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REGULAR ARTICLE

Half of the children with overweight or obesity and attention-deficit/hyperactivity disorder reach normal weight with stimulants

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Abstract

Aim: Treatment of childhood obesity is often insufficient and may be aggravated by high co-occurrence of attention-deficit/hyperactivity disorder (ADHD). We aimed to investigate whether children with overweight or obesity normalised in weight when receiving stimulant treatment for ADHD.

Methods: Growth data of 118 children were obtained from medical records at outpatient paediatric and children's psychiatric services in the Gothenburg area, Sweden. The children were diagnosed with ADHD and were between 6 and 17 years at the start of stimulant treatment. The pre-treatment data act as an internal control where every child is their own control.

Results: At the start of treatment, 74 children had normal weight and 44 had either overweight or obesity. During the year with stimulants, the mean (SD) body mass index (BMI) in standard deviation score (SDS) decreased significantly: −0.72 (0.66) compared with 0.17 (0.43) during the year before treatment ($p < 0.01$). After one year with treatment, 43% of those with overweight or obesity had reached normal weight.

Conclusions: Stimulant treatment for ADHD yields significant weight loss. In children with overweight or obesity and ADHD, this is an important finding showing additional benefit in terms of weight management.

KEYWORDS
attention deficit hyperactivity disorder, body mass index, central stimulants, children, obesity

Key Notes

- The conventional treatment of obesity is often not enough for children with co-occurring attention-deficit/hyperactivity disorder (ADHD).
- After one year with stimulant medication for ADHD, 43% of those with overweight or obesity had reached normal weight.
- Improved regulation of appetite due to increased behaviour control and reduced impulsivity may contribute to the beneficial effect on weight.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; CI, confidence interval; ICD-10, international statistical classification of diseases and related health problems—tenth revision; SDS, standard deviation score.

Karin Fast and Anna Björk have Contributed equally as co-first authors.

1 | INTRODUCTION

There is a significant association between attention-deficit/hyperactivity disorder (ADHD) and obesity.[1-5] Both children and adults with ADHD have an increased risk of obesity compared with the background population.[6] The prevalence of ADHD among children with obesity range from 18%[7] to 58%.[3] In a population-based study, overweight and obesity were twice as common among children with ADHD compared with controls.[2] A meta-analysis with 42 studies that included 728,136 subjects confirmed the association between ADHD and obesity in children with odds ratio of 1.30.[6] Cortese et al showed no higher risk for obesity for either children or adults when ADHD was treated with stimulants.[6]

The recommended obesity treatment during childhood is lifestyle changes for the whole family, including diet, exercise and non-sedentary behaviour, combined with cognitive behavioural therapy, yielding better result when initiated before puberty.[8] However, only a minority of adolescents treated with lifestyle interventions showed successful results.[8] ADHD with inattention and or hyperactivity-impulsivity often involve executive dysfunction, learning problems and sleep disorders.[9] Children with ADHD have an increased risk of depression, and other neuropsychiatric disorders may coexist.[10] A treatment study of adults with obesity showed less weight loss among those who had concurrent symptoms of ADHD.[11] It has been discussed whether obesity treatment should be modulated based on different co-occurring conditions, such as ADHD, and early screening for ADHD in children with obesity has been suggested to enable start of treatment with stimulants as early as possible.[12]

Stimulants are considered first-line pharmacological treatment and effectively reduce ADHD core symptoms.[13,14] Common side effects of stimulants include appetite suppression, increased blood pressure, tachycardia, headache, stomach ache, sleeping problems and dysphoria.[15] Moreover, stimulants improve executive functioning and appetite regulation with less food cravings and consumption of snacks.[16,17] A retrospective study with longitudinal data on 163,820 children ages 3 to 18 years showed lower body mass index (BMI) at a young age when treated with stimulants, but a catch-up during adolescence resulted in higher BMI compared with population-based controls.[18] The effects on growth, however, are inconclusive, and few studies are reporting either non-treated trajectories or long-term outcome with stimulants.[19,20] A publication from the multimodal treatment study of ADHD found reduction in adult height after long-term stimulant medication of children with ADHD.[21]

The main objective of this study was to analyse whether treatment with stimulants due to ADHD results in weight loss among children with normal weight, overweight and obesity. The hypothesis was that children with ADHD and co-occurring overweight or obesity treated with stimulants experience an equally large weight loss as children with ADHD and normal weight. A further objective was to investigate whether the pre-pubertal prescription of stimulants results in greater weight loss than pubescent and postpubescent prescription. The sub-hypothesis was that children with ADHD treated with stimulants experience a greater weight loss when treatment is initiated before puberty.

2 | METHODS

2.1 | Study population

In this retrospective cohort study, data were obtained from medical records at four outpatient paediatric and children's psychiatric services. The services were located in Kungsbacka, Mölnlycke, Partille and Mölndal in the surroundings of Gothenburg in Sweden. The outpatient paediatric and psychiatric services are run by specialists in paediatrics and child psychiatry, respectively. These clinics were responsible for ADHD investigations and the prescription of stimulants by physicians with a special license to prescribe stimulants.

Patients born in 1998-2011 were identified by diagnostic coding for ADHD F90, according to international statistical classification of diseases and related health problems—tenth revision (ICD-10). Prescription of stimulants were found by their respective pharmaceutical trade name. Patient data were extracted from medical records. The inclusion criteria were diagnosis of ADHD (ICD-10 F90), age between 6 and 17 years at the start of treatment, stimulant-naïve, complete growth data and continuous treatment with stimulants during the studied period. Complete growth data include the weight and height at the start of treatment ± three days as well as 12 months ±270 days before and 12 months ±30 days after initiation. Before treatment initiation, the children were expected to follow their respective growth trajectories, explaining the larger tolerance of the pre-treatment inclusion window than the post-treatment.

Participants were not included in the study if they were suspected of noncompliance, indicated by interruptions in medication, during auditing of their medical records. All children included in the study had been prescribed methylphenidate and or lisdexamfetamine, but the dosage and choice of drugs varied. The minimal dose was typically the dose at the start of treatment and all patients had higher doses most of the studied period, after a titration period. Dose variations according to the physicians' recommendation were allowed in the study. Use of non-stimulant atomoxetine was allowed as a supplement, but not as single therapy.

2.2 | Variables

The primary outcome variable was the mean change in BMI standard deviation score (SDS) for the periods twelve months before and twelve months after the start of treatment. The children were classified at the start of treatment according to BMI subgroups, the subtype of ADHD and age. BMI SDS was based on Karlberg et al[22] BMI subgroups responded to the cut-off for overweight (international obesity task force BMI 25) and obesity (international obesity task

force BMI 30) for girls and boys, respectively.[23] The studied population was analysed based on the BMI subgroups: children with normal weight and children with overweight or obesity, respectively. Two of the children (1.7%) grouped as normal weight were in fact underweight according to international cut-offs.[23] Due to the age-corrected nature of BMI SDS, the data included in the study are adjusted for age at collection. A cut-off for the onset of puberty was defined as 9.6 and 10.7 years for girls and boys, respectively, corresponding to −1 SDS in a general population of children from the Swedish references.[24]

Medical records were reviewed for date of birth, sex, the subtype of ADHD, inattentive, hyperactive-impulsive or combined, date of starting treatment, type of stimulants and doses (mg/day), growth charts including weight and height at all time points and compliance to treatment. The subtypes of ADHD are defined in medical records according to ICD-10 F90.1 hyperactive-impulsive, F90.0B combined and F90.0C inattentive. Furthermore, diagnoses other than ADHD and or medications with a possible influence on the outcome, including autism spectrum disorder (ASD) (n = 28), tics (n = 6), Tourette's syndrome (n = 1), pharmacological treatment with antidepressants (n = 8) and levothyroxine (n = 1) were reviewed.

Seven patients had data falling outside the inclusion windows either twelve months ±270 days before or twelve months ±30 days after initiation. Further analysis of these children's growth charts showed multiple measurement points leading up to treatment initiation, but outside the inclusion windows, pointing to channel parallel growth. Hence, it was determined that the children could be included in the study. Their data was extrapolated to fit the required time window.

2.3 | Statistics

A power analysis was carried out ahead of the study using G*Power version 3.1.9.2 (Heinrich Heine Universität, Düsseldorf, DE). The power analysis yielded that the size of the study population should be more than 30 children for describing changes in BMI-SDS during one year after treatment initiation with a confidence index (CI) of 95%.

All statistical analyses were conducted by using SPSS statistics version 25 (IBM Corp). One child had hyperactive-impulsive type ADHD and was handled in the subgroup with the combined type of ADHD. Shapiro-Wilk ($p < 0.05$) and boxplots were used to identify non-normal distributions. Weight SDS, height SDS and BMI SDS were normally distributed, and the remaining continuous variables were assessed as non-normal. The outcome regarding BMI SDS and height SDS was presented using mean and standard deviation (SD). Descriptive statistics were computed for all continuous variables as compared to BMI subgroups by using paired sample t test (if normally distributed) or Kruskal-Wallis (if non-normally distributed). The differences in BMI SDS and height SDS were similarly analysed using paired sample t test; comparing the period on stimulants and the year before the start of treatment. In order to compare the migration between BMI subgroups in the periods before and after the treatment, McNemar's test was used. p values for categorical variables were calculated by Fisher's exact test or McNemar's test, and then the chi-squared statistics determined if significant. p value ≤ 0.05 was considered statistically significant.

2.4 | Ethics

The study was approved by the regional ethical review board in Gothenburg (133–17), and consent was collected from the head of departments. Written informed consent was not requested due to the retrospective design and anonymised data.

3 | RESULTS

Out of 701 patients identified with ADHD in medical records, 118 met the inclusion criteria and were included for further analyses. A flowchart of the study population is shown in Figure 1. One of the children had Down syndrome and was excluded because of a strongly divergent growth pattern.

Table 1 shows the patient characteristics at the start of treatment. The median (range) age was 9.1 (5.3–16.6) years. The majority of all studied children were classified as pre-pubertal (68%) with a median age of 8.2 (5.3-10.6) years. All children included in the cohort were diagnosed with ADHD and had been treated with stimulants for at least one year. At the start of treatment, 63% of the children were classified with normal weight and 37% with overweight or obesity. Among the 44 children in the overweight or obesity group, 19 children (43%) had obesity when treatment started. The combined type of ADHD was diagnosed in 80% of the children. 24% of the children in the study also had ASD, but neither BMI SDS nor age differed from the rest of the sample (Table S1). We found that 10/28 (36%)

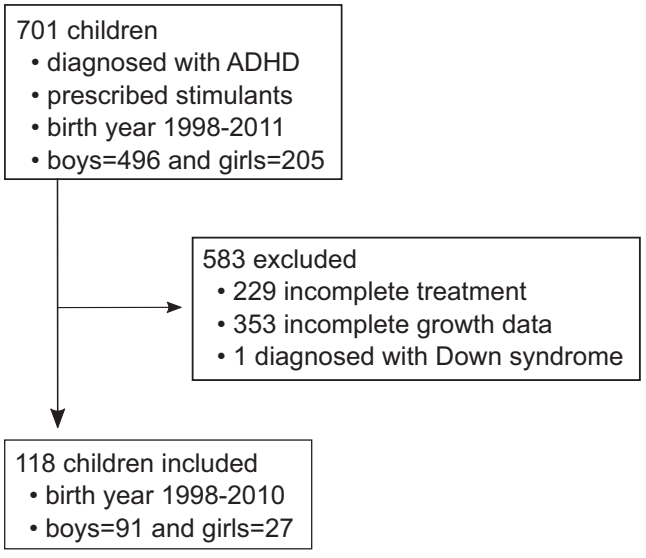

FIGURE 1 Flowchart over the study cohort

TABLE 1 Patient characteristics and BMI subgroups at the start of treatment

	All (*n* = 118)	NW (*n* = 74)	OW/O (*n* = 44)	p
Age (years)	9.1 (5.3–16.6)	8.7 (6.0–15.8)	9.9 (5.3–16.6)	0.05[a]
Sex, *n* (%)				0.82
Boys	91 (77)	58 (78)	33 (75)	
Girls	27 (23)	16 (22)	11 (25)	
Weight (kg)	33.4 (18.9–113.0)	29.0 (18.9–68.8)	45.2 (23.5–113.0)	<0.01[a]
Weight SDS (SDS)	1.1 (−2.4–5.4)	0.0 (−2.4–2.5)	2.8 (−0.6–5.4)	<0.01
Height (cm)	136.0 (111.5–178.5)	133.9 (111.5–178.5)	143.0 (115.7–178.0)	<0.01[a]
Height SDS (SDS)	0.2 (−3.3–6.3)	0.0 (−2.0–3.1)	0.5 (−3.3–3.0)	<0.01
BMI (kg/m^2)	17.6 (13.5–35.7)	16.3 (13.5–24.6)	22.5 (17.6–35.7)	<0.01[a]
BMI SDS (SDS)	0.9 (−2.2–4.6)	0.0 (−2.2–1.8)	2.5 (1.4–4.6)	<0.01
ADHD, *n* (%)				0.16
Combined	94 (80)	62 (84)	32 (73)	
Inattentive	24 (20)	12 (16)	12 (27)	
Age, *n* (%)				0.15
Pre-pubertal	80 (68)	54 (73)	26 (59)	
Pubertal	38 (32)	20 (27)	18 (41)	

Start of treatment is defined as time zero (0). Fisher's exact test was used for all categorical variables.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; *n*, number of participants; NW, normal weight; OW/O, Overweight/Obesity; SDS, standard deviation score.

[a]hypotheses were tested with non-parametric Kruskal-Wallis as the assumption of normality was violated for continuous variables.

of the children with ASD had overweight or obesity, compared with 34/90 (38%) in the group without ASD.

Figure 2A and 2B show the BMI SDS and height SDS, respectively, with corresponding gradients for the children during the year with treatment compared with the year before stimulants. The children were their own controls. The mean (SD) change in BMI SDS for all children during the year with treatment was significantly lower -0.72 (0.66) compared with the change the year before treatment 0.17 (0.43) with p value <0.01 (Table 2a). The difference is also significant when looking at the subgroups normal weight and overweight or obesity separately. During the year with treatment, the children of both subgroups experienced significant decreases in delta BMI SDS, normal weight −0.66 (0.55) and overweight or obesity -0.82 (0.81), with no significant difference between the subgroups (p = 0.25).

The changes in BMI SDS made some children transition between BMI subgroups during the study (Figure 3). Of the 44 children classified as overweight or obesity at the start of treatment, 19 (43%) transitioned into normal weight during the year with treatment. A McNemar test for the consistency in responses to stimulants across normal weight and overweight or obesity was significant with p < 0.01. The percentage of children with normal weight went from 63% at the start of treatment to 79% after a year with stimulant treatment. The corresponding numbers for obesity were 16% and 8%. All 74 children with normal weight when treatment started stayed normal weight when treated with stimulants. Of the 74 children with normal weight two had underweight at the start of treatment, during the year with treatment this increased to four children.

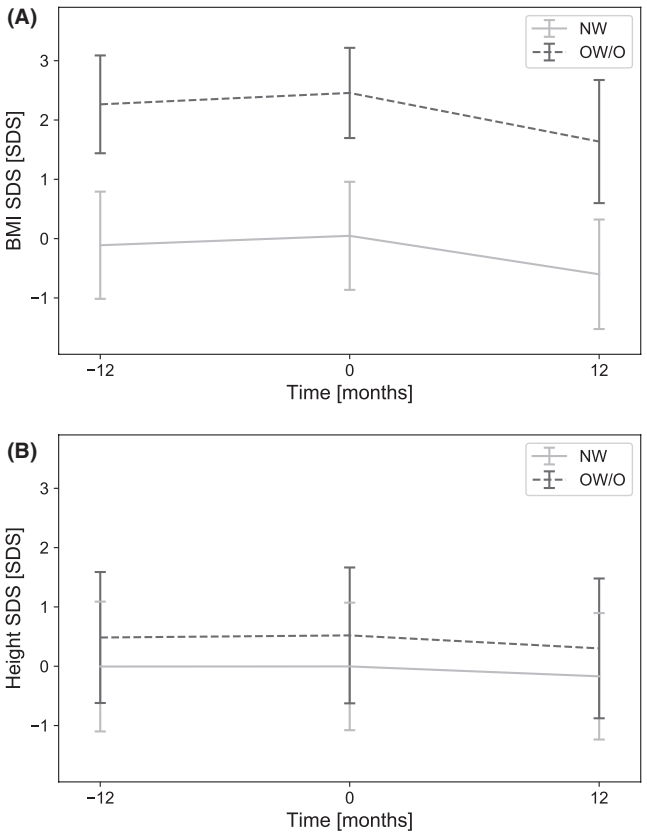

FIGURE 2 Mean change in (A) BMI SDS and (B) height SDS with error bars (1 SD) for children with normal weight, overweight and obesity, respectively. Start of treatment is defined as time zero (0). NW, normal weight; OW, overweight; O, obesity

TABLE 2 Changes over time (months) in (a) BMI SDS, (b) height SDS with corresponding p values

	Δ (0–(−12))	Δ (12–0)	t	CI	p
Overall (118)	0.17 (0.43)	−0.72 (0.66)	−11.25	−1.05; −0.73	<0.001
BMI SDS					
1. Normal weight (74)	0.16 (0.46)	−0.66 (0.55)	−9.55	−0.99; −0.65	<0.001
2. Overweight or obesity (44)	0.19 (0.39)	−0.82 (0.81)	−6.51	−1.33; −0.70	0.001
Comparing Δ (12–0) between 1∉2			1.16	−0.12; 0.44	0.25
Sex					
1. Boys (91)	0.20 (0.45)	−0.72 (0.68)	10.03	0.74; 1.10	<0.001
2. Girls (27)	0.08 (0.35)	−0.72 (0.61)	5.04	0.47; 1.12	<0.001
Comparing Δ (12–0) between 1∉2			−0.02	−0.29; 0.29	0.99
ADHD					
1. Combined (94)	0.22 (0.45)	−0.76 (0.69)	10.62	0.80; 1.16	<0.001
2. Inattentive (24)	−0.03 (0.31)	−0.57 (0.51)	4.40	0.29; 0.79	<0.001
Comparing Δ (12–0) between 1∉2			−1.25	−0.49; 0.11	0.22
Age					
1. Pre-pubertal (80)	0.23 (0.46)	−0.78 (0.65)	10.80	0.83; 1.20	<0.001
2. Pubertal (38)	0.05 (0.33)	−0.59 (0.67)	4.55	0.35; 0.92	<0.001
Comparing Δ (12–0) between 1∉2			−1.5	−0.45; 0.06	0.13
	Δ (0–(−12))	Δ (12–0)	t	CI	p
Overall (118)	0.01 (0.21)	−0.19 (0.24)	7.09	0.15;0.26	<0.001
BMI SDS					
1. Normal weight (74)	0.002 (0.21)	−0.17 (0.22)	4.82	0.10; 0.24	<0.001
2. Overweight or obesity (44)	0.04 (0.22)	−0.22 (0.27)	5.38	0.16; 0.36	<0.001
Comparing Δ (12–0) between 1∉2			1.18	−0.04; 0.14	0.24
Sex					
1. Boys (91)	0.02 (0.20)	−0.19 (0.24)	6.73	0.15; 0.28	<0.001
2. Girls (27)	−0.02 (0.24)	−0.17 (0.61)	2.49	0.03; 0.28	0.02
Comparing Δ (12–0) between 1∉2			−0.37	−0.12; 0.09	0.72
ADHD					
1. Combined (94)	0.02 (0.21)	−0.20 (0.23)	7.27	0.16; 0.28	<0.001
2. Inattentive (24)	0.002 (0.22)	−0.15 (0.29)	1.87	−0.02; 0.31	0.07
Comparing Δ (12–0) between 1∉2			−0.98	−0.16; 0.06	0.33
Age					
1. Pre-pubertal (80)	0.01 (0.20)	−0.20 (0.20)	6.96	0.15; 0.28	<0.001
2. Pubertal (38)	0.02 (0.24)	−0.16 (0.31)	2.92	0.06: 0.30	0.01
Comparing Δ (12–0) between 1∉2			−0.69	−0.15; 0.07	0.50

Start of treatment is defined as time zero (0). Twelve months before and after start of treatment is defined as −12 and 12, respectively. The differences in BMI SDS and height SDS were analysed using paired sample *t* test, mean (SD). For the other categories, independent samples test were used. Fisher's exact test was used for all categorical variables, and then, the chi-squared statistics determines if significant, p-value for interaction.

Abbreviations: ∉, not element of; ADHD, attention-deficit/hyperactivity disorder; BMI, body mass index; CI, confidence interval; SDS, standard deviation score; Δ, delta.

p value ≤ 0.05 was considered statistically significant.

Table 2a and 2b shows the change over time for BMI SDS and height SDS, respectively. We found that delta height SDS went from 0.01 (0.21) during the year without treatment to −0.19 (0.24) during the year with treatment; corresponding to a significant decrease when treated with stimulants ($p < 0.01$). The decrease was significant independent of BMI subgroup, with no difference between the subgroups (Table 2b). While treatment changes due to increased blood pressure and weight loss were found in the medical records, despite oral nutritional supplements, no treatment changes due to changes in height trajectory were found. The delta weight SDS had a similarly significant decrease when treated with stimulants compared with the control. However, in contrast to delta height SDS we

found that delta weight SDS was significantly larger in magnitude for the children with overweight or obesity. For the children with overweight or obesity, the change in weight SDS was −0.96 (0.94), compared with −0.61 (0.48) for children with normal weight when treated with stimulants (p = 0.025, 95% CI). The correlation between delta height SDS and delta weight SDS for the cohort while under treatment with stimulants is shown in a scatter plot (Figure 4), emphasising the wide range in the weight change.

4 | DISCUSSION

This study shows a significant decrease in BMI SDS in the cohort when treated with stimulants. The decrease in BMI SDS is shown to be significant also for children with overweight and obesity, of which 43% dropped to normal weight during the studied period. The children with overweight and obesity also decreased significantly more in weight SDS under treatment compared with children with a normal weight. The results align with previous studies showing a significant decrease in BMI SDS by stimulants.[19,25,26] Diez-Suarez et al studied 342 children between 6 and 18 years with stimulant treatment and found a significant decrease in weight SDS with a magnitude of −0.4 and BMI SDS of −0.58.[19] A study with similar design showed a significant decrease in BMI absolute z-score when treated with stimulants, but no difference in height absolute z-score.[25]

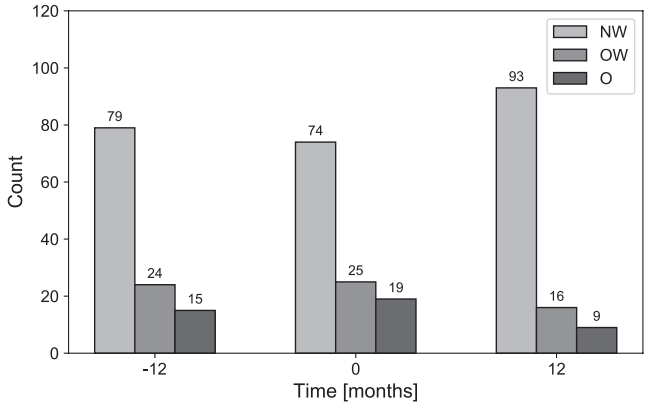

FIGURE 3 The changes in BMI SDS made some children transition between BMI subgroups during the study. NW, normal weight; OW, overweight; O, obesity

We found a significant decrease in height SDS for all children when treated with stimulants compared with the year before treatment. A few individuals decreased more than 0.5 SDS in height. At an individual level, only one child (classified with obesity) had a catch-down in height of more than 1 SDS. While there is a correlation between the individuals losing more in weight being the ones losing more in height, the change in height SDS was less pronounced compared with the decrease in delta weight SDS. The effect of stimulant medication on height has been inconclusive; one previous study found a significant decrease in height during treatment.[20] Diez-Suarez did not show any differences in height SDS before and after stimulant treatment when looking at the full sample (6–18 years), but found slight changes among the subgroup 6-12 years, baseline height SDS: 0.04 (1.14), follow-up: −0.10 (1.11), p < 0.001.[19] A 2020 publication from the multimodal treatment study of ADHD found a reduction in adult height after 16 years of stimulant treatment of children with ADHD.[21] Neither of these studies took into account the pre-stimulant growth trajectory.

Looking at the subgroup analysis of sex, ADHD subgroup and age in Table 2a neither of these variables have a significant influence on changes in BMI SDS, debunking the sub-hypothesis that children with ADHD treated with stimulants experienced a greater weight loss when treatment was initiated before puberty. This might be due to the nature of obesity gradually increasing in severity with time; hence, the correlation should be weaker at a younger age. Even if

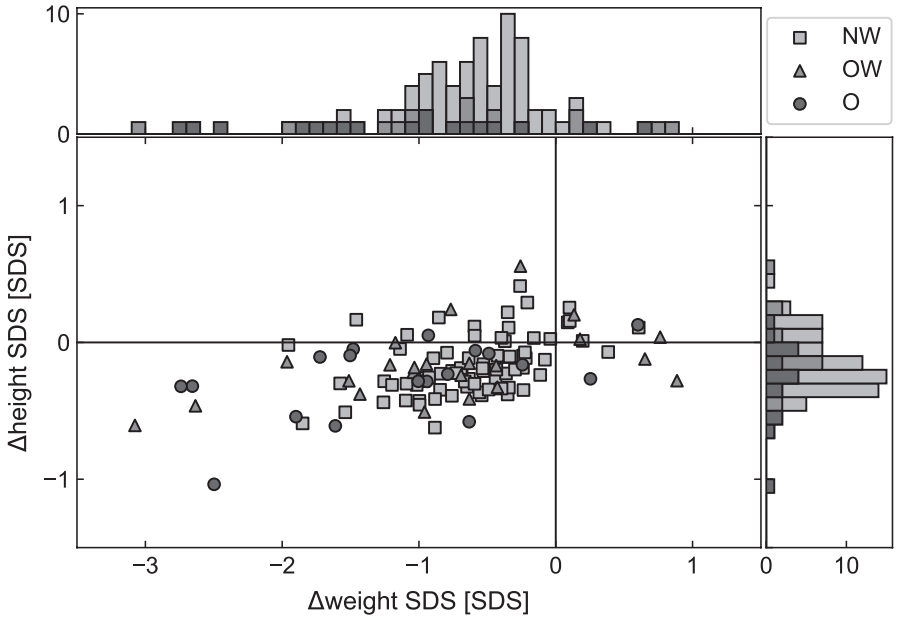

FIGURE 4 The correlation between delta height SDS and delta weight SDS for children under treatment with stimulant marked according to BMI SDS subgroup at the start of treatment. Besides, the histogram shows the number individuals at each point in respective subgroup. NW, normal weight; OW, overweight; O, obesity

we cannot show any significant weight loss with starting treatment earlier, we suggest that an earlier start of treatment may prevent further weight gain.

Impaired executive functions in children with ADHD are associated with higher BMI independent of medication status,[27] highlighting the importance of self-regulation when treating children with ADHD and obesity, or vice versa. One-third of the children with obesity referred to specialist centres had a neurodevelopmental disorder and one-fifth of the parents had ADHD symptomatology.[7] These problems may not only aggravate the morbidity of obesity but also reduce the possibilities to succeed with traditional obesity treatment. Beyond appetite suppression, stimulants enhance motivation and counteract reward dependency,[28] factors that may lead to weight loss. A single dose of stimulant medication has been shown to reduce energy intake.[29] Earlier studies have shown incongruent results regarding dose-dependent weight loss in children with ADHD treated with stimulants, suggesting that a reduction in BMI may be a result of a reduction in ADHD symptoms and improved executive functioning, not just appetite suppression.[19,30]

The strength of this study was the group comparison of non-medicated children with ADHD, where the individuals are their own controls, to our knowledge taking the pre-stimulant growth trajectory into account for the first time. For this type of study, such a design is advantageous compared with, for example, a background population, eliminating hidden differences between the studied cohort and a background population. Another strength is the structured follow-up of each child in the cohort, allowing comparisons over time. There are however some limitations. The cohort is small and the power is insufficient to allow further sub-analysis. As can be seen in Figure 1, most patients extracted from the medical record database did either not receive stimulants continuously during the first year of treatment, or their growth data did not fall within the inclusion criteria. We have no reason to suspect, however, that the influence of stimulants on growth would differ between the study population and the background population.

Our study clearly shows that children with overweight and obesity lose weight when treated with stimulants for ADHD. On a group level, these children lose at least as much weight as children with normal weight. Weight loss caused by loss of appetite is a well-known negative side effect of stimulants,[15] but at an individual level the weight loss should not only be considered a negative side effect due to loss of appetite. Additionally, it might be a consequence of improved executive functioning and appetite regulation,[16,17] potentially avoiding unnecessary weight gain and morbid obesity. We therefore propose neurodevelopmental evaluation to be compulsory at an early stage of the treatment of children with overweight and obesity.

The association between ADHD and a higher risk of obesity is well-known.[2-5,7] Obesity is highly prevalent in individuals with ADHD and vice versa, convincingly showed in a systematic review and meta-analysis[6] and update.[1] A more extensive collaboration has been suggested in the paediatric and psychiatric care for children with ADHD and overweight or obesity, with screening for ADHD among children with weight problems to avoid morbid obesity[12] and for a neurodevelopmental treatment approach to be incorporated in the obesity treatment.[7] Our study illustrates the importance of increased collaboration between the two fields. This would support and enable the discovery of children with ADHD and a high risk for obesity, and, by offering stimulant treatment early in life, this can improve their executive functions and possibly prevent morbid obesity.

5 | CONCLUSION

When treated with stimulants for ADHD, BMI SDS decreases significantly in children, regardless of whether the child has overweight, obesity or normal weight at treatment initiation. Hence, the effect of stimulants in terms of either appetite regulation or improved ability to regulate behaviour can be considered beneficial in children with overweight or obesity.

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CONFLICTS OF INTEREST

The authors report no conflicts of interest in this article.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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