

In Press: Nicotine & Tobacco Research

Running Title: PSYCHOPATHOLOGY, FAMILIAL FACTORS, AND SMOKING

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Psychiatric Disorders, Familial Factors, and Cigarette Smoking:

1. Associations with Smoking Initiation

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This research was supported by DA-11299 to P.R., and MH- 40501, MH-50522, and MH-52858 to P.M.L.

Total number of pages: 50

Word Count: 11,180

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Abstract

Aims. (1) Examine associations between smoking initiation (five cigarettes lifetime) and lifetime psychopathology, regular smoking by family members, and psychopathology in family members. (2) Describe the degree to which the onset of disorder precedes or follows smoking initiation. (3) Examine whether smoking initiators differ as a function of age of smoking onset.

Method. 941 participants were interviewed at three time points, beginning in high school and most recently at age 24. Lifetime psychiatric diagnoses were obtained at each assessment, as were data regarding smoking initiation. Biological parents and full siblings were interviewed for lifetime psychopathology and regular smoking.

Results. Most measures of lifetime psychopathology were associated with smoking initiation. Rates of initiation were especially elevated in participants with multiple disorders. Regular smoking by mother and a sibling (but not father) was associated significantly with smoking initiation, as were two of four measures of psychopathology in relatives. When all significant univariate variables were examined in a single model, drug use disorders, regular smoking by mother, and regular smoking by a sibling remained significantly associated with smoking initiation. Smoking initiation preceded approximately half of the examined diagnostic

categories. Eleven variables differentiated early versus late smoking initiators. Several interactions with sex were found. In every instance, smoking initiation was more strongly associated with the risk factor for young women than for young men.

Conclusions. To our knowledge, this is the first time that the relations of psychopathology and familial factors with smoking initiation have been examined simultaneously or in this much detail. Results underscore the potential importance of assessing and treating psychiatric disorders in smoking prevention and cessation efforts.

Adolescence is a critical time to evaluate the factors associated with cigarette smoking. First, almost all tobacco use begins during this time (e.g., Chassin, Presson, Rose, & Sherman, 1996). Second, cigarette smoking is common among adolescents. By age 18, approximately two-thirds of U.S. adolescents have tried smoking (e.g., Johnston, O'Malley, & Bachman, 1996). Third, despite concerted efforts, adolescent smoking prevention programs have generally resulted in limited success (Rooney & Murray, 1996). Finally, earlier onset of cigarette smoking is associated with heavier use, nicotine dependence, and lower likelihood of smoking cessation (Breslau, Fenn, & Peterson, 1993; Escobedo, Marcus, Holtzman, & Giovino, 1993; Janson, 1999; Khuder, Dayal, & Mutig, 1999; Taioli & Wynder, 1991). Thus, even delaying the onset of smoking may have significant public health benefits.

The primary goal of the present study is to examine psychiatric and familial factors associated with the initiation of cigarette smoking. Cigarette smoking is a complex behavior influenced by multiple factors, which can be categorized as demographic, developmental, psychosocial, cognitive and attitudinal, psychiatric, genetic, and regulatory (Derzon & Lipsey, 1999; Moolchan, Ernst, & Henningfield, 2000). We do not attempt in the present study to inclusively examine all of the potentially salient factors. Instead, we focus on the associations between smoking initiation and three categories of risk factors: (a) lifetime psychopathology in the individual, (b) regular smoking by his or her family members (parents and siblings), and (c) psychopathology in the family members. The relevance of each category is discussed next.

Associations with Lifetime Psychopathology

Associations between most psychiatric disorders and cigarette smoking have been

repeatedly shown for adults (e.g., Breslau, Kilbey, & Andreski, 1991; Covey, Glassman, Stetner, 1998; Degenhardt & Hall, 2001), and to a lesser extent, adolescents (e.g., Brown, Lewinsohn, Seeley, & Wagner, 1996). For example, in the National Comorbidity Study, adults with a lifetime history of mental illness were twice as likely as those with no mental disorder to smoke (Lasser et al., 2000). As much as possible, our review of this literature focuses on the associations with psychopathology at the level of diagnosis, as opposed to a more extensive literature examining associations between smoking and subthreshold levels of psychiatric symptoms.

Probably the strongest associations with smoking have been found for substance use disorders (Breslau et al., 1991; Brown et al., 1996; Kendler et al., 1999; Riggs, Mikulich, Whitmore, & Crowley, 1999) and attention-deficit/disruptive behavior disorders (i.e., attention-deficit/hyperactivity disorder [ADHD], conduct disorder, and oppositional defiant disorder) (Brown et al., 1996; Milberger, Biederman, Faraone, Chen, & Jones, 1997; Pomerleau, Downey, Stelson, & Pomerleau, 1995; Wilens et al., 2000). The associations between smoking and substance use disorder tend to remain significant even when controlling for the presence of comorbidity (e.g., Madden, Bucholz, Martin, & Heath, 2000).

Although smaller in magnitude, associations have also been reported for smoking with internalizing disorders. Compared to nonsmokers, adolescent and adult smokers have higher rates of major depressive disorder (MDD) (Breslau et al., 1991; Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Brown et al., 1996; Glassman et al., 1990; Kendler et al., 1993; 1999), although some exceptions have been noted (e.g., Covey, Hughes, Glassman, Blazer, & George, 1994; Kandel et al., 1997). The association between smoking and MDD remains

significant even when controlling for other psychiatric disorders (Breslau et al., 1991; Kendler et al., 1993), although it may decrease in magnitude (Breslau et al., 1998). The associations between anxiety disorders and smoking in adults appear to be weaker (Breslau et al., 1991; Breslau & Klein, 1999; Covey, Hughes, Glassman, Blazer, & George, 1994; Glassman et al., 1990; Johnson et al., 2000) and have generally been examined only at the level of heavy smoking, although Kendler et al. (1999) reported that generalized anxiety, panic, and phobic disorders were significantly associated with lifetime smoking initiation.

Smoking also has been shown to be associated with eating disorder symptoms and diagnosis (Crisp, Sedgwick, Halek, Joughin, & Humphrey, 1999; Welch & Fairburn, 1998). Lastly, the strongest associations with smoking may involve schizophrenia and certain personality disorders, especially antisocial personality disorder (Barry, Fleming, Manwell, & Copeland, 1997; Goff, Henderson, & Amico, 1992; Hughes, Hatsukami, Mitchell, & Dahlgren, 1986; Johnson, Hyler, Skodol, Bornstein, & Sherman, 1995; Tanskanen, Viinamaeki, Koivumaa-Honkanen, Jaaeskelaenen, & Lehtonen, 1998).

There is some indication that the rates of smoking may be especially elevated given the presence of multiple (comorbid) psychiatric disorders (e.g., Breslau et al., 1991; Hughes et al., 1986; Milberger et al., 1997; Miller-Johnson, Lochman, Coie, Terry, & Hyman, 1998; Riggs et al., 1999). For example, Milberger et al. (1997) reported that approximately 10% of male adolescents with pure (noncomorbid) ADHD were daily smokers, with rates increasing linearly to roughly 40% for those with ADHD plus three psychiatric comorbidities.

Given a significant association between a psychiatric disorder and smoking initiation, we

examine the temporal order of smoking initiation and psychiatric disorder. The temporal order of co-occurring problems may provide important etiologic clues to both difficulties (e.g., one problem may be a risk factor for, or an early manifestation of, the second problem). For example, Breslau et al. (1998) found that smoking (at the level of daily use) and MDD were each predictive of the other outcome in a five-year longitudinal study of young adults. Although many psychiatric disorders develop prior to smoking (Costello, Erkanli, Federman, & Angold, 1999) and smoking has been conceptualized as a self-medication for the distress of psychiatric symptoms (e.g., Carmody, 1989), childhood and adolescent psychopathology have not been found to predict future smoking behavior into early adulthood (e.g., Brook, Cohen, & Brook, 1998; McGee, Williams, & Stanton, 1998).

Associations with Familial Smoking

We also examine the degree to which regular cigarette use in family members (biological parents and full siblings) is associated with smoking initiation. Parental smoking has been found to be significantly associated with adolescent smoking in approximately half of the examined studies (e.g., Bauman, Foshee, Linzer, & Koch, 1990; Brook et al., 1997; Chassin, Presson, Montello, Sherman, & McGrew, 1986; Griesler & Kandel, 1998; Hunter, Croft, Vitzelberg, & Berenson, 1987; Najem, Batuman, Smith, & Feuerman, 1997; Rose, Chassin, Presson, & Sherman, 1999), and estimates of the heritability for cigarette smoking (i.e., the proportion of variance attributable to genetic sources) average approximately 50% (e.g., Han, McGue, & Iacono, 1999; Kendler et al., 1999; Madden et al., 1999; Maes et al., 1999; Swan & Carmelli, 1997; True et al., 1997; True et al., 1999). Studies also indicate that smoking by one's siblings is

significantly predictive of smoking in young people (e.g., Bierut et al., 1998; Pederson, Koval, & O'Connor, 1997; West, Sweeting, & Ecob, 1999). While smoking by family members has been shown to be associated with smoking initiation, the influence of parent and sibling smoking may apply to smoking of mid-adolescents but not young adults and generally is smaller in magnitude than the effect of peer smoking (Unger & Chen, 1999; Wang, Fitzhugh, Westerfield, & Eddy, 1995; West et al., 1999).

Associations with Familial Psychopathology

Given our focus on psychiatric disorders and familial factors in the present study, the last set of variables we examine consists of psychopathology in one's family members. To our knowledge, only a handful of studies have examined the role of familial psychopathology in relation to smoking. With the exception of one study, associations have been positive. Merikangas, Dierker, and Szatmari (1998) reported that the child/adolescent offspring of parents with substance use disorders were at elevated risk for tobacco use, compared to offspring of either parents with anxiety disorders or parents with no mental illness. In another report using the same data set, Dierker, Avenevoli, Merikangas, Flaherty, & Stolar (2001) found that the offspring of parents with substance use disorders had higher rates of regular (weekly or more) smoking and nicotine dependence, but did not differ on rates of smoking experimentation. Kendler and associates (1993) reported that adult women with a family history of MDD had a greater likelihood of smoking, even after controlling for personal history of MDD. In the one study that did not find a significant relationship (Costello et al., 1999), parental mental illness (as assessed by mental health treatment, history of suicide attempts, current maternal depression

symptoms) was not associated with either prevalence or onset of any substance use in offspring, including cigarette smoking.

The Present Study

The present study makes use of data from the Oregon Adolescent Depression Project (OADP; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Lewinsohn et al., 1994). As part of this longitudinal project of depression and other psychopathology, extensive information regarding smoking status of a large, randomly-selected representative sample of older adolescents assessed twice in adolescence (T1 and T2) and at a third point (T3) after age 24. In previous papers, we have reported the cross-sectional and prospective relationships between cigarette use and psychopathology in adolescence (i.e., T1 and T2; Brown et al., 1996; Lewinsohn, Brown, Seeley, & Ramsey, 2000), and the progression from cigarette smoking in adolescence (T1/T2) to the development of substance use disorders by young adulthood (i.e., T3; Lewinsohn, Rohde, & Brown, 1999). Near the time of the T3 assessment, we collected lifetime diagnostic and smoking data on the first-degree relatives of OADP probands. In the current paper, we extend our previous research by using the three waves of psychiatric assessments from the OADP in conjunction with data from family members. Using logistic regression analyses, the univariate and multivariate associations of psychopathology, familial smoking and psychopathology with smoking initiation are examined. Temporal associations between smoking initiation and onset of the psychiatric disorder are examined. In addition, given the deleterious impact of early smoking onset, we investigate the degree to which the variables distinguish early versus late smoking onset.

Throughout the analyses, sex differences are considered. Compared with men, the rate of smoking among women has increased disproportionately, and current smoking prevalence rates for U.S. men and women are roughly comparable (Escobedo & Peddicord 1997; US Department of Health and Human Services 2001). However, the pathways into smoking initiation and maintenance may vary as a function of sex (e.g., Costello et al., 1999; Kandel et al., 1997; Martin, Milich, Martin, Hartung, & Haigler, 1997). In Kandel et al. (1997), the lowest frequency category of smoking (i.e., lifetime smoking but no use in the past six months) was associated with substance use and disruptive behavior disorders for female children and adolescents but not male children/adolescents; conversely, the associations of low levels of smoking with mood disorders were significant for male children/adolescents but not female children/adolescents. Costello et al. (1999) also found that depression was associated with smoking (at the daily level) for males but not females.

Method

Participants and Procedures

OADP participants. A total of 1,709 adolescents, randomly selected from nine high schools in western Oregon (mean age = 16.6, range = 14-18 years of age; 61% participation) completed the initial (T1) assessments between 1987 and 1989 (see Lewinsohn et al., 1993 for additional details). Approximately one year later (T2), 1,507 participants (88%) returned for a re-administration of the interview and questionnaire (mean T1-T2 interval = 13.8 months, SD = 2.3). Although demographic and diagnostic differences between T2 participants and those who discontinued participation were small (Lewinsohn et al., 1993), T1 participants who had initiated

smoking were more likely to drop out of the study compared to T1 participants who had never smoked; 85.6% of T1 participants with smoking initiation completed T2, compared to 90.5% of T1 participants who had never smoked; $\chi^2(1, 1709) = 9.88, p < .01$.

Additional funding was received to follow the course of psychopathology among OADP participants into young adulthood. Between 1993 and 1999, as participants reached their 24th birthday, all individuals with a history of psychopathology and a randomly selected set of participants with no history of mental disorder ($N = 1,101$) were invited to the T3 telephone interview. Sampling of the no-disorder comparison group was proportional to age and sex within age; all participants with non-white ethnicity were retained in the sample. Given our focus on the course of psychopathology into young adulthood, the T3 sample actually contained a slightly larger proportion of smoking initiators (64.7% of participants with smoking initiation by T2 completed T3) compared to T2 participants who either were not invited or failed to complete the T3 interview (59.8% of T2 never smokers completed T3); $\chi^2(1, 1507) = 3.91, p < .05$.

T3 data were obtained from 941 individuals (85% participation of selected T2 sample; 55% of original T1 sample), with a mean interval between T2 and T3 of 6.8 years ($SD = 1.4$). A total of 57.3% of the T3 participants were female, 89.1% were White, 54.3% had lived with both biological parents at T1; and 45.3% had one or more parents with a college education at T1. Attrition at T3 was associated with male sex (57% of T3 participants were female versus 42% of T3 non-participants); $\chi^2(1, 1101) = 13.54, p < .001$, and with younger age at T2 (T3 participants $M = 17.8$ [$SD = 1.3$] versus T3 non-participants $M = 17.5$ [$SD = 1.2$] years old); $t(1095) = 2.64, p < .01$, but was unrelated to race/ethnicity, maximum parental education, or residing with both

biological parents at T2.

Family members. As a separate project, biological parents and full siblings of the T3 participants were recruited and interviewed for lifetime psychopathology and smoking, with the goal of obtaining two sources of data for each family member (either direct and informant interviews or two informant interviews). Data were obtained between 1995 and 1998 on 806 families (86% of T3 interviewed participants), which represented 2,646 individuals (803 mothers, 788 fathers, and 1,055 siblings). Approximately two-thirds of the biological fathers (67.6%) resided with the OADP participant at T1, as had 89.1% of the biological mothers. Mean age of the siblings at the time of their assessment was 25.2 years (SD = 5.7).

Assessment of Psychopathology and Smoking Initiation in the OADP Participant

Psychiatric disorders. Participants were interviewed at T1 with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982) designed to assess all major disorders as per DSM-III-R criteria. Participants at T2 and T3 were interviewed using the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987), which provided detailed information about the onset and course of disorders since the T1 interview.

Almost all diagnostic interviewers (for T1 and T2 initially, and subsequently for T3 and the Family Study project) had advanced degrees in clinical or counseling psychology or social work and all completed a 70-hour didactic and experiential course in diagnostic interviewing. Prior to conducting interviews, all interviewers were required to demonstrate a minimum kappa (Cohen, 1960) of .80 across all symptoms for at least two consecutive training interviews. All

interviews were recorded and a randomly-selected 12% were reviewed by a second interviewer. With few exceptions, inter-rater kappas for current and lifetime diagnoses at T1 and T2 exceeded .80 (Lewinsohn et al., 1993).

The T3 interview repeated the T2 procedures, assessing all disorders as per DSM-IV criteria. Because a significant proportion of participants no longer resided in the area, we shifted to a telephone assessment format for the T3 diagnostic assessments. Although telephone surveys have become standard procedures in behavioral science research, the comparability of telephone and face-to-face interviews for assessing psychiatric disorder had not been clearly established. Therefore, we examined this question in Rohde, Lewinsohn, and Seeley (1997). As part of the T3 assessment, 60 participants were interviewed face-to-face and over the telephone regarding Axis I disorders and an additional 60 participants were interviewed twice regarding all Axis II disorders. Diagnostic agreement in the telephone and face-to-face assessment formats was excellent for anxiety disorder (kappa = .84), and was very good for (non-alcohol) SUD, alcohol use disorders, and MDD (kappas = .73, .70, and .67 respectively). In addition, inter-rater reliabilities for Axis I disorders also were very good to excellent (Rohde et al., 1997).

Antisocial and borderline personality disorders were also assessed in the T3 interview, using relevant portions of the Personality Disorder Examination (PDE; Loranger 1988). Because the rates of antisocial and borderline personality disorders were very low (1.3% each), the PDE dimensional scores were dichotomized at approximately the 90th percentile (antisocial cutoff score ≥ 7 ; borderline cutoff score ≥ 4) and used as an indicator of Axis II psychopathology. Inter-rater reliability of the PDE dimensional scores (i.e., summation of partial and full symptom

criteria) was excellent (Interclass correlations $> .80$).

On the basis of adequate prevalence rates (5% or greater), eight diagnostic categories of lifetime (to T3) psychopathology were examined: (1) MDD ($n = 487$, 51.8%); (2) dysthymia ($n = 47$, 5.0%); (3) alcohol use disorders (ALCOHOL; $n = 310$, 32.9%) (4) drug (other than alcohol) use disorders (DRUG; $n = 216$, 23.0%), (5) anxiety disorders ($n = 194$, 20.6%), (6) ADHD and disruptive behavior disorders (ADHD/DIS; $n = 102$, 10.8%); (7) elevated antisocial personality disorder symptomatology ($n = 88$, 9.4%), and (8) elevated borderline personality symptomatology ($n = 50$, 5.3%). Prevalence rates reflect the oversampling of those with a history of mental illness at T3.

Smoking initiation. As part of the T1-T3 interviews, information was obtained regarding smoking initiation, which was defined as having smoked five or more cigarettes in one's lifetime. Initiation by age 24 was reported by 722 of the 941 T3 participants (76.7%). Information regarding the age of smoking initiation was used from the first available assessment point. The majority of participants who initiated smoking did so before the T1 interview (530, 73.0% of initiators). Of these 530 individuals, smoking initiation occurred on average 4.3 years prior to T1 ($SD = 2.8$). To increase the reliability and validity of reporting, participants at each assessment were assured that smoking information would be kept confidential.

Assessment of Psychopathology and Regular Smoking in Family Members

Parents and siblings over the age of 18 were interviewed with the Structured Clinical Interview for DSM-IV, nonpatient version (SCID-NP; Spitzer, Williams, Gibbon, & First, 1992) and the Antisocial and Borderline Personality Disorders sections of the SCID-II. Siblings

between the ages of 14 and 18 received the version of the K-SADS employed in the T1 OADP assessment, updated to assess disorders as per DSM-IV criteria.

Wherever possible, two sources of lifetime psychiatric information for each family member are obtained: a direct interview with the family member and an informant report. All OADP participants were asked to provide diagnostic information about their first degree relatives as per the Family Informant Schedule and Criteria (FISC; Mannuzza & Fyer 1990). Other family members (most often the mother) were asked to provide additional informant data, if necessary. Interviews were conducted blind to OADP participant diagnoses and each interviewer evaluated no more than two members of any family. Inter-rater reliabilities have previously been shown to be very good to excellent (Lewinsohn, Rohde, Klein, & Seeley, 1999). Best-estimate diagnoses (Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982) were derived for all relatives using all available data (direct and family history interviews and treatment records) by the four doctoral-level diagnosticians.

Family psychopathology, defined as having one or more family members with the disorder of interest, was examined for the following categories: (1) affective disorders (61.8% of participants had family member with depression), (2) drug and alcohol use disorders (70.7% of participants), (3) anxiety disorders (31.9% of participants), and (4) externalizing disorders, which included ADHD, conduct disorder, oppositional defiant disorder, and elevated antisocial personality disorder scores (22.3% of participants). Again, it should be noted that these rates are higher than would be expected in a randomly selected community sample because participants with a history of mental illness were oversampled at T3.

Regular smoking among family members. As part of the diagnostic interview, information regarding tobacco use by the family member was obtained. Three variables were created based on whether the mother, the father, or a sibling (one or more) had ever been a regular smoker, which was defined as smoking 10 or more cigarettes a day for at least one month. Of the 803 families in which psychopathology data were obtained, smoking data were available for 97% of relatives. Lifetime rates of regular smoking were 43.5% for mothers, 59.2% for fathers, and 10.1% for siblings.

Statistical Analyses

Associations between smoking initiation and the lifetime occurrence of psychiatric disorders, family smoking, and family psychopathology were examined using logistic regression (LR) models. The presence or absence of smoking initiation and psychiatric disorder category by age 24 was calculated by combining data from the T1, T2, and T3 waves of assessment. Four T1 demographic variables were examined as potential confounds: Male gender (Yes/No), White ethnicity (Yes/No), Non-intact home (was the participant living with both biological parents at T1? Yes/No), Low parental education (had either parent completed college at T1? Yes/No). The statistical significance of effects was determined by odd ratio (OR), with 95% confidence interval (CI). Because we did not attempt to bring back the entire T2 sample for T3, the univariate analyses using T3 data were re-analyzed using a weighted approach (SUDDAN). Observed and weighted ORs had only trivial differences in magnitude, with none changing in significance. Given the absence of differences, observed associations are reported in the present study. All variables with significant univariate association were examined in a summary multiple logistic

regression (MLR) to determine significant associations controlling for other effects. For the psychiatric disorders that were associated with smoking initiation, temporal patterns between smoking onset and psychiatric disorder onset were examined in participants with both disorder and smoking initiation by comparing whether the proportion of participants in which smoking preceded or followed the disorder significantly differed from 50% (i.e., chance), using a critical ratio z test. Lastly, participants who had initiated smoking were divided into early versus late onset, using a median split (14 years of age), and were compared on the examined variables using LR and MLR analyses. In each set of analyses, the interactions between predictor variables and sex were tested to determine whether sex moderated the observed associations. Given our sample size, we had adequate statistical power to detect small effects in sex interactions. An alpha level of $p < .05$ was set to determine the significance of the associations tested.

Results

Univariate Associations with Smoking Initiation

Univariate associations were examined in a series of LR analyses. In each model, the independent variable was entered, followed by sex, and the interaction with sex (unless noted, interactions with sex were nonsignificant). Significant unadjusted associations (odds ratio; OR) are shown in Table 1. Results are discussed by category.

Insert Table 1 about here

Demographics. None of the four demographic variables had a significant association with

smoking initiation: sex (male/female), race (white/nonwhite), T1 living situation (living with both biological parents/other), or maximum parental education level (one or both parents completed college/neither parent completed college).

Lifetime psychopathology. Most but not all lifetime psychiatric categories were associated with smoking initiation by age 24. Significant associations included MDD, ALCOHOL, DRUG, ADHD/DIS, and the two personality disorder summary scores. Nonsignificant associated include dysthymia ($p = .278$) and anxiety disorders ($p = .061$).

In addition to the significant main effects, two diagnostic categories interacted significantly with sex in their association with smoking initiation. In both cases, the association with smoking initiation was significant for both sexes, but greater in magnitude for the young women. The first interaction was with ALCOHOL, improvement $\chi^2(1, 941) = 4.53, p < .05$. The OR for ALCOHOL was 3.13, $p < .001$; (95% CI = 1.85-5.28) for the male participants, and 8.80, $p < .001$ (95% CI = 3.77-20.52) for the female participants. The second interaction was with DRUG, improvement $\chi^2(1, 941) = 6.45, p < .05$. The OR for DRUG was 3.53, $p < .001$; (95% CI = 2.12-5.88) for the male participants, and 11.15, $p < .001$ (95% CI = 5.06-24.56) for the female participants.

To examine the impact of psychiatric comorbidity, participants were categorized on the basis of number of lifetime psychiatric disorders (0, 1, 2, 3, 4 or more). Differences in smoking initiation rates for the five groups were significant; $\chi^2(4, 941) = 76.36, p < .001$, with 60.5%, 75.3%, 82.7%, 86.5%, and 100.0% of individuals in each of the five categories having tried smoking, respectively. Given the pattern of differences, we computed a single variable

contrasting participants with one disorder versus those with two or more disorders (shown in Table 1).

Family member smoking. Regular smoking by mothers and siblings, but not by fathers, was associated with smoking initiation in the participants.

Family psychopathology. Associations with smoking initiation were present for two of the four diagnostic categories of psychopathology in the relatives (i.e., affective disorders, drug and alcohol use disorders).

Multivariate Associations with Smoking Initiation

To determine the magnitude of associations with smoking initiation controlling for associations with other variables, variables with a significant univariate association were entered simultaneously into a summary MLR analysis. The initial multivariate analysis included the two sex interactions, which were not significant in the summary model. The MLR was recomputed examining only main effects. Three variables remained significant: (a) DRUG, adjusted OR = 9.14, $p < .01$ (95% CI = 2.16-38.70); (b) Regular smoking by mother, adjusted OR = 1.56, $p < .05$, (95% CI = 1.02-2.38); and (c) Regular smoking by a sibling, adjusted OR = 1.44, $p < .05$, (95% CI = 1.02-2.05). To appreciate the magnitude of association, rates of smoking initiation among participants with 0, 1, 2, or 3 of the MLR risk factors were 59.9%, 79.0%, 89.4%, and 89.4%, respectively.

Temporal Order of Smoking Initiation and Psychiatric Disorder

The temporal patterns between smoking and Axis I psychiatric disorder were examined using participants who had both smoking initiation and the disorder of interest. Although not

significant in the previous analyses, anxiety disorders were included because they had approached significance and because they occurred in relatively high frequency (as opposed to dysthymia). For each diagnostic category, participants with both outcomes (i.e., the disorder and smoking initiation) were divided into those who began smoking first versus those who experienced the disorder first. Consistent with scoring conventions for semi-structured diagnostic interview, onset of psychiatric disorder was considered the time at which the participant met full criteria for the disorder. A small number of participants ($n = 32$) experienced the psychiatric disorder and initiated smoking during the same month and were included in “disorder first” category, based on the assumption that some symptoms of the psychiatric disorder probably were present prior to the individual meeting full criteria for diagnosis. Given that diagnoses in the ADHD/DIS and anxiety disorder categories included disorders that had a required childhood onset (e.g., ADHD and separation anxiety disorder, respectively), individual disorders were examined in addition to the summary category. Whether smoking initiation preceded or followed disorder onset was examined using the critical ratio z test (comparing onset to a chance value of 50%). Results are summarized in Table 2. As can be seen, smoking initiation tended to occur prior to the MDD, ALCOHOL, DRUG, CD, and panic disorder. Not surprising given diagnostic requirements, smoking initiation tended to occur after the onset of ADHD and separation anxiety disorder (as well as the summary category of anxiety disorder). No significant temporal patterns were present for ADHD/DIS, oppositional defiant disorder, or phobias.

Insert Table 2 about here

Impact of Early Smoking Initiation

To determine whether the examined variables were associated with early smoking initiation, participants who had initiated smoking were divided into early versus late onset, using a median split (14 years of age; 44.7% had early onset). The two groups were compared on the previously-examined variables using an LR approach. Significant results are shown in Table 3.

Insert Table 3 about here

Eleven of the 20 examined variables were significantly associated with early smoking initiators: two demographic variables (not living with both biological parents, low parental education), six measures of lifetime psychopathology (ALCOHOL, DRUG, anxiety, ADHD/DIS, elevated antisocial personality disorder score, comorbidity), two measures of familial smoking (mother smoking, father smoking), and one measure of familial psychopathology (drug and alcohol).

In addition, four interactions with sex were identified. In each case, the risk factor was more strongly associated with early smoking in the women than in the men. The first interaction was with DRUG; improvement $\chi^2(1, 722) = 6.32, p < .05$. Differences between early versus late initiation for the men were nonsignificant; $\chi^2(1, 306) = 2.85, ns$; OR = 1.51 (95% CI = 0.93-

2.45). For the women, 37.6% of early onset smokers had DRUG compared to 14.4% of late onset smokers; $\chi^2(1, 416) = 29.57, p < .001$; OR = 3.58 (95% CI = 2.23-5.75). The second interaction was with anxiety disorders; improvement $\chi^2(1, 722) = 5.09, p < .05$. The association for men was again nonsignificant; $\chi^2(1, 306) = 1.00, ns$; OR = 0.69 (95% CI = 0.33-1.44). For women, 36.1% of early onset smokers had anxiety disorders compared to 23.9% of late onset smokers; $\chi^2(1, 416) = 7.41, p < .01$; OR = 1.80 (95% CI = 1.18-2.75). The third interaction was with elevated antisocial personality disorder scores; improvement $\chi^2(1, 699) = 4.84, p < .05$. For men, 26.6% of early onset smokers had elevated antisocial personality disorder scores compared to 14.7% of late onset smokers; $\chi^2(1, 298) = 6.46, p < .05$; OR = 2.10 (95% CI = 1.18-3.74). For women, 10.7% of early onset smokers had elevated antisocial personality disorder scores compared to 1.4% of late onset smokers; $\chi^2(1, 401) = 15.94, p < .001$; OR = 8.41 (95% CI = 2.46-28.79). The last interaction was with a family history of affective disorder, improvement $\chi^2(1, 722) = 44.6, p < .05$. The association was nonsignificant for the men; $\chi^2(1, 253) = 0.93, ns$; OR = 0.78 (95% CI = 0.47-1.30). For women, 72.3% of early onset smokers had a family history of affective disorder compared to 61.8% of late onset smokers; $\chi^2(1, 364) = 4.49, p < .05$; OR = 1.61 (95% CI = 1.03-2.51).

To examine the magnitude of association of variables with early smoking initiation when examined simultaneously, all variables with a significant univariate association were entered into a single MLR analysis. Three variables were significant in the multivariate model, two of which were lifetime diagnostic categories: (a) DRUG, adjusted OR = 1.89 (95% CI = 1.47-2.43), and (b) ADHD/DIS, adjusted OR = 1.46 (95% CI = 1.10-1.94). Although none of the sex interactions

were significant in the multivariate model, female sex emerged as a significant main effect when all variables were examined simultaneously (42.4% of male smokers and 46.6% of female smokers had early onset), adjusted OR = 1.22 (95% CI = 1.01-1.46). The rates of early smoking initiation increased from 32.3%, 43.8%, 50.6%, to 57.1% for groups with 0, 1, 2, or 3 risk factors, respectively.

Discussion

Lifetime Psychopathology

Not surprisingly, participants with lifetime psychopathology were more likely to initiate smoking cigarettes. The majority of examined associations with lifetime psychopathology were significant, with significant odds ratios ranging 1.8 (major depressive disorder) to 5.1 (drug use disorders). This suggests that the likelihood of trying cigarette smoking is almost two to over five times greater, given the particular psychiatric disorder.

Associations with smoking initiation were nonsignificant for two categories of lifetime psychopathology: dysthymia and eating disorders. One of these diagnostic categories (i.e., dysthymia) had a lifetime prevalence rate of 5% in the sample, which may have limited statistical power to detect differences. Regarding anxiety disorders, evaluating the associations with smoking is complicated by the fact that the category includes so many different disorders and the exact composition of disorders in our study is both age- and sample-specific. While several studies report highly significant associations between smoking and specific anxiety disorders (e.g., Breslau & Klein, 1999; Johnson et al., 2000), these relationships are generally at heavier levels of cigarette smoking. Consistent with the present study, Cornelius et al. (2001) found no

association between current smoking (at any level) and a summary category of anxiety disorders.

The rates of cigarette initiation were particularly elevated in participants who experienced multiple psychiatric disorders. Of the 92 participants with four or more disorders (9.8% of the T3 sample), 100.0% had tried smoking versus 60% of the participants with no history of a mental disorder. Future research needs to examine whether specific combinations of comorbid psychopathology are associated with especially high rates of smoking initiation.

When all significant univariate variables were examined in a single model, only one category of the participant's psychopathology (drug use disorders) remained significant. Drug use disorders also were one of the categories most strongly associated with earlier smoking initiation. Thus, this diagnostic category is clearly among the most closely associated with smoking initiation. Interestingly, alcohol use disorders had significant univariate associations with both smoking initiation in the total sample and with early initiation among participants who smoked, but was nonsignificant in both MLR analyses. Presumably, this pattern was due to the fact that participants with alcohol use disorders also were likely to have drug use disorders (i.e., the alcohol use disorders accounted for no additional variance beyond drug use disorders). Consistent with the present study, Black, Zimmerman, and Coryell (1999) found that smoking was associated with most psychiatric disorders in univariate analyses but only alcohol and drug use disorders were independently associated with smoking after controlling for comorbidity among disorders. ADHD and the disruptive behavior disorders in the present study had the same pattern as alcohol use disorders (i.e., significant univariate association, nonsignificant in the summary MLR).

Participants with a history of major depressive disorder were more likely to have initiated smoking by age 24, and both major depressive disorder and anxiety disorders were associated with early initiation. Neither category of internalizing disorder, however, contributed significant independent variance in the multivariate analyses. The association between smoking and depression has been reported in some but not all studies. For example, daily cigarette smoking in a cross-sectional study of 9-18 year old adolescents was associated with substance use, attention-deficit/disruptive behavior, and anxiety disorders but not mood disorders (Kandel et al., 1997). The associations of internalizing disorders with smoking may apply to only a subset of young people. For example, Patton and associates (1998) reported that depression and anxiety predicted smoking initiation but only in the presence of peer smoking.

Given that participants with certain psychiatric disorders were more likely to have initiated smoking, our second aim was to describe the degree to which the psychiatric disorder consistently preceded or followed smoking initiation. The interview procedure dated the onset of a psychiatric disorder as the point in time at which the person met full diagnostic criteria. Therefore, we cannot determine if prodromal symptoms were present or if prodromal symptoms were associated with smoking initiation prior to the person developing the psychiatric disorder. While these data may have revealed some interesting associations between symptoms and smoking initiation, we believe that a strength of the present study is our focus on episodes of psychiatric disorder, as opposed to elevated but subthreshold symptomatology. With these caveats in mind, smoking initiation generally occurred before episodes of major depressive, drug use, and alcohol use disorders. The patterns were mixed for the summary categories of attention-

deficit/disruptive behavior and anxiety disorders, presumably due to differences in the average onset age for disorders within these categories. For example, smoking initiation almost always followed the onset of ADHD and always preceded the onset of conduct disorder in the present study. Dierker et al. (2001) examined whether smoking onset preceded or followed onset of disorders. Their results were generally consistent with the present study: anxiety disorders and ADHD tended to precede smoking initiation, alcohol and drug use disorders and MDD tended to follow, and no consistent pattern emerged for conduct and oppositional defiant disorders.

The third aim of our study was to identify factors that differentiated early versus late onset of smoking initiation. Earlier onset smoking has been shown to be associated with greater likelihood of subsequent severe nicotine addiction (Breslau & Peterson 1996; Taioli & Wynder 1991). Approximately half of the examined variables distinguished the early versus late smoking initiators. Whereas the presence of an anxiety disorder was associated with earlier initiation in the present study, Costello et al. (1999) found that the presence of anxiety disorders were associated with later onset of smoking (a delay of approximately one year). As noted earlier, anxiety disorders are a heterogeneous group and the composition may vary as a function of the sample. When examined simultaneously, alcohol and drug use disorders and attention-deficit/disruptive behavior disorders remained significantly associated with early smoking initiation, which is consistent with previous research (Milberger et al. 1997; Riggs et al., 1999).

Familial Smoking

The second major category of predictor variables in the present study consisted of regular smoking by one's mother, father, or siblings. Study participants with mothers or siblings who

were regular smoker were significantly more likely to have initiated smoking by age 24. Associations with regular smoking among fathers were in the same direction but statistically nonsignificant. Regarding early smoking initiation, participants whose parents (either mother or father) regularly smoked were more likely to begin smoking earlier; the association with early onset smoking initiation was unrelated to having a sibling who smoked. Perhaps most interesting, when all variables with a significant univariate association were examined simultaneously in a single model, two of the family smoking variables remained significant in the model predicting smoking initiation in the total sample (although none of the family smoking measures were retained in the model predicting early smoking onset among the initiators). Thus, the variance associated with these factors appears to be uniquely associated with smoking initiation, although the association of psychopathology with early smoking initiation may be more salient than the impact of family smoking.

The association between parent cigarette use and adolescent cigarette use has been inconsistent, found in some studies (e.g., Brook et al., 1997) but not others (e.g., Andrews, Hops, Ary, Tildesley, & Harris, 1993; Wang et al., 1995; 1999). West et al. (1999) examined the impact of smoking by family and friends in predicting uptake of regular smoking in young people at three time points (15-16, 16-18, 18-21 years of age). Parental and sibling smoking were significant at the first assessment point only; the effect of parental smoking became nonsignificant in multivariate analyses (in which peer smoking had the strongest effect in the 15-16 age period). Consistent with this pattern, Britt & Jachym (1996) also found that peer effects were stronger than sibling effects in predicting cigarette use by children.

Familial Psychopathology

The third category of examined variables consisted of lifetime psychopathology in one's family. Although a good deal of research has examined the impact of parental substance abuse on alcohol and drug use problems in offspring, familial psychopathology has seldom been examined in association with adolescent smoking initiation. Participants with family histories of affective and drug/alcohol disorders were more likely to have initiated smoking by 24 years of age. In addition, the presence of drug/alcohol disorders in one's relatives was associated with earlier smoking initiation. Dierker et al. (2001) also reported that adolescents of parents with substance use disorders tended to report an earlier age of smoking initiation (11.5 vs. 13.5 years of age). In the present study, a family history of affective disorders also was associated with earlier smoking initiation, but only among female participants. The occurrence of anxiety disorders or externalizing disorders in one's family members was unrelated to both smoking initiation in general and earlier initiation specifically.

When all significant univariate associations were examined in the multivariate model, none of the family psychopathology variables remained significant, suggesting that the impact of familial psychopathology was accounted for by either psychopathology in the participant or smoking in the family member. Consistent with these possibilities, a family history of substance use disorder was significantly correlated with all three variables retained in summary MLR model ($r_s = .15-.24$, all $p < .001$), a family history of affective disorder was significantly correlated with regular smoking by sibling ($r = .17$), and a family history of externalizing behavior disorder was significantly associated with regular smoking by both mother and sibling

($r_s = .16$ and $.24$, respectively). Longitudinal, prospective research examining the temporal associations between psychopathology and smoking in the parents in relation to psychopathology and smoking in the offspring would be particularly informative.

Sex and Other Demographic Differences

In addition to the major categories of psychopathology and familial factors, we examined the impact of four demographic variables. Somewhat unexpectedly, none were associated with smoking initiation. This is an important negative finding, and we will examine in future studies whether these variables are related to other indices of cigarette smoking, such as progression to regular smoking or cessation. We were particularly interested in whether sex moderated any of the associations with smoking initiation, and six interactions emerged (two with smoking initiation and four with early versus late smoking initiation). In every instance, smoking initiation was more strongly associated with the risk factor for young women than for young men, suggesting that these variables are especially relevant in the initiation of smoking among young women. Regarding smoking initiation, the associations with alcohol and drug use disorders were significant for both genders but the magnitude of the association was greater for young women compared to young men. Regarding early versus later initiation, women who began smoking earlier compared to women who smoked later had more drug use and anxiety disorders, elevated antisocial personality disorder scores, and a family history of affective disorder. The differences for early versus late male initiators were either nonsignificant or smaller in magnitude.

Kandel et al. (1997) examined the associations between four levels of cigarette use (no lifetime use, no use in past 6 months, less than daily use in past 6 months, daily use in past 6

months) and various psychiatric disorders in a large sample of 9-18 year olds, calculating results separately by sex. All disorders (substance use, anxiety, mood, attention-deficit/disruptive behavior disorders) were related to smoking level, although not all adjacent smoking group contrasts were significant. For female participants, differences between never smoking and past smoking groups (the lowest level of smoking examined in their study) were significant for substance use and attention-deficit/disruptive behavior disorders, which parallel findings in the present study. For male participants, mood disorders differentiated the two lowest levels of smoking. The strongest overall associations with smoking group status were for substance use disorders in the females and attention-deficit/disruptive behavior disorders in the males.

Among smoking initiators, three demographic factors were associated with early onset. First, early smoking initiation was more likely among for adolescents who were not living with both biological parents and those in which neither parent had completed college, a proxy measure of measure of socioeconomic status. Neither variable was significant in multivariate analyses. Participants of different racial groups did not differ on smoking initiation or early onset smoking, but the sample contained very few racial or ethnic minorities. Lastly, female sex emerged as significant in the multivariate analysis predicting early initiation. Future research should examine whether early pubertal development is associated with early smoking initiation in female adolescents.

Limitations and Conclusions

The present study has a number of limitations which need to be noted. First, the associations with smoking initiation identified in the present study consisted of lifetime risk

factors. We were not prospectively predicting the onset of smoking initiation among nonsmoking young people. To have conducted a truly prospective study, we would have needed to begin with a much younger sample.

Second, some of the participants who initiated cigarette smoking stopped at minimal experimentation with smoking, whereas others progressed to heavier rates of smoking. In the present study, all of these individuals were combined into a single category. A consensus is emerging that the risk factors predicting transition to one stage of smoking may differ from predictors of transition to another stage of smoking (e.g., Brook et al. 1998; Chassin, Presson, Sherman, Corty, & Olshavsky, 1984; Chassin, Presson, Sherman & Pitts, 2000; Harrell, Bangdiwala, Deng, Webb, & Bradley, 1998; Leventhal & Cleary 1980), and that the associations with psychopathology may be stronger for substance use at heavier or more problematic levels (e.g., Breslau, 1995; Costello, Armstrong, & Erkanli, 2000; Dierker et al., 2001). In future papers in this series, we will examine whether variables from the present study are predictive of progression from smoking initiation to daily cigarette use, and from daily smoking to successful cessation.

Third, while our multivariate analyses may be the most unique aspect of the present study, we did not assess all of the potentially relevant factors predictive of smoking initiation. Instead, our focus was on a subset of potential risk factors which we attempted to assess as thoroughly and rigorously as possible. Multivariate analyses examining an even broader set of variables would be informative.

Fourth, biochemical verification of smoking status was not conducted. Instead, we relied

on retrospective self-report to determine smoking initiation. Fortunately, research suggests that adolescent self-reports of tobacco use are generally reliable and valid, especially when steps such as those used in our study are taken to ensure comprehension and confidentiality (Dolcini, Adler, & Ginsberg, 1996; Warren, McClelland, Elwood, Ferry, & Silva, 1996). While some data suggest that adolescent self-reports of age of smoking initiation are not stable over time, tending to increase with age (Engels, Knibbe, & Drop, 1997; Golub, Lavouvie, & Johnson, 2000), our participants were assessed relatively close to the time of smoking initiation. In addition, our analyses focused on the occurrence of smoking initiation (Yes or No), which appears to be quite reliable over time (e.g., Henriksen & Jackson, 1999; Janson, 1999).

The use of telephone assessments of smoking at T3 may be a potential limitation, given that telephone interviews sometimes result in a small degree of under-reporting of smoking behavior (Luepker, Pallonen, Murray, & Pirie, 1989). However, we previously reported that telephone and face-to-face interviews of psychopathology yielded basically comparable reports (Rohde et al., 1997), and almost all smoking initiation was first reported at the T1 and T2 interviews, were which conducted face-to-face.

Fifth, the sample was predominantly white and adolescents with smoking initiation were less likely to participate in the T2 follow-up assessments. These factors may limit the generalizability of our findings. For example, parental smoking may have a stronger association with offspring smoking for white children than for African-American children (Griesler & Kandel, 1998). Regarding the impact of attrition by adolescent smokers, this potential bias may have lowered the magnitude of associations between smoking initiation and the measures of

individual psychopathology and familial factors.

The present study also has a number of strengths. Results are based on a large community sample, and standardized procedures for assessing smoking and lifetime psychopathology were used. Our use of reliably-assessed DSM diagnostic categories represents an important strength of the study. Most research in the area of smoking has assessed lower levels of substance use or has assessed symptoms of psychopathology by self-report questionnaire. Given the strong associations between smoking initiation and psychopathology shown in the present study, smoking prevention and cessation efforts probably need to take the assessment and treatment of psychiatric disorders into consideration. It is also quite likely that children and adolescents who have tried smoking are at elevated risk for developing various forms of psychopathology and could be selected for secondary prevention efforts aimed at the prevention of psychiatric disorders. This recommendation would appear to be particularly salient in the prevention of future drug and alcohol use disorders.

Another unique contribution of the present study was the inclusion of familial smoking and psychopathology data, generally obtained by direct report (as opposed to the more commonly used informant report). Smoking by mothers and by siblings had unique and robust associations with smoking initiation in the participants. The present findings may help to identify the mechanisms accounting for a transmission of nicotine use and dependence within families. As pointed out in our review of the literature, with the exception of family psychopathology, all of the variables in this study have been examined previously as individual risk factors for smoking. However, to our knowledge, this is the first time that psychopathology and familial factors have

been examined simultaneously or in this much detail. By examining this array of variables simultaneously, our findings illustrate the particularly strong associations between smoking initiation, certain forms of psychopathology, and familial smoking. The causal nature of these associations deserves further attention.

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Table 1

Univariate Associations with Smoking Initiation

Variable	% Smoking Initiation			
	Variable	Variable	Odds	95%
	Present	Absent	Ratio	Confidence Interval
<u>T1 Demographics</u>				
Male gender	76.1	77.2	1.06	NS
White ethnicity	76.7	76.7	1.01	NS
Non-intact home	78.8	75.0	1.24	NS
Low parental education	76.2	76.7	1.03	NS
<u>Lifetime Psychopathology</u>				
MDD	82.1	70.9	1.79***	1.31-2.44
Dysthymia	83.0	76.4	1.54	NS
ALCOHOL	90.3	70.0	4.20***	2.76-6.39
DRUG	94.9	71.3	5.06***	3.39-7.55
Anxiety disorders	81.4	75.5	1.45	NS
ADHD/DIS	91.2	75.0	3.36***	1.66-6.80
Antisocial PD scores	93.2	74.8	4.32***	1.85-10.07
Borderline PD scores	92.0	75.6	3.60*	1.28-10.14
Comorbidity (1 vs. 2+) ^a	87.7	68.1	3.36***	2.36-4.76

(table continues)

Variable	% Smoking Initiation			
	Variable Present	Variable Absent	Odds Ratio	95% Confidence Interval
	<u>Familial Smoking</u>			
Maternal smoking	82.3	73.2	1.68**	1.18-2.40
Paternal smoking	79.1	74.8	1.19	NS
Any sibling smoking	83.8	74.2	1.38*	1.02-1.86
<u>Familial Psychopathology</u>				
Affective	79.8	72.0	1.47*	1.05-2.07
Drug and alcohol	80.5	68.1	1.91***	1.34-2.71
Anxiety	81.4	75.5	0.94	NS
Externalizing _a	82.7	75.2	1.48	NS

* $p < .05$; ** $p < .01$; *** $p < .001$.

___ Note. MDD = major depressive disorder; ALCOHOL = alcohol use disorders; DRUG = drug use disorders; ADHD/DIS = ADHD and disruptive behavior disorders; PD = personality disorder; NS = nonsignificant.

^aThis contrast was restricted to participants with one or more mental disorders.

^aThis category consisted of ADHD, conduct disorder, oppositional defiant disorder, and antisocial personality disorder.

Table 2

Temporal Order of Disorder versus Smoking Initiation for Psychopathology Variables with Significant Univariate Association

Disorder	% Smoking before Disorder	Critical ratio Z
MDD	74.4	9.74***
ALCOHOL	90.0	13.24***
DRUG	84.3	9.80***
ADHD/DIS	48.4	-0.31
ADHD	3.4	-5.01***
Conduct disorder	100.0	2.65**
Oppositional defiant disorder	60.6	1.21
Anxiety disorders	36.3	-3.43***
Separation anxiety disorder	2.3	-6.32***
Phobia disorder	44.1	-0.91
Panic disorder	87.8	4.24***

p < .01; *p < .001.

Note. MDD = major depressive disorder; ALCOHOL = alcohol use disorders; DRUG = drug use disorders; ADHD/DIS = ADHD and disruptive behavior disorders; ADHD = attention-deficit/hyperactivity disorder.

^aDegrees of freedom ranged from 428 to 719.

Table 3

Significant Univariate Associations with Early versus Late Smoking Initiation

Variable	% with Variable		Odds	95%
	Early Onset	Late Onset	Ratio	Confidence Interval
<u>Demographics</u>				
Non-intact home	55.4	40.1	1.86***	1.38-2.50
Low parental education	52.6	36.5	1.92***	1.43-2.63
<u>Lifetime Psychopathology</u>				
ALCOHOL	44.0	34.6	1.48***	1.10-2.01
DRUG	37.8	20.8	2.31***	1.66-3.22
Anxiety	25.4	19.0	1.45*	1.01-2.06
ADHD/DIS	19.5	7.5	2.98***	1.88-4.73
Antisocial PD scores	17.1	7.3	2.63***	1.62-4.27
Comorbidity (1 vs. 2+)	57.9	44.1	1.74***	1.30-2.34
<u>Familial Smoking</u>				
Maternal smoking	51.1	42.6	1.41*	1.02-1.94
Paternal smoking	68.1	54.4	1.79***	1.27-2.51
<u>Familial Psychopathology</u>				
Drug and alcohol	81.5	68.1	2.05***	1.41-3.00

* $p < .05$; *** $p < .001$.

____ Note. ALCOHOL = alcohol use disorders; DRUG = drug use disorders; ADHD/DIS = ADHD and disruptive behavior disorders; PD = personality disorder.