Attention-Deficit/Hyperactivity Disorder (ADHD) and Binge Eating

Samuele Cortese, MD, Bernardo Dalla Bernardina, MD, and Marie-Christine Mouren, MD

Attention-deficit/hyperactivity disorder (ADHD) is characterized by a persistent and pervasive pattern of inattention and/or hyperactivity-impulsivity. Emerging data suggest higher than expected rates of binge eating occur in subjects with ADHD. Several hypotheses may explain this newly described comorbidity: 1) inattention and/or impulsivity foster binge eating, 2) ADHD and binge eating share common neurobiological bases, 3) binge eating contributes to ADHD, or 4) psychopathological factors common to both binge eating and ADHD mediate the association. In patients with ADHD and binge eating, both conditions might benefit from common therapeutic strategies. Further research is needed to gain insight into the association between ADHD and binge eating in order to facilitate more appropriate clinical management and, ultimately, a better quality of life for patients with both conditions.

Key words: attention-deficit/hyperactivity disorder, ADHD, binge eating, bulimic behaviors

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INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is among the most common and impairing childhood psychiatric conditions; it is estimated to affect 5–10% of school-aged children.1 According to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV),2 ADHD is characterized by pervasive and developmentally inappropriate symptoms of inattention and/or impulsivity-hyperactivity. The DSM-IV3 and its updated version (DSM-IV-TR) define four types of ADHD: 1) predominantly inattentive, 2) predominantly hyperactive-impulsive, 3) combined, and 4) not otherwise specified. Delay aversion and deficits in executive functions (i.e., neuropsychological functions including inhibition, working memory, planning, and sustained attention) are common in patients with ADHD.4 Impairing symptoms of ADHD may persist into adulthood in up to 60% of cases.5 Stimulants (methylphenidate and amphetamines) are the first-line FDA-approved pharmacological treatment, followed by the non-stimulant atomoxetine.6 ADHD is often associated with other disorders, which contribute to the functional impairment of the patient. Commonly reported comorbid psychiatric disorders include oppositional defiant disorder, conduct disorder, anxiety disorders, depressive disorders, and speech and learning disorders.4,7 Although overlooked in the past, a potential comorbidity between ADHD and binge eating behaviors has been suggested by recent empirically based evidence.8-10

Better insight into this potential comorbidity is of relevance for two reasons. First, it may contribute to the understanding of possible pathological mechanisms underlying both ADHD and binge eating, at least in a subset of patients. Second, from a clinical standpoint, it might have important implications for the management of patients with both ADHD and binge eating, suggesting common and potentially effective therapeutic strategies for these two conditions when they coexist. This seems particularly noteworthy because of the personal and social adverse outcome associated with both ADHD and binge eating behaviors.

In light of these considerations, the aims of this brief review were as follows: 1) to critically review evidence on the relationship between ADHD and binge eating, 2) to examine the mechanisms proposed to underlie the comorbidity between ADHD and binge eating, and 3) to discuss potential strategies for the clinical management of patients who present with both ADHD and binge eating.
**EVIDENCE ON THE RELATIONSHIP BETWEEN ADHD AND BINGE EATING**

Analyzing data from the Minnesota Adolescent Health Survey, Neumark-Sztainer et al.\(^1\) compared the rates of abnormal eating behaviors (including binge eating) among 2149 adolescents with chronic illness and 1371 controls without chronic illness. The chronic illness group included 689 subjects (286 boys, 403 girls) with attention deficit disorder (ADD). It should be noted that the survey was conducted during the 1986–1987 school year; at that time, the then-current version of the DSM, i.e., the DSM-III Revised,\(^2\) included the definition of “attention deficit disorder with or without hyperactivity”, conditions that are similar to the “combined” and “inattentive” types, respectively, which are outlined in DSM-IV.\(^2\) The authors found that subjects with attention deficit disorder were significantly more likely to binge than were controls (\(P=0.001\)). The major limitation of this study was that, given the large sample size, the diagnosis of ADD and eating practices were self-reported. However, the study’s results are of interest because they set the basis for more accurate further investigation in clinical settings.

In a study of 86 adults with ADHD (diagnosed according to DSM-IV criteria),\(^3\) Mattos et al.\(^4\) found that 8.3% presented with binge eating disorder (BED). The prevalence was higher than expected, since approximately 2.6% of the general adult population present with BED.\(^5\) Interestingly, patients with ADHD plus BED had significantly higher rates of comorbid psychiatric disorders than those with ADHD without BED. The authors correctly pointed out that since only two DSM-IV criteria for BED involve impulsivity or its correlates, ADHD impulsivity per se would not be enough to warrant a BED diagnosis. This suggests that the association between ADHD and BED is not an artefact of similar symptomatology but is a true comorbidity. Although the lack of a control group is a limitation in this study, this was the first study to assess BED rates in subjects with ADHD using a clinical structured interview.

Recently, Surman et al.\(^6\) analyzed data from four large case-control studies (two\(^7,\)\(^8\) in children aged 6–17 years and two\(^9,\)\(^10\) in adults) including, in total, 600 ADHD patients and 664 controls. The diagnosis of ADHD was based on structured or semi-structured interviews conducted according to DSM criteria. The authors found significantly greater rates of bulimia nervosa (which includes binge as well as purging behaviors) in women with versus without ADHD (\(P<0.05\)), but not in men or children. This finding led the authors to hypothesize that significant rates of bulimia nervosa emerge in adulthood in women with ADHD. However, as correctly pointed out by the authors, since bulimia nervosa commonly onsets in late adolescence, and only 20% of the ADHD girls and 26% of the control girls were 15 years old or older, the ability to fully assess bulimia nervosa in this sample of girls was limited.

In a study of 110 women (aged between 25 and 46 years) from the general population, Davis et al.\(^11\) assessed childhood ADHD symptoms and abnormal eating behaviors. Structural equation modelling revealed that ADHD symptoms in childhood significantly predicted abnormal eating behaviors, including binge eating (\(P=0.001\)). Several limitations should be noted, including the retrospective evaluation of ADHD symptoms, the lack of a formal diagnosis of ADHD, and the cross-sectional nature of the study, which prevents a causal interpretation of the relationships among the parameters. However, the structural equation model is well suited to the management of cross-sectional data for inferential purposes; therefore, the results of this study are of interest since they suggest possible mechanisms linking ADHD traits and eating behaviors (i.e., ADHD traits may predict abnormal eating behaviors).

As for the prevalence of ADHD in patients with binge eating behaviors, Sokol et al.\(^12\) evaluated ADHD symptoms in childhood and in the past year in six female bulimic patients with cluster B personality disorders (mean age, 30.8 years) and seven normal control women (mean age, 28.9 years). (Cluster B personality disorders include histrionic personality, narcissistic personality, antisocial personality, and borderline personality.) The subjects fulfilled a modified version of the Conners scale, a widely used questionnaire for the assessment of ADHD symptoms.\(^13\) In terms of current symptoms on the Conners scale, the scores were significantly higher (\(P=0.002\)) in bulimic patients than controls. For ADHD symptoms in childhood, the bulimic patients still had higher scores, with a trend toward statistical significance (\(P=0.085\)). The authors did not report whether any of these subjects met the full DSM criteria for ADHD. Moreover, given the small sample size, the results should be considered with caution. To our knowledge, no other studies to date have assessed the prevalence of ADHD in subjects with binge eating behaviors.

The key results of the above-mentioned studies are summarized in Table 1. In addition to these empirically based studies, several case reports and case series\(^14-\)\(^16\) suggest an association exists between ADHD and binge eating behaviors.

In summary, there is emerging evidence that binge eating or eating disorders with binge eating behaviors (such as BED or bulimia nervosa) occur at higher than expected rates in subjects with ADHD. On the other hand, empirically based evidence on the prevalence of ADHD in subjects with binge eating behaviors is still very limited.
Table 1. Studies on the Relationship between ADHD and Binge Eating Behaviors

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study design</th>
<th>Subjects</th>
<th>Age range (y)/mean age (y) (mean±SD)</th>
<th>Key results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biederman (2004)</td>
<td>Case-control study</td>
<td>219 with ADHD (137 male, 82 female); 215 controls (134 male, 81 female)</td>
<td>Patients, 37.6±10.5; controls, 38.7±4.2</td>
<td>Significantly greater rates of bulimia nervosa (including binge as well as purging behaviors) in women with versus without ADHD ($P&lt;0.05$); bulimia nervosa rates negligible in men</td>
<td>Strength: diagnosis of bulimia nervosa and ADHD according to standardized criteria</td>
</tr>
<tr>
<td>Biederman (1999)</td>
<td>Case-control study</td>
<td>140 girls with ADHD; 122 girls without ADHD</td>
<td>6–17 for both groups</td>
<td>1% of girls with ADHD and no girls without ADHD met full criteria for bulimia nervosa</td>
<td>Strength: diagnosis of bulimia nervosa and ADHD according to standardized criteria; limitation: ability to fully assess bulimia nervosa was limited since only 20% of the ADHD girls and 26% of the control girls were aged 15 years or older (bulimia nervosa commonly onsets in late adolescence)</td>
</tr>
<tr>
<td>Biederman (1994)</td>
<td>Case-control study</td>
<td>59 males with ADHD; 42 females with ADHD; 97 male controls; 110 female controls</td>
<td>36.9±8.4; 39.3±10.0; 40.1±7.1; 38.0±6.9</td>
<td>Significantly greater rates of bulimia nervosa (including binge behaviors) in women with versus without ADHD ($P&lt;0.05$); bulimia nervosa rates negligible in men</td>
<td>Strength: diagnosis of bulimia nervosa and ADHD according to standardized criteria</td>
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<tr>
<td>Biederman (1992)</td>
<td>Case-control study</td>
<td>140 boys with ADHD; 120 boys without ADHD</td>
<td>6–17 for both groups</td>
<td>No boys with or without ADHD had a bulimia nervosa diagnosis</td>
<td>Strength: diagnosis of bulimia nervosa and ADHD according to standardized criteria</td>
</tr>
<tr>
<td>Davis (2006)</td>
<td>Cross-sectional study</td>
<td>110 women from the general population</td>
<td>25–46</td>
<td>Using structural equation modelling, childhood ADHD symptoms significantly predicted abnormal eating behaviors, including binge eating ($P=0.001$)</td>
<td>Limitations: 1) retrospective evaluation of ADHD symptoms; 2) lack of structured interviews to diagnose ADHD</td>
</tr>
<tr>
<td>Mattos (2004)</td>
<td>Cross-sectional study</td>
<td>86 ADHD adult patients (sex ratio not specified)</td>
<td>18–52</td>
<td>Higher than expected prevalence (8.13%) of BED in ADHD subjects (BED prevalence in general population: 2.6%)</td>
<td>Strength: diagnosis of BED and ADHD according to standardized criteria; major limitation: lack of control group</td>
</tr>
<tr>
<td>Neumark-Sztainer (1995)</td>
<td>Cross-sectional survey</td>
<td>689 subjects with ADD (286 boys, 403 girls); 1371 controls (746 boys, 625 girls)</td>
<td>12–20 for both groups</td>
<td>Significantly higher prevalence of binge eating in subjects with attention deficit disorder vs. controls ($P=0.001$, in both boys and girls)</td>
<td>Strength: large sample size; major limitation: self-reported diagnosis of ADD and eating practices</td>
</tr>
<tr>
<td>Sokol (1999)</td>
<td>Case-control study</td>
<td>6 female bulimic patients; 7 normal control women</td>
<td>Patients, 30.8 (SD not specified); controls, 28.9 (SD not specified)</td>
<td>Scores on the Conners scale were significantly higher in bulimic patients than controls ($P=0.002$)</td>
<td>Limitations: 1) no structured interviews were used to diagnose ADHD; 2) limited sample size</td>
</tr>
</tbody>
</table>

*Data from these studies were analyzed by Surman et al.10
POTENTIAL MECHANISMS UNDERLYING THE COMORBIDITY BETWEEN ADHD AND BINGE EATING

Since the above-reviewed studies are cross-sectional, they do not allow an understanding of the causality between ADHD and binge eating. From a theoretical point of view, we think the results of the reviewed studies allow for consideration of the following hypotheses: 1) ADHD fosters binge eating behaviors, 2) ADHD and binge eating are the expression of a common neurobiological dysfunction that manifests itself as binge eating and ADHD in a subset of patients who present with both conditions, 3) binge eating contributes to ADHD symptoms, and 4) psychopathological factors common to both binge eating and ADHD mediate the association between these two conditions.

Hypothesis 1

Both the impulsive and the inattentive component of ADHD might foster disordered eating patterns, including binge eating behaviors. As for impulsivity, Davis et al.\(^8\) suggested that deficient inhibitory control as well as delay aversion, which are both expressions of the impulsivity component of ADHD, may contribute to abnormal eating behaviors, including binge eating. Deficient inhibitory control, which manifests as poor planning and difficulties monitoring one’s behavior effectively, could lead to over-consumption when not hungry associated with the relative absence of concern for daily caloric intake. A strong delay aversion could favor the tendency to binge on “fast food” with high caloric content in preference to home-cooked meals with lower caloric content (which take longer to prepare). Alternatively, deficits in attention and other executive functions (such as planning and working memory, which, as stated in the introduction, are common in ADHD patients) might cause difficulties in adhering to a regular eating pattern, thus favoring abnormal eating behaviors. Fleming and Levy\(^\text{23}\) 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 for the association between inattention and abnormal eating behaviors was provided by Schweickert et al.\(^\text{21}\) According to these authors, compulsive eating may be a compensatory mechanism to help the person control the frustration associated with attention and organizational difficulties.

Hypothesis 2

Another possibility is that ADHD and binge eating are the expression of common neurobiological mechanisms in a subset of patients who present with both of these disorders. The mechanisms involved in the so-called “reward deficiency syndrome” may play a significant role in helping explain the comorbidity between ADHD and binge eating. The reward deficiency 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, remarkably, inappropriate eating.\(^\text{24}\) Several lines of evidence suggest that a subset of patients with ADHD may present with behaviors consistent with the reward deficiency syndrome.\(^\text{25-27}\) This syndrome has also been reported in obese patients with abnormal eating behaviors.\(^\text{24}\) (Abnormal eating behaviors, including binge eating, are often found in obese populations, especially in the severely obese.\(^\text{24}\)) Alterations in the dopamine receptor D2 (DRD2)\(^\text{28}\) and, to a lesser extent, DRD4\(^\text{29,30}\) have been associated with the above-mentioned reward deficiency syndrome. Dysfunction of DRD2 and DRD4 have been found in obese patients with abnormal eating behaviors.\(^\text{31,32}\) Several studies suggest altered DRD4 and DRD2 play a role in ADHD as well\(^\text{33,34}\) (although alterations in DRD2 have not been replicated in other studies\(^\text{33}\)). Therefore, obese patients with abnormal eating behaviors and ADHD may present with common genetically determined dysfunctions in the dopaminergic system.

Interestingly, in a study Levitan et al.\(^\text{35}\) conducted on a sample of women with seasonal affective disorder, the 7R allele of DRD4 was associated with significantly higher scores of childhood inattention and with significantly higher maximal lifetime body mass index scores. Seasonal affective disorder is characterized by marked cravings for high-carbohydrate/high-fat foods, resulting in significant weight gain during winter depressive episodes. A potential implication of the reward system in the pathophysiology of the disorder has been suggested. Therefore, Levitan et al.\(^\text{35}\) hypothesized that childhood attention deficit and adult obesity may be the expression of a common biological dysfunction of the 7R allele of DRD4 associated with a dopamine dysfunction in prefrontal attentional areas and brain circuits involved in the reward pathways.

Another potential common biological mechanism involves alterations in the brain derived neurotropic factor. Lyons et al.\(^\text{36}\) found that in heterozygous brain derived neurotropic factor\(^\text{+/−}\) mice a partial impairment of brain derived neurotropic factor expression caused impaired impulse control (which is a clinical feature found in children with ADHD) associated with aggressiveness and excessive
appetite/food intake. In the study by Kernie et al.,\textsuperscript{37} 50% of heterozygous brain derived neurotropic factor\textsuperscript{+/-} mice became obese and consumed 47% more food than the heterozygous brain derived neurotropic factor\textsuperscript{+/-} non-obese and wild-type mice. Non-obese mice exhibited more than twice the locomotor activity of wild-type and obese mice. Clearly, results from these animal models should be interpreted with caution and may not be generalizable to humans with ADHD. However, Gray et al.\textsuperscript{38} recently found a functional loss of one copy of the brain derived neurotropic factor gene in an 8 year old with hyperphagia, severe obesity, impaired cognitive function, and hyperactivity. Given the limited findings, the role of common genetic mutations underlying shared neurobiological dysfunctions in ADHD and binge eating deserves further investigation.

**Hypothesis 3**

It is also possible that binge eating contributes to ADHD symptoms in patients with both ADHD and binge eating behaviors. It has been reported\textsuperscript{39} that 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 restlessness. Moreover, Rosval et al.\textsuperscript{40} recently reported higher rates of motoric impulsiveness in patients with bulimia nervosa and anorexia nervosa binge/purge subtype in comparison to those with anorexia nervosa restricting subtype and a normal eater control group, confirming the previous findings linking binge eating behaviors with behavioral impulsivity.\textsuperscript{41,42} Therefore, it is possible that this kind of behavioral impulsivity contributes to or manifests as impulsivity of ADHD.

**Hypothesis 4**

Finally, one can not exclude that the association between ADHD and binge eating may be mediated by common psychopathological factors. One possibility involves the role of major depression. It has been reported that major depression is associated with ADHD\textsuperscript{7} as well as with binge eating.\textsuperscript{43} Therefore, the comorbidity of ADHD and binge eating might be mediated by major depressive disorders.

One can not exclude that all of the four above-mentioned hypotheses may hold true and may coexist, at least in certain subjects.

**MANAGEMENT OF PATIENTS WITH ADHD AND BINGE EATING**

From a clinical standpoint, the results of our review suggest that it may be useful to 1) screen for binge eating in patients with ADHD and 2) look for ADHD symptoms in patients with binge eating.

**Screening ADHD Patients for Binge Eating**

ADHD is associated with an enormous personal, familial, and social burden. Well-known comorbid psychiatric disorders, such as anxiety or conduct disorders, which may contribute to the impairment associated with ADHD, are frequently investigated in the assessment of ADHD. However, binge eating behaviors in ADHD patients are scarcely explored in clinical practice. Since binge eating behaviors may aggravate ADHD symptoms and contribute to the adverse outcome of ADHD, as discussed in the previous section, their management may lead to a better quality of life for ADHD patients. However, to our knowledge, no studies assessed the impact of binge eating treatment on the quality of life of ADHD subjects. We think research in this field should be encouraged.

**Screening Binge Eating Patients for ADHD**

Screening for ADHD symptoms in patients with binge eating may be of relevance for the management of their abnormal eating patterns. As reported by Fleming and Levy,\textsuperscript{23} patients with eating disorders feel a great deal of shame over their eating patterns, seeing themselves as ineffectual, stupid, out-of-control, and helpless. Indeed, as reviewed in the previous section, some characteristics erroneously attributed to the supposed “weak personality” of these patients may be due to the core features of ADHD, such as delay aversion, inattention, and/or deficit in response inhibition. Therefore, screening for ADHD in patients with binge eating may be of relevance for two reasons. First, understanding how ADHD impacts eating behaviors can help reduce self-blame and facilitate the process of regaining control in the patient with abnormal eating behaviors, including binge eating. Second, if the hypothesis that ADHD actually contributes to binge eating is true, then the treatment of ADHD might improve eating patterns in patients with binge eating.

As for pharmacological treatment, some reports\textsuperscript{17,19-21} have suggested that stimulants improve ADHD and abnormal eating behaviors in patients with both conditions. Surman et al.\textsuperscript{10} explained the findings of these reports suggesting that the treatment of ADHD-related impulsivity could improve abnormal eating behaviors. Improvement in attention, leading to more regular eating patterns, may also play a significant role. Indeed, these reports also support the hypothesis that ADHD and abnormal eating behaviors share common underlying biological mechanisms, which may be the
target of ADHD medications. Alternatively, ADHD medications might act both on the brain pathways involved in ADHD and on those that mediate abnormal eating behaviors. Interestingly, Meredith et al. found that repeated injections of amphetamine were accompanied by an elevated brain derived neurotropic factor mRNA and brain derived neurotropic factor immunoreactivity in the basolateral amygdala, rostral piriform cortex, and paraventricular nucleus of the hypothalamus. Since there is some evidence that reduction of brain derived neurotropic factor in the hypothalamus causes increased locomotor activity and eating behaviors, the finding of Meredith et al. supports the hypothesis that ADHD medications may also act on brain pathways involved in abnormal eating behaviors. Moreover, Gadde et al. recently published a trial on the efficacy of atomoxetine, the second-line medication for ADHD, in weight reduction in obese women. The positive results of the trial suggested that this ADHD drug may act on the noradrenergic synapses in the medial and paraventricular hypothalamus that are thought to play a major role in modulating satiety and feeding behavior.

As correctly pointed out by Fleming and Levy, stimulant medications alone may not be sufficient to improve the disordered eating habits of patients with ADHD. Non-pharmacological strategies, such as cognitive behavior therapy, should also be considered. An improvement in attentional and organizational strategies, as well as in response inhibition, may substantially improve the eating patterns of the patients. For example, Fleming and Levy reported that many patients with ADHD and eating behaviors find it useful to have visual cues to improve their judgement about appropriate portion size such as using smaller plates, or using their palm or fingers as measurements guides. However, to the best of our knowledge, there are no controlled studies on the impact of ADHD-focused cognitive behavior therapy on binge eating improvement; therefore, this issue deserves further investigation.

CONCLUSION

Clinicians have generally overlooked the comorbidity of ADHD and binge eating. However, emerging data from empirically based studies suggest that the rate of binge eating behaviors in subjects with ADHD is higher than expected. Given the theoretical and clinical relevance of this newly described comorbidity, we think research in this field should be encouraged. More methodologically sound studies, using standardized diagnoses of ADHD, appropriate control groups, and controlling for potential mediators (such as depressive disorders) are greatly needed.

Large prospective studies, which are still lacking at present, could lead to a better understanding of the causality in the relationship between ADHD and binge eating, shedding light on the psychopathological pathways linking the two conditions. Family studies examining the occurrence of ADHD and binge eating and further studies in animal models are necessary to gain insight into the potential common genetic underpinnings. Finally, pharmacological and non-pharmacological treatment studies are greatly needed to find more appropriate and effective therapeutic strategies for patients with both ADHD and binge eating. Therefore, clinical empirically based studies, epidemiological surveys (both cross-sectional and prospective), genetic studies, animal model studies, non-pharmacological treatment studies, and pharmacological trials may advance the knowledge in the field, allowing for better management and, ultimately, a better quality of life for patients with both ADHD and binge eating.

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