

Attention-Deficit/Hyperactivity Disorder And Childhood Obesity

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Introduction

According to the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5)¹, Attention-Deficit/Hyperactivity Disorder (ADHD) is defined by persistent, age inappropriate and impairing levels of inattention and/or hyperactivity-impulsivity. DSM-5 defines four presentations of ADHD: “predominantly inattentive”, “predominantly hyperactive-impulsive”, “combined”, and “not otherwise specified”. Hyperkinetic Disorder (HKD), defined in the International Classification of Diseases, 10th Edition², is a narrower diagnostic category, requiring both symptoms of inattention and hyperactivity/impulsivity and thus overlapping with the combined presentation of ADHD as per DSM-5¹.

The diagnosis of ADHD is currently entirely based on DSM-5 descriptive criteria (Table 1) based on information gathered from the patient, his family, teachers or other people who can provide information on the patient’s behaviour. No biological tests are currently available.

Table 1. DSM-5 criteria for the diagnosis of Attention-Deficit/Hyperactivity Disorder.

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- A. A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):
1. **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:
Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.
 - a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
 - b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
 - c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
 - d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
 - e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
 - f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
 - g. Often loses things needed for tasks and activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
 - h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).

i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. **Hyperactivity and impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

a. Often fidgets with or taps hands or feet or squirms in seat.

b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).

c. Often runs about or climbs in situations where it is inappropriate. (**Note:** In adolescents or adults, may be limited to feeling restless).

d. Often unable to play or engage in leisure activities quietly.

e. Is often "on the go", acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).

f. Often talks excessively.

g. Often blurts out an answer before questions have been completed (e.g., completes people's sentences; cannot wait for turn in conversation).

h. Often has difficulty waiting his or her turn (e.g., while waiting in line).

i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents or adults, may intrude into or take over what others are doing).

B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.

C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school or work; with friends or relatives; in other activities).

D. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained for by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Specify whether:

Combined presentation: If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.

Predominantly inattentive presentation: If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity-impulsivity) is not met for the past 6 months.

Predominantly hyperactive/impulsive presentation: If Criterion A2 (hyperactivity-impulsivity) is met and Criterion A1 (inattention) is not met for the past 6 months.

Specify if

In partial remission: When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

Specify current severity:

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairment in social or occupational functioning.

Moderate: Symptoms or functional impairment between “mild” and “severe” are present.

Severe: Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.

ADHD is one of the most frequent childhood-onset psychiatric conditions, with an estimated worldwide-pooled prevalence exceeding 5% in school-age children ³. Impairing symptoms of ADHD persist into adulthood in up to 65% of childhood-onset cases ⁴, with a prevalence of ADHD in adults estimated at ~2.5% ⁵.

Executive functions are defined as a set of neurocognitive skills that are necessary to plan, monitor and execute a sequence of goal-directed complex actions. They include inhibition, working memory, planning, and sustained attention ⁶. Besides the behavioural core symptoms of inattention, hyperactivity, and impulsivity, deficits in executive functions are common, although not universally, in ADHD ⁷. Additionally, ADHD is usually comorbid with other neurodevelopmental and/or psychiatric conditions, such as learning disorders, oppositional defiant/conduct disorder, mood and anxiety disorders, substance use disorders, and sleep disturbances ^{8,9}.

Currently, the mainstay of treatment, at least for severe cases, is pharmacologic, with psychostimulant medications (methylphenidate and amphetamine derivatives) as the first line, and non-stimulants as secondary option ^{7,10}. Non-pharmacological treatments, such as behavioural therapies, diet regimens, cognitive training, and neurofeedback, are also available. Although the empirical evidence for their efficacy for ADHD core symptoms is so far unclear ¹¹, such treatments may effectively address behavioural or neuropsychological dysfunctions related to ADHD.

Due its core symptoms as well as associated disorders/conditions, ADHD imposes an enormous burden

on society in terms of psychological dysfunction, adverse vocational outcomes, stress on families, and societal financial costs. The U.S. annual incremental costs of ADHD have been recently estimated at \$143-\$266 billion¹² and high costs have been reported in other countries as well (e.g.,¹³).

Whereas the comorbidity between ADHD and psychiatric disorders has been extensively explored⁸, the association with general medical conditions has received much less attention. However, an increasing literature on the association between neuropsychiatric disorders and medical conditions has emerged in the past years. There has been a rising interest, in particular, on possible association of ADHD to obesity and/or overweight¹⁴.

Studies on the relationship between ADHD and obesity/overweight

After the seminal paper by Altfas¹⁵ reporting that 59 out of 215 adults treated in a bariatric clinic presented with DSM-IV ADHD [27.4% vs. 2.5% in the general population], there has been an increasing number of studies, both in adults and children, exploring the relationship between ADHD and obesity. Specifically, two types of studies can be identified: 1) those that assessed the weight status or the prevalence of obesity/overweight in individuals with ADHD and 2) those that focused on the prevalence of ADHD in individuals with obesity. These studies are reported, respectively, in Table 1 and 2. Although this book focuses on childhood ADHD, we report and discuss here also studies in adults, since they may provide useful insights on the relationship between ADHD and obesity in childhood.

Table 2. Studies assessing the weight status of individuals with ADHD.

First author (year)	Sample characteristics	Key results
Spencer ⁵³ (1996)	124 males with ADHD and 109 normal controls Age range: 6–17	ADHD subjects had greater than average body mass (age- and height-corrected weight index: 109 ± 15), although no significant difference was found between the age- and height-corrected weight index of ADHD and control subjects. Age- and height-corrected weight index of untreated ADHD was 115, indicative of overweight.
Biederman (2003) ⁵⁴	140 ADHD girls and 122 female controls Age range: 6–17	The age- and height-corrected weight index was greater than average (1.1), although not indicative of overweight or obesity. No significant differences were found between ADHD girls and controls, as well as between treated and untreated subjects. ADHD girls with comorbid major depression (MD) had a significantly greater average height- and age-corrected weight index relative to ADHD girls without MD ($P = .011$).
Holtkamp (2004) ⁵⁵	97 inpatient and outpatient boys	The mean BMI-SDS of ADHD patients was significantly higher than the age-adapted

	with ADHD in a child and adolescent psychiatric department Mean age (SD): 10 (2)	reference values ($P = 0.038$). The proportion of obese (7.2%) and overweight (19.6%) participants was significantly higher than estimated prevalence ($P = 0.0008$ and $P = 0.0075$, respectively).
Curtin ⁵⁶ (2005)	98 children with ADHD in a tertiary care clinic for developmental, behavioral, and cognitive disorders Age range: 3–18	29% of children with ADHD were at risk for overweight and 17.3% were overweight. No significant difference in comparison to an age-matched reference population. However, the prevalence of at risk for overweight and overweight in children not treated with ADHD drugs (36% and 23%, respectively) was significantly higher than that found in treated participants (16% and 6.3%, respectively) ($P < 0.05$)
Faraone ⁵⁷ (2005)	568 children with ADHD enrolled in a study of the safety of mixed amphetamine salts	At baseline, subjects were heavier than average (mean BMI z-score = 0.41).
Hubel ⁵⁸ (2006)	39 boys with ADHD and 30 healthy controls Age range: 8–14	BMI-SDS was higher in ADHD than in controls. No significant association between group membership (control vs ADHD) and obesity or overweight.
Anderson ⁵⁹ (2006)	655 subjects (general population) younger than 16.6	Subjects with ADHD had higher mean BMI z-scores at all ages compared with individuals who were not observed with a disruptive disorder.
Spencer ⁶⁰ (2006)	178 children with ADHD receiving OROS methylphenidate Age range: 6–13	Subjects were slightly overweight compared with that expected for their age (mean BMI z-score = 0.230).
Swanson ⁶¹ (2006)	140 children with ADHD Age range: 3–5.5	The average BMI was 16.9, which corresponds to the 86th percentile at the baseline assessment.
Ptáček ⁶² (2009)	46 nonmedicated ADHD boys Mean age : 11.03	Boys with ADHD had significantly higher values of percentage of body fat and abdominal circumference.
Pagoto ⁶³ (2009)	6,735 US residents Age range: 18–44	Obesity was more prevalent among persons with adult ADHD (29.4%) than among those with a history of childhood ADHD but no adult

		symptoms (23.7%) and those with no history of ADHD (21.6%).
Yang⁶⁴ (2013)	158 children with ADHD Mean age: 9.2	Prevalence of obesity, overweight, and combined obesity/overweight was 12.0%, 17.1%, and 29.1%, respectively, which were significantly higher than in the general Chinese population (2.1%, 4.5%, and 6.6%, respectively).
Cortese⁶⁵ (2013)	≥ 20	Persistent, lifetime or remitted ADHD were not associated with obesity after controlling for confounders. The number of childhood ADHD symptoms was significantly associated with adult obesity, even after adjustment, in women.

Table 3. Studies assessing the prevalence of ADHD in individuals with obesity.

First author (year)	Sample characteristics	Main findings
Altfas¹⁵ (2004)	215 patients with obesity treated in a specialized obesity clinic (Males: 22; mean age: 43.4 ± 10.9 years)	Prevalence of ADHD in the whole sample: 27.4%. Prevalence of ADHD in individuals with BMI ≥ 40 kg/m ² : 42.6%. Mean BMI loss among patients with ADHD: 2.6 BMI (kg/m ²) vs. 4.0 for non-ADHD (p < 0.002)
Ereerimis⁶⁶ (2004)	30 adolescents with obesity (Males: 14; mean age: 13.8 ± 1.2 years) seeking treatment in a paediatric endocrinology outpatient clinic	Prevalence of ADHD: 13.3%
Agranat-Meged⁶⁷ (2005)	26 adolescents in a tertiary referral centre for obesity (Males: 13; mean age: 13.04 ± 2.8 years), all with morbid obesity (BMI > 95 percentile)	57.7% of the subjects presented with ADHD diagnosed with semi-structured interviews
Fleming⁶⁸ (2005)	75 women with severe obesity (BMI ≥ 35 kg/m ²) (mean age: 40.4 ± 7.25 years) referred for non surgical treatment of obesity	26.7% of women reported impairing symptoms of ADHD in both childhood and adulthood
Alfonsson⁶⁹ (2012)	187 individuals (Males: 50; mean age: 44.28 ± 6.02 years) with obesity, candidate for	10% of the subjects presented with ADHD. ADHD symptoms

	bariatric surgery	significantly correlated with anxiety, depression, and disordered eating (“lack of control over eating”, “eating alone because embarrassed”, “eating until feeling uncomfortable”, and “feeling guilty after overeating”)
Gruss ⁷⁰ (2012)	116 patients (Males: 31; mean age: 44.28 ± 6.02 years) candidate for bariatric surgery	12% of the patients screened positive for ADHD. Rates of Binge Eating disorder did not differ between patients with and without ADHD
Nazar ⁴⁷ (2012)	150 women (mean age: 38.9 ± years)	Prevalence of ADHD: 28.3%. ADHD was significantly correlated with more severe binge eating, bulimic behaviors, and depressive symptoms severity

Whilst results from the first type of studies are overall mixed, probably due to heterogeneity in terms of definition of ADHD, definition and measurement of obesity, and referral bias, the second type of studies consistently showed significantly higher rates of ADHD in individuals with obesity compared to those in the general population. This association might at first seem paradoxical, since one would think that children with ADHD should be thinner than the average due to their hyperactivity. However, it is well known that the motor hyperactivity of ADHD is not constant but is modulated by the context. Of note, little difference in hyperactivity levels between children with ADHD and healthy comparisons was detected while watching television and children with ADHD watch more television and engage in less physical activity than comparisons¹⁴. Moreover, it is supposed that both the inattentive and impulsive component of ADHD may play a role in the association, whilst the hyperactive component may not be relevant (see below). Finally, we note that significantly higher rates of obesity in children with ADHD do not exclude that children with ADHD might present also with significantly higher rates of underweight; in other terms, both overweight and underweight children might be more represented among children with ADHD compared to normal population.

Besides these cross-section studies, to date three studies¹⁶⁻¹⁸ have shown that individuals with ADHD in childhood or young adulthood are at significantly higher risk of obesity/overweight compared to individuals without ADHD.

Possible mechanisms underlying the association between ADHD and obesity

Several mechanisms have been hypothesized to explain a possible association between ADHD and obesity.

From a theoretical point of view, it is possible that: 1) obesity and/or factors associated with obesity (such as sleep disordered breathing) lead to ADHD symptomatology; 2) ADHD and obesity share common biological dysfunction; 3) ADHD increases the risk of obesity.

1) since a subgroup of patients with obesity, especially those with severe obesity¹⁹ may present with binge eating, impulsivity associated with abnormal eating behavior might foster symptoms of inattention and hyperactivity. Indeed, patients with bulimic or abnormal eating behaviors may present with repeated and impulsive interruptions of their activities in order to get food, resulting in ADHD symptoms such as disorganization, inattention, and restlessness²⁰.

Another hypothesis is based on the link between hypoarousal and ADHD symptoms. It has been hypothesized that excessive daytime sleepiness (EDS), due to sleep disordered breathing (SDB) or independent from it, may contribute to ADHD symptoms. According to the “hypoarousal theory” of ADHD proposed by Weinberg et al.²¹, subjects with ADHD behaviors (or at least a subgroup of them) might actually be sleepier than controls and might use motor hyperactivity and impulsivity as a strategy to stay awake and alert, in order to counteract the tendency to fall asleep. Several studies indeed reported a significant association between obesity and sleep-disordered breathing (SDB) or other sleep disorders²². These disorders may cause sleep fragmentation, leading to excessive daytime sleepiness. Moreover, as reported by Vgontzas et al.²³, obesity may be significantly associated with EDS independently of sleep-disordered breathing (SDB) or any other sleep disturbances. Vgontzas et al.²⁴ suggested that, at least in some obese patients, EDS may be related to a metabolic and/or circadian abnormality associated with obesity more than being the consequence of SDB or other sleep disturbances. This hypothesis has been tested by Cortese et al.²⁵ in a sample of 70 obese children (age range: 10-16 years); the authors found that scores of excessive daytime sleepiness on the SDSC were significantly associated with symptoms of inattention, hyperactivity and impulsivity.

2) Another possibility is that obesity and ADHD share common underlying biological mechanisms. The “reward deficiency syndrome” may play a significant role. This syndrome is characterized by an insufficient dopamine-related natural reward that leads to the use of ‘unnatural’ immediate rewards, such as substance use, gambling, risk taking and inappropriate eating. It has been shown that individuals with ADHD may present with behaviors consistent with the “reward deficiency syndrome” (e.g. ^{26, 27}). This syndrome has been reported also in obese patients with abnormal eating behaviors²⁸. Alterations in the dopamine receptor D2 (DRD2)²⁹ and, to a lesser extent, DRD4^{30, 31} have been associated with the above-mentioned “reward deficiency syndrome”. Dysfunctions of DRD2 and DRD4 have been found in obese patients³². Several studies suggest a role of altered DRD4 and DRD2 in ADHD as well (although the alteration in DRD2 has not been replicated in other studies)³³.

Another potential common biological mechanism might involve alterations in the Brain Derived Neurotropic Factor (BDNF). Preliminary evidence from animal model studies points to a potential

dysfunction of BDNF underlying both ADHD and obesity^{34, 35}. Interestingly, Gray et al.³⁶ reported a functional loss of one copy of the BDNF gene in an 8-year old with hyperphagia, severe obesity, impaired cognitive function, and hyperactivity.

Another putative neurobiological pathway relates to the Melanocortin-4-Receptor (MC4R) Deficiency, which has been reported to disrupt the pathway of hunger/ satiety and result in abnormal eating behaviors. Agranat-Meged et al.³⁷ analysed 29 subjects (19 males and 10 females) from 5 “proband nuclear families” with morbid obese children (BMI percentile>97%), and reported that the prevalence of ADHD was significantly higher than expected only in the groups carrying the homozygous or heterozygous mutation.

3) Finally, it is possible that ADHD actually contributes to obesity. Abnormal eating behaviors associated with ADHD may play a significant role. In a study on 110 adult healthy women, Davis et al.³⁸ found that ADHD symptoms and impulsivity were significantly correlated with abnormal eating behaviors, including binge eating and emotionally-induced eating, which, in turn, were positively associated with BMI. This results have been recently replicated in males³⁹. Cortese et al.²⁰ found that, after controlling for potentially confounding depressive and anxiety symptoms, ADHD symptoms, measured by the ADHD-index score of the Conners Parents Rating Scale (CPRS), were significantly associated with bulimic behaviors.

At the present time, it is not clear which dimension of ADHD (inattention, hyperactivity, or impulsivity) may specifically be associated with abnormal eating behaviors. Cortese et al.²⁰ speculated that both a) impulsivity and b) inattention might lead or contribute to abnormal eating behaviors, whereas hyperactivity would not play a significant role.

a) As for impulsivity, Davis et al.³⁸ suggested that both *deficient inhibitory control* could lead to over-consumption when not hungry associated with the relative absence of concern for daily caloric intake.

b) It is also possible that inattention and deficits in executive functions (which, as stated in the introduction, are frequently associated with ADHD), cause difficulties in adhering to a regular eating pattern, favouring abnormal eating behaviors. Davis et al.⁷⁶ pointed out that patients with ADHD may be relatively inattentive to internal signs of hunger and satiety. Therefore, they may forget about eating when they are engaged in interesting activities and they may be more likely to eat when less stimulated, at which point they may be very hungry.

Another explanation on the association between inattention and obesity was provided by Schweickert et al.⁸⁵ who hypothesized that compulsive eating may be a compensatory mechanism to help the person control the frustration associated with attentional and organizational difficulties, although this would not be specific for ADHD. Levitan et al.⁶⁹ hypothesized that difficulties initiating activities linked to attentional and organizational difficulties contribute to decreased caloric expenditure, leading to weight gain over time.

Riverin and Tremblay⁴⁰ hypothesized that, since patients with ADHD are susceptible to commit more

cognitive effort to take in charge standard mental tasks, it is likely that this cognitive effort accentuates their the proneness to hyperphagia and the consequent long-term weight gain.

Finally, Waring et al.⁴¹ evoked also the role of television. Since, as they state, children with ADHD may spend more time watching television or playing computer or video games, this may contribute to weight gain. A large epidemiological study⁴² provided support to this hypothesis.

Clinical and public health implications

Some studies point to the utility of recognizing and possibly treating ADHD in individuals with obesity. In the above-mentioned study by Altfas¹⁵, BMI loss in patients without ADHD (11.2%) was nearly twice that in patients with ADHD (6.5%) or subthreshold ADHD (6.3%). Additionally, although the average number of months in treatment did not differ significantly among the three subgroups, the mean number of visits was significantly higher in patients with ADHD compared to those with subthreshold ADHD or without ADHD. Therefore, the study by Altfas suggested that screening for ADHD in patients with obesity may be useful since it may identify a subgroup that, despite considerable efforts (higher number of visits), struggles with weigh loss significantly more than obese patients without ADHD.

Subsequent data from Pagoto and colleagues⁴³ confirmed and expanded the preliminary observations by Altfas¹⁵. These authors assessed a sample of 63 adults (42 females, mean age 50 ± 10 y) enrolled in a behavioral weight loss program. Of these, 30% reported symptoms consistent with a diagnosis of ADHD, although a formal diagnosis of ADHD was not established. While there was no significant difference in BMI at the beginning of the program between subjects with (ADHD+) and without (ADHD-) an estimated diagnosis of ADHD, the ADHD+ group lost significantly less weight ($3.3\% \pm 3.5$) than the ADHD- group ($5.6\% \pm 3.4$) at 4-month follow-up. Additionally, ADHD+ patients reported significantly more short-lived (fewer than 3 days) weight loss attempts, more fast food meals per week, had higher emotional eating scores, and rated weight loss skills as more difficult to carry out than ADHD- individuals. Another more recent study⁴⁴ extended these results showing that, among patients presenting for bariatric surgery ($n= 60$, 78.3% females), those with comorbid ADHD ($n=19$) had significantly more difficulties in following visits at 12-month follow-up after bariatric surgery than those without estimated comorbid ADHD. One way ADHD hampers weight loss efforts may be through negatively impacting physical activity. Two large epidemiological studies provided support for this hypothesis. In the first, Cook et al.⁴⁵ assessed a nationally representative sample of youth in the U.S. ($n= 45,897$, age range: 10-17 years) and found that youth with ADHD participated in fewer activities and organized sport than those without ADHD ($n=29,801$) [Odds ratio: (95% CI): 1.57 (1.06-2.34)]. ADHD may also be a barrier to effective and persistent weight loss by contributing to dysregulated, abnormal eating patterns, as showed by a clinical study of 51 adults with obesity who screened positive in the adult ADHD (ADHD+) and 127 comparisons without symptoms of ADHD (ADHD-); The study.⁴⁶ found that the ADHD+ group had significantly more abnormal eating behaviors, such as binge eating episodes, waking up at night to eat, and eating in secret and found not only that youths with ADHD were at significantly higher risk of obesity compared to those without ADHD, but also that they were significantly less likely to meet recommended levels of physical activity, even after controlling for some possible confounding factors such as SES and race [Odds ratio: 0.427 (95% CI: 0.245-0.742)]. In the second study, a cross-sectional analysis of children (aged 6-17 years) from the national Survey of Children's Health ($n= 66,707$), Kim et

al.⁴² found that children with ADHD (n= 1757) engaged significantly less in physical activity (p=0.01) than the ADHD- participants. These results are in line with those by Nazar et al.⁴⁷ who, in a clinical sample of 155 women (mean age: 38.9±10.7 years) found a prevalence of ADHD of 28.3% [n= 30, (95% CI: 23.8%-32.8%)].

Although the design of all the previous studies could not establish causality, it is possible that, if ADHD is associated with unsuccessful weight loss in individuals with obesity involved in weight loss programs, ADHD treatment may improve the effectiveness of such programs. A preliminary study by Levy and colleagues⁴⁸ addressed the possible beneficial effects of the pharmacological treatment of comorbid ADHD in terms of obesity outcome. They assessed 242 individuals with a lengthy history of weight loss failure consecutively referred for refractory obesity to a specialized clinic, and found that 78 of them (32.2%) screened positive for ADHD. Of these, 65 started pharmacotherapy for ADHD with psychostimulants, in addition to standard management for weight loss, and were followed up for an average of 466 days. Those who refused pharmacological treatment or who did not tolerate it for adverse events (n=13) were also followed up as comparisons, and received standard care for weight loss management. At follow-up, individuals who received treatment lost 12.36% of their initial weight, whereas comparisons gained an average of 2.78% (p< 0.001). Clearly, a possible important confounder of this study, as well as of those previously mentioned on the effect of ADHD treatment on obesity rates in individuals with comorbid ADHD^{42, 49, 50}, is the anorexigenic effect associated with psychostimulant treatment⁵¹. However, to partially address this methodological concern, Levy et al.⁴⁸ noted that appetite reduction was evident in the first 4–6 weeks of treatment, but then it diminished and vanished in most subjects within 2 months. Therefore, the authors of the study concluded that it is unlikely that the anorexigenic effect of psychostimulants contributed to weight loss at follow-up, after more than one year from the start of treatment. It is important to note that, although the Levy et al.⁴⁸ study was controlled, it was not randomized, and, as such, could not address possible differences at baseline between the two study groups. Indeed, since pharmacological treatment for ADHD is effective and is recommended in several guidelines, randomization to pharmacological treatment vs. placebo could not be performed for ethical reasons.

Finally, a study assessed extent to which improvement of executive dysfunctions, instead of treatment of ADHD, improve obesity treatment outcome. Verbeken and colleagues⁵² evaluated the effects of video games aimed at improving inhibitory control and working memory. They randomized 44 children (8-14 years) who were in the final part of a 10-month inpatient treatment program in an obesity center to either 6-week executive function training or to standard weight control care. At 8 weeks post-training, children in the executive function training group showed significantly better weight loss maintenance than those in the standard care group. However, the effect became non-significant at 12-week follow-up, suggesting that booster sessions of cognitive training may be needed to achieve a longer lasting effect on obesity outcome.

Conclusion

Although more empirical studies are needed, current evidence points to an association between ADHD and obesity and suggest that screening for and treating ADHD in individuals with obesity might significantly improve the outcome of obesity treatment.

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