Published in final edited form as:

J Anxiety Disord. 2011 June ; 25(5): 690–696. doi:10.1016/j.janxdis.2011.03.005.

CBT for childhood anxiety and substance use at 7.4-year follow-up: A reassessment controlling for known predictors

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Abstract

A previous report suggested that successful cognitive behavioral therapy (CBT) for child anxiety reduced substance use problems at 7.4-year follow-up, but that report did not include predictors of: (a) substance use disorder (SUD; e.g., attention deficit-hyperactivity disorder symptoms, negative life events, family substance abuse, additional treatment), or (b) treatment outcome (e.g., severity of internalizing pathology, age). Analyses incorporating these factors tested previously reported findings in 72 participants (ages 15–22 at follow-up; 84% of the 7.4-year follow-up sample), using parent and youth diagnostic interviews and report measures. The majority of previously reported associations between less successful treatment and later substance use problems remained significant after controlling for known predictors of SUD and treatment outcome. Our findings bolster previous conclusions that effective CBT for child anxiety may have ameliorative effects on the target disorder and later substance use problems.

Keywords

Cognitive-behavioral therapy; child; adolescent; anxiety disorder; substance abuse

1. Introduction

Childhood anxiety disorders are associated with strained peer and interpersonal relationships, academic difficulties, and reduced psychological wellbeing (Verduin & Kendall, 2008; Costello, Egger & Angold, 2004). They are common, affecting approximately 18% of children (Kessler, Chiu, Demler, & Walters, 2005) and unlikely to remit without treatment (Pine, Cohen, Gurley, Brook, & Ma, 1998). Rather, the detriments of child anxiety are both immediate and prolonged. In addition to disrupting early functioning, childhood anxiety is a significant risk factor for later negative sequelae, such as substance use disorders (SUDs; Pine et al., 1998; Kaplow, Curran, Angold, & Costello, 2001). Research suggests that childhood anxiety disorders typically precede the onset of SUDs (Merikangas et al., 1998), a primacy that is compelling given the high lifetime co-

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occurrence of these disorders (35% to 45%; Kessler et al., 1996) and the deleterious outcomes associated with SUDs (Toumbourou et al., 2007).

Cognitive behavioral therapy (CBT) for anxiety in youth is an efficacious treatment (Silverman, Pina, & Viswesvaran, 2008) with enduring effects (Glantz et al., 2007; Kendall & Kessler, 2002; Kessler et al., 2007). A previous randomized clinical trial (RCT) of CBT for child anxiety reported that treatment responders (i.e. those whose anxiety diagnoses were either (a) no longer present or (b) no longer the principal diagnosis following treatment) had reduced substance use and fewer associated problems compared to treatment non-responders at 7.4-year follow-up (Kendall, Safford, Flannery-Schroeder & Webb, 2004). Compared to responders, non-responders drank more days per month, were more likely to have unwanted social, physical/psychological consequences from drug use, gave up more activities due to drug use, used larger amounts of drugs, and made more unsuccessful attempts to control their use. Substance dependence is often viewed as chronic, requiring long-term treatment (McLellan, Lewis, O'Brien, & Kleber, 2000). Thus, the claim that CBT for child anxiety may mitigate later substance use, which may in turn lower the risk of SUDs, requires re-examination.

The findings of Kendall and colleagues (2004) did not take into account important predictors of SUDs, such as inattention and impulsivity-hyperactivity, perceived negative life events, and family history of substance abuse. Research supports externalizing behavior and a family history of substance abuse as risk factors for later substance problems (Reinherz et al., 2000; Kendler, Davis, & Kessler, 1997). Attention deficit hyperactivity disorder (ADHD) often predates adolescent substance use (Wilens, et al., 1997). Likewise, increased negative life events have predicted SUDs even when controlling for other critical contributing factors such as genetic vulnerability (Conner, Hellemann, Ritchie, & Noble, 2009; Wills, Vacaro, & McNamara, 1992). Given these associations, it is unclear if the increased substance use of non-responders versus responders reported previously (Kendall et al., 2004) is attributable to their poor CBT treatment outcomes or to these other SUD risk factors.

Approximately 30–45% of children treated with CBT continue to meet diagnostic criteria for an anxiety disorder following treatment (Cartwright-Hatton et al., 2004). Older child age and greater internalizing pathology, but not diagnostic comorbidity, have been associated with poorer treatment outcomes (Rapee, 2003; Southam-Gerow, Kendall & Weersing, 2001; Crawford & Manassis, 2001). Given that internalizing symptoms are also related to increased SUD risk (King, Iacono, & McGue, 2004), it seems plausible that the association between poor treatment response and later substance misuse may be a spurious association resulting from the association of internalizing symptoms with both these outcomes. Similarly, child age might account for both a child's treatment response and later substance use given that older youth are speculated to have a more chronic or developmentally "non-normative" anxiety (Southam-Gerow, et al., 2001) that might also increase their risk for SUDs.

The present study tested whether previously reported associations between differential CBT outcomes and substance use at 7.4-year follow-up would hold after (a) controlling for predictors of SUDs (i.e., ADHD symptoms, negative life events, family substance abuse history), and (b) controlling for predictors of CBT outcome (internalizing pathology, older age). The receipt of additional treatment between the post and 7.4-year follow-up assessment was also included as a novel control variable. Additional treatment may reflect the child's exposure to factors related to increased SUD risk, such as life stressors or emerging child or family psychopathology, or serve as a protective factor against such risk.

We hypothesized that previously reported findings would be strengthened by the inclusion of these control variables.

2. Methods

The present data set, consistent with the initial report, was from prior studies: an earlier 7.4-year follow-up (Kendall et al., 2004) and the original randomized clinical trial (RCT; Kendall et al., 1997). Ninety-four youth (62% male), ages 9–13 years ($M = 11 \pm 1.34$) at treatment), presented for initial intake at an outpatient child anxiety clinic and met criteria for a principal diagnosis of social phobia ($n = 17$; formerly avoidant disorder), separation anxiety disorder ($n = 22$), or generalized anxiety disorder ($n = 55$; formerly overanxious disorder). Children were excluded from the initial sample if they displayed psychotic symptoms and/or if they were taking antianxiety or antidepressant medications.

Sample demographic, diagnostic, and substance use characteristics are summarized in Table 1. Principal diagnoses were assigned via structured interviews administered separately to parents and child. Seventy-two of 94 original participants provided the necessary information at intake and completed substance use assessments at 7.4-year follow-up. There were no significant differences in treatment response, referral source, child age, or child ethnicity for those who did versus did not participate in the long term follow-up. However, nonparticipating children were more likely to be male and from a lower income bracket (Kendall et al., 2004).

2.1. Measures

Anxiety Disorders Interview Schedule for Children (ADIS-C) and for Parents (ADIS-P)—The ADIS-C and ADIS-P (Silverman & Albano, 1987), structured interviews of child mental disorders with high interrater and retest reliability (Silverman & Nelles, 1992; Silverman & Eisen, 1988), were administered independently to parent(s) and child pre- and post-treatment. When parent and child report differed, diagnoses were based on parental report¹. Clinicians made severity ratings (CSRs), ranging from not interfering to clinically interfering, for each diagnostic category, providing an additional continuous measure of endorsed symptoms. CSRs from the ADIS-P ADHD sub-section were used as a continuous quantifier of ADHD pathology in the present analyses.

Family History—At intake, parents responded to the question “not including the child, is there anyone in your family with a substance abuse problem?”

Child Behavior Checklist (CBCL)—The 118-item CBCL (Achenbach & Edelbrock, 1991) is a pretreatment parent report of child problems with documented validity, retest reliability and internal consistency (Achenbach & Rescorla, 2001).

Comprehensive Adolescent Severity Inventory – Alcohol and Other Drug Module (CASI-AOD)—The CASI-AOD (Meyers, et al., 1995), a semi-structured interview with good retest reliability and validity (Meyers et al., 2006), assesses age at first use, pattern of use and social, physical and/or psychological consequences of use over the past year for each substance, the main outcomes of interest in this report.

Adolescent Perceived Events Scale (APES)—The APES (Compas, et al., 1987) assessed (a) the presence or absence and (b) the valence ($-4 = \textit{extremely bad}$, $0 = \textit{neither}$

¹In the case of one adolescent, parent report approached but did not reach a diagnosis. For this youth, the diagnosis was given based on the adolescent's report.

good nor bad, 4 = *extremely good*) of self-reported life events at 7.4-year follow-up. The valences of endorsed items can be totaled to reflect a child's cumulative experience of positive and negative events. It has favorable retest reliability and concurrent validity. For example, 2-week retest reliability for number of reported events and weighted positive events is 0.85 and 0.78 respectively (Compas et al., 1987), and scores have been found to be related to adolescent behavior problems and psychological symptomology.

2.2. Procedure

As part of the original RCT, parents and children provided consent and assent at intake. Children were randomly assigned to treatment condition and therapist. Treatment details are discussed in (Kendall et al., 1997). Doctoral candidates, blind to treatment and diagnoses, conducted diagnostic interviews (inter-rater reliability $\kappa > .85$). As part of the 7.4-year follow-up, participants were located using previously provided information. Adult participants signed informed consent. Parental consent and child assent were obtained for minors. Youth and their parent(s) were interviewed separately at home, the anxiety clinic ($n = 52$), or by phone if they relocated ($n = 20$). Diagnoses were determined based on results of the ADIS-C/P. When parent and child report differed, diagnoses were based on parent report. Fifty dollars was provided for time. For details about the 7.4 year follow-up see (Kendall et al., 2004).

2.3. Statistical Analyses

Consistent with the methodology of Kendall and colleagues (2004), treatment response was tested categorically (responder versus non-responder) in two ways. Treatment was considered a success if the child's principal anxiety (most severe and interfering) diagnosis was either (a) Type I: no longer present (i.e., child no longer met diagnostic criteria) at post-treatment or (b) Type II: no longer principal (i.e., symptoms remained, but it was no longer the main concern) at post-treatment as per child report on the ADIS-C/P. Treatment response as characterized by the parent ADIS was not a significant predictors of substance use at 7.4-year follow-up and thus not included the current re-analysis. Hierarchical linear and logistic regressions tested whether reported associations between child reported CBT outcomes (both Type I and II) and six possible characteristics of substance use (e.g., number of drinking days per month, larger amount of use, attempts to control use, giving up of activities due to use, social/interpersonal consequences of use, and psychological/physical consequences of use) measured by the CASI at 7.4-year follow-up (block two) held as significant after controlling for severity of ADHD symptomology and reported negative life events since posttreatment (block one). Clinician severity ratings, ranging from 0 to 4 on the ADHD subsection of the parent ADIS, provided a continuous measure of ADHD symptomology, whereas the APES total valence score quantified the child cumulative experience of positive and negative events since treatment.

Similarly structured regressions examined the influence of family substance abuse history and receipt of additional outpatient therapy on previously reported associations between Type I and Type II treatment outcomes and CASI substance use characteristics in a smaller sample ($n = 60$) for whom this data were available (data on these items was missing for 12 subjects). All other analyses were conducted with the full sample ($n = 72$).

A final set of regression analyses, structured in the same manner, tested if associations between CASI substance use characteristics and known predictors of treatment outcome (greater internalizing symptoms and older age in first block) explained previously significant relationships between Type I and Type II treatment responsiveness (second block) and substance use. As the aim of the present study was to reevaluate the validity of previously reported findings, treatment response was not examined as a predictor of all CASI

outcomes; rather, only those associations found to be significant by Kendall and colleagues (2004) were re-examined (dashes in Tables 3, 4 and 5 designate analyses excluded for this reason).

3. Results

3.1. Group Comparability and Sample Representiveness

Characteristics of the sample are provided in Table 1. Independent *t* test and chi square analyses revealed no significant differences on ethnicity, family income status, child age or sex for children whose principal anxiety remained present versus not present or principal versus not principal at posttreatment. There were no significant demographic or diagnostic differences in participants from the 7.4-year follow-up with (*n* = 72) versus those without (*n*=14) the necessary data for the current analyses, or in the smaller sample with family history data (*n* = 60).

3.2a. Risk Factors for Substance Abuse: Type I Treatment Response (N = 72; Table 3)

Table 2 presents the zero-order correlations among the variables of interest. Consistent with the previous findings, youth whose principal anxiety disorder remained present after treatment had more drinking days per month ($\beta = 4.78$, $t(65) = 2.41$, $p = .03$; Cohen's $f^2 = .11$), were more likely to make unsuccessful attempts to control their drug use ($B = 3.78$, Wald = 5.19, $p = .02$; Nagelkerke $R^2 = .59$), and used larger amounts ($B = 2.42$, Wald = 7.46, $p = .01$; Nagelkerke $R^2 = .34$) than individuals whose principal diagnosis was no longer present (see Table 3). Total perceived negative life events were significantly associated with the use of larger amounts of drugs ($B = .07$, Wald = 4.70, $p = .03$) and unsuccessful attempts to control drug use ($B = .13$, Wald = 5.54, $p = .02$) at 7.4-year follow-up. Inclusion of negative life events and ADHD pathology in the model undercut previously reported associations between the presence of the principal anxiety disorder at posttreatment and unwanted interpersonal consequences of drug use ($B = 2.21$, Wald = 2.88, $p = .09$). An association between a still-present principal anxiety disorder at posttreatment and psychological or physiological drug use consequences was not previously reported and thus not examined here.

3.2b. Risk Factors for Substance Abuse: Type II Treatment Response (N = 72; Table 3)

Consistent with previous reports, individuals whose principal anxiety disorder remained principal had more drinking days per month ($\beta = 6.33$, $t(65) = 2.75$, $p = .01$; Cohen's $f^2 = .13$) and were more likely to have unwanted interpersonal consequences of drug use ($B = 2.69$, Wald = 3.97, $p = .046$; Nagelkerke $R^2 = .38$) than those whose anxiety was no longer principal, after controlling for ADHD and perceived negative life events. ADHD symptoms and negative life events were not related to number of drinking days or unwanted interpersonal consequences; however, increased negative life events predicted more physical or psychological consequences of substance use ($B = .09$, Wald = 4.02, $p = .045$) and more failed attempts to control drug use ($B = .1$, Wald = 3.86, $p = .03$) at 7.4-year follow-up. Also, greater ADHD symptoms predicted unsuccessful attempts to control drug use ($B = .55$, Wald = 4.59, $p = .03$). After controlling for these relationships, youth whose principal anxiety disorder remained principal were no longer significantly more likely to experience psychological/physical consequences ($B = 1.61$, Wald = 1.87) or to make unsuccessful attempts to control their drug use ($B = 2.27$, Wald = 2.64). By comparison, youth whose principal anxiety disorder remained principal were significantly more likely to report using larger amounts of substances ($B = 2.63$, Wald = 6.28, $p = .01$; Nagelkerke $R^2 = .32$), after controlling for associations between ADHD symptoms ($B = .36$, Wald = 3.43, $p = .06$) and negative life events with larger amounts of drug use ($B = 2.27$, Wald = 4.02, $p = .08$).

3.3a. Family History and Additional Treatment: Type I Treatment Response (N = 60; Table 4)

Neither family history nor additional treatment was a significant predictor of later substance use in this sample. Neither variable diminished previously reported associations. After controlling for these variables, youth whose principal anxiety disorder remained present had more drinking days per month ($B = 5.31$, $t(57) = 2.25$, $p = .03$; Cohen's $f^2 = .11$) and were more likely to have negative social/interpersonal consequences ($B = 2.95$, Wald = 4.58, $p = .03$; Nagelkerke $R^2 = .27$), and to use larger amounts ($B = 2.34$, Wald = 6.21, $p = .01$; Nagelkerke $R^2 = .19$), than individuals whose anxiety disorder remitted.

3.3b. Family History and Additional Treatment: Type II Treatment Response (N = 60; Table 4)

Individuals whose principal anxiety disorder was still principal at posttreatment had more drinking days per month ($B = 7.89$, $t(57) = 2.95$, $p = .01$; Cohen's $f^2 = .18$) and were more likely to have unwanted social/interpersonal ($B = 3.73$, Wald = 5.59, $p = .02$; Nagelkerke $R^2 = .35$) and physical/psychological consequences from drug use ($B = 2.33$, Wald = 4.07, $p = .04$; Nagelkerke $R^2 = .17$), and to use larger amounts ($B = 3.65$, Wald = 7.64, $p = .01$; Nagelkerke $R^2 = .28$) than successfully treated individuals.

3.4a. Predictors of Treatment Response: Type I Treatment Response (N = 72; Table 5)

All previously reported associations remained significant after controlling for child's age at intake and CBCL Internalizing. Though these control variables were generally not related to substance use problems, child age was significantly associated with using larger amounts of drugs ($B = .04$, Wald = 4.6, $p = .03$). Individuals whose principal anxiety disorder remained present had more drinking days per month ($B = 4.38$, $t(68) = 2.24$, $p = .03$; Cohen's $f^2 = .11$), and were more likely to have negative social/interpersonal consequences from drug use ($B = 2.85$, Wald = 4.64, $p = .03$; Nagelkerke $R^2 = .20$), give up activities ($B = 2.68$, Wald = 4.05, $p = .04$; Nagelkerke $R^2 = .29$), make unsuccessful attempts to control their drug use ($B = 2.64$, Wald = 6.38, $p = .01$; Nagelkerke $R^2 = .24$), and use larger amounts ($B = 2.07$, Wald = 6.46, $p = .01$; Nagelkerke $R^2 = .24$) than individuals whose principal anxiety remitted.

3.4b. Predictors of Treatment Response: Type II Treatment Response (N = 72; Table 5)

Individuals whose principal anxiety disorder remained principal drank alcohol more days per month ($B = 5.99$, $t(68) = 2.61$, $p = .01$; Cohen's $f^2 = .13$) and were more likely to have unwanted social ($B = 3.55$, Wald = 6.09, $p = .01$; Nagelkerke $R^2 = .27$) and physical/psychological consequences ($B = 2.44$, Wald = 4.70, $p = .03$; Nagelkerke $R^2 = .15$) of drug use than those whose anxiety was no longer principal. They were also more likely to quit activities due to drug use ($B = 3.68$, Wald = 5.62, $p = .02$; Nagelkerke $R^2 = .39$), make failed attempts to control drug use ($B = 2.23$, Wald = 4.40, $p = .04$; Nagelkerke $R^2 = .16$), and use larger amounts ($B = 2.49$, Wald = 6.59, $p = .01$; Nagelkerke $R^2 = .26$).

4. Discussion

Previous research (Kendall et al., 2004) suggested that successful treatment of childhood anxiety led to less substance use and related problems at a 7.4-year follow-up. The present study tested these associations between successful treatment and later substance use more rigorously by controlling for the presence of comorbid ADHD pathology, perceived negative life events, family history of substance abuse, older child age, additional treatment and severity of internalizing pathology. Inclusion of these crucial variables challenged the strength of the previous findings, testing the possibility that associations between unsuccessful CBT treatment and later substance use resulted from pretreatment and/or

environmental factors independent of the treatment itself. After controlling for these variables, the majority of previous findings held: successfully treated participants drank fewer days per month and were less likely to experience aversive interpersonal consequences of their drug use 7.4 years after treatment than participants who retained their principal diagnosis. They were also less likely to give up activities due to drug use, make unsuccessfully attempts to control their drug use or use larger drug amounts. These associations persisted despite the significant contributions of negative life events, ADHD symptomology and child age to later drug-use related problems. Of all previously reported associations, only the relationship between unsuccessful treatment and physical/psychological consequences of drug use was no longer significant in this more stringent model.

The present results support the notion that effective CBT for child anxiety may have ameliorative effects on target disorders and their sequelae (i.e., substance abuse; Kendall & Kessler, 2002). Despite efforts to identify a spurious relationship between treatment response and later substance use, child characteristics related to substance use risk and poorer prognosis were not better predictors of later substance use than child treatment response. Rather, posttreatment status emerged as a strong, independent indicator of later substance use problems. Manassis and Monga (2001) suggest that adolescents may engage in substance use as a means of managing their anxiety. Present findings suggest that successful CBT may preclude the use of this unhealthy anxiety-management technique by providing anxious individuals with alternative adaptive coping skills. Though this study cannot specify the preventative mechanism instilled by successful CBT, it suggests that the prevention of later substance use problems can occur despite occurrence of negative life events and ADHD difficulties.

The present study has potential limitations. It was not possible to control for some predictors of substance use (e.g., peer influence). Rates of substance use were lower than in normative samples (Grunbaum et al., 2001). Previously reported associations between principal anxiety posttreatment and later quitting of activities due to drug use could not, in some cases, be examined due to an extremely low frequency of this behavior ($n = 3$). Further, family substance abuse history was assessed by a single item and treatment outcome based on child report, both potential limitations. Finally, there was no control group, thus research is needed to determine if there is causality in the reported associations between successful CBT and later substance use.

5. Conclusion

The ability to identify at-risk individuals and reduce substance-related problems in adolescence and young adulthood represents a valuable auxiliary benefit of CBT for child anxiety. Research suggests that substance experimentation peaks during these developmental periods and may predict later negative behavior (Kessler et al., 2007; Chen & Kandel, 1995). The long-term alleviation of anxiety symptoms and/or the preservation of coping mechanisms in this developmentally-critical period may offer a well-timed protection for at-risk children. Though childhood anxiety may be successfully treated as an acute disorder, SUDs are likely chronic disorders (McLellan et al., 2000). CBT may represent not only a treatment for child anxiety, but also a preventative intervention – a means of interrupting a trajectory from anxiety to later substance use that grows increasingly intractable and deleterious with age. This promising possibility underscores the need for further research examining the preventative effects of CBT for child anxiety on later substance problems.

Acknowledgments

This research was supported by NIMH /NIDA grant (MH60653) and facilitated by NIMH grants (64484; 080788) awarded to the fourth author.

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Table 1

Demographic and Diagnostic Characteristics	% (N = 72)	Substance Use Characteristics	% (N = 72)
Race		Alcohol	
Caucasian	85% (62)	Tried	81% (58)
African-American	6% (4)	Regular Use **	46% (33)
Asian	3% (2)	Tobacco	
Self-identified as "Other"	6% (4)	Tried	59% (42)
Family Income		Regular Use **	31% (22)
Below \$20,000	5% (3)	Marijuana	
\$20,001 - \$39,999	28% (20)	Tried	39% (28)
\$40,000 - \$59,999	33% (24)	Regular Use **	22% (16)
\$60,000 - \$79,999	25% (18)	Narcotics	
Above \$80,000	9% (6)	Tried	16% (12)
Principle Diagnoses at Pretreatment		Regular Use **	5% (4)
GAD	63% (45)	Current/Past Substance Abuse	
Social Phobia	17% (12)	Disorder	19% (14)
SAD	20% (15)	Past Substance Dependence	
Comorbid Diagnoses at Pretreatment		Disorder	5% (4)
Specific Phobia	48% (34)	Negative Consequences	
ADHD	14% (10)	Related to Substance Use	22% (16)
ODD	8% (6)	Mean Drinking Days per Month (SD)	
Mood Disorder	14% (10)	Principle Diagnosis No Longer	
Additional Therapy after		Present	2.00 (4.51)
Treatment	48% (34)	Principle Diagnosis Still	
	M = 19.16 (SD = 1.69)	Present	6.67 (9.51)
Child Age 7.4-year	(Range 15 – 22 years)	Principle Diagnosis Still	
Follow-Up*		Principle	8.33 (11.33)

GAD = Generalized Anxiety Disorder, SAD = Separation Anxiety Disorder, ADHD = Attention-Deficit Hyperactivity Disorder, ODD = Oppositional Defiant Disorder;

** Regular use indicates once a month or more.

Table 2

Zero Order Correlations

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
¹ CBCL Internalizing	1	.10	.30*	.18	.26*	.05	.05	.08	-.06	-.02	-.01	.02	-.06	-.01
² Child Age	.10	1	.18	.05	-.17	.004	.14	.19	-.06	-.06	.17	.11	.25*	.17
³ Family History of SA	.30	.18	1	-.06	-.01	-.19	-.07	-.01	-.07	.02	-.15	-.15	-.12	-.09
⁴ Additional Treatment	.18	.05	.00	1	.05	-.03	.23	.16	.13	.14	.09	.08	.003	.05
⁵ Negative Life Events	.26*	-.17	-.17	.05	1	.19	.16	.11	.33	.27	.16	.35**	.30*	.08
⁶ Level of ADHD	.05	.004	.15	-.03	.19	1	-.01	-.05	.14	.21	.07	.33**	.26*	.13
⁶ Principal Still Principal	.05	.14	-.07	.23	.16	-.006	1	.80**	.27*	.37**	.44**	.31**	.38**	.32**
⁷ Principal Still Present	.08	.19	-.01	.16	.11	-.05	.80**	1	.19	.28*	.34**	.39**	.37**	.28*
⁸ Physical or Psychological Consequences	-.06	-.06	-.07	.13	.33**	.14	.27*	.19	1	.80**	.44**	.51**	.51**	.29*
⁹ Unwanted Interpersonal Consequences	-.02	-.06	.02	.14	.27	.21	.37*	.28*	.80**	1	.56**	.65**	.36**	.32**
¹⁰ Gave up Activities	-.01	.17	.15	.09	.16	.07	.44**	.34**	.44**	.56**	1	.49**	.26*	.33**
¹¹ Unsuccessful Attempts to Control	.02	.11	-.15	.08	.35**	.33**	.31**	.39**	.51**	.65**	.49**	1	.44**	.33**
¹² Used Larger Amounts	-.06	.25	-.12	.003	.30*	.26*	.38**	.37**	.51**	.36**	.26*	.44**	1	.42**
¹³ Number of Drinking Day per Month	-.01	.17	-.10	.05	.08	.13	.32**	.28*	.29*	.32**	.33**	.33**	.42**	1

* p < .05,

** p < .01

Treatment Response and Substance Use Variables, Controlling for ADHD severity, and Negative Life Events (N = 72)

Table 3

	Number of Drinking Days per Month		Unwanted Interpersonal Consequences		Physical or Psychological Consequences		Unsuccessful Attempts to Control		Used Larger Amounts of Substances	
	B	SE	B	SE	B	SE	B	SE	B	SE
Type I Treatment										
Outcome					-					
ADHD	0.56	0.46	0.43	0.28			0.75*	.32	.40*	.20
Negative Life Events	0.01	0.06	0.07	0.05	-		0.10	.05	.06	.03
Principal Still Present	4.81*	1.93	2.21	1.30	-		3.77*	1.66	2.42**	0.90
Type II Treatment										
Outcome										
ADHD	0.52	0.46	0.42	0.28	0.18	0.27	0.55*	0.26	0.36	0.20
Negative Life Events	0.002	0.06	0.07	.05	0.09*	0.04	0.10*	0.05	0.06	0.03
Principal Still Principle	6.33**	2.30	2.69*	1.35	1.61	1.17	2.27	1.39	2.63*	1.05

* p < .05,

** p < .01;

ADHD = attention deficit-hyperactivity disorder.

¹ ADHD Clinician Severity Rating from the ADIS-C/P: M = 0.53 (SD = 1.40).

² Negative Life Events: M = 16.73 (SD = 11.16)

Treatment Response and Substance Use Variables, Controlling for Family History of Substance Abuse and Receipt of Additional Outpatient Therapy (N = 60)

Table 4

	Number of Drinking Days per Month		Unwanted Interpersonal Consequences		Physical or Psychological Consequences		Used Larger Amounts	
	B	SE	B	SE	B	SE	B	SE
Type I Treatment Outcome								
Family History of SA	-1.16	1.62	-.20	1.40	-	-	.86	.90
Additional Therapy	.51	1.53	-.30	1.42	-	-	.36	.77
Principal Still Present	5.31*	2.36	2.95**	1.38	-	-	2.34**	.94
Type II Treatment Outcome								
Family History of SA	-0.90	1.56	-.69	1.51	.45	1.22	.77	.94
Additional Therapy	.25	1.49	.06	1.55	-0.23	1.06	.71	.88
Principal Still Principle	7.89*	2.67	3.73*	1.58	2.38*	1.15	3.66**	1.32

*
p < .05,

**
p < .01;

SA = substance abuse.

Table 5
Treatment Response and Substance Use Variables, Controlling for Internalizing Symptoms and Child Age (N = 72)

	Number of Drinking Days per Month		Unwanted Interpersonal Consequences		Physical or Psychological Consequences		Give up Activities		Unsuccessful Attempts to Control		Used Larger Amounts	
	B	SE	B	SE	B	SE	B	SE	B	SE	B	SE
Type I Treatment												
Outcome												
CBCL Internalizing	-0.03	0.09	-0.03	.08	-	-	-0.04	.11	-0.01	.07	-0.05	.05
Child Age	0.04	0.04	-0.05	.05	-	-	-0.04	0.04	0.01	0.03	0.04	.02
Principal Still Present	4.38*	1.96	2.85*	1.32	-	-	2.68*	1.33	2.64*	1.05	2.07*	0.82
Type II Treatment												
Outcome												
CBCL Internalizing	-0.03	.09	-.03	.09	-0.04	0.06	-0.09	.13	-0.01	0.07	-0.05	0.05
Child Age	0.05	.04	-.06	.05	-0.03	0.04	0.07	.05	0.02	0.03	0.04	0.02
Principal Still Principle	5.99*	2.30	3.55*	1.44	2.44*	1.13	3.68*	1.55	2.23*	1.06	2.49*	0.97

* p < .05,

** p < .01;

CBCL = child behavior checklist

*I*_{CBCL Internalizing}: M = 71.99 (SD = 7.38).