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Review

# Attention-deficit/hyperactivity disorder and eating disorders across the lifespan: A systematic review of the literature

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## HIGHLIGHTS

- Childhood ADHD is associated with the disordered eating and eating disorders.
- Support for this relationship is strongest in disordered eating and bulimia nervosa.
- ADHD may increase risk of developing eating pathology among boys more than girls.
- Children with ADHD should be monitored for disordered eating.
- Adolescents and young adults with ADHD should be monitored for eating disorders.

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## ABSTRACT

Attention-deficit/hyperactivity disorder (ADHD) and eating disorders are common and concerning mental health disorders. There is both empirical and theoretical support for an association between ADHD and eating disorders or disordered eating. This systematic review aims to summarize the extant literature on the comorbidity of ADHD and eating disorders across the lifespan, including the influences of sex, age, eating disorder diagnosis, and potential mediators. A total of 37 peer-reviewed studies on diagnosed ADHD and eating disturbances were identified through key research databases. Twenty-six studies supported a strong empirical association between ADHD and eating disorders or disordered eating. The systematic review findings suggest that children with ADHD are at risk for disordered eating, while adolescents, emerging adults, and adults are at risk for both eating disorders and disordered eating. Methodological considerations, future research, and clinical implications are discussed.



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Attention-deficit/hyperactivity disorder (ADHD) and eating disorders (EDs) are among the most common mental health disorders across the lifespan (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Existing research suggests a strong association between these disorders, although the exact nature of this association, including the prevalence of those individuals who present with both disorders, as well as factors that may account for this association, are unclear. Preliminary research suggests a temporal relationship, with childhood ADHD associated with later ED symptoms (e.g., Biederman et al., 2010), although a scan of the literature shows mixed findings to this effect.

ADHD is a neurodevelopmental disorder characterized by persistent symptoms of inattention and/or hyperactivity-impulsivity. There are three subtypes of ADHD, one marked by predominantly inattentive symptoms (ADHD-I), the second by hyperactivity and impulsiveness (ADHD-H), and the third displays a combination of inattentiveness and hyperactivity (ADHD-C; American Psychiatric Association [APA], 2013a). Recent research suggests the prevalence of ADHD among children may be as high as 15.5% (Rowland et al., 2015) with approximately a fifth of cases persisting into adulthood (Klein et al., 2012). While age is related to significant reductions in ADHD symptoms globally, the greatest declines are in symptoms of hyperactivity and impulsivity (Biederman, Mick, & Faraone, 2000). ADHD is diagnosed twice as often in boys as in girls (Polanczyk et al., 2007), although this discrepancy may be due, in part, to sex differences in symptom profiles (Sciutto, Nolfi, & Bluhm, 2004). Boys with ADHD tend to present with more disruptive symptoms, such as hyperactivity, aggressiveness, and other externalizing behaviors, while girls with ADHD tend to have less disruptive behavior and more inattentiveness and internalizing symptoms, so their symptoms may go unnoticed (Sciutto et al., 2004).

Although the precise causes of ADHD are unknown, the most important contributing factors are the interplay between primary biological and secondary environmental risk factors (Kieling, Goncalves, Tannock, & Castellanos, 2008). Twin studies demonstrate a strong genetic component with heritability estimates of approximately 0.80 (Kieling et al., 2008). A variety of genetic-environmental interactions have been implicated in ADHD, particularly those involving dopamine, such as DAT1, a dopamine transporter gene (Gizer, Ficks, & Waldman, 2009), for example, its interaction with prenatal alcohol exposure (Brookes et al., 2006). Also, ADHD imaging studies showed cortical changes, including reductions in size and functional activity in the prefrontal cortex, as well as abnormalities in dopamine transport (Bush, Valera, & Seidman, 2005). Corresponding to these brain differences, neuropsychological studies have demonstrated deficits in executive functioning and alterations in motivation and reward among individuals with ADHD (Tripp & Wickens, 2009). Additional risk factors include temperament (Kerekes et al., 2013), other environmental factors, such as low birth weight (APA, 2013a), and family dysfunction (Paidipati & Deatrick, 2015). ADHD is associated with impairments in academic, social, and vocational functioning (APA, 2013a).

Disordered eating refers to problematic eating behaviors that do not meet diagnostic threshold for an ED. It is a risk factor for developing a clinical ED (Neumark-Sztainer et al., 2006), and is likely far more common than a diagnosable ED. In adolescence, about 24% of girls and 16% of boys report disordered eating symptoms (Hautala et al., 2008) and rates are higher (31%) among adult women (Reba-Harrelson et al., 2009). The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM–5; APA, 2013b), categorizes three primary EDs: anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). AN is marked by persistent caloric restriction, intense fear of weight gain, a distorted view of weight or shape, and a refusal to maintain a minimally healthy body weight (APA, 2013b). There are two subtypes of AN, restricting (AN-R) where weight loss is achieved and maintained primarily through dieting and binge-eating/purging (AN-BP) where the individual regularly engages in overeating and compensatory behaviors (APA, 2013b). AN has the highest mortality rate of any mental illness, at approximately 5% per year (Arcelus, Mitchell, Wales, & Nielsen, 2011). BN is characterized by recurrent episodes of binge eating, inappropriate compensatory behaviors to prevent weight gain, and excessive emphasis on weight or shape in self-evaluation (APA, 2013b). BN is significantly more common than AN (Hoek, 2006). Both AN and BN typically begin in adolescence or young adulthood (Hudson, Hiripi, Pope, & Kessler, 2007). BED consists of recurrent episodes of binge eating without compensatory behaviors (APA, 2013b). The developmental pathway of BED is not well understood but binge eating is found in all age groups (APA, 2013b). The lifetime prevalence of DSM-IV EDs is 0.6% for AN, 1% for BN, and 2.8% for BED (Hudson et al., 2007), but using DSM-5 criteria would likely increase ED diagnoses significantly (Stice, Marti, & Rohde, 2013).

EDs, particularly AN, are highly familial with twin studies reporting heritability estimates ranging from 28% to 83%, demonstrating a significant contribution of genetic factors toward developing an ED (Zerwas & Bulik, 2011). The etiology of EDs may be best understood as a unique interaction between genetics, environment, and personality traits (Le Grange, 2016). Research that examines the link between EDs and other mental health disorders may elucidate common and distinct etiological mechanisms.

There is both empirical and theoretical support for an association between ADHD and EDs or disordered eating. A number of studies have found higher rates of EDs, particularly BN and BED (e.g., Surman, Randall, & Biederman, 2006), as well as disordered eating among individuals with ADHD (Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995). Similarly, there seems to be a higher prevalence of ADHD and ADHD symptoms among those with EDs (Wentz et al., 2005). Further, more boys than girls are diagnosed with ADHD, while the opposite is true of EDs. While boys with ADHD often present with externalizing symptoms such as conduct disorders and substance abuse disorders, girls may be more likely to present with internalizing disorders (Mikami, Hinshaw, Patterson, & Lee, 2008), which are linked to EDs (McDermott, Forbes, Harris, McCormack, & Gibbon, 2006). Indeed,