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Review

Attention Deficit Hyperactivity Disorder (ADHD) and disordered eating behaviour: A systematic review and a framework for future research

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HIGHLIGHTS

- Symptoms of Attention Deficit Hyperactivity Disorder (ADHD) are associated with disordered eating.
- Impulsivity symptoms of ADHD are positively associated with bulimic behaviours.
- A causal effect of ADHD on disordered eating cannot be inferred.
- Mechanistic studies on the link between ADHD and disordered eating are required.

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ABSTRACT

Preliminary findings suggest that Attention Deficit Hyperactivity Disorder (ADHD) may be associated with disordered eating behaviour, but whether there is sufficient evidence to suggest an association between ADHD and specific types of disordered eating behaviour is unclear. Furthermore, it is uncertain whether specific features associated with ADHD are differentially associated with disordered eating behaviour. A systematic review of seventy-five studies was conducted to evaluate the potential association between ADHD symptomatology and disordered eating behaviour and to provide an estimate of the strength of evidence for any association. Overall, a moderate strength of evidence exists for a positive association between ADHD and disordered eating and with specific types of disordered-eating behaviour, in particular, overeating behaviour. There is consistent evidence that impulsivity symptoms of ADHD are positively associated with overeating and bulimia nervosa and more limited evidence for an association between hyperactivity symptoms and restrictive eating in males but not females. Further research is required to assess the potential direction of the relationship between ADHD and disordered eating, the underlying mechanisms and the role of specific ADHD symptoms in the development and/or maintenance of disordered eating behaviour. We propose a framework that could be used to guide the design of future studies.

Contents

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1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood (Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014) and has a worldwide prevalence of 5% in school-age children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The symptoms of ADHD persist in adults in up to 65% of cases (Faraone, Biederman, & Mick, 2006) and the prevalence of ADHD in adults is estimated at 2.5% (Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). Despite the high prevalence of the disorder, fewer than 20% of adults with ADHD are diagnosed or treated (Ginsberg, Quintero, Anand, Casillas, & Upadhyaya, 2014). Moreover, up to 90% of adults with ADHD have comorbid psychiatric disorders (Nutt et al., 2007), which may obscure the symptoms of ADHD. Depression and other mood disorders, anxiety, personality disorders and substance use disorders (SUDs), in addition to oppositional defiant disorder, sleep problems and learning disabilities are often comorbid with ADHD (Corbisiero, Stieglitz, Retz, & Rosler, 2013; Gillberg et al., 2004; Lin, Yang, & Gau, 2015; Miller, Nigg, & Faraone, 2007; Sobanski et al., 2007).

It has also been reported that there is an association between ADHD and eating disorders (EDs) (Bleck & DeBate, 2013; Mikami et al., 2010; Mikami, Hinshaw, Patterson, & Lee, 2008). A scoping search retrieved four reviews relevant to the relationship between ADHD and eating disorders (Cortese, Bernardina, & Mouren, 2007; Curtin, Pagoto, & Mick, 2013; Nazar et al., 2008; Ptacek et al., 2016). Three of these reviews were narrative rather than systematic reviews (Cortese et al., 2007; Nazar et al., 2008; Ptacek et al., 2016). The only published systematic review focussed on individuals aged 12–21 years (Curtin et al., 2013) and thus it is unclear whether disordered eating behaviours are also present in younger children with ADHD. Furthermore, the onset of some EDs such as Binge Eating Disorder (BED) is usually later in adult life (Fairburn & Harrison, 2003), between the ages of 30 and 40 years, highlighting the importance of assessing older individuals. Our aim is to address this gap in the literature by conducting the first systematic review of the association between ADHD and disordered eating in both children and adults.

The present review will also extend knowledge of the relationship between ADHD and disordered eating by including studies that adopt a broader sampling frame than previous reviews. There is evidence that patterns of eating behaviour span a spectrum from extreme over-control of eating to loss of control and binge eating (Lowe et al., 1996; Wierenga et al., 2014), and that disordered eating patterns which do not meet clinical criteria are, nevertheless, often associated with psychopathology, and may eventually develop into a diagnosed eating disorder (Tanofsky-Kraff, Engel, Yanovski, Pine, & Nelson, 2013). Hence, greater insight into the relationship between ADHD and EDs will be gained from studying the full range of variation of eating traits and behaviours, including restrained eating, food craving and loss of control over eating (Herman & Mack, 1975; Nammi, Koka, Chinnala, & Boini, 2004; van Strien, Herman, & Verheijden, 2012; Zeeck, Stelzer, Linster, Joos, & Hartmann, 2011). This is especially relevant for young children and adolescents, for whom diagnostic criteria for EDs may not be applicable (Bravender et al., 2007; Bravender et al., 2010).

We also aim to identify studies that have investigated whether specific features of ADHD are differentially associated with specific types of disordered eating. Recent research has emphasised the role of cognitive processes in the control of eating behaviour (Higgs, 2016). Studying the relationship between individual variations in attention and cognitive control and disordered eating in ADHD provides an opportunity to identify core processes that cut across diagnostic categories and could be targeted by therapeutic interventions.

The first aim of this paper was to systematically review the literature for an association between ADHD and disordered eating. The following four questions were addressed:

(1) Is there an association between ADHD symptoms and disordered eating behaviour?
(2) Are specific features of ADHD differentially associated with specific types of disordered eating behaviour?
(3) Are there factors that affect the direction and/or strength of any relationship between ADHD symptoms and disordered eating (moderators)?
(4) Which factors could explain the relationship between ADHD symptoms and disordered eating behaviour (mediators)?

A second aim was to use our evaluation of the current evidence base to make suggestions for future research. The proposed framework could also be applied to the study of other psychiatric disorders, such as schizophrenia and mood disorders, that are associated with disordered eating.

2. Materials and methods

2.1. Literature search strategy

Original research studies examining the association between ADHD and disordered eating were selected through a literature search in 3 electronic databases: PubMed, Ovid Databases (MEDLINE, PsycINFO, EMBASE + EMBASE CLASSIC) and Web of Science-Core Collection (from 1900). The literature search was performed during May 2016 by a single investigator (PK). Searches included a combination of key words relevant to disordered eating behaviour and ADHD. For the full search strategy used, see Supplemental material. Search limiters

included human subjects and English language. These electronic searches were supplemented by a manual search of reference sections in articles identified by the electronic search and other relevant sources. The search process was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

2.2. Study selection

All original, peer-reviewed studies excluding case series/case studies and drug studies were considered suitable for inclusion. Case series/case studies were excluded as the level of evidence provided by this type of study is recognised as low (http://www.cebm.net/ocebm-levels-of-evidence/). The research evidence relating to the effect of ADHD medication on appetite and disordered eating behaviour is beyond the aim of this review and therefore drug studies were not considered for inclusion. There was no exclusion based on the age of the study participants and the selected studies included children, adolescents and/or adults with either: (1) a categorical diagnosis of ADHD according to the DSM (III, III-R, IV, IV-TR, V) or (2) or Hyperkinetic Disorder (HKD) as per the ICD-10 or previous ICD versions; or (3) for adults, a positive answer to the question: "Did your doctor ever tell you that you have ADHD?" or (4) a diagnosis of ADHD recorded in medical files/registries; or (5) a definition of ADHD using a symptoms threshold measure on a validated ADHD rating scale; or (6) ADHD symptoms measured dimensionally.

2.3. Data extraction

Data extraction was performed using standardized forms created for the review and each article was evaluated by two reviewers. The second reviewer (SH) confirmed the first reviewer's (PK) data extraction for completeness and accuracy. The following data were extracted: age range, sample size, sex distribution, study design, source population (e.g., psychiatrically referred or population-based data), methods of assessing ADHD and disordered eating, findings on the association between ADHD and disordered eating, findings on the association between specific symptoms of ADHD and disordered eating, covariates and moderator and mediators. The fully adjusted measure of association was reported if available.

2.4. Data synthesis

Data were organised according to the type of disordered eating behaviour. Where studies presented results concerning different types of disordered eating behaviour, data were included in all relevant sections. If only a limited number of studies (≤3 studies) presented data for a specific type of disordered eating behaviour, data were not narratively synthesised, as no conclusions could be drawn with confidence at this stage. These included data concerning food addiction (FA) (Davis et al., 2011), meal skipping (Pagoto et al., 2010; Ptacek et al., 2014), meal/eating frequency (Blomqvist, Ahadi, Fernell, Ek, & Dahllof, 2011; Blomqvist, Holmberg, Fernell, Ek, & Dahllof, 2007; Ptacek et al., 2014), Night Eating Syndrome (NES) and/or waking up at night to eat (Docet, Larranaga, Perez Mendez, & Garcia-Mayor, 2012; Nicolau et al., 2014; Runfola, Allison, Hardy, Lock, & Peebles, 2014), snacking (Docet et al., 2012; Ebenegger et al., 2012) and selective/picky eating (Machado, Dias, Lima, Campos, & Gonçalves, 2016; Zucker et al., 2015). Due to the degree of heterogeneity among study designs, particularly with respect to the population characteristics, the assessment methods of EDs and/or eating pathology/disordered eating and outcome measures, a meta-analysis was not conducted.

2.5. Quality and strength of evidence assessment

Critical appraisal checklists were used to evaluate the quality of the studies. Checklists were adapted from the Newcastle–Ottawa Quality Assessment Scale (Normand et al., 2005; Wells et al., 2000) and critical appraisal articles (Grimes & Schulz, 2002; Gurwitz et al., 2005; Mamdani et al., 2005; Normand et al., 2005). The Newcastle–Ottawa Quality Assessment Scale has established content validity and inter-rater reliability (Wells et al., 2000). Items reviewed included the representativeness of sampling procedure, the response rate, the validity of the measurement methods and control for important confounders. A response rate of 60% or above was considered adequate. This cut-off has been used in previous systematic reviews of observational studies (Gariepy, Nitka, & Schmitz, 2010; Wong, Cheung, & Hart, 2008). Reporting on the methodological aspects of the studies rather than relying on a numerical score for quality is considered more appropriate for systematic reviews and meta-analyses (Juni, Witschi, Bloch, & Egger, 1999). Therefore, we rated individual components of the checklist (criteria met, criteria not met, not reported) and provided an overall rating for the quality of the study (low, moderate or high). Quality assessment of studies was conducted independently by two investigators (PK and SH). Any disagreements were resolved by discussion or, if necessary, through a third investigator (CD).

Four categories were used to grade the evidence: (1) "high" grade (indicating high confidence that the evidence reflects a true association, and further research is unlikely to change the confidence level in the estimate of the association); (2) "moderate" grade (indicating moderate confidence that the evidence reflects a true association, and further research may change the confidence level in the estimate of the association and could change the estimate); (3) "low" grade (indicating low confidence that the evidence reflects a true association, and further research is likely to change the confidence level in the estimate of the association and the estimate); and (4) "insufficient" grade (indicating evidence is unavailable or limited, and a conclusion could not be drawn based on the available data).

The body of evidence was considered as consistent in direction if ≥70% of the studies had an effect in the same direction (e.g., showed significant association versus no association).

3. Results

3.1. Study selection

Fig. 1 illustrates the flowchart of study selection (Fig. 1; PRISMA flow chart). The key word search initially identified 6376 citations, 874 from PubMed, 1852 from Web of Science and 3650 from Ovid Databases. After initial screening based on the title and abstract of the article, 184 articles remained for full assessment of which 78 were excluded because they had not evaluated the relationship between ADHD and eating behaviour, 40 were review articles and 1 presented the same results as another report. Seven additional articles (Hudson, Hiripi, Pope, & Kessler, 2007; Levy, Fleming, & Klar, 2009; Mattos et al., 2004; Nazar et al., 2012; Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995; Steadman & Knouse, 2014; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011) identified through searching reference lists identified in the electronic search were also included. One article reported 4 studies that met the inclusion criteria (Surman, Randall, & Biederman, 2006). Thus, a total of 72 articles reporting on 75 suitable studies were reviewed. Due to the limited number of studies (≤3 studies) presenting data for a specific type of disordered eating behaviour, data from 8 studies were not analysed further (Blomqvist et al., 2011; Blomqvist et al., 2007; Davis et al., 2011; Ebenegger et al., 2012; Machado et al., 2016; Ptacek et al., 2014; Runfola et al., 2014; Zucker et al., 2015).

3.2. Characteristics of the included studies

Supplementary material summarizes the characteristics and main findings of the studies included. The total number of participants included in the current review was 115,418. Sample sizes of the 75 studies varied widely between 26 and 12,366. Most studies included both male and

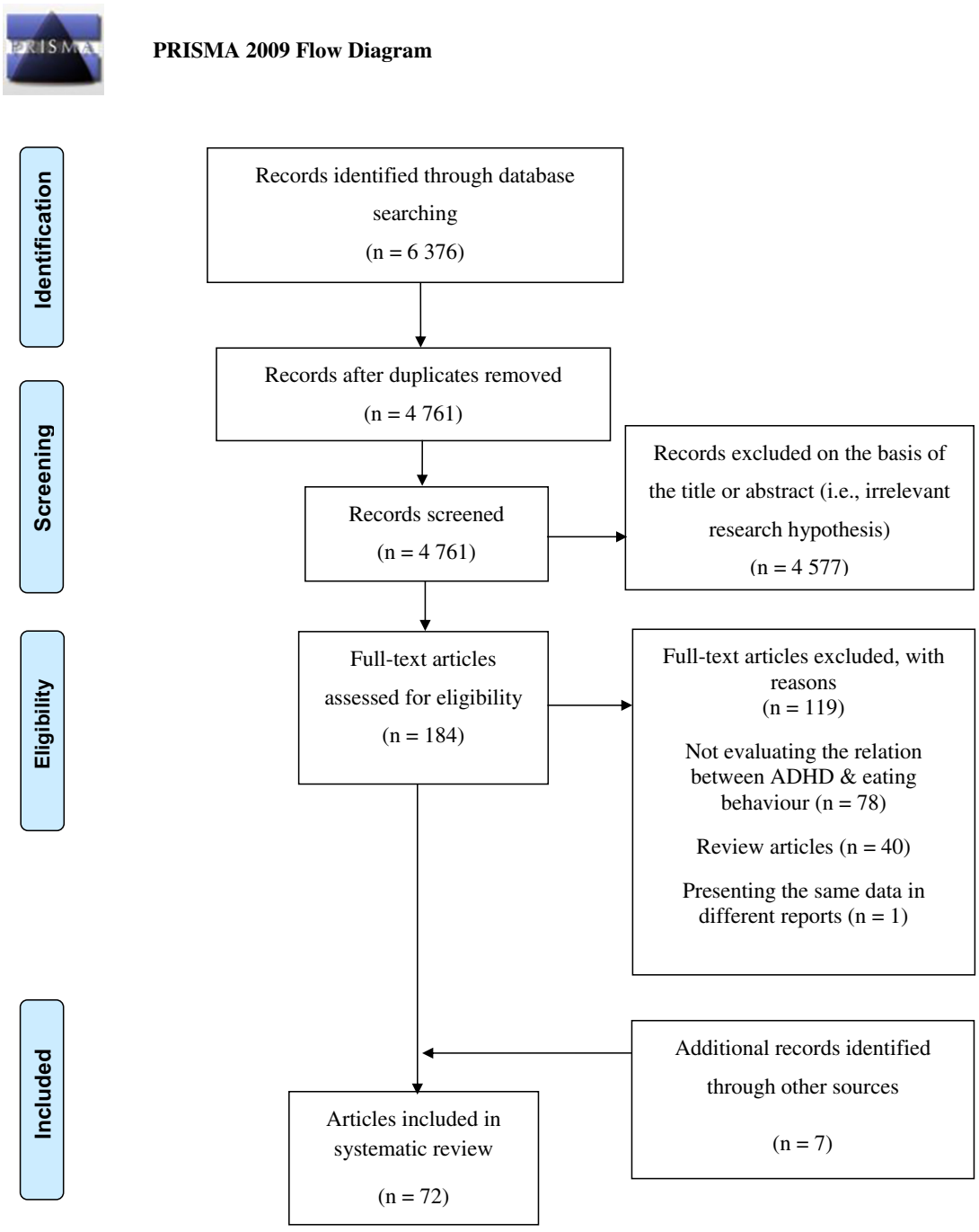

Fig. 1. Flow chart of study selection process.

female participants ($n = 57$) and of those studies the majority ($n = 34$) included more females than males. Fifteen studies included only females and 3 studies only males. The publication year ranged from 1995 to 2016 and 27 of the 75 studies were published within the last 3 years. Most studies were cross-sectional ($n = 37$), 11 studies were case-control, 6 were cohort studies, 5 were prospective, 7 were longitudinal or part of a longitudinal study, 2 were a secondary analysis of data from the National Longitudinal study of Adolescent Health, 3 were experimental studies, 3 studies were retrospective reviews and 1 was an epidemiological study. Thirty-five studies were conducted in Europe, thirty-seven in America and two in Asia. One study used data collected in the United States, Canada, Great Britain and the Netherlands. Thirty studies recruited children and adolescent participants (age range: 1.5–18 years), 35 studies recruited adults (age ≥ 18 years) and 10 studies recruited children and/or adolescents and adults (minimum age: 6 years). Study participants were recruited from the general population ($n = 29$), university students ($n = 3$), children referred through community pediatric mental health clinics, medical clinics and flyers on public bulletin boards ($n = 1$), clinical populations with EDs ($n = 6$) and ADHD ($n = 15$), adults referred with ADHD and/or autism spectrum disorders (ASD) ($n = 1$), tic disorder specialty clinics in the United States, Canada, Great Britain and the Netherlands as from the Tourette Syndrome Association of the United States ($n = 1$), female offenders ($n = 1$), obese children who received, presented or were referred for weight-loss treatment ($n = 5$), obese women seeking non-surgical treatment for obesity ($n = 2$), obese patients attending the Nutrition section of a hospital ($n = 1$), obese patients who underwent or were candidates for bariatric surgery ($n = 6$), severely obese patients who

had been referred for the treatment of refractory obesity ($n = 1$) and obese individuals in community settings ($n = 3$). In 41 studies the association between ADHD and disordered eating was not the main focus of the study and estimates were usually displayed in a descriptive table.

3.3. ADHD and eating disorders (EDs)

Eleven studies reported data relevant to the association between ADHD and EDs, without providing any information about the type of ED (see Supplementary material). Two studies used a case-control design (Bijlenga et al., 2013; Sobanski et al., 2007), 1 study was a two-part longitudinal study with a one-year interval (Viborg, Wangby-Lundh, & Lundh, 2014), 1 study was a longitudinal study with a two-year follow-up (Rojo-Moreno et al., 2015), 1 study was a cohort study (Yoshimasu et al., 2012), 1 was an epidemiological study (Lewinsohn, Shankman, Gau, & Klein, 2004), 3 were cross-sectional studies (Hirschtritt et al., 2015; Karjalainen, Gillberg, Rastam, & Wentz, 2016; Stulz et al., 2013) and 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck, DeBate, & Olivardia, 2015). All studies except two, which included only females (Stulz et al., 2013; Viborg et al., 2014) included both sexes. Overall, significant and positive associations between ADHD and EDs were found in 8 of the 11 studies.

3.4. ADHD and anorexia nervosa (AN)/restrictive eating

Nineteen studies reported data relevant to the association between ADHD and AN/Restrictive eating (see Supplementary material). Two studies were prospective (Biederman et al., 2007; Biederman et al., 2010), 10 studies were cross-sectional (Dempsey, Dyehouse, & Schafer, 2011; Grabarek & Cooper, 2008; Hudson et al., 2007; Karjalainen et al., 2016; Muller, Claes, Wilderjans, & de Zwaan, 2014; Pauli-Pott, Becker, Albayrak, Hebebrand, & Pott, 2013; Rosler, Retz, Yaqoobi, Burg, & Retz-Junginger, 2009; Slane, Burt, & Klump, 2010; Swanson et al., 2011; Wentz et al., 2005), 1 study was an ongoing longitudinal study (Rastam et al., 2013), 2 were part of longitudinal studies (Malmberg, Edbom, Wargelius, & Larsson, 2011; Yates, Lund, Johnson, Mitchell, & McKee, 2009), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck et al., 2015) and 2 studies were retrospective reviews (Blinder, Cumella, & Sanathara, 2006; Welch, Ghaderi, & Swenne, 2015). Fourteen studies included both sexes and 5 studies included only females.

Restrictive eating symptoms investigated across these studies included adoption of behaviours such as use of weight-loss pills, fasting or meal skipping, replacement of meals with food supplements or use of food supplements to reduce appetite in order to lose or maintain body weight, failure to gain sufficient weight for more than a year, fear of gaining weight or growing fat, fear of becoming obese, drive for thinness and restraint eating. Overall, 9 of 19 studies reported significant and positive associations between ADHD and AN/Restrictive eating and for 3 of these studies the associations were significant only for males (Grabarek & Cooper, 2008; Rastam et al., 2013; Welch et al., 2015). Three studies found significant associations only between specific symptoms of ADHD (hyperactivity and hyperactivity/impulsivity) and restrictive eating behaviour (Grabarek & Cooper, 2008; Rastam et al., 2013; Slane et al., 2010).

Population studies with large sample sizes (ranging from 5000 to 12,262 participants) in general reported a non-significant association between ADHD and AN/Restrictive eating symptoms (Bleck & DeBate, 2013; Hudson et al., 2007; Swanson et al., 2011). However, the failure to detect an association in these studies may be due to methodological issues rather than representing a true null result. Specifically, analyses of associations in some studies may have been underpowered owing to the relatively small numbers of participants with AN (Hudson et al., 2007; Swanson et al., 2011) and to the measure used to assess restrictive eating pathology (Bleck & DeBate, 2013).

Overall, the studies that found significant associations were characterised as low quality, with only two studies rated as moderate quality (Bleck et al., 2015; Slane et al., 2010). The most important methodological limitations were small sample sizes (Grabarek & Cooper, 2008; Rosler et al., 2009; Welch et al., 2015; Wentz et al., 2005), study of a forensic population with a high prevalence of Axis I disorders (Rosler et al., 2009), use of non-validated tools to assess eating problems (Bleck et al., 2015; Rastam et al., 2013), absence of a control group (Grabarek & Cooper, 2008; Karjalainen et al., 2016; Malmberg et al., 2011; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015; Wentz et al., 2005) and lack of control for confounding variables (Grabarek & Cooper, 2008; Malmberg et al., 2011; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015; Wentz et al., 2005).

3.5. ADHD and bulimia nervosa (BN)

Twenty-one studies evaluated the association between ADHD and BN and/or BN symptoms (see Supplementary material). Five studies were prospective studies (Biederman et al., 2007; Biederman et al., 2010; Hinshaw et al., 2012; Mikami et al., 2010; Mikami et al., 2008), 5 studies used a case-control design (Seitz et al., 2013; Surman et al., 2006), 8 studies were cross-sectional studies (Cortese, Isnard, et al., 2007; De Zwaan et al., 2011; Grabarek & Cooper, 2008; Hudson et al., 2007; Nazar et al., 2012; Neumark-Sztainer et al., 1995; Rosler et al., 2009; Swanson et al., 2011), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck et al., 2015) and 1 study was a retrospective chart review (Blinder et al., 2006). Eight studies included only females and 13 included both sexes. Overall, significant and positive associations between ADHD and BN/BN symptoms were found in 17 of the 21 studies.

Prospective evidence generally showed a significant positive association between ADHD and BN/BN symptoms (Biederman et al., 2007; Biederman et al., 2010). Only one prospective study did not find a significant association between ADHD and BN/BN symptoms. Hinshaw et al. (2012) in contrast with their previous findings (Mikami et al., 2008) found that girls with childhood-diagnosed ADHD did not differ significantly from controls in terms of BN symptoms at a 10-year follow-up. However, the power of the analysis might have been limited because participants lost in follow-up were poorer, had lower IQ scores and were more symptomatic than the retained sample and there was a large variation observed in continued service utilisation and medication use over the follow-up period.

Findings from case-control studies also pointed to a significant association between ADHD and BN in adult females (Seitz et al., 2013; Surman et al., 2006). Similarly, results from cross-sectional studies suggested a positive association between ADHD and BN/BN symptoms (Cortese, Isnard, et al., 2007; De Zwaan et al., 2011; Hudson et al., 2007; Nazar et al., 2012; Neumark-Sztainer et al., 1995; Rosler et al., 2009; Swanson et al., 2011).

3.6. ADHD and binge eating disorder (BED)

Twenty-seven studies reported data relevant to an association between ADHD and BED symptoms (see Supplementary material). Most studies were cross-sectional ($n = 17$) (Agranat-Meged et al., 2005; Alfonsson, Parling, & Ghaderi, 2012; Davis, Cohen, Davids, & Rabindranath, 2015; De Zwaan et al., 2011; Gruss, Mueller, Horbach, Martin, & de Zwaan, 2012; Hudson et al., 2007; Mattos et al., 2004; Muller et al., 2012; Muller et al., 2014; Nazar et al., 2012; Nazar et al., 2014; Pagoto et al., 2009; Pauli-Pott et al., 2013; Rosler et al., 2009; Slane et al., 2010; Steadman & Knouse, 2014; Swanson et al., 2011), 2 studies used a case-control design (Docet et al., 2012; Nicolau et al., 2014), 1 study used a case-double control design (Davis et al., 2009), 1

study was a retrospective chart review (Reinblatt et al., 2015), 1 study was longitudinal (Goldschmidt, Hipwell, Stepp, McTigue, & Keenan, 2015), 1 was part of a longitudinal clinical intervention study (Levy et al., 2009), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013) and 2 studies were cohort studies (Khalife et al., 2014; Sonneville et al., 2015). Four studies recruited females and the remaining 23 studies recruited both sexes. Overall, significant and positive associations between ADHD and BED/BED symptoms were found in 20 of the 27 studies.

Of the studies that showed significant associations, 3 studies found significant associations between specific symptoms of ADHD (hyperactivity, hyperactivity/inattention and hyperactivity/impulsivity) and binge eating (Goldschmidt et al., 2015; Slane et al., 2010; Sonneville et al., 2015), 2 studies did not differentiate between binging and/or purging behaviour (Bleck & DeBate, 2013; Bleck et al., 2015) and another study found that the association was significant only in a subgroup of participants (Muller et al., 2014). Specifically, Muller et al. (2014), investigating temperament subtypes among treatment seeking obese individuals, found that patients who were characterised as "emotionally dysregulated/undercontrolled" showed more ADHD symptoms compared to "resilient/high functioning" patients. The prevalence of BED was also higher in the "emotionally dysregulated/undercontrolled" group compared to the "resilient/high functioning" group (55.9% vs. 27.3%), suggesting a positive association between ADHD and BED in obese "emotionally dysregulated/undercontrolled" adults.

Findings from population studies generally supported a significant positive association between ADHD and BED symptoms (Bleck & DeBate, 2013; Bleck et al., 2015; De Zwaan et al., 2011; Goldschmidt et al., 2015; Hudson et al., 2007; Pagoto et al., 2009; Slane et al., 2010; Sonneville et al., 2015; Swanson et al., 2011). The 6 studies that did not find a significant association between ADHD and BED and/or BED symptoms recruited children and adolescents who were overweight/obese and hospitalised (Agranat-Meged et al., 2005) or referred for weight reduction (Pauli-Pott et al., 2013) and obese adults (Alfonsson et al., 2012; Davis et al., 2009; Gruss et al., 2012; Muller et al., 2012) who were mainly presenting for bariatric surgery. In general, these studies were limited by very small sample sizes of participants with ADHD (ranging from 8 to 19 individuals). Only one of these 6 studies (Davis et al., 2009) recruited obese adults from a community setting that was not limited by a small sample size.

3.7. ADHD and loss of control over eating (LOC-eating)

Five studies were identified that evaluated an association between ADHD and LOC-eating (see Supplementary material) and significant positive associations were found in all 4 studies that were cross-sectional (Alfonsson et al., 2012; Alfonsson, Parling, & Ghaderi, 2013; Erhart et al., 2012; Reinblatt et al., 2015) and included both sexes.

Pagoto et al. (2010) did not directly measure LOC-eating, but investigated the association between symptoms of ADHD and perceived self-efficacy to control eating in a sample of obese patients who had completed a 16-week clinic-based behavioural weight loss program. In line with the previous findings, Pagoto et al. (2010) reported that individuals who screened positive for adult ADHD reported lower self-efficacy to control their eating compared to controls.

3.8. ADHD and overeating behaviour

Twelve studies reported data relevant to an association between ADHD and overeating (see Supplementary material). Nine studies were cross-sectional (Alfonsson et al., 2012, 2013; Davis, Levitan, Smith, Tweed, & Curtis, 2006; Dempsey et al., 2011; Kim et al., 2014; Pagoto et al., 2010; Patte et al., 2016; Pauli-Pott et al., 2013; Strimas et al., 2008) and 3 used an experimental design (Hartmann, Rief, & Hilbert, 2013; Munsch, Hasenboehler, & Meyer, 2011; Wilhelm et al., 2011). Two studies included only males, one included only females, and the remaining 9 studies included both sexes. Eating behaviour related to overeating, included eating in the presence of emotional distress (emotional eating), eating in response to the palatability and appearance of food (hedonic eating), external eating, eating as a result of increased susceptibility to feelings of hunger, binge eating and tendency to overeat in the presence of palatable foods, other individuals who are overeating, or other disinhibiting stimuli (disinhibited eating). Significant positive associations between ADHD and overeating behaviours were found in 10 of the 12 studies.

Cross-sectional findings generally pointed to a significant association between ADHD and overeating behaviour. In a large study of > 10,000 children from elementary schools in Korea, Kim et al. (2014), used structural equation modelling to investigate associations between ADHD, dietary behaviour and BMI. ADHD was positively associated with consumption of "unhealthy" food, the number of overeating episodes per week and diet speed. Using a similar approach, Davis et al. (2006), in a sample of adult healthy women from the general population, investigated whether ADHD contributes to the obesity risk profile because it fosters a tendency to overeat. Symptoms of ADHD were positively correlated with aspects of overeating, including emotional eating, external eating and binge eating and overeating correlated with higher BMI. Similarly, in a sample of healthy adult males, a positive association between symptoms of ADHD and overeating behaviour was observed, which positively correlated with BMI (Strimas et al., 2008). Recent findings have confirmed these previous reports and together with binge eating, emotional eating and hedonic eating were found to play a significant role in the association between ADHD symptoms and BMI (Patte et al., 2016).

Eating and/or craving food in response to emotional distress was also found to be positively correlated with ADHD in studies of obese adults (Alfonsson et al., 2012, 2013; Pagoto et al., 2010). However, Pauli-Pott et al. (2013) did not find a significant difference in emotional eating between overweight/obese children and adolescents with clinical and/or sub-clinical symptoms of ADHD and overweight/obese children and adolescents without symptoms of ADHD. The very small number of children/adolescents in the study who met the clinical criteria for ADHD ($n = 17$) together with the questions about emotional eating being limited to feelings of loneliness, disappointment and unhappiness suggests that these findings should be interpreted with caution.

Experimental studies are relatively limited in children and adolescents. Two studies reported significant positive associations between ADHD and laboratory measured food intake (Hartmann et al., 2013; Munsch et al., 2011), whereas another study reported no association (Wilhelm et al., 2011). Methodological differences may explain these inconsistent findings. Thus, Wilhelm et al. (2011), in contrast to the other two research groups (Hartmann et al., 2013; Munsch et al., 2011) assessed food intake of hungry participants after an overnight fast. Therefore, it is possible that overeating tendencies are more reliably investigated after a standardized meal in a satiated state.

3.9. Quality ratings and strength of evidence

Inter-rater agreement for quality assessment was good (kappa 0.78, 95% CI: 0.64–0.92). Quality ratings varied significantly across studies although most of the studies included were moderate ($n = 44$) quality. Twelve studies were high quality, and the remaining 19 studies were low quality. Small sample sizes, absence of control groups, non-representative sampling procedures and poor or no control of confounds were the main limitations.

Fig. 2 illustrates the strength of evidence available concerning the association between ADHD and disordered eating behaviour. Overall, there is consistent moderate to high strength of evidence that ADHD is associated positively with BN/BN symptoms. There is consistent moderate strength of evidence that ADHD is associated positively with: EDs,

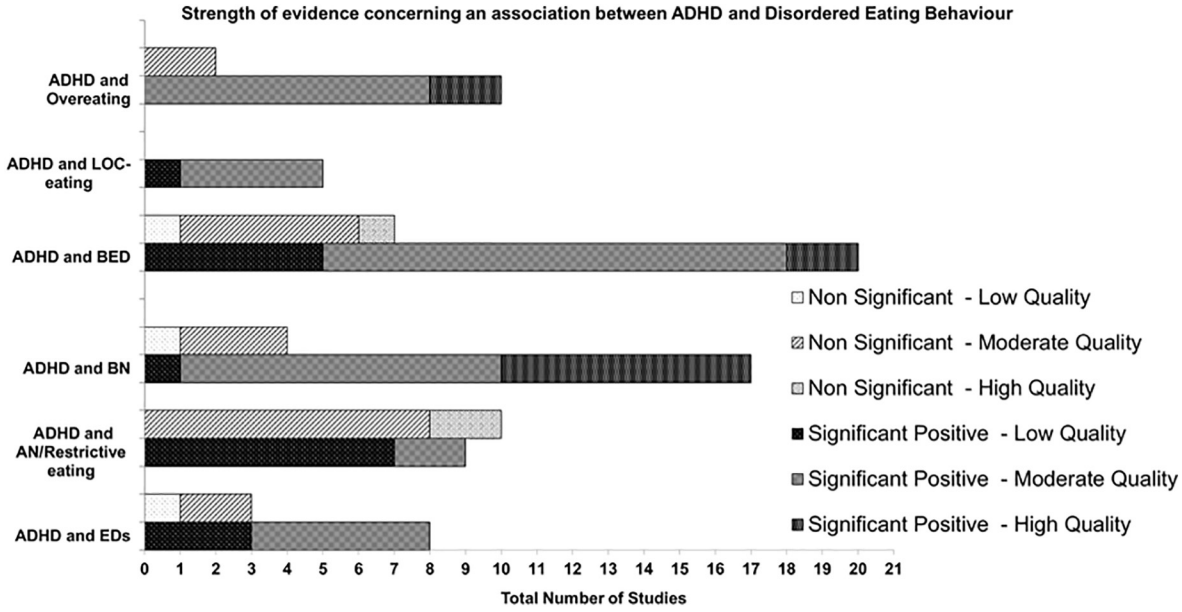

Fig. 2. Strength of evidence concerning an association between ADHD and disordered eating behaviour.

BED/BED symptoms, LOC-eating and overeating behaviour. There is inconsistent evidence whether an association exists between ADHD and AN/Restrictive eating.

3.10. Specific symptoms of ADHD and disordered eating behaviour

Twenty of the 75 studies in this review reported associations between specific symptoms of ADHD and ED types (see Supplementary material). Most studies were cross-sectional studies ($n = 9$) (Alfonsson et al., 2012; Cortese, Isnard, et al., 2007; Ebenegger et al., 2012; Grabarek & Cooper, 2008; Muller et al., 2014; Nazar et al., 2014; Reinblatt et al., 2015; Slane et al., 2010; Yates et al., 2009). Two studies were prospective (Mikami et al., 2010; Mikami et al., 2008), 2 studies were longitudinal (Goldschmidt et al., 2015; Rastam et al., 2013), 2 studies were cohort studies (Khalife et al., 2014; Sonneville et al., 2015), 1 study used a case-control design (Seitz et al., 2013), 1 study was a case double-control study (Davis et al., 2009), 2 studies were experimental (Munsch et al., 2011; Wilhelm et al., 2011) and 1 was a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck et al., 2015). Five studies included only females, 1 included only males and the remaining 14 studies included both sexes.

Overall, evidence was mixed, with the exception of studies that assessed the association between impulsivity symptoms of ADHD and BN/BN symptoms which generally found significant positive associations. Mikami et al. (2010, 2008) found that childhood impulsivity symptoms as opposed to hyperactivity and inattention best predicted adolescent BN symptoms at 5 and 8 years follow-up. Cortese, Isnard, et al. (2007) also found indirect evidence that the association between ADHD symptoms and bulimic behaviour in severely obese adolescents can be accounted for mainly by symptoms of impulsivity and inattention but not hyperactivity. However, Seitz et al. (2013) found that inattentive, rather than impulsivity or hyperactivity symptoms of ADHD, explained the severity of BN symptoms, but the recruitment of a clinical population of females seeking treatment for BN limits the generalisability of these findings. The absence of a relationship between hyperactivity symptoms of ADHD and BN/BN symptoms was also a consistent finding among studies that investigated this potential association (Cortese, Isnard, et al., 2007; Mikami et al., 2010; Mikami et al., 2008; Seitz et al., 2013).

Interestingly, hyperactivity symptoms were significantly associated with restrictive eating symptomatology, particularly in men. Grabarek and Cooper (2008) explored sex-based patterns of relationships between symptoms of ADHD and eating pathology and found that hyperactivity (and not inattention) was significantly related to a drive for thinness in men. However, no significant correlation, for either the inattention or the hyperactivity subscale and drive for thinness was found for women. In line with these findings, hyperactivity/impulsivity symptoms were strongly associated with restrictive eating pathology in boys, but not in girls (Rastam et al., 2013).

Findings on the association between inattentive symptoms of ADHD and specific ED subtypes were generally mixed, with the exception of studies that assessed their association with restrictive eating disorders (Yates et al., 2009) and other symptomatology related to restrictive eating (Grabarek & Cooper, 2008; Rastam et al., 2013), which consistently reported non-significant associations. Studies that assessed the relationship between inattentive symptoms of ADHD and BN symptoms or BED symptoms, and reported significant positive associations were cross-sectional, and included clinical populations; specifically eating disorder inpatients (Yates et al., 2009), females seeking treatment for BN(Seitz et al., 2013), obese women seeking non-surgical treatment for obesity or eating disorders (Nazar et al., 2014), bariatric surgery candidates and obese inpatients (Muller et al., 2014) and severely obese adolescents participating in a 6- to 11-month weight loss program (Cortese, Isnard, et al., 2007).

Findings on the association between specific symptoms of ADHD and LOC-eating, although limited, pointed to a significant association between hyperactivity/impulsivity symptoms and LOC-eating (Alfonsson et al., 2012; Reinblatt et al., 2015).

3.11. Moderators and mediators

Twenty-one of 75 studies reported participant characteristics for moderation. Most studies ($n = 18$) investigated the moderating effect of sex on the association between ADHD and disordered eating behaviour (Alfonsson et al., 2013; Bleck & DeBate, 2013; Davis et al., 2015; Grabarek & Cooper, 2008; Mattos et al., 2004; Mikami et al., 2010; Neumark-Sztainer et al., 1995; Patte et al., 2016; Pauli-Pott et al., 2013; Rastam et al., 2013; Slane et al., 2010; Sobanski et al., 2007; Surman et al., 2006; Welch et al., 2015; Yoshimasu et al., 2012). The findings were mixed: eight studies reported a significant effect of sex

(Grabarek & Cooper, 2008; Mikami et al., 2010; Rastam et al., 2013; Sobanski et al., 2007; Surman et al., 2006), while 8 studies found no significant effect (Alfonsson et al., 2013; Davis et al., 2015; Mattos et al., 2004; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010; Welch et al., 2015; Yoshimasu et al., 2012). One study found a significant effect for diagnosed EDs but not for disordered eating behaviour (Bleck & DeBate, 2013), and another found a significant sex effect only for use of laxatives, diuretics or emetics for weight loss (Neumark-Sztainer et al., 1995).

When stratified by type of ED, the results were more consistent. Thus, Mikami et al. (2010) found that the positive association observed between childhood ADHD symptoms of impulsivity and parent-reported BN symptoms at 8-year follow-up was stronger for girls than for boys. Surman et al. (2006), in an analysis of two samples of adults, reported significantly greater rates of BN in women with ADHD compared to women without ADHD; however, rates of BN did not differ between men with and without ADHD. Neumark-Sztainer et al. (1995) found that adolescents with ADHD reported more symptoms related to BN pathology than controls. However, only girls with ADHD reported the use of laxatives, diuretics or emetics for weight loss. Findings on the moderating effect of sex on the association between ADHD symptomatology and binge eating generally pointed to a non-significant effect (Davis et al., 2015; Mattos et al., 2004; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010). Similarly, Alfonsson et al. (2013) reported no significant gender effect on the relationship between ADHD and LOC-eating, a core feature of binge eating. Five studies assessed the moderating effect of sex on the association between symptoms of ADHD and AN/restraint eating symptomatology (Grabarek & Cooper, 2008; Pauli-Pott et al., 2013; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015). Findings from adults consistently indicated that hyperactivity symptoms of ADHD were more strongly related to dietary restraint and/or drive for thinness in men than women (Grabarek & Cooper, 2008; Slane et al., 2010). Hyperactivity/impulsivity symptoms of ADHD were also found to be strongly associated with restrictive eating pathology in boys, but not in girls (Rastam et al., 2013). However, in overweight/obese children and adolescents, Pauli-Pott et al. (2013) did not find a significant sex effect on the relationship between ADHD and dietary restraint.

Findings on the moderating effect of age were limited as no studies tested the effect of age using moderation analysis, but 4 studies reported relevant data. Surman et al. (2006) suggest a significant effect of age on the association between ADHD and BN, especially for women. Further, Mikami et al. (2010, 2008) assessed the longitudinal association between ADHD and BN in girls and found that although no girls met full diagnostic criteria for BN at 5 and 8 years follow-up, those with ADHD were more likely to have disordered eating behaviour and BN symptoms.

Three studies reported data on the moderating effect of weight (Erhart et al., 2012; Mikami et al., 2010; Wilhelm et al., 2011) suggesting that the effect of ADHD on eating behaviour does not depend on the weight of the individual.

Other moderators explored included race, punitive parenting, parental expression of emotion (a construct measuring parental criticism of and emotional over-involvement with the child) and peer rejection (Bleck & DeBate, 2013; Mikami et al., 2008). The only study to examine the moderating effect of race, reported that Asian females with ADHD were significantly less likely to have an ED than White females with ADHD (IRR: <0.01; 95% CI: <0.01–<0.01) (Bleck & DeBate, 2013). Punitive parenting, parental expression emotion and peer rejection were assessed as potential moderators by Mikami et al. (2008). Punitive parenting was reported by Mikami et al. (2008) to be a significant predictor of pathological eating in girls with ADHD, but not in controls (significant interaction between ADHD diagnosis and punitive parenting, $B = 0.24$; $p < 0.01$). Interactions between expression of emotion or peer rejection and ADHD were not significant.

Only three of the 75 studies included in this review tested for mediation. Sonneville et al. (2015), in a sample of children/adolescents assessed the relationship between ADHD and binge eating, and found that symptoms of ADHD (hyperactivity/inattention) during late childhood were significantly associated with binge-eating in mid-adolescence, and this relationship was mediated via overeating in late-childhood and a strong desire for food in early adolescence. To investigate whether impulsivity can explain the relationship between ADHD symptoms and binge eating, Steadman and Knouse (2014) tested this mediation pathway in undergraduate students. As impulsivity is a multifactorial construct, Steadman and Knouse (2014) used different approaches to measure impulsivity (the Barratt Impulsiveness Scale, the Impulsiveness Questionnaire, the Barkley Deficits Executive, Self-Restraint subscale and a behavioural Go/No-Go task), which are thought to represent different underlying processes. However, none of the measures of impulsivity were found to be significant mediators between ADHD and binge eating symptoms. Davis et al. (2015) using a community-based sample of young men and women found that a high-risk personality profile associated with impulsive and rash responding, and with anxiety proneness may, in part, account for the relationship between ADHD symptomatology and the use/abuse of a broad range of addictive behaviours including food binging.

4. Discussion

To the best of our knowledge, this is the first review that has systematically assessed the association between ADHD and disordered eating. Our aim was to evaluate whether there is sufficient evidence to suggest an association between ADHD and disordered eating behaviour, and to examine if ADHD is associated with specific types of disordered eating behaviour. Seventy-five studies were identified and included in this review and overall, the evidence suggests that ADHD is positively associated with disordered eating. However, the strength of evidence is moderate because the majority of studies were limited by methodological issues, including non-representative sampling of participants, small sample sizes, the absence of control groups and poor control for confounds. In addition, there was heterogeneity across studies, particularly in the sample characteristics and the measurement of disordered eating and ADHD, which may confound interpretation of the results. Furthermore, most studies were cross-sectional, and therefore a causal relationship between ADHD and disordered eating cannot be inferred, and the possibility of reverse causality cannot be excluded. The research evidence relating to the effect of ADHD medication on appetite and disordered eating behaviour is beyond the aim of this review. However, medication for ADHD is potentially a significant confounder when assessing a possible association between ADHD and EDs and/or eating pathology/disordered eating. Nevertheless, within the studies reviewed here only five controlled for stimulant medication (Davis et al., 2015; Hinshaw et al., 2012; Pagoto et al., 2009; Reinblatt et al., 2015; Wilhelm et al., 2011), and none of these controlled specifically for the type of stimulant medication, while only one controlled specifically for the dose of stimulant medication (Wilhelm et al., 2011). There is consistent moderate to high strength of evidence that ADHD is positively associated with BN symptoms, and consistent moderate strength of evidence that ADHD is positively associated with eating behaviour related to overeating, including BED symptoms, emotional, external, disinhibited eating and LOC-eating. Evidence for an association between ADHD and AN/Restrictive eating is inconsistent.

Of the studies reviewed only 21 out of the 75 reported participant characteristics for moderation. Sex was one of the commonly reported moderators (Davis et al., 2015; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010). Although all of the studies that stratified by sex provide evidence for a stronger association between ADHD and BN/BN symptoms in females than males, it is unclear whether the associations found between sex and BN/BN symptoms are unique to ADHD or simply indicate general population patterns of differential sex prevalence of BN/BN symptoms. In addition, although it is consistently reported that the association between hyperactivity symptoms of ADHD and

restrained eating symptomatology is stronger for males than females, it is plausible that this is a reflection of the higher hyperactivity symptoms in males compared to females (Gaub & Carlson, 1997; Williamson & Johnston, 2015) and not a higher predisposition for males with ADHD to be diagnosed with restrained eating. There is currently insufficient evidence to determine whether age, weight and ADHD medication moderate the relationship between ADHD and disordered eating behaviour.

Mechanism testing was very limited in the studies reviewed and only three of the 75 studies tested for mediation (Davis et al., 2015; Sonneville et al., 2015; Steadman & Knouse, 2014). Sonneville et al. (2015) found that the association between ADHD and binge eating in mid-adolescence was mediated by childhood over-eating and strong desire for food in early-adolescence, suggesting that early childhood ADHD symptoms in, in addition to an overeating phenotype, may contribute to risk for adolescent binge eating via a strong desire for food in early adolescence. However, several other pathways could explain the association between ADHD and disordered eating behaviour.

It is conceivable that disordered eating behaviours are not directly related to ADHD and may be mediated by other, often common, co-existing health-related conditions, such as depression and other mood disorders (Austerman, 2015) and EDs (Blinder et al., 2006; Casper, 1998; Keel, Klump, Miller, McGue, & Iacono, 2005). Adjustment for comorbid conditions that are common in ADHD was limited in the studies reviewed and only 8 of the 75 studies reported an adjusted measure of association. Of these studies a significant association between pathological eating and ADHD, persisted after adjusting for a variety of covariates, including depression, anxiety, mood, antisocial behaviour, stage of development and internalizing and externalizing conditions (Biederman et al., 2010; Cortese, Isnard, et al., 2007; Mikami et al., 2008; Reinblatt et al., 2015), suggesting that ADHD is a specific risk factor for eating pathology.

Alternatively, biological factors such as shared common genetic variants could partly explain the link between ADHD and disordered eating. For example, dysfunctions in the dopamine pathways of the brain have been found among both individuals who are obese (Val-Laillet et al., 2015) and individuals with ADHD (Badgaiyan, Sinha, Sajjad, & Wack, 2015). Recent findings also suggest that individuals who have ADHD symptoms and carry genetic profiles associated with greater dopamine activation in brain reward areas are more likely to engage in overeating behaviour, such as binge, emotional and hedonic eating (Patte et al., 2016).

Personality factors should also be considered. An avoidant coping style has been associated with impaired eating behaviour (Martyn-Nemeth, Penckofer, Gulanick, Velsor-Friedrich, & Bryant, 2009; Troop, Holbrey, Trowler, & Treasure, 1994). Furthermore, eating psychopathology has been associated with decreased use of adaptive coping strategies (e.g., problem-focused coping) (Mayhew & Edelmann, 1989; Troop et al., 1994). Therefore, the association between ADHD and disordered eating may be complex and compounded by feedback loops including an ability to cope with challenges in life.

It is also plausible that core processes underlying ADHD give rise to pathological eating patterns. Impulsivity, a main feature of ADHD has been found to be a characteristic of patients with BN and BED (Dawe & Loxton, 2004; Fischer, Smith, & Anderson, 2003; Penas-Lledo & Waller, 2001). Four of the studies reviewed reported associations between impulsivity symptoms of ADHD and BN/BN symptoms (Cortese, Isnard, et al., 2007; Mikami et al., 2010; Mikami et al., 2008; Seitz et al., 2013) and there is evidence that impulsivity symptoms of ADHD are positively associated with BN symptoms and with BED (Muller et al., 2014). Hyperactivity/impulsivity symptoms of ADHD were also consistently positively associated with LOC-eating, a core symptom of BED (Alfonsson et al., 2012; Reinblatt et al., 2015). Impulsivity has been suggested to positively influence overeating (Jasinska et al., 2012; Meule, 2013; Nederkoorn, Braet, et al., 2006; Nederkoorn, Smulders, et al., 2006), as it may increase the susceptibility that highly palatable food-cues attract attention and thereby trigger eating behaviour (Castellanos et al., 2009; Hou et al., 2011; Polivy, Herman, & Coelho, 2008). Therefore, impulsivity symptoms of ADHD may relate to binge eating, and the binge eating episodes of BN, via an enhanced susceptibility to food-cues.

Inattention symptoms of ADHD may also be associated with disordered eating and it has been suggested that compulsive eating may be a compensatory mechanism to help control frustration associated with attention and organization difficulties (Schweickert, Strober, & Moskowitz, 1997). Davis et al. (2006) proposed that patients with ADHD may be inattentive to internal signs of hunger and satiety. Thus, they may forget about eating when they are engaged in other activities and may be more likely to eat when less stimulated, at which point they may be very hungry. Eating in response to feelings of hunger and satiety has been found to be associated with BMI (Gast, Campbell Nielson, Hunt, & Leiker, 2015; Madden, Leong, Gray, & Horwath, 2012), suggesting that inattention to internal signs of hunger and satiety may cause overeating. However, the evidence to date concerning the association between inattention symptoms of ADHD and disordered eating is mixed. In this review, the studies that found significant positive associations were cross-sectional in nature. Therefore, reverse causality may also be possible, as nutritional deficiencies that are often common in EDs (Mitchell & Crow, 2006; Setnick, 2010) may impact on a patients' ability to focus attention, resulting in symptoms that mimic attention deficits in ADHD.

The role of hyperactivity symptoms of ADHD in disordered eating has generally been neglected. However, in this review, hyperactivity symptoms of ADHD were found to be significantly associated with restrictive eating behaviours, particularly in men (Grabarek & Cooper, 2008; Rastam et al., 2013; Slane et al., 2010). Excessive exercise is often characteristic of patients suffering from AN, especially in the acute phase of the disorder (Kohl, Foulon, & Guelfi, 2004). Therefore, the observed association between hyperactivity symptoms of ADHD and restrictive eating symptomatology may be mediated by (over)exercising behaviour.

4.1. Strengths and limitations of the systematic review

This systematic review has a number of strengths and some limitations. Eating behaviour was conceptualised as a continuum ranging from normal eating to eating disorders, and therefore a range of eating behaviour was included. ADHD was also conceptualised dimensionally, allowing a large number of studies to be systematically reviewed. Certain limitations require that the results of this review should be interpreted with some caution. Most studies published in this area were not specifically designed to explore an association between ADHD and disordered eating. In many cases this has resulted in suboptimal study designs, with the potential for biased results. Language and publication bias may also be relevant, as the search was limited to studies published in the English language. Finally, due to the heterogeneity of the studies, particularly with respect to methodologies, outcomes and populations, a meta-analysis was not feasible.

4.2. Clinical implications

EDs can significantly impact both the physical and the psychological health of individuals, and thus it may be advisable to assess the risk for disordered eating behaviour in the management of ADHD. Recent research has focussed on the association between ADHD and obesity (Cortese et al., 2015), but the current findings highlight the importance of considering the risk that individuals with ADHD may develop a range of disordered eating behaviour. Individuals with ADHD can present with normal weight, but suffer from eating pathologies (Fairburn & Cooper, 1982). Furthermore, hyperactivity symptoms of ADHD in children may be protective against obesity, leading to an underestimation of problematic eating behaviours that could co-exist with ADHD. However, as symptoms of ADHD manifest differently with age, and symptoms of hyperactivity typically decrease and become more subtle (Holbrook

et al., 2016; Todd et al., 2008) the continuation of problematic eating behaviour can result in the development of overweight/obesity later in adolescent/adult life due to an age-related decrease in energy expenditure. Therefore, weight management issues need to be taken into account when considering medication for ADHD. Recently, lisdexamfetamine, a central nervous system stimulant used for the treatment of ADHD, has been approved for the treatment of moderate to severe BED (US Food and Drug Administration, 2015). It is unclear if this medication would be effective for individuals with ADHD who exhibit restrictive eating, and assessment of eating behaviours would be an important factor to consider when making treatment decisions. Ultimately, a better understanding of the range of specific eating problems experienced by individuals with ADHD and their underlying mechanisms will facilitate more effective and personalised treatment.

4.3. Future research

Research on the association between ADHD and disordered eating is in its infancy and some methodological limitations of previous studies could be addressed in future research. In this review, assessment of eating pathologies varied substantially across studies, even among studies of individual EDs, making comparisons across samples difficult. Studies used DSM criteria, symptom counts, ED questionnaires, or single questions about eating behaviours, while few studies assessed eating behaviour in a laboratory setting. Use of uniform and robust methods of assessing disordered eating would be preferable in future studies. As most studies in this review included a population comprised mainly or only of females, future studies would benefit from adequate representation of both sexes in samples. This would enable a better understanding of the role of sex in the association between ADHD and EDs and/or eating pathology/disordered eating.

Confounding factors such as health characteristics, stimulant medication and ADHD-related comorbidities should be included in the models in addition to the confounds already identified in previous research (e.g., age, sex, socioeconomic status). Drug therapies for ADHD such as methylphenidate can have a pronounced effect on appetite (Findling et al., 2008), while lisdexamfetamine is clinically effective in treating both ADHD and BED (Brownley et al., 2016). To disentangle the effect of ADHD from any effect of stimulant medication on disordered eating, future studies should control for factors such as type of stimulant medication, length of stimulant treatment and medication dosage.

Further research is also required to clarify the nature of the mechanisms underlying the association between ADHD and disordered eating. An emerging question is whether the relationship between ADHD and disordered eating is influenced by conditions that often co-exist with EDs and eating pathologies, including anxiety and depression or whether core features of ADHD may lead to pathological eating behaviour. Given the extensive body of evidence that suggests an association between impulsivity symptoms of ADHD and BN symptoms, future studies should address the role of core processes such as impulsivity in disordered eating. As impulsivity involves at least two identifiable cognitive/emotional processes, reward–driven behaviours and poor inhibition (Solanto et al., 2001), future research should address whether food-cue induced overeating may be the result of overwhelming, reward-related, bottom-up processes or deficient inhibitory, top-down control mechanisms, or both (Appelhans, 2009; Heatherton & Wagner, 2011; Price, Higgs, Maw, & Lee, 2016). Further studies are also needed to explore whether inattention and hyperactivity symptoms may contribute to disordered eating and the underlying processes of any relationship.

We propose a research framework to guide future studies on ADHD and disordered eating based on the National Institute of Mental Health Research Domain Criteria Initiative (RDoC), which encourages research on dimensions of observable behaviour and neurobiology rather than a categorical, symptom-based approach to the study of mental health (Insel et al., 2010). Our proposed research framework comprises multi-modal, laboratory-based assessment of cognitive constructs across different RDoC systems. Studying the relationship between individual variations in specific RDoC cognitive constructs of attention and cognitive control and disordered eating in ADHD provides an opportunity to identify core processes that cut across diagnostic categories and could be targeted by therapeutic interventions. We propose recruitment of participants from the community to span the range of variation in cognitive processes associated with ADHD. This dimensional approach ensures that potential confounds associated with clinical research (e.g., medication status) can be minimised. Participants would complete behavioral tasks that assess specific cognitive constructs including cognitive control and attention as well as questionnaire items assessing ADHD symptoms. In addition, functional brain imaging could provide a link from behavioral and self-report measures to neural units of analysis. Disordered eating behaviour would be assessed by both self-report measures and laboratory based assessment of loss of control over eating. Tanofsky-Kraff et al. (2013) have proposed a similar approach to the study of disinhibited eating in obese adolescents but an RDoC compatible approach has yet to be adopted widely in the study of disordered eating. While high quality longitudinal studies are the gold standard for understanding the temporal relationship between ADHD and disordered eating behaviour, such designs are often impractical and unfundable. Our proposed framework enables testing for causal relationships between cognitive constructs and disordered eating because processes such as attention and cognitive control can be manipulated and the effects on laboratory measures of eating (such as measures of eating rate captured using a Universal Eating Monitor) assessed. This framework could also be used to study disordered eating in other mental health conditions associated with disordered eating such as schizophrenia and depression and may be extended from cognitive constructs to other RDoC domains and their interaction. For example, it is likely that cognitive control systems and positive valance systems interact to influence food responsiveness (Higgs, 2016; Wildes & Marcus, 2015).

5. Conclusions

There is a consistent moderate strength of evidence that ADHD is positively associated with disordered eating and with specific types of disordered-eating behaviour, in particular, overeating behaviour. In addition, there is evidence that impulsivity is positively associated with BN symptoms. There is also more limited evidence to suggest an association between hyperactivity symptoms and restrictive eating in males but not females, although this requires further investigation. Increased awareness of this phenomenon could enhance clinical management and therapeutic options for individuals with ADHD. Greater understanding of the relationship between core cognitive constructs and disordered eating behaviour potentially using the RDoC framework will be useful for informing therapeutic options for individuals with ADHD and other mental health conditions that are associated with eating disorders.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.cpr.2017.03.002.

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