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

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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.

**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 studies2,3 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,2,3,5,10–18 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 ≤ BMI <25), overweight (25 ≤ 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 \( \chi^2 \) 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 \( \chi^2 \) 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. 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 (SES\textsuperscript{22} at FU18) and adult SES (participant SES\textsuperscript{22} at FU41) (t\textsubscript{221} = −6.95, \( P < .01 \), and t\textsubscript{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. Analyses were repeated excluding these subjects. Finally, to be consistent with others’ standards for diagnosing ADHD in adults, 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, or 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 (\( P = .08 \)). 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

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men Without Childhood ADHD (all) (n = 111)</th>
<th>Men With Childhood ADHD (all) (n = 111)</th>
<th>P (2-tailed)</th>
<th>Men With Childhood ADHD Persistent ADHD (n = 24)</th>
<th>Remitted ADHD (n = 87)</th>
<th>Group Differences Across Persistent ADHD, Remitted-ADHD, and Men Without Childhood ADHD (P [2-tailed])</th>
<th>Persistent-ADHD Versus Men Without Childhood ADHD (P [2-tailed])</th>
<th>Remitted-ADHD Versus Men Without Childhood ADHD (P [2-tailed])</th>
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</thead>
<tbody>
<tr>
<td>Demographic</td>
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<tr>
<td>Age, y</td>
<td>41.6 ± 3.2</td>
<td>41.3 ± 3.0</td>
<td>.02</td>
<td>41.3 ± 2.8</td>
<td>41.3 ± 3.1</td>
<td>.09</td>
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</tr>
<tr>
<td>Mean parental SES (at FU18)*</td>
<td>2.8 ± 1.1</td>
<td>3.1 ± 1.0</td>
<td>.03</td>
<td>2.8 ± 1.1</td>
<td>3.2 ± 1.0</td>
<td>.03</td>
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<tr>
<td>Mean SES (at FU41)*</td>
<td>2.4 ± 1.1</td>
<td>3.4 ± 1.0</td>
<td>&lt;.001</td>
<td>3.7 ± 1.1</td>
<td>3.3 ± 1.0</td>
<td>&lt;.001</td>
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<td>&lt;.001</td>
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<td>Lifetime mental disorders</td>
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<tr>
<td>Any mood disorder</td>
<td>45 (40.5)</td>
<td>47 (42.3)</td>
<td>.78</td>
<td>14 (58.3)</td>
<td>33 (37.9)</td>
<td>.19</td>
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<td>Any anxiety disorder</td>
<td>19 (17.1)</td>
<td>20 (18.0)</td>
<td>.86</td>
<td>6 (25.0)</td>
<td>14 (16.1)</td>
<td>.59</td>
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<td>Any alcohol-related disorder</td>
<td>39 (35.1)</td>
<td>47 (42.3)</td>
<td>.33</td>
<td>9 (37.5)</td>
<td>38 (43.7)</td>
<td>.46</td>
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<tr>
<td>Any nonalcohol substance use disorder</td>
<td>37 (33.3)</td>
<td>61 (55.0)</td>
<td>.02</td>
<td>16 (68.7)</td>
<td>45 (51.7)</td>
<td>.02</td>
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<td></td>
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<tr>
<td>Nicotine dependence</td>
<td>27 (24.3)</td>
<td>55 (49.5)</td>
<td>&lt;.001</td>
<td>16 (68.7)</td>
<td>39 (44.8)</td>
<td>&lt;.001</td>
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<td>&lt;.001</td>
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<tr>
<td>Anthropometric values</td>
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<tr>
<td>Height, in</td>
<td>70.1 ± 2.9</td>
<td>70.3 ± 2.9</td>
<td>.64</td>
<td>69.7 ± 3.3</td>
<td>70.5 ± 2.8</td>
<td>.40</td>
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<tr>
<td>Weight, lb</td>
<td>193.8 ± 34.9</td>
<td>212.6 ± 49.7</td>
<td>.001</td>
<td>199.5 ± 37.8</td>
<td>216.1 ± 52.1</td>
<td>.001</td>
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<tr>
<td>BMI</td>
<td>27.6 ± 3.9</td>
<td>30.1 ± 6.3</td>
<td>&lt;.001</td>
<td>28.9 ± 4.8</td>
<td>30.4 ± 6.6</td>
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<td>Weight status</td>
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<td></td>
<td>.0096</td>
<td>.02</td>
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<tr>
<td>Underweight</td>
<td>0 (0)</td>
<td>1 (0.9)</td>
<td>.90</td>
<td>0 (0)</td>
<td>1 (1.1)</td>
<td>.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>28 (25.2)</td>
<td>20 (18.0)</td>
<td>.25</td>
<td>4 (18.7)</td>
<td>16 (18.4)</td>
<td>.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>59 (53.2)</td>
<td>44 (39.8)</td>
<td>.59</td>
<td>15 (54.2)</td>
<td>31 (35.6)</td>
<td>.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>24 (21.6)</td>
<td>46 (41.4)</td>
<td>.002</td>
<td>7 (28.2)</td>
<td>39 (44.8)</td>
<td>.003</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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 (%).

* Hollingshead and Redlich index. Range = 1 (high SES) to 5 (low SES); FU18, follow-up at mean age 18; FU41, follow-up at mean age 41.

The 3 groups differed significantly in weight (P = .001) and BMI (P = .001) but not in height (P = .40) (Table 1). Mean parental SES was significantly higher in men with childhood ADHD (1.6 ± 3.6) than men without childhood ADHD (0.6 ± 2.3), and significantly higher BMIs were documented (49.7 ± 1.9 vs 49.8 ± 2.6, P = .001). Group differences remained after adjustment for parental SES and lifetime mental disorders. BMI was still significantly elevated in men with childhood ADHD (29 ± 3.5) compared with men without childhood ADHD (25 ± 3.1), and was significantly greater in height (P = .001) (Table 1). Group differences remained after adjustment for parental SES and lifetime mental disorders. BMI was still significantly elevated in men with childhood ADHD (29 ± 3.5) compared with men without childhood ADHD (25 ± 3.1).
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%) (Table 1). 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: 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: 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].

### Sensitivity Analyses

Results were unchanged when the 5 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
two psychological hypotheses have been proposed to explain a possible link between ADHD and obesity. 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. In addition, ADHD-related inattention and deficits in executive functions may produce difficulties in adherence to regular eating patterns, leading to abnormal eating behaviors.

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 ADHD as well as in obesity. 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 pathways and related systems, such as melanocortin, which are also implicated in both ADHD and obesity.

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 (i.e., 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%.

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 States 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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(Continued from first page)

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/content/131/6/e1731.full.html