

Attention Deficit Hyperactivity Disorder Symptoms Predict Nicotine Dependence and Progression to Regular Smoking from Adolescence to Young Adulthood

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Objective To examine the association between retrospectively reported attention deficit hyperactivity disorder (ADHD) symptoms and progression to smoking and the association with nicotine dependence. **Methods** Study sample consisted of a nationally representative cohort of U.S. adolescents ($n = 13,494$). Logistic regression was used to examine ADHD symptoms from both the inattentive (IN) and hyperactive-impulsive (HI) domains and smoking trajectories. Linear regression was used to examine nicotine dependence. **Results** HI symptoms were associated with progression from nonsmoking to regular smoking ($OR = 1.14$, 95% $CI = 1.07-1.21$), and with progression from experimentation to regular smoking ($OR = 1.16$, 95% $CI = 1.08-1.26$). IN and HI symptoms were associated with nicotine dependence among current smokers (IN: $\beta = 0.17$, $SE = 0.03$, $p < 0.0001$; HI: $\beta = 0.10$, $SE = 0.04$., $p < .001$). **Conclusions** These results have important implications for the development of prevention and treatment modalities.

Key words ADHD; adolescence; smoking.

Introduction

It is well established that there are numerous negative health consequences due to smoking and that there is an increased risk of smoking among those with a psychiatric disorder (Breslau, Novak, & Kessler, 2004). Attention Deficit Hyperactivity Disorder (ADHD) is one of the more common psychiatric disorders in childhood and adulthood affecting between 2% and 5% of the population (American Psychiatric Association, 1994). In addition to having serious consequences for academic and other outcomes, ADHD is a significant risk factor for smoking among both adults (41–42% vs. 26% for ADHD and nonADHD, respectively) and adolescents (19.0–46% vs. 10–24% for ADHD and nonADHD, respectively) (Lambert & Hartsough, 1998; Milberger, Biederman, Faraone, Chen, & Jones, 1997; Milberger, Biederman, Faraone, Wilens, & Chu, 1997; Molina & Pelham, 2003; Pomerleau, Downey, Stelson, & Pomerleau, 1995; Rohde, Kahler, Lewinsohn, & Brown, 2004). Moreover,

individuals with ADHD report earlier initiation of smoking and report more difficulty quitting than individuals in the general population (Milberger et al., 1997; Molina & Pelham, 2003; Pomerleau et al., 1995; Rohde et al., 2004).

There is considerable evidence that core ADHD symptoms of inattention and hyperactivity-impulsivity confer risk for smoking related behaviors, independent of clinical diagnosis. For example, in a recent study in a large population-based sample of young adults, retrospectively-reported ADHD symptoms were significantly and linearly related to lifetime smoking risk, age of onset of regular smoking and, among current smokers, number of daily cigarettes smoked (Kollins, Fuemmeler, & McClernon, 2005). The findings of this epidemiologic study are consistent with other reports in the literature using community and nonclinical samples and with laboratory studies of the effects of nicotine on attention and other executive functions (Burke, Loeber, & Lahey,

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2001; Lerman et al., 2001; Levin, McClernon, & Rezvani, 2006; Tercyak, Lerman, & Audrain, 2002).

The separate domains of ADHD symptoms [i.e., inattention and hyperactive-impulsive (HI)] may be differentially related to smoking outcomes among both clinical and nonclinical populations. For example, the risk for smoking has been shown to be more strongly associated with the number of reported inattentive (IN) symptoms than by the diagnosis of ADHD itself in a clinical sample (Burke et al., 2001). In contrast, a study in a nonclinical population has reported that HI symptoms are more strongly associated with lifetime risk of smoking than IN symptoms (Kollins et al., 2005). The differences in findings between these studies may reflect differences in how relations between ADHD symptoms interact with smoking at various points in the life-course. For instance, Burke et al. employed an adolescent sample, whereas the study by Kollins et al. was a cross-sectional analysis of smoking in a young adult sample. As such, different ADHD symptoms may contribute to different aspects of smoking behavior, and these effects may be influenced by developmental level.

One potential approach for understanding the differential contribution of HI and IN symptoms to the development of smoking and related outcomes is to examine the progression of smoking behavior prospectively. Smoking behavior does not progress similarly for all adolescents. For instance, some adolescents may experiment with smoking but never become addicted, whereas others may progress to regular smoking. Smoking experimentation is typically initiated during this early adolescent phase with those who begin at an earlier age more likely to become addicted and more likely to have more difficult time quitting (Chassin, Presson, Pitts, & Sherman, 2000; Khuder, Dayal, & Mutgi, 1999). Although several studies have begun to show an association between smoking behaviors and ADHD symptoms, few studies have explored the degree to which symptoms influence progression to smoking. Establishing an association between psychiatric symptoms and the progression to addictive behaviors is a first step toward examining potential causal mechanisms and planning optimal methods and time periods for intervention. At least one study has examined the association between psychiatric disorders and risk to progression to daily smoking during the transition between adolescence and young adulthood and found a lifetime diagnosis of ADHD to be a significant predictor of progression, although this study did not report the differential effects of IN and HI symptoms, and did not report on data from a population-base sample (Rohde et al., 2004).

The purpose of the present study was twofold. First, we assessed the differential effects of retrospectively reported HI and IN symptoms on the progression of smoking behaviors from adolescence through young adulthood. Second, we examined the relation between HI and IN symptoms on reports of nicotine dependence in individuals who were current regular smokers. Based on our previous work and the work of others suggesting hyperactive symptoms to be important predictors of lifetime smoking risk, we hypothesized that although both HI and IN symptoms would be associated with progression to smoking, HI symptoms would confer a greater risk. We examined data from the National Longitudinal Study of Adolescent Health (Add Health)—a nationally representative cohort of youth—to accomplish these goals (Bearman, Jones, & Udry, 1997; Resnick et al., 1997).

Methods

Data Source

The study population was drawn from 20,747 adolescents from the National Longitudinal Study of Adolescent Health, a nationally representative study of adolescents. The longitudinal cohort includes 14,388 eligible respondents who participated in in-home surveys on three separate occasions (April–December, 1995, April–August, 1996, and August, 2001–August, 2002). The mean age of survey participants on the three waves of data collection was 15.65 ($SD = 1.75$) years, 16.22 ($SD = 1.64$) years, and 22.96 ($SD = 1.77$) years. By design, the Add Health survey included a sample stratified by region, urbanicity, school type, ethnic mix and size to garner a nationally representative sample. Precise details regarding the design and data collection have been described elsewhere (Bearman et al., 1997; Resnick et al., 1997).

Study Sample

For the present study, the subpopulation of individuals with available population weights and who had reported data on the smoking items that comprised the trajectory categories included 13,494 participants. Women who were pregnant ($n = 379$) were excluded, as it was believed that women who were pregnant may alter their smoking behaviors. Further, participants who were regular smokers at Wave I ($n = 2670$) were excluded from analysis because they would not provide unique information about progression to smoking. Thus, the available study sample included 10,445 participants who included the following: nonsmokers at Wave I who continued to be nonsmokers at Wave III ($n = 4810$), nonsmokers at Wave I who went on to be experimenters at Wave III

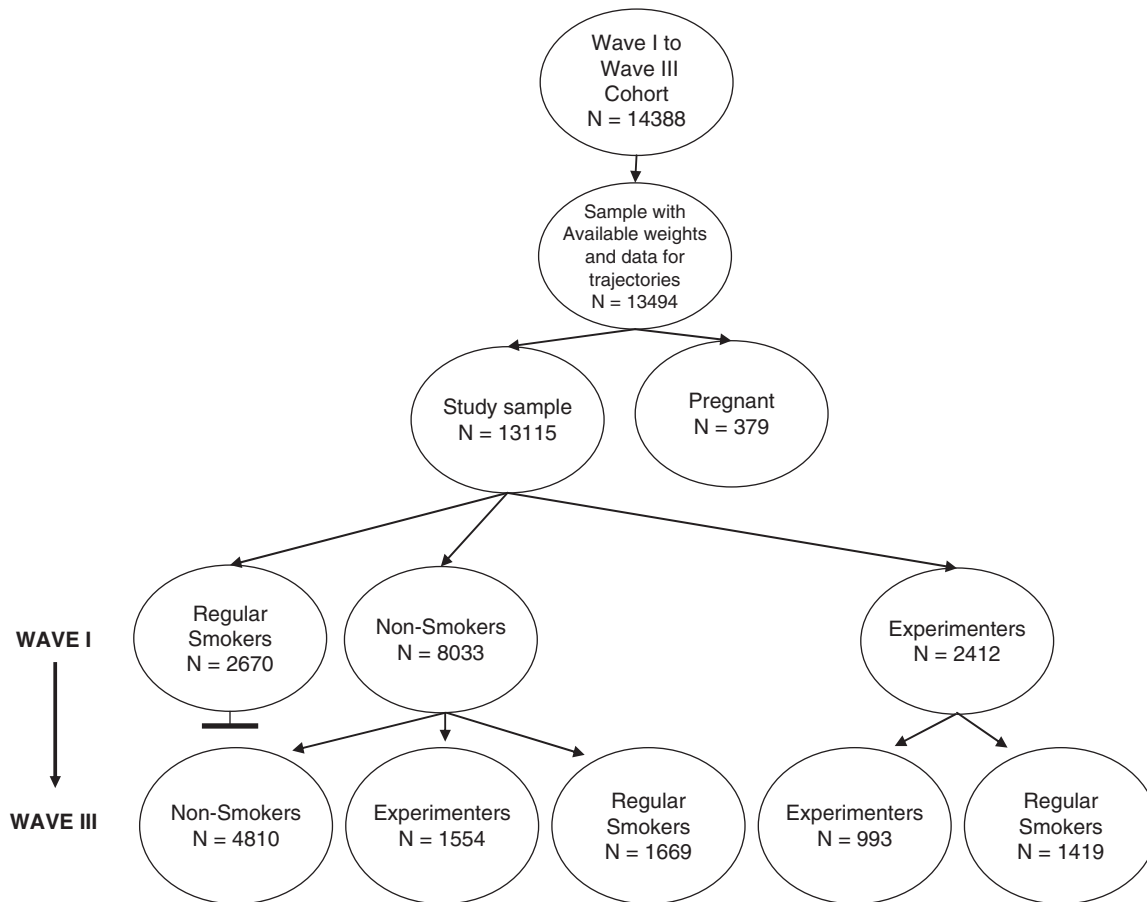


Figure 1 Trajectories of smoking progression and Ns for each group.

($n = 1554$), nonsmokers at Wave I who went on to be regular smokers at Wave III ($n = 1669$), experimenters at Wave I who continued to be experimenters at Wave III ($n = 993$), and experimenters at Wave I who went on to be regular smokers at Wave III ($n = 1419$). See Fig. 1 for details.

Measures

Smoking Progression and Dependence. In our analyses, smoking experimentation was defined as those subjects who had smoked a whole cigarette at least once. Regular smoking was defined as ever having smoked at least one cigarette daily for 30 consecutive days. Based on these definitions, three separate dichotomous smoking trajectories were used to describe progression of nicotine use across Waves I and III. First, an Experimentation (EXP) trajectory was defined in which adolescents who reported no experimentation with smoking at either Waves I or III were classified as 0; those who reported no experimentation at Wave I, but experimentation at Wave III (but not regular use) were classified as 1. A second trajectory,

Regular Smoking Initiation (RegSmk) was defined as follows: those subjects who reported no experimentation at either Waves I or III were classified as 0; those who reported no experimentation at Wave I, but regular smoking at Wave III were classified as 1. Finally, a third trajectory of Progression to Regular Smoking (ProgSmk) was defined. In this trajectory, subjects who reported experimentation at Wave I and regular smoking at Wave III were classified as 1, while all others were classified as 0. These three trajectories, therefore, captured different patterns of smoking progression from adolescence to young adulthood.

Participants who reported current regular smoking at Wave III also completed the six items that comprise the Fagerström Test of Nicotine Dependence (FTND), a widely used measure of nicotine use (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). Sample items of the FTND include, “do you smoke more frequently during the first hours after waking than during the rest of the day” and “do you still smoke even if you are so ill that you are in bed most of the day.” The Fagerstrom

Table 1. Retrospective Attention Deficit Hyperactivity Symptoms assessed in Add Health

Questions began with the following instructions: Think back when you were between 5 and 12 years of age. For each of the following statements, which answer best describes your behavior when you were that age? When you were between 5 and 12, you:

Inattention symptoms

- failed to pay close attention to details or made careless mistakes in your homework
- had difficulty sustaining your attention in tasks or fun activities
- didn't listen when spoken to directly
- didn't follow through on instructions and failed to finish work
- had difficulty organizing tasks and activities
- avoided, disliked, or were reluctant to engage in work requiring sustained mental effort
- were easily distracted
- lost things that were necessary for tasks or activities
- were forgetful

Hyperactive symptoms

- fidgeted with your hands or feet or squirmed in you seat
- left your seat in the classroom or in other situations when being seated was expected
- felt restless
- had difficulty doing fun things quietly
- felt "on the go" or "driven by a motor"
- blurted out answers before the questions had been completed
- had difficulty awaiting your turn
- talked too much

questionnaire is used widely to characterize levels of nicotine dependence and use. Scores from this instrument have been found to be correlated with biomarkers for nicotine levels (C. S. Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994; Radzius, Moolchan, Henningfield, Heishman, & Gallo, 2001). Summed scores from these six items were used as the primary measure of nicotine dependence for analysis.

ADHD

In Wave III, participants were asked to retrospectively report ADHD symptoms experienced between the ages of 5 and 12 years. Respondents were asked to report the frequency of a symptom using a 4-point Likert scale: never or rarely, sometimes, often, very often. For reasons that are not clear, one item asked in the retrospective ADHD section of Wave III—"you were spiteful or vindictive"—is not a DSM-IV ADHD symptom and was excluded from analyses; while one DSM-IV impulsivity symptom, "often interrupts or intrudes on others" was not included in the retrospective ADHD section. Thus, our analyses included responses to nine IN and eight HI

symptoms. Table 1 displays symptoms of ADHD assessed in Add Health.

A symptom was considered present if it was experienced "often" or "very often." This approach to dichotomizing symptoms has been used in other community-based studies of ADHD symptomatology, is considered clinical convention (Murphy & Barkley, 1996) and was also used in a previous study on the relation between ADHD and smoking outcomes (Kollins et al., 2005). For our primary analyses, the total number of symptoms reported (0–9 for IN; 0–8 for HI) was used as a measure of ADHD severity in order to assess the relation between symptoms and smoking progression and dependence. Individuals were also classified into one of four clinically relevant groups based on the number of reported symptoms: (a) IN subtype: ≥ 6 IN symptoms, < 6 HI symptoms (2.69%); (b) HI subtype: ≥ 6 HI symptoms, < 6 IN symptoms (2.93%); (c) Combined subtype: ≥ 6 IN symptoms AND ≥ 6 HI symptoms (2.52%); and (d) Control subtype: < 6 HI AND < 6 IN symptoms (91.86%). The six-symptom cutoff was chosen to be consistent with DSM-IV ADHD criteria requiring the presence of six or more symptoms from either the IN or HI symptom domains. As such, it does not represent a proxy for the diagnosis of ADHD.

The use of retrospective report is common in clinical practice when working with adults with ADHD and data exist that support the reliability and validity of these self-reports (Epstein & Kollins, 2005; P. Murphy & Schachar, 2000; Stein et al., 1995; Ward, Wender, & Reimherr, 1993; Zucker, Morris, Ingram, Morris, & Bakeman, 2002). In our previous report, we also demonstrated adequate reliability and validity of this approach by demonstrating good internal consistency of the items ($\alpha = .86$) as well as showing that parents of respondents reporting six or more symptoms on either or both HI and IN scales were more likely to indicate learning or behavioral problems at Wave 1; and these individuals also were more likely to report taking medications for ADHD at Wave III (Kollins et al., 2005).

Demographic and Other Variables

Demographic factors that have been shown to contribute to smoking risk were also assessed. These included items assessing gender, race (white vs. nonwhite), and indicators of socioeconomic status (parental report of the highest education level of either themselves or their spouse and whether the family of the participants had received public assistance at Wave I).

Since conduct disorder (CD) has been shown in previous studies to be an important mediator of the risk

Table II. Demographics of Analysis Sample

Wave I ↓ Wave III	Experimentation Initiation				Regular Smoking Initiation				Progression to Regular Smoking			
	Never smoker ↓ Never smoker		Never smoker ↓ Experimenter		Never smoker ↓ Never smoker		Never smoker ↓ Regular smoker		Experimenter ↓ Experimenter		Experimenter ↓ Regular smoker	
	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%
Total	4810	75.57	1554	24.42	4810	74.23	1669	24.77	993	41.10	1419	58.90
Gender												
Male	2426	51.57	761	49.05	2426	51.57	811	50.27	472	45.01	700	49.47
Female	2377	48.43	791	50.95	2377	48.43	856	49.73	518	54.99	719	50.53
Ethnicity												
White	2392	38.13	597	26.83	2392	38.13	543	23.99	289	20.82	383	17.68
Non-white	2405	61.87	954	73.17 ^a	2405	61.87	1126	76.01 ^a	700	79.18	1035	82.32
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
CD symptoms	0.470	0.924	0.481	0.874	0.470	0.924	0.702	1.179 ^b	0.985	1.361	1.211	1.492 ^b
Age												
Wave I	15.45	1.890	14.95	1.799 ^b	15.45	1.890	14.59	1.744 ^b	15.78	1.752	15.19	1.696 ^b
Wave III	21.84	1.940	21.34	1.804 ^b	21.85	1.940	20.97	1.770 ^b	22.14	1.760	21.56	1.739 ^b

Columns indicate the number of people (or mean) and percentages (or standard deviation) for each given trajectory.

Means and percentages are weighted.

Numbers may not add to total due to missing data on the covariate.

^aMantel-Haenszel chi-square $p < .001$.

^bt-test $p < .01$.

between ADHD and substance use (Barkley, Fischer, Edelbrock, & Smallish, 1990), we also included a measure of CD symptoms in multivariate analyses. This variable consisted of responses to 13 questions that corresponded to DSM-IV symptom criteria for CD that have been used in previous studies with the Add Health database to characterize CD (Miles, van den Bree, & Pickens, 2002). This CD variable derived from the Add Health data set has also been shown to have adequate internal consistency ($\alpha = 0.67$) (Kollins et al., 2005).

Statistical Analysis

Statistical analyses were conducted using SAS-callable SUDAAN (version 8.0) statistical software (SUDAAN user's manual, release 8.0., 2001). SUDAAN allows for control of survey design effects of individuals clustered in sampling unit of school and stratification of geographic region. Poststratification weights were applied in order to allow the results to be comparable to young adults in the U.S. population. Logistic regression analyses were used to predict smoking trajectories from self-reported ADHD symptoms. Two models were tested for each trajectory. In the first, the total number of IN and HI symptoms were used as predictors and in the second, the subtype classification of ADHD symptoms was used (i.e., IN, HI, Combined, and Control). In all models, age, sex,

ethnicity, SES variables, and CD were included to control for their effects.

The relation between ADHD symptom domains and self-reported nicotine dependence (as measured by the FTND) among those who progressed to regular smoking and who were currently smoking at Wave III was analyzed using a multiple linear regression model controlling for age, sex, ethnicity, SES, and CD. A second regression analysis examined the subtype classification of ADHD as a predictor of nicotine dependence.

Results

Figure 1 illustrates the composition of the different smoking trajectories used for our analyses. Demographic characteristics and CD symptoms for these trajectory classes are presented in Table II.

Associations Between ADHD Symptoms and Smoking Progression

The first set of multiple logistic regression analyses evaluated the relation between ADHD symptoms and the three trajectories (progression to experimentation, regular smoking initiation, and progression to regular smoking from experimentation) controlling for potentially important predictor variables (age, race, SES, sex, and CD)

Table III. Logistic Regression Analyses

	Experimentation initiation			Regular smoking initiation			Progression to regular smoking		
	OR	95% CI		OR	95% CI		OR	95% CI	
		Lower limit	Upper limit		Lower limit	Upper limit		Lower limit	Upper limit
Model I									
Demographic variables									
Highest level of education in family	1.06	1.01	1.11	0.96	0.92	1.00	1.00	0.94	1.06
Receiving public assistance	0.78	0.54	1.13	0.94	0.70	1.25	1.10	0.66	1.81
Age	0.87	0.83	0.92	0.78	0.74	0.82	0.82	0.77	0.88
Race	1.48	1.18	1.85	1.81	1.44	2.29	1.18	0.89	1.57
Sex	1.18	0.99	1.40	1.09	0.89	1.32	0.85	0.66	1.10
Conduct disorder	1.08	0.98	1.20	1.28	1.18	1.40	1.13	1.02	1.24
# of ADHD symptoms									
Inattention symptoms	0.99	0.92	1.06	1.04	0.98	1.11	0.96	0.89	1.04
Hyperactivity symptoms	1.05	0.99	1.11	1.14	1.07	1.21	1.16	1.08	1.26
Model II									
Demographic variables									
Highest level of education in family	1.05	1.00	1.11	0.96	0.92	1.00	1.00	0.94	1.06
Receiving public assistance	0.78	0.54	1.13	0.96	0.71	1.29	1.13	0.68	1.87
Age	0.87	0.83	0.92	0.78	0.74	0.82	0.82	0.77	0.88
Race	1.48	1.18	1.85	1.86	1.47	2.34	1.20	0.90	1.60
Sex	1.18	0.99	1.40	1.09	0.90	1.33	0.87	0.67	1.13
Conduct disorder	1.09	0.98	1.20	1.31	1.21	1.43	1.14	1.03	1.25
ADHD subtype									
Inattentive subtype	1.51	0.81	2.80	1.47	0.84	2.57	0.95	0.48	1.89
Hyperactive subtype	1.39	0.76	2.55	1.90	1.10	3.27	3.25	1.54	6.86
Combined subtype	1.21	0.55	2.68	2.64	1.48	4.72	1.88	0.78	4.53
Control subtype (reference)	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00

Odds ratios (OR) and Confidence Intervals (CI) indicate odds of being on each trajectory compared to not being on the trajectory. In the Experimental Initiation column, ORs indicate odds of progressing from nonsmoking to experimentation across Waves I and III, in the Regular smoking initiation column, ORs indicate odds of progressing from nonsmoking to regular smoking, and in the Progression to regular smoking column, ORs indicate odds of progressing from experimentation to regular smoking. Model I included ADHD symptoms from each domain (IN and HI) as a continuous variable. Model II used subtypes of ADHD symptoms (see text for details).

(Table III). IN symptoms did not predict any of the progression trajectories. In contrast, HI symptoms were significantly associated with progression from no smoking to regular smoking (OR = 1.14; 95% CI: 1.07–1.21) and from experimentation to regular smoking (OR = 1.16; 95% CI: 1.08–1.26).

Results from analyses examining ADHD subtype as a predictor of smoking trajectories are presented in Table III. Similar to findings obtained using ADHD symptoms as a continuous indicator, the IN subtype was not significantly associated with any of the progression trajectories. The HI subtype was associated with progression from no smoking to regular smoking initiation (OR = 1.90; 95% CI: 1.10–3.27) and with progression from experimentation to regular smoking (OR = 3.25; 95% CI: 1.54–6.86). The Combined subtype was associated with progression from no smoking to regular smoking initiation (OR = 2.64; 95% CI: 1.48–4.72).

Impact of ADHD Symptoms on Nicotine Dependence Among Current Smokers

For individuals reporting smoking in the past 30 days at Wave III, both IN and HI symptoms were significantly associated with nicotine dependence, after controlling for CD, demographic factors, and the other symptom domain (IN: $\beta = 0.17$, $SE = 0.03$, $p < .0001$; HI: $\beta = 0.10$, $SE = 0.04$, $p < .001$). The model accounted for 9% of the variance in the FTND measure. When subtype of ADHD was examined as a predictor of nicotine dependence, all three subtypes were significantly associated with FTND scores (IN: $\beta = 0.74$, $SE = 0.37$, $p < .05$; HI: $\beta = 1.26$, $SE = 0.28$, $p < .0001$; Combined: $\beta = 1.24$, $SE = 0.31$, $p < .0001$). To explore the possibility that ADHD symptoms might be better predictors of nicotine dependence among people without high levels of either IN or HI symptoms, we conducted an exploratory

regression analysis restricting the sample to those people reporting ≤ 6 IN and HI symptoms. Results suggest that in this model IN symptoms were significant predictors of nicotine dependence ($\beta = 0.24$, $SE = 0.06$, $p < .0001$), while HI symptoms were not ($\beta = 0.08$, $SE = 0.05$, $p = .08$).

Discussion

Results from this study suggest that retrospectively reported ADHD symptoms are significantly associated with progression of smoking behavior and levels of nicotine dependence in a large, population-based sample of young adults. These findings extend previous work by our group which found that ADHD symptoms, most notably HI symptoms, increased lifetime risk of regular smoking (Kollins et al., 2005). In the present study, HI symptoms were significantly associated with the progression from nonsmoking to regular smoking, and with the progression from experimentation to regular smoking. These results were significant even after controlling for a range of other variables associated with smoking risk, as well as controlling for IN symptoms.

We also found that among current smokers, the number of self-reported IN symptoms was significantly associated with the level of nicotine dependence as measured by items from the FTND. However, when the clinically relevant subtypes of symptoms were examined, all three symptom clusters (IN, HI, and Comb) were significantly associated with nicotine dependence.

Consistent with other complementary studies, we also found in our models that white adolescents were significantly more likely to progress to experimentation from nonsmoking and regular smoking from nonsmoking than members of other racial/ethnic groups (Flint, Yamada, & Novotny, 1998; Robinson, Berlin, & Moolchan, 2004). We did not find that Whites were significantly more likely to be in the trajectory group from experimentation to regular smoking. It may be that racial differences are mitigated among those who are initially more experienced with tobacco use during adolescence, although this deserves further study. This finding may also reflect differences across racial categories with respect to onset of regular smoking. For example, one study found that African-American youth exhibited a 1-year delay in onset of both initiation and daily smoking, compared to nonAfrican-American youth (Robinson et al., 2004). We also found that CD symptoms were significantly associated with smoking progression in all three trajectory groups. This is consistent with the

documented literature suggesting that CD symptoms enhance risk of many types of substance use, including tobacco (Lynskey & Fergusson, 1995). The findings extend the literature as few studies have examined the role of CD on smoking progression in longitudinal cohorts (Mayhew, Flay, & Mott, 2000).

One of the unique aspects of the current study was that we examined the role that ADHD has on various patterns of smoking progression. In this study, ADHD symptoms expressed both continuously and as clinically relevant subtypes were associated with smoking progression, but not with progression to experimentation. Although future studies are needed in this area, the findings suggest that HI symptoms play a key role in the adoption of regular smoking, but HI and IN symptoms do not seem to play a role in the progression to experimentation. In our previous study, we found that ADHD symptoms were associated with earlier age of onset of smoking (Kollins et al., 2005). To the extent that ADHD symptoms were associated with earlier onset, it is possible that ADHD symptoms are associated with earlier experimentation. Notably, in order to study progression, we excluded some individuals from our analyses because they had already had a history of regular smoking prior to the baseline data collection. Future prospective studies will be needed that can assess ADHD symptoms and smoking behaviors from an earlier age in order to more definitely determine and describe this ADHD symptom—smoking relation from early childhood, to adolescence, to young adulthood. Overall, the findings are important as they extend previous work by demonstrating not only that ADHD symptoms are associated with lifetime risk of smoking (Kollins et al., 2005), but also that they influence the developmental progression of smoking behavior.

Another unique aspect of the study was that we examined what effect retrospectively reported ADHD symptoms had on nicotine dependence in current smokers. When we assessed ADHD symptoms as a continuous factor, IN and HI symptoms were related to FTND scores and, when the ADHD subtype variable was used in the regression analyses, all three subtypes predicted FTND scores. We also found that when considering symptom presentation that is below traditional clinical levels (i.e., less than six symptoms), only IN symptoms regulate nicotine dependence. These findings are consistent with the literature documenting the beneficial effects of nicotine on attention (Levin et al., 2006; Levin & Rezvani, 2002; Rezvani & Levin, 2001) as they suggest individuals with inattention symptoms may

be administering nicotine to improve attention. These preliminary findings suggest more research is needed on the role that IN and HI have on regulating dependence in both clinical and nonclinical ADHD samples.

There are several limitations to the present study that warrant comment. First, the primary independent variable was based on retrospective self-report of ADHD symptoms in childhood. The nature of the Add Health survey was such that individuals were asked to report on symptoms occurring ~10 years before the time of report. As such, the extent to which this self-report reliably and validly reflects ADHD symptoms in childhood is unknown. Complicating this issue is the degree to which individuals with true ADHD symptoms may be able to accurately report on the presence or absence of such symptoms. However, as mentioned, the use of retrospective report is very common in clinical practice when working with adults with ADHD, and these approaches have been found to be reliable and valid (Epstein & Kollins, 2005; Murphy & Schachar, 2000; Stein et al., 1995; Ward et al., 1993; Zucker et al., 2002). Furthermore, the Add Health survey of HI symptoms lacked one item included in the Diagnostic and Statistical Manual of Mental Disorders nomenclature (i.e., interrupts or intrudes on others) (American Psychiatric Association, 1994). If this is a low prevalent symptom, in the population, there may be negligible effects; however, if it is a highly prevalent symptom, the data may underestimate HI symptoms. It is difficult to know for certain what effect these survey limitations may have on our findings. However, as we have noted previously, the approach to quantifying ADHD symptoms within the Add Health does appear demonstrate consistency among the items and promising evidence of measurement validity (i.e., parents of respondents reporting six or more symptoms on either or both HI and IN scales were more likely to indicate that their participating child had a learning or behavioral problem when they were children and respondents also were more likely to report taking medications for ADHD symptoms at Wave III) (Kollins et al., 2005).

A second limitation was the use of the FTND as the primary measure of nicotine dependence. The FTND has been criticized as more a measure of “use” than a measure of “dependence” (Colby, Tiffany, Shiffman, & Niaura, 2000). Assessment of craving or the use of biological verification may have been a more precise way of characterizing nicotine use and dependence, but these endpoints were unavailable in the context of the available data set.

A third limitation is that the Add Health study is not a clinical sample. Thus, although we categorized individuals based on subtypes of ADHD symptom (predominately inattention, predominately hyperactive, and combined) this cannot be considered a true diagnostic estimate of prevalence in the population. Rather, this method was primarily chosen to explore how the potentially relevant “clinical categories” relate to smoking progression. A further limitation of using a nonclinical sample is that alternative comorbid diagnoses could not be evaluated. Therefore, the impact of clinically relevant symptoms and categories as well as other potential related diagnoses (e.g., Pervasive Developmental Disorders, Anxiety Disorders) could not be fully examined in the current models. The effect of these conditions will need to be evaluated with clinic-based rather than population-based samples. Nevertheless, the approach taken in this study provides evidence of a potential relation between ADHD symptoms (along with subtypes) and smoking progression at a population-level and thus, adds to and extends research in clinical samples. Finally, a myriad of factors have been associated with smoking initiation and progression including, family, peer, environmental, and policy. While a full treatment of these factors is outside the scope of the current study, this area is ripe for future investigation.

In spite of its limitations, the present study has a number of strengths. This is the first study to systematically examine the link between ADHD symptoms and progression of smoking behaviors in a nationally representative longitudinal cohort. As such, these findings add to the literature that has previously identified risk conferred by ADHD symptoms to smoking outcomes, in general (Kollins et al., 2005; Tercyak et al., 2002). Also, these findings highlight the potential unique contributions of the IN and HI symptom domains in the development of smoking-related behaviors. Understanding such differences will be important to extend this work and to consider how the present data may be used to develop viable prevention or intervention strategies.

The present results suggest that the observed effects have important public health implications. The specific results indicate that a one unit increase in hyperactive symptoms was associated with a 14% increased odds of progressing from no smoking in adolescence to initiating regular smoking and a 16% increased odds of progressing from experimentation to regular smoking. Moreover, having six or more symptoms of both types of ADHD symptoms was associated with a two and a half fold increased odds of progressing from no smoking

to initiating regular smoking and a nearly two fold increased odds of progressing from experimenting to regular smoking. If these associations represent a true relationship, they could have an important implication at a population level, as ADHD symptoms are common among children. Continued research that can verify these associations would be needed to accurately estimate the population effect. Nevertheless, an implication of the present study is that measuring these symptoms in the general population may prove useful in identifying individuals at higher risk for smoking, regardless of whether they are also at risk for ADHD diagnosis. Such individuals might then be selectively targeted for early prevention/intervention efforts. Likewise, our findings could inform treatment and prevention efforts and suggest that the effects of such efforts may be moderated by the presence of ADHD symptoms. That is, traditional prevention and intervention programs may be less effective among individuals with high levels of ADHD symptomatology.

These results highlight the need for additional work in this area. Notably, the study results are correlational. Ideally, research that uses prospective longitudinal designs to more fully characterize the relationship between ADHD symptoms in youth with smoking outcomes later in life will address some of the limitations of the current study. Given the convergence in the literature with respect to the link between ADHD symptoms and nicotine use, as well as laboratory studies of nicotine effects on attention and related process, basic laboratory studies that investigate the potential mechanisms underlying these associations are warranted.

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