Published in final edited form as:
Drug Alcohol Depend. 2014 November 1; 144: 160–169. doi:10.1016/j.drugalcdep.2014.09.002.

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 5 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.

*Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

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Contributors

Carlos Blanco and Kenneth S. Kendler designed the study. Olaya García-Rodríguez provided summaries of previous research and wrote the first draft of the manuscript. Melanie M. Wall, Shuai Wang and Chelsea J. Jin conducted the statistical analysis. Olaya García-Rodríguez Carlos Blanco and Kenneth S. Kendler provided comments to the manuscript. All authors contributed to and have approved the final manuscript.

Conflict of Interest

All the authors declare that they have no conflicts of interest.

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.

Keywords

smoking initiation; nicotine dependence; risk factors; developmental model

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).

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, 2004c; 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 3,000 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= 4,512).

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 (κ=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 (κ=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 5 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 ND, current religious service attendance, marital problems (whether the respondent got separated, divorced or broke off a steady relationship in the last 12 months), number of stressful life events divided into independent (those the respondent is unlikely to have caused such as a death of a family member, range: 0–9) and dependent (those in which the respondent is likely to play an active role such as serious problems with a neighbor, range 0–5), and social deviance (measured as the number of ASPD behaviors in which the respondent engaged between Waves 1 and 2).

2.3. Statistical analyses

To obtain a thorough understanding of the relative importance of each variable and group of variables in the final model, we conducted our analysis in two stages, first identifying predictors of lifetime smoking initiation and then predictors of 12-month ND among those with a history of smoking initiation. To identify predictors of lifetime smoking initiation, we compared data from respondents with lifetime smoking versus those with no lifetime smoking. We used odds ratios (ORs) to examine the bivariate relationships between each predictor and lifetime smoking initiation (Table 1: Model 1). We then examined the main effects and the interactions of each predictor with sex (using men as the reference group), by

constructing one logistic regression model for each tier, and including age, ethnicity and sex as covariates in each model (Supplementary Model 1[1]). In the last step we constructed one logistic regression model of main effects and one of interactive effects including all variables that were significant in the prior step (Table 2: Model 2).

The second stage in our model development was to identify predictors of 12-month ND from the subsample with lifetime smoking initiation. To do that, we followed procedures similar to those used to construct our smoking initiation model, examining first the bivariate associations between each predictor and ND (Table 3: Model 3), then the main effects and the interactions of each predictor with sex, including age, ethnicity, and sex as covariates in each tier (Supplementary Model 2[1]) and another model of main effects and interactions with sex using all of the variables that were significant in the prior step (Supplementary Model 3[2]). Lastly, we constructed a final model that also included ND in adulthood but not in the past-year as a predictor (Table 4: Model 4). Predictive accuracy of lifetime smoking initiation and 12-month ND across the different models was assessed with the c-index (Harrell et al., 1984). The c-index is a measure of concordance between the predicted and the observed outcome and equals the area under the receiver operating characteristic curve such that values of 0.50 represent prediction no better than chance and values of 1.0 represent perfect prediction.

Finally, to examine whether the magnitude of the effect of the predictors was the same on the risk of lifetime smoking initiation as it was on the risk of 12-month ND conditional on initiation, we tested the continuation ratio (CR) with a logistic regression with three levels: no lifetime smoking, lifetime smoking with no ND and lifetime initiation with ND (MacLean, 1988). The model used in the logistic regression is a specially structured model, with the outcome variable a pass/fail indicator, and with a specially defined threshold variable. All the variables together with age, ethnicity, sex, and their interactions with the threshold were used as covariates. The adjusted betas are the coefficients of their corresponding interactions. Further details of this method can be found elsewhere (Colea and Ananthb, 2001). All analyses, including ORs and 95% confidence intervals (95% CI) were estimated using SUDAAN (Research Triangle Institute, 2007) to adjust for the design effects of the NESARC.

3. RESULTS

3.1. Lifetime smoking initiation

Table 1 presents the bivariate analyses of the variables included in our theoretical model in the sample with and without lifetime history of smoking initiation (Model 1). The results indicate that most predictors were significantly associated with increased odds of lifetime smoking initiation: being Native American; a family history of SUD, any sexual abuse, a vulnerable family environment and parental divorce or death during childhood; impulsivity, low self-esteem, anxiety and social deviance during early adolescence; history of trauma and

[1]Supplementary Models 1, 2, 3 and complete results of the sex interactions analyses are presented in Supplementary material and can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...
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a diagnosis of any Axis I or II disorder during late adolescence; being divorced, history of any SUD and social deviance during adulthood; higher social support, a diagnosis of any Axis I disorder (including SUDs), marital problems, stressful life events and social deviance during last year. However, being female, younger than 50 years old, being Black, Asian or Hispanic, having higher educational attainment, and attending religious service significantly decreased the odds of lifetime smoking initiation.

In Model 2 (Table 2), which examined the effect of each variable adjusted for age, race/ethnicity, sex and all significant variables in Supplementary Model 1[3], 13 out of 27 variables had significant main effects. The strongest association of smoking initiation was history of AUD (AOR= 2.14; 95% CI = 1.96–2.33) and CUD (AOR= 1.90; 95% CI = 1.62–2.24) whereas the AOR of the other significant associations ranged between 1.06 (independent stressful life events during last year) and 1.45 (being divorced). Higher educational attainment, a higher number of personality disorders and religious service attendance significantly decreased the odds of smoking in this model. The childhood and adulthood tiers included the majority of significant predictors. In the early- and late-adolescence tiers, only impulsivity, number of personality disorders and educational attainment, respectively, remained significant and in the past-year tier, neither SUD comorbidity or dependent stressful life events remained significant. Sex interactions with family history of SUD (AOR= 1.23; 95% CI = 1.07–1.42) and social deviance in adulthood (AOR= 1.09; 95% CI = 1.05–1.13) indicated stronger effects of these variables in women than in men. Past year AUD was found to have stronger effect in women than in men (AOR= 1.40; 95% CI = 1.06–1.84) even though the main effect of past year AUD was not significant (the significant interaction with OR>1). The protective effect of educational attainment was not as strong in women (AOR= 1.06; 95% CI = 1.04–1.08), while the protective effect of religiosity was even stronger in women (AOR= 0.84; 95% CI = 0.74–0.96). Complete results of the sex interaction analyses are provided in the Supplementary tables[4].

3.2. Twelve-month nicotine dependence

As in the bivariate model of smoking initiation, 12-month ND among individuals with lifetime smoking initiation was significantly associated with most predictors in the model (Table 3; Model 3). Prior history of ND (OR=8.11; 95% CI= 7.39–8.90) and past-year CUD (OR= 4.14; 95% CI = 3.12–5.48) were the variables with the strongest association with last year ND. The ORs of the other significant variables ranged from 1.02 (vulnerable family environment) and 3.11 (being younger than 30 years). Being Hispanic, parental divorce or death during childhood, higher educational attainment, and religious service attendance decreased odds of last year ND. In Model 4 (Table 4), which adjusted for age, race/ethnicity, sex and all significant variables in Supplementary Model 2 and 3[5], late adolescence, adulthood and past-year tiers included the majority of significant predictors. Past history of ND (AOR= 7.06; 95% CI = 6.37–7.82) was the strongest predictor of 12-month ND, followed by past-year AUD (AOR= 1.45; 95% CI = 1.23–1.70), marital problems and

[3]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...
[4]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...
[5]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

psychiatric comorbidity in the last year (AOR= 1.33; 95% CI = 1.06–1.66, AOR= 1.27; 95% CI = 1.19–1.35, respectively), impulsivity (AOR= 1.21; 95% CI = 1.07–1.37) and regular physical exercise (AOR= 1.19; 95% CI = 1.04–1.36) among others with lower AORs. Religious service attendance, history of AUD, number of Axis I disorders during late adolescence, higher educational attainment and vulnerable family environment had protective odds of 12-month ND among lifetime smokers. The protective effect of educational attainment (AOR= 0.95; 95% CI = 0.92–0.98) was stronger in women, while the effect of social support (AOR= 0.98; 95% CI = 0.96–1.00) was stronger in men,. Complete results for the sex interaction analyses are provided in the Supplementary tables[6].

3.3. Differential effects of predictors on smoking initiation and ND

Some predictors differed significantly in their impact on lifetime smoking initiation and 12-month ND (Table 5). The continuation ratio tests whether the impact of the risk factor on the initial stage of no smoking versus smoking initiation differs from the impact on the second stage, smoking initiation versus nicotine dependence. A positive adjusted beta indicates a greater impact on dependence than on initiation, while a negative adjusted beta indicates a greater impact on initiation than on dependence. All predictors in the childhood tier had a significantly stronger association with smoking initiation than with ND. Family history of SUD and sexual abuse increased risk for smoking initiation but not of ND and having a vulnerable family environment, which decreased risk for ND, was not associated to smoking initiation. Of particular interest, parental divorce or death significantly increased the risk for smoking initiation, but decreased the risk of ND. There were no significant differences on the association of smoking initiation and ND with any of the early adolescence tier variables. History of trauma and number of personality disorders during late adolescence had a stronger association with ND than with smoking initiation. Most variables in the adulthood tier (being divorced, history of AUD, CUDand social deviance) were significantly more strongly associated to smoking than to ND. The only exception was engaging in regular physical exercise, which was associated with decreased risk of smoking initiation but increased risk of ND. Five variables in the past year tier (alcohol use disorders, marital problems, dependent life events during past year, number of axis I disorders and social support during last year) were all more strongly associated with ND than with initiation. Of particular interest was having higher social support during last year, which increased the risk for ND, but decreased the risk of smoking initiation.

4. DISCUSSION

In a nationally representative sample of US adults, a broad range of variables in several developmental tiers individually predicted lifetime smoking initiation and 12-month ND. However, after mutually adjusting for the effect of other covariates, the number of significant predictors was reduced. The predictive power of the models for lifetime smoking initiation and for 12-month ND was high and there were few sex differences in both models. However, although lifetime smoking initiation and 12-month ND shared several predictors, the final models and the continuation ratio highlighted the importance of different tiers for

[6]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

each outcome. The models also highlighted that even when a variable predicted both outcomes it sometimes had a differential effect on each outcome.

Consistent with previous research (Garcia-Rodriguez et al., 2010; Griesler and Kandel, 1998; Hu et al., 2006), we found that numerous variables when examined individually increased the likelihood of smoking initiation. After adjusting for the effect of other covariates, a more restricted set of variables independently contributed to the probability of smoking. Specifically, most childhood variables predicted lifetime smoking initiation, while early- and late-adolescence variables did not, suggesting the strong influences of experiences in earlier developmental periods. Most adulthood variables also predicted lifetime smoking while most past-year variables were not significantly associated with smoking initiation. The c-index of the final model was 0.75, suggesting good predictive power.

In line with previous findings about the etiology of MDD (Kendler et al., 2002, 2006), AUD (Kendler et al., 2011), and CUD (Blanco et al., 2014) we also found that although multiple variables predicted 12-month ND in the bivariate analyses, more distal predictors were no longer significant after taking into account the effect of more proximal ones. With the exception of impulsivity, early-tiers variables did not predict 12-month ND. Similar results have been found in previous genetic studies about developmental trajectories of nicotine dependence, finding that social and familial environmental factors, more closely related to childhood and early adolescent tiers do not predict lifetime ND (Kendler et al., 2008). However, adverse event during childhood and early adolescence may act as risk factors for low educational achievement (Breslau et al., 2009), marital problems (Theobald and Farrington, 2012), personality disorders (Fryers and Brugha, 2013), or other psychiatric disorders, including SUD (Friestad et al., 2012; Fryers and Brugha, 2013; Levitan et al., 1998; Schilling et al., 2007), all of which increase the risk for ND.

As expected, having a previous lifetime history of ND had the largest OR in the model for 12-month ND. Smoking cessation and relapse to smoking is by far the most common course of ND (Garcia-Rodriguez et al., 2013; Piasecki, 2006; Rafful et al., 2013). Individuals with past history of ND may be more vulnerable to current ND due to dysregulation of neurochemical mechanisms involved in brain reward systems (Koob, 2006), psychological factors such as craving (Shadel et al., 2011), or mechanisms related to drug-associated cues (Shaham et al., 2003). Although prior history of ND was the strongest predictor of current ND, other risk factors such as past-year AUD, marital problems, psychiatric comorbidity and impulsivity, also independently increased the risk of 12-month ND, pointing to the multifactorial etiology of ND and stressing the importance of these variables as amenable to intervention in multicomponent smoking cessation programs.

A novel contribution of our study was that, by using the continuation ratio, we were able to identify substantial differences in the predictors of lifetime smoking initiation versus 12-month ND. Several variables across most tiers differed significantly in their association with smoking initiation versus ND. For example, family history of SUD, sexual abuse during childhood, history of any SUD and social deviance predicted lifetime smoking initiation but not 12-month ND. By contrast, history of trauma during late adolescence, psychiatric

comorbidity (including AUD), marital problems and dependent stressful life events during last year predicted 12-month ND but not lifetime smoking initiation. More dramatically, some variables, such as engaging in regular physical activity, parental divorce or death or social support had opposite effects on risk of smoking initiation versus risk of ND. Although physical activity has been shown to protect against smoking initiation in adolescents (Audrain-McGovern et al., 2003; Garcia-Rodriguez et al., 2010), recent findings suggest that adults who engaged in vigorous exercise had greater liability to psychiatric disorders, including SUD (Dakwar et al., 2012). Common reward-related vulnerabilities, such as sensation seeking and reward dependence may account for this association (Boecker et al., 2008). Parental divorce or death have been related to drug use initiation due to lack of parental supervision and other factors associated with divorce (D'Onofrio et al., 2005). However the opposite influence of this variable on transition to dependence may be reflecting other familial factors, such as genetic variation in drug metabolism (Kendler et al., 1999), that may protect against substance dependence. While specific abstinence-related social support has been associated with better outcomes when trying to quit (Lawhon et al., 2009), general social support as measure in the current study seems to have opposite effects on initiation than on ND. Greater general social support before smoking onset may compete with social pressure for smoking, thus protecting against initiation. In contrast, those who initiated and progress to ND are likely to have smoking peers, increasing social support by means of shared behaviors (Kobus, 2003).

Taken together, childhood tier variables appear to be more relevant for lifetime smoking initiation than for 12-month ND, whereas past-year tier maybe more important for 12-months ND, mediating the effect of earlier tiers. These findings are consistent with prior studies documenting that the risk factors for drug initiation and dependence differ and that social and familiar childhood experiences influence initiation but not dependence (Blanco et al., 2014; Kendler et al., 2011, 2008; Rhee et al., 2003). These differences in predictors of smoking initiation versus 12-month ND show the potential complexity of pathways from smoking initiation to ND and that some risk factors may be shared while others impact only at one stage or have even an inverse effect depending on the stage. The differential effects of predictors on smoking initiation and ND suggest that interventions may need to differ depending on whether they target prevention smoking initiation versus achieving smoking cessation.

Most of the strongest predictors of 12-month ND overlapped with those recently reported for CUD (Blanco et al., 2014). AUD and psychiatric comorbidity during last year and impulsivity in early adolescence are strong predictors for both CUD and ND. Similarly, religious service attendance decreased the risk for both disorders, pointing to shared pathways to the development and maintenance of these disorders, as previously suggested (Lopez-Quintero et al., 2011; Palmer et al., 2012). By contrast, predictors of lifetime smoking and lifetime cannabis use have substantial differences, possibly suggesting substance-specific risk factors for drug use initiation but common liability for drug dependence (Palmer et al., 2012).

There were few significant sex interactions, indicating that overall, the models for both lifetime smoking initiation and 12-month ND did not differ much by sex. However, we

identified some variables more strongly associated with one sex or the other. For example, family history of SUD and past-year AUD increased the risk of lifetime smoking initiation even more females than in males, while higher education attainment and religious service attendance were more protective for males. Similarly, history of other drug use disorders was more strongly associated to the risk of 12-month ND among females than males. Few sex differences have been also reported in prior comprehensive developmental models of the etiology of MDD (Kendler et al., 2002, 2006) and CUD (Blanco et al., 2014).

Our study has limitations that should be mentioned. First, as in most large-scale surveys, information of substance use, included tobacco, was based on self-report and not confirmed by objective methods and variables included in the models were assessed retrospectively which may have led to recall bias. Second, impulsivity and self-esteem variables were assessed with a single item, which may reduce criterion validity given the multifaceted nature of these constructs. However, one single-item assessments of self-esteem have shown strong convergent validity with gold-standard scales (Robins et al., 2001) and the single item "I act on impulse" has systematically showed the highest loadings in factor structure studies (Chahin et al., 2010; Patton et al., 1995; Spinella, 2007) and good predictive validity (Chamorro et al., 2012). Third, although we organized the predictors into five discrete developmental tiers, there is clear overlap and considerable between-subject variability across those periods. Nevertheless, the use of those periods provides a structure that has been successfully tested with other disorders, such as MDD and CUD.

Despite these limitations, our study shows that an adaptation of Kendler's model for MDD had high predictive power, providing a useful developmental model for the etiology of smoking initiation and ND. The model included five developmental tiers in which childhood tier appears to be more determinant for lifetime 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 12-months ND, in both male and females and with few sex differences. We hope these findings may be helpful in the development of future, theory-based prevention smoking initiation and ND treatment programs.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Role of Funding Sources

Funding for this study was provided in parts by NIH grants DA019606, DA023200, CA133050 and the New York State Psychiatric Institute (Dr. Blanco and Melanie M. Wall). The NIH and the NYSPI had no further role in the study design, collection, analysis or interpretation of the data, the writing of the manuscript or the decision to submit the paper for publication.

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Highlights

- A developmental model of smoking initiation and nicotine dependence is proposed
- Risk factors were divided into 5 developmental tiers from childhood to last year
- Substantial differences for smoking initiation versus nicotine dependence were identified
- Childhood tier appears to be more determinant for smoking initiation
- More proximal tiers tempered the effect of more distal tiers in nicotine dependence

Table 1

Bivariate associations of risk factors and prevalence of lifetime smoking initiation (SI) in the general population. NESARC wave 2 (n= 34,653).

	No smoking initiation (n=18,356)			Smoking initiation (n=16,297)			Model 1[a]
	%/mean		CI	%/mean		CI	OR (CI 95%)
Age							
18–29	18.39	17.51	19.30	14.18	13.43	14.97	**0.62 (0.57–0.68)**
30–39	21.29	20.40	22.20	16.00	15.31	16.71	**0.61 (0.56–0.66)**
40–49	21.42	20.66	22.19	21.60	20.74	22.47	**0.81 (0.76–0.87)**
>=50	38.91	37.72	40.11	48.22	47.18	49.27	1.00 (1.00–1.00)
Race/ethnicity							
White, non-Hispanic	64.66	60.58	68.53	77.48	75.37	79.47	1.00 (1.00–1.00)
Black, non-Hispanic	12.76	11.23	14.47	9.25	8.14	10.49	**0.60 (0.55–0.67)**
Native American	1.68	1.34	2.11	2.72	2.29	3.23	**1.35 (1.07–1.70)**
Asian/Pacific Islander	6.09	4.86	7.61	2.36	1.74	3.21	**0.32 (0.27–0.39)**
Hispanic	14.81	11.88	18.31	8.18	6.82	9.80	**0.46 (0.41–0.52)**
Sex							
Male	39.08	38.12	40.05	57.20	56.18	58.22	**1.00 (1.00–1.00)**
Female	60.92	59.95	61.88	42.80	41.78	43.82	**0.48 (0.45–0.51)**
Childhood							
Family history of SUD	33.12	31.88	34.39	46.34	45.08	47.61	**1.74 (1.66–1.83)**
Sexual abuse	8.84	8.23	9.48	11.80	11.14	12.50	**1.38 (1.26–1.51)**
Vulnerable family environment (mean)	2.94	2.84	3.04	3.56	3.47	3.66	**1.03 (1.03–1.04)**
Parental divorce or death	8.70	8.21	9.23	10.46	9.93	11.01	**1.23 (1.13–1.33)**
Early adolescence							
Impulsivity	12.88	12.26	13.53	21.49	20.62	22.37	**1.85 (1.72–1.99)**
Low self-esteem	11.35	10.56	12.19	12.91	12.17	13.69	**1.16 (1.06–1.27)**
Early-onset anxiety	9.13	8.40	9.92	10.97	10.16	11.83	**1.23 (1.12–1.34)**
Social deviance[b] (mean)	0.32	0.30	0.35	0.79	0.75	0.83	**1.33 (1.29–1.37)**
Late adolescence							
Education years (mean)	14.13	13.98	14.27	13.45	13.36	13.55	**0.95 (0.93–0.96)**
History of trauma	49.87	48.34	51.41	58.30	57.08	59.51	**1.41 (1.33–1.48)**

	No smoking initiation (n=18,356)			Smoking initiation (n=16,297)			Model 1[a]
	%/mean		CI	%/mean		CI	OR (CI 95%)
Number of Axis I disorders excluding ND (mean)[b]	0.34	0.32	0.36	0.66	0.63	0.69	**1.49 (1.43–1.54)**
Number of Personality disorders (mean)	0.31	0.29	0.33	0.49	0.47	0.52	**1.23 (1.19–1.26)**
Adulthood[c]							
Ever divorced	27.55	26.68	28.43	40.81	39.62	42.01	**1.81 (1.72–1.91)**
History of SUD							
Alcohol	20.04	18.73	21.43	47.53	45.92	49.14	**3.61 (3.36–3.89)**
Cannabis	3.63	3.22	4.09	15.66	14.77	16.60	**4.93 (4.37–5.57)**
Other drugs	2.27	1.97	2.61	10.04	9.32	10.81	**4.81 (4.18–5.54)**
Regular physical exercise	71.04	69.64	72.41	70.47	69.42	71.50	0.97 (0.92–1.03)
Social deviance (mean) [b]	1.10	1.04	1.16	2.63	2.52	2.73	**1.25 (1.23–1.27)**
Past year							
Social support (mean)	17.28	17.14	17.43	17.66	17.54	17.78	**1.01 (1.01–1.02)**
Alcohol use disorders	6.05	5.53	6.61	13.48	12.78	14.21	**2.42 (2.19–2.67)**
Cannabis use disorders	0.62	0.48	0.79	2.63	2.31	3.00	**4.35 (3.31–5.72)**
Number of Axis I disorders excluding ND (mean)	0.28	0.26	0.29	0.41	0.39	0.43	**1.23 (1.19–1.28)**
Religious service attendance	61.03	59.58	62.45	45.46	44.09	46.83	**0.53 (0.50–0.57)**
Marital problems	4.26	3.92	4.62	5.35	4.98	5.76	**1.27 (1.13–1.43)**
Stressful life events (mean)	1.34	1.30	1.38	1.60	1.56	1.65	**1.10 (1.08–1.12)**
Independent (mean)	0.61	0.59	0.63	0.75	0.72	0.77	**1.19 (1.15–1.23)**
Dependent (mean)	0.69	0.66	0.71	0.81	0.78	0.83	**1.11 (1.09–1.14)**
Social deviance (mean) [b]	0.29	0.27	0.31	0.66	0.63	0.70	**1.33 (1.28–1.37)**

[a] Bivariate model;

[b] in early adolescence antisocial behavior before age 15; in adulthood since age 15; and past year between Wave 1 and Wave 2;

[c] lifetime but before 12 month. OR= odds ratio; SUD= substance use disorders; ND= nicotine dependence; for continuous variables, ORs reflect the change in the odds per unit of predictor; Significant results are bolded (p<0.05).

Drug Alcohol Depend. Author manuscript; available in PMC 2015 November 01.

Table 2

Multivariable associations of risk factors and lifetime smoking initiation (SI). NESARC wave 2 (n=34,653).

	Model 2[a] AOR (CI 95%)
Childhood	
Family history of SUD	**1.23 (1.15–1.31)**
Sexual abuse	**1.20 (1.08–1.34)**
Vulnerable family environment[b]	1.00 (0.99–1.01)
Parental divorce or death	**1.10 (1.01–1.21)**
Early adolescence	
Impulsivity	**1.24 (1.13–1.36)**
Low self-esteem	-
Early-onset anxiety	0.94 (0.83–1.06)
Social deviance [b,c]	1.01 (0.98–1.05)
Late adolescence	
Education years[b]	**0.93 (0.92–0.94)**
History of trauma	1.07 (1.00–1.14)
Number of Axis I disorders excluding ND[b]	1.00 (0.94–1.06)
Number of Personality disorders[b]	**0.87 (0.84–0.91)**
Adulthood[d]	
Ever divorced	**1.45 (1.35–1.55)**
History of SUD	
Alcohol	**2.14 (1.96–2.33)**
Cannabis	**1.90 (1.62–2.24)**
Other drugs	**1.27 (1.06–1.53)**
Regular physical exercise	0.95 (0.89–1.01)
Social deviance[b,c]	**1.12 (1.09–1.14)**
Past year	
Social support[b]	-
Alcohol use disorders	1.00 (0.88–1.13)
Cannabis use disorders	1.00 (0.68–1.46)
Number of Axis I disorders excluding ND[b]	**1.09 (1.04–1.15)**
Religious service attendance	**0.66 (0.61–0.70)**
Marital problems	-
Stressful life events[b]	
Independent[b]	**1.06 (1.02–1.10)**
Dependent[b]	1.00 (0.96–1.03)
Social deviance[b,c]	**1.07 (1.03–1.11)**
	c index[e]=0.75

[a] Only significant variables in Supplementary Model 1 (Supplementary material) were included. Controlling for age, ethnicity, sex and all significant variables in Supplementary Model 1;

[b] for continuous variables, AORs reflect the change in the odds per unit of predictor;

[c] in early adolescence antisocial behavior before age 15; in adulthood since age 15; and past year between Wave 1 and Wave 2;

[d] lifetime but before 12 month.

[e] c-index for Model 2 represent predictability with all variables included; AOR= adjusted odds ratio; SUD= substance use disorders; ND= nicotine dependence; significant variables are bolded ($p<0.05$). Reference group= no smoking initiation.

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Table 3

Bivariate associations of risk factors and prevalence of 12-months nicotine dependence (ND) among individuals with lifetime smoking initiation. NESARC wave 2 (n=16,297).

	Smoking initiation with no ND (n=11,785)			Nicotine Dependence (n=4,512)			Model 3[a]
	%/mean	CI		%/mean	CI		OR (CI 95%)
Age							
18–29	11.42	10.58	12.31	21.13	19.70	22.63	**3.11 (2.74–3.53)**
30–39	14.55	13.82	15.32	19.62	18.01	21.34	**2.27 (1.98–2.59)**
40–49	19.52	18.52	20.56	26.81	25.10	28.58	**2.31 (2.02–2.63)**
>50	54.51	53.28	55.74	32.44	30.62	34.32	1.00 (1.00–1.00)
Race/ethnicity							
White, non-Hispanic	77.70	75.42	79.82	76.95	74.68	79.07	1.00 (1.00–1.00)
Black, non-Hispanic	8.97	7.91	10.16	9.94	8.49	11.60	**1.12 (1.00–1.25)**
Native American	2.17	1.81	2.60	4.10	3.20	5.24	**1.90 (1.46–2.48)**
Asian/Pacific Islander	2.43	1.77	3.33	2.20	1.44	3.33	0.91 (0.63–1.33)
Hispanic	8.73	7.18	10.57	6.82	5.65	8.21	**0.79 (0.67–0.93)**
Sex							
Male	58.59	57.36	59.80	53.72	51.79	55.65	1.00 (1.00–1.00)
Female	41.41	40.20	42.64	46.28	44.35	48.21	**1.22 (1.11–1.34)**
Childhood							
Family history of SUD	44.18	42.77	45.60	51.77	49.78	53.75	**1.36 (1.24–1.48)**
Sexual abuse	10.05	9.28	10.88	16.18	14.92	17.53	**1.73 (1.51–1.98)**
Vulnerable family environment (mean)	3.46	3.35	3.56	3.83	3.66	3.99	**1.02 (1.01–1.03)**
Parent's divorce or death	11.00	10.38	11.66	9.09	8.09	10.20	**0.81 (0.70–0.93)**
Early adolescence							
Impulsivity	17.98	17.05	18.95	30.24	28.72	31.81	**1.98 (1.81–2.17)**
Low self-esteem	11.11	10.35	11.92	17.41	15.96	18.97	**1.69 (1.49–1.91)**
Early-onset anxiety	9.49	8.75	10.28	14.68	13.14	16.38	**1.64 (1.44–1.88)**
Early-onset of smoking	27.10	26.08	28.14	36.73	34.72	38.78	**1.56 (1.42–1.71)**
Social deviance[b] (mean)	0.65	0.61	0.69	1.14	1.04	1.24	**1.15 (1.12–1.18)**
Late adolescence							

	Smoking initiation with no ND (n=11,785)			Nicotine Dependence (n=4,512)			Model 3[a]
	%/mean	CI		%/mean	CI		OR (CI 95%)
Education years (mean)	13.64	13.52	13.75	12.98	12.88	13.08	**0.93 (0.92–0.95)**
History of trauma	54.20	52.84	55.55	68.61	66.79	70.36	**1.85 (1.69–2.02)**
Number of Axis I disorders excluding ND (mean)	0.54	0.51	0.57	0.95	0.89	1.00	**1.37 (1.32–1.42)**
Number of Personality disorders (mean)	0.35	0.33	0.37	0.84	0.78	0.90	**1.47 (1.41–1.53)**
Adulthood[c]							
Ever divorced	39.91	38.57	41.25	43.08	41.18	44.99	**1.14 (1.05–1.24)**
History of SUD							
Nicotine	26.47	25.37	27.60	74.48	72.74	76.15	**8.11 (7.39–8.90)**
Alcohol	44.26	42.64	45.90	55.73	53.36	58.08	**1.59 (1.45–1.74)**
Cannabis	12.66	11.80	13.58	23.19	21.48	25.00	**2.08 (1.86–2.33)**
Other drugs	7.62	6.93	8.37	16.12	14.72	17.62	**2.33 (2.05–2.65)**
Regular physical exercise	68.28	67.12	69.42	75.94	74.26	77.55	**1.47 (1.33–1.61)**
Social deviance[b] (mean)	2.22	2.13	2.31	3.66	3.46	3.86	**1.11 (1.10–1.12)**
Past year							
Social support (mean)	17.43	17.30	17.56	18.24	18.02	18.46	**1.03 (1.02–1.03)**
Alcohol use disorders	10.30	9.65	10.99	21.46	19.84	23.18	**2.38 (2.11–2.68)**
Cannabis use disorders	1.43	1.18	1.73	5.66	4.71	6.78	**4.14 (3.12–5.48)**
Number of Axis I disorders excluding ND (mean)	0.27	0.26	0.29	0.75	0.71	0.80	**1.71 (1.63–1.80)**
Religious service attendance	49.77	48.20	51.34	34.65	32.66	36.69	**0.53 (0.49–0.59)**
Marital problems	3.75	3.38	4.15	9.38	8.45	10.41	**2.66 (2.26–3.13)**
Stressful life events (mean)	1.33	1.29	1.38	2.28	2.20	2.37	**1.33 (1.29–1.37)**
Independent (mean)	0.65	0.63	0.67	0.98	0.93	1.02	**1.44 (1.37–1.50)**
Dependent (mean)	0.64	0.61	0.67	1.21	1.16	1.27	**1.53 (1.47–1.60)**
Social deviance[b] (mean)	0.43	0.40	0.45	1.25	1.16	1.35	**1.37 (1.33–1.42)**

[a] Bivariate model;

[b] in early adolescence antisocial behavior before age 15; in adulthood since age 15; and past year between Wave 1 and Wave 2;

[c] lifetime but before 12 month; OR= odds ratio; SUD= substance use disorders; ND= nicotine dependence; for continuous variables, ORs reflect the change in the odds per unit of predictor; significant variables are bolded (p<0.05).

Table 4

Multivariable associations of risk factors and 12-months nicotine dependence (ND) among individuals with lifetime smoking initiation. NESARC wave 2 (n=16,297).

	Model 4[a] AOR (CI 95%)
Childhood	
Family history of SUD	0.92 (0.82–1.03)
Sexual abuse	0.91 (0.77–1.08)
Vulnerable family environment[b]	**0.99 (0.98–1.00)**
Parental divorce or death	-
Early adolescence	
Impulsivity	**1.21 (1.07–1.37)**
Low self-esteem	0.94 (0.79–1.11)
Early-onset anxiety	0.92 (0.75–1.12)
Early-onset of smoking	1.03 (0.92–1.15)
Social deviance[b,c]	0.99 (0.96–1.03)
Late adolescence	
Education years[b]	**0.93 (0.91–0.94)**
History of trauma	**1.17 (1.05–1.31)**
Number of Axis I disorders excluding ND[b]	**0.92 (0.86–0.99)**
Number of Personality disorders[b]	**1.07 (1.00–1.14)**
Adulthood[d]	
Ever divorced	1.10 (0.99–1.23)
History of SUD	-
Nicotine	**7.06 (6.37–7.82)**
Alcohol	**0.79 (0.70–0.90)**
Cannabis	-
Other drugs	0.96 (0.80–1.15)
Regular physical exercise	**1.19 (1.04–1.36)**
Social deviance[b,c]	0.98 (0.96–1.00)
Past year	
Social support[b]	1.01 (1.00–1.02)
Alcohol use disorders	**1.45 (1.23–1.70)**
Cannabis use disorders	-
Number of Axis I disorders excluding ND[b]	**1.27 (1.19–1.35)**
Religious service attendance	**0.65 (0.58–0.72)**
Marital problems	**1.33 (1.06–1.66)**
Stressful life events[b]	
Independent[b]	**1.08 (1.02–1.14)**
Dependent[b]	**1.11 (1.05–1.17)**

	Model 4[a] AOR (CI 95%)
Social deviance[b,c]	**1.12 (1.07–1.16)**
	c index[e]=0.82

[a] Only significant variables in Supplementary Model 2 (Supplementary material) were included. Controlling for age, ethnicity, sex and all significant variables in Supplementary Model 2 and including history of ND;

[b] for continuous variables, AORs reflect the change in the odds per unit of predictor;

[c] in early adolescence antisocial behavior before age 15; in adulthood since age 15; and past year between Wave 1 and Wave 2;

[d] lifetime but before 12 month;

[e] c-index for model 4 represent predictability with all variables included; AOR= adjusted odds ratio; SUD= substance use disorders; ND= nicotine dependence; significant variables are bolded ($p<0.05$). Reference group= smoking initiation with no ND.

Table 5

Differential effects of predictors on lifetime smoking initiation and 12-months nicotine dependence (ND). NESARC wave 2 (n=34,653).

	Smoking initiation with no ND[a] (n=11,785)	Nicotine Dependence[b] (n=4,512)	Continuation ratio	
	AOR (CI 95%)	AOR (CI 95%)	Adjusted beta[c]	AOR (CI 95%)[d]
Childhood				
Family history of SUD	**1.23 (1.15–1.31)**	1.03 (0.93–1.15)	**−0.17**	**0.84 (0.74–0.96)**
Sexual abuse	**1.19 (1.07–1.33)**	0.96 (0.82–1.14)	**−0.21**	**0.81 (0.66–0.99)**
Vulnerable family environment[e]	1.00 (0.99–1.01)	**0.98 (0.97–0.99)**	**−0.02**	**0.98 (0.97–0.99)**
Parental divorce or death	**1.10 (1.00–1.20)**	**0.84 (0.71–0.99)**	**−0.27**	**0.77 (0.64–0.92)**
Early adolescence				
Impulsivity	**1.23 (1.12–1.35)**	**1.20 (1.07–1.35)**	−0.02	0.98 (0.83–1.14)
Low self-esteem	**0.90 (0.82–1.00)**	0.98 (0.84–1.14)	0.08	1.09 (0.90–1.31)
Early-onset anxiety	0.94 (0.83–1.06)	1.02 (0.85–1.22)	0.08	1.08 (0.87–1.35)
Social deviance[e,f]	1.01 (0.98–1.05)	1.01 (0.97–1.04)	0.00	1.00 (0.95–1.04)
Late adolescence				
Education years[e]	**0.93 (0.92–0.94)**	**0.93 (0.91–0.94)**	0.00	1.00 (0.98–1.02)
History of trauma	1.06 (0.99–1.14)	**1.21 (1.10–1.34)**	**0.14**	**1.15 (1.01–1.30)**
Number of Axis I disorders excluding ND[e]	1.00 (0.95–1.06)	0.95 (0.89–1.02)	−0.05	0.95 (0.87–1.04)
Number of Personality disorders[e]	**0.89 (0.85–0.93)**	**1.09 (1.03–1.16)**	**0.21**	**1.23 (1.14–1.33)**
Adulthood[g]				
Ever divorced	**1.46 (1.36–1.56)**	**1.16 (1.05–1.29)**	**−0.23**	0.80 (0.71–0.90)
History of SUD				
Alcohol	**2.13 (1.96–2.32)**	1.05 (0.94–1.18)	**−0,71**	**0.49 (0.43–0.57)**
Cannabis	**1.91 (1.62–2.25)**	0.98 (0.82–1.17)	**−0,67**	**0.51 (0.40–0.66)**
Other drugs	**1.27 (1.07–1.53)**	1.03 (0.87–1.22)	−0,21	0.81 (0.64–1.02)
Regular physical exercise	**0.93 (0.87–1.00)**	**1.26 (1.11–1.42)**	**0.30**	**1.35 (1.18–1.53)**
Social deviance[e,f]	**1.12 (1.09–1.14)**	1.02 (1.00–1.04)	**−0.09**	**0.91 (0.89–0.94)**
Past year				
Social support[e]	**0.99 (0.99–1.00)**	**1.01 (1.00–1.02)**	**0.02**	**1.02 (1.01–1.03)**
Alcohol use disorders	1.01 (0.89–1.14)	**1.34 (1.15–1.56)**	**0.28**	**1.32 (1.10–1.59)**
Cannabis use disorders	0.98 (0.66–1.44)	1.02 (0.70–1.48)	0.04	1.04 (0.65–1.64)
Number of Axis I disorders excluding ND[e]	**1.10 (1.05–1.16)**	**1.32 (1.24–1.40)**	**0.18**	**1.20 (1.11–1.30)**
Religious service attendance	**0.65 (0.61–0.70)**	**0.65 (0.58–0.72)**	−0.01	0.99 (0.88–1.12)
Marital problems	0.93 (0.81–1.08)	**1.22 (1.00–1.49)**	**0.27**	**1.31 (1.01–1.69)**
Stressful life events[e]				
Independent[e]	**1.06 (1.02–1.10)**	**1.06 (1.01–1.11)**	0,00	1.00 (0.94–1.06)

	Smoking initiation with no ND[a] (n=11,785)	**Nicotine Dependence[b] (n=4,512)**	**Continuation ratio**	
	AOR (CI 95%)	**AOR (CI 95%)**	**Adjusted beta[c]**	**AOR (CI 95%)[d]**
Dependent[e]	1.00 (0.97–1.04)	**1.14 (1.08–1.20)**	**0.13**	**1.14 (1.06–1.21)**
Social deviance[e,f]	**1.07 (1.04–1.11)**	**1.10 (1.06–1.14)**	0.03	1.03 (0.98–1.08)

Controlling for age, ethnicity, sex and other covariates in the model;

[a] Reference group: never smokers;

[b] reference group: smokers without nicotine dependence;

[c] derived from the logistic regression model used to calculate the continuatio ratio (see text and Colea and Ananthb (2001) for explanation of the continuation ratio);

[d] AOR for the continuation ratio are derived from exponential function of the adjusted beta;

[e] for continuous variables, AORs reflect the change in the odds per unit of predictor;

[f] In early adolescence antisocial behavior before age 15; in adulthood since age 15; and past year between Wave 1 and Wave 2;

[g] lifetime but before 12 month; AOR= adjusted odds ratio; SUD= substance use disorders; ND= nicotine dependence; significant differences are bolded ($p<0.05$).