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REVIEW

Attention deficit hyperactivity disorder and disordered eating behaviors: links, risks, and challenges faced

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Abstract: Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder that often persists in adulthood. It is defined by inattention and/or hyperactivity–impulsivity. ADHD is associated with many comorbidities, including eating disorders (EDs). In the last decade, studies have reported that ADHD is linked with binge EDs, bulimia nervosa, and anorexia nervosa. Many postulates have been proposed to explain the association: 1) impulsive behavior in ADHD patients leads to disordered eating behavior; 2) other psychologic comorbidities present in ADHD patients account for eating behavior; 3) poor eating habits and resulting nutritional deficiencies contribute to ADHD symptoms; and 4) other risk factors common to both ADHD and EDs contribute to the coincidence of both diseases. Additionally, sex differences become a significant issue in the discussion of EDs and ADHD because of the higher incidence of bulimia nervosa and anorexia nervosa in females and the ability of females to mask the symptoms of ADHD. Interestingly, both EDs and ADHD rely on a common neural substrate, namely, dopaminergic signaling. Dopaminergic signaling is critical for motor activity and emotion, the latter enabling the former into a combined motivated movement like eating. This linkage aids in explaining the many comorbidities associated with ADHD. The interconnection of ADHD and EDs is discussed from both a historical perspective and the one based on the revealing nature of its comorbidities.

Keywords: ADHD, eating disorders, obesity, disordered eating, dopamine, motivation

Introduction

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by an age inappropriate level of attention, impulsivity, and hyperactivity.[1] The prevalence of ADHD is ~5.3% in the pediatric population and 3.4% in the adult population.[2,3] Treatment for ADHD includes cognitive behavioral therapy and pharmacologic treatment. The drug of choice is methylphenidate, a psychostimulant. Other drugs include amphetamine and nonpsychostimulant drugs, such as atomoxetine and guanfacine.[4]

Interestingly, ADHD is rarely present as an isolated disorder. Previous studies have reported that ~70% of ADHD patients display at least one other comorbid disorder or specific neurological problems.[5–7] In the last decade, researchers report a significant coincidence of ADHD and eating disorders (EDs).[5,8,9] The EDs mostly associated with ADHD are binge eating disorder (BED) and bulimia nervosa (BN). BED is characterized by recurrent binge eating episodes and associated feelings of guilt and lack of control. BN is characterized by recurrent binge eating episodes followed by self-induced vomiting or other compensatory behavior. Anorexia nervosa (AN) is characterized by

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distorted body image and excessive dieting.[1] Although the link between AN and ADHD is more controversial, some studies suggest an association between the two.[10]

Other psychiatric comorbidities of ADHD include conduct disorders, oppositional defiant disorders (ODDs), mood disorders, anxiety disorders, learning disabilities, mental retardation, Tourette's syndrome, borderline personality disorders, etc.[11] In this extensive literature review, Biederman et al proposed that ADHD patients with different comorbidities represent unique subgroups with different clinical courses and require different treatment strategies. Since 1991, many researchers have echoed this belief.[11]

In addition to psychiatric comorbidities, ADHD is associated with devastating health outcomes: substance abuse, suicide, physical injuries, risky sexual behavior, obesity, diabetes type 2, hypertension, higher health care visits, and early mortality.[12,13] Although the reason behind these associations is not clear, impairment of executive function in patients with ADHD is hypothesized to be the most simple explanation.[14] Thus, they are unable to plan and execute healthy behaviors, including caring for their own hygiene and health. The adverse health outcome mostly related to disordered eating is obesity. Besides this, it is unclear whether ADHD may be connected with specific changes in somatic growth.[15–17]

The topic of sex differences in ADHD also becomes significant. In pediatric populations, ADHD is estimated to be three times more common in boys than in girls. In adults, the incidence of ADHD is similar in both sexes.[18] Males with ADHD are more likely to externalize their symptoms, and females are more likely to internalize their symptoms.[19,20] Compounding these observations some believe that, in reality, the prevalence of ADHD in girls is higher than reported, because of their ability to better "mask" their symptoms during childhood.[21] BN and AN are disorders that mainly affect females. The phenomena that ADHD is underdiagnosed in girls and EDs are more prevalent have clinical implications.

Eating patterns and nutrition for subjects with ADHD

Recent studies have found a significant link between the ADHD and an abnormal dietary pattern, ie, the consumption of "junk food" and nonadherence to a "traditional" three-meal daily diet. The first of such studies was the Raine study, which analyzed the eating patterns of 1,799 adolescents.[22] Of the participants, 115 adolescents were found to have ADHD. Subjects were classified as "western" or "traditional" at the 14-year follow-up after birth. The western diet pattern was linked with higher intake of fat, sugar, and sodium and lower intake of omega-3 fatty acids, fiber, and folate. The study found that the subjects with ADHD symptoms were more likely to have a western style diet than a traditional healthy diet (odds ratio [OR] =2.21), potentially suggesting dietary problems, which may be associated with processed foods.

Similar to the Raine study, a Korean group studied the dietary patterns of children with ADHD.[23] The four dietary patterns discovered were "seaweed-egg", "traditional-healthy", "traditional", and "snack". The seaweed-egg pattern was characterized with high intakes of fats and sweets. The study found a significant association between the ADHD and the seaweed-egg dietary pattern. Another Korean study of 12,350 participants reports a significant association between ADHD and unhealthy foods, such as soft drinks, westernized fast food, and instant noodles.[24] Our group studied the eating behaviors of premedicated newly diagnosed ADHD boys using structured interviews of parents. We found that patients with ADHD are more likely to skip breakfast and dinner and have more than five meals throughout the day. We also found that these disruptive dietary patterns were accompanied by diminished consumption of fruits and vegetables and increased consumption of sweetened beverages.[25] Even prior to the Raine trial, many nutritional deficiencies had been associated with ADHD.[26] Iron deficiency and low serum ferritin levels have been linked to ADHD and impaired cognitive behavioral development.[27–29] Zinc deficiency, especially in the Middle East, has also been associated with ADHD. Zinc is an important cofactor implicated in the metabolism of dopamine, a neurotransmitter involved in ADHD pathophysiology. Arnold et al reported that zinc supplementation decreased the optimal dose of amphetamine treatment.[30,31] Consumption of food additives and artificial sweeteners has been reported to contribute to abnormal levels of hyperactivity in developing children.[32] Considerable research has been devoted to the omega-3 and -6 polyunsaturated fatty acid (PUFA) supplementation. Similar to zinc, polyunsaturated fatty acids are also involved in neuronal development and have protective effects against ADHD symptoms.[33–35] As a result of the previous findings, there has been an interest for dietary interventions with hopes of improvement in symptoms or prevention of ADHD in children.[36]

Coupling of obesity and ADHD

The link between ADHD and disordered eating behavior is evident by the observation that obesity is more prevalent in individuals with ADHD compared to the general population. Altfas was the first to describe the comorbidity of the two

disorders.[37] Altfas found an unusual prevalence of ADHD (27.4%) among obese adults. After treatment of symptoms, weight loss was greater in the treated ADHD obese adults compared to non-ADHD obese adults.[37] Shortly after, two other groups reported similar finding in hospitalized obese children and obese women. Further studies reported that obese patients with ADHD had predominantly inattentive symptoms.[38,39]

The comorbidity was later supported by massive community surveys, smaller clinical cross-sectional studies, and some longitudinal studies. A large cross-sectional study of 43,297 US adolescents revealed a statistically significant adjusted OR of 1.5.[13] Similar results were found in adolescent population of 9,619 adolescents aged.[40] A study with 1,633 adult German participants found that the prevalence of ADHD was 9.3% in individuals who are obese. This abnormally high prevalence was not observed in overweight (3.8%) and normal weight individuals (4.3%). Conversely, the study reported that participants with ADHD were twice as likely to be obese than the general population (22.1% vs 10.2%).[41] Furthermore, Cortese et al found that obese adults were more likely to have had a diagnosis of childhood ADHD in the past. Notably, their study consisted of 34,653 face-to-face interviews of young adults and also found that impulsive and inattentive symptoms, but not hyperactive symptoms, mediated the association.[42] Also notable was a 33-year longitudinal study of 207 participants, by the same author, which found that men who had childhood ADHD had higher BMI and obesity rates.[43]

Other smaller cross-sectional studies of children and adolescents seeking treatment for ADHD suggest a comorbidity between higher BMI and ADHD. A cross-sectional study by a group in Poland examined boys aged 6–18 years who were diagnosed with ADHD and demonstrated that overweight status but not obesity was statistically significant in the ADHD group.[44] Another study of 158 children with ADHD aged 6–16 years found that patients with ADHD had a higher prevalence of obesity. The study also found that the patients with combined subtype of ADHD were significantly more likely to be obese and overweight compared to those with only inattentive or only hyperactive symptoms.[45]

A possible explanation for the comorbidity between the two disorders is a common genetic and neurobiological pathway. Obesity genes in the pathways of dopaminergic circuitry, such as FTO (fat mass–and obesity-associated variant) and melanocortin 4 receptor, have been associated with ADHD.[46–48] A case study reported that a 13-year-old obese boy with ADHD (BMI =47.2) and a melanocortin 4 receptor mutation showed a dramatic decrease in BMI after atomoxetine treatment.[49] Co-occurrence of ADHD and obesity has also been attributed to common immune and inflammatory processes, common fetal programming mechanisms, and common perinatal risk factors.[12,50]

Coupling of BEDs and ADHD

Various investigators propose that the link between obesity and ADHD lies in the common symptom of impulsivity. Here, it is surmised that ADHD predisposes an individual to BED, again due to common impulsivity symptoms. Impulsivity is defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions.[51] Inhibition, working memory, planning, and sustained attention are necessary for executive function or the ability to perform a sequence of goal-oriented tasks.[52,53] Cortese et al argue that the impulsivity of ADHD patients causes executive dysfunction, which prevents the patient from executing the goal of controlling eating behavior or losing weight.[14]

Clinically, impulsivity in ADHD patients can be found with the Barratt Impulsiveness Scale (BIS). It is a 30-item self-reporting questionnaire.[54] Impulsiveness can also be demonstrated using the go–no-go paradigm. ADHD patients with predominantly impulsive symptoms have more commitment errors in the go–no-go task. For example, when receiving a cue for a string of consecutive "go" responses, the subject is more likely to commit to go even after presented with a cue for "no-go". Commitment errors represent a lack of response inhibition, an essential ingredient of executive functioning.[55] Because of impulsiveness, patients with ADHD and BED are not able to "inhibit" their impulse to eat food, even when faced the task of dieting and losing weight.[56] This hypothesis is corroborated by many cross-sectional studies of BED patients in weight loss or prebariatric surgery clinics. It is found that symptoms of impulsiveness and inattention correlate with severity of BED symptoms.[57,58] For example, Docet et al found that obese patients with ADHD were more likely to have binge eating episodes, to eat snacks between meals, to eat large amounts of food, and to eat in secret.[59] In another study, methylphenidate treatment in obese individuals with ADHD resulted in significant weight loss in obese patients with ADHD.[60]

The link between impulsivity and BED was also demonstrated using a combination of magnetoencephalography, the go–no-go task, and the BIS.[61,62] The study used food stimuli or toys as the go–no-go cues and a population of subjects with and without BED. It was found that, in BED, impulsiveness according to BIS scores correlated with decreased

food-specific response inhibition in the prefrontal control network during the go–no-go task.[63] This is consistent with other studies on response inhibition to food cues in BED using functional magnetic resonance imaging.[64,65]

Often young children may not have BED at the time of diagnosis of ADHD; however, some have a similar condition called "loss of control eating" (LOC).[66] It is defined by subjective feelings of binge eating or subjective loss of control while consuming a normal or small amount of food. Thus, a subject who may not have an official diagnosis of BED may still be considered to have LOC. Reinblatt et al reported that LOC was much higher in children with ADHD (adjusted OR =12.68).[67] LOC eating in childhood may be a predictor of disordered eating behavior in adolescence.[68] Although promising and educational, it is still not known whether a combination of LOC and ADHD in childhood longitudinally translate into objective BED. Food addiction, or eating behavior that involves overconsumption of specific foods that are highly palatable in an addiction-like manner, has also been associated with inattentive impulsive symptoms. It is thought that ADHD consists of dopamine and norepinephrine circuits in the brain, which are involved in reward processing. This accounts for the high prevalence of substance abuse disorders in ADHD subjects. Thus, food addiction, like substance abuse, is also comorbid with ADHD.[69,70]

Although executive dysfunction has been shown to play a role in BED, it does not encompass the entire picture. Steadman and Knouse found that although impulsivity in ADHD correlates with BED symptoms, impulsivity alone does not mediate the association.[71] Other comorbidities in ADHD patients are thought to contribute to binge eating behavior. Affective diseases, mainly depression, have been associated with higher incidence of BED. Many studies found that the presence of depressive symptoms in obese adults with ADHD correlated with severity of BED symptoms.[72–75] The other comorbidity that may mediate the association between BED and ADHD is ODD. Pauli-Pott et al reported that ODD symptoms, not ADHD symptoms alone, were associated with disordered eating behaviors.[76,77] This suggests that patients with ADHD and another psychiatric comorbidity are necessary for the development of BED. Expectedly, in the same study, participants with symptoms of anxiety and depression showed emotional and binge eating.

ADHD symptomatology in women with BN

BN, as noted for the other disorders, is associated with eating and has also been linked to ADHD. In the case of BN, sex differences become significant and apparent from the literature. The prevalence of ADHD is three times higher in boys than girls.[42] This may be due to the phenomena that girls are more likely to internalize and "mask" their symptoms, while boys externalize them. There is also a higher level of clinical suspicion of the disorder in boys, which may contribute to underdiagnosis in girls.[18,21] On the other hand, BN is nearly 12 times more common in girls than boys.[78]

Owing to underdiagnosis of ADHD in girls, by the time a female realizes she has a psychiatric disorder, she may already be in late adolescence or adulthood. This phenomenon is evident in a series of case studies, which were the first presentations of the association between BN and ADHD. These studies paint a common picture of a young adult female who had a seemingly normal childhood. Her attention was sufficient to perform her school duties and manage her family and social life during childhood and early adolescence.[79] When faced with greater challenges, such as attending university, managing her time, having a relationship, and making her own life decisions, her deficits in executive functions became apparent. Symptoms of depression and/or anxiety were present, and bulimic symptoms of binging and purging were out of control. Pharmacologic therapy for ADHD symptoms in this patient improved her executive functions and, interestingly, decreased her purging behaviors.[79–84] A number of studies support the initial findings of the previously mentioned case studies. In a study of 20 women with BN and 20 age-matched controls, symptoms of impulsivity measured with the BIS were significantly higher in the BN group.[85] In a larger study of 89 women with ED, Yates et al found that inattentive symptoms of ADHD were common among women with BN. Furthermore, Yates et al also confirmed that inattentive symptoms correlated with bulimic behavior and depressive symptoms.[86] A number of other studies reported similar findings.[87,88] In addition to depression, the presence of anxiety and disruptive disorders was reported. Impulsivity alone does not account for the link between ADHD and BN. Other comorbidities, such as depression, anxiety, and disruptive behavior, were also found to mediate the association between BN and ADHD.[11] Girls with ADHD and depressive symptoms were found to be at the highest risk of BN.[11] Notably, among studies of BN, there are two longitudinal studies by Mikami et al in a 5-year prospective longitudinal study of ADHD girls aged 6–12 years.[89] Mikami et al found that baseline impulsivity symptoms predicted adolescent pathology. Interestingly, the group found that baseline peer rejection and parent–child relationship predicted eating pathology. Punitive parenting

in childhood also predicted pathological eating behaviors.[89] In an 8-year follow-up, the Eating Disorder Inventory-II was used to collect data about body image dissatisfaction and personality characteristics associated with EDs. It was found that boys and girls with ADHD were at risk with symptoms of BN in mid-adolescence. The association was stronger in girls than in boys.[90] Similar studies have the same results.[84,85] In summary, girls with ADHD and symptoms of depressive, anxiety, and disruptive disorders are at risk of developing BN. In adolescents with BN, there is a high prevalence of suicide attempts, alcohol consumption, and illegal drug use.[91]

Evidences from modern neurobiology methods corroborate the abovementioned findings. For example, the Catechol-O-methyltransferase gene, involved in the dopaminergic pathways of ADHD, was also implicated in BN.[92] From a neuroimaging perspective, ADHD and BN share many neural pathways. Abnormalities in these pathways in the frontostriatal circuit may account for the coincidences in these two disorders.[93] Much is not known about the effect of puberty on ADHD and BN. It is thought that sex differences may be accounted for by different hormones in boys and girls during puberty.[94–96] Biederman et al found that girls with ADHD tend to have an earlier onset of menarche; however, the reason behind this finding is currently not understood.[95] The link between AN and ADHD is not clear despite the hypothesis of a common neural substrate.[97–100] The number of studies is small, and the studies that do exist have a small number of subjects.[10,101,102] Thirty-two female patients diagnosed with ED had no correlation between severity of ADHD and severity of ED symptoms; however, there was an association between impulsivity and avoidance of fattening food.[101] A larger study of 191 patients reports that girls with AN had a higher correlation with ADHD symptomatology than girls with BN.[10] The symptoms with the highest correlation included novelty seeking, impaired self-directedness, and impaired cooperativeness. Similarly, Wentz et al found that in a small study of individuals with EDs, there was a high prevalence of ADHD.[102]

Hazards and challenges

The literature suggests that it may be beneficial to interview and counsel patients and parents regarding eating behaviors. We surmise, from the data analysis, that special attention should be given to females since they are more likely to internalize their symptoms and display depressive behaviors, thus masking symptoms of ADHD. This may account for the observation that the ratio of ADHD children who seek treatment is ~3:1 (girls to boys).[2] This was especially evident in the series of case studies on BN. These case studies paint the picture of a young adult female who was able to mask her ADHD symptoms throughout childhood. During adulthood, when faced with tasks that required higher executive function (for example, time management in university), the symptoms of ADHD caused havoc in her life. This is compounded by the problem that it is a challenge to diagnose ADHD early, regardless of the population. The solution for this problem may be in educating the general population, which will allow people to seek early advice if symptoms of ADHD are present. However, this may lead to an overdiagnosis of ADHD but is better than underdiagnosing this debilitating condition. Moreover, the solution for early detection is in the hands of neonatologists or pediatricians. In this regard, many risks factors for ADHD are perinatal, for example, prematurity.[103] Furthermore, research should be focused on identifying babies who are more likely to have ADHD.

In addition to early diagnosis, it is also important to monitor affective symptoms of ADHD. Most of the literature on BN and AN suggest that depression may mediate bulimic and anorexic symptoms. Monitoring depressive symptoms in addition to eating behaviors in girls, especially, appears to be critical. In addition to depressive symptoms, these patients have a distorted body image.[89,90] Importantly, regarding dietary contributing factors, care must be taken when counseling girls about diet.[8] Impulsive behavior is related to avoidance of food in anorexic girls.[101] Anorexic and bulimic girls are already obsessed and guilty due to their eating behaviors. Counseling eating behavior should be done in a positive manner, which rehabilitates self-confidence, not in a manner that makes the girls feel even more "guilty".

A challenge in the study of EDs and ADHD is the cyclical nature of the symptoms of both diseases. Investigators have found that predominant symptoms of ADHD may change, persist, or remit throughout the lifetime.[104,105] The general trend is that inattentive symptoms are more persisting.[103] Patients with EDs have been found to cycle through symptoms of BN, AN, and EDs not otherwise specified.[106,107] No study to our knowledge has longitudinally examined the complex interaction that may arise due to cyclical shifting between different subtypes of EDs and ADHD. One can only imagine that it is very complex, and more research and understanding about the relationship between the two is needed.

Conclusion

ADHD and eating behavior are strongly tied together and correlated throughout age groups.[5,8,9] Furthermore, unhealthy eating habits as well as food-associated additives, in general,

are directly correlated with ADHD and healthier diets have been associated with improved symptomatology.[22] ADHD is also correlated with pathological eating behaviors that are characterized as mental illnesses, such as, BN, AN as well as abnormal BMI ranges known as obesity.[1,13,18] It is important to note that catecholamines, eg, dopamine, are heavily involved in both motor regulation and emotions, working together to provide the motivation for motor activity, using few chemical messengers in both invertebrates and vertebrates, especially humans.[108] Thus, it is not a surprise that ADHD comorbidities involve EDs, which may result from altered cognitive and emotional neural substrates.[5,11] Furthermore, it is not a surprise that ADHD comorbidities involve EDs, which may result from altered cognitive and emotional neural substrates given their dependencies.[109] This is also somewhat evident from the COMT data, which involve ADHD behavioral states and EDs.[110,111] Taken together, although ADHD is complex and multifaceted as are EDs, it appears that they may represent a logical comorbidity. Hence, early diagnostic indicators for ADHD may be at hand in the form of novel discoveries in gene expression patterns.

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References

1. American Association of Pediatrics. *Diagnostic and Statistic Manual of Mental Disorders*. 5th ed. Washington DC: American Psychiatric Publishing; 2013.
2. Polanczyk G, de Lima MS, Horta BL, Biederman J, Rohde LA. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *Am J Psychiatry*. 2007;164(6):942–948.
3. Fayyad J, De Graaf R, Kessler R. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. *Br J Psychiatry*. 2007;190:402–409.
4. Pliszka S. Practice parameter for the assessment and treatment of children and adolescents with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry*. 2007;46(7):894–921.
5. Nazar BP, Pinna CM, Coutinho G, et al. Review of literature of attention-deficit/hyperactivity disorder with comorbid eating disorders. *Rev Bras Psiquiatr*. 2008;30(4):384–389.
6. Goetz M, Schwabova J, Hlavka Z, et al. Cerebellar symptoms are associated with omission errors and variability of response time in children with ADHD. *J Atten Disord*. 2014.
7. Nováková M, Paclt I, Ptáček R, Kuželová H, Hájek I, Sumová A. Salivary melatonin rhythm as a marker of the circadian system in healthy children and those with attention-deficit/hyperactivity disorder. *Chronobiol Int*. 2011;28(7):630–637.
8. Quinn PO. Attention-deficit/hyperactivity disorder and its comorbidities in women and girls: an evolving picture. *Curr Psychiatry Rep*. 2008;10(5):419–423.
9. Cortese S, Angriman M, Maffeis C, et al. Attention-deficit/hyperactivity disorder (ADHD) and obesity: a systematic review of the literature. *Crit Rev Food Sci Nutr*. 2008;48(6):524–537.
10. Fernández-Aranda F, Agüera Z, Castro R, et al. ADHD symptomatology in eating disorders: a secondary psychopathological measure of severity? *BMC Psychiatry*. 2013;13(1):166–173.
11. Biederman J, Newcorn J, Sprich S. Comorbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety, and other disorders. *Am J Psychiatry*. 1991;148(5):564–577.
12. Nigg JT. Attention-deficit/hyperactivity disorder and adverse health outcomes. *Clin Psychol Rev*. 2013;33(2):215–228.
13. Halfon N, Larson K, Slusser W. Associations between obesity and comorbid mental health, developmental, and physical health conditions in a nationally representative sample of US children aged 10 to 17. *Acad Pediatr*. 2013;13(1):6–13.
14. Cortese S, Comencini E, Vincenzi B, Speranza M, Angriman M. Attention-deficit/hyperactivity disorder and impairment in executive functions: a barrier to weight loss in individuals with obesity? *BMC Psychiatry*. 2013;13:286–292.
15. Ptacek R, Kuzelova H, Stefano G, Raboch J, Kream R, Goetz M. ADHD and growth: questions still unanswered. *Neuro Endocrinol Lett*. 2014;35(1):1–6.
16. Ptacek R, Kuzelova H, Paclt I, Zukov I, Fischer S. ADHD and growth: anthropometric changes in medicated and non-medicated ADHD boys. *Med Sci Monit*. 2009;15(12):CR595–CR599.
17. Ptacek R, Kuzelova H, Paclt I, Zukov I, Fischer S. Anthropometric changes in non-medicated ADHD boys. *Neuro Endocrinol Lett*. 2009;30(3):377–381.
18. Quinn PO, Madhoo M. A review of attention-deficit/hyperactivity disorder in women and girls: uncovering this hidden diagnosis. *Prim Care Companion CNS Disord*. 2014;16(3):CC.13r01596.
19. Gershon J. A meta-analytic review of gender differences in ADHD. *J Atten Disord*. 2002;5:143–154.
20. Biederman J, Faraone SV, Spencer T, Wilens T, Mick E, Lapey KA. Gender differences in a sample of adults with attention deficit hyperactivity disorder. *Psychiatry Res*. 1994;53(1):13–29.
21. Quinn P, Wigal S. Perceptions of girls and ADHD: results from a national survey. *MedGenMed*. 2004;6(2):2.
22. Howard AL, Robinson M, Smith GJ, Ambrosini GL, Piek JP, Oddy WH. ADHD is associated with a "western" dietary pattern in adolescents. *J Atten Disord*. 2011;15(5):403–411.
23. Woo HD, Kim DW, Hong YS, et al. Dietary patterns in children with attention deficit/hyperactivity disorder (ADHD). *Nutrients*. 2014;6(4):1539–1553.
24. Kim EJ, Kwon HJ, Ha M, et al. Relationship among attention-deficit hyperactivity disorder, dietary behaviours and obesity. *Child Care Health Dev*. 2014;40(5):698–705.
25. Ptacek R, Kuzelova H, Stefano GB, et al. Disruptive patterns of eating behaviors and associated lifestyles in males with ADHD. *Med Sci Monit*. 2014;20:608–613.
26. Konikowska K, Regulska-Ilow B, Rozanska D. The influence of components of diet on the symptoms of ADHD in children. *Rocz Panstw Zakl Hig*. 2012;63(2):127–134.
27. Halterman JS, Kaczorowski JM, Aligne CA, Auinger P, Szilagyi PG. Iron deficiency and cognitive achievement among school-aged children and adolescents in the United States. *Pediatrics*. 2001;107(6):1381–1386.
28. Konofal E, Lecendreux M, Arnulf I, Mouren MC. Iron deficiency in children with attention-deficit/hyperactivity disorder. *Arch Pediatr Adolesc Med*. 2004;158(12):1113–1115.
29. Millichap JG, Yee MM, Davidson SI. Serum ferritin in children with attention-deficit hyperactivity disorder. *Pediatr Neurol*. 2006;34(3):200–203.
30. Arnold LE, Disilvestro RA, Bozzolo D, et al. Zinc for attention-deficit/hyperactivity disorder: placebo-controlled double-blind pilot trial alone and combined with amphetamine. *J Child Adolesc Psychopharmacol*. 2011;21(1):1–19.

31. Toren P, Eldar S, Sela BA, et al. Zinc deficiency in attention-deficit hyperactivity disorder. *Biol Psychiatry*. 1996;40(12):1308–1310.
32. Bateman B, Warner JO, Hutchinson E, et al. The effects of a double blind, placebo controlled, artificial food colourings and benzoate preservative challenge on hyperactivity in a general population sample of preschool children. *Arch Dis Child*. 2004;89(6):506–511.
33. Mitchell EA, Aman MG, Turbott SH, Manku M. Clinical characteristics and serum essential fatty acid levels in hyperactive children. *Clin Pediatr*. 1987;26(8):406–411.
34. Colter AL, Cutler C, Meckling KA. Fatty acid status and behavioural symptoms of attention deficit hyperactivity disorder in adolescents: a case-control study. *Nutr J*. 2008;7:8–19.
35. Richardson AJ, Montgomery P. The Oxford-Durham study: a randomized, controlled trial of dietary supplementation with fatty acids in children with developmental coordination disorder. *Pediatrics*. 2005; 115(5):1360–1366.
36. Millichap JG, Yee MM. The diet factor in attention-deficit/hyperactivity disorder. *Pediatrics*. 2012;129(2):330–337.
37. Altfas JR. Prevalence of attention deficit/hyperactivity disorder among adults in obesity treatment. *BMC Psychiatry*. 2002;2:9–17.
38. Agranat-Meged AN, Deitcher C, Goldzweig G, Leibenson L, Stein M, Galili-Weisstub E. Childhood obesity and attention deficit/hyperactivity disorder: a newly described comorbidity in obese hospitalized children. *Int J Eat Disord*. 2005;37(4):357–359.
39. Fleming JP, Levy LD, Levitan RD. Symptoms of attention deficit hyperactivity disorder in severely obese women. *Eat Weight Disord*. 2005;10(1):e10–e13.
40. Phillips KL, Schieve LA, Visser S, et al. Prevalence and impact of unhealthy weight in a national sample of US adolescents with autism and other learning and behavioral disabilities. *Matern Child Health J*. 2014;18(8):1964–1975.
41. de Zwaan M, Gruss B, Müller A, et al. Association between obesity and adult attention-deficit/hyperactivity disorder in a German community-based sample. *Obes Facts*. 2011;4(3):204–211.
42. Cortese S, Faraone SV, Bernardi S, Wang S, Blanco C. Adult attention-deficit hyperactivity disorder and obesity: epidemiological study. *Br J Psychiatry*. 2013;203(1):24–34.
43. Cortese S, Ramos Olazagasti MA, Klein RG, Castellanos FX, Proal E, Mannuzza S. Obesity in men with childhood ADHD: a 33-year controlled, prospective, follow-up study. *Pediatrics*. 2013;131(6): e1731–e1738.
44. Hanć T, Słopień A, Wolańczyk T, et al. ADHD and overweight in boys: cross-sectional study with birth weight as a controlled factor. *Eur Child Adolesc Psychiatry*. 2015;24(1):41–53.
45. Yang R, Mao S, Zhang S, Li R, Zhao Z. Prevalence of obesity and overweight among Chinese children with attention deficit hyperactivity disorder: a survey in Zhejiang Province, China. *BMC Psychiatry*. 2013; 13(1):133–140.
46. Albayrak Ö, Pütter C, Volckmar AL, et al; Psychiatric GWAS Consortium: ADHD Subgroup. Common obesity risk alleles in childhood attention-deficit/hyperactivity disorder. *Am J Med Genet B Neuropsychiatr Genet*. 2013;162b(4):295–305.
47. Choudhry Z, Sengupta SM, Grizenko N, et al. Association between obesity-related gene FTO and ADHD. *Obesity (Silver Spring)*. 2013; 21(12):E738–E744.
48. Agranat-Meged A, Ghanadri Y, Eisenberg I, Ben Neriah Z, Kieselstein-Gross E, Mitrani-Rosenbaum S. Attention deficit hyperactivity disorder in obese melanocortin-4-receptor (MC4R) deficient subjects: a newly described expression of MC4R deficiency. *Am J Med Genet B Neuropsychiatr Genet*. 2008;147b(8):1547–1553.
49. Pott W, Albayrak O, Hinney A, Hebebrand J, Pauli-Pott U. Successful treatment with atomoxetine of an adolescent boy with attention deficit/hyperactivity disorder, extreme obesity, and reduced melanocortin 4 receptor function. *Obes Facts*. 2013;6(1):109–115.
50. Verlaet AA, Noriega DB, Hermans N, Savelkoul HF. Nutrition, immunological mechanisms and dietary immunomodulation in ADHD. *Eur Child Adolesc Psychiatry*. 2014;23(7):519–529.
51. Moeller FG, Barratt ES, Dougherty DM, Schmitz JM, Swann AC. Psychiatric aspects of impulsivity. *Am J Psychiatry*. 2001;158(11):1783–1793.
52. Barkley RA. Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychol Bull*. 1997; 121(1):65–94.
53. Willcutt EG, Doyle AE, Nigg JT, Faraone SV, Pennington BF. Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review. *Biol Psychiatry*. 2005;57(11):1336–1346.
54. Barratt ES. Factor analysis of some psychometric measures of impulsiveness and anxiety. *Psychol Rep*. 1965;16(2):547–554.
55. Trommer BL, Hoeppner JA, Lorber R, Armstrong KJ. The go-no-go paradigm in attention deficit disorder. *Ann Neurol*. 1988;24(5):610–614.
56. Schag K, Schonleber J, Teufel M, Zipfel S, Giel KE. Food-related impulsivity in obesity and binge eating disorder – a systematic review. *Obes Rev*. 2013;14(6):477–495.
57. Gruss B, Mueller A, Horbach T, Martin A, de Zwaan M. Attention-deficit/hyperactivity disorder in a prebariatric surgery sample. *Eur Eat Disord Rev*. 2012;20(1):e103–e107.
58. Reinblatt SP, Leoutsakos JM, Mahone EM, Forrester S, Wilcox HC, Riddle MA. Association between binge eating and attention-deficit/hyperactivity disorder in two pediatric community mental health clinics. *Int J Eat Disord*. 2014;48(5):505–511.
59. Docet MF, Larranaga A, Perez Mendez LF, Garcia-Mayor RV. Attention deficit hyperactivity disorder increases the risk of having abnormal eating behaviours in obese adults. *Eat Weight Disord*. 2012; 17(2):e132–e136.
60. Levy LD, Fleming JP, Klar D. Treatment of refractory obesity in severely obese adults following management of newly diagnosed attention deficit hyperactivity disorder. *Int J Obes*. 2009;33(3):326–334.
61. Wilson TW, Franzen JD, Heinrichs-Graham E, White ML, Knott NL, Wetzel MW. Broadband neurophysiological abnormalities in the medial prefrontal region of the default-mode network in adults with ADHD. *Hum Brain Mapp*. 2013;34(3):566–574.
62. Hege MA, Stingl KT, Kullmann S, et al. Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks. *Int J Obes*. 2015;39(2):353–360.
63. Hege MA, Preissl H, Stingl KT. Magnetoencephalographic signatures of right prefrontal cortex involvement in response inhibition. *Hum Brain Mapp*. 2014;35(10):5236–5248.
64. Balodis IM, Grilo CM, Kober H, et al. A pilot study linking reduced fronto-striatal recruitment during reward processing to persistent binge-ing following treatment for binge-eating disorder. *Int J Eat Disord*. 2014; 47(4):376–384.
65. Lock J, Garrett A, Beenhakker J, Reiss AL. Aberrant brain activation during a response inhibition task in adolescent eating disorder subtypes. *Am J Psychiatry*. 2011;168(1):55–64.
66. Blomquist KK, Roberto CA, Barnes RD, White MA, Masheb RM, Grilo CM. Development and validation of the eating loss of control scale. *Psychol Assess*. 2014;26(1):77–89.
67. Reinblatt SP, Mahone EM, Tanofsky-Kraff M, et al. Pediatric loss of control eating syndrome: association with attention-deficit/hyperactivity disorder and impulsivity. *Int J Eat Disord*. 2015;48(6):580–588.
68. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *Int J Eat Disord*. 2009;42(1):26–30.
69. Gearhardt AN, Yokum S, Orr PT, Stice E, Corbin WR, Brownell KD. Neural correlates of food addiction. *Arch Gen Psychiatry*. 2011;68(8):808–816.
70. Meule A, Hermann T, Kubler A. Food addiction in overweight and obese adolescents seeking weight-loss treatment. *Eur Eat Disord Rev*. 2015;23(3):193–198.
71. Steadman KM, Knouse LE. Is the relationship between ADHD symptoms and binge eating mediated by impulsivity? *J Atten Disord*. 2014.
72. Muller A, Claes L, Wilderjans TF, de Zwaan M. Temperament subtypes in treatment seeking obese individuals: a latent profile analysis. *Eur Eat Disord Rev*. 2014;22(4):260–266.

73. Nazar BP, Suwwan R, de Sousa Pinna CM, et al. Influence of attention-deficit/hyperactivity disorder on binge eating behaviors and psychiatric comorbidity profile of obese women. *Compr Psychiatry*. 2014; 55(3):572–578.
74. Alfonsson S, Parling T, Ghaderi A. Self-reported symptoms of adult attention deficit hyperactivity disorder among obese patients seeking bariatric surgery and its relation to alcohol consumption, disordered eating and gender. *Clin Obes*. 2013;3(5):124–131.
75. Khalife N, Kantomaa M, Glover V, et al. Childhood attention-deficit/hyperactivity disorder symptoms are risk factors for obesity and physical inactivity in adolescence. *J Am Acad Child Adolesc Psychiatry*. 2014; 53(4):425–436.
76. Pauli-Pott U, Becker K, Albayrak O, Hebebrand J, Pott W. Links between psychopathological symptoms and disordered eating behaviors in overweight/obese youths. *Int J Eat Disord*. 2013;46(2):156–163.
77. Pauli-Pott U, Neidhard J, Heinzel-Gutenbrunner M, Becker K. On the link between attention deficit/hyperactivity disorder and obesity: do comorbid oppositional defiant and conduct disorder matter? *Eur Child Adolesc Psychiatry*. 2014;23(7):531–537.
78. Surman CB, Randall ET, Biederman J. Association between attention-deficit/hyperactivity disorder and bulimia nervosa: analysis of 4 case-control studies. *J Clin Psychiatry*. 2006;67(3):351–354.
79. Dukarm CP. Bulimia nervosa and attention deficit hyperactivity disorder: a possible role for stimulant medication. *J Womens Health (Larchmt)*. 2005;14(4):345–350.
80. Schweickert LA, Strober M, Moskowitz A. Efficacy of methylphenidate in bulimia nervosa comorbid with attention-deficit hyperactivity disorder: a case report. *Int J Eat Disord*. 1997;21(3):299–301.
81. Sokol MS, Gray NS, Goldstein A, Kaye WH. Methylphenidate treatment for bulimia nervosa associated with a cluster B personality disorder. *Int J Eat Disord*. 1999;25(2):233–237.
82. Guerdjikova AI, McElroy SL. Adjunctive methylphenidate in the treatment of Bulimia nervosa co-occurring with bipolar disorder and substance dependence. *Innov Clin Neurosci*. 2013;10(2):30–33.
83. Keshen A, Ivanova I. Reduction of bulimia nervosa symptoms after psychostimulant initiation in patients with comorbid ADHD: five case reports. *Eat Disord*. 2013;21(4):360–369.
84. Loannidis K, Serfontein J, Muller U. Bulimia nervosa patient diagnosed with previously unsuspected ADHD in adulthood: clinical case report, literature review, and diagnostic challenges. *Int J Eat Disord*. 2014; 47(4):431–436.
85. Wolfe BE, Jimerson DC, Levine JM. Impulsivity ratings in bulimia nervosa: relationship to binge eating behaviors. *Int J Eat Disord*. 1994; 15(3):289–292.
86. Yates WR, Lund BC, Johnson C, Mitchell J, McKee P. Attention-deficit hyperactivity symptoms and disorder in eating disorder inpatients. *Int J Eat Disord*. 2009;42(4):375–378.
87. Seitz J, Kahraman-Lanzerath B, Legenbauer T, et al. The role of impulsivity, inattention and comorbid ADHD in patients with bulimia nervosa. *PLoS One*. 2013;8(5):e63891.
88. Nazar BP, Pinna CM, Suwwan R, et al. ADHD rate in obese women with binge eating and bulimic behaviors from a weight-loss clinic. *J Atten Disord*. 2012.
89. Mikami AY, Hinshaw SP, Patterson KA, Lee JC. Eating pathology among adolescent girls with attention-deficit/hyperactivity disorder. *J Abnorm Psychol*. 2008;117(1):225–235.
90. Mikami AY, Hinshaw SP, Arnold LE, et al. Bulimia nervosa symptoms in the multimodal treatment study of children with ADHD. *Int J Eat Disord*. 2010;43(3):248–259.
91. Fischer S, le Grange D. Comorbidity and high-risk behaviors in treatment-seeking adolescents with bulimia nervosa. *Int J Eat Disord*. 2007;40(8):751–753.
92. Yilmaz Z, Kaplan AS, Zai CC, Levitan RD, Kennedy JL. COMT Val158Met variant and functional haplotypes associated with childhood ADHD history in women with bulimia nervosa. *Prog Neuropsychopharmacol Biol Psychiatry*. 2011;35(4):948–952.
93. Broft A, Shingleton R, Kaufman J, et al. Striatal dopamine in bulimia nervosa: a pet imaging study. *Int J Eat Disord*. 2012;45(5): 648–656.
94. Ostojic D, Miller CJ. Association between pubertal onset and symptoms of ADHD in female university students. *J Atten Disord*. 2014.
95. Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL, Zeitlin S. Are girls with ADHD at risk for eating disorders? Results from a controlled, five-year prospective study. *J Dev Behav Pediatr*. 2007; 28(4):302–307.
96. Katayama K, Yamashita Y, Yatsuga S, Koga Y, Matsuishi T. ADHD-like behavior in a patient with hypothalamic hamartoma. *Brain Dev*. 2016;38(1):145–148.
97. Stefano GB, Ptáček R, Kuželová H, et al. Convergent dysregulation of frontal cortical cognitive and reward systems in eating disorders. *Med Sci Monit*. 2013;19:353–358.
98. Stefano G, Ptacek R, Kuzelova H. Endogenousmorphine – up-to-date review 2011. *Folia Biol*. 2012;58(2):49–56.
99. Ptacek R, Kuzelova H, Stefano G. Genetics in psychiatry up-to-date review 2011. *Neuro Endocrinol Lett*. 2011;32(4):389–399.
100. Kream R, Stefano G, Ptacek R. Psychiatric implications of endogenous morphine: up-to-date review. *Folia Biol*. 2010;56(6):231–241.
101. Stulz N, Hepp U, Gachter C, Martin-Soelch C, Spindler A, Milos G. The severity of ADHD and eating disorder symptoms: a correlational study. *BMC Psychiatry*. 2013;13(1):44–50.
102. Wentz E, Lacey JH, Waller G, Rastam M, Turk J, Gillberg C. Childhood onset neuropsychiatric disorders in adult eating disorder patients. A pilot study. *Eur Child Adolesc Psychiatry*. 2005;14(8):431–437.
103. Mick E, Biederman J, Prince J, Fischer MJ, Faraone SV. Impact of low birth weight on attention-deficit hyperactivity disorder. *J Dev Behav Pediatr*. 2002;23(1):16–22.
104. Hart EL, Lahey BB, Loeber R, Applegate B, Frick PJ. Developmental change in attention-deficit hyperactivity disorder in boys: a four-year longitudinal study. *J Abnorm Child Psychol*. 1995;23(6):729–749.
105. Biederman J, Mick E, Faraone SV. Age-dependent decline of symptoms of attention deficit hyperactivity disorder: impact of remission definition and symptom type. *Am J Psychiatry*. 2000;157(5):816–818.
106. Anderluh M, Tchanturia K, Rabe-Hesketh S, Collier D, Treasure J. Lifetime course of eating disorders: design and validity testing of a new strategy to define the eating disorders phenotype. *Psychol Med*. 2009;39(1):105–114.
107. Castellini G, Lo Sauro C, Mannucci E, et al. Diagnostic crossover and outcome predictors in eating disorders according to DSM-IV and DSM-V proposed criteria: a 6-year follow-up study. *Psychosom Med*. 2011;73(3):270–279.
108. Stefano GB, Kream RM. Endogenous morphine synthetic pathway preceded and gave rise to catecholamine synthesis in evolution (review). *Int J Mol Med*. 2007;20(6):837–841.
109. Esch T, Kim JW, Stefano GB. Neurobiological implications of eating healthy. *Neuro Endocrinol Lett*. 2006;27(1–2):21–33.
110. Mantione KJ, Kream RM, Stefano GB. Catechol-O-methyltransferase: potential relationship to idiopathic hypertension. *Arch Med Sci*. 2010; 6(3):291–295.
111. Mantione K, Kream RM, Stefano GB. Variations in critical morphine biosynthesis genes and their potential to influence human health. *Neuro Endocrinol Lett*. 2010;31(1):11–18.