

Autistic Traits in Children With and Without ADHD



WHAT'S KNOWN ON THIS SUBJECT: Studies examining the prevalence and associated features of autistic traits (ATs) in children with ADHD with exclusionary autism spectrum disorders suggest that children with ATs exhibit more severe social and interpersonal dysfunction reminiscent of the deficits in children with autism spectrum disorders.



WHAT THIS STUDY ADDS: Our results suggest that ATs are overrepresented in ADHD children when compared with control subjects. They also suggest that the presence of ATs is associated with more severe psychopathology as well as more impaired interpersonal, school, family, and cognitive functioning.

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KEY WORDS

ADD, ADHD, attention deficit disorder, attention-deficit/hyperactivity disorder, AT, autistic traits, autism traits, comorbidity, social disability

ABBREVIATIONS

ADD—attention deficit disorder
ADHD—attention-deficit/hyperactivity disorder
ASD—autism spectrum disorder
ATs—autistic traits
CBCL—Child Behavior Checklist
DSM-III-R—*Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition*
MGH—Massachusetts General Hospital
OR—odds ratio
SAICA—Social Adjustment Inventory for Children and Adolescents
SES—socioeconomic status
WISC-R—Wechsler Intelligence Scale for Children—Revised

Dr Kotte was involved significantly in the acquisition of data, analysis and interpretation of data, in the drafting and revising of the article, and in the final approval of the to-be-published version; Drs Joshi, Fried, Uchida, and Spencer were involved in the interpretation of data, in revising it critically for important intellectual content, and final approval of the version to be published; Ms Woodworth and Ms Kenworthy were involved in the drafting and revising of the manuscript, analysis and interpretation of the data, and final approval of the version to be published; Dr Faraone was significantly involved in the interpretation of data, in the drafting and revising of the article, and in the final approval of the to-be-published article; and Dr Biederman made a substantial contribution to the conception and design of the study, was significantly involved in the acquisition and interpretation of data, in the revision of the manuscript, and in the final approval of the to-be-published version.

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abstract



OBJECTIVE: To assess the implications of autistic traits (ATs) in youth with attention-deficit/hyperactivity disorder (ADHD) without a diagnosis of autism.

METHODS: Participants were youth with ($n = 242$) and without ($n = 227$) ADHD and controls without ADHD in whom a diagnosis of autism was exclusionary. Assessment included measures of psychiatric, psychosocial, educational, and cognitive functioning. ATs were operationalized by using the withdrawn + social + thought problems T scores from the Child Behavior Checklist.

RESULTS: A positive AT profile was significantly overrepresented among ADHD children versus controls (18% vs 0.87%; $P < .001$). ADHD children with the AT profile were significantly more impaired than control subjects in psychopathology, interpersonal, school, family, and cognitive domains.

CONCLUSIONS: A substantial minority of ADHD children manifests ATs, and those exhibiting ATs have greater severity of illness and dysfunction. *Pediatrics* 2013;132:e612–e622

Twin, family, and linkage studies indicate that attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorders (ASDs) share a portion of their heritable etiology.^{1–4} Genome-wide association studies found rare copy number variants shared between the 2 disorders,⁵ raising the possibility that some children with ADHD may manifest symptoms of autism even in the absence of a diagnosis of ASD. Recent studies have identified that symptoms of autism or autistic traits (ATs) appear in 20% to 30% of children with ADHD^{4,6,7} and that such children are more impaired than other children with ADHD, particularly in the domains of interpersonal communication and empathy. However, these findings require replication.

The main aim of the current study was to examine the prevalence and correlates of ATs in youth with ADHD by using data from an existing, large-scale sample of referred youth with and without ADHD in whom the diagnosis of autism was exclusionary. We hypothesized that ATs would be prevalent in children with ADHD and that their presence would be associated with higher levels of morbidity and disability.

METHODS

Subjects

Subjects were youth of both genders derived from longitudinal, case-control family studies conducted at Massachusetts General Hospital (MGH).^{8,9} These studies included participants aged 6 to 18 years with ($n = 280$) and without ($n = 242$) *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition* (DSM-III-R), ADHD ascertained from pediatric clinics at a large health maintenance organization and referrals to a pediatric psychopharmacology clinic. Within each setting, we selected non-ADHD normal controls from pediatric medical clinics

based on structured interview diagnoses. We did not exclude controls having other psychiatric disorders.

ADHD cases were identified from either a major academic medical center, in which we selected ADHD subjects from consecutive referrals to its pediatric psychopharmacology clinic and from a major health maintenance organization, in which ADHD subjects were selected from consecutively ascertained pediatric clinic outpatients. Healthy controls were ascertained from outpatients referred for routine physical examinations to its pediatric medical clinics at each setting identified from their computerized records as not having ADHD. Further information on the ascertainment of the sample have been published in detail elsewhere.^{10–13} In previous articles,^{14,15} we reported that the rates of other psychiatric disorders in the control sample were low and consistent with expectations from population studies.

Participants had a mean \pm SD age of 11.3 ± 3.2 years, were 99% white, and had a mean socioeconomic status (SES) score of 1.7 ± 0.9 . The sample included 274 children (52%) who reported that he or she was tutored (ADHD: 172 [61%]; controls: 54 [22%]), repeated a grade (ADHD: 69 [25%]; controls: 18 [7%]), or took a special class (ADHD: 74 [26%]; controls: 5 [2%]). Of the control children with academic difficulty (27% [$n = 65$]), 34% ($n = 22$) met DSM-III-R criteria for ≥ 1 psychiatric disorder versus 28% ($n = 49$) of those without academic difficulty ($n = 177$). Of the ADHD children with academic difficulty (75% [$n = 209$]), 81% ($n = 169$) met DSM-III-R criteria for ≥ 1 psychiatric disorder versus 73% ($n = 52$) of those without academic difficulty ($n = 71$). These numbers suggest that the presence of a psychiatric illness may account for the increased prevalence of academic functioning difficulties. Adoption, unavailable nuclear

family, major sensorimotor handicaps, psychosis, autism, language barriers, or an estimated IQ < 80 were exclusionary for both ADHD and control participants. Parents provided written informed consent, and children and adolescents provided written assent. The institutional review board at MGH approved the study.

Assessment Procedures

Psychiatric assessments relied on the Kiddie Schedule for Affective Disorders and Schizophrenia–Epidemiologic Version,^{16,17} conducted directly and individually with the mothers and the children. For children aged < 12 years who could not provide reliable self-reports of their symptoms, interviews were conducted with their mothers (indirect interviews). Combining data from direct and indirect interviews, we considered a diagnosis positive if it was endorsed in either interview. Social class was assessed by using the Hollingshead and Redlich scale¹⁸

Interviews were administered by highly trained and supervised psychometricians, blinded to referral source or diagnostic status (ADHD or control). Based on 500 assessments from interviews of children and adults, the median κ coefficient of agreement between a psychometrician and an experienced clinician was .98.

We used an empirically derived profile from the Child Behavior Checklist (CBCL) to define ATs (CBCL-AT) by using a cutoff of 195 from the combined T scores of the withdrawn, social problems, and the thought problems subscales¹⁹ that correctly classified 78% of all subjects with ASD from a psychiatrically referred sample with and without ASD. Two subscales were created from the CBCL by summing the anxiety/depression, aggression, and attention scales (severe dysregulation: sum of T scores ≥ 210 ; deficient emotional self-regulation: sum of T scores of 180–210).²⁰

Psychosocial functioning was assessed by using the Social Adjustment Inventory for Children and Adolescents (SAICA).²¹ Using methodology recommended by Reynolds²² and used previously by this team,²³ we identified children who were socially disabled on the basis of the discrepancy between the expected SAICA scaled score (derived from the estimated Full Scale IQ) and the actual SAICA scaled score. We first converted the estimated Full Scale IQ and SAICA scores to the Z scores Z_{IQ} and Z_S . We then estimated the expected SAICA score, Z_{ES} , by the regression equation $Z_{ES} = r_{IQS} \times Z_{IQ}$, where r_{IQS} is the correlation between the IQ and SAICA scores. We used the value from our control sample ($r = 0.25$, $P < .05$). We then calculated the discrepancy score as $Z_{ES} - Z_S$ and its SD, $\sqrt{(1 - r^2_{IQS})}$. We defined as socially disabled any subject who had a value > 1.65 on the standardized discrepancy score, $Z_{ES} - Z_S / \sqrt{(1 - r^2_{IQS})}$.

Family functioning was assessed by using the Moos Family Environment Scale.²⁴ Mothers provided information regarding their child's history of school problems (ie, grade retention, special placements, remedial assistance) and treatment history (ie, counseling, medication, hospitalization). For analytic purposes, we treated this information as follows: "counseling" classified those who had received any type of psychosocial treatment for their ADHD; "counseling + medication" classified those who had received any type of psychosocial treatment for their ADHD as well as any medication treatment for their ADHD; and others were classified as "no treatment." Mothers also provided information regarding their history of pregnancy, delivery, and their child's infancy.

Intellectual functioning was assessed through the vocabulary, block design, digit span, digit symbol, digit coding, and arithmetic subtests of the Wechsler

Intelligence Scale for Children—Revised (WISC-R)²⁵ and the perseverative errors subtest of the Wisconsin Card Sorting Test.²⁶ Using procedures suggested by Sattler,²⁷ we estimated Full Scale IQ from the block design and vocabulary subtests of the WISC-R by using age-corrected scaled scores. We computed the Freedom From Distractibility IQ by using the digit span, digit coding, and oral arithmetic subscales of the WISC-R. Reading and arithmetic achievement was assessed by using subtests of the Wide Range Achievement Test—Revised.²⁸

Statistical Analysis

We used *t* tests, Pearson's χ^2 test, Wilcoxon rank-sum test, and analysis of variance as indicated. We controlled for any demographic confounder that reached significance at an α level of .05 (ie, age, SES). Logistic and linear regression was used in the adjusted analyses. Given the many statistical tests computed, the .01 α level was used to assert statistical significance for omnibus comparisons. All tests were 2-tailed. Bonferroni corrections were used to control for chance findings in pairwise comparisons.

RESULTS

Because 53 participants did not have CBCL information, our final sample included 227 controls and 242 ADHD subjects. More ADHD than control participants had a positive AT profile (44 [18.18%] vs 2 [0.87%]; Fisher's exact test, $P < .001$). Because there were only 2 control subjects with an AT profile, we did not include these in analyses. Comparisons were made between ADHD subjects with (ADHD + CBCL-AT, $n = 44$) and without (ADHD, $n = 198$) a CBCL-AT profile and controls without ADHD or the CBCL-AT profile (controls, $n = 227$).

Sociodemographic Characteristics

ADHD + CBCL-AT participants were slightly younger than control subjects and were of a more disadvantageous SES status, scoring an average of 2.18 on the Hollingshead measure of SES, than ADHD participants (1.8 on the Hollingshead) and controls (1.6) (Table 1). Therefore, all subsequent analyses controlled for age and family SES.

Clinical Correlates of ADHD

ADHD + CBCL-AT and ADHD participants had similar ages of ADHD onset, similar proportions of ADHD-associated level of impairment (ie, classified as "mild" and "moderate or severe" as defined by the structured interview procedure and relating to the impairment caused by ADHD in multiple areas of functioning as perceived by the child, or the mother, if an indirect interview was conducted), similar rates of ADHD symptoms, similar proportions of any medication treatment for ADHD, similar family impairments (ie, expression, conflicts, cohesion as measured by the Moos Family Environment Scale), and similar school functioning (eg, tutoring, repeated grades) (all $P > .01$). ADHD + CBCL-AT participants had increased rates of placement in a special class (2.2% controls; 22.8% ADHD; 50% ADHD + CBCL-AT; $\chi^2_{[4]} = 81.1$, $P < .001$) (Table 1). ADHD + CBCL-AT participants experienced more family conflict as measured by the Moos Family Environment Scale than control subjects (ADHD + CBCL-AT: 61.5 ± 11.2 ; ADHD: 56.9 ± 12.3 ; $F_{[4, 257]} = 10.9$; $P < .001$; Table 1). There were no differences between the ADHD + CBCL-AT and ADHD groups in individual DSM-III-R ADHD symptoms (Fig 1A), but ADHD + CBCL-AT participants had higher rates of additional ADHD-related symptoms captured during the structured interview, including "clumsiness" (odds ratio [OR]: 2.9, $P = .02$), "an illness equal in part inattention and hyperactivity" (OR: 3.7,

TABLE 1 Sociodemographic and Clinical Characteristics

Characteristic	Controls (<i>n</i> = 227)	ADHD (<i>n</i> = 198)	ADHD + CBCL-AT (<i>n</i> = 44)	Test Statistic	<i>P</i>
Age [age range], y	11.9 ± 3.4 [6–18]	11.0 ± 3.14 ^a [6–18]	9.8 ± 2.7 ^{a,b} [6–18]	$F_{(2466)} = 9.8$	<.001
Male	117 (52)	107 (54)	26 (59)	$\chi^2_{(2)} = 0.9$.6
White	213 (94)	193 (97)	44 (100)	$\chi^2_{(2)} = 5.7$.06
SES	1.59 ± 0.74	1.8 ± 0.95	2.18 ± 1 ^{a,b}	$\chi^2_{(2)} = 13.1$.001
Intactness of family (divorced/separated)	40 (18)	50 (25)	14 (32) ^a	$\chi^2_{(2)} = 6.2$.045
IQ	114.2 ± 11.6	108.5 ± 12.6 ^a	102 ± 14.9 ^{a,b}	$F_{(2, 466)} = 23.2, \chi^2_{(2)} = 6.2$	<.001, .045
ADHD characteristics					
Age of onset	–	2.9 ± 2.4	3.0 ± 1.8	$F_{(3, 238)} = 1.90, t = -0.3$.7, .97
ADHD impairment					
Moderate or severe ^c	–	185 (94)	43 (98)	$\chi^2_{(3)} = 6.7$, Fisher's exact test	.083
Mean no. of symptoms	–	11 ± 2	12 ± 2	$F_{(3, 238)} = 0.8, t = -2.1$.5, .04
Treatment history					
Medication (any treatment)	0 (0)	546 (278) ^a	10 (23) ^a	Fisher's exact test	.04
Counseling + medication	0 (0)	91 (46) ^a	298 (664) ^{a,b}	Fisher's exact test	<.001
School functioning					
Tutoring	52 (23)	122 (62) ^a	24 (55) ^a	$\chi^2_{(42)} = 69.768.8$	<.001
Placement in special class	5 (2)	45 (23) ^a	22 (50) ^{a,b}	$\chi^2_{(42)} = 79.281.1$	<.001
Repeated grade	17 (7)	53 (27) ^a	8 (18) ^a	$\chi^2_{(42)} = 31.328.4$	<.001
Family functioning (FES)					
Expression	50.2 ± 14.6	47.8 ± 14.0	42.5 ± 14.0 ^{a,b}	$F_{(24, 4579)} = 4.45.4$.0025
Conflict	50.6 ± 12.5	56.9 ± 12.3 ^a	61.5 ± 11.2 ^{a,b}	$F_{(42, 45760)} = 10.921.3$	<.001
Cohesion	54.2 ± 16.6	44.2 ± 20.9 ^a	39.4 ± 21.6 ^a	$F_{(42, 45760)} = 13.019.8$	<.001

Data are presented as mean ± SD or *n* (%). FES, Family Environment Scale.

^a Compared with the control group.

^b Compared with the ADHD group.

^c Severity reflected the number of symptoms endorsed and the associated difficulties with them at home, in school, and in the social context.

$P = .03$), “hyperactivity” (OR: 8.9, $P = .04$), “fights with peers” (OR: 6.8, $P = .002$), and “rejection by peers” (OR: 24, $P = .003$) (Fig 1B).

Pregnancy, Delivery, and Infancy Complications

ADHD + CBCL-AT mothers reported more infections during pregnancy (ADHD + CBCL-AT: 28%; ADHD: 11%; controls: 8%; $\chi^2_{(4)} = 10.7$, $P = .001$), switching formulas during their children's infancies (ADHD + CBCL-AT: 25%; ADHD: 9%; controls: 6%; $\chi^2_{(4)} = 13.6$, $P < .001$), and described their infants as “stiffened” during infancy (ADHD + CBCL-AT: 25%; ADHD: 5%; controls: 1%; $\chi^2_{(4)} = 32$, $P < .001$) (Table 2).

Patterns of Psychiatric Comorbidity

ADHD participants with and without ATs had significantly higher prevalence of all comorbid psychiatric disorders versus control subjects (ie, disruptive behavior disorders, $\chi^2 = 142.4$, $P <$

.001; mood disorders, $\chi^2 = 132.5$, $P < .001$; multiple anxiety disorders, $\chi^2 = 63.9$, $P < .001$; language disorders, $\chi^2 = 25.5$, $P < .001$; elimination disorders, $\chi^2 = 40.5$, $P < .001$; and substance use disorders, $\chi^2 = 20.9$, $P < .001$). Compared with ADHD participants, ADHD + CBCL-AT participants had a significantly higher prevalence of disruptive behaviors (OR: 3.7, $P = .001$), mood disorders (OR: 5.4, $P < .001$), multiple anxiety disorders (>2) (OR: 3.7, $P < .001$), and language disorders (OR: 2.6, $P = .01$) (Fig 2A).

ADHD participants with and without ATs had significantly more impaired scores on each CBCL clinical and composite scale compared with controls (all $P < .001$). ADHD + CBCL-AT participants had significantly more impaired scores on all CBCL clinical and composite scales than ADHD participants, including scales that were not used to define ATs (all $P < .001$) (Fig 2B).

Deficits in Emotion Regulation and Social Disability

ADHD + CBCL-AT participants had a significantly higher prevalence of the CBCL–severe emotional dysregulation profile than both ADHD and control participants (controls: 0%; ADHD: 6.57%; ADHD + CBCL-AT: 72.73% [all $P < .001$]). In contrast, ADHD and ADHD + CBCL-AT participants did not differ from each other in the prevalence of the CBCL–deficient emotional self-regulation profile, which assesses a lower level of emotional dysregulation compared with the CBCL–severe emotional dysregulation profile. However, both groups had a significantly higher prevalence of the CBCL–deficient emotional self-regulation profile than control subjects (controls: 1.32%; ADHD: 38.89%; ADHD + CBCL-AT: 20.45% [all $P < .001$]) (Fig 3A).

Social Functioning

Both ADHD groups had a significantly higher prevalence of social disability

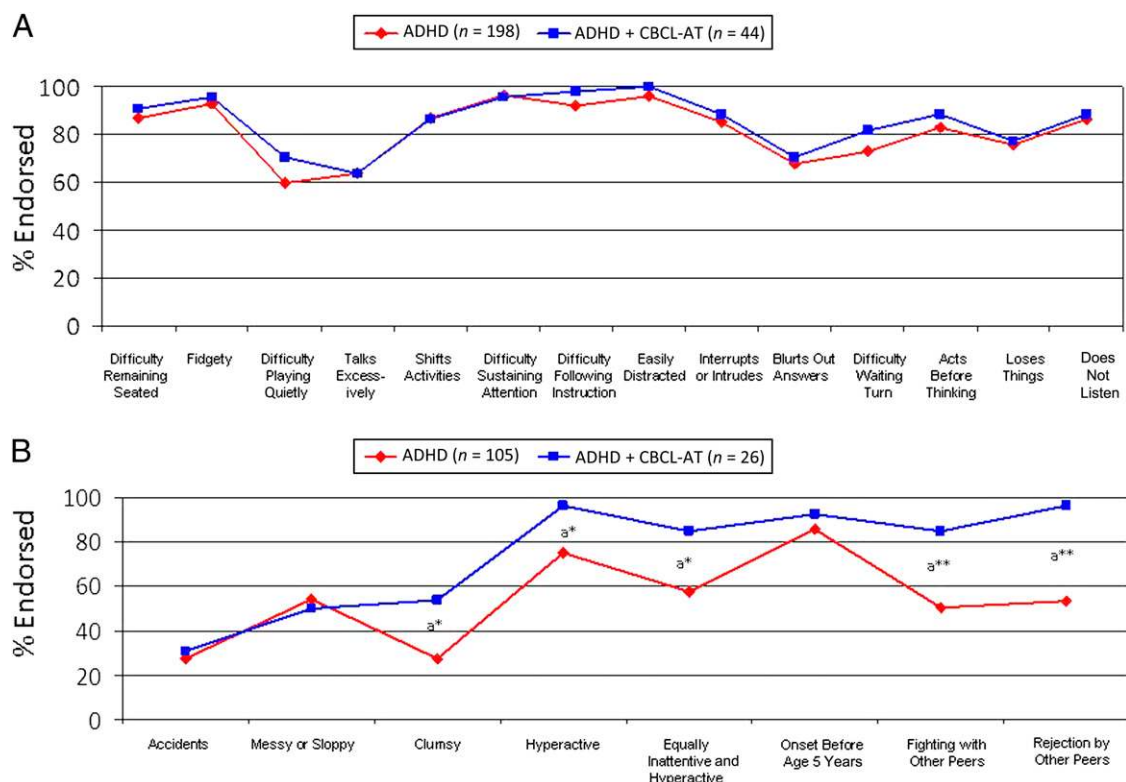


FIGURE 1

ADHD symptoms in the ADHD and ADHD + CBCL-AT groups. A, DSM-III-R symptoms. B, Additional related symptoms. ^aCompared with the ADHD group. * $P < .05$; ** $P < .005$.

TABLE 2 Pregnancy and Infancy Characteristics (Adjusting for Age and SES)

Characteristic	Controls (n = 225)	ADHD (n = 188)	ADHD + CBCL-AT (n = 40)	Test Statistic	P
Pregnancy characteristics					
Excessive nausea	25 (11)	40 (21) ^a	10 (25) ^a	$\chi^2_{(4)} = 10.4$.03
Infection	19 (8)	20 (11)	11 (28) ^{a,b}	$\chi^2_{(4)} = 10.7$.03
High blood pressure	24 (11)	39 (21) ^a	11 (28) ^a	$\chi^2_{(4)} = 14.1$.007
Accidents	2 (1)	9 (5) ^a	4 (10) ^a	$\chi^2_{(4)} = 11$.003
Family problems	20 (9)	39 (21) ^a	12 (30) ^a	$\chi^2_{(4)} = 21.2$.003
Medications	47 (21)	58 (31) ^a	18 (45) ^a	$\chi^2_{(4)} = 13.8$.008
Smoking (3 mo at gestation)	16 (7)	27 (14) ^a	9 (23) ^a	$\chi^2_{(4)} = 22.2$.002
Infancy characteristics					
Switch formulas	13 (6)	16 (9)	10 (25) ^{a,b}	$\chi^2_{(4)} = 13.6$.009
Crying infant	29 (13)	48 (25) ^a	12 (30) ^a	$\chi^2_{(4)} = 13.7$.008
Stiffened infant	2 (1)	9 (5) ^a	10 (25) ^{a,b}	$\chi^2_{(4)} = 32.0$	<.001

Data are presented as n (%).

^a Compared with the control group.

^b Compared with the ADHD group.

as defined by the SAICA (all $P < .01$) than control subjects. However, rates were significantly higher in ADHD + CBCL-AT participants versus ADHD participants (controls: 6.77%; ADHD: 34.94%; ADHD-AT: 68.57%) (Fig 3B).

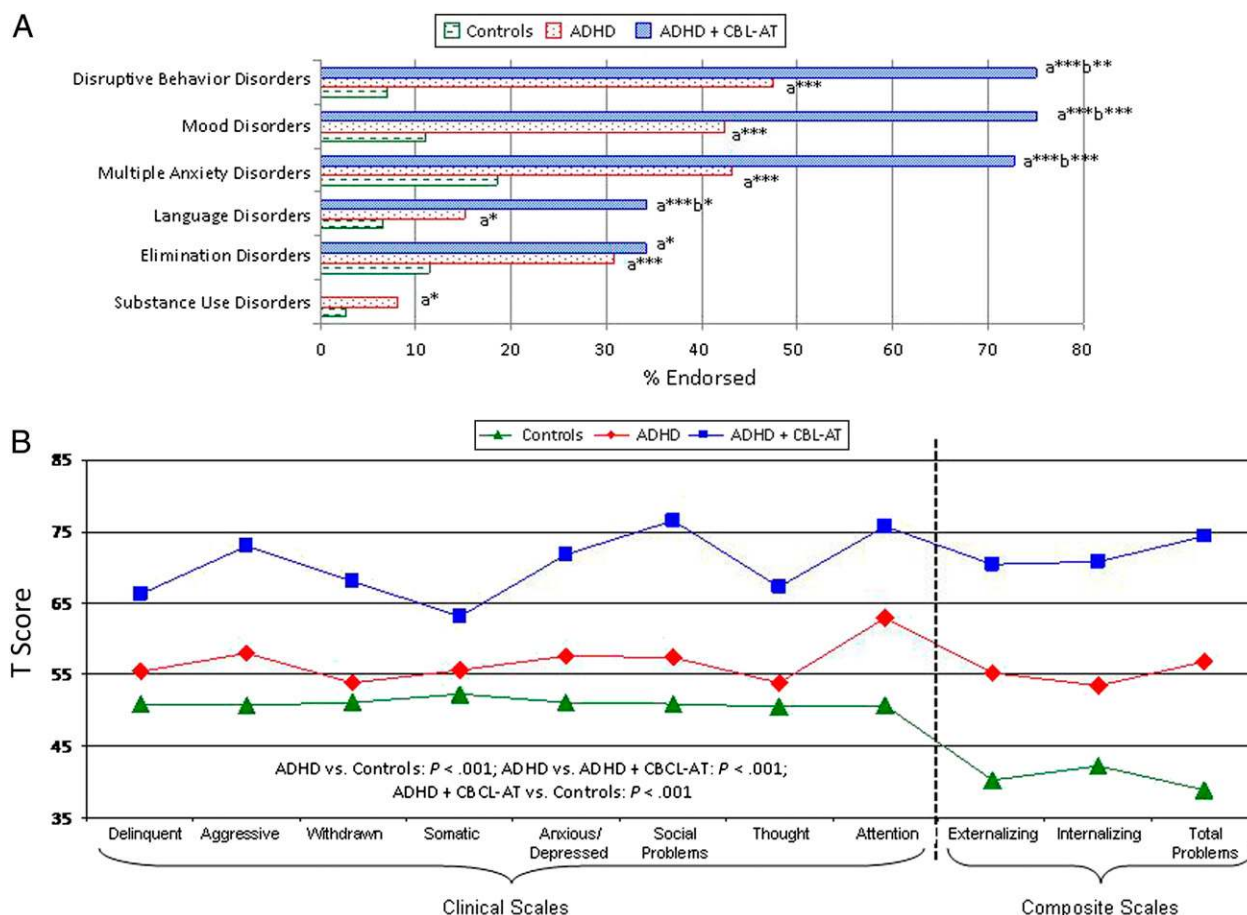
ADHD + CBCL-AT participants had significantly more impaired SAICA scaled scores than ADHD participants in mea-

sures of school behavior, spare time problems, activities and problems with peers, and problems with siblings and parents (all $P < .001$) (Fig 4A). A similar pattern was observed when analyzing findings from the CBCL social functioning scales, which consist of the activities, social, and total competence scales. Of these, only the social problems scale was used to define ATs.

ADHD + CBCL-AT participants had significantly more impaired scores than ADHD participants on the CBCL social and total competence scales (Fig 4B).

Cognitive Findings

ADHD + CBCL-AT participants scored significantly worse than ADHD participants on the WISC-R Full IQ (ADHD +

**FIGURE 2**

Additional related symptoms in the ADHD, ADHD + CBCL-AT, and control groups. A, Prevalence of psychiatric disorders (lifetime). B, CBCL profile. ^aCompared with the control group. ^bCompared with the ADHD group. * $P < .05$; ** $P < .005$; *** $P < .001$.

CBCL-AT: 101.96 ± 14.90 ; ADHD: 108.54 ± 12.55), freedom from distractibility (ADHD + CBCL-AT: 92.71 ± 16.11 ; ADHD: 100.28 ± 14.35), block design (ADHD + CBCL-AT: 10.61 ± 3.82 ; ADHD: 12.70 ± 3.48), digit symbol scaled scores (ADHD + CBCL-AT: 8.86 ± 3.99 ; ADHD: 10.57 ± 3.13) (all $P < .05$), and Wisconsin Card Sorting Test perseverative errors subtest (T scores: ADHD + CBCL-AT: 28.86 ± 16.88 ; ADHD: 17.71 ± 12.53 ; controls: 15.84 ± 10.92) (Fig 5).

DISCUSSION

Our findings revealed that ATs are present in children with ADHD, and their presence heralds a significantly more compromised clinical presentation characterized by higher rates of psychopathological, neuropsychological,

and interpersonal deficits. These results are highly consistent with those of 3 previous reports^{4,6,7} that examined ATs in children with ADHD.

The current study found that ADHD children with a positive AT profile do not differ from other ADHD children in the core symptoms of ADHD, but they do present with a more severe clinical picture when additional and ADHD-relevant symptoms in the diagnostic criteria are considered, including clumsiness, messiness, and social difficulties with peers. Because these symptoms are commonly reported in children with ASDs, it is possible that they reflect ASD tendencies as well as ADHD ones.^{29–32} Of note, the presence of clumsiness in children with ADHD and ATs may be closely related to the dis-

ordered movement kinetics observed in Asperger's syndrome^{33–36} and what has been previously described in children with the neuropsychological profile of deficits in attention, motor control, and perception.^{37–39}

Also consistent with the extant literature are our findings on the Kiddie Schedule for Affective Disorders and Schizophrenia–Epidemiologic Version showing that ADHD + AT children exhibited significantly higher rates of comorbid psychiatric disorders versus ADHD children, especially in the domain of disruptive behavior disorders, which included the diagnoses of conduct and oppositional defiant disorder.^{4,6} Also reflecting the impairment observed in children with ASDs^{40,41} is the finding that ADHD + CBCL-AT

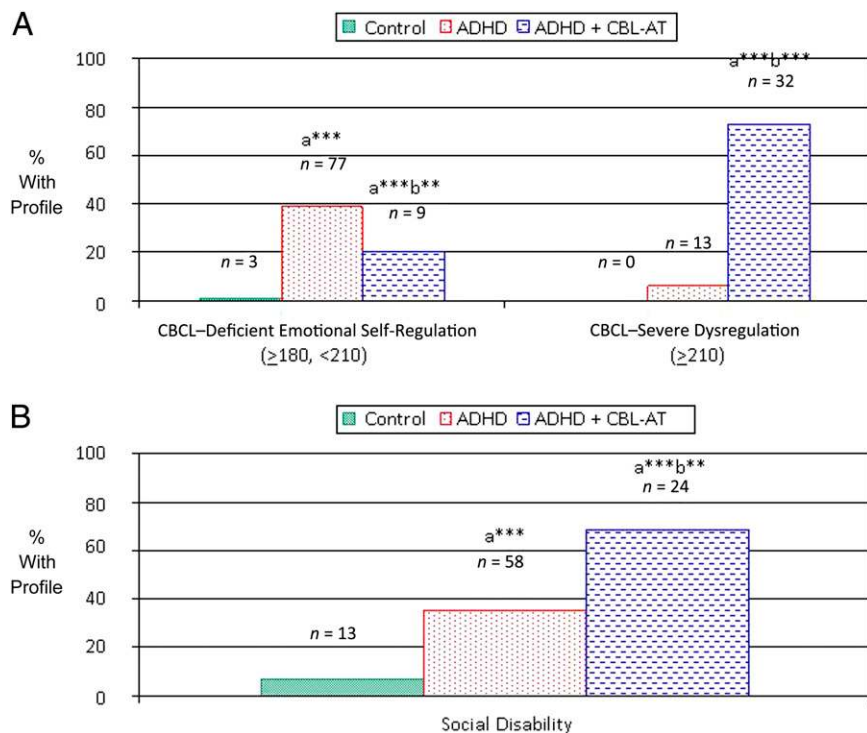


FIGURE 3

Clinical Features in the ADHD, ADHD + CBCL-AT, and control groups. A, Emotion dysregulation: CBCL AAA profile (attention + aggression + anxiety/depressed T scores). B, Social disability. ^aCompared with the control group. ^bCompared with the ADHD group. * $P < .005$; *** $P < .0001$.

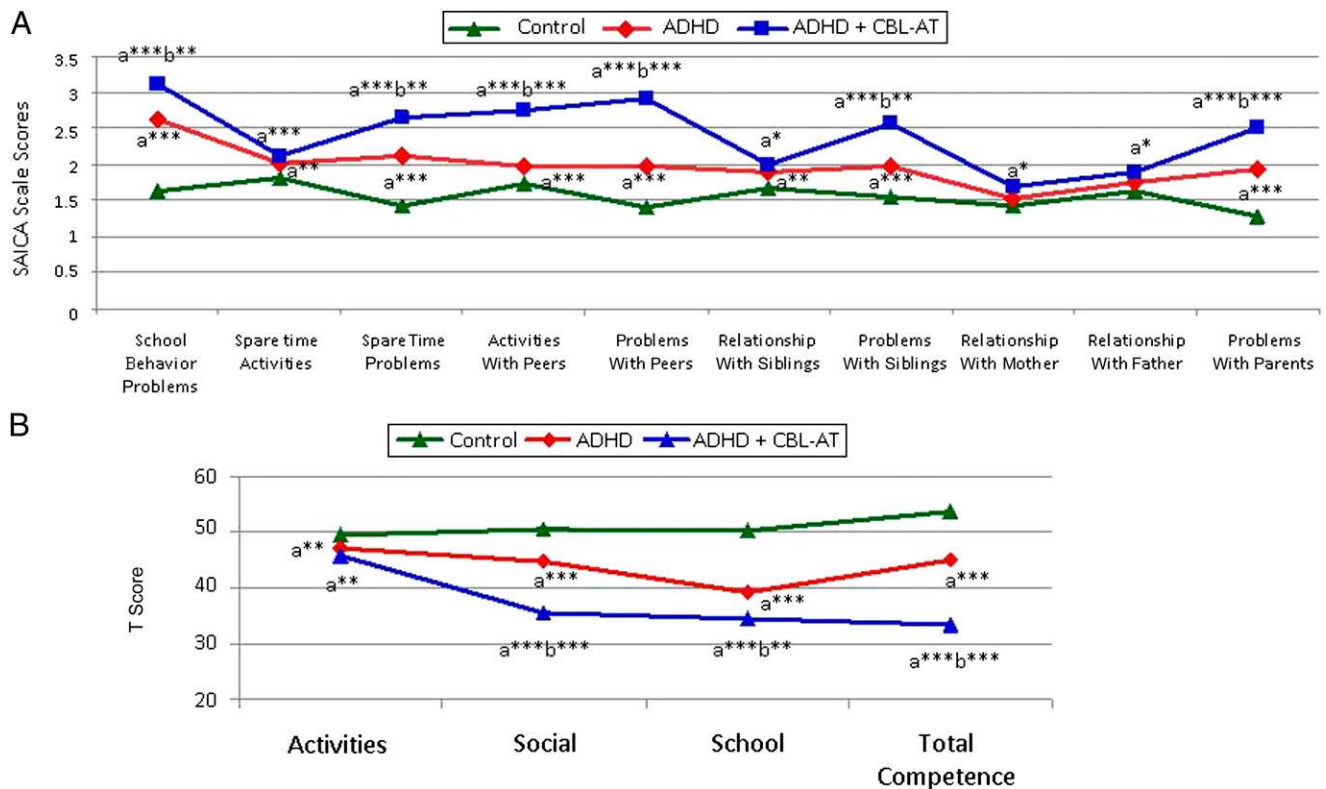
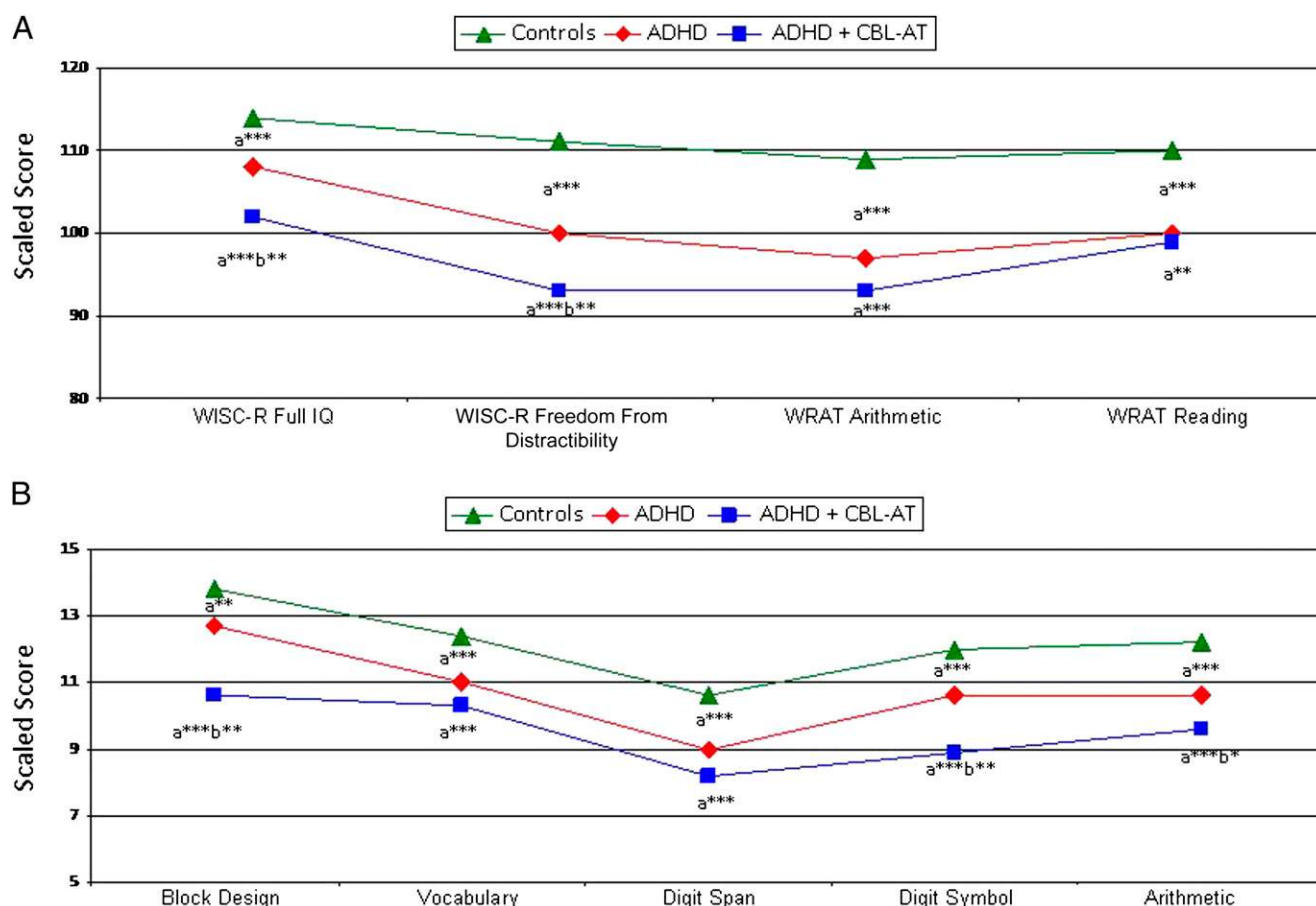


FIGURE 4

Social functioning in the ADHD, ADHD + CBCL-AT, and control groups. A, SAICA individual item scores. B, CBCL social functioning scales. ^aCompared with the control group. ^bCompared with the ADHD group. * $P < .05$; ** $P < .005$; *** $P < .0001$.

**FIGURE 5**

Neuropsychological functioning in the ADHD, ADHD + CBCL-AT, and control groups. A, WISC-R and Wide Range Achievement Test (WRAT). B, WISC-R. ^aCompared with the control group. ^bCompared with the ADHD group. * $P < .05$; ** $P < .005$; *** $P < .0001$.

children were more likely to have a positive CBCL-severe dysregulation profile versus ADHD children, indicating that these children experience very severe behavioral, emotional, and educational problems.^{20,42} The high rates of mood dysregulation in the ADHD + CBCL-AT children are also consistent with an emerging body of literature documenting high rates of mood disorders in children with ASDs.⁴³ Further research is needed to better understand the role ATs confer on emotion regulation in children with ADHD.

ADHD + CBCL-AT children also had significantly lower Full IQ, freedom from distractibility, block design, and digit symbol WISC-R scores as well as differences in perseverative errors on the Wisconsin Card Sorting Test than ADHD

children. These findings suggest impairments in executive functioning and cognitive flexibility, a pattern observed in other populations positive for ATs,^{44,45} as well as in children with ASDs.^{46–48}

Also consistent with the extant literature are our findings showing that ADHD + AT children were more likely than ADHD children to fight with and be rejected by peers, to have more school behavior problems, more difficulties utilizing their spare time, and more problems with siblings. Considering the well-established evidence that social difficulties are a core component of ASDs,^{49–51} our findings also suggest that the social disability observed in the ADHD + CBCL-AT group may be more a reflection of underlying ATs than the presence of ADHD itself. The

higher rates of mood, anxiety, disruptive, and substance use disorders,⁵² and school failure, school dropout, and delinquent offenses⁵³ in ADHD + AT children is particularly worrisome.

Our findings that ADHD children with ATs had a higher rate of pregnancy and infancy complications than other ADHD children could suggest that prenatal and perinatal complications alone or in combination with genetic risk factors could account for the development of ATs in some children with ADHD. These findings are intriguing in light of previous reports yielding support for the role of maternal infection during pregnancy^{54–57} and the behavioral characteristics of infants later classified with ASDs.⁵⁸

The ability to identify a subgroup of ADHD children with ATs may facilitate the development of more individualized clinical interventions. For instance, special care could be made to target treatment on the domain of social difficulties by focusing and expanding on the development of social skills training in addition to contingency management typically used for the psychosocial treatment of ADHD. Scientifically, this research will help inform future work targeted at identifying biomarkers for a potentially distinct subtype of ADHD. For example, twin, family, linkage, and genome-wide association studies already suggest that ADHD and ASD may share a common heritable etiology.^{1–4,59} Of note, Williams et al⁵ found that rare copy number variants identified in ADHD subjects were significantly enriched for loci implicated in autism.

Our findings need to be viewed in light of some limitations. Our sample did not contain a comparison group of children with a diagnosed ASD. Such a group would be useful to determine the degree to which our ADHD + CBCL-AT cohort exhibits features that are similar to the diagnostic class. However, because our primary diagnosis of interest was ADHD impacted by traits of autism, the absence of an autism-only control group does not detract from the finding that ADHD children with ATs exhibit more impairments than those with ADHD only. Although autism was excluded, it was done by using subject history as opposed to validated measures such as the Autism Diagnostic Observation Schedule, Social Communication Questionnaire, or the Social Responsiveness Scale, thus allowing for the possibility that some children with undiagnosed ASDs could have been included in our sample. However, con-

sidering that the mean age of our sample at baseline was 11 years, it is not very likely that children with a clear diagnosis of ASD would have remained undiagnosed. Because our measure of ATs has not been validated, it is possible that its external validity may be compromised because findings may not generalize to other samples. Finally, because our sample was referred and largely white, our findings may not generalize to community samples or other ethnic groups.

CONCLUSIONS

Despite these limitations, our work found that the CBCL-AT profile identifies a sizeable minority of ADHD children at high risk for significant morbidity and disability. More work is needed to replicate these findings in children with and without ADHD and to further examine their prognostic utility.

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