

# A Naturalistic 10-Year Prospective Study of Height and Weight in Children with Attention-Deficit Hyperactivity Disorder Grown Up: Sex and Treatment Effects

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**Objective** To assess the effect of attention-deficit/hyperactivity disorder (ADHD) and its treatment on growth outcomes in children followed into adulthood.

**Study design** Two identically designed, longitudinal, case-control studies of males and females with and without ADHD were combined; 124 and 137 control and subjects with ADHD, respectively, provided growth information at the 10- to 11-year follow-up. We used linear growth curve models to estimate the effect of time on change in height and whether this effect differed by sex and ADHD status. We also examined the effect of stimulant treatment on growth outcomes.

**Results** We found no evidence that ADHD was associated with trajectories of height over time or differences at follow-up in any growth outcomes. Similarly, we found no evidence that stimulant treatment was associated with differences in growth. However, among subjects with ADHD, major depression was associated with significantly larger weight in females and smaller height in males.

**Conclusions** Our results do not support an association between deficits in growth outcomes and either ADHD or psychostimulant treatment for ADHD. These findings extend the literature on this topic into young adulthood and should assist clinicians and parents in formulating treatment plans for children with ADHD. (*J Pediatr* 2010; ■: ■-■).

A recent review examined height and weight in children with attention-deficit/hyperactivity disorder (ADHD)<sup>1</sup> and found that although stimulant medication is associated with delays in expected growth, this effect may attenuate over time. The authors suggested that growth deficits in children with ADHD may normalize over time, but because none of the studies followed children into adulthood, they were not able to directly assess this issue. For example, in our cross-sectional study of boys with and without ADHD, we reported small but significant differences in height. However, height deficits were only evident in early adolescence and were not related to psychopharmacological treatment. Also, we did not detect evidence of weight differences between boys with and without ADHD.<sup>2</sup>

Another important question is the effect of ADHD and its treatment on growth outcomes in females because the overwhelming majority of subjects from the studies reviewed by Faraone et al<sup>1</sup> were male. In a previous cross-sectional study, we did not detect a significant difference between girls ages 6 to 17 years with and without ADHD on age-specific height, z scores, or weight (corrected for height and age). However, a significant difference was found in absolute weight, with medicated girls with ADHD being heavier than treatment naïve girls with ADHD.<sup>3</sup> Additional prospective data are needed to further examine growth outcomes in girls with ADHD, and, given sex effects in the pattern of growth in height through childhood and adolescence, it is important to know whether sex moderates the associations between ADHD, its treatment, and growth outcomes. To address these issues, we tested the following hypotheses: (1) that the trajectory of growth in height differs by ADHD status; (2) that subjects with and without an ADHD diagnosis in childhood will differ on height and weight measurements at follow-up; (3) the association between growth measurements at follow-up and ADHD differs by sex; and (4) psychostimulant treatment is associated with altered growth in subjects with ADHD.

## Methods

Subjects were derived from two identically designed, longitudinal, case-control studies. The first study ascertained 140 male cases (ADHD) and 120 control

ADHD	Attention-deficit/hyperactivity disorder
BMI	Body mass index
GAF	DSM-IV Global Assessment of Functioning
MD	Major depression

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subjects (non-ADHD) ages 6 to 17 years. Subjects were assessed at baseline and at 4- and 10-year follow-up. The second study ascertained 140 females with ADHD and 122 control subjects ages 6 to 17 years. Subjects were assessed at baseline and at 5- and 10- to 11 year follow-up times. We used data from both studies, across all waves of assessment. Potential probands were excluded if they had been adopted, if their nuclear family was not available, if they had major sensorimotor handicaps, if they had psychosis or autism, or if they were unable to participate in the assessments due to language barriers or an estimated IQ <80. After a complete description of the study, the parents or guardians of the subjects or the subjects if aged >18 years provided written informed consent. The Institutional Review Board at Massachusetts General Hospital approved this study.

For both studies, two independent sources provided the index children. The “psychiatric referral source” was an academic medical center, where we selected subjects with ADHD from consecutive referrals to its pediatric psychopharmacology clinic. We selected control subjects from outpatients receiving routine physical examinations. The “pediatric referral source” was a health maintenance organization, where we selected subjects with ADHD from consecutively ascertained pediatric outpatients, identified from their records as having ADHD. We selected control subjects from outpatients receiving routine physical examinations identified from their records as not having ADHD. We used a 3-stage ascertainment procedure to select probands.<sup>4</sup> For subjects with ADHD, the first stage was their referral. The second stage was confirmation of the diagnosis of ADHD through a telephone questionnaire administered to the mother. The third stage was a diagnostic assessment with a structured interview. Patients who received a positive diagnosis at all three stages were included. Only subjects classified as not having ADHD at all 3 stages were included in the control group.

### Psychiatric Assessment

Psychiatric assessments relied on the K-SADS-E (Epidemiologic Version)<sup>5</sup> for subjects younger than 18 years of age and the Structured Clinical Interview for DSM-III-R (SCID)<sup>6</sup> (supplemented with modules from the K-SADS-E to assess childhood diagnoses) for subjects ages 18 and older. Diagnoses were based on direct interviews with the mothers and the offspring. We combined data from direct and indirect interviews by considering a diagnosis positive if it was endorsed in either interview. Diagnoses were considered positive if DSM-IV criteria were unequivocally met. A committee of blinded board-certified child and adult psychiatrists resolved diagnostic uncertainties.

All interviewers had undergraduate degrees in psychology and were trained to high levels of inter-rater reliability. First, they learned interview mechanics, diagnostic criteria, and coding algorithms. Then, they observed interviews by experienced raters and clinicians. They subsequently conducted at least 6 practice (nonstudy) interviews and at least two study interviews while being observed by senior interviewers. The principal investigator (J.B.) supervised the interviewers. We

computed  $\kappa$  coefficients of agreement by having child and adult psychiatrists and clinical psychologists diagnose subjects from audiotaped interviews. Based on 500 assessments of children and adults, the  $\kappa$  coefficient for ADHD was 0.88.

Our approach to defining persistent and subthreshold ADHD is consistent with our previous studies of these samples.<sup>7-9</sup> Persistent ADHD was defined at the 10-year follow-up as meeting full or subthreshold criteria for DSM-IV ADHD currently (ie, during the past month). A subthreshold ADHD case was defined as endorsing only 4 or 5 ADHD symptoms but meeting all other diagnostic requirements (ie, age of onset).

### Demographic Assessment

Socioeconomic status was assessed with the 5-point Hollingshead scale,<sup>10</sup> using the parents' occupational and educational status. As a measure of overall functioning, we used the DSM-IV Global Assessment of Functioning (GAF).<sup>5</sup>

### Assessment of Psychostimulant Treatment

We identified subjects with a lifetime history of treatment with any stimulant (amphetamine products [mixed amphetamine salts, d-amphetamine], methylphenidate products [IR methylphenidate, OROS methylphenidate, transdermal methylphenidate, d-methylphenidate, extended release methylphenidate], and pemoline). Among these subjects with a positive lifetime history, we calculated the total duration of treatment in years.

### Assessment of Growth Measures

Weights were obtained using a Physician's Beam Scale (Detecto, Webb City, Missouri). Measurements were obtained with the subjects lightly clothed but without shoes. Subjects were erect with height examined at the vertex. We used data from growth tables provided by the National Center for Health Statistics from 2000,<sup>11</sup> which are sex-specific and standardized from ages 2 to 20, and the program *zanthro* as implemented in STATA 10.1 (StataCorp, College Station, Texas),<sup>12</sup> to calculate the following height and weight indices (we used the reference data from age 20 for subjects older than this age).

Height values were converted to a height  $z$  score, the difference of an individual height from the mean height, for children of the same age and sex, divided by the standard deviation of height for that subgroup. We examined the relationship of the child's height to the parent's height by regression analysis in the control sample, within sex and assessment wave. The estimated regression equation was used to determine the child's predicted height from their parents' heights. The difference between the child's actual height ( $z$  score) and the child's predicted height based on parental height ( $z$  score) was defined as the parent and age corrected height.

Weight values were converted to a weight  $z$  score defined as the difference of an individual weight from the mean weight, for children of the same age and sex, divided by the standard deviation of weight for that subgroup. First, we calculated each subject's body mass index (BMI; kilograms/meters<sup>2</sup>).

Then, BMI values were converted to a BMI z score defined as the difference of an individual BMI from the mean BMI, for children of the same age and sex, divided by the standard deviation of BMI for that subgroup.

### Statistical Analysis

To test our first hypothesis, we used linear growth curve models to estimate the effect of time on the change in growth outcomes, restricted to subjects with at least 2 growth measurements.<sup>13</sup> We estimated height as a function of age, ADHD, and the age-by-ADHD interaction as fixed effects, with random intercept and random slopes for age. The interaction term tested whether the trajectory height across age differs by ADHD status. To test our second hypothesis, we estimated a series of regression models with the growth outcomes measured at follow-up as the dependent variable and ADHD status as the independent variable. To test our third hypothesis, we included a sex-by-ADHD interaction term to the model described above for hypothesis 2. To test our fourth hypothesis, we estimated a series of regression models among subjects with ADHD with the growth outcomes measured at final follow-up as the dependent variable and duration of lifetime stimulant treatment as the independent variable. All tests were 2-tailed, and the  $\alpha$  level was set at 0.05.

## Results

Of the 140 with ADHD and 120 control boys recruited at baseline, 112 (80%) and 105 (88%), respectively, were successfully reassessed at the 10-year follow-up ( $\chi^2_{(1)} = 2.6, P = .11$ ). Previously, we reported no differences between boys followed up and not followed up on a range of demographic characteristics, except social class.<sup>7</sup> There was no significant difference between the ADHD and control groups in the proportion providing growth data ( $n = 78$  (70%) and  $n = 68$  (65%), respectively;  $\chi^2_{(1)} = 0.59, P = .44$ ). Within the strata of ADHD status, we found no differences between subjects with and without growth data on demographic variables (all  $P$  values  $>.05$ ).

Of the 140 girls with ADHD and 122 control girls recruited at baseline, 96 (69%) and 91 (75%), respectively, were successfully reassessed at the 10-year follow-up ( $\chi^2_{(1)} = 1.16, P = .28$ ). We found no differences between girls followed up and not followed up on demographic characteristics.<sup>14</sup> There was no significant difference between the ADHD and control groups in the proportion providing growth data ( $n = 59$  [61%] and  $n = 56$  [62%], respectively;  $\chi^2_{(1)} < 0.01, P = .99$ ). Within the strata of ADHD status, we found no differences between subjects with and without growth data on demographic variables (all  $P$  values  $>.05$ ). Thus, the sample utilized for this study was comprised of 78 and 68 males with and without ADHD, respectively, and 59 and 56 females with and without ADHD, respectively.

**Table I** shows the demographic characteristics of ADHD and control subjects at the 10-year follow-up. Males with ADHD had a significantly lower mean GAF scores and less affluent social class scores compared with control subjects.

**Table I.** Demographic characteristics of ADHD and control probands with growth data at 10- to 11-year follow-up, stratified by sex

Demographic characteristics	ADHD probands	Control probands	Test statistic (df), $P$ value
	Males		
	$n = 78$	$n = 68$	
Social class*	$2.0 \pm 1.0$	$1.7 \pm 0.9$	$t(144) = -2.28, P = .024$
Age	$21.5 \pm 3.5$	$22.3 \pm 4.1$	$t(144) = 1.34, P = .181$
Past GAF score	$49.6 \pm 8.2$	$59.2 \pm 7.7$	$t(144) = 7.29, P < .001$
Ascertainment source <sup>†</sup>	40 (51)	20 (29)	$\chi^2_{(1)} = 7.18, P = .007$
Females			
	$n = 59$	$n = 56$	
Social class*	$2.1 \pm 1.0$	$1.8 \pm 0.7$	$t(109) = -1.30, P = .194$
Age	$21.1 \pm 3.3$	$22.2 \pm 2.8$	$t(113) = 2.07, P = .041$
Past GAF score	$52.3 \pm 8.6$	$61.9 \pm 8.9$	$t(111) = 5.76, P < .001$
Ascertainment source <sup>†</sup>	25 (43)	18 (32)	$\chi^2_{(1)} = 1.46, P = .227$

Values represent mean  $\pm$  SD or frequency (percent).

\*Hollingshead scale; 1 = most affluent, 5 = least affluent.

<sup>†</sup>Either a major academic medical center or pediatric clinics of a major health maintenance organization. Rates are subjects ascertained from the major academic medical center.

Also, males with ADHD were more likely to have been recruited from the academic medical center relative to male control subjects. Females with ADHD were significantly younger and had lower mean GAF scores compared with female control subjects.

### Trajectories of Growth in Height

First, we estimated a linear growth curve model among the male subjects with two height measurements ( $n = 61$  and  $68$  for control and ADHD probands, respectively). The age coefficient was significant ( $\beta = 1.93, z = 13.8, P < .001$ ), but we found no evidence for a difference in height across age associated with ADHD ( $\beta = -0.67, z = -0.6, P = .560$ ). Next, we added a quadratic age term to the model, which resulted in an improvement in model fit (likelihood ratio test of the nested model: LR  $\chi^2_{(1)} = 174.0, P < .001$ ). Next, we estimated the model using ADHD status, age, quadratic age, and the quadratic age-by-ADHD interaction as fixed effects. The quadratic age-by-ADHD interaction term was not significant. In another model, the test of whether both the age-by-ADHD and the quadratic age-by-ADHD interaction terms were jointly equal to zero was not significant (Wald  $\chi^2_{(2)} = 3.79, P = .150$ ), again providing no evidence that the trajectory of growth in height across age was different between males with and without ADHD.

Next, we repeated this analysis in the female subjects who provided at least two height measurements ( $n = 102$  and  $106$  for control and ADHD probands, respectively). Again, the age coefficient was significant ( $\beta = 1.74, z = 15.6, P < .001$ ), and we found no evidence for a difference in height across age associated with ADHD ( $\beta = -0.30, z = -0.3, P = .762$ ). Next, we added a quadratic age term to the model, which resulted in an improvement in model fit (LR  $\chi^2_{(1)} =$

280.1,  $P < .001$ ). Next, we estimated the model using ADHD status, age, quadratic age, and the quadratic age-by-ADHD interaction as fixed effects. The quadratic age-by-ADHD interaction term was not significant. In another model, the test of whether both the age-by-ADHD and the quadratic age-by-ADHD interaction terms were jointly equal to zero was not significant (Wald  $\chi^2_{(2)} = 2.29$ ,  $P = .319$ ), again providing no evidence that the trajectory of growth in height across age was different between females with and without ADHD. Mean fitted values of metric height from these models across quartiles of age are displayed (Table II).

### Growth in Height and Weight at Most Recent Follow-up

We found no significant differences between subjects with and without ADHD in height, age-corrected height, age- and parent-corrected height, weight, age-corrected weight, or age-corrected BMI (Table III). We next estimated a series of models to test whether the effect of ADHD on growth outcomes was conditional on sex. None of the interactions were significant (all  $P$  values  $> .05$ ). When combining the male and female subjects into one sample for increased statistical power, we still did not detect any significant ADHD effects in growth outcomes. We repeated these analyses including only subjects who were age 20 or older at the most recent follow-up ( $n = 54$  and  $n = 47$  for males with and without ADHD, respectively;  $n = 38$  and  $n = 45$  for females with and without ADHD, respectively) and found no significant differences between subjects with and without ADHD in any growth outcome (all  $P$  values  $> .05$ ).

We also compared growth outcomes between ADHD probands with ( $n = 86$ ; 50 males and 36 females) and without ( $n = 50$ ; 27 males and 23 females) persistent ADHD at the most recent follow-up assessment. Adjusting for sex, there were no significant differences on any growth outcome between ADHD probands with and without persistence (all  $P$  values  $> .05$ ).

### Effect of Psychostimulant Treatment for ADHD on Growth Outcomes

Among males and females with ADHD at the 10-year follow-up, the rate of lifetime stimulant treatment was 76% ( $n = 59$ ) and 86% ( $n = 51$ ), respectively ( $\chi^2_{(1)} = 2.5$ ,  $P = .116$ ). Among boys, the mean age treatment onset was  $8.4 \pm 3.3$  years, with a mean duration of  $7.4 \pm 4.5$  years (range, 0.5 to 18 years).

Among females, the mean age treatment onset was  $8.7 \pm 3.3$  years, with a mean duration of  $6.1 \pm 3.8$  years (range, .5 to 16 years). There were no significant associations between duration of stimulant treatment and any growth outcomes in the combined sample of male and female subjects, adjusting for sex (all  $P$  values  $> .05$ ).

### Effect of Comorbid Major Depression on Growth Outcomes

In our previous assessment of growth outcomes in our sample of female youth with and without ADHD, we reported significantly increased weight in girls with ADHD with comorbid major depression (MD), compared with other girls with ADHD.<sup>3</sup> We attempted to replicate this finding in the current study. At the 10-year follow-up, females with ADHD and MD had a mean age-corrected BMI that was 0.55 standard deviations greater than girls with ADHD without MD ( $0.77 \pm 0.9$  versus  $0.23 \pm 0.9$ , respectively;  $t(57) = 2.15$ ,  $P = .036$ ). Also, males with ADHD and MD had a mean age- and parent corrected height  $z$  score that was 0.46 standard deviations smaller than boys with ADHD without MD ( $-0.18 \pm 0.7$  versus  $0.28 \pm 0.8$ , respectively;  $t(57) = -2.41$ ,  $P = .019$ ). Similar results were found for the age-corrected height  $z$  score ( $t(75) = -2.60$ ,  $P = .012$ ).

## Discussion

Our results showing no differences in trajectories of growth outcomes between subjects with and without ADHD are consistent with our baseline assessment of our female sample with and without ADHD.<sup>3</sup> These findings are also congruent with the mean baseline height  $z$  score from studies of stimulant-naïve children with ADHD ( $z = 0.08$ , 95% CI =  $-0.01$  to  $0.17$ ).<sup>1</sup>

However, our results are not consistent with evidence showing an increased risk for overweight in children with ADHD<sup>15</sup> nor with the Multimodal Treatment Study of ADHD, which found height deficits in children with ADHD and prolonged medication treatment.<sup>16,17</sup> Several differences between our and other studies may have contributed to this discrepancy, such as the sampling strategy and degree of precision in the measurement of treatment data. However, the most likely cause is the age differences between the samples. The MTA study followed school-aged children for up to 3 years, whereas the mean age of our ADHD sample at the 10-year follow-up was 22 years, where we found no evidence that a history of stimulant treatment influenced ultimate height or weight. Taken together, these studies are consistent with Faraone et al<sup>1</sup> who found that, although stimulant medication is associated with delays in expected growth,<sup>18</sup> this effect may attenuate over time, so that final height may not be affected.

Confirming our prior results in the baseline assessment of our female sample,<sup>3</sup> we found an association between comorbidity with MD and significant weight gain in females with ADHD. These results can be interpreted as suggesting that depression in female youth with ADHD may lead to

**Table II.** Mean fitted values of metric height from linear growth curve models across quartiles of age

	Quartiles of age in years			
	9-14	15-18	19-21	22-30
Males with ADHD	157.2	175.2	179.9	178.7
Male control subjects	152.2	175.6	180.1	181.1
	6-12	13-15	16-19	20-30
Females with ADHD	141.1	158.9	166.6	166.5
Female control subjects	145.1	160.4	165.5	164.8

**Table III.** Growth outcomes of ADHD and control probands at follow-up, stratified by sex

Growth measure	Control probands		ADHD probands		Test statistic, <i>P</i> value
	Males*				
	n = 78		n = 68		
Height (cm)	179.0 ± 6.0		178.9 ± 7.7		t(145) = 0.20, <i>P</i> = .844
Age-corrected height (z score)	0.35 ± 0.8		0.27 ± 1.0		t(142) = -0.20, <i>P</i> = .844
Parent- and age-corrected height	0.02 ± 0.7		-0.03 ± 0.8		t(138) = -0.79, <i>P</i> = .434
Weight (kg) (z score)	82.2 ± 15.6		81.8 ± 17.3		t(144) = -0.47, <i>P</i> = .638
Weight for age (z score)	0.80 ± 0.9		0.73 ± 0.9		t(141) = -0.57, <i>P</i> = .571
BMI for age (z score)	0.60 ± 0.9		0.55 ± 1.0		t(141) = -0.49, <i>P</i> = .628
Females†					
	n = 59		n = 56		
Height (cm)	164.6 ± 6.5		166.4 ± 7.0		t(111) = 1.10, <i>P</i> = .275
Age-corrected height (z score)	0.20 ± 1.0		0.49 ± 1.1		t(111) = 1.12, <i>P</i> = .266
Parent- and age-corrected height (z score)	0.00 ± 0.9		0.29 ± 1.0		t(101) = 1.36, <i>P</i> = .178
Weight (kg)	67.5 ± 18.6		70.8 ± 18.3		t(111) = 0.51, <i>P</i> = .614
Weight for age (z score)	0.46 ± 1.1		0.70 ± 1.2		t(111) = 0.83, <i>P</i> = .411
BMI for age (z score)	0.38 ± 1.0		0.61 ± 0.9		t(111) = 0.70, <i>P</i> = .486

Values represent mean ± SD.

\*Adjusted for social class, past GAF score, and ascertainment source.

†Adjusted for past GAF score.

overeating, limited physical activity, or both. Alternatively, being over-weight or obese may place girls with ADHD at heightened risk for depression. Among males with ADHD, we found that MD was associated with a significant reduction in height. This finding is consistent with a recent study from the National Longitudinal Study of Adolescent Health, which found a negative relationship between height and depression among males ages 12 to 19 years of age.<sup>19</sup> It could be that shorter height relative to peers confers a risk for affective symptoms among adolescent males. Because there was no relationship between height and MD in our sample of male controls, this effect may be stronger among males with comorbid psychiatric disorders, such as ADHD, or other psychosocial vulnerabilities. Additional studies are needed to clarify these issues.

Our results should be considered in the light of some methodological limitations. We were only able to collect growth data on a subset of subjects in the follow-up assessments, which may have introduced sampling bias. Also, our null findings may represent a type II error, suggesting that a larger sample with better statistical power may be able to detect significant differences. However, our samples had 85% and 99% power to detect medium and large-sized (ie, 0.5 and 0.8 mean standard deviation differences) effects, respectively. Because some subjects were initially assessed at baseline with a history of stimulant treatment, we were unable to obtain growth measurements on all subjects prior to treatment onset. Also, our assessment of the effects of stimulant treatment would have benefited from additional information regarding dose and interruptions of treatment, which were unavailable in sufficient detail in these data. Finally, while the effect of non-stimulant psychopharmacological treatments on growth outcomes is a clinically important issue to study, our sample was primarily treated with stimulant medications. There were rel-

atively few subjects treated with other classes (eg, antidepressants, mood stabilizers, antipsychotic agents) of psychopharmacological medications. Thus, because we do not have the power to conduct these analyses in this sample, future studies should examine this issue.

Despite these limitations, our results do not support an association between deficits in growth outcomes in height and weight and either ADHD or psychostimulant treatment for ADHD. These findings support the extant literature on this topic, extend it into young adulthood, and can inform patients, clinicians, and parents about the growth trajectories of children with ADHD. ■

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## Appendix

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tis Pharmaceuticals. T.S. has been on the advisory board for the following pharmaceutical companies: Shire Laboratories Inc., Cephalon, Eli Lilly & Company, Glaxo-Smith Kline, Janssen, McNeil Pharmaceutical, Novartis Pharmaceuticals, and Pfizer. In the past year, S.F. has received consulting fees and has been on Advisory Boards for Eli Lilly and Shire and has received research support from Eli Lilly, Pfizer, Shire, and the National Institutes of Health. In previous years, S.F. has received consulting fees or has been on Advisory Boards or has been a speaker for the following sources: Shire, McNeil, Janssen, Novartis, Pfizer and Eli Lilly. In previous years he has received research support from Eli Lilly, Shire, Pfizer, and the National Institutes of Health.

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