Effects of Inattention and Hyperactivity/Impulsivity Symptoms on Development of Nicotine Dependence from Mid Adolescence to Young Adulthood

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Objective The relationship of Attention-Deficit/Hyperactivity Disorder (ADHD) and smoking is well documented. However, it is unclear whether ADHD symptoms relate to nicotine dependence (ND) symptoms from adolescence to young adulthood. We sought to assess the relationship of ADHD Hyperactivity–Impulsivity (H/I) and Inattention (I) symptoms with ND symptoms in a community sample from adolescence to young adulthood (ages 18–21).

Methods Participants were adolescents (n = 672) smoking at least one whole cigarette in their lifetime. Participants are members of a prospective cohort study (ages 14–22) evaluating the bio-behavioral predictors of smoking. Data were analyzed with a two-piece latent growth curve model.

Results ADHD-I symptoms were associated with ND symptoms acceleration in adolescence, but slowing acceleration in young adulthood, whereas ADHD-H/I symptoms were associated with ND symptoms acceleration in young adulthood.

Conclusions The results suggest the relationship of ADHD and ND symptoms may differ by symptom type, and the developmental period assessed.

Key words adolescent; ADHD symptoms; nicotine dependence; LGCM.

Adolescence is a critical period for smoking. Of adults ever smoking regularly, the majority began smoking during adolescence and progressed to a regular habit by age 18 (Chassin, Presson, Rose, & Sherman, 1996; USDHHS, 1994). It is estimated that 23% of high school students (CDC, 2006), and 24% of adults aged 18–24 are current smokers (CDC, 2005a).1 Research suggests nicotine dependence (ND) can follow shortly after smoking initiation, and develop well before adolescents progress to weekly or daily smoking. One study found that several symptoms of ND, including mental and physical addiction, and cravings, occurred within the first 2–5 months after smoking initiation (Gervais, O’Loughlin, Meshefedjian, Bancej, & Tremblay, 2006). Another study found that the first symptom of ND occurred within 2 weeks of first reported monthly smoking (DiFranza et al., 2000). Individuals with ND smoke 57% of all cigarettes smoked in the United States, while accounting for 13% of the population (Grant, Hasin, Chou, Stinson, & Dawson, 2004). Thus, it is important to understand the factors that increase the likelihood and rate of progression to ND from adolescence into young adulthood.

Attention-Deficit/Hyperactivity Disorder (ADHD) is one factor associated with an increased risk of adolescent smoking initiation and progression (Clark & Cornelius, 2004; Dani & Harris, 2005; Galera, Fombonne, Chastang, & Bouvard, 2005; Molina & Pelham, 2003; Rohde, Kahler, Lewinsohn, & Brown, 2004). ADHD is a developmental disorder characterized by age inappropriate levels of hyperactivity and impulsivity, and an inability to sustain directed attention (Barkley, 2003; Spencer, Biederman, & Mick, 2007). The DSM-IV (APA, 1994) identifies three subtypes of ADHD: Predominantly inattentive, predominantly hyperactive and impulsive, and a
combined subtype. Placement in a given subtype requires endorsing a minimum of six of the nine symptoms within a given subtype. The combined subtype is the most prominent accounting for 50–75% of all individuals with ADHD, followed by ADHD—Inattention (20–30%) and ADHD—Hyperactivity/Impulsivity (15%) subtypes (Spencer, Biederman, Mick, 2007). Although it is estimated that almost 4 million children and adolescents aged 3–17 (7%) have ADHD (CDC, 2003b; Dey et al., 2004), a larger number of youth have ADHD symptoms without meeting diagnostic criteria.

Research has established the relationship of ADHD and smoking in clinical samples (Lambert, 2005; Lambert & Hartsough, 1998; Molina & Pelham, 2001, 2003; Palacio et al., 2004; Rohde, Kahler, Lewinsohn, & Brown, 2004) and ADHD symptoms and smoking in community samples (Galera, Fombonne, Chastang, & Bouvard, 2005; Kollins, McClernon, & Fuemmeler, 2005; Whalen, Jamner, Henker, & Dellino, Lozano, 2002). Youth with ADHD and youth higher in ADHD symptoms tend to start smoking earlier than youth without ADHD and youth with lower ADHD symptoms (Kollins et al., 2005; Milberger, Biederman, Farahone, Chen, & Jones, 1997; Molina & Pelham, 2003). Among adults, those with ADHD tend to smoke more than adults without ADHD, and they tend to have more difficulty quitting (Lambert & Hartsough, 1998; Pomerleau, Downey, Stelson, & Pomerleau, 1995; Rohde et al., 2004). Moreover, ADHD symptoms are associated with a higher daily smoking rate among young adolescents (Kollins et al., 2005).

Although research has focused on the relationship between ADHD symptoms and smoking, little is known about the effects of ADHD symptoms on the development of nicotine dependence. One recent prospective study found that clinically diagnosed ADHD children were significantly more likely to have nicotine dependence at 28-year follow-up than their non-ADHD counterparts (Lambert, 2005). A second study found that clinically diagnosed ADHD youth were significantly more likely than non-ADHD controls to be nicotine dependent (Biederman et al., 2006). Unfortunately, neither study differentiated ADHD—H/I from ADHD—I symptoms. However, adolescent and adult research has explored the relationship between ADHD symptoms subtypes and smoking. For instance, research supports an association between inattention (ADHD-I) symptoms and smoking, but not hyperactivity/impulsivity (ADHD-H/I) symptoms in community samples (Lerman et al., 2001; Tercyak, Lerman, & Audrain, 2002). Retrospective reports of childhood ADHD symptoms among a community sample of young adults, however, suggests that ADHD-H/I symptoms are stronger predictors of regular smoking than ADHD-I symptoms (Kollins et al., 2005). Thus, it is possible that the relationship between ADHD symptoms and ND symptoms would differ by ADHD subtype.

There are at least two reasons ADHD symptoms may be related to symptoms of ND. First, research on clinically diagnosed adolescents with ADHD found that ADHD indirectly affected substance use through association with deviant peers (Laucht, Hohm, Esser, Schmidt, & Becker, 2007; Marshal, Molina, & Pelham, 2003). Having deviant peers is associated with greater substance use, including cigarette smoking (Simons-Morton, Haynie, Crump, Eitel, & Saylor, 2001). Research with community samples has found that impulsivity and poor control, characteristics of ADHD—H/I, are associated with having peers who smoke (Audrain-McGovern, Rodriguez, Tercyak, Neuner, & Moss, 2006; Wills & Cleary, 1999; Wills, Vaccaro, & McNamara, 1994). Research with community samples has found that good control, by contrast, is directly associated with less substance use (Wills et al., 2001), and indirectly related to less adolescent substance use through having fewer deviant peer relationships (Wills, Sandy, & Shinar, 1999).

In addition to the potential pathway through peer relationships, a second possibility is the negative reinforcing effects of nicotine in that it may reduce inattention and problems with self-control. One study found that nicotine alone (administered by patch), and in conjunction with stimulant medication, decreased inattention symptoms, while increasing control in a sample of clinically diagnosed adults with ADHD (Gehricke, Whalen, Jamner, Wigal, & Steinhoff, 2006). Another study found that nicotine patch administration to a sample of nonsmoking adolescents clinically diagnosed with ADHD improved cognitive/behavioral inhibition with effects comparable to methylphenidate (Potter & Newhouse, 2004). Thus, it is possible that adolescents and adults with ADHD symptoms may smoke because nicotine alleviates ADHD symptoms (negative reinforcement). This notion is supported by possible shared genetic factors linking ADHD and smoking (Laucht et al., 2007).

Given the limitations of past studies assessing the relationship between ADHD symptoms and smoking and the lack of studies assessing the relationship between ADHD symptoms and symptoms of nicotine dependence,
this study adds to the literature in three critical ways. First, we assessed whether one subtype more strongly predicts ND symptoms and change in ND symptoms than the other. More specifically, does one ADHD symptom subtype (ADHD-H/I or ADHD-I) as measured by self-report in a community sample, affect the development of ND symptoms more so than the other, while controlling for the opposite subtype.

Second, unlike previous studies (Milberger et al., 1997), this study explores developmental trajectories of ND symptoms, allowing for both inter- and intra-individual variability in ND symptoms, and exploring the effects of ADHD symptoms subtype on change in ND symptoms over time, instead of predicting end state ND symptoms (e.g., ND symptoms 3 years posthigh school). Third, this study employs a community-based sample, thereby allowing for generalizing findings to a nonclinical sample of adolescents and young adults. This is important as there are differences in characteristics of adolescents with clinical ADHD and adolescents from community samples (Bauermeister et al., 2007). For instance clinically diagnosed ADHD participants are more likely to have family members with a history of psychiatric disorders, have school suspensions and expulsions, and special education counseling, all of which have not been studied in community samples. As such, the objective of this study was to evaluate the effect of ADHD symptoms on longitudinal change in ND symptoms, and whether this effect differed by ADHD-H/I and ADHD-I symptoms, and developmental period (adolescence or young adulthood). We hypothesized that ADHD symptoms (assessed in the fall of 10th grade, study baseline) would be related to a higher level of ND symptoms at baseline and a faster rate of acceleration in nicotine dependence symptoms from baseline, across adolescence to young adulthood.

Methods
Participants and Procedures
Participants were high school students (50% female and 68% Caucasian) taking part in a longitudinal study of the biobehavioral predictors of adolescent smoking adoption. Participants were enrolled in one of five public high schools in northern Virginia. This cohort was drawn from the 2,393 students identified through class rosters at the beginning of ninth grade. Students were ineligible to participate in this study if they had a special classroom placement (e.g., severe learning disability). Based on the selection criteria, a total of 2,120 (89%) students were eligible to participate, and of these, 1,533 (72%) parents provided a response regarding their teen’s participation. Of the 1,533, 1,151 (75%) parents consented to their teen’s participation, yielding an overall consent rate of 54%. Analysis of differences between students whose parents did and did not consent revealed a significant race by education interaction. The likelihood of consent was greater for Caucasian parents with more than a high school education than for Caucasian parents with a high school education or less (Audrain, Tercyak, Goldman, & Bush, 2002). Although the overall difference was small (89% vs. 77%), this result suggests some caution in generalizing these findings.

The cohort was formed in the 9th grade and is being followed until the end of the fourth year post high school (age range from 14 to 22-years old). Five data collection waves were completed during high school: spring 2000, 9th grade; fall 2000, 10th grade; spring 2001, 10th grade; spring 2002, 11th grade; spring 2003, 12th grade (age range 14 to 18-years old), and three data waves have been collected post high school (age range 19 to 21-years old). Data for this study included waves two through eight only, as the ADHD symptoms questionnaire was completed in the fall of 10th grade. Thus, for this study, 10th grade fall (wave 2) is baseline. Data were collected on-site the five high school waves, during compulsory classes (e.g., health), and by phone the three waves post high school. The questionnaires took ~30 min to complete.

At 10th grade baseline, 1,072 participants had data on smoking, whereas 733 participants had data on smoking at last follow-up 3 years post high school. We analyzed participation bias comparing participants with a monotonic missing data pattern (missing data at follow-up waves to 10th grade baseline; 31.02%) to those with any other missing data pattern (e.g., wave nonresponse; 16.27%) or no missing data (complete data pattern; 52.31%). We focused on monotonic missing data as it represented the greatest source of missing data in our sample. Moreover, it represents loss to follow-up, whereas with other patterns, subjects are retained despite absence at least one wave, or are merely missing data on specific variables. Binomial logistic (1 = monotonic missing data pattern) regression analysis revealed that females were 66% (OR = .66, 95%CI = .50, .88) as likely as males, whereas non-Caucasians were 67% more likely (OR = 1.67, 95%CI = 1.25, 2.22) than Caucasians, to have a monotonic missing data pattern. Living with at least one smoker (OR = 1.70, 95%CI = 1.25, 2.31) was also associated with increased odds of monotonic missing
There was no effect for baseline smoking or any other predictor variable in the logistic regression analysis. Thus, males, non-Caucasians, and adolescents living with a smoker were more likely to be lost to follow-up, warranting some caution generalizing results.

Participants in the present study were adolescents \((n = 672)\) who reported smoking at least one whole cigarette in their lifetime at any one of the six waves from 10th grade fall to the third year posthigh school (age range 15 to 21-years old). Exposure to nicotine was a necessary prerequisite to evaluate progression in ND. University Institutional Review Board approval of the study protocol was obtained.

**Measures**

**Nicotine Dependence**

ND was measured with the seven-item modified Fagerstrom Tolerance Questionnaire for adolescents (mFTQ) (Prokhorov, Pallonen, Fava, Ding, & Niaura, 1996). Scores can range from 0 to 9. The mFTQ has been found to correlate significantly with intensity and duration of smoking, and salivary cotinine in an adolescent sample, and to have satisfactory item structure and internal consistency (Cronbach’s coefficient \(\alpha = .75\)) (Prokhorov et al., 2000). We assessed ND all seven waves. The mFTQ items were completed only if participants smoked a whole cigarette “1 month ago or less,” any given wave. Participants received a zero mFTQ score any wave for which mFTQ items were not completed, provided they completed mFTQ items at least one of the seven waves surveyed. ND was log transformed to correct for the resulting univariate nonnormality, however we will discuss the results without alluding to the log transformation except where necessary.

**ADHD Symptoms**

ADHD symptoms were assessed with the 18-item Current Symptoms Scale-Self Report Form (Barkley & Murphy, 1998). The measure asks individuals to describe their behavior during the past 6 months, on 18 clinically relevant ADHD symptoms (e.g., “Leave my seat in situations in which seating is expected,” “Am easily distracted”) using a four-point Likert scale \((0 = \text{Never or rarely}, 3 = \text{often})\). We used two subscales in this study, summing nine items to assess inattention symptoms (Cronbach’s coefficient \(\alpha = .84\)), and nine items to assess hyperactivity/impulsivity symptoms (Cronbach’s coefficient \(\alpha = .78\)). We standardized the two subscales to facilitate interpreting the results, using SAS version 9.1.3. Thus, a unit increase in ADHD symptoms heretofore refers to a 1SD increase in ADHD symptoms. ADHD symptoms were assessed in the 10th grade fall semester only.

**Covariates**

Demographic variables assessed included gender \((1 = \text{“male,”} 2 = \text{“female”})\) and race \((0 = \text{“Caucasian,”} 1 = \text{“non-Caucasian”})\). Items to assess exposure to smoking were the binary variable household smokers \((0 = \text{nobody living in the household smokes,} 1 = \text{at least household member smokes})\) and the continuous variable peer smoking \((0–8 \text{ best male and female friends smoke})\).

Past month marijuana use was assessed with one item asking how many times during the past 30-days the adolescent used marijuana. Past month alcohol use was assessed with one item asking, “During the past 30 days, on how many days did you have at least one drink other than a few sips?” Response choices ranged from 0 to all 30 days for alcohol use and 0 to 40 or more times for marijuana use (Grunbaum et al., 2004). We controlled for these covariates in the model as they have been shown to be associated with smoking and ND (Audrain-McGovern et al., 2004; Conrad, Flay, & Hill, 1992; Pate, Heath, Dowda, & Trost, 1996; Riggs, Mikulich, Whitmore, & Crowley, 1999; Wills et al., 1994).

**Results**

**Statistical Analysis**

Statistical analysis employed growth curve modeling in a latent variable framework (latent growth curve modeling; LGCM), using Mplus 4.21. LGCM models repeated measures of an observed variable on latent variables (factors) representing baseline level and developmental trends (e.g., linear, quadratic) (Duncan & Duncan, 1999; Muthén, 2002). The factors are random effects. Therefore, LGCM allows researchers to assess developmental heterogeneity in initial status and rate of change from baseline and regress factors on select covariates.

**Two-piece Model**

In this study, there were seven repeated measurements (waves) of ND, spanning ages 15–21. The first two waves occurred 6 months apart, the remaining waves a year apart. As the seven waves included a transition from adolescence to young adulthood, we modeled development with a two-piece model including a linear trend for high school development from the fall of 10th grade to the spring of 12th grade, and a second nonlinear trend for development post high school, from the spring
of 12th grade to last follow up 3 years post high school, with the last two factor loadings estimated freely.  

Missing Data Analysis
To account for missing data, multivariate modeling used all available data. Mplus allows modeling with missing data using maximum likelihood estimation of the mean, variance, and covariance parameters, when requested, employing the Expectation Maximization (EM) algorithm, assuming data are missing at random (Muthén, 1998–2004). We only accounted for missing data on the repeated measure of nicotine dependence. Thus, cases with missing data on the covariates were not included in the analysis. Parameter estimation in this study employed a Maximum likelihood robust (MLR) estimator.

Intra-class Correlations
As data were collected from five public high schools, a multi-level analysis may be most appropriate for this data, as participants are clustered within schools. However, intra-class correlations were below .05, suggesting a multi-level model was not necessary (Heck, 2001).

Evaluating Model Fit
Model fit was evaluated with model chi-square ($\chi^2$), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Residual (SRMR). Suggested fit criteria are nonsignificant $\chi^2$, CFI > .95, RMSEA < .05–.08 (Curran, Bollen, Chen, Paxton, & Kirby, 2003; Loehlin, 2004; Muthén & Muthén, 2001). An RMSEA value zero represents exact model fit (Curran et al., 2003).

Descriptive Statistics
The proportion of participants smoking at each level by wave is presented in Table I. Means, standard deviations, and bivariate correlations for ND symptoms (not log transformed) and covariates appear in Table II. Comparable to Youth Risk Behavior Survey (YRBS) findings (CDC, 2006), 20% of participants were current smokers (smoked on ≥1 day in the past 30 days) in the fall of 10th grade (YRBS, 21.4%), whereas this proportion increased to 30% by 12th grade (YRBS, 28%). The proportions were also comparable to National Health Interview Survey (NHIS) findings for young adults (aged 18–24), with 25.39% of study participants smoking over 100 cigarettes in their lifetimes and on 1 or more days in the past month (NHIS, 23.6%).

The proportion of participants with household members smoking was 33%. The average number of peers smoking was 2.25 (SD = 2.48). The average ADHD-H/I and ADHD-I symptoms scores were 6.91 (SD = 4.53) and 6.25 (SD = 4.84), respectively. The results of a previous study with this cohort revealed that the proportion of participants with clinically significant inattention, hyperactivity/impulsivity, and combined type symptoms were 2, 2, and 6%, respectively (Tercyak et al., 2002).

Model Fit
Measurement Model
The two piece latent growth curve measurement model fit the data well, $\chi^2$($17, 728$) = 27.85, $p = .05$, CFI = .99, RMSEA = .03, SRMR = .03. As noted, two growth factors were used, in addition to the level factor ($\eta_0$) because of the developmental transition from high school to post high school. The first growth factor ($\eta_1$) represented development of ND symptoms during high school from the fall of 10th grade to the end of 12th grade. The second growth factor ($\eta_2$) was used to model the transition from high school to post high school. The second growth factor was used to account for the higher levels of ND symptoms observed in the fall of 10th grade.

Table I: Proportion Smoking at Each Level by Wave

<table>
<thead>
<tr>
<th>Smoking level</th>
<th>High school waves</th>
<th>Waves post-high school</th>
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</thead>
<tbody>
<tr>
<td>Did not smoke in past 30-days</td>
<td>80.69</td>
<td>80.24</td>
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<tr>
<td>Smoked weekly</td>
<td>7.54</td>
<td>7.60</td>
</tr>
<tr>
<td>Smokes daily ≤10 cigarettes</td>
<td>5.38</td>
<td>4.41</td>
</tr>
<tr>
<td>Smokes daily &gt;10 cigarettes</td>
<td>4.98</td>
<td>6.08</td>
</tr>
</tbody>
</table>

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after high school.³ Factor loadings for the second growth factor (τₙ₂) were for waves 1–7, respectively, λ₁ₙ₂ = 0, λ₂ₙ₂ = 0, λ₃ₙ₂ = 0, λ₄ₙ₂ = 0, λ₅ₙ₂ = 2, λ₆ₙ₂ = 2.571, λ₇ₙ₂ = 2.043, reflecting no contribution during high school, and a slowing of growth followed by a decline thereafter. Factor loadings for the level factor (π₀) are constrained to 1 by default, as the level does not change with time. Figure 1 presents the two-piece LGCM with factor loadings and standardized path coefficients for the significant model paths.

Mean log ND symptoms level (π₀) was significantly different from zero at baseline (π₀ = 41, z = 14.81, p < .0001), as was log ND symptoms linear trend 1 (π₁ = .01, z = 2.05, p < .05). However, trend 2 was not significant (p > .05), suggesting nonsignificant change in ND symptoms in young adulthood (ages 18–21). There was, though, significant variability in all three growth factors (p < .05), indicating significant developmental heterogeneity in ND symptoms at baseline, and acceleration during and after high school.

Full Model
The full two-piece LGCM fit the data well with the three growth factors regressed on ADHD H-I and ADHD-I, and the remaining covariates, χ²(49, 672) = 56.89, p = .20, CFI = .99, RMSEA = .02, SRMR = .02.

Table II. Bivariate Correlation Matrix (n = 672)

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<td>.11</td>
<td>.14</td>
<td>1.00</td>
</tr>
<tr>
<td>15.</td>
<td>ADHD-I</td>
<td>.21</td>
<td>.10</td>
<td>.20</td>
<td>.20</td>
<td>.11</td>
<td>.11</td>
<td>.13</td>
<td>.02</td>
<td>.05</td>
<td>.07</td>
<td>.06</td>
<td>.21</td>
<td>.18</td>
<td>.70</td>
</tr>
<tr>
<td>Means</td>
<td></td>
<td>1.16</td>
<td>1.06</td>
<td>1.20</td>
<td>1.10</td>
<td>1.04</td>
<td>1.08</td>
<td>.96</td>
<td>1.50</td>
<td>.32</td>
<td>2.25</td>
<td>.33</td>
<td>1.16</td>
<td>1.46</td>
<td>6.90</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>1.72</td>
<td>1.67</td>
<td>1.60</td>
<td>1.53</td>
<td>1.43</td>
<td>1.53</td>
<td>1.46</td>
<td>.50</td>
<td>2.48</td>
<td>.47</td>
<td>.54</td>
<td>.97</td>
<td>4.55</td>
<td>4.84</td>
</tr>
</tbody>
</table>

³Each unit of time represented 6 months, thus the first two waves were spaced one unit apart, whereas the remaining waves were spaced two units apart; data collection occurred 1 year apart for waves 3–6.
to 12th grade), after an initial positive effect on baseline ND symptoms. This is likely the result of the strong positive effect of peer smoking on baseline level, leaving little room for further average ND symptoms growth after baseline. There were no other significant effects on linear trend 1.

### Table III. Nonstandardized Model Parameter Estimates, Standard Errors, and z-test Statistics for Log Nicotine Dependence Level and Two Developmental Trends

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>β</th>
<th>SE</th>
<th>z</th>
<th>β</th>
<th>SE</th>
<th>z</th>
<th>β</th>
<th>SE</th>
<th>z</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>-0.09</td>
<td>.056</td>
<td>-1.68</td>
<td>0.06</td>
<td>.064</td>
<td>1.04</td>
<td>-0.08</td>
<td>.055</td>
<td>-1.56</td>
</tr>
<tr>
<td>Non-Caucasian</td>
<td>0.06</td>
<td>.057</td>
<td>1.10</td>
<td>-0.02</td>
<td>.014</td>
<td>0.14</td>
<td>0.01</td>
<td>.056</td>
<td>0.18</td>
</tr>
<tr>
<td>Peer smoking</td>
<td>0.11</td>
<td>.012</td>
<td>9.55</td>
<td>-0.01</td>
<td>.003</td>
<td>-0.56</td>
<td>0.01</td>
<td>.065</td>
<td>0.16</td>
</tr>
<tr>
<td>Household smoking</td>
<td>0.095</td>
<td>.054</td>
<td>1.74</td>
<td>-0.005</td>
<td>.001</td>
<td>-0.15</td>
<td>0.01</td>
<td>.021</td>
<td>0.18</td>
</tr>
<tr>
<td>30-day marijuana use</td>
<td>0.147</td>
<td>.048</td>
<td>3.06</td>
<td>0.008</td>
<td>.003</td>
<td>0.24</td>
<td>0.01</td>
<td>.014</td>
<td>0.04</td>
</tr>
<tr>
<td>30-day alcohol use</td>
<td>-0.016</td>
<td>.025</td>
<td>-0.68</td>
<td>0.009</td>
<td>.007</td>
<td>1.24</td>
<td>0.004</td>
<td>.013</td>
<td>0.35</td>
</tr>
<tr>
<td>ADHD-H/I symptoms</td>
<td>0.006</td>
<td>.036</td>
<td>0.157</td>
<td>-0.007</td>
<td>.010</td>
<td>-0.73</td>
<td>0.046</td>
<td>.019</td>
<td>2.37</td>
</tr>
<tr>
<td>ADHD-I symptoms</td>
<td>0.001</td>
<td>.036</td>
<td>0.024</td>
<td>0.021</td>
<td>.010</td>
<td>2.09</td>
<td>-0.06</td>
<td>.019</td>
<td>3.10</td>
</tr>
</tbody>
</table>

**ND (nicotine dependence); ADHD-H/I symptoms (hyperactivity/impulsivity symptoms); ADHD-I symptoms (inattention symptoms); female (1, male; 2, female); non-Caucasian (0, Caucasian; 1, non-Caucasian); peers smoking (number of friends smoking, ranging from 0 to 9 friends smoking); household smoking (1, household member smokes; 0, no household members smoke).**

**aWe present the results for the log ND symptoms as ND symptoms were log transformed in the LGCM. Nonlog ND means, standard deviations, and correlations with other model variables are presented in Table II.**

**bND level—Log nicotine dependence symptoms level factor.**

**cND trend1—Log nicotine dependence symptoms linear trend factor representing development age 15 to 18-years old.**

**dND trend2—Log nicotine dependence symptoms linear trend factor representing development age 18 to 20-years old.**

**eADHD-H/I and ADHD-I symptoms were assessed in the fall of 10th grade only.**

**p ≤ .05; **p ≤ .01; ***p ≤ .001.**

### Nicotine Dependence Symptoms Trend, Waves 4–7 (Trend 2)

ADHD-H/I symptoms had a significant positive effect on ND symptoms trend 2, $\beta = 0.05$, $z = 2.37$, $p = .0177$, indicating that each unit increase in ADHD-H/I symptoms at age 15, was associated with an increase in...
ND symptoms change after high school (i.e., ages 18 to 21-years old). ADHD-I symptoms had a significant negative effect on ND symptoms trend 2, $\beta = -.06$, $z = -3.10$, $p = .0019$, suggesting that each unit increase in ADHD-I symptoms at age 15 was associated with a decrease in change from age 18 to 21-years old. It should be noted though that interpreting the effects of ADHD symptoms on trend 2 is not as straightforward as interpreting the effects on linear trend 1, as the last two factor loadings indicate gradual decrease across time. However, as the average intercept value is positive ($\eta_1 = .18$, $z = 3.11$, $p = .0019$), positive effects can be interpreted roughly as increasing the trend value, essentially resulting in ND symptoms increase for positive values and decrease in negative values. Finally, being female had a negative effect on ND symptoms trend 2, $\beta = -.06$, $z = -3.10$, $p = .0019$, suggesting that being female was associated with a decrease in ND symptoms change from ages 18 to 21-years old. There were no other significant effects on trend 2.

The Effects of ADHD Total Score on ND

As the DSM-IV includes a combined type in addition to ADHD predominantly inattentive and ADHD predominantly Hyperactive-Impulsive, we assessed the effect of ADHD total score on ND. There were no significant effects for ADHD total score on ND at baseline, trend 1, or trend 2.

Effect Sizes

Although the effect of ADHD–I ($\beta = .021$, 95% CI = .001, .041) on ND symptoms trend 1, and ADHD–I ($\beta = -.06$, 95% CI = -.099, -.022) and ADHD–H/I ($\beta = .046$, 95% CI = .008, .084) on ND symptoms trend 2 were significant, the results are tempered by the sizes of the 95% CI. Thus, although the effect of ADHD–I on change in ND symptoms (log transformed) across high school was .021 for each standard deviation increase, it could have been as small as .001 or as large as .041. Moreover, collectively, the predictor variables accounted for 13 and 12% of the variability in trends 1 and 2, respectively. Removing ADHD symptoms from the model, the effects on the same first and second trends were 9 and 6% respectively, accounting for between 3 and 6% of the ND symptoms trend variances.

Discussion

The objective of this study was to evaluate whether ADHD symptoms measured at age 15 affected progression in nicotine dependence symptoms prospectively in mid adolescence and during the transition to young adulthood, and whether this effect differed by ADHD symptoms (inattentive or hyperactivity–impulsivity). Although recent findings indicate that children diagnosed with ADHD are more likely to be nicotine dependent 10 years later (Biederman et al., 2006), this is the first study to model the effects of ADHD symptoms on the development of nicotine dependence symptoms over time in a community sample. Moreover, we found the effects of ADHD symptoms on nicotine dependence symptoms differed by ADHD symptoms subtypes and developmental period.

Although ADHD symptoms were unrelated to ND symptoms level at baseline, higher inattention symptoms were related to acceleration in ND symptoms during adolescence and deceleration in young adulthood. Further, ADHD-H/I symptoms were associated with acceleration in ND symptoms in young adulthood. These findings are somewhat consistent with previous research assessing the differential effects of ADHD symptoms with smoking. For instance, Molina & Pelham (2003) found inattention rather than hyperactivity–impulsivity prospectively predicted subsequent smoking from mid to late adolescence. However, effects beyond age 18 were not considered. Although speculative, the observed leveling off of ND symptoms might suggest that individuals with nonclinical attention deficits may rely less on nicotine to aid attention as they enter adulthood; it is possible that they developed alternative coping strategies to deal with attention difficulties. Only a study modeling repeated measures of both ADHD symptoms and ND symptoms over time (e.g., associated processes LGCM) could answer this question. In contrast, retrospective reports of childhood ADHD symptoms among young adults found that ADHD H/I symptoms were better predictors of ever regular smoking (ever smoking at least one cigarette per day for 30 days) than ADHD-I symptoms (Kollins et al., 2003). These somewhat disparate findings may be related to study design (prospective vs. retrospective) as well as definitions of regular smoking. Another possibility is that participants in this study who were clinically diagnosed with ADHD underreported symptoms which could have skewed the effects of ADHD symptoms on ND symptoms in adolescence and young adulthood (Barkley, Fischer, Smallish, & Fletcher, 2002). Unfortunately, since this is a community sample, there is no way to confirm this possibility.

Clinically, the inattentive and hyperactivity–impulsivity features of ADHD make distinctive subtypes
of this disorder. It is not entirely surprising that the ADHD-I and ADHD-H/I symptoms have unique effects on ND symptoms at different points in development. Indeed, data from a prior report suggests the importance of separating these symptom profiles in relation to smoking among unimpaired teenagers (Tercyak et al., 2002), and among adults attempting to stop smoking (Lerman et al., 2001). Consistent with the hypothesis that some smokers self-medicate with nicotine, these results suggest that those with more frequent symptoms of inattention may use nicotine as a stimulant drug to help some ADHD symptoms.

Although the findings of this study suggest a prospective relationship between ADHD symptoms in mid adolescence and change in ND symptoms in adolescence and young adulthood, it is not known why the two are related. There are at least two possibilities. First, adolescents with ADHD and higher ADHD symptoms may tend to affiliate with more deviant peers (Laucht et al., 2007; Marshal et al., 2003). Greater deviant peer relationships have been associated with substance use, including tobacco (Simons-Morton et al., 2001). A second possibility is the negative reinforcing effects of nicotine. That is, nicotine appears to reduce symptoms of inattention and poor control (Gehricke et al., 2006; Potter & Newhouse, 2004). Thus, it is possible that adolescents and adults with ADHD symptoms smoke to alleviate ADHD symptoms. Only further prospective research with clinical and community samples can assess the viability of these speculations.

The finding that ADHD-H/I symptoms were associated with greater acceleration in ND symptoms during young adulthood (ages 18 to 20-years old) was revealed through the analysis of data beyond adolescence allowing us to capture the development and progression in ND symptoms that occurs in emerging adulthood. ADHD-H/I symptoms may be more highly associated with other problematic behaviors, such as drug use that promote greater levels of smoking and dependence on nicotine in emerging adulthood (Barkley, Fischer, Edelbrock, & Smallish, 1990; Disney, Elkins, McGuie, & Iacono, 1999; Fergusson, Lynskey, & Horwood, 1996; Flory & Lynam, 2003; Milligan et al., 1997). Methodologically, the differential results for ADHD-H/I and ADHD-I symptoms during adolescence and young adulthood indicate the importance of piecewise modeling when dealing with developmental transitions. A single linear growth factor would not have captured the differential effects across the transition to young adulthood.

These findings need to be considered within the context of the study limitations. Most notably ADHD symptoms were obtained via self-report. However, unlike other studies relying on self-reported symptoms (Kollins et al., 2005) that asked participants to report symptoms occurring approximately 10 years prior, this study asked adolescents to report symptoms occurring within the past 6 months. Moreover, as noted previously, adolescents with clinically diagnosed ADHD tend to underreport ADHD symptoms in self report (Barkley et al., 2002). A second limitation is ADHD symptoms were reported for the fall of 10th grade only (age 13), and it is possible that ADHD symptoms changed over time. Third, unlike others studies (Galera et al., 2005; Kollins et al., 2005), we did not measure or control for conduct disorder. Controlling for disruptive behavior disorders or personality traits might alter or better explain the association between hyperactivity-impulsivity symptoms and nicotine dependence (Dinn, Aycicegi, & Harris, 2004; Wilson & Levin, 2005). However, we did control for substance use, peers and family smoking, and baseline smoking, which have been shown to relate to adolescent nicotine dependence and ADHD symptoms (Rodriguez & Audrain-McGovern, 2004; Saules, Pomerleau, & Schubiner, 2003; Whalen et al., 2002). In addition, a recent study found that controlling for conduct disorder did not remove the effects of ADHD symptoms (Kollins et al., 2005), which is consistent with studies that have considered the independent effects of inattention (Milligan et al., 1997). Fourth, the overall consent rate of 54% suggests caution generalizing these findings to the population as a whole. However, it is also important to note that the sample was nationally representative based on demographics (U.S. Census Bureau, 2001b) and regionally and locally representative based on smoking behavior (2001a, 2006). Moreover, those lost to follow-up were more likely to be male and non-Caucasian. Although this is not uncommon in longitudinal studies of health behavior (Divaik, Wahl, O’Keefe, Mermelstein, & Flay, 2006), it is not clear how the current findings apply to these two subgroups. Finally, the study design may not adequately capture development of ND in adolescents, as recent studies suggest that ND develops quite early and rapidly in adolescence (DiFranza et al., 2007). Future studies should assess the qualitative differences of ND measures for adolescents, and assess ND more frequently to better capture its development and assess its predictors.

This is the first study to our knowledge to assess the effects of ADHD symptoms on ND symptoms prospectively in a community sample of adolescents, and to assess
how these symptoms impact adolescents during the transition into young adulthood. Future research should
assess how ADHD symptoms and ND symptoms covary over time, gathering repeated measures of ADHD
symptoms along with information about smoking. Moreover, future studies should attempt to replicate these
findings with community and clinical samples.

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