The Association between Attention Deficit Hyperactivity Disorder in Adolescence and Substance Use Disorders in Adulthood

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Abstract

Objectives—To determine whether attention deficit hyperactivity disorder in adolescence (ADHD) is related to substance use disorders (SUDs) in adulthood, and whether conduct disorder (CD) mediates this relationship.

Design—A prospective design incorporating five assessments, spanning the mean ages of 14 to 37. Two baseline assessments were taken at ages 14 and 16, and three outcome assessments were taken between the ages 27–37.

Setting—United States.

Participants—A community sample of participants initially drawn from upstate New York in 1975, and followed to a mean age of 37.

Intervention(s) or Main Exposure(s)—The Diagnostic Interview Schedule for Children was used for assessing ADHD and CD, and the University of Michigan Composite International Diagnostic Interview (UM-CIDI) was used for assessing SUDs.

Main Outcome Measure(s)—A diagnosis of a SUD was given to the participants in adulthood.

Results—Attention deficit hyperactivity disorder in adolescence was related to SUDs in adulthood. This association, however, was indirect, as CD served as a mediator between ADHD and SUDs.

Conclusions—Although adolescent ADHD is not prospectively associated with SUDs in adulthood, it remains a significant risk factor for SUDs through the correlations between ADHD and CD, and CD with SUDs.

INTRODUCTION

Substance use disorders (SUDs) are associated with psychopathology as well as low educational levels, unemployment, and risky sexual behavior.1–3 Given the negative consequences of SUDs, it is important to identify its predictors. One factor researchers have identified as related to the development of SUDs is the presence of attention deficit hyperactivity disorder (ADHD). Cross-sectional and longitudinal research indicates that ADHD in childhood or adolescence is associated with an increased risk for substance use or dependence later in adolescence or in early adulthood.4–10 Another factor investigators have shown to be related to SUDs is conduct disorder (CD).11

In addition to their relationship to SUDs, ADHD and CD are strongly correlated with one another.12,13 Researchers have attempted to identify the independent association of ADHD
with the onset of SUDs, most often by statistically controlling for the presence of CD and/or other psychopathology.\textsuperscript{7–10}

These attempts to link diagnoses of ADHD to SUDs have generally failed to find an independent prospective association between ADHD and SUDs. For instance, Elkins et al.\textsuperscript{9} examined the relationship between diagnoses of ADHD on the one hand, and three types of SUDs (nicotine dependence, alcohol abuse/dependence, and cannabis abuse/dependence) on the other. Diagnoses of ADHD were not related to the diagnosis of SUDs at age 18, once analyses were adjusted to control for childhood CD. Similarly, after controlling for other psychopathology, including CD, Biederman et al.\textsuperscript{8} found that ADHD in childhood and adolescence was related to lifetime and one-year prevalence of nicotine dependence at a 10-year follow-up, but not to drug or alcohol dependence.

To our knowledge, only one study\textsuperscript{10} has found that a diagnosis of ADHD is independently, prospectively linked to the subsequent development of SUDs. Gau and colleagues\textsuperscript{10} found that a diagnosis of ADHD at age 12 was related to the development of a SUD within the next three years, even after controlling for other psychopathology, including CD. One possible explanation for the discrepancy between Gau et al.’s findings and those of other researchers noted above is that Gau et al. employed a shorter longitudinal time-frame than other studies, and their outcome assessment took place at a younger age. Since ADHD is most prevalent in childhood and early adolescence,\textsuperscript{14,15} it may be that the disorder exerts a direct influence on SUDs at these early ages, but the effect is mediated over time.

Given that the literature suggests a stronger relationship between CD and SUDs than between ADHD and SUDs, we hypothesize a mediational model.\textsuperscript{16} In order to test our mediational hypothesis we apply Baron and Kenny’s\textsuperscript{16} three conditions for a possible mediational pathway: (1) the independent variable (ADHD) is related to the potential mediator (CD), (2) the mediator (CD) is related to the dependent variable (SUDs), despite control on the independent variable, and, (3) the relationship between the independent and dependent variable is rendered non-significant once there is statistical control for the effect of the mediator.

Another issue is whether there is an interactive effect of ADHD and CD on SUDs. There is a basis for this supposition, as Lynam\textsuperscript{17} has maintained that ADHD and CD combine multiplicatively to put individuals at particular risk for poor developmental outcomes. There has been limited research investigating whether this interaction extends to symptom counts of ADHD and CD combining to create a high risk of SUDs or symptoms of dependence.\textsuperscript{18,19} However, these studies have attained mixed results. Moreover, there have been no attempts to test for an interaction between diagnoses of ADHD and CD leading to diagnoses of SUDs. Given the limited prior research on this issue, we propose to test for such an interaction.

Following the mediational model cited above, we test the following hypotheses: (1) ADHD is related to CD; (2) CD in adolescence is associated with SUDs in adulthood, even after controlling for ADHD; and (3) ADHD in adolescence is associated with SUDs in adulthood, but this effect no longer holds after controlling for CD.

We extend the literature in three important respects. First, to our knowledge, our study is the first to formally test for CD as a mediator between ADHD and SUDs. As noted above, we proposed CD is the mediator, as its relationship with SUDs is more powerful than the relationship between ADHD and adult SUDs.\textsuperscript{7–9} Second, we follow our sample to a much later age of SUDs than have other researchers who have studied the relationship between ADHD and SUDs. Thus, our data allows us to investigate the association of ADHD in adolescence and SUDs in adulthood.
METHODS

Participants and Procedure

Data for the participants in this study came from a community-based random sample residing in one of two upstate New York counties first assessed for drug use in 1983. The sample was taken from an earlier study using maternal interviews in 1975 (T1). The original maternal/youth study assessed problem behavior among youngsters. The sampled families were generally representative of the population of families in Albany and Saratoga, two upstate New York counties, with respect to gender, family intactness, family income, and education. Interviews of both mothers and youths were conducted in 1983 (T2, N=756), 1985–1986 (T3, N=739), and 1992 (T4, N=750). Three more interviews of the second generation were conducted in 1997 (T5, N=749), 2002 (T6, N=673), and 2005–2006 (T7, N=607). Some of the participants who were not interviewed in previous years were interviewed in later waves of data collection. The mean ages (SDs) of participants at the interviews were 14.1 (2.8) at T2, 16.3 (2.8) at T3, 22.3 (2.8) at T4, 27 (2.8) at T5, 31.9 (2.8) at T6, and 36.6 (2.8) at T7, respectively. In the current analyses, we included the participants whose measurements of SUDs at T5, T6, and T7 and ADHD and CD at T2 and T3 were available (N=515). Among these participants, 290 (56.3%) were females. Using the T2 sample as the baseline, there was no significant association (p-value>0.05) between those included in the analysis and those who were excluded (N=241) from it with respect to any T2 substance use, e.g., tobacco use (t=0.03, p-value=0.98), marijuana use (t=0.6, p-value=0.57), and other illicit drug use (t=0.9, p-value=0.35). However, there were a significantly higher percentage of female participants in the 515 sample than in the 241 sample (56.3% and 42.7%, respectively; χ²(1)=19, p-value<0.001).

Extensively trained and supervised lay interviewers administered interviews in private. Written informed consent was obtained from participants and their mothers in 1983, 1986, and 1992, and from participants only in 1997, 2002, and 2007. The Institutional Review Board of New York University School of Medicine authorized the use of human subjects in this research study. Additional information regarding the study methodology is available from prior publications.20

Measures

ADHD and CD at T2–T3: The parent and youth versions of the Diagnostic Interview Schedule for Children (DISC-I) were administered in 1983 (T2), and again in 1985–1986 (T3) to assess psychiatric disorders including ADHD and CD. Symptoms of ADHD include an unusually high and chronic level of inattention, hyperactivity, or both. Some items from other parts of the questionnaire were added to the Disc-I to make the diagnosis of ADHD consistent with the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).21 Criteria from DSM-IV were used to classify the participants with respect to ADHD.

According to the DSM-IV, conduct disorder is a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms are violated, as manifested by the presence of three (or more) of several criteria in the past 12 months, with at least one criterion present in the past six months.

Substance Use Disorder at T5–T7: At T5, T6 and T7, substance use disorder (dependence or abuse) was assessed by use of the University of Michigan Composite International Diagnostic Interview (UM-CIDI) substance use disorder measure.22 We adapted this measure to make it consistent with the criteria used in the DSM-IV.21 We ascertained substance dependence by the presence of three or more of the following criteria for each substance used during the same 12-month period before the interviews at both T6 and T7: (1) use of more of...
the substance than usual to get the same effect, or the same amount has less of an effect than
before; (2) the presence of withdrawal symptoms or the use of the substance to avoid
withdrawal symptoms; (3) the use of much larger amounts of the substance than intended, or
use for a longer period of time than intended; (4) the presence of such a strong desire or urge
to use the substance that the person could not resist using it; (5) a period of a month or more
in which the person spent a great deal of time using the substance or getting over its effects;
(6) the person gave up activities because of use of the substance; or, (7) emotional or
psychological problems resulting from using the substance such as feeling uninterested in
things, feeling depressed, suspicious of people, paranoid, or having strange ideas. If a
participant did not meet the criteria for substance dependence, substance abuse was ascertained
by the presence of at least one of the following four criteria during the same 12-month period:
(1) being under the effects of the substance or suffering its after-effects while at work or school
or while taking care of children; (2) being under the effects of the substance or feeling its after-
effects in a situation which increased the user’s chances of getting hurt – i.e., when driving a
car or boat, using knives or guns or machinery, crossing against traffic, climbing or swimming;
(3) having legal problems because of use of the substance; or (4) having problems getting along
with other people because of use of the substance.

Analytical Plans—Chi-square tests were used to test whether gender and age were associated
with SUDs, and whether gender was associated with earlier ADHD and CD. We conducted
four separate logistic regression analyses to examine the association between earlier adolescent
ADHD (at T2 or T3) and later adult SUDs (at T5, T6, or T7): (1) without controls; (2)
controlling for earlier adolescent CD; (3) controlling for gender, age, and adolescent SUDs
and major depressive disorder (MDD); and, (4) controlling for earlier adolescent CD, gender,
age, and adolescent SUDs and MDD. We also conducted four separate logistic regression
analyses to examine the association between earlier adolescent CD (at T2 or T3) and later adult
SUDs (at T5, T6, or T7): (1) without controls; (2) controlling for earlier adolescent ADHD;
(3) controlling for gender, age, and adolescent SUDs and MDD; and, (4) controlling for earlier
adolescent ADHD, gender, age, and adolescent SUDs and MDD. Next, we conducted a logistic
regression analysis to examine the relationship between adolescent ADHD and CD, with CD
as the dependent variable and ADHD as the independent variable. Using the formula created
by MacKinnon and Dwyer23 we calculated the mediational effect of earlier adolescent CD,
which operated between earlier adolescent ADHD and later adult SUDs. Finally, we used the
likelihood ratio test (LRT) to examine the significance of the following interactional effects
on later adult SUDs: between adolescent ADHD and CD, between adolescent ADHD and
gender, and between adolescent CD and gender.

RESULTS

At T5, T6, or T7, 12% of the adults were diagnosed as having SUDs according DSM-IV criteria.
The percentage of SUDs for males was significantly higher than that for females [17.8% and
7.6%, respectively; χ²(1) = 12.4, p-value < 0.001]. Participants from the younger age group
(<36 years at T7) had a similar percentage of SUDs as that of participants from the older age
group (>36 years at T7) [11.7% and 12.3%, respectively; χ²(1) = 0.05, p-value = 0.8]. At T2
or T3, 12.8% of the adolescents were diagnosed as having ADHD and 13.6% of the adolescents
were diagnosed as having CD. In total, 20.6% of the adolescents were diagnosed as having
either ADHD alone (7%), CD alone (7.8%), or both ADHD and CD (5.8%). The percentage
of males who had ADHD was not significantly different from the percentage of females who
had the disorder [14.2% and 11.7%, respectively; χ²(1) = 0.7, p-value = 0.4]. At T2 or T3, the
percentage of males who had CD was significantly higher than the percentage of females who
had CD [17.3% and 10.7%, respectively; χ²(1) = 4.8, p-value = 0.03].
As noted in Table 1, the logistic regression analyses revealed that, without controls, adolescents who had earlier ADHD at T2 or T3 were 2.5 times (95% C.I. = [1.3, 4.8], p=0.005) more likely to have a later adult SUDs than those who did not have earlier ADHD. However, the association between earlier ADHD and later SUDs was no longer significant with earlier CD controlled (A.O.R. = 1.6, 95% C.I. = [0.8, 3.4], p=0.18). In addition, earlier adolescent ADHD at T2 or T3 was significantly related to later adult SUDs at T5, T6, or T7, with gender, age, earlier SUDs, and earlier MDD controlled (A.O.R. = 2.1, 95% C.I. = [1.0, 4.3], p=0.04). The relationship between ADHD in adolescence and later SUDs in adulthood was no longer significant with control on CD, gender, age, earlier SUDs, and earlier MDD (A.O.R =1.6, 95% C.I. = [0.7, 3.4], p=0.26).

As noted in Table 2, the logistic regression analyses revealed that, without controls, adolescents who had earlier CD at T2 or T3 were 3.5 times (95% C.I. = [1.9, 6.4], p<0.001) more likely to have later adult SUDs than those who did not have earlier CD. The participants who had earlier CD remained significantly more likely to have later SUDs with earlier ADHD controlled (A.O.R. = 2.9, 95% C.I. = [1.5, 5.7], p=0.002). In addition, earlier adolescent CD at T2 or T3 was significantly associated with later adult SUDs (A.O.R. = 2.9, 95% C.I. = [1.5, 5.6], p=0.002) with gender, age, earlier SUDs, and earlier MDD controlled. CD remained significantly associated with later adult SUDs with control on ADHD, gender, age, earlier SUDs, and earlier MDD (A.O.R. = 2.5, 95% C.I. = [1.3, 5.1], p=0.009).

We then conducted logistic regression analyses to examine the association between ADHD (T2 or T3) and CD (T2 or T3). The results indicated that adolescents with ADHD are 8.5 times more likely to have CD (p<0.001) than those who did not have ADHD. Therefore, the findings suggest a mediational effect of ADHD on adult SUDs via CD. Using the formula created by MacKinnon and Dwyer,\textsuperscript{23} we calculated the mediational effect, which was 0.6 and statistically significant (SE=0.2, t=2.9, p=0.004).

As noted in the analysis section, we then tested the following interaction term: ADHD at T2 or T3 by CD at T2 or T3. The results indicated that the interaction term (ADHD by CD) was not significantly related to SUDs with control on ADHD, CD, gender, age, earlier SUDs, and earlier MDD ($\chi^2(1) = 0.4$, p-value = 0.55). Thus, there was no indication of an interaction between earlier adolescent ADHD and CD in leading to later adult SUDs. The results also indicated that the interaction terms (ADHD by gender and CD by gender) were not significantly related to SUDs with control on ADHD, CD, gender, age, earlier SUDs, and earlier MDD ($\chi^2(1) = 1.5$, p-value = 0.22 and $\chi^2(1) = 2.6$, p-value = 0.1, respectively)

**DISCUSSION**

Our findings support our hypotheses. First, ADHD is related to CD. Second, CD is highly related to SUDs, even with control on ADHD. Third, ADHD is related to SUDs, but this effect is not maintained after controlling for CD. Thus, our findings support a mediational model; namely, the association between ADHD and SUDs is mediated by CD.

Our study contributes to the literature, as it is the first investigation to longitudinally study a sample of adolescents, with and without ADHD, CD, and SUDs, followed to a later stage of development, namely, the thirties.\textsuperscript{7–10} In following our sample to a later stage of development than have prior investigators,\textsuperscript{7–10} we confirmed that the relationship between adolescent ADHD and SUDs is indirect at this developmental stage (i.e., adulthood). Our findings related to the mediational model are consistent with those of several researchers who have emphasized the importance of CD in predicting SUDs.\textsuperscript{7,15,24} For instance, Fergusson et al.\textsuperscript{15} have maintained that, after allowing for their high inter-correlation, attentional problems and
conduct problems have differential consequences for later development. Specifically, conduct problems are directly related to later substance abuse, while attentional problems are not.

Although ADHD and CD are separate dimensions, they are significantly correlated. Several factors may contribute to this association. Family and peer factors may mediate the relationship between ADHD and CD. More specifically, individuals with ADHD may have difficulty in relating to family members and may associate with deviant peers, which, in turn, may be related to the development of CD.

Our findings have important implications for the risk factors that predict SUDs. Given the symptom profile which characterizes CD, it is perhaps not surprising that adolescent CD is independently associated with the development of SUDs. That is, conduct disorder is typified by aggression (e.g., showing physical cruelty to people or using a dangerous weapon) and/or delinquent behavior (e.g., destroying property, lying, or stealing), both of which are reliable predictors of the development of SUDs. The association between aggressive and delinquent behavior on the one hand, and SUDs on the other, may also be accounted for by problem behavior theory, which posits that problem behaviors cluster within individuals. In the current study, it is particularly striking that the interval between the first baseline assessment and the final outcome assessment spanned a period of over twenty years. Therefore, the adverse effects of CD on the risk for SUDs are not temporally limited, but persist over a considerable length of time.

Several factors may serve to mediate the relationship between CD and SUDs. Adolescents with CD may seek out peers who are delinquent and use drugs. The peer group may then supply the individual with drugs or otherwise reinforce their drug use. Conduct disorder in adolescents may evoke difficulty in the mutual attachment relationship between parent and child. A distant parent-child mutual attachment relationship has been found to predict substance use.

We did not find an interactive effect of ADHD and CD on SUDs. Though there is some evidence that individuals with both ADHD and CD are at particular risk for poor outcomes, our findings suggest that this interaction may not extend to substance dependence. More research is needed, however, to investigate this issue.

**Limitations**

The study has several limitations. First, our sample is predominantly White. Our results, therefore, may not be generalizable to samples of ethnic minorities. Second, we are limited to self-report measures of substance abuse and dependence. Self-report measures of substance use, however, have generally been found to be reasonably accurate. Third, we did not address how ADHD and CD can be handled in school and clinical programs without the negative label assigned to adolescents with these disorders. Finally, we did not investigate the underlying mechanisms that may be responsible for the association between ADHD and CD. Several factors may contribute to their association. Shared genetic influences are probably a contributing factor. For instance, Nadder, Rutter, Silberg, Maes, and Eaves noted that twin studies suggest a common genetic component underlying both ADHD and CD. Perinatal complications, temperament, and impaired cognitive functioning have also been shown to be associated with both ADHD and CD. Future research should focus on these underlying mechanisms.

**Conclusions**

Our findings demonstrate that the presence of either adolescent ADHD, CD, or both are related to SUDs in one’s twenties and thirties. However, where CD has a direct effect in predicting the development of substance abuse and dependence by adulthood, the effect of ADHD is
mediated through CD. In sum, adolescents with ADHD remain at risk for developing SUDs, through the mediating effect of CD. For both ADHD and CD, either diagnosis indicates that the individual may be at risk to develop SUDs not only in the several years following diagnosis, but for a period lasting over twenty years. Since ADHD generally develops earlier than CD, ADHD may be an important diagnostic cue for later SUDs. At the same time, CD, because of its proximal influence on SUDs, serves as a major risk factor for SUDs. Given the clinical and public health importance of the progression of ADHD, CD, and SUDs, further research about the specific mechanisms explaining this developmental pattern is indicated. Finally, a focus on ADHD and CD where relevant should be included in therapeutic interventions.

Acknowledgments

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References


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Table 1
Separate logistic regression analyses predicting SUDs in the late twenties to thirties: Earlier adolescent ADHD (N=515).

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>SUDs in the late twenties to thirties</th>
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</thead>
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<tr>
<td></td>
<td>Coefficient (SE)</td>
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<tr>
<td>ADHD at T2 or T3</td>
<td>0.9 (0.3)</td>
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<tr>
<td>ADHD at T2 or T3 with control on CD at T2 or T3</td>
<td>0.5 (0.4)</td>
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<td>ADHD at T2 or T3 with control on demographic factors &amp; SUDs and MDD at T2</td>
<td>0.8 (0.4)</td>
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<td>ADHD at T2 or T3 with control on demographic factors &amp; SUDs and MDD at T2, and CD at T2 or T3</td>
<td>0.4 (0.4)</td>
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</table>

Note: 1. Demographic factors include gender, age, and educational level in the late thirties.
2. SE = standard error; OR=odds ratio; CI=confidence interval.
## Table 2
Separate logistic regression analyses predicting SUDs in the late twenties to thirties: Earlier adolescent CD (N=515).

<table>
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<th>Independent variables</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient (SE)</td>
<td>Wald Statistic</td>
<td>Adjusted OR (95% CI)</td>
<td>p value</td>
<td></td>
</tr>
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<td>CD at T2 or T3</td>
<td>1.3 (0.3)</td>
<td>15.9</td>
<td>3.5 (1.9–6.4)</td>
<td>&lt;0.001</td>
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<td>CD at T2 or T3 with control on ADHD at T2 or T3</td>
<td>1.1 (0.3)</td>
<td>9.7</td>
<td>2.9 (1.5–5.7)</td>
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<td>CD at T2 or T3 with control on demographic factors &amp; SUDs and MDD at T2</td>
<td>1.1 (0.3)</td>
<td>9.8</td>
<td>2.9 (1.5–5.6)</td>
<td>0.002</td>
<td></td>
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<tr>
<td>CD at T2 or T3 with control on demographic factors &amp; SUDs, and MDD at T2, and ADHD at T2 or T3</td>
<td>0.9 (0.4)</td>
<td>6.8</td>
<td>2.5 (1.3–5.1)</td>
<td>0.009</td>
<td></td>
</tr>
</tbody>
</table>

Note: 1. Demographic factors include gender, age, and educational level in the late thirties.

2. SE = standard error; OR=odds ratio; CI=confidence interval.