 1990; Glassman et al., 2001, 1990; Tsoh et al., 2000) and to avoid the potential of intermixing of the effects of other smoking transitions with the effects of heavy smoking persistence and continued heavy smoking cessation on the risk of MDEs, subjects who changed their smoking status during the follow-up period from that reported at baseline (3059) were excluded from the current analyses (Fig. 1). Therefore, the present sample is composed of 3824 respondents who did not change their smoking status throughout the follow-up duration (i.e. only those who stayed current, former, and never smokers). This criterion for selection and other restriction rules are shown in Fig. 1. Both death and the inability to trace subjects (see Supplementary Table 1) constituted a large proportion of the missing data in the original cohort. A comparison between the original and the present analytical sample on various bio-psycho-social variables associated with heavy smoking status and depression is available online (see Supplementary Table 2).

2.3. Long-term MDE risk assessments among ever-heavy smokers

Ever-heavy smokers (current and former) may share similar genetic, behavioral, and environmental vulnerabilities, at least for heavy smoking initiation. In turn, if these SV factors were dominant characteristics that also convey risk for MDE, then we would expect former-heavy smokers to continue to have elevated risks of MDE similar to those predicted of current-heavy smokers. However, if the persistence of the exposure (current as opposed to former) had the dominant effect on the risk for MDE, then current-heavy smokers would be expected to have higher risks of MDE relative to former-heavy smokers.

2.4. Measures

2.4.1. Smoking status (exposure)

The number of cigarettes smoked per day (CPD) was dichotomized based on reporting current or former daily consumption of 20 or more CPD (Fagerstrom, 1978). Current or former smokers were considered ever-heavy smokers if they reported smoking greater than 20 CPD at any time-point during the study. Only individuals consistently reporting smoking 20 CPD or less throughout follow-up were considered light smokers (past or current). We carried out dose–response assessments. Among current smokers, we assessed any dose–response effects of smoking amount on the risk of MDE over the 12-years of follow-up. Among former-heavy smokers, we assessed the risk of MDE as a function of the number of years since smoking cessation.

2.4.2. Major Depressive Episode (outcome)

The NPHS included a brief fully structured diagnostic interview for MDE, the Composite International Diagnostic Interview Short Form (CIDI-SF) (Kessler et al., 1998), which assesses the presence of depressive symptoms lasting a minimum of 2 weeks in the 12 months prior to the interview. The CIDI-SF algorithm is scored