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Attention Deficit/Hyperactivity Disorder and the Clinical Management of Obesity

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Abstract Attention deficit/hyperactivity disorder (ADHD) has been associated with increased risk for obesity and obesity treatment failure. The present paper discusses how features of ADHD, including inattention, reward sensitivity, and impulsivity, may impact obesity risk and have implications for the management of obesity. In addition, we review emerging research on how obesity may contribute to brain changes that are associated with ADHD-like symptoms. Finally, suggestions for improving the clinical management of obesity in patients with ADHD are discussed, including pharmacological treatment, exercise, and cognitive behavior therapy. ADHD is a barrier to the clinical management of obesity and more research is needed to further understand the link between ADHD and obesity. Effective treatment approaches are needed given the significant difficulty patients with ADHD encounter in their attempts to regulate their weight in the context of an obesogenic environment.

Keywords ADHD · Attention deficit/hyperactivity disorder · Obesity · Impulsivity · Treatment

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Introduction

Attention deficit/hyperactivity disorder (ADHD) is a neurobehavioral disorder, essential features of which include a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than typically observed in individuals at a comparable level of development. The *Diagnostic and Statistical Manual 4th edition* (DSM-IV) [1] specifies that these symptoms must have persisted for at least 6 months, originated before the age of 7 years, and impairment is present in two or more settings. The DSM-IV describes three subtypes of ADHD: 1) inattentive type; 2) hyperactive-impulsive type; and 3) combined type. ADHD has been shown to persist into adulthood in half of children with ADHD [2] and affects over 9 million US adults [3]. An association between ADHD and obesity is well documented both in population [4, 5•, 6–8] and clinical samples [9–11].

In addition to ADHD being linked to greater risk for obesity, evidence is also emerging that ADHD may pose a significant barrier to the management of obesity [10, 12]. In the present paper we discuss the neurocognitive features of ADHD that may lead to obesogenic behavior and affect the

patient's ability to engage in the self-management skills that are instrumental to weight control. Treatment strategies for patients with comorbid ADHD and obesity are discussed as well as future directions for research on the clinical management of obesity in the patient with ADHD.

Inattention, Reward Sensitivity, and Impulsivity May Lead to Obesogenic Behavior

Some research has explored neurocognitive features of ADHD, including inattention, reward sensitivity, and impulsivity, to determine the extent to which these problems are associated with obesity and/or interfere with the ability to lose weight. Inattention, a hallmark of ADHD, refers to distractibility, reduced ability to sustain attention, and susceptibility to interference [13]. Inattention may adversely impact the individual's capacity to execute goal-directed dietary and physical activity behaviors and the ability to regulate one's weight resulting in irregular eating habits and poor adherence. Inattention might also affect awareness of internal hunger and satiety cues, especially when the individual is engaged in other activities [14, 15]. This could result in externally cued eating, which tends to be associated with overeating. Inattention may also interfere with other behaviors critical to successful weight loss, such as meal planning, implementation of specific behavioral skills (eg, consistently keeping a diet diary), following through with planned exercise, and the ability to detect changes in food portion sizes [16]. In some studies, the inattentive type of ADHD has been found to be more prevalent than the hyperactive/impulsive type in obese samples [12, 17, 18]. Additional research is needed to further describe specifically how inattention affects one's ability to regulate their weight, as well as interventions that could be helpful in this contest (eg, cues/reminders, frequent feedback, etc.).

Reward sensitivity is a biologically based personality trait originally described by Gray [19] that may stem from hypo- or hyper-reactivity to reward at the neurobiological level. High "reward sensitivity" describes individuals who are especially sensitive to rewards in their environment, while diminished reward sensitivity describes individuals who are especially insensitive to rewards in their environment. Recent evidence has emerged that individuals with ADHD, particularly those with the inattentive type, are highly reward sensitive [20]. Reward sensitivity is implicated in appetitive motivation, such that it may mediate responses to appetitive stimuli, including drugs, food, and sex [21]. Mesolimbic dopaminergic (DA) processes are central to reward sensitivity [22, 23], with greater sensitivity to reward at the behavioral level being associated with either a hyper- or hyporesponsive mesolimbic DA system [24–26]. Because palatable foods enhance dopamine activation, eating might serve a "self-medicating" function for some individuals with ADHD [27]. Given that ADHD is generally characterized by reward sensitivity, obese adults with ADHD may be more sensitive to rewards relative to their obese counterparts without ADHD, which might manifest in eating pathology. For example, Davis et al. [28•] found that individuals with ADHD were more likely to meet criteria for "food addiction" than those without ADHD. Similarly, binge eating disorder has been shown to play a mediating role in the relationship between ADHD and obesity [10].

Impulsivity is another common feature of ADHD that has implications for obesogenic behavior and possibly risk for obesity. Although it is a multifaceted construct, impulsivity can simply be characterized as action without foresight [29]. Some evidence suggests that people high in impulsivity are prone to overeating, weight gain [30–32], and obesity [33]. Among 228 adolescent girls with ADHD, impulsivity was a much stronger predictor of eating pathology than inattention, although both inattentive and impulsive ADHD subtypes were equally likely to be obese [34]. This suggests that impulsivity might be instrumental in the observed association between ADHD and binge eating disorder [5•, 35, 36]. Greater impulsivity could also lead to greater difficulty inhibiting the consumption of high energy–dense palatable foods, especially in response to stress and other cues, as described by Lowe et al. [37]. Cognitive models of ADHD emphasize the role of inhibitory control as a critical requirement for other executive functions that subserve self-regulation and goal-directed behavior [38]. Consuming a healthy diet in an obesogenic environment or restricting intake to lose weight can be thought of as goal-oriented behavior, thus any impairment in inhibitory control could hijack their optimal execution. The increasingly obesogenic environment may be particularly challenging for people high in impulsivity (ie, low inhibitory control), given the omnipresence of food cues. Supporting this contention is a study showing that impulsivity was associated with greater food consumption in an environment characterized by high food variety, but not in one characterized by monotonous foods [39]. This impulsivity-by-environment interaction suggests that persons with ADHD might be disproportionately vulnerable to obesogenic environments.

Impulsive individuals also appear to have less inhibitory control during hunger, as evidenced in a study that found that impulsive individuals consumed more food when exposed to highly palatable food than their less impulsive counterparts under conditions of hunger, but not under conditions of satiety [40]. These findings suggest that restrictive diets in the context of an obesogenic environment might be especially challenging for people with ADHD. Accordingly, Nederkoorn et al. [41] reported that impulsivity predicted less weight loss following treatment in children.

One particular manifestation of impulsivity, delay discounting, seems particularly relevant to understanding the association between ADHD and obesity. Delay discounting reflects the degree to which an individual undervalues rewards with increasing delays [42]. For both humans and animals, immediate rewards have a disproportionately strong influence on our decisions and actions. Less valuable immediate rewards (eg, $200 today) can often be preferred to more valuable future rewards (ie, $240 in 3 months). A number of studies have found steeper delay discounting among children and adults with ADHD relative to controls [43, 44]. These effects appear to be more robust among those with the hyperactive/impulsive or combined subtypes [44–46], which is consistent with the conceptualization of delay discounting as a facet of impulsivity. The tendency to discount delayed rewards steeply is associated with several conditions in which impulsivity and hyper-responsivity to appetitive cues are thought to play an etiological role, such as compulsive gambling, tobacco use, and drug addiction [47–49].

Recently, delay discounting has also been linked to eating behavior and obesity [50•, 51]. For example, obese women show steeper discounting of delayed rewards than normal weight women [52], and greater delay discounting has been associated with higher intake of palatable food among lean, overweight, and obese subjects who are most sensitive to the rewarding properties of food [53, 54]. It has been hypothesized that a greater predisposition for delay discounting would likely manifest as heightened sensitivity to the immediate reward from palatable food and decreased valuation of the long-term rewards associated with a healthy diet and weight maintenance, which in combination would contribute to obesity [55, 56]. The neurobiological mechanisms underlying individual differences in delay discounting have also been linked to the neurocognitive deficits in ADHD and the brain regulation of eating behavior. Specifically, alterations in mesolimbic dopamine function have been associated with a stronger preference for immediate rewards in the context of delay discounting tasks, palatable food intake, and ADHD [20, 57–60]. In contrast, "executive" brain regions found to be altered in ADHD, particularly the lateral prefrontal cortex, appear to play a critical role in inhibiting impulsive behaviors and pursuing long-term goals, including sustained weight loss [50•, 61–65]. Thus, heightened delay discounting appears to stem from a neurocognitive profile implicated in both ADHD and obesity. Developing a regular pattern of eating and the removal of food cues in the home environment may be helpful for the impulsive individual, in addition to assistance in setting shorter-term goals. Additional research is needed to identify effective weight loss strategies for individuals who are high in impulsivity.

Chicken or the Egg? Obesity May Produce ADHD-Like Symptoms

Aside from common underlying mechanisms between ADHD and obesity and the impact that ADHD may have on weight gain and obesity, there is some, albeit limited, evidence for the possibility that obesity contributes to the development of ADHD-like symptoms. For example, short-term experimental overfeeding in lean humans has been shown to lead to brain changes including an increase in the activation of the default mode network (DMN), a set of areas that are active typically at rest and seem to be involved in self-referential cognition and introspection [66]. This effect goes in the direction of ADHD-like brain changes, characterized by DMN overactivation and difficulty in suppressing this network during effortful tasks [67, 68]. Additional evidence comes from a study showing improvements in attention/executive functioning in obese subjects following weight loss [69]. Animal studies provide more direct evidence in support of this association. Diet-induced obesity in a swine model leads to changes in brain perfusion that are selectively located in areas implicated in ADHD, such as the dorsolateral prefrontal cortex, and the amount of weight gain negatively correlates with perfusion in these locations [70]. Lastly, a brain effect of obesity is supported by biological plausibility, as obesity-related changes can affect neuroplasticity and cognitive function through different mechanisms (eg, the release of proinflammatory substances to the systemic circulation) [71]. Altogether, the evidence suggests that obesity may worsen ADHD symptomatology or facilitate the development of ADHD-like symptoms via brain changes; however, direct evidence for this association in humans is not available yet and future studies are warranted. To the extent that obesity-induced brain changes impair weight control, patients with long-standing obesity and more severe obesity may be the most treatment resistant. Research is needed to determine whether these brain changes are reversible via weight loss and/or lifestyle changes (eg, caloric restriction, exercise).

Obesity Management in the Patient with ADHD

Because the association between ADHD and obesity has only recently been observed, specialized interventions have not been developed; however, some evidence suggests that individuals with ADHD are less responsive to standard behavioral interventions for obesity. Only two studies have examined weight loss treatment outcomes in individuals with ADHD and both found that these patients lost less weight in a behavioral weight loss program than their counterparts without ADHD [10, 12]. Altfas [12] conducted a retrospective chart review for 215 weight loss clinic patients

and observed that patients who met diagnostic criteria for ADHD achieved only 65% of the weight loss of patients without ADHD (2.6 vs 4.0 kg, respectively). In spite of poorer weight loss outcomes, patients with ADHD had a greater number of visits than their non-ADHD counterparts (mean, 56.6 vs 39.4 visits), and although not statistically significant, longer treatment duration (38.7 vs 28.6 months). Pagoto et al. [10] also documented weight loss in 63 patients of an outpatient behavioral weight loss program. Similar to Altfas [12], patients screening positive for ADHD lost only 60% of the weight of their counterparts who screened negative (mean weight loss, 3.72 vs 6.17 kg, respectively). Participants with ADHD symptoms reported significantly more short-lived (< 3 days) weight loss attempts than those who denied such symptoms. They had significantly more frequent fast food consumption, higher emotional eating scores, rated weight loss skills as more difficult, and reported lower self-efficacy to control their eating. Although more research is needed comparing weight loss outcomes among patients with and without ADHD, these studies suggest that patients with ADHD lose significantly less weight than their counterparts and may have more difficulty managing their intake.

Improving Clinical Management of Obesity in Patients with ADHD

Given that symptoms of ADHD appear to be associated with obesogenic behavior and greater difficulty losing weight, obesity treatment approaches that offset these symptoms are needed. Research supports the potential for psychopharmacology, physical activity, and cognitive behaviorial therapy (CBT) to assist in the clinical management of obesity in this population.

Psychopharmacology

Psychopharmacology, including psychostimulants, some antidepressants, and norepinephrine reuptake inhibitors, is considered the first-line treatment in ADHD for both children and adults [72, 73]. These agents have been shown to improve clinical and core symptoms of ADHD, including vigilance, divided attention, focused attention, and cognitive flexibility in adults [74]. Medications appear to have an impact on risk for obesity among children with ADHD. Two observational studies of children showed that ADHD was associated with obesity only in children who were not using medication for their ADHD, but not in children who were medicated [6, 75]. Other research suggests that medications for ADHD may cause weight loss. For example, an observational study by Levy et al. [76] followed a sample of 78 obese adult patients diagnosed with ADHD who underwent pharmacological treatment (primarily stimulants) for ADHD for a mean of 466 days. Patients who stayed on the medication (83%) lost a mean of 15.05 kg (12.36%), while patients who went off the medication gained 3.26 kg (2.78%). Although not a randomized controlled trial, this amount of weight loss among individuals with ADHD is far higher than observed in other studies that did not utilize medication (eg, [10, 12]). Although concerns had been raised about the cardiovascular side effects of stimulant medications, a large population-based study recently showed no impact of stimulants on cardiovascular events in young and middle-aged adults [77].

Others studies have shown a weight loss effect of medications that are often used to treat ADHD in obese patients who do not have ADHD. Anderson et al. [78] conducted a double-blind, placebo-controlled, randomized trial of obese adults who were assigned to placebo, bupropion sustained-release 300 or 400 mg/d. All participants received behavioral weight loss counseling and meal replacements. Results indicated that bupropion yielded statistically and clinically significant net weight losses relative to placebo. A second study by Gadde et al. [79] randomized 30 obese women to atomoxetine, a norepinephrine reuptake inhibitor, or placebo with both conditions receiving a behavioral weight loss program. Patients in the atomoxetine group lost significantly more weight than did controls (-3.6 [1.0] vs 0.1 [0.4] kg, respectively). Three other studies have shown that methylphenidate acutely suppressed appetite and energy intake [80–82]. Although only 25% of adults with ADHD are estimated take medication to control their symptoms [3], stimulants and other ADHD medications may be a particularly important treatment option to consider for adults with comorbid ADHD and obesity.

Exercise

Exercise may be especially beneficial for patients with comorbid ADHD and obesity. Both human and animal studies strongly support the impact of exercise on improved cognitive function [83]. Exercise has been found to improve executive function and increase prefrontal activation during executive function tasks in children [84•], and some initial work has documented that exercise in children with ADHD has yielded improvements in overall behavior, sustained attention, vigilance, and impulsivity [85, 86]. Exercise increases brain-derived neurotrophic factor, a molecule involved in synaptic plasticity, learning, and memory, which may be particularly important for individuals with ADHD [83]. Additionally, Gapin et al. [87] reported some preliminary, albeit indirect, evidence that exercise increases dopamine levels in children with ADHD [87]. Exercise might be a first step in treatment for patients with comorbid obesity and ADHD given that its cognitive benefits might also result

in improved self-regulation, which could facilitate adherence to other weight loss strategies [88].

Cognitive Behavioral Therapy

A version of CBT [89] has been developed specifically for adults with ADHD to reduce ADHD symptoms and improve overall functioning. Because ADHD symptoms interfere with the execution of weight loss skills, CBT for ADHD might be a useful adjunct to obesity treatment among adults with ADHD. CBT provides patients with the opportunity to learn the functional skills that do not come naturally to them (eg, time/task management, organizational skills, and task analysis), resulting in new skills that can enhance functioning and effectiveness in occupational, scholastic, and interpersonal domains. CBT also incorporates cognitive modification strategies that assist the patient in identifying automatic thoughts, recognizing the ensuing feelings and behaviors that lead to maladaptive responses, and in reframing and problem solving to devise and implement more adaptive behaviors. Several studies have shown the efficacy of CBT in improving ADHD symptoms [89–92], organizational skills [91], depression and anxiety [89] in patients with ADHD. Research is needed to determine whether the incorporation of CBT strategies for ADHD into a behavioral weight loss program would improve both ADHD symptoms and weight loss.

Bariatric Surgery

Although bariatric surgery is often indicated for individuals who have repeatedly failed to lose weight in structured programs and/or on their own, individuals with ADHD may experience the same difficulties with weight control following bariatric surgery, and therefore may be at higher-than-average risk for regain. Assessment for ADHD symptoms should be included in the pre-surgery screening process and extra support provided for these patients post surgery. The Adult ADHD Self-Report Scale is a brief, validated measure that is feasible for clinical settings [93]. We are aware of no studies that have evaluated bariatric surgery outcomes among individuals with ADHD; such research is needed. Research on whether bariatric surgery impacts executive function, inhibitory control, and other symptoms of ADHD would also broaden our understanding of how obesity and weight loss affect these symptoms.

Conclusions

ADHD is a risk factor for both obesity and obesity treatment failure. Inattention, reward sensitivity, and impulsivity are features of ADHD that may contribute to overeating, eating pathology, poor adherence to structured attempts to lose weight, and increased vulnerability to obesogenic environments. When ADHD is suspected in an obese patient, referral for assessment and treatment is highly recommended. The clinician should note that people with ADHD may experience greater than average difficulty with organization, self-monitoring, planning, follow through on goals, resisting food temptations, and consistency. These symptoms should not be confused with poor motivation or irresponsibility in the patient who is unsuccessful at controlling his/her weight. It should also be considered that even in the absence of an ADHD diagnosis, obesity may be associated with brain changes that produce symptoms that mimic ADHD. Patients with comorbid obesity and ADHD likely require extra support and more intensive weight loss intervention to overcome these challenges. In addition to pharmacological treatment, exercise and CBT are two potentially helpful adjuncts to weight loss treatment for those with ADHD. Further research is needed to understand the association between obesity and ADHD and/or ADHD-like symptoms.

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