Parental bonding and eating disorders: A systematic review

Authors: Dr Amanda Tetley*, Dr Nima G. Moghaddam, Dr David L. Dawson & Dr Mike Rennoldson

Trent Doctorate in Clinical Psychology, University of Nottingham and University of Lincoln

*Corresponding author: Dr Amanda Tetley, lwxacte@nottingham.ac.uk

Institute of Work, Health and Organisations, Yang Fuji Building, Jubilee Campus, Wollaton Road, Nottingham, NG8 1BB

RUNNING HEAD: PARENTAL BONDING AND EATING DISORDERS

Abstract

Objective: This article systematically reviews studies of parental bonding in people with eating disorders. **Method:** MEDLINE, PsychINFO, EMBASE and CINAHL were searched to identify studies that compared parental bonding in people diagnosed with an eating disorder relative to non-clinical controls. **Results:** Twenty-four studies were identified. Women with eating disorders typically reported lower parental care and higher parental protection compared to non-clinical, but not psychiatric, controls. Interestingly, these relationships were mediated by avoidant problem solving style and several schemas from the Young Schema Questionnaire (YSQ[1]). **Discussion:** While there are methodological limitations associated with the reviewed studies, they do offer some support for the proposal that difficulties in parent-child relationships predispose women to eating disorders and other psychiatric diagnoses.

Parental bonding and eating disorders: A systematic review

There are a number of theoretical accounts of the development of eating disorders. The most influential of these for psychological therapy arise from the cognitive behavioural tradition[2]. This theoretical approach assumes that dysfunctional beliefs underlie psychological distress and most crucially that these beliefs arise from negative early life experiences[3]. Consistent with this theme, a recent cognitive model of eating disorders suggests that dysfunctional self-loathing beliefs are key to the development of eating disorders and arise from negative childhood experiences, such as parental neglect or indifference[4].

Cognitive behavioural theories are not the only theoretical contributions suggesting that difficult relationships between parents and children could be implicated in the onset of eating disorders. Using attachment theory[5] as an explanatory framework, it has been suggested that insecure attachments to caregivers are common in those with eating disorders. The symptoms of their eating disorder are assumed to represent an attempt to maintain physical and psychological proximity to a caregiver[6,7]. Likewise, psychodynamic theories suggest that parents of those who develop eating disorders are demanding/over-controlling and emotionally unresponsive. This is assumed to result in either; 1) a need for the adolescent to remain child-like to avoid abandonment, 2) a refusal to eat to subdue the internalised controlling parent, or 3) weight-control behaviours to maintain closeness to the parent [8,9].

It is important to assess the empirical support for the hypothesised link between early parent-child relationships and eating disorders. To date, four systematic literature reviews have sought to synthesise data on this empirical question[6,10,7,11]. Before considering their conclusions, it is important to note that they conceptualise existing empirical studies as either considering the "attachment construct" as defined by Bowlby[5,12], or considering "parental bonding" as defined by Parker and colleagues[12].

Briefly, the "attachment construct" referred to in these reviews is defined by Bowlby's attachment theory[5]. This suggests that children respond to caregiver's behaviour in ways that most effectively achieve care and security. If attachment figures are experienced as unresponsive, frightening, or neglectful, children are assumed to develop one of three insecure attachment styles that continue across the lifespan, namely avoidant, preoccupied, or disorganised. *Avoidant attachment* is associated with withdrawal and an avoidance of emotional intimacy; *preoccupied attachment* is associated with attempts to avoid rejection and extreme distress on separation from others; *and finally, disorganised attachment* is characterised by a combination of seeking care and avoiding it and dissociating from the environment in the face of this dilemma.

Parental bonding, in contrast to the attachment construct, has been defined by Parker and colleagues[12] as the parental contribution to parent-child relationships and is typically assessed using the Parental Bonding Instrument (PBI)[12]. Parker and colleagues [12] define maternal and paternal contribution to bonding along two dimensions, namely care and protection. The dimension of care ranges from affection, emotional warmth, empathy, and closeness, to emotional coldness, indifference and neglect. The dimension of overprotection/control ranges from

control, overprotection, intrusion, infantilisation, and prevention of independent behaviour, to allowance of independence and autonomy. Parker and colleagues[12] suggest that this parental contribution to the parent-child bonding is an area that is neglected, or at best only briefly considered in attachment theory[12]. This indeed appears to be the case given that consideration of parental behaviour in attachment theory revolves around the emotional responsiveness to the child and fails to consider the effects of parental protection/control.

Two of the reviews addressing parent-child relationships in people with eating disorders have included studies assessing both Bowlby's attachment construct and parental bonding as defined by Parker and colleagues[12]. The initial review concluded that, compared to non-clinical controls, those with eating disorders remember both parents as less caring, but only their father as more protective – with this latter finding more common in women with Bulimia Nervosa (BN)[6]. By contrast, an updated review found that clients with eating disorders consistently remember their parents as more controlling and less affectionate than their non-clinical counterparts (so called affectionless control)[11]. Both reviews also find that those with eating disorders encounter separation anxiety and are more likely than controls to be insecurely attached. The most recent of these reviews also finds that women with Anorexia Nervosa (AN) tend to have an avoidant attachment style while women with BN tend to be preoccupied in their attachment style[11]. This latter finding has also been supported in more recent reviews of attachment in those with eating disorders[7,10].

In the most recent reviews in this area,[7,10] the authors have chosen to focus solely on studies assessing the Bowlbian attachment construct, thereby excluding studies assessing parental bonding. The present article will therefore review

empirical studies assessing parental bonding in those with eating disorders, updating the previous review (which was conducted in 1999[11]). An updated review is crucial for three reasons. First, only 11 studies were reviewed up to 1999 and many more have been undertaken since. Second, while the authors of the review noted a predominance of "affectionless control" parenting in people with eating disorders, careful inspection of the reviewed studies reveals contradictory findings. Third, both previous reviews in this area[6,11] highlighted limitations of the studies they reviewed. In particular, the studies did not incorporate a psychiatric control group, failed to select healthy controls in such a way to limit the confounding aspects of disordered eating behaviours, and did not consider how parental bonding might result in the manifestation of eating disorders.

This review aims to update these previous reviews. The primary aims were to: (1) identify the extent to which parental bonding, as defined by Parker and colleagues[12], is found to differ for people with eating disorders relative to non-clinical samples in studies published since 1999; and (2) assess the methodological quality of this research and identify what further research is required. The secondary aim was to identify any mediators of the relationship between parental bonding and eating disorders. This will serve to extend our understanding of the potential developmental pathways of disordered eating behaviours.

Method

Searching

Medline, EMBASE, PsycINFO and CINAHL databases were searched to identify relevant English-language journal articles published between 1999 and June 2012. Reference lists of all full-text articles included in the review were also searched.

Electronic searches were based on both medical subject heading (MeSH) terms and textwords. The concepts included in the search strategies were “eating disorders” and “parent-child relationships” (see Appendix A for search terms for PsycINFO). “Attachment” was not included as a term in the search strategy because it reduced the specificity of searches and did not appear to identify any relevant articles not identified using other search terms for parent-child relationships.

Inclusion and exclusion criteria

Types of studies

English-language peer-reviewed articles were included in this review if they assessed parental bonding in people with eating disorders and compared this to bonding in non-clinical participants. The types of studies relevant for inclusion were cross-sectional, case-control, longitudinal, or comparative twin studies. The review was restricted to English-language peer-reviewed articles for practical reasons.

Types of participants

Studies were included if they recruited participants who have been diagnosed with an eating disorder at some time in their life (AN, BN, Binge Eating Disorder [BED] or Eating Disorder Not Otherwise Specified [EDNOS]) using criteria outlined in DSM or ICD. Alternatively, they may have been recruited from a specialist eating

disorder service/organisation because these individuals will most likely have received an eating disorder diagnosis. Studies could include male or female participants of any age (child or adult).

Studies were excluded if they simply reported on the association between parental bonding and measures of subsequent eating difficulties in non-clinical samples. This ensured that the review focused on a consistently defined population of those with eating disorders.

Measurement of parental bonding

Studies were included if they assessed parental bonding as defined by Parker and colleagues[12] for the period of childhood up to age 16 or time of enrolment into the study (if before age 16). Parental bonding as defined by Parker and colleagues[12] is best operationalised in the PBI because it was designed explicitly to map onto this construct. However, there are other assessment tools that assess the overlapping constructs of PBI-care and protection. In this review, we included studies that employed the PBI or a tool assessing constructs similar to PBI-care and protection. Where a tool appeared on first observation to be measuring parental bonding, the items were carefully inspected to determine their correspondence with the constructs of care and protection as defined by Parker and colleagues[12]. The assessment of care must reflect to some extent the PBI dimension of care ranging from affection, emotional warmth, empathy, and closeness, to emotional coldness, indifference and neglect. The assessment of parental overprotection/control must reflect to some extent a dimension ranging from control, overprotection, intrusion, infantilisation, and prevention of independent behaviour, to allowance of independence and autonomy.

Articles were excluded if they assessed (1) only a narrow element of the constructs of “care” or “overprotection/control”, for example only assessing parental invasion of privacy, (2) the constructs of “care” and/or “overprotection/control” as part of a broader measure and failed to separately report the analyses for these constructs, or (3) parental bonding for only a short period of childhood. The reason for this latter exclusion is that accounts of parental bonding at one moment in time might not reflect bonding over the entire period of childhood. Thus, such assessments may fail to present robust tests of hypotheses linking childhood parental bonding with eating disorders.

Screening and data extraction

The title and abstracts of all citations identified by the searches were read by one reviewer to identify those that clearly did not meet inclusion criteria. The full-text articles of all remaining citations were obtained and screened for inclusion by the same reviewer. Two independent reviewers extracted the relevant information from papers included in the review. A standard data collection proforma was used to extract the following information: study authors and year of publication, study design, participant details (including demographic information and definition of eating disorder), measure of parental bonding including who completed it (e.g., participant, parent), and study findings. Where possible, effect sizes (*r* values) were calculated for differences in parental bonding across study groups by extracting the relevant test statistics from the article.

Assessment of susceptibility to bias

Given that there is no accepted “gold standard” tool to assess the susceptibility to bias of empirical studies[13], a bespoke assessment tool was developed for this review. The tool was designed on the basis of published guidance[13] in this area. This guidance suggests that tools should be based upon the recent recommendations for reporting on observational studies (STROBE[14]). Two relevant checklists were identified as STROBE-compliant[13] and these formed the basis for the bespoke tool designed for the current review, namely the Critical Appraisal Skills Programme (CASP) for observational studies[15] and the Guidelines and Checklist for appraising a medical article[16].

The bespoke tool assessed five elements: (1) design issues, including validity and reliability of the measure of parental bonding and introduction of bias due to missing data, (2) sample representativeness, including the representativeness and appropriateness of the clinical and comparison groups and appropriate sample selection, (3) confounding factors, including matching of clinical and non-clinical groups and controls for effects of comorbidity, (4) suitability of statistical methods, and (5) conflicts of interest. For each of these areas, a decision was made as to the presence or absence of bias according to specific criteria (see Table 1). These criteria were in the most part based on the guidance outlined in the CASP and Guidelines and Checklist for appraising a medical article[15,16]. Novel criteria were devised for areas specific to the current review and justifications for these are detailed in Table 1.

An overall judgement of susceptibility to bias was made (low, medium, high). A numerical scoring of bias was not adopted because this involves weighting of individual components and the accuracy of such scoring procedures is unclear[13].

Results

Overview

The findings in relation to each of the aims specified in the introduction (above) will be considered in turn. Before this, the characteristics of identified studies will be described. Studies will be referenced throughout by numbers in parentheses which relate to the study numbers in Table 2.

Characteristics of identified studies

Twenty-four studies were included in this review (see Figure 1 for the outcomes of article screening). The studies included are presented in Table 2. Included studies were conducted in the UK (n=7), Australia (n=3), US (n=3), Israel (n=2), Spain (n=2), Italy (n=2), Poland (n=1), Canada (n=1), Japan (n=1) New Zealand (n=1), and Portugal (n=1). The studies adopted cross-sectional designs (n=16), case-control designs (n=5), and monozygotic twin-pair designs (n=3). The number of participants ranged from 18 to 622; all participants were females and mean ages (where reported) ranged from 14.7to 40.3 years. Participants with eating disorders were diagnosed using DSM-IV (n=14), DSM-IV-TR (n=1), DSM-III/DSM-III-R (n=3), or using other criteria (e.g., membership of the Eating Disorder Association). Parental bonding was assessed in the studies using the PBI[12] (n=17), the EMBU (memories of parental rearing)[43] (n=4), the Parental Attitude Scale (PAS[26]) (n=1), the Young

Parenting Inventory-Revised[44] (n=1), and the Childhood Experience of Care and Abuse Interview (CECA[45]) (n=1) (see Table 3 for details of these measures).

1. Methodological quality of included studies

Results of the susceptibility to bias analysis are presented in Table 4. The findings are considered for each area of bias in turn.

Design bias:

A relatively small number of studies had missing data (17%) or used a potentially unreliable assessment of parental bonding (13%).

Sample representativeness:

The eating-disordered samples and comparison psychiatric samples were typically considered representative of these populations. In fact, the eating disorder group was judged to be highly representative in 33% of studies due to selection from the general population. While the non-clinical groups were typically assessed for eating disorders (67%), in 33% of studies this was not made explicit. In almost half of the studies (46%) it was impossible to determine if the sample selection minimised susceptibility to bias.

Confounding factors:

Only 8% of studies controlled for psychiatric comorbidity. Likewise, clinical and non-clinical groups were only matched on important demographic characteristics (e.g., age, education, socioeconomic status) in 25% of the studies. In 17% of

studies, it was clear that the groups were not demographically similar, and in a further 25% there was no explicit statement about the similarity of groups.

Statistical analysis:

None of the studies reported whether the analysis was adequately powered, 58% failed to control adquetely for Type I errors, and 42% failed to state whether the data were appropriate for parametric analyses.

Overall susceptibility to bias assessment:

Seventeen percent of studies were judged to have a high susceptibility to bias, 58% to have a medium susceptibility, and 25% to have a low susceptibility. Those with a high susceptibility to bias employed a potentially unreliable measure of parental bonding (25%), recruited an inappropriate non-clinical comparison group (50%) or suffered both these limitations (25%). Those with a low susceptibility to bias typically employed a suitable measure of parental bonding, sampled the population with moderate representativeness, and studied groups that were considered to be sufficiently similar with regards to their demographic characteristics.

2. Parental bonding in women with eating disorders relative to comparison groups

In addition to comparing parental bonding in women with eating disorders relative to non-clinical controls, a number of studies also compared against women with other psychiatric diagnoses, and compared parental bonding across different eating disorder diagnostic groups (i.e., AN versus BN). Each of these comparisons will be considered separately.

Comparison 1: Non-clinical samples

Included studies compared levels of maternal care and overprotection in non-clinical samples to women with BN (n=8), women with AN (n=13), women with BED (n=1), and women characterised more generally as having an eating disorder diagnosis (n = 4). All these studies also compared levels of paternal care in these groups, with an additional two studies comparing paternal care between groups categorised as having an eating disorder; one of these studies also assessed paternal protection between eating disorder groups.

Parental Care: A substantial proportion of studies reported lower maternal and paternal care in women diagnosed with an eating disorder. Specifically, lower maternal care was reported in 50% for comparisons involving women with BN (2,4,13,14), 46% of comparisons involving women with AN (5,6,10,11,13,17), 100% of comparisons involving women with BED (18), and 100% of women characterised more generally as having an eating disorder diagnosis (19,20,24). Similarly, lower paternal care was reported in 38% of comparisons involving women with BN (2,13,14), 53% of comparisons involving women with AN (5,6,7,10,11,13),100% of comparisons involving women with BED (18), and 100% of women characterised more generally as having an eating disorder (19,20,21,22,24). In addition to this, CECA-parental antipathy was lower in women diagnosed with both AN and BN (so-called mixed AN and BN) (15).

There are three additional notable findings of these studies. First, differences in paternal and maternal care across clinical and non-clinical groups reflected small effect sizes for women with BED (18), small to almost large effect sizes for women

with AN (5,6,7), and medium to large effect sizes for women categorised more generally as having an eating disorder diagnosis (24). Second, one study found that paternal care was no longer significantly lower in women with eating disorders relative to non-clinical samples after controlling for psychiatric comorbidity (24). Third, one study found that parental care was only significantly lower in chronically ill women with AN, but not in partially recovered or recovered women with AN (11).

Parental overprotection: A substantial proportion of studies reported higher maternal and paternal overprotection in women diagnosed with an eating disorder. Specifically, higher maternal protection was reported in 38% of comparisons involving women with BN (2,4,13), 38% of comparisons involving women with AN (10,12,13,17, 8),100% of comparisons involving women with BED (18), and 75% of comparisons involving women characterised more generally as having an eating disorder diagnosis (19, 23, 24). Similarly, higher paternal protection was reported in 25% of comparisons involving women with BN (4,13,2), 31% of comparisons involving women with AN (6,16,17, 8), 0% of comparisons involving women with BED (18), and 60% of comparisons involving women with an eating disorder diagnosis (19, 23,24). In addition to this, a comparison of parental control more generally suggests that it is higher in a group of women with both AN and BN (so-called mixed AN and BN) (15).

Importantly, where significant differences in paternal and maternal protection are reported, large effect sizes have been found for women with BN (4), small to medium effect sizes for women with AN (6), and medium effect sizes for women categorised more generally as having an eating disorder (24). Despite this, it is apparent from one study that differences in paternal and maternal overprotection are

no longer significant in women with BN after controlling for age, scores on the Beck Depression Inventory (BDI[3]), and BMI (2).

Studies with low susceptibility to methodological bias: The findings synthesised thus far suggest that the differences in parental care and protection are not consistently reported across studies. Therefore, it is interesting to synthesise findings from those studies that we judged to have a low susceptibility to methodological bias, separately. This suggests that only one of the six studies with a low susceptibility to bias failed to find evidence of lower parental care (16) in women with eating disorders relative to non-clinical controls. Likewise, only one of the studies failed to find evidence of lower parental protection (11).

Comparison 2: Psychiatric controls

Six studies compared levels of care and protection in women with eating disorders relative to psychiatric controls. In the most part, these comparisons consistently revealed little evidence of different levels of parental care or overprotection across these two groups (10, 16, 17 18). There were however two exceptions to this. One study found that women with AN recalled significantly lower CECA-Parental-Antipathy (15) and higher CECA-Parental-Control (15) than women with depression (15). Another study found that women with AN recall significantly higher paternal and maternal care compared to women with Borderline Personality Disorder (BPD), (12).

Comparison 3: AN versus BN

Seven studies compared levels of care and protection in women with AN and BN. These studies found that the two groups did not differ significantly in parental care (13,14,15,16,17), but two studies found evidence of significantly greater paternal protection in women with BN (13,15).

3. Factors that mediate the relationship between parental bonding and ED symptomatology

Five of the studies assessed mediators of the relationship between parental bonding and the severity of eating pathology (assessed by the Eating Disorder Inventory-II;EDI-2[55], Eating Disorder Examination; EDE[56,57] or Eating Disorder Inventory;EDI[59]). All studies employed the Baron and Kenny[17] method to assess mediation.

The findings provide little evidence to suggest that personality traits defined by the Temperament and Character Inventory (TCI[54]) mediate the relationship in women with BN (2). Likewise, beliefs relating to being defective (e.g., bad, inferior), being a failure, and beliefs that one must strive to meet high internalised standards (assessed by YSQ;[1]) were not found to mediate the relationship in women with AN (10). Finally, neither a tendency to avoid experienced affect (assessed by Young-Rygh Avoidance Inventory; YRAI[57]) or the use of compensation strategies to overcome negative core beliefs (assessed by Young Compensatory Inventory;YCI[58]) were found to mediate the relationship in women characterised broadly as having an eating disorder.

By contrast, one study found evidence to suggest that maternal care and protection significantly mediate the relationship between the tendency to avoid dealing with problems (assessed by the Social Problem Solving Inventory-Revised; SPSI-R[60] Avoidance style) and eating disorder symptomatology in women with AN (5). Likewise, another study found that in women with eating disorders the association between paternal rejection and drive-for-thinness and body-dissatisfaction are significantly mediated by beliefs that one is defective and that abandonment by others is likely (assessed by YSQ[1]). The same study found that the association between paternal protection and drive-for-thinness was significantly mediated by beliefs that one is vulnerable to an imminent catastrophe (assessed by YSQ)(22).

Discussion

The first aim of this review was to assess the extent to which studies find that parental bonding differs for people with eating disorders relative to non-clinical controls. Consistent with findings from the most recent review in this area[11], we find that a substantial proportion of studies report evidence of lower parental care and higher parental protection in women with eating disorders. We also find that parental bonding does not differ significantly in women diagnosed with eating disorders relative to women with other psychiatric diagnoses, nor does it typically differ significantly across eating disorder diagnostic categories. This latter finding is perhaps unsurprising given that there is much overlap between the diagnostic categories for eating disorders[2].

A second aim of this study was to assess the methodological quality of reviewed studies. Our findings suggest that some studies suffer from serious methodological limitations. For example, they fail to ensure that the non-clinical sample is free of eating disorder symptomatology or that it is demographically similar to the eating disorder group. Furthermore, some studies compare only small sample sizes and fail to comment on whether their statistical analysis is sufficiently powered. While these methodological limitations are somewhat widespread across studies, they were not characteristic of all studies. Indeed, some studies recruited samples representative of the population from which they were drawn and ensured that clinical and non-clinical groups were adequately matched on important demographic characteristics. Interestingly, it was these studies with a lower susceptibility to methodological bias that were more likely to report evidence of lower parental care and higher parental control in women with eating disorders relative to non-clinical controls. This was also the case in studies incorporating larger sample sizes, which most often included samples of women characterised more generally as having an eating disorder. As a result of their larger sample size, these studies were presumably more likely to be representative of women with eating disorders and to be sufficiently powered for analysis.

The secondary aim of this review was to identify any mediators of the relationship between parental bonding and eating disorder symptomatology in an attempt to understand the developmental pathway. The two previous reviews in this area[6,11] found no evidence of attempts to address this issue and interestingly our review suggests that it has only recently begun to receive empirical attention. Those studies that have begun to consider this issue have found evidence to suggest that deficiencies in maternal bonding could contribute to an avoidance of dealing with

social problems which contributes to eating disorder symptomatology. They also suggest that lower parental bonding generates unhelpful beliefs/schemas which in turn contribute to the onset of eating disorders. Specifically, the evidence is indicative of high paternal protection generating beliefs that one is vulnerable to harm and high parental rejection contributing to feeling internally flawed (i.e., defective) and to beliefs that close relationships will end imminently.

Before drawing conclusions from these findings, it is important to consider the general limitations of the research in this area. Most crucially, all studies typically rely on retrospective reporting of parental bonding over the period of childhood after the woman has developed an eating disorder. This is potentially problematic because it relies on the general assumption that people can accurately recall this, and that the onset of the eating disorder does not affect perception or experience of bonding. Indeed, these assumptions could be incorrect. For instance, there is a possibility that recall could be affected by experiences of current relationships. It is also possible that parental care and protection change after the onset of an eating disorder as parents feel the need to protect their daughter, for example. Another possibility is that the sufferer's perception of their parents' behaviour changes after onset perhaps because they blame their parents for the disorder.

We also find that there are no studies to date that consider parental bonding across childhood in men with eating disorders, limiting the applicability of findings to women. A further limitation concerns the way in which studies have considered evidence for mediating variables. These studies base their conclusions on mediation analyses incorporating only the eating disorder sample. Thus, by their nature, these comparisons are in fact only providing evidence of mediators of the association

between bonding and eating disorder symptom severity in those diagnosed with an eating disorder.

Notwithstanding these limitations, the findings offer some support to the hypothesis that problematic parental bonding could present a contributing factor to the onset of eating disorders. This is articulated in a variety of theories from different theoretical persuasions (e.g., attachment, psychodynamic, cognitive behavioural)[4,6-9]. However, it is also important to note that the findings suggest that difficulties in parental bonding are not limited to women with eating disorders per se but are also found in women with other psychiatric diagnoses. This suggests that it may represent a more general risk factor for psychological difficulties. Thus, following on from this it is crucial to explore the developmental pathway of eating disorders to identify what causes low parental bonding to result in eating disorders rather than other psychological difficulties.

Preliminary evidence reviewed here appears to indicate that unhelpful beliefs relating to defectiveness, low self-efficacy, abandonment and vulnerability to harm could contribute to eating disorder symptomatology and it is these which are generated to some extent by problematic parental bonding. This is in part consistent with cognitive behavioural theories which suggest that negative early relationships with parents result in dysfunctional beliefs about self/others and ultimately result in the development of an eating disorder[4]. However, these theories hypothesise that it is self-loathing and perfectionist beliefs that are responsible for the onset of eating disorders and result from early negative experiences with parents[4,61]. Yet our findings suggest that there is limited evidence to support the role of perfectionist beliefs as a mediating variable. While one study in our review could be considered to provide some support for the role of self-loathing beliefs/defectiveness beliefs as a

mediating variable, this study is limited by its focus on mediation in the eating disorder group (see above) and by its reliance on cross-sectional data.

If further research can elucidate the mechanisms by which parental bonding affects disordered eating, then consideration of this is likely to be important in therapeutic work with clients. It may require consideration in formulating a client's difficulties or be more integral to a therapeutic intervention. It may be that it is particularly relevant to interventions delivered to clients who continue to reside within the family unit. Consistent with this, recent advances in family therapy for young people with eating disorders encourage therapists to consider parent-child relationships and how they affect current family functioning[62]. It is particularly crucial to note that proponents of this approach recognise the importance of not blaming parents for their child's difficulties; rather, they advocate understanding the origins of the parents' attachment styles, how these impact on their relationships with their offspring, and how these may be addressed.

There are limitations associated with this review which require consideration. First, it focussed solely on parental bonding as defined by Parker and colleagues[12]. This focuses on broad concepts of parental care and protection and does not allow consideration of more specific aspects of parenting such as parental criticism and encouragement of perfectionist standards. It may be that these more specific components of parenting differentiate people with eating disorders from people with other psychological difficulties, suggesting that this should be addressed by future studies and reviews. Second, the review was restricted to comparisons of parental bonding in people diagnosed with eating disorders and thereby the conclusions are not generalisable to women who may not meet diagnostic criteria yet engage in behaviours characteristic of eating disorders. Fourth, the research to date excludes

conclusions about how cultural factors and gender moderate the effect of parental bonding on eating disorder symptomatology. Finally, this review only included published studies, perhaps reflecting a publication bias.

Taken together, the conclusions from this review highlight a number of recommendations for future research. First, studies would benefit from identifying variables that mediate the relationship between parental bonding and eating disorder symptomatology in a large sample of people with and without eating disorders. Second, they should address issues of parental bonding in men, as well as in women. Third, study designs should be optimal with researchers ensuring that non-clinical and clinical groups are adequately matched, that samples are sufficiently large to guarantee that statistical power is achieved, and that the use of longitudinal designs is considered. Finally, it may be fruitful to determine how specific aspects of parenting relate to development of eating disorders.

To conclude, the findings from this review suggest that women with eating disorders and other psychiatric diagnoses often report lower parental bonding relative to non-clinical controls. It is important for future studies to consider mediators of this relationship in women with eating disorders to elucidate the developmental pathways. In addition to this, it may be fruitful to consider how specific aspects of parental behaviour may be particularly pertinent to the development of eating disorders.

References

*Denotes included in review

1. Schmidt NB, Joiner TE, Young JE, Telch MJ. The schema questionnaire - investigation of psychometric properties and the hierarchical structure of a measure of maladaptive schemas. Cognitive Ther Res. 1995;19(3):295-321.
2. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361(9355):407-16.
3. Beck A, Rush A, Shaw BF, Emery G. Cognitive therapy of depression. New York, NY: The Guildford Press; 1979.
4. Cooper MJ, Wells A, Todd G. A cognitive model of bulimia nervosa. Brit J Clin Psychol. 2004;43:1-16.
5. Bowlby J. Attachment and loss: Vol 1. Attachment. New York: Basic Books; 1969.
6. O'kearney R. Attachment disruption in anorexia nervosa and bulimia nervosa: A review of theory and empirical research. Int J Eat Disorders. 1996;20(2):115-27.
7. Zachrisson HD, Skarderud F. Feelings of Insecurity: Review of Attachment and Eating Disorders. Eur Eat Disord Rev. 2010;18(2):97-106.
8.Sugarman A, Kurash C. The body as a transitional object in bulimia. Int J Eat Disorder. 1982;1(4):57-67.
9. Swift WJ, Stern S. The psychodynamic diversity of anorexia-nervosa. Int J of Eat Disorder. 1982;2(1):17-35.
10. O'Shaughnessy R, Dallos R. Attachment Research and eating disorders: A review of the literature. Clinical Child Psychology and Psychiatry. 2009;14(4):559-74.
11. Ward A, Ramsay R, Treasure J. Attachment research in eating disorders. Brit J Med Psychol. 2000;73:35-51.

12. Parker G, Tupling H, Brown LB. Parental bonding instrument. Brit J Med Psychol. 1979;52(MAR):1-10.

13. Sanderson S, Tatt LD, Higgins JPT. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: a systematic review and annotated bibliography. Int J Epidemiol. 2007;36(3):666-76.

14. Von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP, Initiative S. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. J Clin Epidemiol. 2008;61(4):344-9.

15. Critical Appraisal Skills Programme NHS. Critical Appraisal Skills Programme. England, UK: National Health Service; 2003.

16. Fowkes FGR, Fulton PM. Critical-appraisal of published research - introductory guidelines. Brit Med J. 1991;302(6785):1136-40.

17. Baron RM, Kenny DA. The moderator mediator variable distinction in social psychological-research - conceptual, strategic, and statistical considerations. J Pers Soc Psychol. 1986;51(6):1173-82.

18. MacKinnon DP, Fairchild AJ. Current Directions in Mediation Analysis. Curr Dir in Psychol Sci. 2009;18(1):16-20.

19. Bonne O, Lahat S, Kfir R, Berry E, Katz M, Bachar E. Parent-Daughter Discrepancies in Perception of Family Function in Bulimia Nervosa. Psychiat. 2003;66(3):244-54. *

20. Fassino S, Amianto F, Rocca G, Daga GA. Parental bonding and eating psychopathology in bulimia nervosa: personality traits as possible mediators. Epidemiol Psichiat S. 2010;19(3):214-222

21. Bulik CM, Wade TD, Kendler KS. Characteristics of monozygotic twins discordant for bulimia nervosa. Int J Eat Disorder. 2001;29(1):1-10.

22. Wade TD, Treloar SA, Martin NG. A comparison of family functioning, temperament, and childhood conditions in monozygotic twin pairs discordant for lifetime bulimia nervosa. Am J Psychiat. 2001;158(7):1155-7.

23. Swanson H, Power K, Collin P, Deas S, Paterson G, Grierson D, et al. The relationship between parental bonding, social problem solving and eating pathology in an anorexic inpatient sample. Eur Eat Disorder Rev. 2010;18(1):22-32.

24. Canetti L, Kanyas K, Lerer B, Latzer Y, Bachar E. Anorexia nervosa and parental bonding: The contribution of parent-grandparent relationships to eating disorder psychopathology. J Clin Psychol. 2008;64(6):703-16.

25. Castro J, Toro J, Cruz M. Quality of rearing practices as predictor of short-term outcome in adolescent anorexia nervosa. Psychol Med. 2000;30(1):61-7.

26. Rostowska T, Rostowski J, Lewandowska-Walter A. Neuropsychological and family factors conditioning anorexia nervosa. Acta Neuropsychologica. 2009;7(4):264-82.

27. Wade TD, Treloar, SA, Martin NG, Statham D, Heath AC. . Monozygotic twin pairs discordant for lifetime anorexia nervosa: An exploratory investigation. Aust J Psy. 2004;56(2):127-32.

28.Deas S, Power K, Collin P, Yellowlees A, Grierson D. The relationship between disordered eating, perceived parenting, and perfectionistic schemas. Cognitive Ther Res. 2011;35(5):414-424. *

29. Bulik CM, Sullivan PF, Fear JL, Pickering A. Outcome of anorexia nervosa: eating attitudes, personality, and parental bonding. Int J Eat Disorder. 2000;28(2):139-47. *

30. Laporte L, Guttman H. Recollections of parental bonding among women with borderline personality disorder as compared with women with anorexia nervosa and a control group. Aust JPsych. 2007;59(3):132-9. *

31. Leung N, Thomas G, Waller G. The relationship between parental bonding and core beliefs in anorexic and bulimic women. Brit J Clin Psych. 2000;39(Pt 2):205-13. *

32. Tereno S, Soares I, Martins C, Celani M, Sampaio D. Attachment styles, memories of parental rearing and therapeutic bond: a study with eating disordered patients, their parents and therapists. Eur Eat Disorder Rev. 2008;16(1):49-58. *

33. Webster JJ, Palmer RL. The childhood and family background of women with clinical eating disorders: a comparison with women with major depression and women without psychiatric disorder. Psychol Med. 2000;30(1):53-60. *

34. Wade TD, Gillespie N, Martin NG. A comparison of early family life events amongst monozygotic twin women with lifetime anorexia nervosa, bulimia nervosa, or major depression. Int J Eat Disorder. 2007;40(8):679-86. *

35. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: three integrated case-control comparisons. Arch Gen Psychiatry. 1999;56(5):468-76. *

36. Striegel-Moore RH, Fairburn CG, Wilfley DE, Pike KM, Dohm FA, Kraemer HC. Toward an understanding of risk factors for binge-eating disorder in black and white women: A community-based case-control study. Psychol Med. 2005;35(6):907-17. *

37. De Panfilis C, Rabbaglio P, Rossi C, Zita G, Maggini C. Body image disturbance, parental bonding and alexithymia in patients with eating disorders. Psychopathology. 2003;36(5):239-46. *

38. Fujimori A, Wada Y, Yamashita T, Choi H, Nishizawa S, Yamamoto H, Fukui K. Parental bonding in patients with eating disorders and self-injurious behavior. Psychiat Clin Neuros. 2011;65(3):272-9. *

39. Gutzwiller J, Oliver JM, Katz BM. Eating dysfunctions in college women: the roles of depression and attachment to fathers. J Am Coll Health. 2003;52(1):27-32. *

40. Jones CJ, Leung N, Harris G. Father-daughter relationship and eating psychopathology: the mediating role of core beliefs. Brit J Clin Psychol. 2006;45(Part 3):319-30. *

41. Sheffield A, Waller G, Emanuelli F, Murray J, Meyer C. Do schema processes mediate links between parenting and eating pathology? Eur Eat Disorder Rev. 2009;17(4):290-300. *

42. Rojo-Moreno L, Livianos-Aldana L, Conesa-Burguet L, Cava G. Dysfunctional rearing in community and clinic based populations with eating problems: Prevalence and mediating role of psychiatric morbidity. Eur Eat Disorder Rev. 2006;14(1):32-42. *

43. Perris C, Jacobsson L, Lindstrom H, Knorring LV, Perris H. Development of a new inventory for assessing memories of parental rearing behavior. Acta Psychiat Scand. 1980;61(4):265-74.

44. Sheffield A, Waller G, Emanuelli F, Murray J, Meyer C. Links between parenting and core beliefs: Preliminary psychometric validation of the young parenting inventory. Cognitive Ther Res. 2005;29(6):787-802.

45. Bifulco A, Brown GW, Harris TO. Childhood experience of care and abuse (CECA) - a retrospective interview measure. J Child Psychol Psyc. 1994;35(8):1419-35.

46. Parker G. The parental bonding instrument - a decade of research. Soc Psych Psych Epid. 1990;25(6):281-2.

47. Wilhelm K, Niven H, Parker G, Hadzi-Pavlovic D. The stability of the Parental Bonding Instrument over a 20-year period. Psy Med. 2005;35(3):387-93.

48. Pedersen W. Parental relations, mental-health, and delinquency in adolescents. Adolescence. 1994;29(116):975-90.

49. Arrindell WA, Emmelkamp PMG, Brilman E, Monsma A. Psychometric evaluation of an inventory for assessment of parental rearing practices - a dutch form of the embu. Acta Psychiat Scand. 1983;67(3):163-77.

50. Gerlsma C, Das J, Emmelkamp PMG. Depressed-patients parental representations - stability across changes in depressed mood and specificity across diagnoses. J Affect Disorders. 1993;27(3):173-81.

51. Arrindell WA, Gerlsma C, Vandereycken W, Hageman W, Daeseleire T. Convergent validity of the dimensions underlying the parental bonding instrument (PBI) and the EMBU. Pers Indiv Differ. 1998;24(3):341-50.

52. Garner DM, Garfinkel PE. Eating attitudes test - index of the symptoms of anorexia-nervosa. Psy Med. 1979;9(2):273-9.

53. Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. The eating attitudes test - psychometric features and clinical correlates. Psy Med. 1982;12(4):871-8.

54. Cloninger CR, Svrakic DM, Przybeck TR. A psychobiological model of temperament and character. Arch Gen Psychiatry. 1993;50(12):975-90.

55. Garner DM. Eating Disorder Inventory 2: Professional Manual. Odessa, FL: Psychological Assessment Resources; 1993.

56. Fairburn CG, Cooper Z. The Eating Disorders Examination. In: Fairburn CG, Wilson GT, editors. Binge eating:Nature, assessment and treatment. (12th ed). New York, NY: Guildford Press; 1993.57. Young J, E, Rygh J. Young-Rygh Avoidance Inventory (YRAI). New York, NY: Cognitive Therapy Centre; 1994.

58. Young JE. Young Compensatory Inventory (YCI). New York, NY: Cognitive Therapy Centre; 1995.

59. Garner DM, Olmsted MP, Polivy J. Eating disorder inventory. Psychopharmacol Bull. 1985;21(4):1009-10.

60. D'zurilla TJ, Maydeuolivares A. Conceptual and methodological issues in social problem-solving assessment. Behav Ther. 1995;26(3):409-32.

61. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behav Res Ther. 2003;41(5):509-28.

62. Dallos R. Attachment narrative therapy: integrating ideas from narrative and attachment theory in systemic family therapy with eating disorders. J Fam Ther. 2004;26(1):40-65.

Appendix

Search strategies

PsycINFO database search terms*

1. (exp Eating Disorders/ OR exp Binge Eating/ OR eating disorder$ OR eating pathology OR eating psychopathology OR disordered eat$ OR anorexi$ OR anorectic OR bulimi$ OR hyperphagia OR binge eat$ OR Eating Disorder not Otherwise Specified OR EDNOS) AND (Parent Child Relations/ OR parental bond$ OR parental rearing OR parent child relation$ OR father daughter relation$ OR mother daughter relation$ OR father son relation$ OR mother son relation$ OR father child relation$ OR mother child relation$ OR maternal rearing OR paternal rearing OR paternal bond$ OR maternal bond$ OR maternal relation$ OR parental relation$ OR paternal relation$ OR mother child interaction$ OR father child interaction$ OR parent child interaction$ OR father son interaction$ OR father daughter interaction$ OR mother son interaction$ OR mother daughter interaction$. OR child rearing)

*Equivalent search strategies were developed for EMBASE, MEDLINE and CINAHL databases.

Tables

Table 1. Susceptibility to bias assessment: Areas of bias

Area of bias	***Details of assignment categories***
Design bias	
Does the measure of parental bonding represent a valid/reliable assessment?	***Yes***=Evidence that the measure administered is valid and reliable ***No***=Measure not psychometrically sound OR administered in such a way that introduces bias (e.g., interviewer not blind to whether participant belongs to clinical or comparison group)
Is there any missing data that might introduce bias?	***Yes***=Data for a specific measure not reported for entire study sample, OR data missing for some participants on specific measures ***No***=No data missing
Sample representativeness	
How representative are the Eating Disorder Group?[a]	***Highly***=Recruited from the general population ***Sufficiently***=Recruited from treatment-seeking populations ***Unlikely=***Exclusion/inclusion are likely to deem group unrepresentative

Area of bias	***Details of assignment categories***
Is the non-clinical control group representative of the population without a diagnosed eating disorder? [b]	***Yes***=Evidence to confirm that members of the non-clinical group do not meet diagnostic criteria for an eating disorder ***No***=Evidence that members of the non-clinical group meet diagnostic criteria ***Unclear***=No information available on whether the eating behaviours of the non-clinical group have been assessed for diagnostic criteria
If a non-eating disordered clinical group are included, are they representative of this clinical population? [b]	***Highly***=Recruited from the general population or treatment-seeking population, meeting diagnostic criteria for a psychiatric condition but clearly not meeting diagnostic criteria for an eating disorder ***No***=No evidence to suggest that member of group meet diagnostic criteria for a psychiatric disorder OR evidence they meet diagnostic criteria for an eating disorder
Is the selection method for study participants appropriate?	***Highly***=Random selection OR based on a random criteria (such as every third person attending a clinic or all admissions to a clinic) ***Sufficiently*** =Self-selected randomly ***No***=Non-random selection ***Unclear***=No information provided

Area of bias	***Details of assignment categories***
Confounding factors	
Is matching of the groups suitable?	***Highly***=Groups matched on important demographic characteristics (e.g., age, education) ***Sufficiently=***Groups do not differ on important demographic characteristics OR where they do these factors have been controlled for statistically in the analysis ***No***=Evidence that groups differ significantly on one or more of these characteristics ***Unclear***=No explicit consideration of the similarity of groups on all important demographic characteristics
Has psychiatric comorbidity been accounted for? [c]	***Yes***=Controlled for symptoms of anxiety and/or depression ***No***=No reported controlling for this
Statistical analysis	
Is the analysis adequately powered?	***Yes***=Explicit comment by authors to state that study achieved at least 80% statistical power ***No***=Explicit comment by authors to state that study was underpowered ***Unclear***=No explicit comment to state whether the study achieved 80% statistical power

Area of bias	***Details of assignment categories***
Have Type I errors been controlled for?	***Yes*** = Appropriate adjustment for Type I errors ***No*** = Type I errors were not controlled
Are parametric/non-parametric tests used appropriately?	***Yes*** = Consider suitability of parametric/non-parametric tests and justify choice accordingly ***No*** = Use parametric tests without explicitly stating that data meets assumptions for these tests
Are tests of mediation appropriate? [d]	***Yes***=Use the Baron & Kenny (1986) method ***No***=Do not use Baron & Kenny (1986) method
Conflict of interest	
Is there a conflict of interest?	***Yes*** = Reported conflict of interest or likely conflict given the funding source ***No*** = Clear explicit comment that there is no conflict of interest ***Unclear*** = No comment on funding source OR not comment on conflict of interest

Note: [a] Studies recruiting from the general population are considered to be "highly" representative because it is known that treatment-seeking populations are not typically representative of the population of people with eating disorders[2]; [b] It is important that control groups (clinical and psychiatric) do reach criteria of a diagnosis of eating disorder because this would suggest that they are not appropriate controls; [c] Controlling for comorbid anxiety and depression ensures that differences between groups with an eating disorder diagnosis and non-clinical controls are

not in fact a result of the fact that the eating disorder group suffer from anxiety and depression; [d] The Baron and Kenny[17] method of testing statistical mediation is the commonly accepted approach[18].

Table 2. Included studies characteristics and findings

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
1 Medium	19	Israel	32 (100%)	CS	**BN(16)** 23 (2) **C (16)** 23(2)	DSM-IV	PBI (P,M,F)	***MC/MP/PP:*** NS for P,M,F ***PC:*** BN<C (*r* = .39) for P; NS for M,F
2 Low	20	Italy	308 (100%)	CS	**BN (purging type) (154)** 32.7 (10.4) **C (154)** 24.4 (3.6)	DSM-IV-TR	PBI (P)	***MC/PC:*** BN<C [a] ***MP/PP:*** BN>C [b] *No evidence that TCI scales mediate associations between PB & EDI-II Drive-for-thinness,-Bulimia or -Body-dissatisfaction in BN group*
3 Medium	21	US	40 100%	MZ twin	**BN (20)** 35.4 **C (20)** [c] 35.4	DSM-III-R	PBI (P, twin)	***MC/MP/PC/PP:*** NS

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
4 Medium	22	Australia	18 100%	MZ twin	**BN (9)** 40.3 (5.8) **C (9)** 40.3 (5.8)	No information [d]	PBI (P)	***MC***: BN<C (*r* =.69) ***MP*:** BN>C (*r* =.58) ***PC/PP*:** NS
5 Medium	23	UK	119 100%	CS	**AN (43)** 24.7 (6.8) **C (76)** 20.5 (5.1)	DSM-IV	PBI-S (P)	***MC/PC***: AN<C (both *r* = .22) ***PP/MP***: NS *MC & MP separately mediated the relationship between SPSR-I-Avoidance style and Total-EDE in AN group*
6 Medium	24	Israel	76 100%	CS	**AN (43)** 21.3 (3.7) **C (33)** 22.3 (4.5)	DSM-IV	PBI (P)	***MC/PC*:** AN<C (MC *r* =.32; PC *r* =.44) ***PP*:** AN>C (*r* =.22) ***MP*:** NS

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
7 Medium	25	Spain	317 100%	CS	**AN (158)** 14.9 (1.4) **C (159)** 14.7 (1.3)	DSM-IV	EMBU (P)	***MR/PR/MO/PO/MW*:** NS ***PW*:** AN>C (*r* =.14)
8 High	26	Poland	50 100%	CS	**AN (20)** 60% aged 15-17 25% aged 12-14 15% aged 18-19 **C (30)** 40% aged 15-17 50% aged 12-14 10% aged 18-19	No information [e]	PAS (P)	***MA/PA*:** AN<C (PA *r* =.33) [f]

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
9 High	27	Australia	18 100%	MZ twin	**AN (9)** 32.6 (2.6) **C (9)** 32.6 (2.6)	DSM-III-R	PBI (P)	***MC/MP/PC/PP*:** NS
10 Medium	28	UK	162 100%	CS	**AN (40)** 29 (10.3) **Dep/Anx (DA) (44)** 37 (12.1) **Control (78)** 20 (5.1)	DSM-IV	PBI-S (P)	***MC/PC:*** AN<C; AN=DA; DA=C ***MP:*** AN>C; AN=DA; DA=C ***PP:*** NS *YSQ-defectiveness, -Unrelenting standards, & -Failure not found to mediate relationship between PBI & EDE-total in AN group*

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
11 Low	29	New Zealand	168 100%	CC [g]	**AN:recovered (AN-r)(21)** **AN:partial recovery (AN-pr) (34)** **AN:chronically ill (AN-ci) (15)** **C (98)**	DSM-III / DSM-III-R	PBI (P)	***MC*:** AN-ci<AN-pr=AN-r=C ***PC*:** AN-ci<AN-r=C; AN-pr=all other groups ***MP/PP*:** NS
12 Medium	30	Canada	102 (100%)	CS	**AN (34) (Restricting)** 23.5 (7) **C (33)** 23.4 (7) **Borderline Personality Disorder (BPD) (35)** 31.7 (6)	DSM-III-R	PBI (P) [h]	***MC/PC*:** BPD<AN=C ***Maternal denial of freedom:*** AN=BPD>C ***Paternal denial of freedom*:** BPD>AN=C ***Maternal/Paternal denial of autonomy:*** BPD>C=AN

Study No. (Bias judgement)	Ref	Country	Total N (% female)	Study design	Diagnostic group Mean age in years (SD)	Diagnostic tool	PB measure (Raters)	Comparison of PB across groups *Evidence of mediation*
13 Medium	31	UK	80 100%	CS	**AN (30)** Restrictive 26.1 (7.8) Bulimic 22.6 (3.5) **BN (27)** 25.6 (5.1) **C (23)** 26.4 (4.7)	DSM-IV	PBI (P)	***MC/PC:*** AN=BN<C ***MP***: BN=AN>C ***PP***: BN>AN=C
14 Medium	32	Portugal	92 100%	CS	**AN (30)** 19.3 (3.4) **BN (27)** 21.6 (3.6) **C (35)** 19.0 (3.0)	DSM-IV	EMBU	***MR/PR****:* AN=BN; AN=C; BN>C ***MW/PW/MP/PP:*** NS

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
15 Medium	33	UK	160 100%	CS	**AN (28)** 29 no SD reported **BN (32)** 30 **Mixed (Diagnosis of AN+BN) (20)** 30 **Depressed (40)** 34 **C (40)** 34	DSM-III-R	CECA	***Proportion marked/moderate on P-antipathy:*** Mixed>C; BN=AN=C; Depressed>AN, Depressed =Mixed, BN=Depressed; ***Proportion marked/ moderate on PC:*** Mixed, BN & AN=C; Mixed & BN=Depressed; Depressed>AN; BN>AN

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
16 Low	34	Australia	622 100%	CC [i]	**AN (23)** **BN (20)** **MD (186)** **C (393)** Age of sample=35 (2.1)	DSM-IV	PBI (P)	***MC/MP/PC/PP***: NS **MZ twin comparison *PP***: AN>unaffected twin (n=7; *r* = 0.63) ***MC/MP/PC:*** NS
17 Low	35	UK	475 100%	CC [j]	**AN (67)** 22.4 (4.8) **Other psychiatric Disorder (OPD) (102)** [k] Not reported **BN (102)** 23.7 (4.9) **C (204)** Not reported	DSM-III-R	PBI (P)	***Low maternal care & high protection:*** AN>C (odds ratio = 3.5); AN=BN; AN=OPD ***Low paternal care & high protection:*** AN>C (odds ratio = 2.9); AN=BN; AN=OPD [l]

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
18 Low	36	US	321 100%	CC [m]	**BED (107)** **Psychiatric (P) (107)** **C (107)** All range in age from 18-40	DSM-IV	PBI (P)	***Maternal problematic parenting***: [n] BED=P>C (*r* = 0.15) ***Paternal problem parenting:*** NS
19 Medium	37	Italy	132 100%	CS	**ED (64)** 32.2 (11.5) **C (68)** 29.8 (8.9)	DSM-IV	PBI (P)	***MC/PC/Mean PC:*** ED<C ***MP/PP/Mean PO:*** ED>C

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
20 Medium	38	Japan	200 100%	CS	**ED-self harm: (ED=SH) (25)** 24.3 (5.6) **ED-no self harm (ED-no SH) (55)** 26.9 (7.9) **C (120)** 19.5 (1.2)	DSM-IV	PBI (P)	***PC:*** ED+SH<C = ED no SH ***MC:*** C>ED+SH = ED no SH ***PP/MP:*** NS
21 Low	39	US	306 100%	CS	**ED (36)** **ED symptomatic (ED-s) (69)** **C (201)** Mean age reported for sample=19.4 (2.4)	DSM-IV	PBI (P) [o]	***PC:*** ED-s=ED<C

Study No. (Bias judgement)	ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of parental bonding across groups *Evidence of mediation*
22 Medium	40	UK	116 100%	CS	**ED (66)** 32.5 (9.7) **C (50)** 30.8 (16.1)	Not reported [p]	EMBU-short form	***PR:*** ED>C ***PW:*** ED<C ***PP:*** NS *Association between PR & EDI-Drive for thinness & -body-dissatisfaction mediated by YSQ-abandonment, -& defectiveness. Association between PP & EDI-drive-for-thinness mediated by YSQ-vulnerability-to-harm*
23 High	41	UK	477 100%	CS	**ED (124)** 27.6 (7.8) **C (353)** 24.4 (SD=8.0)	DSM-IV	YPI-R YCI	***MED/PED/MP/PP/MCon/PCon:*** ED>C *YRAI scales or YCI not found to mediate relationship between PB & EDI-Drive-for-thinness, -Body-satisfaction, or -Bulimia*

Study No. (Bias judgement)	Ref	Country	Total N (% Female)	Study design	Diagnostic groups (N) Mean age in years (SD)	Diagnostic tool	PB Measure (Raters)	Comparison of PB across groups *Evidence of mediation*
24 High	42	Spain	101 100%	CC [q]	**ED-community (ED-c) (29)** 15.0 (1.8) **ED-inpatient (ED-p) (43)** 15.8 (2.0) **C (29)** 15.0 (1.8)	DSM-IV	EMBU	***MW/PW:*** ED-c=ED-p<C *(MW r =.32 for ED-c vs C; PW r =.31 for ED-c vs C; MW r =.36 for ED-p vs C; PW r =.49 for ED-p vs C)* ***MR/PR:*** ED-c=ED-p>C (MR *r* =.51 for ED-c vs C; PR *r* =.49 for ED-c vs C & ED-p vs C; MR *r* =.32 for ED-p vs C) ***MP/PP:*** ED-c>C; ED-p=C; ED-p=ED-c [r] (MP *r* = .32; PP *r* = .28) Odds ratio for having an ED: PR = 32.3, MR = 9.29, MW = 4.56, PW =4.04

Note: All findings are significant at $p < 0.05$; *r* values indicate effect sizes, where *r* =0.1 indicates a small effect size, *r* =0.30 indicates a medium effect size, and *r* = 0.50 indicates a large effect size.

CS = Cross-sectional; CC = Case-control; MZ twins = Monozygotic twin study; BN = Bulimia Nervosa; AN = Anorexia Nervosa; BED = Binge Eating Disorder; ED = Eating Disorder; C = Non-clinical; DSM = Diagnostic and Statistical Manual; PBI = Parental Bonding Instrument; EMBU = own memories of child rearing inventory; PAS = Parental Attitude Scale; CECA = Childhood Experience of Care and Abuse; YPI-R = Young Parenting Inventory Revised; P = Participant; M

= Mother; F = Father; PB = Parental Bonding; MC = Maternal care; PC = Paternal care; MP = Maternal overprotection; PP = Paternal overprotection; MA = Maternal Autonomy; PA = Paternal Autonomy; MR = Maternal rejection; PR = Paternal rejection; MW = Maternal warmth; PW = Paternal warmth; MED = Maternal emotional deprivation; PED = Paternal emotional deprivation; PCon = Paternal control; MCon = Maternal control; NS = not statistically significantly different; EDE=Eating Disorder Examination; SPSI-R = Social Problem Solving Inventory-Revised; EDI-II = Eating Disorder Inventory – II; YSQ = Young Schema Questionnaire; TCI = Temperament and Character Inventory ; YRAI = Young-Rygh Avoidance Inventory ; YCI =Young compensatory Inventory

[a] Findings remained statistically significant after controlling for age, Beck Depression Inventory (BDI;[3]), and BMI; [b] Findings were no longer statistically significant after controlling for age, BDI, and BMI; [c] Controls are unaffected twins from MZ twin pair; [d] Assessed lifetime BN using adapted questions from EDE; [e] Authors state that the sample was drawn from a treatment-seeking population; [f] Appears *t* value for MA not correctly reported in paper so there is no calculation of effect size, findings for other subscales of accepting/rejecting and overprotecting are unclear. [g] Participants matched on age and gender; [h] PBI divided into three factors rather than the usual two factors; [i] Authors do not make it explicit what demographic factors participants are matched upon; [j] Participants matched on age and social class; [k] This group included people with major depressive (81%), bipolar disorder (1%), and anxiety (18%); [l] Compared AN group to other three groups using separate logistic regression analyses, controlling statistically for current age, parental social class, and age at onset of disorder(no comparison of BN and non-clinical group); [m] All groups matched on ethnicity, age (within 2 years) and education; [n] Maternal and paternal problematic parenting included low care, overprotection and affectionless control and were derived from the PBI; [o] Authors only use the PC scale; [p] Authors report that sample have a current ED and were recruited from Eating Disorder Association; [q] Participants matched by age and school; [r] EMBU data were categorized in such a way that a subject were considered to have experienced lack of emotional warmth when the average EMBU score on this sub-scale was below the 25th percentile of the total sample score. For overprotection or rejection, scores on these sub-scales had to be above the 75th percentile.

Logistic regression revealed that MW, MR, PR, but not PW, MP, PP, remain statistically significantly associated with presence of ED (categorised as yes/no) after controlling for psychiatric comorbidity.

Table 3. Measures of parental bonding

Measure	No. of items	Self-report (SR) OR Interview (I)	Scales overlapping with PBI construct	Psychometric properties
Parental Bonding Instrument (PBI)	25	SR	NA	Concurrent validity Internally consistent Good test re-test reliability[12,46,47]
PBI-short form	10	SR	NA	Good internal consistency Test re-test reliability[48]
EMBU	81	SR	Warmth Rejection Protection [a]	Validity[49] Test re-test reliability[50]
Parental Attitude Scale (PAS)	50	SR	Accepting/Rejecting Autonomy Overprotection	No evidence

Measure	No. of items	Self-report (SR) OR Interview (I)	Scales overlapping with PBI construct	Psychometric properties
Young Parenting Inventory-Revised	72	SR	Emotionally-depriving parenting Overprotective parenting Controlling parenting	Test-retest reliability Construct validity established through correlations with negative core beliefs (44)
CECA	NA	I	Antipathy Control	Good concurrent validity Inter-rater reliability(45)

[a] Warmth reflects the same construct as the PBI-care scale, rejection reflects the polar opposite of PBI-care, and protection scale overlaps with the PBI-overprotection scale[51].

Table 4. Susceptibility to bias results

Bias indicator	***Study***																						
	1 [19]	2 [20]	3 [21]	4 [22]	5 [23]	6 [24]	7 [25]	8 [26]	9 [27]	10 [28]	11 [29]	12 [30]	13 [31]	14 [32]	15 [33]	16 [34]	17 [35]	18 [36]	19 [37]	20 [38]	21 [39]	22 [40]	23 [41]	24 [42]
Design issues																							
Missing data	Y	N	Y	N	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
PB measure	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	N	Y
Sample representative																							
ED group	S	S	H	H	S	S	S	S	H	S	S	U [a]	S	S	S	H [b]	H [c]	H	S	S	H	S	*	H
Non-clinical group	Y	Y	Y	Y	U	Y	U	N	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	U	Y	U	U	N [d]
Other group	-	-	-	-	-	-	-	-	-	-	-	Y	-	-	Y	Y	Y	Y	-	-	-	-	-	-
Appropriate selection	U	U	S	S	U	H	U	U	U	U	H	S	U	U	S	S	S	S	S	U	S	S	U	H
Confounding factors																							
Groups similar	H	H	S	S	N	S	U	U	S	N	S	S	U	N	S	H	H [e]	H	N	U	S	S	U	H
Control for comorbidity	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N

Bias indicator	***Study***																							
	1 [19]	2 [20]	3 [21]	4 [22]	5 [23]	6 [24]	7 [25]	8 [26]	9 [27]	10 [28]	11 [29]	12 [30]	13 [31]	14 [32]	15 [33]	16 [34]	17 [35]	18 [36]	19 [37]	20 [38]	21 [39]	22 [40]	23 [41]	24 [42]
Statistical analysis																								
Adequate power	U	U	U	U	U	U	U	U	U	N [f]	U	U	U	U	U	U	U [g]	U	U	U	U	U	U	U
Type I errors	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N [h]	Y	Y	Y	N	N	Y	Y	Y	N	Y
Parametric	Y	N	N	N	Y	N	N	N	N	Y	N	N	Y	Y	Y	Y	Y	N	N	N	N	Y	N	Y
Mediation analysis	-	Y	-	-	Y	-	-	-	-	Y	-	-	Y	Y	-	Y	-	-	-	-	-	Y	-	Y
Conflict of interest	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
Overall bias judgement	M	L	M	M	M	M	M	H	H	M	L	M	M	M	M	L	L	L	M	M	L	M	H	H

Note: Y=Yes; N=No; U=Unclear; H=Highly appropriate; S=Suitability appropriate; L=Low likelihood of susceptibility to bias ; M=Medium likelihood of susceptibility to bias; H=High likelihood of susceptibility to bias; *no recruitment information provided ; [a] Those with Borderline Personality Disorder were excluded from the Eating Disorder group, no reports of the number excluded on this basis; [b] Authors reports response rate is less than 50% which could introduce bias; [c] Bulimia Nervosa group recruited from the general population but the Anorexia Nervosa group recruited from Eating Disorder clinics; [d] Group not deemed appropriate because include those who scored less than 30 on the Eating Attitudes Test-26[52], albeit a score of 20 or above indicates possibility of an Eating Disorder[53]; [e] Bulimia Nervosa group matched to controls, but Anorexia Nervosa group were not; [f] Authors acknowledge that the study was not powered for mediation analysis but no comment on extent to which other analyses were statistically powerered; [g] Statistical power may have reduced by

categorising the continuous parental bonding measure; [h] A MANOVA controlling for multiple dependent variables being tested, (i.e., emotional support, overprotection and rejection) was not statistically significant and consequently the univariate analyses may have been more susceptible to Type 1 errors.

Figures

Figure 1. Flow diagram outlining article screening and inclusion