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Impulsive personality traits mediate the relationship between adult attention deficit hyperactivity symptoms and alcohol dependence severity

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Abstract

Background—While the role of attention deficit hyperactivity disorder (ADHD) as a risk factor for developing alcohol use disorder (AUD) has been established, the underlying pathways connecting the two are still not fully understood. Overlapping constructs such as impulsivity may explain the increased risk for developing AUD in individuals with ADHD.

Methods—In this study, we assessed whether adult ADHD symptoms increase the odds of having a diagnosis of AUD. Furthermore, we tested whether facets of impulsivity explained the relationship between ADHD symptoms and alcohol dependence (AD) severity.

Results—In a logistic regression of 749 participants (464 = AD, 285 = controls), overall adult ADHD symptoms, and more specifically, symptoms of hyperactivity/restlessness and problems with self-concept, increased the odds of having a diagnosis of AD. Within the AD sample, we found that impulsivity mediated the relationship between adult ADHD symptoms and AD severity. In particular, negative and positive urgency mediated the relationship of overall adult ADHD

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Contributors

LL was responsible for the concept of this study analysis. AMD, SAA, MLS and LL performed and/or assisted with the statistical analyses. All authors assisted with data analysis and interpretation of findings. AMD wrote the first draft of the manuscript. All authors critically reviewed content and approved the final version for publication.

symptoms, and symptoms of hyperactivity/restlessness and problems with self-concept with AD severity.

Conclusions—These results highlight the importance of looking at cohorts of ADHD symptoms and facets of impulsivity to assess the risk of developing AUD. They also suggest potential avenues for intervention strategies in individuals with pre-existing adult ADHD symptoms who are seeking treatment for AUD.

Keywords

alcohol use disorder; attention deficit hyperactivity disorder; impulsivity

Introduction

Alcohol use disorder (AUD) affects approximately 15.1 million adults in the U.S. (Substance Abuse and Mental Health Services Administration (SAMHSA), 2016). In 2010, alcohol-related costs were \$240 billion in the U.S. alone (Sacks, Gonzales, Bouchery, Tomedi, & Brewer, 2015). However, only about 1.3 million (8.3%) seek treatment for AUD, among which most individuals often have other psychiatric comorbidities. For example, about 23% of individuals seeking treatment for AUD have comorbid attention deficit hyperactive disorder (ADHD) (van Emmerik-van Oortmerssen et al., 2012). Individuals seeking treatment for AUD with comorbid ADHD have an earlier age of onset of dependence, greater likelihood to relapse, and increased social problems, severity of dependence, and number of hospital visits compared to individuals without a diagnosis of ADHD (Ercan, Co kunol, Varan, & Toksöz, 2003; Moura et al., 2013; Salama, Ibrahim, El Magd, & Kerim, 2015). In addition, the presence of ADHD symptoms, without a formal diagnosis, has been linked to increased risk for AUD (De Alwis et al., 2014). Thus, there is a critical need to further understand the relationship between AUD and ADHD.

While the role of ADHD as a risk factor for developing AUD is established, the underlying pathways connecting the two are still not fully understood. Overlapping constructs between ADHD and AUD may explain the connection between these two disorders. Several lines of evidence support the role of impulsivity in this regard. Impulsivity is a broad construct contained of multiple factors that lead to overall rash behavior (Lynam & Miller, 2004). First, impulsivity is present in the symptomology of both disorders. AUD can be characterized by an individual's inability to inhibit their need to drink despite negative consequences. Symptoms may also include unsuccessful attempts to cut down drinking and/or frequently drinking more than one intends to. Impulsivity in ADHD is characterized by the impairment of an individual's social and work life due to difficulties in waiting one's turn and failure to inhibit inappropriate responses.

Second, both ADHD and AUD share similar neurocircuitry and genetic variations related to increased impulsive behavior and personality traits. For example, reduced connectivity between the prefrontal cortex and striatum neural circuits has been associated with individual differences in impulsivity (Bjork, Smith, Chen, & Hommer, 2011; Diekhof et al., 2012). Individuals with ADHD show a reduction in white matter microstructure connectivity of prefrontal cortex and striatum (Liston, Cohen, Teslovich, Levenson, & Casey, 2011), and

this is particularly pertinent in individuals whose ADHD symptoms persist into adulthood (Shaw et al., 2015). Additionally, as compared to controls, reduced fronto-striatal connectivity has been described both in individuals with AUD (Park et al., 2010) and ADHD (for review see Liston et al., 2011). Hypoactivation of the dorsal lateral prefrontal cortex and the anterior cingulate cortex during behavioral inhibitory control tasks has been found in ADHD and AUD populations (Cortese et al., 2012; Luijten et al., 2014). Overall, these findings indicate both reduced connectivity and reduced activity in overlapping neurocircuits involved in impulsive personality and behavior. Moreover, genetic variations of the dopamine D4 receptor gene (*DRD4*) have been linked to impulsive personality, such that carriers of the 7 or greater repeat (*DRD4L*) show increased trait impulsivity (Jonas & Markon, 2014). Studies show a higher frequency of *DRD4L* carriers in individuals with ADHD (Wu, Xiao, Sun, Zou, & Zhu, 2012), AUD (Franke et al., 2000), and addictive-related behaviors (McGeary, 2009). Thus, there are biological components linked to impulsivity that are shared between ADHD and AUD.

Recent studies have investigated the role of impulsivity in the relationship between ADHD and problematic drinking behavior. In undergraduates, hyperactivity-impulsive and inattentive symptoms of ADHD were related to increases in weekly alcohol consumption (Roberts, Peters, Adams, Lynam, & Milich, 2014). However, after controlling for components of impulsivity (e.g., sensation seeking, lack of premeditation) this relationship was no longer significant, stipulating that impulsivity may contribute to the relationship of these two variables. Undergraduates with ADHD were more likely to experience negative consequences of alcohol compared to undergraduates without ADHD and this relationship was fully mediated by disinhibition (Rooney, Chronis-Tuscano, & Huggins, 2015). Another study found that within young adults diagnosed with ADHD in childhood, positive and negative urgency (the tendency to act out impulsively to positive and negative emotions) mediated the relationship between ADHD and alcohol related problems (Pedersen et al., 2016). Overall, these studies indicate that separate facets of impulsivity (e.g., disinhibition, negative urgency) may differentially contribute to the relationship between ADHD and AUD. However, these results are limited to either undergraduates or adults with a formal diagnosis of ADHD in childhood. Whether and how impulsivity may play a role in the relationship between ADHD symptoms and AUD within a sample of adult individuals diagnosed with AUD has not been investigated.

Therefore, the current study addresses two main questions. First, we aimed to validate past reports that show an association between ADHD and AUD. Specifically, this study evaluated whether the presence of adult ADHD symptoms increased the odds of an individual being diagnosed with AUD. Second, we evaluated whether separate facets of impulsivity may explain the relationship between ADHD symptoms and alcohol dependence severity. We hypothesized that having a larger number of adult ADHD symptoms would put an individual at greater odds for a diagnosis of AUD, and that the underlying pathways of impulsive behavior would account for the relationship between ADHD symptoms and AUD severity.

Methods

Participants

Participants initially considered for this analysis were 872 (35% female; 56% Caucasian) individuals who participated in Institutional Review Board (IRB)-approved screening protocols (98-AA-0009 and 05-AA-0121) of the National Institute on Alcohol Abuse and Alcoholism (NIAAA), conducted at the National Institutes of Health Clinical Center between 2008 and 2015. Participants fell into one of two categories: 526 individuals seeking treatment for current alcohol dependence (AD) and 346 controls with no lifetime history of AD. All participants provided written informed consent prior to participation. Participants seeking treatment for AD were admitted to an inpatient treatment program of approximately four weeks while controls were studied in an outpatient clinic. All assessments in the AD sample were done approximately one week after admission to prevent potential confounding responses from alcohol withdrawal.

Assessments

Alcohol Dependence and Other Psychological Disorders—The Structural Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002) was administered to participants to assess for the history of participants' psychological disorders. The SCID-I is a semi-structured interview to assess the presence of current and past alcohol and other substance abuse and dependence, depressive mood disorders, and anxiety disorders. Of note, we defined history of depressive disorder as any history of the following: a major depressive episode, major depressive disorder, or dysthymia as measured by the SCID-I. Since the DSM-IV was used, we refer to individual's diagnosis as AD, instead of the AUD label from the DSM-5.

Adult ADHD symptoms—The Conners' Adult ADHD Rating Scales–Self-Report Scale: Short Version (*CAARS-S:S*; Conners, Erhardt, & Sparrow, 1998) was used to assess adult ADHD-related symptoms. The CAARS-S:S was designed to act as a screening form for adult ADHD. It is a 26-item self-report measure and each item is rated on a 4-point Likert scale (0 – not at all true, to 3 – very much true). The CAARS-S:S has 4 factor-derived subscales each containing 5-items: inattention/memory problems ($\alpha = 0.80$), hyperactivity/restlessness ($\alpha = 0.78$), impulsivity/emotional lability ($\alpha = 0.79$), and problems with self-concept ($\alpha = 0.82$). The inattention/memory problems factor indexes high-order executive functioning. The hyperactivity/restlessness factor measures primarily cognitive restlessness, and to a lesser extent, hyper-mobility. The impulsivity/emotional lability factor refers to stress intolerances, hot temper, and verbal impulsivity, and the problems with self-concept factor measures anxiety, lack of self-confidence, and social isolation. The CAARS-S:S provides information on two indexes: the ADHD index and the inconsistency index. The ADHD index contains 12 questions, some of which are pulled from the 4 subscales above, and measures the most pertinent ADHD symptoms to distinguish clinical and non-clinical individuals. A score of ≥ 23 indicates probable adult ADHD (Dakwar et al., 2012). The inconsistency index compares answers within the assessment. An inconsistency index of ≥ 8 suggests that an individual's answers may be inconsistent. Therefore, we excluded individuals with an inconsistency index of ≥ 8 from analysis. This led to the removal of 61

individuals (Figure S1). Of note, the CAARS-S factor structure has been replicated in substance users seeking outpatient treatment (Cleland, Magura, Foote, Rosenblum, & Kosanke, 2006). Thus, the CAARS-S:S subscale factor structure is valid in both individuals affected by substance use disorder and those who are not.

Impulsivity—The Negative urgency, (lack of) Premeditation, (lack of) Perseverance, Sensation Seeking, and Positive Urgency Impulsivity Scale (UPPS-P; Lynam, Smith, Whiteside, & Cyders, 2006) was used to assess impulsivity. The UPPS-P assesses different pathways of impulsivity and is designed to consolidate and provide a more thorough evaluation of the numerous theories, operationalizations, and measures of impulsivity. The UPPS-P consists of 59 items rated on a 4-point scale (1 – agree strongly to 4 – disagree strongly) and is a reliable measure of five latent processes that predispose impulsive behavior (Whiteside & Lynam, 2001). Negative urgency ($\alpha = 0.94$) tests an individual's tendency to reactive impulsively to negative affect. Premeditation ($\alpha = 0.87$) measures an individual's tendency to act without contemplating the consequences of said action. Perseverance ($\alpha = 0.87$) measures an individual's ability to remain attentive to difficult or boring tasks. Sensation seeking ($\alpha = 0.88$) evaluates an individual's openness and tendency to seek out positive or high reward experiences, regardless of possible dangers. Positive urgency ($\alpha = 0.96$) was later added to the original UPPS impulsivity scale to measure an individual's aptitude to act out rashly in response to positive mood (Cyders et al., 2007).

Alcohol Dependence Severity—The Alcohol Dependence Scale (ADS; Skinner & Allen, 1982) was used to assess alcohol dependence severity. The ADS is a 25-item self-report questionnaire, with a range of total score from 0 to 47. A score of 0 indicates no evidence of AD reported, a score of 1–13 indicates a low level of AD, a score of 14–21 indicates an intermediate level of AD, a score of 22–30 indicates a substantial level of AD, and finally a score of 31–47 indicates a severe level of AD.

Family history of alcohol dependence—We assessed family history of problematic drinking using the Family Tree Questionnaire (FTQ; Mann, Sobell, Sobell, & Pavan, 1985), because it has been shown to be a strong predictor of AD (e.g., Schuckit & Smith, 2000). The FTQ queries individuals about full-blood, first-degree (i.e., siblings and parents) and second-degree relatives (i.e., grandparents). Individuals were asked not to include step-relatives, half-siblings, or individuals that were adopted. Relatives were then rated on 4 categories: 1) abstainer 2) social drinker 3) possible problematic drinker, and 4) definite problem drinker. Individuals also had the opportunity to rate a relative as “unknown drinking status”. Definite problem drinkers include relatives that have received treatment for their drinking or have experienced several negative consequences for their drinking behavior. From this data, a family history density (FHD; Stoltenberg, Mudd, Blow, & Hill, 1998) was calculated. FHD weights each family member identified as a definite problematic drinker based on how closely they are related to the participant (i.e., parent/sibling = 0.5, grandparent = 0.25). Relatives were scored zero if they were identified as abstainers, social drinkers, possible problematic drinkers, or had an unknown drinking status.

Statistical Analysis

Case Control Logistic Regression: We tested whether presence of self-reported adult ADHD symptoms predicted the probability of an individual being diagnosed with AD. Two logistic regressions were run. The first tested the CAARS:S-S ADHD index as a predictor of AD, while the second tested the four subscales of the CAARS:S-S as predictors of AD. In each model we also included the following variables known to impact the probability of having a diagnosis of AD: lifetime history of depressive disorder (yes/no), lifetime history of anxiety disorder (yes/no), lifetime history of other substance dependence (yes/no), FHD, age, years of education, gender (female versus male), and race (Caucasian versus African-American and other racial minorities). Sixty-two individuals were excluded from the analysis due to missing data, leaving a total of 749 (35% female; 57% Caucasian) participants included in the analysis (Figure S1). Additional information on checks for statistical assumptions and coding of dichotomous variables can be found in the supplementary materials. A Bonferroni multiple comparisons correction was made such that to be considered statistically significant an alpha level of 0.025 must have been met.

Multiple Mediation: Next, we tested the hypothesis that impulsivity mediates the relationship of adult ADHD symptoms (via CAARS:S-S) and severity of AD (via ADS). We specifically examined whether different facets of impulsivity (e.g., sensation seeking, negative urgency) mediate the relationship of ADHD symptoms and AD severity. Further, we tested whether individual facets of impulsivity accounted for more mediation than others. These analyses only included individuals diagnosed with AD. Of the 464 AD individuals, 135 were excluded because UPPS-P was missing, as it was not part of the NIAAA screening protocol until 2010 (Figure S1). This left a total of 329 participants (31.9% females; 51.7% Caucasians) included in the analysis. First, we tested whether impulsivity (via UPPS-P subscales) mediated the relationship of the ADHD index and ADS scores. We then subsequently tested whether impulsivity mediated the relationship of CAARS:S-S subscales and ADS scores. In order to test the independent effects of each subscale, we added the remaining subscales as covariates in the mediation model. For example, when testing the problems with self-concept subscale's relationship with the ADS, the remaining three subscales of the CAARS:S-S were treated as covariates. In addition, all mediation analyses controlled for gender, race, lifetime history of another substance dependence, anxiety disorder or depression, age, years of education, and FHD.

Simple step multiple mediation analysis was conducted using PROCESS macro (Hayes, 2013) for SPSS. Unstandardized indirect effects were computed for each of the mediators using bootstrapping with 5,000 iterations. Bootstrapping has been found to be a more valid and powerful test for estimating indirect effects (Mackinnon et al., 2004; Williams & Mackinnon, 2008), and does not rely on the assumption of normality. Although, it was previously suggested that there must be a significant relationship of the independent variable (X) on the dependent variable (Y) for mediation to occur (Baron & Kenny, 1986), it has since been argued that mediation can occur without the total effect of X on Y (e.g., Hayes, 2009; Rucker, Preacher, Tormala, & Petty, 2011). Therefore, mediation was established if one of the following three conditions occurred: 1) *Indirect effects:* 95% bias-corrected confidence intervals (CI) for indirect effects of mediating variables did not contain zero, 2)

Full mediation: there was a significant relationship of X on Y (total effect), but when mediating variables were added to the model the effect of X on Y lost significance (direct effect), or 3) *Partial mediation:* there was a significant total and direct effect of X on Y, however the direct effect was mitigated with the addition of mediation variables.

Results

Case-control

Table 1 and 2 contain descriptive of cases versus controls and correlations of variables of interest. The first logistic regression tested whether overall adult ADHD symptoms increased the probability of having a diagnosis of AD (Table 3). In this analysis, it was found that an increased ADHD index (Odds Ratio [95% CI]): (1.293 [1.183, 1.414], $p < 0.001$), higher FHD (548.835 [22.079, 13642.565], $p < 0.001$), and being older (1.156 [1.117, 1.196], $p < 0.001$) increased odds of an individual having a diagnosis of AD. Having no lifetime history of substance dependence (0.011 [0.004, 0.034], $p < 0.001$), no lifetime history of any anxiety disorder (0.042 [0.013, 0.129], $p < 0.001$), more years of education (0.862 [0.760, 0.979], $p = 0.022$) and being female (0.336 [0.148, 0.765], $p = 0.009$) decreased the odds of an individual having a diagnosis of AD. Finally, race did not predict the odds of an individual having a diagnosis of AD.

In the second logistic regression, we tested whether the individual subscales of the CAARS:S-S increased the probability of having a diagnosis of AD (Table 4). This analysis revealed that increased scores on the hyperactivity/restlessness (1.291 [1.091, 1.528], $p = 0.003$) and problems with self-concept (1.562 [1.328, 1.838], $p < 0.001$) subscales increased the odds of an individual having a diagnosis of AD. Increased scores on the inattention/memory problems subscale (0.772 [0.627, 0.951], $p = 0.015$) reduced the odds of an individual having a diagnosis of AD. Also, similar to the first logistic regression, a higher family history density (548.835 [22.079, 13642.565], $p < 0.001$) and being older (1.156 [1.117, 1.196], $p < 0.001$) increased the odds of having a diagnosis of AD, while no lifetime history of other substance dependence (0.007 [0.002, 0.024], $p < 0.001$), no lifetime history of anxiety disorder (0.051 [0.016, 0.162], $p < 0.001$), and being female (0.269 [0.111, 0.654], $p = 0.009$) decreased the odds of having a diagnosis of AD. In this model, years of education and race did not predict the odds of having a diagnosis of AD.

Multiple Mediation

Table 5 reports all indirect effects of the individual mediators on the relationship of adult ADHD symptoms to AD severity.

ADHD Index—In the first analysis, we investigated whether impulsivity mediated the relationship of the number and severity of self-reported ADHD adult symptoms on AD severity. There was a significant total effect of ADHD Index on ADS score ($\beta = 0.395$, 95% CI [0.272, 0.518], $p < 0.0001$) and a significant direct effect of ADHD Index on ADS score ($\beta = 0.159$, 95% CI [0.005, 0.318], $p < 0.049$). This indicates that impulsivity partially mediated the effect of the ADHD Index on ADS score (Figure 1). There was a total indirect effect (indirect effect of all mediators as a group; $\beta = 0.236$, 95% CI [0.122, 0.357]) and

indirect effects of both the negative (parameter estimates = 0.164, 95% CI [0.06, 0.27]) and positive (parameter estimates = 0.073, 95% CI [0.006, 0.157]) urgency subscales. When comparing indirect effects between mediators, negative urgency accounted for greater mediation compared to the (lack of) premeditation (indirect effect contrast = 0.163 [0.044, 0.306]), (lack of) perseverance (indirect effect contrast = 0.159 [0.036, 0.279]), and sensation seeking (indirect effect contrast = 0.1701 [0.067, 0.277]), whereas sensation seeking was a worse mediator than positive urgency (indirect effect contrast = -0.079 [-0.164, -0.007]).

Subscales of CAARS:S-S—In the remaining analysis, we tested whether impulsivity mediated the relationship of different groups of ADHD symptoms (via subscales of the CAARS:S-S) on AD severity. There was no total effect ($\beta = 0.019$, 95% CI [-0.299, 0.338], $p = 0.904$) of the inattention/memory problems subscale on ADS score, nor was there a direct effect ($\beta = -0.057$, 95% CI [-0.405, 0.292], $p = 0.7496$). There were also no total indirect or individual indirect effects found. Thus, pathways to impulsive behavior did not mediate the relationship of inattention/memory problem adult ADHD symptoms on AD severity.

There was a no total effect ($\beta = 0.258$, 95% CI [-0.024, 0.539], $p = 0.072$) of hyperactivity/restlessness adult ADHD symptoms on AD severity, and no direct effect ($\beta = 0.139$, 95% CI [-0.147, 0.424], $p = 0.340$). There was also no total indirect effect (parameter estimate 0.119, 95% CI [-0.007, 0.251]). However, there were indirect effects found for negative (parameter estimates = 0.083, 95% CI [0.019, 0.194]) and positive (parameter estimates = 0.063, 95% CI [0.012, 0.156]) urgency subscales. Similar to the results of the ADHD index, negative urgency accounted for greater mediation compared to (lack of) premeditation (indirect effect contrast = 0.082, 95% CI [0.016, 0.197]), (lack of) perseverance (indirect effect contrast = 0.082, 95% CI [0.012, 0.192]), and sensation seeking (indirect effect contrast = 0.110, 95% CI [0.002, 0.232]) subscales of the UPPS-P. Unlike the ADHD index, (lack of) premeditation (indirect effect contrast = -0.062, 95% CI [-0.156, -0.003]) and (lack of) perseverance (indirect effect contrast = -0.063, 95% CI [-0.158, -0.004]) exerted less of an effect than positive urgency in mediation of hyperactivity/restlessness ADHD symptoms on AD severity. Thus, mediation was met for both negative and positive urgency.

There was no total effect ($\beta = 0.235$, 95% CI [-0.089, 0.579], $p = 0.150$) or direct effect ($\beta = -0.025$, 95% CI [-0.374, 0.324], $p = 0.8873$) of emotional lability/impulsivity ADHD symptoms on AD severity. There was a total indirect effect (parameter estimate = 0.270, 95% CI [0.116, 0.450]) and an indirect effect of negative urgency (parameter estimate = 0.214, 95% CI [0.077, 0.364]). There were no indirect effects found for (lack of) premeditation, (lack of) perseverance, sensation seeking or positive urgency. Negative urgency accounted for greater mediation compared to (lack of) premeditation (indirect effect contrast = 0.201, 95% CI [0.077, 0.364]) and (lack of) perseverance (indirect effect contrast = 0.214, 95% CI [0.073, 0.363]). Thus, mediation was met for impulsivity, as a whole, as well as negative urgency.

A significant total effect ($\beta = 0.450$, 95% CI [0.224, 0.676], $p < 0.001$) and significant direct effect ($\beta = 0.315$, 95% CI [0.080, 0.550], $p = 0.009$) of problems with self-concept adult

ADHD symptoms on AD severity was found. Thus, partial mediation of pathways of impulsive behavior on the relationship of problems with self-concept ADHD symptoms on AD severity was established (Figure 2). There were also a total indirect effect (parameter estimate = 0.135, 95% CI [0.025, 0.264]) and indirect effects of negative (parameter estimate = 0.080, 95% CI [0.022, 0.168]) and positive (parameter estimate = 0.036, 95% CI [0.002, 0.093]) urgency on the relationship of adult ADHD problems with self-concept symptoms to AD severity. More specifically, it was found that negative urgency accounted for greater mediation than (lack of) premeditation (indirect effect contrast = 0.083, 95% CI [0.019, 0.167]) and (lack of) perseverance (indirect effect contrast = 0.079, 95% CI [0.009, 0.175]). Positive urgency, however, did not better account for mediation compared to other components of impulsivity.

Discussion

The results from this study are two-fold: 1) the presence of overall ADHD symptoms, and more specifically the presence of hyperactivity/restless and problems with self-concept symptoms, increase an individual's odds of having a diagnosis of AD, and 2) general and individual facets of impulsivity (i.e., negative and positive urgency) uniquely contribute to the relationship between adult ADHD symptoms and AD severity. This study extends prior research by showing that this relationship is true regardless of formal ADHD diagnosis within a sample of adults seeking treatment for AD. It also highlights the role of impulsivity in increasing the odds of having more severe AD if there are pre-existing ADHD symptoms.

Of note, the three predictors found to increase the odds of having a diagnosis of AD (i.e., ADHD index, hyperactivity/restlessness, and problems with self-concept), were also found to be mediated by negative and positive urgency when looking at AD severity. That is, one's tendency to rashly act on negative or positive emotions mediated the relationship between overall ADHD symptoms and both hyperactivity/restlessness, as well as problems with self-concept symptom categories and AD severity. These results are consistent with prior work that found positive and negative urgency mediated the relationship between a childhood diagnosis of ADHD and alcohol-related problems (Pedersen et al., 2016). Together, these studies indicate that impulsivity mediates the relationship between ADHD and AUD, regardless of whether symptomatic behaviors or diagnostic cut offs are considered. These results also underscore the need to further understand the role of emotional dysregulation in individuals with comorbid ADHD and AUD symptoms.

Interestingly, adult ADHD symptoms of impulsivity/emotional lability did not predict the likelihood of having AD. Furthermore negative urgency and, to a lesser extent, positive urgency indirectly affected the relationship between these ADHD symptoms and AD severity. These results denote that general symptoms related to emotional dysregulation and impulsivity are not by themselves exacerbating the probability of having more severe AD. Instead, it is the combination of pre-existing adult ADHD symptoms and the tendency to act out impulsively, particularly in response to emotions, that intensifies the likelihood to develop problematic patterns of alcohol-seeking behaviors.

While speculative, individuals exhibiting ADHD symptoms may adopt maladaptive emotional regulation strategies, which can increase the likelihood to react impulsively to high valence states. One mechanism to regulate these emotions may be drinking to either cope with negative emotions or to enhance positive emotions. Thus, individuals with ADHD symptoms may experience emotion dysregulation, and have increased likelihood to respond to high valence impulsively, which in turn may result in excessive alcohol use in order to regulate their emotions. To some extent, there is support for this conceptual framework in the current literature. Individuals with ADHD are prone to emotional dysregulation. It has been suggested that this may be a core feature of adult ADHD, and is often among the main reasons why adult individuals with ADHD seek treatment (Retz, Stieglitz, Corbisiero, Retz-Junginger, & Rösler, 2012). Moreover, individuals with ADHD have increased negative urgency compared to controls (Lopez, Dauvilliers, Jaussent, Billieux, & Bayard, 2015). Although drinking motives in individuals with ADHD symptoms have not been investigated to our knowledge, research indicates that the most common drinking motives are to enhance or cope with current emotional states (Hammarberg, Öster, & Nehlin, 2017). Additionally, qualitative research on alcohol expectancies has found that adults with ADHD are more likely to drink to enhance current positive emotions or mitigate negative emotions (Nehlin, Nyberg, & Öster, 2015). Future research should further probe this line of work.

In addition to maladaptive emotion regulation strategies, individuals with ADHD symptoms may drink in order to improve their problems with self-concept. Our results show that problems with self-concept symptoms predicted AD and was directly related to AD severity. In a qualitative study, adults with ADHD reported that drinking alcohol increased their feeling of belonging, made them perceive themselves as more normal, and improved their social interactions (Nehlin et al., 2015). In another study, it was found that ADHD symptoms moderated the relationship of positive alcohol expectancies (e.g., alcohol will make it easier to talk to people and make one feel calm) and alcohol related problems in undergraduates (Dattilo, Murphy, Van Eck, & Flory, 2013). Specifically, individuals with more ADHD symptoms and more positive alcohol expectancies had increased alcohol related problems. Our results, also suggest that this relationship was mediated by impulsive personality traits as a whole, and positive and negative urgency. Individuals with more problems with self-concept symptoms may initially start drinking to feel more “normal” and then continue to drink due to increase positive emotions associated with belonging or reduce strong negative emotions of social rejection

Limitations

Important study limitations need to be kept in mind. First, while mediation analysis is designed to test for causation, the cross-sectional nature of this study impedes one from concluding that impulsivity is a causal pathway of the relationship between ADHD symptoms and AUD. To elucidate whether impulsivity is a true causal mechanism, future research should test whether mood induction in individuals high and low on ADHD symptoms affects alcohol self-administration. We also did not assess formal diagnosis of ADHD or the onset of ADHD symptoms. Due to the cross-sectional nature of the study and absence of this data, we cannot conclusively delineate the temporal relationship between ADHD, impulsivity, and AUD severity. The developmental trajectories of these two

disorders suggest that ADHD would be an antecedent to AUD, and impulsive personality traits are one of the many risk factors for the development of each disorder (American Psychiatric Association, 2013; Verdejo-García, Lawrence, & Clark, 2008). However, some evidence suggests that chronic alcohol use may increase ADHD symptoms (Karam et al., 2017) and further may increase impulsive personality traits (Quinn, Stappenbeck, & Fromme, 2011). Moreover, assessment of ADHD symptoms approximately one week after alcohol withdraw removes acute withdrawal's potential confounding effects, but not the effects associated with long-term abstinence. The relationships found may change if ADHD symptoms were assessed later in an individual's recovery.

Second, the generalizability of these results is limited to individuals seeking treatment for AUD. We also did not assess conduct disorder (CD), antisocial personality disorder (ASPD), or nicotine dependence symptoms that are often comorbid with ADHD (Korsgaard, Torgersen, Wentzel-Larsen, & Ulberg, 2016). These results may be reduced if CD, ASPD, or nicotine dependence symptoms are tested within the same model. Thus, future research should look at the mediating relationship between impulsivity factors and individuals exhibiting ADHD, symptoms as well as CD, ASPD, or nicotine dependence symptoms, to further expand the generalizability of these results. Furthermore, subgroup analyses were beyond the scope of the current study. Future research, should investigate how the relationships found in this study are influenced by race and gender.

One strength of this study is that it supports the notion that impulsivity may be a link contributing to individuals with ADHD's heightened risk in developing AUD. However, this study did not investigate the exact neurobiological mechanisms that underlie these reciprocal phenotypic relationships. Behavioral control inhibition (a behavioral subcomponent of impulsivity) may be one line of research to investigate in order to understand these mechanisms. A meta-analysis examining the relationship between negative urgency and different impulsive behavior tasks found that negative urgency was only significantly related to response inhibition (Cyders & Coskunpinar, 2011). Functional brain imaging studies on response inhibition tasks have found hypoactivation of the dorsal lateral prefrontal cortex and anterior cingulate cortex in both disorders (Cortese et al., 2012; Luijten et al., 2014). It is unclear whether this hypoactivation is worsened when individuals have comorbid ADHD and AUD. Future studies, may want to investigate the neurocircuitry of comorbid ADHD and AUD within behavioral inhibition tasks, and positive and negative urgency.

Clinical Implications

Despite the existing limitations, the current study provides fruitful avenues for intervention research. If experimental and longitudinal studies replicate these results, pharmacological and/or behavioral interventions that target emotion regulation and prosocial coping skills to these emotions may be such avenues to create more efficacious treatment for individuals with ADHD symptoms seeking treatment for AUD. In particular, therapies that teach one how to recognize emotions, triggers to those emotions, and how to regulate strong emotions may help reduce ADHD symptoms, positive and negative urgency, and harmful use of alcohol. One such therapy is dialectical behavior therapy (DBT). DBT is a comprehensive therapy that focuses on teaching individuals how to regulate emotions, maintain attention,

and navigate interpersonal skills. DBT thus may help reduce one's positive and negative urgency, pre-existing ADHD symptoms (e.g., inattention), and increase problems with self-concept via improving interpersonal skills. Targeting all three of these factors may then increase the likelihood of remission for AUD.

Conclusion

Overall, this study suggests that the subcomponents of impulsivity to react rashly to negative and positive emotions (negative and positive urgency, respectively) contribute to the relationship between ADHD symptoms and AUD severity. It further implies that negative and positive urgency contribute more to the relationship between ADHD symptoms and AUD severity than sensation seeking, lack of premeditation and lack of perseverance.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, (DSM-5). 5. Washington, DC: American Psychiatric Association; 2013.
- Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*. 1986; 51(6):1173. [PubMed: 3806354]
- Bjork JM, Smith AR, Chen G, Hommer DW. Psychosocial problems and recruitment of incentive neurocircuitry: exploring individual differences in healthy adolescents. *Developmental Cognitive Neuroscience*. 2011; 1(4):570–577. [PubMed: 21927631]
- Cleland C, Magura S, Foote J, Rosenblum A, Kosanke N. Factor structure of the Conners Adult ADHD Rating Scale (CAARS) for substance users. *Addictive Behaviors*. 2006; 31(7):1277–1282. [PubMed: 16169157]
- Conners, C., Erhardt, D., Sparrow, E. Conners adult attention rating scale—self-report: Long version. North Tonawanda, NY: Multi-Health Systems; 1998.
- Cortese S, Kelly C, Chabernaud C, Proal E, Di Martino A, Milham MP, Castellanos FX. Toward systems neuroscience of ADHD: a meta-analysis of 55 fMRI studies. *American Journal of Psychiatry*. 2012; 169(10):1038–1055. [PubMed: 22983386]
- Cyders MA, Coskunpinar A. Measurement of constructs using self-report and behavioral lab tasks: Is there overlap in nomothetic span and construct representation for impulsivity? *Clinical psychology review*. 2011; 31(6):965–982. [PubMed: 21733491]

- Cyders MA, Smith GT, Spillane NS, Fischer S, Annus AM, Peterson C. Integration of impulsivity and positive mood to predict risky behavior: Development and validation of a measure of positive urgency. *Psychological Assessment*. 2007; 19(1):107. [PubMed: 17371126]
- Dakwar E, Mahony A, Pavlicova M, Glass A, Brooks D, Mariani JJ, Levin FR. The utility of attention-deficit/hyperactivity disorder screening instruments in individuals seeking treatment for substance use disorders. *J Clin Psychiatry*. 2012; 73(11):e1372–1378. [PubMed: 23218166]
- Dattilo L, Murphy KG, Van Eck K, Flory K. Do ADHD symptoms moderate the relation between positive alcohol expectancies and alcohol-related outcomes? *ADHD Attention Deficit and Hyperactivity Disorders*. 2013; 5(2):93–104. [PubMed: 23229745]
- De Alwis D, Agrawal A, Reiersen AM, Constantino JN, Henders A, Martin NG, Lynskey MT. ADHD symptoms, autistic traits, and substance use and misuse in adult Australian twins. *Journal of studies on alcohol and drugs*. 2014; 75(2):211–221. [PubMed: 24650814]
- Diekhof EK, Nerenberg L, Falkai P, Dechent P, Baudewig J, Gruber O. Impulsive personality and the ability to resist immediate reward: An fMRI study examining interindividual differences in the neural mechanisms underlying self-control. *Human Brain Mapping*. 2012; 33(12):2768–2784. [PubMed: 21938756]
- Ercan ES, Co kunol H, Varan A, Toksöz K. Childhood attention deficit/hyperactivity disorder and alcohol dependence: a 1-year follow-up. *Alcohol and Alcoholism*. 2003; 38(4):352–356. [PubMed: 12814903]
- First, M., Spitzer, R., Gibbon, M., Williams, JB. Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition. (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute; 2002.
- Franke P, Wang T, Möthen MM, Knapp M, Neith H, Lichtermann D, Maier W. Susceptibility for alcoholism: DRD4 exon III polymorphism: a case-control and a family-based association approach. *Addiction biology*. 2000; 5(3):289–295. [PubMed: 20575843]
- Hammarberg A, Öster C, Nehlin C. Drinking motives of adult patients seeking treatment for problematic alcohol use. *Journal of Addictive Diseases*. 2017; 36(2):127–135. [PubMed: 28166486]
- Hayes AF. Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication monographs*. 2009; 76(4):408–420.
- Hayes, AF. Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. Guilford Press; 2013.
- Jonas KG, Markon KE. A meta-analytic evaluation of the endophenotype hypothesis: Effects of measurement paradigm in the psychiatric genetics of impulsivity. *Journal of Abnormal Psychology*. 2014; 123(3):660–675. [PubMed: 24978691]
- Karam R, Rovaris D, Breda V, Picon F, Victor M, Salgado C, Meller M. Trajectories of attention-deficit/hyperactivity disorder dimensions in adults. *Acta Psychiatrica Scandinavica*. 2017
- Korsgaard HO, Torgersen S, Wentzel-Larsen T, Ulberg R. Personality disorders and Axis I comorbidity in adolescent outpatients with ADHD. *BMC Psychiatry*. 2016; 16(1):175. [PubMed: 27245754]
- Liston C, Cohen MM, Teslovich T, Levenson D, Casey B. Atypical prefrontal connectivity in attention-deficit/hyperactivity disorder: pathway to disease or pathological end point? *Biological Psychiatry*. 2011; 69(12):1168–1177. [PubMed: 21546000]
- Lopez R, Dauvilliers Y, Jaussent I, Billieux J, Bayard S. A multidimensional approach of impulsivity in adult attention deficit hyperactivity disorder. *Psychiatry Research*. 2015; 227(2):290–295. [PubMed: 25895489]
- Luijten M, Machielsen MW, Veltman DJ, Hester R, de Haan L, Franken IH. Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *Journal of psychiatry & neuroscience: JPN*. 2014; 39(3): 149. [PubMed: 24359877]
- Lynam DR, Miller JD. Personality pathways to impulsive behavior and their relations to deviance: Results from three samples. *Journal of Quantitative Criminology*. 2004; 20(4):319–341.
- Lynam, DR., Smith, GT., Whiteside, SP., Cyders, MA. The UPPS-P: Assessing five personality pathways to impulsive behavior. West Lafayette, IN: Purdue University; 2006.

- Mann RE, Sobell LC, Sobell MB, Pavan D. Reliability of a family tree questionnaire for assessing family history of alcohol problems. *Drug and Alcohol Dependence*. 1985; 15(1–2):61–67. [PubMed: 4017879]
- McGeary J. The DRD4 exon 3 VNTR polymorphism and addiction-related phenotypes: a review. *Pharmacology Biochemistry and Behavior*. 2009; 93(3):222–229.
- Moura HF, Faller S, Benzano D, Szobot C, Von Diemen L, Stolf AR, Pechansky F. The effects of ADHD in adult substance abusers. *Journal of Addictive Diseases*. 2013; 32(3):252–262. [PubMed: 24074191]
- Nehlin C, Nyberg F, Öster C. The patient's perspective on the link between ADHD and substance use: a qualitative interview study. *J Atten Disord*. 2015; 19(4):343–350. [PubMed: 25359762]
- Park SQ, Kahnt T, Beck A, Cohen MX, Dolan RJ, Wrase J, Heinz A. Prefrontal cortex fails to learn from reward prediction errors in alcohol dependence. *Journal of Neuroscience*. 2010; 30(22):7749–7753. [PubMed: 20519550]
- Pedersen SL, Walther CA, Harty SC, Gnagy EM, Pelham WE, Molina BS. The indirect effects of childhood attention deficit hyperactivity disorder on alcohol problems in adulthood through unique facets of impulsivity. *Addiction*. 2016; 111(9):1582–1589. [PubMed: 26999438]
- Quinn PD, Stappenbeck CA, Fromme K. Collegiate heavy drinking prospectively predicts change in sensation seeking and impulsivity. *Journal of Abnormal Psychology*. 2011; 120(3):543. [PubMed: 21443288]
- Retz W, Stieglitz R-D, Corbisiero S, Retz-Junginger P, Rösler M. Emotional dysregulation in adult ADHD: what is the empirical evidence? *Expert Review of Neurotherapeutics*. 2012; 12(10):1241–1251. [PubMed: 23082740]
- Roberts W, Peters JR, Adams ZW, Lynam DR, Milich R. Identifying the facets of impulsivity that explain the relation between ADHD symptoms and substance use in a nonclinical sample. *Addictive Behaviors*. 2014; 39(8):1272–1277. [PubMed: 24813555]
- Rooney M, Chronis-Tuscano AM, Huggins S. Disinhibition mediates the relationship between ADHD and problematic alcohol use in college students. *J Atten Disord*. 2015; 19(4):313–327. [PubMed: 23117860]
- Rucker DD, Preacher KJ, Tormala ZL, Petty RE. Mediation analysis in social psychology: Current practices and new recommendations. *Social and Personality Psychology Compass*. 2011; 5(6):359–371.
- Sacks JJ, Gonzales KR, Bouchery EE, Tomedi LE, Brewer RD. 2010 national and state costs of excessive alcohol consumption. *American Journal of Preventive Medicine*. 2015; 49(5):e73–e79. [PubMed: 26477807]
- Salama H, Ibrahim S, El Magd OA, Kerim AA. The impact of attention-deficit hyperactivity disorder across the lifespan on substance use disorders. *Egyptian Journal of Psychiatry*. 2015; 36(2):66.
- Schuckit MA, Smith TL. The relationships of a family history of alcohol dependence, a low level of response to alcohol and six domains of life functioning to the development of alcohol use disorders. *Journal of Studies on Alcohol*. 2000; 61(6):827–835. [PubMed: 11188488]
- Shaw P, Sudre G, Wharton A, Weingart D, Sharp W, Sarlls J. White matter microstructure and the variable adult outcome of childhood attention deficit hyperactivity disorder. *Neuropsychopharmacology*. 2015; 40(3):746–754. [PubMed: 25241803]
- Simon, L. [Accessed March 26, 2017] Detecting multicollinearity using variance inflation factors. Available from: http://online.stat.psu.edu/online/development/stat501/12multicollinearity/05multico_vif.html
- Skinner HA, Allen BA. Alcohol dependence syndrome: measurement and validation. *Journal of Abnormal Psychology*. 1982; 91(3):199–209. [PubMed: 7096790]
- Stoltenberg SF, Mudd SA, Blow FC, Hill EM. Evaluating measures of family history of alcoholism: density versus dichotomy. *Addiction*. 1998; 93(10):1511–1520. [PubMed: 9926555]
- Substance Abuse and Mental Health Services Administration (SAMHSA). Results from the 2015 National Survey on Drug Use and Health: Detailed Tables. Table 5.6 A Substance Use Disorder in Past Year among Persons Aged 18 or Older, by Demographic Characteristics: Numbers in Thousands, 2014 and 2015. 2016. Retrieved from www.samhsa.gov/data/sites/default/files/NSDUH-DetTabs-2015/NSDUH-DetTabs-2015/NSDUH-DetTabs-2015.htm#tab5-6a

- van Emmerik-van Oortmerssen K, van de Glind G, van den Brink W, Smit F, Crunelle CL, Swets M, Schoevers RA. Prevalence of attention-deficit hyperactivity disorder in substance use disorder patients: a meta-analysis and meta-regression analysis. *Drug and Alcohol Dependence*. 2012; 122(1):11–19. [PubMed: 22209385]
- Verdejo-García A, Lawrence AJ, Clark L. Impulsivity as a vulnerability marker for substance-use disorders: review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience & Biobehavioral Reviews*. 2008; 32(4):777–810. [PubMed: 18295884]
- Whiteside SP, Lynam DR. The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*. 2001; 30(4):669–689.
- Wu J, Xiao H, Sun H, Zou L, Zhu L-Q. Role of dopamine receptors in ADHD: a systematic meta-analysis. *Molecular neurobiology*. 2012; 45(3):605–620. [PubMed: 22610946]

Figure 1.

Standardized regression coefficients from bootstrapping are provided along each path. A) Total effect of ADHD adult symptoms on AD severity (without mediators), $p < 0.0001$, $R^2 = 0.276$. B) Single-step multiple mediation model of ADHD adult symptoms on AD severity via pathways to impulsive behavior, $p < 0.0001$, $R^2 = 0.338$. Total effect of all mediators combined = 0.235 (95% CI [0.121, 0.356]). * $p < 0.05$, ** $p < 0.001$

Figure 2.

Standardized regression coefficients (standard error) from bootstrapping are provided along each path. A) Total effect of problems with self-concept on AD severity (without mediators), $p < 0.0001$, $R^2 = 0.276$. B) Single-step multiple mediation model of problems with self-concept on AD severity via pathways to impulsive behavior, $p < 0.0001$, $R^2 = 0.350$. Total effect of all mediators combined = 0.135 (95% CI [0.025, 0.254]). * $p < 0.05$, ** $p < 0.001$.

Table 1

Descriptive characteristics of treatment-seeking AD individuals and controls

Variable	Controls (n = 285)	AD Participants (n = 464)	Cohen's <i>d</i> (95% CIs)
CAARS-S:S			
ADHD Index, Mean (SD)	5.88 (3.77)	12.28 (3.77)	-1.7 (-1.97, -1.43)
Inattention/Memory Problems, Mean (SD)	2.81 (2.33)	4.79 (3.15)	-0.69 (-0.89, -0.48)
Hyperactivity/Restlessness, Mean (SD)	4.40 (2.55)	6.90 (3.34)	-0.78 (-1.00, -0.56)
Impulsivity/Emotional Liability, Mean (SD)	1.53 (1.32)	3.72 (2.75)	-0.94 (-1.11, -0.78)
Problems with Self-Concept, Mean (SD)	2.15 (2.48)	6.73 (4.198)	-1.26 (-1.52, -1.00)
Family History Density, Mean (SD)	0.03 (0.07)	0.17 (0.18)	-0.94 (-0.95, -0.93)
Age, Mean (SD)	29.65 (8.61)	42.95 (10.17)	-1.38 (-2.07, -0.69)
Years of Education, Mean (SD)	16.20 (2.78)	13.82 (2.79)	0.85 (0.65, 1.05)
No History of Depressive Disorder	267 (94%)	353 (76%)	0.46 (0.32, 0.61)
No History of Anxiety Disorder	278 (98%)	260 (56%)	1.00 (0.84, 1.16)
No History of Other Substance Use Disorders	280 (98%)	139 (30%)	1.79 (1.60, 1.99)
Female	122 (45%)	142 (31%)	0.25 (0.11, 0.39)
Male	163 (55%)	322 (69%)	-0.25 (-0.39, -0.11)
Caucasian	173 (61%)	260 (56%)	0.09 (-0.05, 0.24)
Racial Minority [±]	112 (39%)	204 (44%)	-0.09 (-0.24, 0.05)

Note: CAARS-S:S = Conners' Adult ADHD Rating Scales–Self-Report Scale: Short Version; CI = confidence interval; SD = Standard Deviation

Small, medium, and large effects were considered $d = 0.20$, 0.50 , and 0.80 respectively. The null for 95% CIs = 0.00

[±]Racial demography breakdown can be found in the supplementary materials

Table 2

Zero-order bivariate correlations of continuous independent variables

	M (SD)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. ADHD Index	9.85 (6.34)	-												
2. Inattention/Memory Problems	4.03 (3.02)	0.79	-											
3. Hyperactivity/Restlessness	5.95 (3.29)	0.74	0.52	-										
4. Impulsivity/Emotional Liability	2.89 (2.54)	0.76	0.52	0.59	-									
5. Problems with Self-concept	4.99 (4.26)	0.81	0.67	0.48	0.53	-								
6. Family History Density	0.12 (0.16)	0.32	0.22	0.26	0.28	0.30	-							
7. Age	37.89 (6.34)	0.15	0.05	0.02	0.14	0.23	0.18	-						
8. Years of Education	14.72 (3.02)	-0.22	-0.09	-0.16	-0.18	-0.21	-0.18	-0.18	-					
9. Negative Urgency *	2.65 (0.77)	0.63	0.47	0.48	0.59	0.47	0.16	-0.12	-0.10	-				
10. (lack of) Premeditation *	1.96 (0.63)	0.45	0.48	0.34	0.42	0.29	0.10	-0.15	-0.08	0.43	-			
11. (lack of) Perseverance *	1.98 (0.59)	0.51	0.57	0.24	0.31	0.44	0.13	-0.23	-0.06	0.37	0.56	-		
12. Sensation Seeking *	2.66 (0.72)	0.13	0.16	0.31	0.11	-0.07	0.06	-0.26	0.03	0.13	0.14	0.00	-	
13. Positive Urgency *	2.05 (0.83)	0.47	0.34	0.41	0.35	0.32	0.13	-0.10	-0.19	0.58	0.37	0.34	0.19	-
14. Alcohol Dependence Scale *	20.53 (7.65)	0.42	0.33	0.33	0.32	0.40	0.31	-0.19	-0.04	0.46	0.27	0.28	0.04	0.36

Note: ADHD = Attention deficit hyperactivity disorder

Bolded values indicate $P < 0.05$

Information on tests of multicollinearity can be found in the supplementary material

* $N = 329$ for correlations with the UPPS subscales and Alcohol Dependence Scale, while the remainder of the variables use the full sample used in the logistic regressions (i.e., $N = 749$)

Table 3

Logistic regression results for ADHD Index and other factors associated with AD

Variable	β Coefficient	OR (95% CI)	Significance
CAARS-S:S ADHD Index	0.257	1.293 (1.183–1.414)	$p < 0.0001$
History of Depressive Disorder	0.612	1.845 (0.559–6.093)	$p = 0.315$
History of Anxiety Disorder	−3.175	0.042 (0.013–0.129)	$p < 0.001$
History of Other Substance Use Disorders	−4.991	0.011 (0.004–0.034)	$p < 0.001$
Family History Density	6.308	548.835 (22.079–13642.565)	$p < 0.001$
Age	0.145	1.156 (1.1171.196)	$p < 0.001$
Education	−0.148	0.862 (0.760–0.979)	$p = 0.022$
Gender	−1.091	0.336 (0.148–0.765)	$p = 0.009$
Race	−0.026	0.974 (0.474–2.001)	$p = 0.944$

Note: AD = alcohol dependence; ADHD = attention-deficit hyperactive disorder; CAARS-S:S = Conners' Adult ADHD Rating Scales–Self-Report Scale: Short Version; CI = confidence interval; OR = odds ratio; History of depressive disorder includes a major depressive episode, major depressive disorder, or dysthymia as measured by the DSM-IV-TR SCID-I; gender is females compared to males; race is Caucasian compared to other racial minorities, history of other mental illnesses is no history compared to present history

$R^2 = 0.866$, Hosmer and Lemeshow Test $\chi^2(8) = 3.575$, $p = 0.893$.

Table 4

Logistic regression results for CAARS:S-S subscales and other factors associated with AD

Variable	β Coefficient	OR (95% CI)	Significance
CAARS-S:S			
Inattention/Memory Problems	-0.259	0.772 (0.627–0.951)	$p = 0.015$
Hyperactivity/Restlessness	0.255	1.291 (1.091–1.528)	$p = 0.003$
Impulsivity/Emotional Liability	0.198	1.219 (0.956–1.555)	$p = 0.109$
Problems with Self-Concept	0.446	1.562 (1.328–1.838)	$p < 0.001$
History of Depressive Disorder	0.829	2.291 (0.633–8.296)	$p = 0.207$
History of Anxiety Disorder	-2.973	0.051 (0.016–0.162)	$p < 0.001$
History of Other Substance Use Disorders	-4.976	0.007 (0.002–0.024)	$p < 0.001$
Family History Density	6.621	750.328 (25.949–21695.968)	$p < 0.001$
Age	0.149	1.161 (1.115–1.208)	$p < 0.001$
Education	-0.105	0.901 (0.786–1.032)	$p = 0.132$
Gender	-1.313	0.269 (0.111–0.654)	$p = 0.004$
Race	0.167	1.182 (0.574–2.555)	$p = 0.671$

Note: CAARS-S:S = Conners' Adult ADHD Rating Scales–Self-Report Scale: Short Version; CI = confidence interval; OR = odds ratio; History of depressive disorder includes a major depressive episode, major depressive disorder, or dysthymia as measured by the DSM-IV-TR SCID-I; gender is females compared to males; race is Caucasian compared to other racial minorities, history of other mental illnesses is no history compared to present history

$R^2 = 0.884$, Hosmer and Lemeshow Test $\chi^2(8) = 6.682$, $p = 0.571$.

Table 5

Indirect effects of UPPS-P subscales

Independent Variable	Mediating Variable	Dependent Variable	Parameter Estimates	Standard Error	95% CI
ADHD Index →	Negative Urgency		0.163	0.053	0.060, 0.269
	(lack of) Premeditation		0.001	0.028	-0.054, 0.057
	(lack of) Perseverance	→ ADS Score	0.004	0.038	-0.075, 0.078
	Sensation Seeking		-0.006	0.008	-0.034, 0.002
	Positive Urgency		0.073	0.036	0.006, 0.156
Inattention/Memory Problems →	Negative Urgency		0.045	0.036	-0.005, 0.147
	(lack of) Premeditation		0.019	0.057	-0.095, 0.138
	(lack of) Perseverance	→ ADS Score	0.001	0.080	-0.159, 0.158
	Sensation Seeking		-0.020	0.033	-0.105, 0.031
	Positive Urgency		0.031	0.031	-0.007, 0.114
Hyperactivity/ Restlessness →	Negative Urgency		0.082	0.042	0.018, 0.193
	(lack of) Premeditation		0.000	0.009	-0.016, 0.027
	(lack of) Perseverance	→ ADS Score	-0.000	0.012	-0.031, 0.025
	Sensation Seeking		-0.027	0.038	-0.108, 0.043
	Positive Urgency		0.063	0.036	0.010, 0.155
Emotional Liability/ Impulsivity →	Negative Urgency		0.214	0.073	0.077, 0.364
	(lack of) Premeditation		0.012	0.04	-0.055, 0.117
	(lack of) Perseverance	→ ADS Score	0.000	0.01	-0.024, 0.024
	Sensation Seeking		0.001	0.013	-0.017, 0.040
	Positive Urgency		0.041	0.037	0.000, 0.156
Problems with Self-Concept →	Negative Urgency		0.079	0.036	0.022, 0.168
	(lack of) Premeditation		-0.003	0.013	-0.046, 0.013
	(lack of) Perseverance	→ ADS Score	0.000	0.018	-0.039, 0.038
	Sensation Seeking		0.022	0.034	-0.035, 0.099
	Positive Urgency		0.035	0.023	0.002, 0.093

Note. ADHD = attention-deficit hyperactive disorder; ADS = alcohol dependence severity; CI = confidence interval