Stefano Pallanti[1,2,3], Luana Salerno[4]

[1] Dipartimento di Neuroscienze, Psicologia, Area del Farmaco e Salute del Bambino (NEUROFARBA), University of Florence, Florence, Italy;
[2] Department of Psychiatry and Behavioral Sciences, UC Davis Health System, Sacramento, CA, USA;
[3] Department of Psychiatry and Behavioral Sciences, Albert Einstein College of Medicine, New York, NY, USA;
[4] Institute of Neurosciences, Florence, Italy

ADHD: THE DARK SIDE OF EATING DISORDERS

Abstract

Objectives: Adult Attention Deficit Hyperactivity Disorder (ADHD) is a neurobiological disorder that is in most cases accompanied by other psychiatric conditions, and the latter often constitutes the reason for which adults seek professional help. Among ADHD co-occurrent conditions, Binge Eating Disorder (BED) has recently received more attention. However, there is evidence suggesting that ADHD may be a risk factor for developing not only binge eating behaviors but also other eating disorders, make them more difficult to treat with standard interventions. The aim of this review is to collect findings regarding the impact that an unrecognized and untreated ADHD may have on the onset of Eating Disorders (EDs), and explore the possibility that disordered eating may be another clinical feature of ADHD presentation.

Materials and Methods: For this aim, a PubMed search was conducted in June 17, 2016 for English-language publications from the previous 10 years. Search terms included: attention deficit hyperactivity disorder, ADHD, eating disorders, and comorbidity. Other articles have been obtained and included for their clinical and scientific relevance.

Results: Collected findings suggest that ADHD and EDs share some neurobiological and clinical features, and ADHD can predict the development of an ED. It may be possible that ADHD may foster the development of a particular form of ED that is more resistant to treatment and tends to relapse.

Conclusions: Implications of collected findings pertain to prevention of eating disorders in ADHD children and adolescents and in implementing appropriate treatment plans for adults with both ADHD and ED. Indeed, people with both ADHD and ED need specific treatment interventions, that target symptoms of ADHD and not only those of EDs. New evidence on the role of ADHD medications in the treatment of EDs has been also discussed.

Keywords: Attention deficit hyperactivity disorder, ADHD, Eating disorders, Comorbidity

Introduction

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental condition characterized by severe and age-inappropriate levels of hyperactivity, impulsivity and inattention. The core symptoms of ADHD are present in approximately 5% of children and adolescents, with an over-representation of male subjects[1]. There is evidence showing that symptoms tend to persist over the lifespan in up to 50% of cases [2], leading to lower educational, occupational, social and clinical outcomes in adulthood [3]. ADHD is a heterogeneous disorder, and up to 70% of people affected present at least one comorbid psychiatric condition, increasing social and occupational distress [3].

Eating Disorders (EDs) consist in disordered eating behaviors characterized by a clinical as well phenotypic heterogeneity. DSM-5 made several changes to their classification, recognizing Binge Eating Disorder (BED) as a distinct condition, and modifing criteria for Anorexia (AN) and Bulimia Nervosa (BN) [4]. Moreover, DSM-5 included in the chapter "Feeding and Eating Disorders" some conditions usually diagnosed in

Correspondence

Stefano Pallanti
stefanopallanti@yahoo.it

the developmental age, i.e. avoidant/restictive food intake disorder, elimination disorder, pica and rumination disorder.
Among comorbid conditions of adult ADHD, mood, anxiety and substance use disorders are the most frequently reported. Despite some researchers suggested a central role of impulsiveness in causing bulimic and binge eating behaviors [5 6] and others described the presence of attention deficits in patients with AN or BN[7], up until now very little is known about the impact that unrecognized and untreated ADHD might have on the onset, course and treatment of EDs.
Some research findings demonstrated the presence of common personality traits between ADHD individuals and those with EDs [8], but the fact that ADHD is a disorder emerging early in infancy whereas EDs tend to present in adolescence and later in life may suggest that disordered or excessive eating behaviors can be, in some cases, another expression of the same disorder, that is ADHD. In this case, being ADHD a neurodevelopmental disorder, such particular form of ED could be more difficult to treat with standard interventions, because not targeting cognitive deficits ADHD-related.
Therefore, the objective of this review is to raise awareness of the potential presence of ADHD in some EDs, that may account for some difficulty in treatment and remission.

Methods

PubMed was searched using the following combination of keywords: "Attention Deficit Hyperactivity Disorder" *OR* "ADHD*" AND* "Eating Disorders" *AND* "Comorbidity", published in English language in the last 10 years. The primary criteria for inclusion in this article were that each study had an adequate number of subjects, assessed symptoms using acceptable scales and tests, and was published during the past 10 years. Several older articles have been obtained from references and included for their scientific relevance to the aim of our paper.

Results

We found only 53 articles published in the last 10 years matching keywords and inclusion criteria, that became 28 limiting results to papers regarding adult population. Collected findings have been integrated with evidence derived from older research studies, and results have been divided in the following sections: prevalence of comorbid ADHD and EDs, the nature of comorbid ADHD and EDs, neurobiological substrates of comorbid ADHD and ED. Aggregated data have been finally discussed, informing for clinical implications and indications for future research have been also provided.

Prevalence of Comorbid ADHD and ED

Studies performed in women from the general population report a prevalence rate of 0.9% for Anorexia Nervosa (AN), of 1.5% of Bulimia Nervosa (BN) and of 3.5% of Binge Eating Disorder (BED) [9]. The vast majority of studies investigating the potential comorbidity between ADHD and ED reported higher prevalence rates: some studies found a prevalence of 11-16% of EDs (particularly Bulimia Nervosa) in people with ADHD [10-13], whereas ADHD has been found in 10-17% of subjects affected by AN purging type[14]. Previous studies [15 16] reported an increased tendency to binge in subjects with ADHD compared to controls and a prevalence of 8.3% for BED in ADHD individuals. However, other studies did not find increased ADHD rates in people with EDs [17 18].
There is evidence supporting a negative impact of ADHD on EDs. Biederman et al. [11] found that not only girls with ADHD presented a higher risk to develop an eating disorder, but in presence of both they experienced more mood, anxiety and disruptive behaviours in respect to those with only ADHD [11]. Data from this study showed females with ADHD to be 3.6 times more likely to suffer for an eating disorder compared to controls [11], and 5.6 times more likely to develop bulimia nervosa. Data from a nationally representative sample revealed that females had higher rates of comorbid ADHD and received more diagnoses of eating disorders than males (1.05% *vs* 0.20%, $p < .01$). Interestingly, in such study ADHD predicted the diagnosis of eating disorders in females but not in males [19]. These data are consistent with those by Davis et al. [20], who found childhood symptoms of ADHD to predict disordered eating in women aged 25-46 years [20], including BED.

The nature of comorbid ADHD and EDs

It has been suggested that ADHD and EDs are linked by some neuropsychological features, such as varied degrees of impulsivity, low self-esteem as well deficits in attention and impaired executive functions [9 21 22].
A higher level of impulsivity in ED subjects than healthy people have been described in several studies [5 6], and a correlation between impulsivity and severity of BN [23 24] has been also noticed. Although

Stulz et al. did not find an association between the severity of ADHD symptomatology and the severity of EDs, they found a statistically significant correlation between the level of impulsivity and the avoidance of fattening food, and also excessive fasting [25]. The latter association was completely unexpected, being excessive control of caloric intake the tipical feature of AN patients. However, authors supposed that the excessive control on what AN subjects eat may work as a sort of protection by a primary impulsivity [25], and it could explain the frequently described "diagnostic flux" within EDs category [26], particularly between AN and BN [27 26].

The important role of impulsivity in comorbid ADHD and ED has been indicated also by Mikami et al. [28], when they found that childhood impulsivity predicted BN symptoms onset in adolescence. Such data have been recently confirmed by research showing people with clinical ADHD to be more prone to disordered eating, including binge/purge and restictive eating behaviour [29], whereas individuals with a subclinical ADHD were more prone to suffer from binge/purge behaviours and not from restrictive ones [30].

A recent study confirmed the role of cognitive deficits other than impulsivity in patients with BN: those with childhood ADHD not only presented more impulsivity than those with BN alone, as measured by the total BIS score, but they showed more inattentive symptoms on the BIS subscale "Attentional Impulsivity" [31 32]. Such results lead authors to suggest an additive effect of ADHD and BN with regard to impulsivity and inattention [31]. Attention and executive deficits (EF) have suggested to play a role in disordered eating in several ways: poor inhibitory control, poor planning and impaired self-monitoring – i.e. impaired executive functioning – may foster overeating even when not hungry and without caloric concerns [20 33], whereas attentional deficits may impede to adhere to a regular dietetic regime, because of the lack of attention to the internal signs of hunger as well satiety in individuals with ADHD [34]. Moreover, compulsive eating characterizing subjects with ADHD has been interpreted as a compensatory behaviour for controlling the frustration experienced for failures in organization [35].

However, other researchers emphasized the role of motivational or reward processing problems [36 37] in emerging and mainteinance of eating disorders. This perspective finds support in the fact that food is a natural reward, and palatable food stimulates bingeing by activation of the dopaminergic reward system [38], without concerns about consequences of such exagerated eating. The reward system has a key role also in ADHD [39] and together with impairments in EF, attention deficits and poor inhibitory control, it is considered another overlapping neurobehavioral factor underlying the frequent co-occurrence of ADHD and EDs.

The association between ADHD and obesity has been more studied. Evidence shows that ADHD is a risk factor for obesity [40 41]: it has been found in 25% of treating seeking obese individuals [42], and in a 33-year follow-up study males with ADHD resulted 2 times more likely to become obese than controls [43]. However, it would be noted that binge eating behaviors are frequent in obese patients, and that overweight – as well BN and binge eating – is more frequent in people with ADHD than in the general population [20 31 33].

Neurobiological substrates of comorbid ADHD and ED

Data from neuroimaging and pharmacological studies show some shared neurobiological substrates which can give us some insights about the link between ADHD and EDs.

An explanation for comorbid ADHD and ED, particularly BED, may be found in the so-called Reward Deficiency Syndrome. The Reward deficiency syndrome is characterized by reward-seeking behavior, and it is caused by genetic variations leading to insufficient numbers of D2 receptors in the brain of people carrying the D2A1 allele. A dysfunction in DRD2 and DRD4 underlying a reward deficiency system [44-46] has been described for both ADHD [47-49], and obese people with altered eating behaviours [50-54].

We know from accumulated evidence that the cognitive deficits associated with ADHD emerge from dysfunctions particularly in fronto-striatal or mesocortical brain networks, and the alterations in reward processing have been attributed to dysfunctions in the mesolimbic dopaminergic system [37 55]. Interestingly, FMRI studies performed on adults with BN and BED also demonstrated the presence of a dysfunctional frontostriatal circuitry, responsible for self-control and impulsive behaviors [56 57], and a decreased recruitment of reward pathways in patients with persistent binge eating episodes even after treatment [58]. However, these studies did not take into account comorbid ADHD and EDs, although such investigation would be of value.

The fundamental role of dopamine (DA), as well norepinephrine (NE) systems in regulating eating behavior and reward [59 60] have been also confirmed by findings from a recent neuroimaging study, using the administration of methylphenidate in order to amplify the signals of dopamine, and showing that

food stimuli significantly increased DA in the caudate and putamen in obese subjects with BED, but not in those without BED [61]. In another study performed on subjects with BN using PET, the striatal dopamine release resulted associated with the frequency of binge eating [62]. However, in this case it is not possible to exclude the presence of an undiagnosed ADHD and its influence in such findings.

Studies on animal models suggested an involvement of alterations in the brain derived neurotropic factor (BDNF) in the relationship between ADHD and binge eating. Indeed, altered BDNF was found to cause excessive food intake in mice, as well impairments in impulse control and a tendency to become obese [63-65]. However, findings from animal models need caution to be interpreted, and generalization to humans is not possible.

It would be noted that there is some discordance about the association between severity of ADHD and severity of ED, with some studies indicating no association [25], and others finding a positive one between eating episodes and ADHD symptoms [66]. This discordance suggests the presence of other factors that could mediate the ADHD-ED interconnection. Depression may be one of them [67], and also disordered sleep may exert an effect. Indeed, disordered sleep patterns and daily sleepiness have been found associated to unhealthy eating habits leading to overweight and obesity, fostering unhealthy food consumption behaviours in children [68 69]. In this line of research, interestingly a link between disrupted circadian rhythm and obesity has been recently proposed in adults with ADHD [70]. This finding gives support to what previously suggested by Cortese and colleagues [33 71], who indicated an involvement of the hypocretin/orexin pathways in the relationship between eating, sleep and ADHD. Even though the orexin system has recently received increased attention for its importance in the regulation of emotion, reward, and energy homeostasis [72], its role in comorbid ADHD and EDs needs specific investigation.

Discussion

We believe that there is enough evidence supporting the existence of shared neurobiological underpinnings explaining the frequent presence of EDs in ADHD. Less is still known regarding the presentation of ADHD in EDs patients and its impact change to EDs emergence and maintenance. It has been largely reported how people with EDs feel embarassement, sense of guilt, depression and a sense of weakness which makes them less likely to ask for help. This is the reason for which binge eating disorder (BED) is frequently unrecognized, and it is more frequently treated when it is associated with obesity. However, as Cortese and colleagues [33] pointed out, such feelings of frustration and ineptitude frequently derives by the core deficits of ADHD, and are usually reported by adults affected by the disorder. People with ADHD suffer from their problems to persist toward goals, for their difficulty to inhibit their actions even when these may compromise their desired goal. Such features led Barkley to define ADHD as a disorder of self-regulation [73], and such impaired capacity to regulate own behavior is also a reported characteristic of people with BN and BED [74]. Evidence shows that ADHD-related deficits may create obstacles in adhering to a dietetic regime [43], and this is confirmed by recent data reporting ADHD as the main cause of treatment failure in refractory obesity candidates for bariatric surgery [75].

We believe that screening for ADHD symptoms individuals with disordered eating may help clarify difficulty in ED management and may offer new treatment options. Recent pharmacological reports suggested that ADHD medications, by acting on the brain areas involved in both ADHD and EDs, can improve not only attention and impulsive behavior but also abnormal eating [35 43 76-78]. Interestingly, recently the stimulant lisdexamfetamine (LDX) has been approved by FDA as the first medication indicated to treat moderate to severe BED in adults [79]. The rationale of its use relies on accumulated evidence of the already reported dysfunction of the dopamine (DA) and norepinephrine (NE) systems in binge eating, and in this context LDX resulted effective in facilitating DA and NE neurotransmission, and consequently in reducing pathological excessive eating [80].

The potential benefit of ADHD medication for disordered eating is not a new thing, being already suggested years ago, when Meredith et al. [81] found that the repeated amphetamine administration increased BDNF expression in the rat amygdala, piriform cortex and hypothalamus, targeting those brain pathways that are impaired in EDs. Methylphenidate resulted effective in reducing sugar craving and consequently bingeing in several studies [82-84], whereas the non-stimulant atomoxetine demonstrated positive effects on weight control in obese women [85]. By acting on noradrenergic synapses, atomoxetine showed its effect by reducing binge eating and promoting weight loss also in adults with BED [85].

Taken together all findings seem to confirm that the

presence of ADHD may influence EDs presentation, its response to treatment and also relapse. It seems the people with ADHD and ED present a double impairment because they are affected by more cognitive deficits influencing their emotional status. For this reason, people with both ADHD and ED need specific treatment interventions, targeting those symptoms of ADHD which usually are not considered in the treatment of only ED. Pharmacological intervention should consider the recent evidence regarding the effectiveness of ADHD medications in disordered eating, and need to be complemented by other non-pharmacological treatments, as psychoeducation, CBS and coaching, in order to improve those areas – attention, planning, organization, emotional control – that constitute the dysfunctional core of ADHD for facilitating the attainment of target goal, as well mainteinance of results.
Current evidence cannot exclude what we suggested, i.e. the existence of a particular subtype of ED that is expression of cognitive deficits of ADHD, and not a simple co-morbid condition. Such hypothesis finds some support in data from Fernandez-Aranda and colleagues [66] showing a different severity of ADHD among ED subtypes. Specifically, it has been found a more severe ADHD in people with BN, BED and EDNOS and a lower prevalence of ADHD in the AN group. Authors explained such findings as the expression of the common impulsivity trait characterizing ADHD and BN/BED individuals, whereas AN subjects were less affected by ADHD symptoms because of their rigidity and perfectionism, that did not match with an ADHD profile. However, it should be noted that this interpretation does not take into account the frequent diasgnostic switches of people with EDNOS (currently divided into the Other Specified Feeding or Eating Disorder or OSFED, and Unspecified Feeding or Eating Disorder or UFED) among EDs diagnostic categories. Additionally, perfectionism has been also reported in adult ADHD, as a coping strategy to overcome mental chaos [86].
It would be noted that the vast majority of reported studies present some limitations that need to be taken into account. Main limitations are the different population studied and diagnostic instruments used, many of them did not differentiate between a diagnosis of ADHD and the solely presence of symptoms of ADHD, and they usually lack of a control group. Therefore, future research should address these limitations with adequate methodology, using control groups and investigating the role of potential mediating factors.To test our hypothesis, longitudinal and perspective studies are needed.

From a clinical point of view, our review indicate how can be of value the clinical assessment of ADHD in patients with EDs, particularly in females seeking help, in light of evidence showing that girls and women are three times more likely to be treated for depression before receiving ADHD diagnosis [13].
In presence of both ADHD and ED, it should be necessary to treat ADHD first, in order to normalize those cognitive dysfunctions, such as executive deficits, impaired attention, poor impulse and emotional control, that otherwise will hamper effectiveness of treatment. New evidence from pharmacological studies reporting ADHD medications effective in people with EDs should be also considered, as well the implementation of those psychological intervention that resulted effective in ADHD individuals, because aimed to improve those executive functions (planning, organizing, control of behavior) that are compromised by the presence of such disorder.

Conclusion

In summary, aggregated evidence shows that ADHD may be a potential risk not only for binge eating, but for all EDs as well for obesity. Data from neuroimaging and pharmacological studies have given new insight on shared neurobiological underpinnings which may explain the link between ADHD and EDs, and indicate new treatment options. Clinicians should be aware of the higher prevalence of ADHD in EDs, and screen for the presence of the disorder in order to implement more efficacious interventions, by targeting those cognitive deficits characterizing people with ADHD, which if not recognized could compromise treatment results. Up until know, there are still few studies investigating comorbid ADHD and EDs, and available data cannot exclude the presence of a different subtype of ED, that may emerge in life as another clinical expression of untreated ADHD, and that is for this reason more resistant to standard treatments. Further research is needed to explore this possibility, by comparing individuals with both ADHD and EDs and comparing them with subjets affected by only ADHD or ED and matched controls. In the meanwhile, in light of the evidence indicating ADHD to be a predictor of EDs and obesity, prevention programs in ADHD population should be implemented, whereas pharmacological and non-pharmacological treatments generally proposed for adult ADHD should be considered in EDs management.

Take home messages for psychiatric care

- ADHD and EDs share some neurobiological features
- Evidence shows that ADHD can predict the development of ED
- Because of its earlier onset and cognitive deficits, ADHD may foster the development of a more resistant form of ED, and causes the relapse
- New evidence shows efficacy of ADHD medications in the treatment of EDs
- Clinicians should assess for the presence of ADHD individuals with EDs and obesity
- Future research is needed for the implementation of more targeted intervention

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