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Toward a comprehensive developmental model of smoking initiation and nicotine dependence

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ABSTRACT

Background: This study aims to identify predictors of smoking initiation and nicotine dependence (ND) to develop a comprehensive risk-factor model based on Kendler's development model for major depression.
Methods: Data were drawn from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), Wave 2 (n = 34,653). Risk factors were divided into five developmental tiers according to Kendler's model (childhood, early adolescence, late adolescence, adulthood, past-year). Hierarchical logistic regression models were built to predict the risk of smoking initiation and the risk of ND, given initiation. The continuation ratio (CR) was tested by ordinal logistic regression to examine whether the impact of the predictors was the same on smoking initiation or ND.
Results: The final models highlighted the importance of different tiers for each outcome. The CR identified substantial differences in the predictors of smoking initiation versus ND. Childhood tier appears to be more determinant for smoking initiation while the effect of more distal tiers (i.e. childhood and early adolescence) was tempered by more proximal ones (i.e. late adolescence, adulthood and past-year) in ND, with few sex differences.
Conclusions: The differential effect of some predictors on each outcome shows the complexity of pathways from smoking initiation to ND. While some risk factors may be shared, others impact only at one stage or have even an inverse effect. An adaptation of Kendler's developmental model for major depression showed high predictive power for smoking initiation and ND.

1. Introduction

Tobacco use continues to be the world's leading cause of preventable disease, disability, and premature death (World Health Organization, 2012). Despite substantial decreases in the prevalence of smoking in the last decades (Centers for Disease Control and Prevention, 2011; Secades-Villa et al., 2013), 27% of U.S. adults still reports some form of tobacco use (SAMHSA, 2012), nearly half of current smokers (12.8% of the U.S. population) are nicotine dependent (Grant et al., 2004a; Lopez-Quintero et al., 2011), quit attempts are often unsuccessful (Chiappetta et al., 2014), and rates of relapse are high (Garcia-Rodriguez et al., 2013). A better understanding of the etiology of smoking and nicotine dependence (ND) is essential to develop more effective prevention efforts and smoking cessation treatment programs.

A substantial body of research has identified a broad range of risk factors for smoking initiation. Several variables, including some personality traits, such as impulsivity (Bickel et al., 1999; Mitchell, 1999), neuroticism (Munafo et al., 2007), sexual abuse and other adverse childhood experiences (Anda et al., 1999; Perez-Fuentes et al., 2013; Sugaya et al., 2012), stressful life events (Pomerleau and Pomerleau, 1991) and most axis *I* and *II* psychiatric disorders (Center for Behavioral Health Statistics and Quality, 2013; Lasser et al., 2000; Secades-Villa et al., 2013; Ziedonis et al., 2008) have been strongly associated with tobacco use. By contrast, some lifestyle variables such as moderate physical activity have been linked to a decreased risk for regular smoking (Audrain-McGovern et al., 2003; Hedman et al., 2007), mostly in males (Garcia-Rodriguez et al., 2010).

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In contrast with this extensive research on risk factors for smoking initiation, to date there has been relatively little research examining risk factors for ND. The available research, largely focused on comorbid psychiatric disorders, has also found that axis *I* (Breslau et al., 2004; Kandel et al., 2007; Kendler et al., 1999; Wing et al., 2012) and axis *II* (Grant et al., 2004a; Lopez-Quintero et al., 2011) disorders strongly increase the likelihood of developing ND. Some demographic characteristics such as being white, female, of younger, and never married (Goodwin et al., 2011; Kandel and Chen, 2000; Lopez-Quintero et al., 2011) also increase the risk of ND among smokers.

However, risk factors are unlikely to act in isolation (Clark and Winters, 2002). For instance, adverse childhood experiences are strongly related to many psychiatric disorders, including ND (Perez-Fuentes et al., 2013; Sugaya et al., 2012), but this association may be moderated by recent stressful life events (Balk et al., 2009), as it is true for other psychiatric disorders (McLaughlin et al., 2010). In addition, some sex differences in risk factors for smoking have been previously reported (Flay et al., 1994; Garcia-Rodriguez et al., 2010; Mermelstein, 1999). Thus, a thorough understanding of the etiology of smoking initiation and ND requires an integrative developmental model, examining several risk factors, their joint and independent effects and sex interactions from a life-span perspective.

A key limitation of existing research in the etiology of substance use disorders (SUD) is the lack of integrated, empirically-supported models that examine risk factors from a life-span perspective. A few theoretical models have attempted to integrate a reduced number of variables to predict SUD, but used to be focus on a single set of variables (Kalivas and Volkow, 2005; Kessler and Price, 1993; Nathan, 1988). We sought to build on prior work by examining whether a promising candidate, Kendler's developmental model for major depression disorder (MDD) could be adapted to advance our understanding of smoking initiation and ND. Both MDD and ND appear to have multifactorial etiology and share at least some risk factors (Grant, 1995; Grant et al., 2004a,c; Kendler et al., 1993). In addition, Kendler's model has been successfully adapted to alcohol use disorders (AUD; Kendler et al., 2011), and cannabis use disorders (CUD; Blanco et al., 2014). Kendler's model, which is based on over two decades of work on the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders and over 3000 twin pairs posits that: (1) the etiology of MDD is multifactorial; (2) contemporary risk factors tend to influence each other; and, (3) the effect of earlier risk factors such as childhood sexual abuse is partially mediated through later risk factors such as childhood-onset anxiety and psychiatric comorbidity.

Kendler's model organizes predictors into tiers roughly approximating five developmental periods: childhood, early adolescence, late adolescence, adulthood, and the last year. The model seeks to be comprehensive and parsimonious, rather than exhaustive, and recognizes that several variables can have effects beyond their tier and possibly exert different effects by sex and across individuals.

Prior to our analyses, we adapted Kendler's model to incorporate aspects more important in the etiology and course of ND than of MDD. First, we substituted impulsivity, which plays a larger role in smoking initiation and ND (Audrain-McGovern et al., 2009; Ryan et al., 2013) for neuroticism. Second, we included a measure of early-onset of smoking (defined as onset of use prior to age 14), which appears to increase the risk of ND (Kendler et al., 2013). Third, we included a measure of regular physical activity in the adulthood tier, which has been previously reported to decrease the risk of smoking maintenance (Audrain-McGovern et al., 2003). Fourth, we included social support in the last-year tier, given its relevance in smoking initiation, maintenance and relapse (Lawhon et al., 2009). Fifth, we added religious service attendance to the last-year tier, given its inverse association with smoking initiation and ND (Ford and Hill, 2012; Kendler et al., 1999).

Given that predictors of smoking initiation may differ from those of ND (Dierker and Donny, 2008; Lopez-Quintero et al., 2011), we examined predictors of smoking initiation in the full population, and examined predictors of ND only among those with a history of smoking initiation.

2. Methods

2.1. Participants and procedures

The National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) (Grant et al., 2009, 2004c) was the source of data. The NESARC target population at Wave 1 was the civilian non-institutionalized population 18 years and older residing in households and group quarters. Blacks, Hispanics, and adults 18 to 24 years were oversampled, with data adjusted for oversampling and household and person-level non-response. Interviews were conducted with 43,093 participants by experienced lay interviewers. All procedures, including informed consent, received full ethical review and approval from the US Census Bureau and the US Office of Management and Budget. The Wave 2 interview was conducted approximately 3 years later (mean interval, 36.6 months). Excluding ineligible respondents (e.g. deceased), the Wave 2 response rate was 86.7% reflecting 34,653 completed interviews (Grant et al., 2009). Wave 2 NESARC weights include a component that adjusts for non-response, demographic factors, and psychiatric diagnoses to ensure that the Wave 2 sample approximated the target population, that is, the original sample minus attrition between the two waves. As described previously (Grant et al., 2009), adjustment for non-response was successful, as the Wave 2 respondents and the original target population did not differ on age, race/ethnicity, sex, socioeconomic status, or the presence of any substance, mood, anxiety, or personality disorder.

Participants included in this analysis were those with Wave 2 data ($n = 34,653$) and the primary outcomes were lifetime smoking initiation ($n = 16,297$) (defined as smoking 100 or more cigarettes in their entire life or any other tobacco product at least 20 times) and Wave 2 diagnosis of current (i.e. 12-month) DSM-IV nicotine dependence ($n = 4512$).

2.2. Measures

Data were collected using the Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV Version (AUDADIS-IV; Grant et al., 2001), a structured diagnostic interview that includes computer algorithms to generate DSM-IV diagnoses. AUDADIS-IV methods to diagnose axis *I* and *II* disorders are described in detail elsewhere (Grant et al., 2005a, 2005b; Hasin et al., 2005; Stinson et al., 2007). Test-retest reliabilities for AUDADIS-IV DSM-IV axis *I* and *II* diagnoses in the general population and clinical settings were fair to good ($\kappa = 0.40–0.77$; Canino et al., 1999; Grant et al., 2003; Ruan et al., 2008). Convergent validity was good to excellent for all affective, anxiety, and personality disorders diagnoses (Grant et al., 2004b; Hasin et al., 2005), and selected diagnoses showed good agreement ($\kappa = 0.64–0.68$) with psychiatrist reappraisals (Canino et al., 1999).

Based on Kendler's original model, we divided the potential risk factors for smoking initiation and past year ND into five developmental tiers: childhood, early adolescence, late adolescence, adulthood, and past year.

Childhood tier including family history of SUD (lifetime history of alcohol or drug use disorders [AUD or DUD, respectively] in the biological parents or siblings), any sexual abuse, vulnerable family environment (assessed using the childhood emotional neglect scale of the Childhood Trauma Questionnaire, (Bernstein et al., 1994)), and parental divorce or death of at least one parent during childhood.

Early adolescence tier including impulsivity (dichotomous, scored 1 if the respondents considered that they had often done things impulsively), low self-esteem (dichotomous, scored 1 if respondents believed they were not as good, smart, or attractive as most other people), age of onset of anxiety disorders (with early onset before age 18), age of smoking onset (with early onset defined as before age 14) (Dawson et al., 2008; Lopez-Quintero et al., 2011), and social deviance (assessed as the number of conduct disorder or antisocial personality disorder (ASPD) behaviors in which the respondent engaged before age 15, according to DSM-IV-TR criteria (American Psychiatric Association, 2000), range 0 to 33).

Late adolescence tier including educational attainment (in years), any history of trauma which occurred before age 21 out of the list of 23 traumatic events that measure post-traumatic stress disorder (PTSD), number of personality disorders and number of axis *I* disorders with onset before age 21.

Adulthood tier including history of divorce, history of SUD (AUD, CUD and other DUD), engagement in regular physical activity (using queries about frequency and intensity from the short-form International Physical Activity Questionnaire, (Craig et al., 2003; Dakwar et al., 2012) and social deviance (measured as the number of ASPD behaviors in which the individual engaged after age 15 but prior to the Wave 1 assessment).

Past year tier including social support (assessed with the Interpersonal Social Support Evaluation List; ISEL-12 (Cohen et al., 1985), a 12-item likert scale, range 12 to 48), past year AUD and CUD, comorbidity with psychiatric disorders other than