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Obesity in Men With Childhood ADHD: A 33-Year Controlled, Prospective, Follow-up Study

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KEY WORDS

ADHD, adults, BMI, longitudinal follow-up, obesity, weight

ABBREVIATIONS

ADHD—attention-deficit/hyperactivity disorder

CI—confidence interval

DSM—*Diagnostic and Statistical Manual of Mental Disorders*

OR—odds ratio

SES—socioeconomic status

Dr Cortese made substantial contributions to analysis and interpretation of data, and drafted the article; Dr Ramos Olazagasti made substantial contributions to analysis and interpretation of data, and revised the article critically; Drs Klein and Mannuzza made substantial contributions to conception and design, acquisition of data, and interpretation of data, and revised the article critically; Dr Castellanos made substantial contributions to conception and design and interpretation of data, and revised the article critically; Dr Proal made substantial contributions to interpretation of data and revised the article critically; and all authors approved the final version as submitted.

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WHAT'S KNOWN ON THIS SUBJECT: Cross-sectional studies in children and adults have reported a significant positive association between attention-deficit/hyperactivity disorder (ADHD) and obesity.



WHAT THIS STUDY ADDS: This controlled, prospective, follow-up study of boys with ADHD found significantly higher BMI and obesity rates in adulthood, compared with men without childhood ADHD, regardless of socioeconomic status and other lifetime mental disorders.

abstract

FREE

OBJECTIVE: To compare BMI and obesity rates in fully grown men with and without childhood attention-deficit/hyperactivity disorder (ADHD). We predicted higher BMI and obesity rates in: (1) men with, versus men without, childhood ADHD; (2) men with persistent, versus men with remitted, ADHD; and (3) men with persistent or remitted ADHD versus those without childhood ADHD.

METHODS: Men with childhood ADHD were from a cohort of 207 white boys (referred at a mean age of 8.3 years), interviewed blindly at mean ages 18 (FU18), 25 (FU25), and 41 years (FU41). At FU18, 178 boys without ADHD were recruited. At FU41, 111 men with childhood ADHD and 111 men without childhood ADHD self-reported their weight and height.

RESULTS: Men with childhood ADHD had significantly higher BMI (30.1 ± 6.3 vs 27.6 ± 3.9 ; $P = .001$) and obesity rates (41.4% vs 21.6%; $P = .001$) than men without childhood ADHD. Group differences remained significant after adjustment for socioeconomic status and lifetime mental disorders. Men with persistent ($n = 24$) and remitted ($n = 87$) ADHD did not differ significantly in BMI or obesity rates. Even after adjustment, men with remitted (but not persistent) ADHD had significantly higher BMI (B: 2.86 [95% CI: 1.22 to 4.50]) and obesity rates (odds ratio: 2.99 [95% CI: 1.55 to 5.77]) than those without childhood ADHD.

CONCLUSIONS: Children with ADHD are at increased risk of obesity as adults. Findings of elevated BMI and obesity rates in men with remitted ADHD require replication. *Pediatrics* 2013;131:e1731–e1738

Elevated rates of obesity have been reported in children with attention-deficit/hyperactivity disorder (ADHD).¹ Two studies^{2,3} obtained similar results in adults; they relied on retrospective reports of childhood ADHD, however, which may have limited their validity.⁴ A prospective study⁵ reported significantly higher BMI in men with ADHD diagnosed in childhood relative to non-ADHD comparisons (mean age: 32 years). Another prospective study found that at a mean age of 27 years, adults with persistent ADHD, but not those with remitted ADHD, had significantly higher BMI than community comparisons; the 2 ADHD groups did not differ significantly.⁶ To the best of our knowledge, no study has reported obesity rates in adults (beyond the final growth period) diagnosed with ADHD in childhood.

We present data on BMI and obesity in adulthood from a prospective follow-up of 6- to 12-year-old boys with ADHD and boys without childhood ADHD. Our first objective was to examine BMI and obesity rates in men with and without childhood ADHD after taking into account variables potentially associated with obesity, ie, socioeconomic status [SES]^{7,8} and lifetime mood, anxiety, and substance use disorders.⁹ Consistent with the literature,^{2,3,5,10–16} we hypothesized that there would be significantly higher BMI and obesity rates in men with childhood ADHD versus those without childhood ADHD, even after adjusting for SES and lifetime mental disorders. We also hypothesized that BMI and obesity rates would be significantly greater in men with persistent ADHD (“persistent-ADHD”) than in those with remitted ADHD (“remitted-ADHD”) and in both ADHD subgroups compared with men without childhood ADHD.

METHODS

Participants

Participants with childhood ADHD originally comprised 207 children ages

6 to 12 years; they were middle class, medically healthy, white boys, referred to a research clinic at a mean age of 8.3 years.^{17,18} Inclusion criteria were: referral by schools because of behavior problems, elevated hyperactivity ratings by teachers and parents, history of behavior problems, IQ ≥ 85 , and English-speaking parents. Children with aggressive or antisocial behavior were excluded to rule out comorbid conduct disorder. Participants had to have met criteria for *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (DSM-IV-[TR])¹⁹ ADHD, combined type because: (1) they were clinically impaired by inattention, and hyperactivity/impulsivity, symptoms; (2) cross-situationality was required; (3) mean ratings on the Conners' Teacher Rating Scale²⁰ items of restless/overactive, inattentive/distractible, and excitable/impulsive (rated 0–3) were high (2.8, 2.6, and 2.4, respectively); and (4) classroom observations by “blind” observers showed highly significant differences between index and “normal” children on hyperactivity, inattention, and impulsivity.²¹ Follow-up waves were conducted at mean ages of 18.4 ± 1.3 years (FU18), 25.0 ± 1.3 years (FU25), and 41.2 ± 2.7 years (FU41). White, medically healthy boys ($n = 178$) were recruited at FU18 (mean age: 18.3 ± 1.5 years) and matched for age, parental social class,²² and geographic residence with boys with ADHD. They had been seen at the same medical center for minor acute ailments or routine physical examinations and, based on chart review and parent reports, had no behavior problems in childhood. At FU41, on average 33 years after the initial diagnosis of ADHD, participants were invited to take part in a study assessing clinical outcomes²³ and brain correlates²⁴ of adults with and without childhood ADHD.

The study was approved by the institutional review board of the NYU

Langone Medical Center. Participants were informed fully of study procedures and provided signed consent.

Psychiatric Assessment

At FU41, ADHD was diagnosed by using the Assessment of Adult Attention Deficit Hyperactivity Disorder (text available online²⁵), which has been found to generate reliable and valid clinical diagnoses.²⁶ In addition, the Structured Clinical Interview for DSM-IV Axis I Disorders–Non-Patient Edition²⁷ and the Psychiatric Research Interview for Substance and Mental Disorders, Version 6.0,²⁸ were administered by clinicians who were blind to childhood data as well as to all other data. Disorders were defined as “lifetime” if they occurred at any time.

Ongoing ADHD was defined as: (1) meeting DSM-IV-(TR) symptom criteria (at least 6 of 9 inattention symptoms and/or at least 6 of 9 hyperactivity/impulsivity symptoms reported as occurring “often”) during the past 6 months; (2) ADHD symptoms causing clinically significant functional impairment; and (3) symptoms not accounted for by another mental disorder.

Other Assessments

BMI was calculated from self-reported height and weight and was categorized as underweight (BMI < 18.5), normal weight ($18.5 \leq \text{BMI} < 25$), overweight ($25 \leq \text{BMI} < 30$), or obese (BMI ≥ 30).²⁹

The Hollingshead and Redlich index²² was used as a measure of SES. “Parental SES” indicates parents' SES at FU18, and “adult SES” is the participants' SES at FU41.

Data Analyses

For independent samples, *t* tests were used to contrast demographic values, height, weight, and BMI between men with and without childhood ADHD. Group

differences in mental disorders and obesity rates relied on χ^2 tests. A 1-way analysis of variance tested differences across persistent-ADHD, remitted-ADHD, and men without childhood ADHD. Pairwise contrasts relied on least significant difference tests for demographic values, height, weight, and BMI or χ^2 tests for categorical variables (prevalence of mental disorders and obesity rates).

Further analyses contrasted obesity rates and BMI, adjusting for SES and lifetime mental disorders (other than ADHD). These covariates were selected a priori, based on studies reporting significant associations between obesity and SES, lifetime mood, anxiety, and substance use disorders.^{7–9} Because error variance in BMI scores differed between men with and without childhood ADHD (Levene's test: $F = 20.11$, $P < .001$), we fitted a mixed model that estimated separate residual variances for each group. Specifically, we compared men with and without childhood ADHD on BMI, adjusting for SES and lifetime mood, anxiety, and substance use disorders (alcohol, nonalcohol, and nicotine dependence). Although men with and without childhood ADHD differed in both parental SES (SES²² at FU18) and adult SES (participant SES²² at FU41) ($t_{221} = -6.95$, $P < .01$, and $t_{221} = -2.20$, $P = .02$, respectively), only parental SES was significantly, albeit modestly, correlated with BMI in adulthood ($r = 0.14$, $P = .03$). Therefore, group contrasts were adjusted for parental SES but not adult SES. The mixed model was also used to compare BMI across the 3 subgroups, adjusting for covariates.

Finally, the likelihood of having obesity was compared between men with and without childhood ADHD, and across the 3 subgroups, by using binary logistic regression. "Obesity" was predicted according to "group," adjusting for parental SES and lifetime mood, anxiety, and substance use disorders.

For sensitivity analyses, 5 men without childhood ADHD were diagnosed as meeting DSM-IV criteria for ADHD, based on blind clinical assessments using the Assessment of Adult Attention Deficit Hyperactivity Disorder, despite reports of not having behavioral problems in elementary school when originally recruited at FU18. We repeated the analyses without these 5 participants. In addition, at FU41, 5 men with persistent-ADHD were being treated with psychostimulants, which may decrease weight.³⁰ Analyses were repeated excluding these subjects. Finally, to be consistent with others' standards for diagnosing ADHD in adults,^{5,31} we reanalyzed the data by using a threshold of 4 of 9 ADHD symptoms, instead of 6 of 9.

Mixed model analyses were conducted in SAS version 9.2 by using PROC MIXED. All other analyses were conducted by using SPSS version 16.0 (IBM SPSS Statistics, IBM Corporation, Armonk, New York). Significance was defined as $P \leq .05$ (2-tailed).

RESULTS

Sample

Of 385 men (207 with childhood ADHD, 178 without), 114 (29.6%) were lost to FU41 (72 [34.8%] with childhood ADHD and 42 [23.6%] without). Twenty-one men with and 20 men without childhood ADHD were not located; 13 men with and 15 men without childhood ADHD declined to participate; 11 men with and 2 without childhood ADHD were deceased; interviews were denied in 4 men with and in 1 without childhood ADHD who were incarcerated; and grant support ended before evaluation of 23 men with and 4 men without childhood ADHD. We initiated the inquiry about weight and height after the first 24 men with and 25 men without childhood ADHD had been evaluated. As a result, self-reported height and weight were provided by 111 of 135 men with

childhood ADHD (82.2% of those at FU41), and 111 of 136 (81.6%) men without childhood ADHD. Participants with and without height/weight data did not differ significantly in age ($P = .58$) or SES ($P = .50$). Men with childhood ADHD assessed or lost at FU41 did not differ significantly, at FU25, in prevalence of ADHD, any substance use disorder, any mood disorder, any anxiety disorder, and any DSM-III-R disorder except ADHD at FU25 (all: $P > .10$). Similarly, men without childhood ADHD assessed or lost at FU41 did not differ significantly, at FU25, in rates of ADHD, any mood disorder, and any anxiety disorder (all: $P > .10$). At FU25, 8% of the men without childhood ADHD, versus 19% of those lost to follow-up at FU41, had been diagnosed as having a substance use disorder ($P = .07$). In addition, 10% of the men without childhood ADHD, versus 22% of those lost to follow-up at FU41, had been diagnosed with a mental disorder other than ADHD at FU25 ($P = .08$).²³ Therefore, men without childhood ADHD assessed or lost at follow-up did not differ significantly in previously assessed mental disorders.

Demographic and Clinical Characteristics at FU41

At FU41, mean age did not differ significantly between men with and without childhood ADHD ($P = .62$) (Table 1). Men without childhood ADHD had significantly higher previous parental, and current, SES ($P = .03$ and $P < .001$, respectively). The rates of lifetime mood and anxiety disorders did not differ significantly between groups, but lifetime rates of nonalcohol substance use disorders and nicotine dependence were significantly higher in men with childhood ADHD than men without ($P = .02$ and $P < .001$, respectively).

Eight men with persistent-ADHD presented with ADHD combined type, 8 with inattentive type, and 8 with hyperactive-impulsive type. The remitted-ADHD

TABLE 1 Characteristics of the Sample in Relation to Childhood and Persistent ADHD

Characteristic	Men Without Childhood ADHD (all) (n = 111)	Men With Childhood ADHD (all) (n = 111)	P (2-tailed)	Men With Childhood ADHD		Group Differences Across Persistent-ADHD, Remitted-ADHD, and Men Without Childhood ADHD (P [2-tailed])	Persistent-ADHD Versus Men Without Childhood ADHD (P [2-tailed])	Remitted-ADHD Versus Men Without Childhood ADHD (P [2-tailed])
				Persistent ADHD (n = 24)	Remitted ADHD (n = 87)			
Demographic								
Age, y	41.6 ± 3.2	41.3 ± 3.0	.62	41.3 ± 2.8	41.3 ± 3.1	.89		
Mean parental SES (at FU18) ^a	2.8 ± 1.1	3.1 ± 1.0	.03	2.8 ± 1.1	3.2 ± 1.0	.03		.01
Mean SES (at FU41) ^a	2.4 ± 1.1	3.4 ± 1.0	<.001	3.7 ± 1.1	3.3 ± 1.0	<.001	<.001	<.001
Lifetime mental disorders								
Any mood disorder	45 (40.5)	47 (42.3)	.78	14 (58.3)	33 (37.9)	.19		
Any anxiety disorder	19 (17.1)	20 (18.0)	.86	6 (25.0)	14 (16.1)	.59		
Any alcohol-related disorder	39 (35.1)	47 (42.3)	.33	9 (37.5)	38 (43.7)	.46		
Any nonalcohol substance use disorder	37 (33.3)	61 (55.0)	.02	16 (66.7)	45 (51.7)	.02	.02	
Nicotine dependence	27 (24.3)	55 (49.5)	<.001	16 (66.7)	39 (44.8)	<.001	<.001	.002
Anthropometric values								
Height, in	70.1 ± 2.9	70.3 ± 2.9	.64	69.7 ± 3.3	70.5 ± 2.8	.40		<.001
Weight, lb	193.8 ± 34.9	212.6 ± 49.7	.001	199.5 ± 37.8	216.1 ± 52.1	.001		<.001
BMI	27.6 ± 3.9	30.1 ± 6.3	<.001	28.9 ± 4.8	30.4 ± 6.6	.001		<.001
Weight status			.0096			.02		
Underweight	0 (0)	1 (0.9)	.99	0 (0)	1 (1.1)	.46		
Normal	28 (25.2)	20 (18.0)	.25	4 (16.7)	16 (18.4)	.40		
Overweight	59 (53.2)	44 (39.9)	.59	13 (54.2)	31 (35.6)	.04		.02
Obesity	24 (21.6)	46 (41.4)	.002	7 (29.2)	39 (44.8)	.003		.001

Persistent-ADHD and remitted-ADHD did not differ significantly in demographic values, rates of lifetime mental disorders, or anthropometric values. Data are presented as mean ± SD or n (%).

^a Hollingshead and Redlich index. Range = 1 (high SES); FU18, follow-up at mean age 18; FU41, follow-up at mean age 41.

group had significantly lower parental SES than men without childhood ADHD ($P = .01$). Both ADHD subgroups had significantly lower adult SES than men without childhood ADHD (all: $P < 0.001$); persistent-ADHD and remitted-ADHD did not differ significantly in adult SES ($P = .15$) or in rates of mental disorders.

BMI and Obesity Rates in Men With and Without Childhood ADHD

Compared with men without childhood ADHD, those with childhood ADHD had significantly greater weight (212.6 ± 49.7 vs 193.8 ± 34.9 pounds; $P = .001$), BMI (30.1 ± 6.3 vs 27.6 ± 3.9 ; $P < .001$), and rates of obesity (41.4% vs 21.6% ; $P = .002$). Groups did not differ significantly in height ($P = .64$) (Table 1).

Group differences remained after adjustment for parental SES and lifetime mental disorders. BMI was still significantly elevated in men with childhood ADHD (unadjusted model, B: 2.56 [95% confidence interval (CI): 1.18 to 3.95]; $P < .001$; adjusted model, B: 2.47 [95% CI: 1.02 to 3.92]; $P = .001$) (Table 2), who were also significantly more likely to have obesity (unadjusted model, odds ratio [OR]: 2.57 [95% CI: 1.42 to 4.62]; adjusted model, odds ratio: 2.62 [95% CI: 1.39 to 4.93]; $P = .003$) (Table 3).

BMI and Obesity Rates in Persistent-ADHD, Remitted-ADHD, and Men Without Childhood ADHD

The 3 groups differed significantly in weight ($P = .001$) and BMI ($P = .001$) but not in height ($P = .40$) (Table 1). Mean BMI in persistent-ADHD and remitted-ADHD was 28.9 ± 4.8 and 30.4 ± 6.6 , respectively. The hypothesis that persistent-ADHD would have greater BMI than remitted-ADHD, and greater than men without childhood ADHD, was not supported ($P = .27$, and $P = .18$, respectively). Only remitted-ADHD had significantly higher BMI than men without childhood ADHD (30.4 ± 6.6 vs 27.6 ± 3.9 ; $P < .001$). The average difference

TABLE 2 BMI at 41 Years in Relation to Childhood and Persistent ADHD

Predictor Variable	Unadjusted Model		Adjusted Model		Predictor Variable	Unadjusted Model		Adjusted Model	
	B	95% CI	B	95% CI		B	95% CI	B	95% CI
Childhood ADHD	2.56	1.18 to 3.95	2.47	1.02 to 3.92	Persistent ADHD	1.31	−0.78 to 3.40	0.82	−1.27 to 2.91
					Remitted ADHD	2.91	1.33 to 4.49	2.86	1.22 to 4.50
Parental SES			0.44	−0.15 to 1.02				0.41	−0.16 to 0.99
Any mood disorder			0.94	−0.38 to 2.26				0.88	−0.42 to 2.19
Any anxiety disorder			0.68	−0.99 to 2.35				0.78	−0.84 to 2.40
Any alcohol-related disorder			−1.39	−2.85 to 0.06				−1.62	−3.04 to −0.19
Any nonalcohol substance use disorder			−0.61	−2.03 to 0.81				−0.56	−1.97 to 0.85
Nicotine dependence			0.81	−0.71 to 2.34				1.14	−0.37 to 2.65

TABLE 3 Obesity at 41 Years in Relation to Childhood and Persistent ADHD

Predictor Variable	Unadjusted Model		Adjusted Model		Predictor Variable	Unadjusted Model		Adjusted Model	
	OR	95% CI	OR	95% CI		OR	95% CI	OR	95% CI
Childhood ADHD	2.57	1.42 to 4.62	2.62	1.39 to 4.93	Persistent ADHD	1.49	0.56 to 4.02	1.38	0.47 to 4.09
					Remitted ADHD	2.95	1.59 to 5.47	2.99	1.55 to 5.77
Parental SES			1.36	1.01 to 1.83				1.34	0.99 to 1.81
Any mood disorder			1.21	0.65 to 2.24				1.26	0.68 to 2.36
Any anxiety disorder			1.82	0.86 to 3.86				1.91	0.90 to 4.08
Any alcohol-related disorder			0.69	0.34 to 1.41				0.63	0.30 to 1.31
Any nonalcohol substance use disorder			0.82	0.41 to 1.63				0.86	0.43 to 1.73
Nicotine dependence			0.97	0.47 to 1.97				1.07	0.52 to 2.21

in BMI was 1.5 between persistent-ADHD and remitted-ADHD, 1.3 between persistent-ADHD and men without childhood ADHD, and 2.8 between remitted-ADHD and men without childhood ADHD. For a man measuring 70 inches tall (the average height in this sample), a BMI difference of 2.8 equals 19.5 pounds.

As with BMI, obesity was not elevated in persistent-ADHD relative to remitted-ADHD (29.2% vs 44.8%) and men without childhood ADHD (21.6%) ($P = .24$ and $.43$). However, the obesity rate was significantly higher in remitted-ADHD than in men without childhood ADHD ($P = .001$) (Table 1).

These differences remained significant after adjusting for SES and lifetime mental disorders. BMI did not differ significantly between ADHD subgroups. Relative to men without childhood ADHD, persistent-ADHD did not have significantly higher BMI (unadjusted model, B: 1.31 [95% CI: −0.78 to 3.40]; $P = .22$; adjusted model, B: 0.82 [95% CI: −1.27 to

2.91]; $P = .44$), but remitted-ADHD did (unadjusted model, B: 2.91 [95% CI: 1.33 to 4.49]; $P < .001$; adjusted model, B: 2.86 [95% CI: 1.22 to 4.50]; $P < .001$) (Table 2). The persistent-ADHD men did not differ significantly in obesity rates from men without childhood ADHD (unadjusted model, OR: 1.49 [95% CI: 0.56 to 4.02]; $P = .77$; adjusted model, OR: 1.38 [95% CI: 0.47 to 4.09, $P = .66$). In contrast, the remitted-ADHD group was significantly more likely to be obese than the group of men without childhood ADHD (unadjusted model, OR: 2.95 [95% CI: 1.59 to 5.47]; $P = .008$; adjusted model, OR: 2.99 [95% CI: 1.55 to 5.77]; $P = .008$) (Table 3). Persistent-ADHD and remitted-ADHD did not differ significantly in obesity rates (unadjusted model, OR: 1.49 [95% CI: 0.56 to 4.02]; adjusted model, OR: 1.38 [95% CI: 0.47 to 4.09]).

Sensitivity Analyses

Results were unchanged when the 5 men without childhood ADHD, but with

current ADHD, were excluded (data available on request). Excluding 5 persistent-ADHD subjects receiving stimulant treatment at FU41 yielded mean BMI values of 29.0 ± 4.2 (vs 28.9 ± 4.8) and did not alter differences in BMI and obesity rates across the 3 subgroups. Results were also unchanged when we applied a threshold of 4 of 9 ADHD symptoms, instead of 6 of 9, to define ongoing ADHD (data available on request). There were no outliers (BMI > 3 SDs beyond the mean) among men with childhood ADHD. Group differences were unaffected by removing 2 outliers among men without childhood ADHD (data not shown).

DISCUSSION

In this controlled, prospective longitudinal study, childhood ADHD in boys predicted significantly higher BMI, and a twofold increase in obesity rates in adulthood, at a mean age of 41 years relative to men without childhood ADHD. Group differences remained

significant after adjustment for SES and lifetime mental disorders. Unlike men with persistent-ADHD, who did not differ significantly from men without childhood ADHD, those with remitted-ADHD had significantly higher BMI and obesity rates than men without childhood ADHD, even after controlling for SES and lifetime mental disorders.

Elevated obesity in adults with a childhood diagnosis of ADHD extend findings of increased obesity in adults with retrospectively diagnosed ADHD.^{2,3} Results also confirm that anxiety or depressive disorders are unlikely to explain the association between childhood ADHD and obesity in adulthood.^{2,16} In addition, lifetime substance use disorders, which have not been taken into consideration in previous studies, did not affect the relationship between childhood ADHD and obesity in adulthood.

At ages well beyond the potential for further growth, men with and without childhood ADHD did not differ significantly in height. This finding extends a previous report in the same cohort, which found no significant height differences at FU18, despite the finding that methylphenidate treatment had reduced growth velocity in childhood.³²

Our hypothesis that men with persistent ADHD would have significantly higher BMIs and obesity rates than their counterparts, who no longer had ADHD, was not confirmed. This finding is in contrast to that of Barkley et al,⁶ who reported significantly higher BMI in adults with persistent ADHD (at mean age 27 years), but not remitted ADHD, relative to community comparisons (obesity rates were not indicated); as in our study, individuals with persistent and remitted ADHD did not differ significantly. Thus, this result must be interpreted tentatively pending replication.

Two psychological hypotheses have been proposed to explain a possible link between ADHD and obesity.¹ First, deficient inhibitory control and delay aversion, expressions of the impulsivity intrinsic to ADHD, may foster poor planning and difficulty in monitoring eating behaviors, leading to abnormal eating patterns and consequent obesity.^{33,34} In addition, ADHD-related inattention and deficits in executive functions may produce difficulties in adherence to regular eating patterns, leading to abnormal eating behaviors.¹ The finding of significantly higher BMI and obesity rates in men with childhood ADHD, compared with those without, suggests that childhood ADHD is a risk factor for obesity in adulthood, regardless of adult ADHD status.

Regarding neurobiological correlates, dysfunction of fronto-striatal dopaminergic pathways has been implicated in ADHD³⁵ as well as in obesity.³⁶ These neuronal circuits underpin impulse control, executive functions, and reward sensitivity. Limited preliminary evidence in a few illustrative individuals with both ADHD and obesity suggests genetic alterations in chromosome regions encompassing genes possibly involved in dopaminergic regulation pathways³⁷ and related systems, such as melanocortin,^{38,39} which are also implicated in both ADHD and obesity.³⁶

This is the first controlled, prospective, follow-up study of obesity in men with a diagnosis of ADHD established in childhood that includes individuals who all reached their maximal height. Findings of elevated obesity rates in those with childhood ADHD are not likely due to an unusually low obesity rate in comparisons (ie, 21.4%), as the obesity rate in New York state among white adult males in 2006–2008, a period overlapping with that of the study, was reported to be ~24%.⁴⁰

We note several limitations. Weight and height were not obtained for the entire cohort, but individuals with and without weight and height data did not differ significantly in parental SES, which was significantly associated with BMI at follow-up. Height and weight values relied on self-report. However, a study of a large representative sample of adults in the United States⁴¹ found that self-reported and measured weight did not differ significantly among men ages 40 to 49 years (72% of our sample) and differed slightly (<1 pound) among men ages 30 to 39 years (28% of our sample), thus suggesting that bias in self-reported weight is unlikely to be an influential factor. Complete data on height and weight of participants at FU18 were not available because only the first one-half of the cohort was selected for measurements of height and weight at FU18. This missing data prevented us from determining whether the association between childhood ADHD and weight status at FU41 was attributable to weight status in adolescence or whether it developed later. The limited number of persistent-ADHD ($n = 24$) subjects precluded analyses of DSM-IV ADHD types. The sample was limited to white males, and results cannot be generalized to women or other ethnic/racial groups. Finally, we did not evaluate physical activity, sedentary time, or sleep problems, any of which might affect weight.

CONCLUSIONS

White boys with ADHD are at substantially elevated risk for obesity in adulthood. The long-term risk for obesity should be considered when managing children with ADHD.

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