A Sobering Fact: ADHD Leads to Substance Abuse

Timothy E. Wilens, M.D.

The link between attention-deficit/hyperactivity disorder (ADHD) and alcohol or drug abuse or dependence in adolescents and adults has been an area of increasing clinical, research, and public health interest. ADHD continues to be among the most common neurobehavioral disorders presenting for diagnosis and treatment in children. ADHD occurs in early childhood and affects 6% to 9% of juveniles and 4% to 5% of adults (for review, see Wilens and Spencer). Persistent from childhood into adulthood in approximately half of cases, ADHD is associated with learning disorders, psychiatric comorbidity, and substantial educational, occupational, and interpersonal impairments across the life span.1 Substance use disorders (SUDs) usually occur in adolescence or early adulthood and affect 15% to 20% of American adults and a less defined but sizable number of juveniles.2 What has been an intense area of research over the previous decade has been the link between ADHD and SUDs. The thorough and informative meta-analysis by Charach and associates3 has been very helpful in answering an important question: does ADHD really put you at risk for later SUDs?

Understanding this fundamental question is really quite important. Given the catastrophic effects of SUDs on an individual and his or her family, anything we can do as clinicians to ameliorate the risk of developing SUDs is crucial. ADHD uniformly occurs before SUDs. Hence, in the spirit of prevention, by understanding the risk for SUDs, we can educate, monitor, and potentially modify the course of SUDs in youths growing up with ADHD. Clinicians need to start discussing this risk with our patients and their families, beginning in the fifth grade and before the onset of SUDs.

Although Charach et al.3 have shed important light on ADHD risk for SUDs, we can certainly benefit from similar analyses examining the risk for ADHD in substance abusers. Qualitative inspection of clinical and epidemiologic studies of adolescents and adults with SUDs seems to show high rates of ADHD in SUD: 15% to 25% of adults with drug- and alcohol use disorders have ADHD.4,5

In addition to highlighting the risk for SUDs in ADHD, Charach et al.3 indicated the importance of understanding these associations. We know that having ADHD makes SUDs more problematic: ADHD is associated with early initiation of cigarette smoking and SUDs, higher risk for cigarette use and SUDs, more severe cigarette smoking and SUDs, and less likelihood of remitting from the addiction compared with individuals without ADHD.5,6

So how is SUD linked to ADHD? Short answer—not sure; longer answer—multifactorial. The available literature shows that siblings, parents, and offspring of individuals with SUDs share the etiologies of ADHD and SUDs7 and evidence for the involvement of several genes in the etiology of ADHD may be relevant to SUDs: the D2 dopamine receptor gene, the dopamine-β-hydroxylase gene, the dopamine transporter (DAT) gene, the SNAP-25 gene, the D4 dopamine receptor gene, and others.7 ADHD and SUDs may have a polygenic mechanism or represent variable expressivity of a shared risk factor7 at higher risk for ADHD. In addition, individuals with ADHD are at higher risk for SUDs, and those with ADHD plus SUDs are at very high risk for both disorders compared with controls.5 Family, twin, adoption, and molecular genetic studies have shown that genes influence the likelihood of developing each disorder.

Neuropsychological deficits have been consistently documented in studies of adults with ADHD that are remarkably similar to those observed in adults with SUDs.8 These adults tend to have impaired performance on tasks assessing vigilance, motoric speed, response inhibition, verbal learning, and working memory. Studies evaluating the relation of the neuropsychological...
dysfunction observed in ADHD to SUDs are underway. For instance, is it ADHD or the cognitive executive dysfunction in ADHD that results in SUDs?

The neuroimaging literature shows evidence of structural brain abnormalities in individuals with ADHD, including smaller volumes in the frontal cortex, cerebellum, and subcortical structures. Functional imaging studies have implicated frontosubcortical systems and deficits in anterior cingulate activation in the pathophysiology of ADHD, findings that have also been observed in studies of SUDs. Three subcortical structures implicated by the imaging studies (i.e., the caudate, putamen, and globus pallidus) are part of the neural circuitry underlying motor control, executive functions, inhibition of behavior, and the modulation of reward pathways—critical in SUDs. These frontal-striatal-pallidal-thalamic circuits provide feedback to the cortex for the regulation of behavior. ADHD is thought to be mediated by catecholaminergic dysregulation of norepinephrine and dopamine—the latter implicated as a common pathway for SUDs.

Evidence also exists that a subgroup of individuals with ADHD may be self-medicating. Adults with nicotine dependence often describe improved attention and executive functioning—consistent with the literature on using nicotinic agents for ADHD. ADHD may be associated with or result in conduct symptoms and eventual SUDs that may be related to demoralization and failure. Other evidence of self-medication includes preference of drugs over alcohol in adolescents and adults with ADHD and a higher risk for cigarette smoking and SUDs in individuals with ADHD with residual ADHD symptoms. Conversely, Biederman et al. found that adults with ADHD were indistinguishable from their peers without ADHD in the type of substance abused, with no differences evident in indices of self-medication (e.g., for mood, sleep, other) between young adults with and without ADHD. The specific choice of substances by individuals does not appear to correlate with any particular aspect of ADHD, with marijuana being the most available and common drug of abuse in ADHD.

Now that ADHD is a definitively known risk for SUD, future research needs to begin distinguishing within ADHD what the predictors are of those children with ADHD that put them at highest risk for later SUDs. For instance, what is inherent in ADHD that leads to SUD: is it a conduct disorder, aggression, attentional dysfunction, executive dysfunction, self-medication, or common shared neurobiological mechanisms between ADHD and SUD? What are treatment effects on later SUDs? Studies through adolescence have shown decreases or delays in SUD; however, studies into adulthood have not shown major differences. Are these differences in outcome a result of the strong risk for SUDs in ADHD or a lack of persistent treatment (e.g., stopping one’s antihypertensive places them at higher risk for subsequent stroke); and in whom does persistent treatment assist—those with ADHD and no comorbid psychopathology or those with ADHD and a comorbidity such as conduct disorder?

So at the end of the day, thanks in part to the work of Charach et al., one realizes that like conduct disorder, ADHD begets a substantial risk for SUDs and that one needs to plan, educate, and intervene appropriately. Future work will assist greatly in understanding mechanistically the link, prevention, and early intervention steps.

REFERENCES


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Correspondence to Timothy E. Wilens, M.D., Massachusetts General Hospital, 55 Fruit Street, YAW 6A, Boston, MA 02114; e-mail: twilens@partners.org

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