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ADHD, Impulsivity and Alcohol Abuse: Methods, Results and Implications

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Abstract

In this Special Section, the relationships among attention deficit hyperactivity disorder (ADHD), impulsivity, and alcohol abuse are explored by four diverse studies. Among these excellent studies, the constructs indicated in the issue title varied in definitions, measurement methods and interpretive implications. The experimental approaches also varied, including cross-sectional examination of candidate genes in children, determination of long-term outcomes, laboratory tasks to measure attention and inhibition, and alcohol administration. These diverse topics and approaches yielded insights into the etiology and effects of substance abuse and indicated research directions that will advance this field.

Keywords

attention deficit hyperactivity disorder; impulsivity; alcohol

In this Special Section, the relationships among attention deficit hyperactivity disorder (ADHD), impulsivity, and alcohol abuse were explored by four diverse studies. These studies addressed a broad range of issues. Lee and Humphries examined ADHD characteristics related to children's expectations about alcohol effects. Molina et al. studied characteristics in adolescence and early adulthood that, in the context of childhood ADHD, predicted later problematic alcohol use. In young adults, Endres et al. investigated neurocognitive impairments related to clinical diagnoses encompassing externalizing disorders (EXT), including ADHD and alcohol use disorders. Roberts et al. studied the immediate effects of alcohol consumption effects on attention and inhibition.

These new data provided novel information on the determinants of alcohol involvement as well as alcohol effects. Each informed different facets of our understanding of ADHD,

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impulsivity and alcohol involvement. These excellent studies considered the constructs indicated in the issue title with various definitions and measurement methods. The experimental approaches also varied, including cross-sectional examination of candidate genes in children, determination of long-term outcomes, laboratory tasks to measure attention and inhibition, and alcohol administration. These diverse topics and approaches yield insights into the etiology and effects of alcohol abuse and indicate research directions that will advance this field. What emerges are commonalities in inhibitory control impairments pertinent for understanding ADHD, impulsivity and alcohol abuse.

Three articles (Molina et al., Lee and Humphries, Endres et al.) examined ADHD as measured by items in the American Psychiatric Association Diagnostic and Statistical Manual (DSM), but each study used these indicators as somewhat different constructs. In a study of relatively young school-aged children, Lee et al. measured ADHD symptoms and DRD4 genotype at age 6-9 years, then again determined ADHD symptoms as well as alcohol expectancies (AE) about two years later. ADHD items were considered indicators of a continuous variable over multiple time points. At the second assessment, most children were sufficiently young (i.e., age 8-13 years) as to make significant alcohol use unlikely. Accordingly, AE did not reflect alcohol consumption experiences. However, even at this young age, ADHD symptoms were associated with AE, including expectations that alcohol would result in negative arousal, sedation and being 'wild and crazy.' Remarkably, DRD4 genotype predicted expectations of being 'wild and crazy' after alcohol consumption. In addition, DRD4 7+ girls, compared to DRD4 7- girls, had more negative AE while DRD4 7- girls showed a positive association between ADHD symptoms and negative AE.

Since AE predicts alcohol involvement, the determinants of childhood AE may identify risks or suggest etiological mechanisms that could be targeted in preventive interventions. The authors proposed several mechanisms by which DRD4 genotype might affect AE. First, "wild and crazy" AE may be part of a broader phenotypic constellation related to dopamine function, including reward sensitivity and risk taking. Alternatively, the genotype may be associated with parental alcohol involvement, which may, in turn, influence offspring AE. Finally, the designation of 'wild and crazy' as a positive or a negative description of alcohol effects varies across development, and at the age of testing in this study may reflect a tendency of some children (e.g., the DRD4 7 repeat genotype) to assign a positive valence to alcohol use. Whatever the mechanism, the finding that this genotype is associated with specific expectations about alcohol use experiences is notable. Although this finding is best considered preliminary, the genetic influence on the predisposition to use alcohol may operate through alcohol-related cognitions that pre-date the first alcohol consumption experience.

By focusing on diagnoses, Molina et al. utilized a categorical approach to the ADHD construct. The ADHD children in this study were identified through a clinical setting, and thus may have been more severe or may have had other unique characteristics when compared to ADHD children representative of those in the general population. Molina studied the pathways from childhood ADHD to problematic alcohol use (i.e., heavy drinking), with a focus on the mediating effects of social impairment and delinquency. Comparing these long-term outcomes of children with (n=148) and without (n=117) ADHD,

childhood ADHD alone did not predict young adult heavy drinking. However, childhood ADHD predicted adolescent and young adulthood delinquency which, in turn, did predict heavy drinking. In these analyses, adolescent delinquency, defined to include conduct disorder characteristics and other related problems, was included as a construct distinct from ADHD. This approach reflects a different ADHD conceptualization than was evident in Enders et al., where ADHD was considered an indicator of a broader construct (i.e., EXT) including these conduct disorder characteristics. Molina et al. noted that prior observations on childhood ADHD and later problematic alcohol involvement have produced mixed results. Different approaches to considering ADHD and conduct disorder as distinct or joined constructs may, to some extent, explain apparently different results in studies on ADHD predicting alcohol involvement outcomes.

Molina et al. observed that childhood ADHD predicted social impairment, defined by parental response to a global item on the child's peer relationships. In the overall model, social impairment in adolescence predicted less heavy drinking at age 20. This observation was complicated by two additional findings: 1) when delinquency was not included in the model, the effect of adolescent social impairment was not evident, and 2) at age 20, social impairment was not associated with heavy drinking. Further research is needed to characterize social impairment in the context of childhood ADHD and adolescent developmental trajectories to understand the mechanisms whereby social impairment influences problematic alcohol use.

Whereas Molina et al. and Lee et al. considered ADHD as a construct distinct from conduct disorder, Endres et al. defined ADHD not as a distinct entity, but as part of a broader construct labeled "externalizing psychopathology" (EXT). In a young adult sample, EXT manifestations included DSM-IV lifetime problems with ADHD, conduct disorder, adult antisocial behavior, as well as alcohol and other substance use disorders. The indicators were summarized as a latent variable for analyses. Endres et al. examined neurocognitive characteristics associated with EXT in young adults using a go/no-go associative learning task presented with or without a working memory component. Individuals high on EXT had poorer working memory capacity, poorer inhibition, and poorer information processing capacity. Adding working memory load in the task impaired performance, especially in high-risk individuals. This project used a novel analytic approach, a linear ballistic accumulator computational model, to disentangle the complex interrelations among these cognitive functioning constructs. Higher EXT was associated with more false alarms (i.e., poor passive avoidance learning), which indicated impaired evidence accumulation during cues signaling inhibition. Disinhibited decision making was related to lower executive working memory capacity, and lower working memory capacity partially mediated the association between EXT and disinhibited decision making. Working memory load dramatically increased disinhibited decision making in all subjects. In essence, EXT was associated with not deliberating long enough in contexts where response inhibition was required and cognitive capabilities were challenged. This difficulty may be partly due to perseveration and/or insensitivity to punishment. Since EXT included substance use disorders, and the design was cross-sectional, the study could not determine the extent to which substance use effects may have influenced the observed relationships. These findings extend our understanding of the cognitive deficits that may underlie EXT, and suggest

targets for remediating problem behaviors through cognitive training. However, a mechanistic understanding of the relationships among neurocognitive characteristics, clinical symptoms indicating pathological difficulties with inhibiting behavior, and substance effects will require studies more optimally designed to disentangle these relationships.

While “impulsivity” may be defined as part of the ADHD construct, the term may also be applied to characteristics that are conceptually and psychometrically distinct. Endres et al. as well as Roberts et al. utilized behavioral tests in laboratory settings to define inhibitory capability and attention under specific task demands. In a study directly examining alcohol consumption effects in an adult sample, Roberts et al. found that alcohol consumption may disrupt goal-directed behavior by disinhibiting attention toward irrelevant distracters. Subjects with problematic alcohol involvement were particularly sensitive to this effect. Roberts et al. measured attention control in an adult sample (ages 21 through 35 years old) through their Delayed Ocular Response Task. In a controlled laboratory setting, the participant was challenged to inhibit a reflexive saccade (i.e., eye movement) toward a distracter stimulus. Responses were examined in trials with and without alcohol consumption. Alcohol consumption resulted in disinhibition. Another task involved responding (i.e., left or right side) to neutral or alcohol-related visual probes, where faster responses to the alcohol-related stimulus indicated “attentional bias.” Disinhibiting effects of alcohol were exaggerated in association with high attentional bias and a history of heavy drinking. The authors suggested that sensitivity to alcohol effects on attentional control may contribute to problematic alcohol use. The results were consistent with an addiction model where impaired inhibitory control and exaggerated salience of alcohol-related cues contribute to problematic alcohol use. Improving inhibitory control and reducing effects of alcohol-related stimuli are both characteristics that may be amenable to interventions.

As would be expected, each study has limitations and each raises important issues. While three of the studies included a sufficient representation of both genders to support analyses, Molina et al. included few females (less than 10% of the subjects). Even in the studies with significant female representation, however, gender effects received relatively little attention. Another important issue is that substances other than alcohol may also be pertinent. Three of the four articles specifically focus on alcohol as the substance of interest. The outcomes of ADHD children with respect to involvement with other substances, such as nicotine and cannabis, are important and related topics (e.g., Elkins et al., 2007) that are not addressed in this issue.

In conclusion, these four studies may be conceptualized as contributing to our understanding of the developmental progressions involving ADHD, impulsivity and alcohol abuse. Lee and Humphries examined ADHD and AE in children younger than the typical age of alcohol use initiation. If their findings are confirmed, and ADHD and DRD4 genotype are found to influence childhood AE, their observations suggest novel approaches to the identification of risks, and a possible avenue for preventive intervention. Their insights may lead to tailored interventions to alter specific AE perceptions in children who present with particular risk profiles. By noting that childhood ADHD, viewed as a distinct construct, does not predict problematic alcohol involvement, Molina et al. emphasize the need to consider ADHD in the

context of developmental progressions involving other characteristics, including conduct disorder or delinquency. Prior publications from this program have provided important information on treatment effects (Molina et al., 2013), and their examination of social impairment suggests that additional research on this complicated topic may be important for understanding alcohol involvement trajectories. Endres et al. reminds us that ADHD, conduct disorder, adult antisocial characteristics and substance use disorders may be conceptualized as constituting a coherent characteristic. Their study suggests that individuals with these outcomes have neurocognitive impairments that may need to be addressed for successful outcomes to occur. Complementing these studies, Roberts et al. examined alcohol consumption effects, showing that alcohol consumption can directly produce disinhibition, an effect that is well known to clinicians but may be misperceived by patients. Alcohol consumption may produce exaggerated disinhibition among adults with ADHD (Weafer et al., 2009). As a group, these four studies provide an illustration of the developmental progressions involving attention, inhibitory control and alcohol involvement. They suggest targets for preventive and therapeutic interventions, and point to further research that may provide further insights into the relationships among ADHD, impulsivity and alcohol abuse.

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