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Short Communication

Smoking patterns and abstinence effects in smokers with no ADHD, childhood ADHD, and adult ADHD symptomatology

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Abstract

Cigarette smokers are known to be overrepresented among adults with Attention Deficit Hyperactivity Disorder (ADHD). To date, however, no attempt has been made to determine the extent to which a lifetime diagnosis of ADHD may be associated with smoking even in the absence of current symptomatology. We hypothesized that nicotine dependence and abstinence effects—especially effects relevant to ADHD symptomatology—would be more pronounced in adult ADHD smokers in comparison with those who reported childhood ADHD symptoms only. Results indicated that, in contrast to controls without ADHD symptomatology, both adult and childhood ADHD groups were significantly more likely to experience a number of nicotine withdrawal symptoms, including irritability and difficulty concentrating; in no instance did the ADHD groups differ from one another in this regard. Thus, studying people with childhood symptoms of ADHD, even in the absence of an adult diagnosis, may shed light on the known association between smoking and ADHD.

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1. Introduction

Despite changing nomenclature over the past several decades, Attention Deficit Hyperactivity Disorder (ADHD) has long been recognized as a disorder of poor impulse control and distractibility that affects approximately 5–15% of children in the general population (Nolan, Gadow, & Sprafkin, 2001; Scahill & Schwab-Stone, 2000; Wolraich, Hannah, Baumgaertel, & Feurer, 1998), with considerably higher rates among disadvantaged sectors such as the child welfare, mental health, and juvenile systems (Garland et al., 2001). ADHD in childhood and adolescence is associated with a variety of self-management deficits and impairments in cognitive, social, family, and school functioning (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman, Faraone, Milberger, Guite, et al., 1996; Faraone & Biederman, 1998), including conduct and substance use disorders (Milberger, Biederman, Faraone, Wilens, & Chu, 1997). Increased smoking prevalence and earlier onset of cigarette smoking have also been demonstrated in adolescents with ADHD in comparison with controls (Lambert & Hartsough, 1998; Milberger, Biederman, Faraone, Chen, & Jones, 1997).

More recently, it has been found that ADHD persists into adulthood in approximately 65% of those diagnosed with the childhood syndrome (Biederman, Faraone, Milberger, Curtis, et al., 1996; Weiss & Hechtman, 1993); among the remaining 35%, the condition appears to abate or resolve. Factors related to the persistence of ADHD include family history of ADHD, psychosocial adversity, and comorbidity with conduct, mood, and anxiety disorders (Biederman, Faraone, Milberger, Curtis, et al., 1996). As in childhood, ADHD in adulthood is associated with numerous complications, including social, intellectual, and neuropsychological deficits, impulsivity, marital dissolution, and criminality (Biederman et al., 1993; Downey, Pomerleau, & Pomerleau, 1996; Downey, Stelson, Pomerleau, & Giordani, 1997; Mannuzza, Klein, Konig, & Giampino, 1989; Seidman, Biederman, Weber, Hatch, & Faraone, 1998). Furthermore, ADHD in adulthood is associated with an increased likelihood of mood and substance use disorders (Biederman, Wilens, Mick, Faraone, & Spencer, 1998; Carroll & Rounsaville, 1993; Faraone et al., 2000; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990), earlier onset of substance use disorders (Wilens, Biederman, Mick, Faraone, & Spencer, 1997), and poorer prognosis in terms of recovery from substance use disorders (Wilens, Biederman, & Mick, 1998).

Cigarette smokers are also known to be overrepresented among adults with ADHD (Pomerleau, Downey, Stelson, & Pomerleau, 1995). Since nicotine has been shown to improve performance and decrease distractibility in both smokers and never-smokers (Conners et al., 1996; Levin et al., 1996, 1998), the idea that smoking represents a form of self-medication of ADHD symptomatology among adult smokers with this disorder (Khantzian, 1997; Pomerleau, Marks, & Pomerleau, 2000) has face validity. Interpretation of this relationship, however, is complicated by the fact that establishment of an adult diagnosis of ADHD requires a positive childhood diagnosis as well. Among other psychiatric cofactors for smoking—e.g., depression—a lifetime diagnosis is associated with smoking (Glassman et al., 1988, 1990), leading some researchers to speculate about the possibility of shared etiologies rather than (or in addition to) a causal relationship (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998), and to suggest that genetic factors predispose to both smoking and depression (Kendler et al.,

1993). To the best of our knowledge, however, no attempt has been made to date to determine the extent to which a lifetime diagnosis of ADHD may be associated with smoking even in the absence of current symptomatology.

As a preliminary exploration of this issue, we administered a questionnaire based on DSM-IV criteria, querying both childhood and adult symptomatology, to daily smokers recruited from the local community, as well as a variety of measures of smoking, nicotine dependence, and abstinence effects. The sample included individuals with no evidence of ADHD, with childhood symptomatology only, and with symptoms consistent with an adult diagnosis of ADHD. We hypothesized that experimentation and onset of regular smoking would occur at an earlier age in both childhood and adult ADHD smokers, compared with smokers with no ADHD. Because we have found that smokers often report nicotine withdrawal symptoms pathognomonic of underlying psychological disturbances (e.g., depression, anxiety, disordered eating; Pomerleau et al., 2000), we hypothesized that nicotine dependence and abstinence effects—especially effects relevant to ADHD symptomatology—would be more pronounced in adult ADHD smokers in comparison with those who reported childhood ADHD symptoms only.

2. Methods

2.1. Subjects

Subjects were 282 daily smokers (74.8% female; 79.1% Caucasian) recruited from the local community to participate in either laboratory investigations of smoking behavior and effects or clinical trials for smoking cessation. Consent for the collection of baseline data was obtained, and individuals who did not qualify for subsequent participation in a laboratory experiment were paid for completing the questionnaire battery. Because some of these individuals were specifically recruited to participate in studies of ADHD in smokers, we were able to assemble a sample enriched for both childhood-only (13.1%; $n = 37$) and childhood/adult (16.7%; $n = 47$) ADHD.

2.2. Procedure

Prior to their participation in a study, subjects completed a questionnaire battery that included standard demographic data, data on smoking patterns and history, the Fagerström Test for Nicotine Dependence (FTND; Fagerström, Heatherton, & Kozlowski, 1991; Heatherton, Kozlowski, Frecker, & Fagerström, 1991), the Center for Epidemiological Studies—Depression (CES-D; Radloff, 1977; Weissman, Sholomakis, Pottenger, Proshoff, & Locke, 1977), and the CAGE (Mayfield, McLeod, & Hall, 1974), a widely used four-item scale validated as a screening instrument for alcohol dependence. In addition, subjects filled out a questionnaire designed to assess ADHD, a brief case finding instrument that has been validated using a semistructured psychiatric interview as the “gold standard” (Mehring et al., *in press*). Both childhood (by age 12) and current DSM-IV symptomatology were

queried. Participants were classified as having either (1) no evidence of ADHD history (abbreviated henceforth as NH, i.e., <6 childhood hyperactive symptoms, <6 childhood inattentive symptoms, <6 adulthood hyperactive symptoms, and <6 adulthood inattentive symptoms); (2) having evidence of childhood ADHD only (abbreviated CH; i.e., at least six childhood hyperactive symptoms and/or at least six childhood inattentive symptoms but <6 adulthood hyperactive symptoms and <6 adulthood inattentive symptoms); or (3) having evidence of ADHD persisting into adulthood (abbreviated AD; at least six childhood hyperactive symptoms and/or at least six childhood inattentive symptoms; and at least six adulthood hyperactive symptoms and/or at least six adulthood inattentive symptoms).

2.3. Data analyses

Group differences in continuous variables were tested using one-way analysis of variance, with post hoc testing using Tukey's HSD correction to detect pairwise between-group

Table 1

Demographic and baseline data for participants with no history of ADHD, childhood ADHD only, and childhood/adult ADHD

	No ADHD (NH) [<i>n</i> = 198]	ADHD (childhood only) (CH) [<i>n</i> = 37]	ADHD (adult) (AD) [<i>n</i> = 47]	Significance (<i>P</i> value) ^a	
Age (years)	33.1 ± 11.7	29.4 ± 9.4	29.4 ± 9.4	NS	
Sex (percent female)	78.3%	64.9%	68.1%	NS	
Race (percent Caucasian)	79.8%	81.1%	74.5%	NS	
Education (years completed)	14.2 ± 1.9	13.1 ± 1.8	14.0 ± 1.9	<i>F</i> = 5.09	<i>P</i> < .01
				NH vs. CH	<i>P</i> < .01
Childhood hyperactivity symptoms	1.5 ± 1.6	5.4 ± 2.3	6.1 ± 2.4	<i>F</i> = 165.5	<i>P</i> < .001
				NH vs. CH	<i>P</i> < .001
				NH vs. AD	<i>P</i> < .001
Childhood inattentiveness symptoms	1.6 ± 1.7	5.7 ± 1.5	6.4 ± 1.6	<i>F</i> = 220.3	<i>P</i> < .001
				NH vs. CH	<i>P</i> < .001
				NH vs. AD	<i>P</i> < .001
Adult hyperactivity symptoms	1.7 ± 1.6	2.5 ± 1.9	6.1 ± 2.4	<i>F</i> = 136.3	<i>P</i> < .001
				NH vs. CH	<i>P</i> < .05
				NH vs. AD	<i>P</i> = .001
				CH vs. AD	<i>P</i> < .001
Adult inattentiveness symptoms	1.7 ± 1.8	2.7 ± 1.8	6.0 ± 1.6	<i>F</i> = 112.9	<i>P</i> < .001
				NH vs. CH	<i>P</i> < .01
				NH vs. AD	<i>P</i> < .001
				CH vs. AD	<i>P</i> < .001
Depression (CES-D score)	10.4 ± 9.4	8.9 ± 8.3	16.9 ± 10.5	<i>F</i> = 9.61	<i>P</i> < .001
				NH vs. AD	<i>P</i> < .001
				CH vs. AD	<i>P</i> < .01
History of alcohol dependence (percent with CAGE score ≥ 1)	42.8%	51.4%	65.2%	NH vs. AD	<i>P</i> < .01

^a For continuous variables, post hoc comparisons were made using Tukey's HSD; for categorical variables, group differences were tested using logistic regression.

differences. Group differences in categorical variables were tested using logistic regression, with the ADHD category (no history of ADHD, childhood ADHD, and childhood/adult ADHD) entered as a predictor.

3. Results

Demographic and baseline characteristics for smokers with no history of ADHD symptomatology (NH, $n = 198$), a history of childhood but no adult symptomatology (CH, $n = 37$), and with symptomatology persisting into adulthood (AD, $n = 47$) are shown in Table 1. There were no significant group differences in age, or in sex and race distribution. Small but significant differences in education were observed, with CH having fewer years of education than NH. As expected, AD scored significantly higher on current depression than either of the other groups, and significantly higher on alcohol problems than NH. Although AD scored significantly higher than both NH and CH on adult hyperactive and inattentive symptomato-

Table 2

Patterns of smoking and withdrawal symptomatology for participants with no history of ADHD, childhood ADHD only, and childhood/adult ADHD

	No ADHD (NH) [$n = 198$]	ADHD (childhood only) (CH) [$n = 37$]	ADHD (adult) (AD) [$n = 47$]	Significance (P value) ^a	
Age at first experimentation (years)	14.6 ± 3.3	12.8 ± 2.9	13.8 ± 2.6	$F = 5.29$	$P < .01$
Age started smoking regularly (years)	17.0 ± 3.0	15.6 ± 3.4	16.5 ± 3.2	$F = 3.59$	$P < .05$
Cigarette consumption (cigarettes/day)	17.9 ± 7.8	20.9 ± 9.7	18.4 ± 8.5	NS	
Nicotine dependence (FTND score)	4.3 ± 2.4	5.0 ± 2.4	4.9 ± 2.4	NS	
Percent reporting craving as a withdrawal symptom	77.3%	89.2%	87.0%	NS	
Percent reporting DSM-IV withdrawal symptoms					
Depressed mood	13.6%	18.9%	29.8%	NH vs. AD	$P < .01$
Insomnia	9.6%	24.3%	21.3%	NH vs. CH	$P < .05$
Irritability	51.0%	70.3%	68.1%	NH vs. AD	$P < .05$
Anxiety	42.9%	40.5%	53.2%	NH vs. CH	$P < .05$
Difficulty concentrating	25.8%	43.2%	55.3%	NH vs. AD	$P < .01$
Restlessness	61.6%	89.2%	76.6%	NH vs. CH	$P < .01$
Decreased heart rate	4.5%	5.4%	4.3%	NS	
Increased appetite	36.4%	54.1%	40.4%	NH vs. CH	$P < .05$

^a For continuous variables, post hoc comparisons were made using Tukey's HSD; for categorical variables, group differences were tested using logistic regression.

logy, as expected from the classification strategy, CH also scored significantly higher than NH on these dimensions.

Data on patterns of smoking initiation and withdrawal symptomatology are shown in Table 2. CH participants recalled having started smoking earlier than NH participants ($P < .05$), but were not significantly different from AD participants. AD participants were more likely than NH participants to experience the following DSM-IV nicotine withdrawal symptoms: depressed mood ($P < .01$), insomnia ($P < .05$), irritability ($P < .05$), and difficulty concentrating ($P < .01$). CH participants were more likely than NH participants to experience withdrawal symptoms of insomnia ($P = .014$), irritability ($P = .034$), difficulty concentrating ($P < .05$), restlessness ($P < .01$), and increased appetite ($P < .05$). CH participants were not significantly different from AD participants on frequency of any withdrawal symptoms.

4. Discussion

Our findings confirm that ADHD is associated with increased severity along several dimensions of nicotine dependence and smoking in comparison with unaffected individuals. As hypothesized, higher levels of depression were observed in smokers with adult ADHD symptomatology. Our results further (and more surprisingly) suggest that a *history* of ADHD is at least as strongly predictive of these differences as is a current diagnosis. Both AD and CH participants were significantly more likely to experience a number of nicotine withdrawal symptoms, but in no instance did AD and CH groups differ from one another in this regard.

There are several possible explanations for these findings. (1) At least some childhood-only smokers may not show adult symptomatology because they are successfully using nicotine to manage their symptoms (i.e., they are “nicotine responders”). (2) Even though the CH group did not meet criteria for AD, they had significantly more adult symptomatology than did the NH group; thus, it is possible that the inclusion of subclinical cases of AD ADHD in the CH sample contributed to the results. (3) Childhood ADHD may have triggered tobacco use (as suggested by earlier age of experimentation and regular smoking), and these individuals remained dependent and continued smoking even after outgrowing the need or trigger for initiating and maintaining smoking during adolescence. (4) There may be a noncausal relationship between ADHD and smoking, with some common underlying variable driving both conditions, as has been suggested for depression (Breslau et al., 1998; Kendler et al., 1993). The fact that CH and AD do not differ with respect to self-reported smoking withdrawal symptomatology, but both differed from NH along several dimensions, lends support to the first two explanations. Further research in larger samples, however, will be needed to clarify these issues. Information on whether smoking is overrepresented among adults with a history of childhood ADHD but no adult ADHD in population-based samples would also be helpful in interpreting our results.

Some limitations of our study should be noted: Participants were not formally diagnosed with ADHD but rather evaluated using a screening measure that, in keeping with its intended use as a case finding measure, tends to overdiagnose. As such, conclusions regarding the diagnosis of ADHD must remain tentative until results can be confirmed diagnostically.

Second, our assessments of withdrawal are subject to the shortcomings of all retrospective data, such as recall bias. Third, the relatively smaller size of the two ADHD groups resulted in less power to detect differences between these two groups; e.g., our failure to detect differences between these two groups in depressed mood as a withdrawal symptom, despite fairly substantial differences in the percentage reporting such symptoms, may be an artifact of this lower power, especially given significant differences in current depression between the two groups. Finally, results from this sample of relatively well-educated, largely Caucasian, predominately female, young adult sample may not generalize to other subpopulations of smokers.

Nonetheless, our findings indicate that studying people with childhood symptoms of ADHD, even in the absence of an adult diagnosis, may shed light on the known association between smoking and ADHD. Because childhood ADHD is more prevalent and more widely studied than adult ADHD, data from large-scale childhood samples may be able to provide important insights into the smoking–ADHD relationship. Future research should be directed towards better defining such relations in childhood, and determining if they persist into adulthood, in order to better tailor smoking cessation and/or harm minimization efforts for this challenging population.

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