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The Complicated Relationship Between Attention Deficit/ Hyperactivity Disorder and Substance Use Disorders

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Abstract

Adolescents and young adults with substance use disorders (SUD) and attention deficit/ hyperactivity disorder (ADHD) are increasingly presenting in clinical practice. The overlap and role of treatment for these co-occurring disorders remains unclear. A review of the literature was conducted to highlight and update recent evidence on the overlap of ADHD and SUD, the role of ADHD medication on later SUD, and the treatment of ADHD and SUD in adolescents and young adults. Recent work continues to highlight the high risk for comorbid ADHD in patients with SUD; and conversely, the high risk for SUD developing in ADHD across the lifespan, particularly in the context of comorbid conduct disorder. Although the data remains discordant, it appears that ADHD pharmacotherapy does not increase the risk for SUD. Medication treatment alone does not appear to be particularly effective in treating SUD in currently active substance abusing

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individuals with ADHD. Structured therapies may be effective in treating adolescents and young adults with ADHD and SUD. Further controlled trials evaluating the sequence and effect of structured psychotherapies and/or ADHD pharmacotherapy on SUD relapse in these groups are warranted.

Keywords

Adolescence; Substance use disorders; Attention deficit/hyperactivity disorder; Stimulants comorbidity; Cognitive-behavioral therapy

Introduction

Attention deficit/hyperactivity disorder (ADHD) is among the most prevalent neurobehavioral disorders presenting for treatment in children and adolescents [1•]. ADHD affects between 6 to 9 % of children and adolescents and up to 5 % of adults worldwide [2, 3]. Historically, ADHD was not thought to continue beyond adolescence however, long-term controlled follow-up studies have demonstrated the persistence of the disorder with childhood ADHD continuing into adolescence for approximately three-quarters of cases and into adulthood for half of cases (for review see [4]). Compared to their non-ADHD peers, adolescents with ADHD have more disturbances in social relations and academic underachievement despite adequate intellectual abilities [5]. ADHD is also frequently associated with co-occurring learning and psychiatric problems across the lifespan [4]. Likewise, substance use disorders (SUD) remain among the most problematic co-occurring disorders with ADHD [6•].

SUD usually begins in adolescence or early adulthood and affects up to 30 % of U.S. adults [3, 7]. It is estimated that 9 % of adolescents manifest a drug use disorder and 6 % meet criteria for an alcohol use disorder [3]. Childhood-onset SUD predicts increased severity of SUD, decreased efforts to seek treatment, and prolonged duration of SUD in adulthood [8, 9]. Early onset SUD is associated with elevated rates of academic failure, suicidal behaviors, and other dangerous behaviors [10–12]. The misuse of marijuana, alcohol, or the combination of the two are the most common substances of abuse in adolescents with ADHD [6•]. Given the apparent risks associated with having a SUD, as well as high rate of ADHD in adolescents, the overlap between the two disorders is relevant to research as well as clinical practice.

Are ADHD and SUD Linked?

A longstanding literature has suggested that, compared to the general population, adolescents with SUD have a higher risk for having ADHD [4, 13]. For example, in a large multisite National Institute of Health (NIH) study of cannabis use disorders, ADHD was reported to occur in 38 % of both adolescent girls and boys [14]. In a recent international met-analysis by van Emmerik and associates [15•], 23 % of treatment-seeking largely young adult substance abusers had ADHD. Furthermore, the International ADHD in Substance Use Disorders Prevalence (IASP) cross-sectional study sought to determine the prevalence of ADHD in adult treatment seeking patients with SUD in different countries and SUD

populations [16•]. Studying 3558 subjects from ten countries, they found that 40 % of subjects screened positive for ADHD. McAweeney et al. [17] found a significant difference in the prevalence rate of previously diagnosed youth with ADHD (3 %) and those diagnosed while in treatment (44 %). Whether the reason for this under reporting of rates is caused by differences in substance of abuse, in diagnostic assessment, or between populations is unclear at this time.

Children with ADHD have been found to be at increased risk for developing an SUD [18, 19•, 20•, 21•, 22•]. The risk of SUDs have been shown to be twice as high among people with ADHD and four times as high among those with ADHD and comorbid conduct disorder [6•, 23]. In a meta-analytic review, Charach and collegeaues [20•] noted a substantially higher likelihood of cigarette smoking (2.4x's) and SUD (1.5x's) in youth with ADHD compared to those without ADHD. Groenman et al. [22•] recently reported in a four-year follow-up that youth with ADHD were at highest risk for developing an SUD or nicotine dependence, independent of conduct disorder. Furthermore, 1480 pairs of twins were followed from childhood to adolescence in a population-based prospective twin study conducted by Chang et al. [21•]. Results revealed that hyperactive/impulsive symptoms of ADHD predicted early onset-tobacco use, and that those children with persistent hyperactivity/impulsivity were at a pronounced risk for both early-onset tobacco and alcohol use [21•].

One important antecedent to developing SUD in children and adolescents with ADHD is cigarette smoking. For instance, in one study, over half of adolescent ADHD smokers developed SUD in young adulthood [24]. Interestingly, explanations for this may be that exposure to peers using nicotine may increase the risk for SUD due to availability and convenience. Furthermore, animal data suggests that there may be neuroplastic changes in the developing brain associated with nicotine use increasing the likelihood of both behavioral changes and later SUD [25]. Of interest, recent work has also demonstrated that cigarette smoking increases the likelihood for adolescents with and without ADHD to develop new neuropsychologically-defined executive function deficits [26•].

Adolescents with ADHD, compared to peers without ADHD, have been found to be at increased risk for SUD as well as have an earlier onset and more chronic path [18, 27–29]. In a case-control study, Kousha et al. [30•] recently showed that adolescents with ADHD had a younger age of onset for SUD, a shorter period between the first use of a substance and developing a fulminate SUD, greater functional impairment, and more severe use of substances. In many of the above studies, comorbid conduct disorder was related to the highest rates and earliest onset of cigarette smoking and SUD in adolescence [20•, 31].

What Explains the Link Between ADHD and SUD?

Why SUD is linked to ADHD remains unclear. Adolescents with active SUD report using substances frequently in order to attenuate their moods and to help them sleep [32]. Adults with nicotine dependence have described improvement in attention and executive functioning, which is consistent with the literature suggesting the use of nicotinic agents as

treatment for ADHD [33]. Considering this, and the fact that ADHD is chronic and often associated with demoralization and failure, the self-medication hypothesis is compelling.

The neurobiological link between ADHD and SUD has shown evidence of structural brain abnormalities in individuals with ADHD. These abnormalities have included smaller volumes in the frontal cortex, cerebellum, and subcortical structures [4]. Functional imaging studies have demonstrated that there may be deficits in anterior cingulate activation and the frontosubcortical systems, in both individuals with ADHD and SUD. Speculated differences in the differential development of the frontal/executive/inhibitory and the limbic/reward systems in ADHD (and conduct disorder) may also account for the increased risk for SUD [34]. Furthermore, dopamine systems and striatal involvement are similar for the two disorders [13]. However, these compelling theories need to be balanced with results of recent work from a large multisite study indicating that there is completely different neurocircuitry involved in both disorders [35•].

Other theorized explanations for the link between the two disorders include family/genetic contributions [36, 37] as well as exposure to parental SUD [38•]. Gestational exposure to nicotine or alcohol has been linked consistently to an elevated risk for ADHD in offspring [39, 40]. In addition, the literature has also shown that siblings, parents, and offspring of individuals with SUD share the etiologies of ADHD and SUD as well as several genes [37].

The Role of Co-Morbid Disorders

The link between SUD and ADHD is often studied in the context of additional co-morbid disorders. Prospective data shows that co-occurring conduct or bipolar disorders convey a heightened risk for SUD among ADHD subjects [31, 41–43]. A few clinically based studies have suggested that the link between SUD and ADHD disappears after controlling for co-morbid conduct disorder (CD) [44, 45•]. However, caution is warranted when interpreting these findings due to selection and referral biases that may have introduced higher rates of conduct disorders [44, 45•]. Population-based studies using dimensional approaches to selection have shown a significant independent link between ADHD and SUD, even though, at times, part of the association may have been mediated by conduct problems [46–48]. Szobot et al. [49], studying a community-based sample of 968 adolescents, found that after controlling for CD, adolescents with ADHD continued to present a significantly higher risk for SUD compared to peers without ADHD. Interestingly, in our 10 year follow-up of prepubescent children into young adulthood, while showing that ADHD and conduct disorder at baseline predicted SUD, we failed to find any other predicators of later SUD, such as family history of SUD, cognitive impairment, executive dysfunction, socialization, or family environment [6•].

Does Early Treatment of ADHD Influence the Risk for Later SUD?

Pharmacotherapy is a well-established treatment for ADHD, however, medications, especially stimulants, have raised significant concern surrounding their potential for abuse [50] and possible role in the later onset of SUD [51]. This, however, does not appear to be the case based on existing literature. Humphreys et al. [52•] recently conducted a large meta-analysis of 15 longitudinal studies consisting of 2565 individuals to test whether treatment

with medication for ADHD predicted later substance outcomes. Conducting separate random-effects analysis for various substance outcomes, results suggested that treatment for ADHD with medications did not influence substance use outcomes. Our group conducted the only other meta-analysis on this topic ten years ago. We found that stimulant treatment for ADHD significantly *reduced* later substance problems – particularly in adolescents [53]. Hence, one meta-analysis found no effect, and another found that treatment reduced SUD.

Some new studies not included in the Humphreys review [52•] are noteworthy and also either do not show an effect of medication treatment on SUD or show that medication treatment improves SUD and related outcomes. A recent prospective, longer-term open trial of an extended release form of methylphenidate (OROS-MPH), showed that stimulant-treated ADHD adolescents were at lower risk than a matched group of untreated ADHD youth for developing cigarette smoking [54•] and SUD [55•]. Moreover, Lichtenstein et al. [56•] recently reported on a Swedish national registry study of 25,656 ADHD young adults who were followed for 5 years through the age when criminality and SUD would be likely to develop. In this study, approximately half of the sample was treated pharmacologically for their ADHD. The authors reported a significant 41 % (females) and 32 % (males) reduction in criminality, of which approximately 36 % of crimes were drug related-and potentially a proxy of SUD [56•]. Additionally, further analysis found that, among patients with ADHD, rates of criminality were lowest during periods when the patients were receiving medication for their ADHD. Very recently, a multisite study from Europe also showed diminished SUD in treated ADHD groups [57•]. In this study, 54 men who were currently incarcerated, with ADHD and amphetamine dependence, were randomized to receive either methylphenidate (MPH) or placebo in conjunction with weekly cognitive behavioral therapy (CBT). The medication period was initiated 14 days prior to release from prison and continued for 24 weeks while released on probation. The study found that the MPH treated group reduced their ADHD symptoms during the trial and had a significantly higher proportion of drug negative urines screens compared to the placebo group. Furthermore, the MPH treated group had better retention in treatment [57•]. Even though the findings on risk reduction have been mixed, it appears that early medication treatment for ADHD does not increase the risk for later SUD. Moreover, it appears that a preponderance of studies demonstrate that pharmacological treatment of ADHD is related to improved SUD in adolescence [53] with the question of the persistence of the protective effect into adulthood. It may be that, in many of the longer-term studies, only a minority of subjects remain on their medication [58, 59••, 60••]; hence, the protective effect dissipates. It is noteworthy that all of the clinical trials including controlled studies have demonstrated a protective effect of pharmacotherapy through adolescence and young adulthood. Given the current state of research, it appears parsimonious to recommend continuation of ADHD pharmacological treatment through later adolescence and young adulthood to maintain the reduction in SUD risk.

How do you Approach Substance Abusing Adolescents and Young Adults with ADHD?

Prior to commencing any treatment of adolescents and young adults with reported SUD and ADHD, a thorough diagnostic workup needs to be undertaken. Given that active SUD may exacerbate ADHD symptoms by as much as 30 % [61•], in patients who are not well known to the practitioner, a period of brief abstinence or low level use of substances is usually necessary to help establish a valid diagnosis [62]. As discussed elsewhere, the diagnosis of adolescents and young adults with ADHD is by clinical evaluation of the individual and/or caregiver as well as review of past performance, testing, medical/neurological issues, and consideration of adjunct completion of rating scales [63•, 64].

Treatment of adolescents and adults with ADHD is predicated upon empirically based data demonstrating improvement with cognitive behavioral therapies [65••, 66, 67••, 68••, 69] as well as pharmacological therapies [70]. The role of therapies for substance abusing individuals with ADHD has been largely predicated upon the use of medications with psychotherapies- this strategy is less well developed and more inferential. Below we highlight some of the studies that have been completed being particularly mindful of the potential role of adjunctive CBT to the ultimate outcomes for both SUD and ADHD.

The Role of Medication Stimulants, noradrenergic agents, and catecholaminergic antidepressants have all been found to be effective agents in reducing ADHD symptoms [4]. Several review studies have suggested that the use of nonstimulant agents (atomoxetine), antidepressants (bupropion), and extended-release or longer acting stimulants with lower abuse liability and diversion potential is preferable when treating adolescents with co-occurring ADHD and SUD [29, 71–73]. There have been some differences in the literature regarding atomoxetine, a nonstimulant that is not associated with abuse and that has had success in treating ADHD. In patients with ADHD and SUD, results in outcome have seemed to vary depending upon the time the treatment was initiated (e.g., whether the study subjects were actively engaging in substance abuse or dependence versus being engaged in brief abstinence). In a 12-week multisite randomized controlled trial (RCT) of briefly abstinent alcoholic adults not receiving CBT, atomoxetine reduced ADHD, alcohol craving scores, and heavy drinking but not relapse [74]. Furthermore, neither serious adverse events nor evidence of impaired liver functioning was found in heavy (versus light) drinkers receiving atomoxetine [75]. In contrast, in a 12-week single site RCT of 70 current substance-abusing adolescents with ADHD receiving motivational interviewing and CBT, both placebo and atomoxetine groups improved similarly with no significant differences in either substance use or ADHD outcomes between groups [76]. McRae-Clark [77] found similar results in a recent 12-week RCT of atomoxetine or placebo in conjunction with motivational interviewing (MI). All adults with ADHD and concurrent cannabis dependence showed a significant decrease in symptoms by the end of the study, with no difference between treatment groups.

Commensurate with the Thurstone [76] and McRae-Clark [77] findings with atomoxetine, several other recent stimulant studies have shown that when it comes to providing pharmacotherapy to current substance abusing individuals with ADHD, results suggest only

minimal effects on the ADHD and substance use (See Table 1) [59••, 60••, 76–81]. Riggs et al. [59••] published results from a well-conducted, large NIH multisite study focused on the treatment of adolescents with ADHD and SUD. In this 16-week RCT, 300 adolescents with mixed SUD received CBT along with either 72 mg/day of osmotic release oral system-methylphenidate (OROS MPH) or placebo. Significant improvement in symptoms was found for both treatment arms; however, there were no group differences between improvement in ADHD or SUD related to OROS MPH or placebo. Of note, stimulant medication was not found to be misused or abused and was reported to be of relatively low abuse liability [82•] as well as side effects being similar to other studies in non substance abusing adolescents with ADHD [59••].

Similar results have also been reported in adult populations. For example, in a 12-week three-arm RCT of methylphenidate, bupropion, or placebo in conjunction with weekly CBT, Levin et al. [78] found that there were clinically significant decreases in ADHD scores in all three arms. Furthermore, in a RCT study assessing adults with ADHD and cocaine dependence currently in CBT the authors reported that MPH did not improve outcome measures compared to placebo [79]. In summary, several controlled stimulant and nonstimulant trials in non-abstinent adolescent and adult subjects with co-occurring ADHD and SUD have reported some reductions in ADHD and SUD symptoms, but no difference between treatment arms attributable to the medication when participants are concurrently receiving psychotherapy. These data seem to suggest that psychotherapies, such as CBT, may be the active treatment that is associated with the improvement in ADHD symptoms. This hypothesis clearly merits further examination.

The Role of Psychotherapy Emerging research suggests that CBT for ADHD in adolescents and adults is effective. Antshel et al. [65••] recently examined a modified CBT intervention based on adult work [83], and observed improvements in core and associated symptoms across 82 adolescents who participated in the program. This study found similar results as adult studies demonstrating the success of CBT in RCTs for reducing ADHD symptoms (see Table 2) [66, 67••, 68••, 69, 83–85] and SUD [14, 86, 87].

The success of CBT for treating ADHD in previous RCTs brings into question whether psychotherapies can contribute to treatment among adolescents and adults with comorbid ADHD and SUD. As mentioned earlier, several pharmacological RCTs on subjects with ADHD and SUD (see Table 1) have shown overall improvement among study subjects and failure for group differences (medication versus placebo) in ADHD outcomes. All of these studies have had adjunct psychotherapies, CBT or MI, for subjects warranting the idea that the psychotherapy, and not the medication, explains the overall improvement in ADHD among substance abusers with ADHD. However, previous study designs prevent further determination of the potential contribution of CBT and MI calling into question the need for further investigation into the role of psychotherapy alone for comorbid ADHD and SUD [59••].

Recommended Treatments for Substance Abusing Adolescents and Young Adults with ADHD

The strategy for caring for adolescents and young adults with SUD and ADHD should include consideration of both disorders. First, a thorough assessment of the substance use and ADHD needs be completed prior to treatment. Furthermore, adolescents and young adults with SUD benefit have been shown to benefit from both family and individual intervention [88, 89, 90•]. If possible it may be best to control the addiction initially, so that the individual with ADHD is not actively using or in a harm reduction model, is using substances in a less perilous and more “controlled” manner [29, 72]. Once the substance use is better controlled, the uses of structured psychotherapies appear to be the preferred line of treatment for addressing both the ADHD and SUD. It appears that useful psychotherapeutic intervention for ADHD and SUD adolescents and adults includes motivational interviewing and CBT that would incorporate structured and goal-directed sessions as well as active therapist involvement. Additionally, pharmacological agents can be used in conjunction with psychotherapy in order to alleviate ADHD symptoms and further substance use. Since there is a dearth of literature examining psychotherapy specifically for adolescents and young adults with SUD and ADHD, more work examining the efficacy of CBT for the treatment of both active disorders and/or relapse prevention is necessary.

Conclusion

In conclusion, a convincing literature shows that children and adolescents with ADHD are at an increased risk for various substance use disorders, including cigarette smoking and developing early-onset SUD. Children and adolescents with ADHD and their caregivers need to be educated about this increased risk. Medication treatment of ADHD may protect against SUD, but only if the medications are taken continuously. When approaching the care of adolescents and young adults with co-occurring SUD and ADHD, engagement, support, and treatment of the families are critical. Stabilization of the substance use in comorbid individuals is a notable first aim of treatment. Results of recent studies suggest that the use of structured psychotherapies, like CBT, should be the first lines for addressing ADHD with comorbid SUD. Studies of CBT for ADHD alone are promising, although it is still unclear if CBT is most effective as a stand-alone treatment or in conjunction with medication. Initiating medication for ADHD in the SUD population appears to be best used following some stabilization of the SUD. Future studies need to assess the sequence and effectiveness of psychotherapies alone and in combination with medications to treat ADHD and SUD in adolescents and adults.

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Table 1

Pharmacotherapy trials for individuals with SUD and ADHD

Author, year [ref]	Individuals (N/sex)	Age range	Sample description	Intervention	Retention	Outcome	Psychotherapy sessions	Psychotherapy comments
Riggs *et al.*, 2011 [59••]	*N*=303 78.9 % male	13–18	ADHD & 1 non-tobacco SUD (excluding opiods/meth)	16 week multi-site double-blind RCT of OROS-MPH + CBT or placebo + CBT	78.1 % of OROS & 71.7 % of placebo completed; 79 % medication compliance	Clinically significant decrease in ADHD scores and reduction in drug use in both groups; no group difference	Individual CBT 16 sessions 1X a week	Targeted at *substance use* Manual-standardized CBT using motivational enhancement approaches
Thurstone *et al.*, 2010 [76]	*N*=70 78.6 % male	13–19	ADHD & 1 non-tobacco SUD	12 week double-blind RCT of ATMX + CBT or placebo + CBT	92 % completed the trial, 8 % were lost to follow-up [3 ATMX, 2 placebo]	Clinically significant decrease in ADHD scores in both groups; no group difference. No group difference in drug use	Individual CBT 12 sessions 1 hour-long 1X a week	Targeted at *substance use* Core modules included goal setting, a functional analysis of drug use, and coping with cravings
Levin *et al.*, 2006 [78]	*N*=98 57.1 % male	18–60	ADHD & opiod dependence (53 % cocaine)	12 week double-blind 3 arm RCT of either MPH, BPR, or placebo + weekly CBT	71 % completed the trial	Clinically significant decrease in ADHD scores in all 3 groups; no group difference. Proportion of positive drug use screens during trial high in both groups	Individual CBT 12 sessions 1X a week	Used the structured relapse prevention manual established by Carroll et al. [86] targeted at preventing *relapse to cocaine*. Modified it for ADHD
Levin *et al.*, 2007 [79]	*N*=106 83 % male	23–52	ADHD & cocaine dependence	14 week double-blind RCT of MPH + CBT or placebo + CBT	84 % completed at least 4 weeks [PBO 83 %, MPH 85 %], & 44 % completed the entire trial [PBO 45 %, MPH 43 %]	Clinically significant decrease in ADHD scores; no group difference. MPH group had slightly higher increase in probability of screening negative on tox screen compared to placebo	Individual CBT 14 sessions 1X a week	Used the structured relapse prevention manual established by Carroll et al. [86] targeted at preventing *relapse to cocaine*. Modified it for ADHD
Levine *et al.*, 2009 [80]	*N*=20 95 % male	18–60	ADHD & cocaine dependence	12 week open trial of ATMX + CBT	65 % reached the maintenance	Significant reduction in ADHD	Individual CBT 12 sessions 1X a week	Used the structured relapse prevention manual established by Carroll et al.

Author, year [ref]	Individuals (N/sex)	Age range	Sample description	Intervention	Retention	Outcome	Psychotherapy sessions	Psychotherapy comments
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
					dose phase, 45 % completed at least 6 weeks of treatment & 25 % completed all 12 weeks			
Schubiner *et al.*, 2002 [81]	*N*=48 89.6 % male	18–55	ADHD & cocaine dependence	12 week double-blind RCT of MTP + CBT or placebo + CBT	58 % of the placebo group & 45 % of the MTP group completed the trial	No group difference in ADHD symptoms. MTP group had significant decline in SUD symptoms compared to controls	Individual CBT 24 sessions 2X a week	Based on strategies outlined in the Project MATCH CBT manual [91] and a CBT manual for cocaine treatment [86]. Targeted *substance use*
McRae-Clark *et al.*, 2010 [77]	*N*=38 76 % male	18–65	ADHD & cannabis dependence	12 week double-blind placebo controlled trial of ATXM or placebo in conjunction with Motivational Interviewing	42 % completed the trial [16 completed in ATMX group and 7 completed in placebo group]	ATM group had greater improvement in ADHD on the CGI scale compared to placebo; no group differences in self-rated ADHD symptoms, overall Wender-Reimherr Adult ADHD Scale, or marijuana use	Motivational Interviewing (MI) 3 sessions 3 hours each (2 hours of testing and 1 hour of feedback)	Modeled after the Drinker's Check-UP33 [92] which combines MI with personal feedback of assessment findings in relation to population or clinical norms
Tamm *et al.*, 2013 [60••]	*N*=299 79 % male	13–18	ADHD & 1 non-tobacco SUD	16 week double-blind RCT of OROS-MPH + CBT or placebo + CBT	75 % completed the trial; 86 % medication adherence; 75 % CBT adherence	Significant improvement in SUD for those with CD in OROS group compared to those with CD in placebo group. In either group significant baseline affect for ADHD	Individual CBT 16 sessions 1X a week	Targeted at *substance use* Manual-standardized CBT using motivational enhancement approaches

All Psychotherapy was concurrent

RCT = Randomized controlled trial

TAU = Treatment as usual

Table 2

Cognitive behavioral therapy trials for individuals with ADHD

Author, year [ref]	Individuals (N/sex)	Age range	Sample description	Intervention	Retention	Outcome	Psychotherapy sessions	Psychotherapy comments
Weiss *et al.*, 2012 [84]	*N*=48 N/A	18–66	ADHD	Secondary analysis of multisite double-blind RCT, parallel study where subjects randomized to Dextroampheta-mine + CBT or placebo + CBT	62 % of medication arm and 77 % for placebo completed the trial (no significant difference)	Both groups showed improvement in functioning; no group differences	Individual CBT 9 sessions (session 8 & 9 were “booster sessions”)	Addressed specific issues of ADHD such as emotional dysregulation, sleep, *addiction*, anger outbursts, and other common problems in ADHD
Emilsson *et al.*, 2011 [67••]	*N*=54 37 % male	18-up	ADHD with stable psychopharm	RCT of 15 sessions of CBT + stable psychopharm or TAU	74 % completed the trial	Significant decrease in symptoms for CBT group that decreased further at 3-month follow-up. Comorbid problems also improved in CBT group	Group and Individual CBT 15 sessions	Structured, manualized program that aims to decrease impairment of core ADHD symptoms and improve social, problem solving, and organizational skills
Solanto *et al.*, 2010 [68••]	*N*=88 34 % male	18–65	ADHD	12 week RCT of either meta-cognitive therapy (MCT) or support group	84 % in MCT group & 63 % in the support group completed the trial	Improvement in MCT group compared to support group. Higher scores at baseline meant larger drop with MCT	Group MCT 12 sessions 2 hours each	Designed to enhance time management, organization, and planning in adults with ADHD
Philipsen *et al.*, 2007 [85]	*N*=66 59.7 % male	18–53	ADHD (some had SUD and 43 patients on stable psychopharm)	Open multi-site study of weekly sessions of structured skills training	92 % completed the trial	Improvement in ADHD & depression symptoms; did not see medication effect	Group Structured Skills Training 13 sessions	“Control ADHD rather than to be controlled by ADHD” was defined as the overall objective of the program. The first part of each session served as a discussion of home exercises, and during the second part new topics were presented.
Bramham *et al.*, 2009 [66]	*N*=61 in CBT group *N*=37 on waitlist 65.6 % male 57 % male	18–45	ADHD (all but 2 on stable psychopharm)	Non randomized intervention of 6 sessions CBT compared to a group on waitlist not receiving CBT	67 % in CBT group completed the trial	CBT group had significant improvements on measures of ADHD, self efficacy, and self- esteem	Group CBT (average 10 people per workshop) 6 sessions 1X a month	Designed to treat ADHD as well as comorbid anxiety, depression, and low self-esteem

Author, year [ref]	Individuals (N/sex)	Age range	Sample description	Intervention	Retention	Outcome	Psychotherapy sessions	Psychotherapy comments
Antshel *et al.*, 2012 [65••]	*N*=68 66.2 % male	Adolescents Mean age 16	ADHD with stable psychopharm	All received manualized CBT program	100 % completed all modules of CBT, 55 % never missed a session & remaining 45 % only missed 1–3 sessions	Variables all improved; lower improvement for those with ODD	Individual CBT 7 core sessions and 4 optional sessions	Downward extension of Safren *et al*. 2005 [83] CBT for adults with ADHD
Safren *et al.*, 2005 [83]	*N*=31 45 % male	23–59	ADHD with stable psychopharm (clinically severity of at least moderate)	Randomized double-blind, 12 sessions CBT + psychopharm or TAU	100 % completed the trial; no dropouts	CBT group had lower IE-rated ADHD, anxiety, & depression and lower self-reported ADHD and anxiety	Individual CBT 12 sessions 3 core modules & 3 optional modules	Directed at ADHD containing elements of motivational interviewing, and practice, repetition and review of previously learned skills
Safren *et al.*, 2010 [69]	*N*=86 55.8 % male	18–65	ADHD with stable psychopharm for at least 2 months (but still significant symptoms)	RCT of either 12 sessions of CBT or relaxation with educational support (RES)	2 subjects in the CBT group and 6 subjects in the RES group were not retained through the post-tx assessment	CBT group received lower scores on CGI and ADHD rating scale rated by blind IE. F/U maintained gains over 6 and 12 months	Individual CBT 12 sessions 3 core modules & 2 optional modules	Consistent with their prior models (see [83])

All Psychotherapy was concurrent

RCT = Randomized controlled trial

TAU = Treatment as usual