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Psychiatric diagnoses among quitters versus continuing smokers 3 years after their quit day

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ABSTRACT

Background: People with psychiatric disorders are more likely to smoke and smoke more heavily than the general population, and they suffer disproportionally from smoking-related illnesses. However, little is known about how quitting versus continuing to smoke affects mental health and the likelihood of developing a psychiatric diagnosis. This study used data from a large prospective clinical trial to examine the relations of smoking cessation success with psychiatric diagnoses 1 and 3 years after the target quit day.

Methods: This study enrolled 1504 smokers (83.9% white; 58.2% female) in a cessation trial that involved the completion of the Composite International Diagnostic Interview to assess psychiatric diagnoses and biochemical confirmation of point-prevalence abstinence at Baseline and Years 1 and 3.

Results: Regression analyses showed that, after controlling for pre-quit (past-year) diagnoses, participants who were smoking at the Year 3 follow-up were more likely to have developed and maintained a substance use or major depressive disorder by that time than were individuals who were abstinent at Year 3.

Conclusions: Quitting smoking does not appear to negatively influence mental health in the long-term and may be protective with respect to depression and substance use diagnoses; this should encourage smokers to make quit attempts and encourage clinicians to provide cessation treatment.

1. Introduction

Cigarette smoking is strongly linked with psychopathology (Aubin et al., 2011). Smoking is especially prevalent among individuals with psychiatric disorders and some psychiatric disorders are related to both smoking heaviness and increased relapse likelihood (Aubin et al., 2011; Grant et al., 2004; Lasser et al., 2000; Piper et al., 2010). Clinicians who treat patients with mental illness all too rarely provide these patients with smoking cessation interventions (Hall, 2007; Lembke et al., 2007; Richter, 2006), despite evidence that such smokers are as motivated to quit smoking as are other smokers (McClave et al., 2010) and suffer disproportionately from smoking-related diseases (Schroeder and Morris, 2010).

While it is clear that smoking and psychopathology are strongly comorbid (Aubin et al., 2011), some aspects of their relationship remain relatively unexplored. For instance, it is unclear what smoking cessation, versus continued smoking, portends for the future likelihood of mental illness. The evidence relating cessation to future trajectories of psychiatric symptomatology is very limited. The extant studies tend to: comprise small samples that are restricted to psychiatric populations (Baker et al., 2010); rarely assess or report formal diagnoses; and employ cross-sectional rather than longitudinal designs.

From the extant data and various theories of psychopathology, it is possible to argue that smoking cessation could either increase or decrease the incidence and/or severity of subsequent psychopathology. For instance, both clinicians and smokers have suggested that smoking cessation might deprive smokers of an important affective coping strategy and might, therefore, exacerbate mental illness or jeopardize sobriety from other additive substances (Aubin et al., 2011; Brandon and Baker, 1991; Hitsman et al., 2009; Johnson et al., 2010; Prochaska, 2011; Solway, 2011; Weinberger et al., 2010; Ziedonis et al., 2008). Research shows that nicotine withdrawal is stressful and exacerbates negative affect (Hughes, 2006; Piasecki et al., 2000), and cessation may exacerbate the symptoms of depression (Glassman et al., 2001; Pomerleau et al., 2000) and/or schizophrenia (Cole et al., 2010). There is also evidence that loss of reinforcement may induce or worsen depression (e.g., Lewinsohn, 1974; see Aubin et al., 2011 for a discussion of other possible mechanisms), which suggests

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that cessation-related loss of smoking reinforcement could increase depression.

Conversely, some theories and data suggest that smoking cessation should either reduce psychopathology, or at least *not* worsen it. For instance, some evidence suggests that smokers with diagnosed psychopathology are able to quit smoking without significant increases in their psychiatric symptomatology (e.g., Baker et al., 2006, 2010; McFall et al., 2010; Schroeder and Morris, 2010). In theory, quitting smoking might reduce psychopathology through a variety of mechanisms. For instance, smoking could be considered a stressor, especially in light of the iterative cycles of withdrawal smokers experience between cigarettes (e.g., Hendricks et al., 2006). Therefore, ongoing smoking might result in affective dysregulation from which abstinent smokers would eventually recover (Parrott, 1994). Also, smoking may increase the likelihood that smokers are exposed to cues and opportunities that foster psychopathology (e.g., smoking may promote entry into social networks where other types of substance use and abuse are common) and quitting could reduce such exposure.

Bearing in mind the limitations of cross-sectional research, the available evidence from such research on the impact of cessation on mental health yields a fairly consistent pattern in which current smokers report worse mental health than never-smokers or former smokers (Murphy et al., 2003). Virtually, no cross-sectional data show that quitters have more severe psychopathology than those who continue to smoke (Berlin et al., 2010). However, it is difficult to draw strong inferences from such findings. It might be that smokers with less severe psychopathology are the ones able to quit; i.e., quitting does not influence psychopathology, but rather severe psychopathology impedes quitting. If it could be established that smoking cessation does not negatively affect long-term mental health, such findings could be used to boost smokers' willingness to make quit attempts and clinicians' willingness to intervene with their patients who smoke.

The present research uses data from a large prospective clinical trial to examine the relation of smoking status with psychiatric diagnoses, as well as changes in affect, 1 and 3 years following smoking cessation treatment. Strengths of the trial include its longitudinal design with pre-cessation baseline assessments, the lengthy follow-up interval, the relatively large sample size, and the use of formal psychiatric diagnoses based on structured interviews. These data will allow us to explore whether successful quitters versus continuing smokers have different likelihoods of meeting criteria for psychiatric diagnoses in the 3 years following their target quit day and whether cessation influences self-reported positive and negative affect. These findings could inform theories that attempt to account for links between psychopathology and tobacco use (e.g., Aubin et al., 2011).

2. Method

2.1. Recruitment and inclusion/exclusion criteria

Participants were recruited in Madison and Milwaukee, WI, to participate in a comparative efficacy smoking cessation clinical trial. Recruitment methods included TV, radio, and newspaper advertisements, community flyers, and earned media (e.g., interviews, press releases). Inclusion criteria included smoking ≥10 cigarettes per day on average for the past 6 months and being motivated to quit smoking. Exclusion criteria included current use of any medications contraindicated for use with the study's smoking cessation pharmacotherapies (e.g., current use of monoamine oxidase inhibitors, bupropion, lithium, anticonvulsants, or antipsychotics); any history of psychosis, bipolar disorder, or an eating disorder (contraindications for bupropion); consuming six or more alcoholic beverages daily 6 or 7 days a week (again, a contraindication for bupropion); pregnancy or breastfeeding; and serious health conditions that would prevent participation in or completion of the study. This study received human subjects' approval from the University of Wisconsin Health Sciences Institutional Review Board, and was registered with clinicaltrials.gov as number: Clinical trial registration: Smoking Cessation Medications: Efficacy, Mechanisms and Algorithms: NCT00332644. For more details see Piper et al. (2010).

2.2. Procedure

Participants who passed a phone screen attended an information session and provided written informed consent. Next, participants underwent multiple screenings, including a medical history screening, vital signs assessment, and a carbon monoxide breath test and completed demographic, smoking history, the Positive and Negative Affect Scale (PANAS; Watson et al., 1988) and tobacco dependence questionnaires. At a subsequent baseline visit, participants completed the World Mental Health Survey Initiative version of the Composite International Diagnostic Interview (CIDI; Kessler and Ustun, 2004; World Health Organization, 1990). Finally, eligible participants were randomized to one of six treatment conditions: bupropion sustained release ($n = 264$); nicotine lozenge ($n = 260$); nicotine patch ($n = 262$); nicotine patch plus nicotine lozenge ($n = 267$); bupropion sustained release plus nicotine lozenge ($n = 262$), or placebo (five placebo conditions that matched the five active conditions; $n = 189$). All participants received six individual 10–20 min counseling sessions led by bachelor's-level case managers supervised by a licensed clinical psychologist. All medications were provided for 8 weeks after the target quit day except the nicotine lozenge, which was provided for 12 weeks after the target quit day (consistent with prescribing instructions). Randomization was conducted in a double-blind fashion using a blocked randomization scheme with blocking on gender and race (White versus non-White). At 1 and 3 years after the target quit day participants completed the CIDI interview, additional questionnaires, including the PANAS, and a smoking status assessment.

2.3. Measures

2.3.1. Smoking status. Participants provided a breath sample at all study visits to permit alveolar carbon monoxide (CO) analysis, using a Bedfont Smokerlyzer (Bedfont Scientific, Rochester, England). Cessation outcomes were defined as biochemically confirmed (CO < 10 ppm) 7-day point-prevalence abstinence at 1 and 3 years after the target quit day. We used the intent-to-treat principle such that smokers who did not provide outcome data were assumed to be smoking.

2.3.2. World mental health survey initiative version of the CIDI. The CIDI (Kessler and Ustun, 2004; World Health Organization, 1990) is a structured clinical interview administered with Computer Assisted Personal Interviews (CAPI), Version 20 by trained study personnel certified by a CIDI trainer. The CIDI modules used provided both past-year diagnoses (i.e., within the past 12 months) as well as lifetime diagnoses (i.e., ever in the participant's lifetime, including in the past-year) for Depression, Mania, Panic Disorder, Social Anxiety Disorder (SAD), Generalized Anxiety Disorder (GAD), Alcohol Abuse and Dependence/Drug Abuse and Dependence, and Attention Deficit Disorder (ADD). The CIDI used in this research did not allow assessment of current psychiatric illness (e.g., occurring within the past 2 weeks; cf. First et al., 1998). Therefore, a smoker with a past-year diagnosis may or may not have been experiencing clinically significant symptoms at the time of the interview.

2.4. Analytic plan

Analyses were conducted using Predictive Analytics SoftWare (PASW) Statistics version 18.0 (SPSS Inc., Chicago, IL, USA). Logistic regression was used to examine the relation between smoking status (quit = 1 versus smoking = 0) and psychiatric diagnoses at Years 1 and 3 (diagnosis = 1 versus no diagnosis = 5). Linear regression was used to conduct similar analyses using negative and positive affect as the dependent variables. We analyzed diagnoses separately and then, to increase power, we created composite diagnostic categories by combining panic attack, GAD, and SAD to form an anxiety disorders category and combining alcohol and drug abuse or dependence disorders into a substance use disorder (SUD) category.

We also examined how diagnoses changed over time and how those changes were related to smoking status. Multinomial logistic regression was used to examine whether smoking status was related to differences in five diagnostic transition categories over three time points (Baseline, Year 1, and Year 3) with those with no past-year diagnosis across all three time points serving as the reference group. The likelihood of having no past-year diagnosis across all three time points was compared against the likelihood of having: (1) a past-year diagnosis at all three time points; (2) no past-year diagnosis at baseline but developing and maintaining a past-year diagnosis by Year 3 (i.e. no diagnosis at baseline, but diagnosis at Year 1 and Year 3, or diagnosis only at Year 3); (3) a baseline past-year diagnosis that resolved by Year 3 (i.e. a baseline diagnosis but no diagnosis at Year 1 and Year 3, or a diagnosis at baseline and Year 1 but not at Year 3); or (4) a fluctuating diagnosis (i.e. no past-year diagnosis at baseline and Year 3 but a past-year diagnosis at Year 1; or a past-year diagnosis at baseline and Year 3 but no past-year diagnosis at Year 1). Assessment of the relation between smoking status and diagnostic transitions was conducted separately for major depression, SUD, and anxiety diagnoses.

In the above analyses, we controlled for diagnosis in the past 12 months to address the possibility that a smoker might currently be symptomatic or vulnerable to an increase in psychiatric symptoms, given their recent diagnosis. We also conducted the same analyses controlling for lifetime diagnosis and with and without controlling for smoking cessation treatment condition. We did not control for all variables that differed significantly among diagnostic groups so as not to partial out variance in the naturally occurring diagnostic groups that was intrinsic to the nature