Attachment and mentalization and their association with child and adolescent eating pathology: A systematic review.

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Abstract

Objective: Insecure attachment and mentalizing difficulties have been associated with eating pathology in adulthood. However, it is unclear whether eating pathology is associated with attachment or mentalization in children. The aim of this study is to systematically review the literature in this emerging field.

Method: Electronic databases were used to search for articles.

Results: 22 studies were identified. In the 15 studies investigating attachment, an association with eating pathology was found in all studies. Mentalizing difficulties and eating pathology were found to be correlated in the seven studies which examined their association.

Discussion: In keeping with the adult literature, cross-sectional studies of children and adolescents consistently report associations with eating pathology. There is some evidence from prospective studies that insecure attachment may be a risk factor for the development of eating pathology in adolescence. The literature on mentalization and eating pathology suggests that adolescents with anorexia nervosa may have difficulties in recognizing emotions. Further research utilizing clinical samples and well-validated measures of attachment and mentalization are required to shed further light on this area.
Attachment and mentalization and their association with child and adolescent eating pathology: A systematic review.

In recent years the concepts of attachment and mentalization have attracted increasing research interest in the field of eating disorders. An association between attachment insecurity and eating disorders has been a consistent finding of numerous studies over the past quarter century. Meanwhile, several studies have reported findings of mentalizing difficulties in adults with eating disorders. Attachment and mentalization represent two aspects of social and emotional development which have been considered to be salient to eating disorders, either as foci for treatment, or through inclusion in causal models. Whilst the two concepts are distinct, there are areas of both theoretical and empirical overlap. Importantly, both attachment and mentalization are developmental processes, which appear to be in a state of flux during the adolescent period.

The number of studies which have examined attachment and mentalization in adolescent samples is relatively small, and no systematic review has been conducted in this area. The primary aim of this paper is therefore to systematically review the literature on attachment and mentalization as it relates to child and adolescent eating pathology. The secondary aim is to consider the implications of the evidence in relation to etiological models for eating disorders.

Attachment

Bowlby proposed attachment as a theory of human behavior and relationships across the lifespan. He suggested that infants develop mental representations of the self and others in response to their repeated experiences of the availability and responsiveness of their caregivers. These ‘internal working models’ (IWMs) were postulated to form a lasting template for appraisals of self and others in close relationships across the life-course.

The evidence for stability in attachment representations across the lifespan is complex. Pinquart et al.’s meta-analysis of longitudinal studies of attachment from infancy to adulthood found no significant stability in attachment classification in intervals over 15 years. However, levels of stability were greater in lower-risk samples. Possible reasons for the lack of attachment stability across time include difficulties around measurement, and changes in caregiver quality over time leading to re-appraisal of IWMs. More recently, a
twin study by Fearon et al\textsuperscript{15} showed a strong genetic influence on attachment representations in adolescence. The development of adult attachment representations therefore appears to be a complex process, with significant amounts of change across developmental periods, and a role for genes as well as environment.

In adults, attachment can be assessed by interview or self-report. The Adult Attachment Interview\textsuperscript{16} (AAI) yields four categories of attachment representations, labelled secure, dismissive, preoccupied and disorganized. An alternative approach to assessment has been the use of self-report measures of attachment style, which assess thoughts and behavior in relation to adult romantic relationships. Brennan, Clark and Shaver\textsuperscript{17} found two factors to underlie all the extant adult self-report measures included in their meta-analysis.

Attachment anxiety relates to an attachment style in which an individual is highly motivated to engage in close relationships and has a tendency to idealize others while devaluing themselves. By contrast, individuals with attachment avoidance tend to minimize their own attachment needs and avoid close relationships with others.

The measurement of attachment in middle childhood and adolescence is complex, and there is an absence of a widely agreed ‘gold standard’ measure. However, findings increasingly support the use of interview-based measures of attachment for adolescents\textsuperscript{18} such as the Child Attachment Interview.\textsuperscript{19, 20} A small number of well-validated self-report measures of attachment for children and adolescents have been developed,\textsuperscript{21, 22} but many of the measures for this age group have only limited data on validity.\textsuperscript{23}

In adult samples of patients with eating disorders, higher rates of insecure attachment have been consistently found compared with rates in community samples.\textsuperscript{24, 25, 26} Many studies have used cross-sectional study designs to examine associations between attachment security and eating pathology, using either self-report\textsuperscript{4, 27} or interview-based measures of attachment.\textsuperscript{5, 6, 28} Several more recent studies have explored potential mediators of the relationship between attachment and eating pathology (see 1 for a review). For example, both Tasca et al\textsuperscript{29} and Ty and Francis\textsuperscript{30} have reported affect regulation to be one such mediator of the relationship. Finally, a small number of studies have begun to examine the role of attachment in the treatment process for adults with eating disorders. For instance, Illing et al\textsuperscript{31} found that higher attachment anxiety was significantly related to greater
severity of eating disorder symptoms and poorer treatment outcome in a clinical sample with a range of eating disorder diagnoses.

**Mentalization**

Mentalization refers to the capacity to reflect and interpret one’s own and others’ behavior in terms of intentional internal mental states such as thoughts, feelings, and beliefs. The concept thus includes, but is not limited to, the concept of theory of mind (TOM). The concepts of attachment and mentalization are at their closest point theoretically within the developmental model proposed by Fonagy et al, who postulated that mentalization develops within the social matrix of attachment relationships.

The links between attachment and mentalization have been explored empirically with adults using the concept of *reflective function* (RF). This refers to the ability to reflect on the mind of self and others in the context of attachment relationships. RF can be rated from AAI transcripts using the Reflective Function Scale, and a small body of research has examined RF in eating disorder samples.

As compared with community controls, patients with anorexia nervosa were found to have lower RF – meaning poorer mentalization - in studies by Ward et al and Rothschild-Yakar et al. Fonagy et al also reported lower RF in a sample of adults with eating disorders, although the diagnostic profile of the sample were not specified. Rothschild-Yakar et al found that higher RF was associated with lower drive for thinness in their community sample. However, in their clinical sample, which consisted of inpatients with anorexia nervosa binge/purge subtype, no correlation was found between mentalizing and drive for thinness, and a positive correlation was found between mentalization and bulimic symptomology. A complex relationship between mentalization and bulimia is also suggested by Pedersen et al’s study, in which no difference in mean RF scores was found between a sample of women with bulimia nervosa and healthy controls. However, patients with bulimia were more polarized in their RF abilities, with more scores in both the low and high range.

Other researchers have investigated the concept of TOM within eating disorders, with a recent systematic review concluding that patients with eating disorders have a poorer
understanding of the mental states of others, as compared with controls. For example, Harrison et al found that adult women with anorexia nervosa (AN) had significantly lower scores on the Reading the Mind in the Eyes task as compared with a clinical comparison group. However, Oldershaw et al found almost complete normalization of emotion recognition ability and emotional TOM in patients who had recovered from AN, suggesting that impairments in these domains may be a consequence of starvation.

Rationale for this review

Attachment and mentalization represent two areas of social and emotional functioning that are salient in adult eating disorders, and findings support their inclusion in explanatory models both as causal and maintaining factors. However, the existing research on both attachment and mentalization is beset by a number of limitations. Firstly, studies using clinical samples may be biased by study designs in which chronically unwell patients are recruited to studies using non-random sampling methods. Secondly, studies have not always adequately controlled for potential confounders, such as depression or chronicity. Thirdly, the cross-sectional study designs used in the adult literature do not provide strong evidence about the role of either attachment or mentalization in the etiology of eating disorders. Moreover, the relationship between attachment and mentalization is unclear and likely to be complex. From a psychoanalytic perspective, both Bruch and Fonagy et al postulated a role for misattuned early attachment relationships in the development of mentalizing difficulties in patients with eating disorder. Empirically, there is evidence that RF predicts attachment status. However, mentalizing difficulties such as impairments in TOM have also been conceptualized as intermediate phenotypes that are shared between eating disorders and autism spectrum disorders (see Treasure for a review). A review of studies of child and adolescent eating pathology and its association with attachment and mentalization therefore has the potential to shed light on etiological models of eating disorders.

A second rationale for this review is that there is evidence to suggest that attachment and mentalizing may have a bearing on treatment outcome in eating disorders. It has been suggested that subgroups of eating disorder patients that differ in symptom profile,
attachment classification and mentalizing abilities might require a different therapeutic approach. These considerations are likely to be salient in the treatment of adolescent eating disorders, not least because both attachment representations and mentalizing capacity appear to be in a state of flux during adolescence. Indeed, emerging evidence from brain imaging studies (e.g. Blakemore) suggests that adolescence may be a critical stage in the development of mentalizing and social cognition. Furthermore, changes in attachment and mentalizing over the course of therapy have been shown to mediate treatment response in adolescents who self-harm, raising the possibility that attachment and mentalizing may be worthwhile treatment targets in other adolescent clinical populations, such as those with eating disorders.

**METHOD**

A systematic review of studies examining attachment and/or mentalization with children and/or adolescents with eating pathology was conducted using the Medline (1946-Present), PsychInfo (1806-Present) and Embase (1974-Present) databases. The search was conducted according to PRISMA guidelines. We searched for relevant articles in English using the keywords attachment, mentalization, social cognition, theory of mind, reflective function, eating disorders, eating pathology, anorexia nervosa, bulimia nervosa and binge eating, up until May 2015. Limits were set to exclude studies of infants, and to exclude dissertations. Reference-checking of located articles was conducted. Searches were conducted independently by HC and TG. Study eligibility was assessed by TJ. In cases where eligibility was unclear, decisions on inclusion/exclusion were made by TJ and IE.

Studies were included if they used a measure of eating pathology and also a measure of attachment or mentalization. The upper age limit for inclusion was set at 20. There is no agreed definition of adolescence, with chronological age being just one of several ways to define it. We were guided by the World Health Organization’s definition of adolescence as ending at age 19. In order to include studies of 19 year-olds, we decided on 20 as the upper age limit. Data on age range, mean age and standard deviation are presented in Tables 1 and 2. For studies which did not present a full range of data on age, we made a
judgement based on the available data and sampling method to determine inclusion. Case studies and studies of obesity were excluded.

Assessment of bias

We assessed bias within individual studies using a critical appraisal checklist adapted from Fowkes and Fulton, using the following criteria: (a) representativeness of study sample; (b) acceptability of the control group; (c) quality of the measure of eating pathology; (d) quality of the measure of attachment or mentalization; (e) attrition; (f) blindness of assessors where this was relevant to methodology. Bias was assessed by TJ and HC, with IE providing additional advice.

RESULTS

The initial search yielded a total of 460 studies, which were screened for suitability by reading the title and abstract. Of these, 111 studies were identified as potentially being suitable for inclusion, and the full texts of these studies were appraised. Following this phase, 22 studies were identified as meeting criteria, and 89 were excluded. A flowchart detailing the search and the reasons for exclusion can be found in Figure 1. Details of attachment studies can be found in Table 1, and mentalization studies in Table 2.

Risk of bias within studies

Results for the assessment of bias within individual studies are presented in Tables 3 and 4 for attachment and mentalization studies respectively. Two issues stood out as raising the risk of bias for many of the studies in this review. Firstly, the representativeness of samples was uncertain in most of the studies in the review, with most studies using non-random sampling methods. Only the studies by Le Grange et al and Milan and Acker, both of which were drawn from large population studies, used sampling methods to try to ensure a representative sample. Of the studies using clinical samples, only the studies by Zonnevyle-Bender and Zonnevijlle-Bender were rated as having a lower risk of bias, which was due to their use of a consecutive sampling approach. The second risk of bias came from the use of poorly validated measures, an issue which will be discussed further in the Results and Discussion sections.
Cross-sectional studies of attachment and eating pathology

Eleven studies used cross-sectional designs to investigate associations between eating pathology and attachment in children and adolescents. In all eleven studies, an association between these two variables was reported.

Three studies investigated attachment and eating pathology in community samples of pre-adolescent or early adolescent children. In Sharpe et al’s\textsuperscript{59} study of 305 girls, participants who were classed as insecurely attached reported significantly higher weight concerns. However, attachment was assessed using Hazan & Shaver’s\textsuperscript{60} attachment item, in which participants are asked to choose which out of three short paragraphs best described their feelings. The paragraphs are based on adult romantic attachment styles, and it is unclear whether this is a valid measure of attachment in pre-adolescent children. A similar problem applies to the study by Meesters et al,\textsuperscript{61} in which 405 children aged 10-16 were given a modified version of the Relationship Questionnaire,\textsuperscript{62} which is another measure of adult romantic attachment styles. The modified measure aims to assess attachment to mother, father and peers with one item for each attachment figure. The validity and reliability of the adapted children’s version of the instrument is low. The study found a correlation between insecure attachment towards mother and food preoccupation/dieting in boys only.

Cate et al\textsuperscript{63} used the Inventory of Parent and Peer Attachment (IPPA) in their study.\textsuperscript{21} In a sample of 76 girls, a significant negative correlation between eating pathology and attachment security was found, when the latter was measured on a single dimension of security. The IPPA was also used by Orzolek-Kronner\textsuperscript{64}, who investigated attachment and eating pathology within three samples aged 12-20 – a sample with eating disorders, a clinical comparison group with other psychiatric diagnoses and a healthy control sample. Scores for attachment security were similar in the eating disorder and clinical comparison groups, but were significantly lower in the control group. This suggests that there is an association between attachment insecurity and psychiatric morbidity that is not specific to eating pathology.

Three studies investigated associations between attachment and eating pathology in late-adolescents. Pace et al\textsuperscript{65} examined binge eating symptoms and attachment in a community sample of 233 late adolescent students. The group of students with high self-reported binge...
eating symptoms was found to have significantly lower scores on the secure attachment scale of the Relationship Questionnaire.\textsuperscript{62} On the insecure attachment sub-scales, the group reporting binge eating symptoms had significantly higher scores on the preoccupied and fearful subscales of the attachment measure, but not on the avoidant subscale.

Cole-Detke and Kobak\textsuperscript{66} investigated both eating disorder and depressive symptoms in a sample of college women. Based on a three-way coding of attachment classification derived from the AAI Q-Sort,\textsuperscript{67} the group with high self-reported eating pathology was found to have a high proportion of participants in the Dismissing category (67\%). Once depressive tendencies were controlled for in the analysis, eating disorder symptoms were associated with deactivating attachment strategies, such as attempting to divert attention away from attachment cues in the interview.

Mayer et al\textsuperscript{68} investigated the direct and indirect effects of risk factors for eating behavior problems in a non-clinical sample of late adolescent females. In keeping with other studies, insecure attachment was positively correlated with eating behavior problems. However, in the regression analysis, insecure attachment was not found to have a direct effect on eating behavior problems, but to have indirect effects via self-esteem and depression. This raises the possibility of self-esteem and depression as potentially important mediators of the relationship between attachment and eating pathology.

Cross-sectional studies involving tests of mediation

Three cross-sectional studies testing mediation models were identified.

Back\textsuperscript{69} examined eating behaviors, attachment and parental upbringing in a sample of 80 high school students. The negative relationship between secure mother attachment and eating pathology was fully mediated by body and weight dissatisfaction, whereas the positive relationship between fearful attachment and eating was only partially mediated by body dissatisfaction.

Van Durme et al\textsuperscript{70} investigated the role of emotional regulation as a potential mediator of the relationship between insecure attachment and eating pathology in a non-clinical sample of 952 children aged 10-15. Maladaptive emotional regulation was found to partially mediate the effect of both attachment avoidance and anxiety on eating restraint and eating
attachment and mentalization

Pathology. A limitation of the study is the use of the Experiences in Close Relationships Scale-Revised\(^7\), which has poor evidence of construct validity\(^2\).

Boone\(^2\) examined the association between attachment and binge eating symptoms in a community sample of 328 students aged 14-20. Perfectionistic self-promotion was found to fully mediate the relationship between avoidant attachment towards father and binge eating. Socially prescribed perfectionism fully mediated the relationship between attachment avoidance towards mother and binge eating.

Finally, attachment itself was investigated as a mediator of the relationship between self-esteem and loss of control of eating in a study by Goossens et al.,\(^3\) in a community sample of 555 pre-pubertal children aged 8-11 years. The relationship between self-esteem and loss of control was fully mediated by attachment toward mother and partially mediated by attachment toward father. A limitation of all four studies is that, by the nature of their cross-sectional design, they provide evidence of associations. Further testing within longitudinal study designs is needed to confirm the hypothesized mediating role of these variables.

Prospective studies of attachment and eating pathology

Five prospective studies were identified in this review.

Milan and Acker\(^4\) investigated the effect of attachment insecurity in early infancy on responsivity to risk factors for eating disorders in a sample of 447 girls aged 15. Attachment was assessed at 36 months, using Cassidy et al.’s\(^5\) modified Strange Situation. Eating attitudes were assessed at age 15 using the Eating Attitudes Test.\(^6\) Maternal affect was measured at ages 11, 12 and 15, and averaged across time-points in the mediation analysis. At age 15 the study also recorded the child’s relational style, body mass index (BMI) and pubertal weight change, as compared with the BMI recorded at age 9. The study found no evidence of a direct association between attachment status in early childhood and eating pathology in adolescence. However, BMI predicted disordered eating for those adolescents with a history of insecure attachment in early childhood. Among adolescents with a history of insecure attachment in early childhood, pubertal weight gain and maternal negative affect had an indirect effect on eating pathology via a preoccupied relational style in adolescence.
Goossens et al\textsuperscript{76} investigated attachment as a predictor of weight gain and eating pathology in a community sample of 688 preadolescents. Attachment was assessed using Kerns et al.'s\textsuperscript{22} Security Scale, which yields an overall score of security. Eating pathology was assessed using a Dutch adaptation of the Children’s Eating Disorder Examination Questionnaire (ChEDE-Q).\textsuperscript{77} Measurement of variables was repeated one year later. At baseline, attachment to mother and attachment to father were both negatively correlated with eating pathology, but the magnitude of the correlation was greater for maternal attachment. Attachment toward mother at baseline significantly predicted increases in adjusted BMI, eating restraint and eating, weight and shape concerns one year later.

Le Grange et al\textsuperscript{55} report a study investigating infant and child pathways to abnormal eating attitudes in adolescence, drawing on a large Australian cohort of children. Eleven waves of data were collected from infancy to age 15-16. At age 13-14, participants completed the IPPA.\textsuperscript{21} A path analysis was performed to identify developmental correlates of abnormal eating as measured at age 15-16. Peer attachment, but not parent attachment, was included in the final path model. Surprisingly, higher scores for peer attachment, representing stronger relationships, were associated with an increase in abnormal eating at age 15-16. Correlations between parent and peer attachment and eating pathology are not reported.

Two further longitudinal studies met criteria for review, both of which are limited by small sample sizes. Colton et al\textsuperscript{78} investigated attachment and eating pathology in a sample of 106 girls with Type 1 diabetes. Attachment security to mother and father, as rated on the IPPA\textsuperscript{21}, were both predictors of new-onset disturbed eating behaviour one year later. However, only 10 girls in the sample reported new disturbed eating behaviour at the second time point, and external validity is compromised by the specificity of the sample. Burge et al\textsuperscript{79} conducted a study of attachment and psychopathology in a community sample of 137 late adolescent women. Attachment was assessed by self-report measures, but psychopathology was assessed via structured interview. The study found no main effect for attachment on eating pathology one year later. However, when interaction terms were entered into the regression analysis, the interaction of attachment with initial symptomology was predictive of increases in eating pathology. However, only two women in the sample met criteria for eating disorders, with a further five women exhibiting eating disorder symptomology.
Overall, the longitudinal studies in this review have a number of limitations. Firstly, the relationship between self-report measures of eating pathology and caseness is problematic, in part because eating disorders meeting diagnostic threshold are relatively uncommon. The EAT questionnaire, which was used in Milan and Acker’s study, has been found to have high rates of false positives and false negatives, and Eisler and Szmukler found that social class was a confounding variable in their community sample of British schoolgirls who completed the EAT. Similar doubts apply to the ChEDE-Q used in the Goossens et al study. The ChEDE-Q was validated in a sample of obese youngsters and was found to produce significantly different results as compared with interview measures in respect of binge eating symptoms. Indeed, agreement levels between the ChEDE-Q and interview were lower than those reported for the adult self-report EDE-Q on which it is based, and its developers suggest that further validation work is necessary, using samples with a broader range of eating disorders. The EDI used in Le Grange et al’s study, is also not a diagnostic tool. The one longitudinal study which used a diagnostic interview to assess eating pathology, by Burge et al, did not find attachment to predict increases in eating pathology when controlling for previous eating pathology. Thus the findings of the longitudinal studies suggesting that attachment insecurity predicts later eating pathology should be interpreted with caution, given the uncertain relationship between high scores on self-report measures of eating pathology and clinically significant eating disorder symptoms. This limitation also applies to all the other studies in this review using self-report measures of eating pathology in community samples.

A further limitation of the Goossens et al study is the confound between eating pathology and weight gain. The authors take categorical BMI increases (e.g. from overweight to obese) to be indicative of eating pathology, yet they do not differentiate between weight gain of those who are underweight and those who are normal or overweight. In Milan and Acker’s study, pubertal weight change measured between the ages of 9 and 15 was found to indirectly predict increased disordered eating attitudes, although only in the sample with an insecure attachment history at 36 months. Pubertal weight change is reported without reference to BMI categories, such as overweight. A fair reading of Milan and Acker’s findings is that insecure attachment may have a moderating effect on the impact of weight gain around puberty on eating attitudes, albeit the effect is modest. As acknowledged by
the authors, it is not clear to what extent such a finding applies to clinical samples with eating disorders.

The study by Le Grange et al\textsuperscript{55} is notable in being the only study in this review that did not report an association between parent attachment and eating pathology. Also of interest is the finding that stronger peer attachment was associated with increases in eating pathology. However, the conclusion validity of this study is threatened by the fact that the attachment measure used was significantly shortened.

**Studies of mentalization and eating pathology**

Seven studies of mentalization and eating pathology were identified.

**Studies investigating emotion recognition**

Four studies were identified which investigated emotion recognition abilities in adolescents with eating disorders. Three of these studies\textsuperscript{57,58,87} used images of facial expressions developed by Matsumoto and Ekman.\textsuperscript{88} These are a well-validated set of images to assess facial recognition, with several studies supporting their validity and reliability.\textsuperscript{89}

Zonnevijlle-Bender et al\textsuperscript{57} investigated emotion recognition in a clinical sample of 30 adolescent girls with an eating disorder aged 12 – 18, and compared this with a healthy control sample. Participants were shown images of female models showing one of seven emotions (happiness, anger, sadness, fear, disgust, surprise and contempt). The eating disorder sample performed significantly worse than healthy controls in recognising emotions. This procedure was repeated in a second study\textsuperscript{58} by this research group, this time comparing the performance of adolescent and adult samples with AN. The authors found no significant difference between the two groups in their ability to recognise emotions.

Lulé et al\textsuperscript{87} investigated accuracy and speed of emotion labelling in a sample of 15 adolescent females with AN, as compared to 15 age and sex-matched healthy controls. Participants were shown images of faces displaying the six basic emotions (anger, fear, sadness, surprise, disgust and happiness) on a computer. Adolescents with AN showed a shorter reaction time for recognition of emotions relative to controls. The accuracy of perception did not differ between the clinical and control groups for fear, surprise, sadness
and anger. Adolescents with AN showed less accuracy in recognizing disgust, but greater accuracy in recognizing happiness. This study has a risk of bias due to the small sample size. Moreover the clinical sample included both inpatients and outpatients, whose treatment ranged from 8 to 736 days. Data on how many of the clinical sample had been admitted to hospital are not provided. It is therefore hard to assess the representativeness of the clinical sample. A strength of the study is that the clinical sample were physically stable, thus implying that their performance was not compromised by effects of starvation.

Lang et al\textsuperscript{90} investigated emotion recognition in adolescents and adults with AN using a body motion paradigm, as compared with health controls. Participants viewed video clips of actors walking from left to right whilst portraying one of four emotions (anger, fear, happiness or sadness) or an emotionally neutral state. The actors were filmed in the dark and had small lights attached to various body parts, so that the stimuli appeared to participants as white dots on a dark background. The study found no differences between the clinical and healthy control samples with respect to recognising emotions, with the exception of sadness. Adolescents with AN were less accurate than adolescent healthy controls in recognising sadness. Both adolescents and adults with AN were less accurate than the healthy control group in recognising sadness. However, within the clinical sample, adolescents were poorer than adults in recognising sadness.

One limitation of the study concerns the validity and reliability of the emotion recognition task. The authors developed the scoring method using a small reference group of 15 healthy controls. This reference group did not view the clips portraying ‘neutral’ emotions, which could have introduced bias, as respondents may give different answers regarding emotion when the stimuli is changed, as the sequence and content of the materials may influence interpretation.

**Other studies of mentalization and eating pathology**

Three other studies were identified which used disparate study designs and measures to investigate mentalization and eating pathology.

In the study by Cate et al,\textsuperscript{63} which examined both mentalization and attachment, 76 girls (aged 9-12) were administered self-report measures of eating pathology. Subjects scoring at
the highest and lowest extremes of eating pathology were administered a picture task developed by Murray\textsuperscript{91} in which they had to make up a story relating to each of the five pictures. Mentalizing capacity was assessed using a coding system applied to the transcripts of the children’s responses.\textsuperscript{92} The total mentalization score was based on the girls’ use of mentalistic language. The group which scored highly on the measure of eating pathology was found to have significantly lower mentalization scores compared with the group with low scores. Significant negative correlations were found between eating disorder risk and attachment styles, and between eating disorder risk and mentalization.

Rothschild-Yakar et al\textsuperscript{93} examined mentalizing in a sample of female inpatients with eating disorders. The clinical sample comprised 71 female inpatients aged 14–19 and an age-matched sample of healthy controls. Mentalizing in self and others was assessed using the Object Representation Inventory (ORI).\textsuperscript{94} Patients with eating disorders presented with a significantly lower level of symbolic representation and with more malevolent representations of their parents in comparison to controls. Across all study participants, a more benevolent parental representation, specifically with father, combined with better mentalization abilities, was found to indirectly predict lower eating disorder symptoms, via the reduction of distress.

Schulte-Rüther et al\textsuperscript{47} conducted a study of 19 adolescent patients (aged 12–18) with AN and 21 age-matched controls. Participants were investigated using functional magnetic resonance imaging during TOM tasks at two time-points – for the patients with anorexia this was at admission and discharge from hospital. TOM was assessed using an experimental paradigm involving 15-second videos of three white geometric shapes moving against a black background. Participants were asked whether the shapes were ‘friends’ or not based on the contingency of the interactions, which were designed to be interpreted as social. No differences were found between the clinical and comparison groups. However, irrespective of time-point, patients with AN showed reduced activation in the middle and anterior temporal cortex and in the medial prefrontal cortex. Hypoactivation in the medial prefrontal cortex at admission to hospital was correlated with poor clinical outcome at 1-year follow-up. The medial prefrontal cortex is known to be a region of the brain that is activated during mentalizing tasks.\textsuperscript{49, 95}
In all three studies, the external validity of the findings is compromised by the relatively poor validity of the instruments used to assess mentalization. In Cate et al’s study, the use of Symons et al’s system to code responses to Murray’s Thematic Apperception Test is a novel but not yet validated test of mentalization in 9-12 year olds. No other measure of mentalization was used, thus the concurrent validity of this measurement approach is unknown. Similarly, the use of Blatt et al’s ORI also lacks validity data in adolescent samples. Finally, behavioral performance on the experimental measure used in Schulte-Rüther et al’s study did not discriminate between AN and non-clinical samples in either this study, or in the study by McAdams and Krawczyk, although both studies reported differences at the level of neural function. As such, this measure has been rated as presenting a lower risk of bias, although it should be noted that the sample sizes in these brain imaging studies were small.

DISCUSSION

This review has found limited evidence of an association between mentalizing difficulties and eating pathology in children and adolescents. Thus far, the most replicated finding is that of emotion recognition difficulties in adolescent samples with eating disorders, particularly AN, although the heterogeneity of the samples in this sub-group of studies reduces the strength of this finding. All the studies in this review reported an association between mentalizing difficulties and eating pathology, and an association was found between insecure attachment and mentalizing difficulties in the one study which examined the two variables. However, caution is warranted in interpreting findings given that small sample sizes and poorly-validated measures of mentalization were a limitation of several studies. A gap in the adolescent literature is the absence of studies comparing mentalizing ability in clinical samples of young people with anorexia nervosa whilst ill and following recovery. The evidence in this review thus does not speak to the issue of mentalizing difficulties as a trait or state. Within the taxonomy developed by Kraemer et al, current evidence suggests that mentalizing difficulties may be a correlate of eating pathology in children and adolescents.

With regard to attachment, a positive correlation between attachment insecurity and eating pathology in childhood and adolescence was found in 14 of the 15 studies in this review. The validity of this association is strengthened by the use of developmentally appropriate,
well-validated measures of attachment, such as Kerns et al’s security scale, within large samples, such as in the study by Goossens et al. However, there is a risk that the association between attachment and eating pathology may be confounded by variables such as low self-esteem and depression, and future studies in this area need to look more closely at potential confounders which might impact on appraisals of self and others.

The evidence of an association between attachment insecurity and eating pathology replicates the findings of the adult literature. Of note, this association has been found in non-clinical samples across the age range from pre-adolescence to late adolescence. The studies in this review also found an association between insecure attachment and a broad range of eating pathology.

In terms of the question of etiology, the findings of the longitudinal studies in this review merit particular attention. Milan and Acker found that attachment insecurity in infancy is a weak predictor of later eating pathology, whereas Goossens et al found that attachment insecurity in the pre-adolescent period predicted the development of eating pathology one year later much more robustly. Le Grange et al found attachment to peers, but not parents, predicted eating pathology in mid-adolescence. This latter study has the lowest reliability of the three longitudinal studies in this review, on account of its use of a significantly shortened version of its attachment measure. The Milan and Acker study further discredits what O’Kearney has referred to as unitary causation models – quite clearly, early attachment insecurity is not the cause of eating disorders. The contrast between the findings of Milan and Acker and Goossens et al also emphasizes the point that insecure attachment as assessed in adolescence or adulthood cannot be taken to be evidence for insecure attachment in infancy. The stability of attachment from infancy to adulthood is limited and Fearon et al recently found evidence for a strong genetic influence on attachment representations in adolescence. These findings help make sense of why the predictive value of insecure attachment appears to be stronger in the pre-adolescent phase as compared with infancy. The available evidence suggests that insecure attachment is both a correlate and risk factor for eating pathology. However, given that no study has demonstrated that change in attachment alters the risk of eating pathology, insecure attachment has not been established as a causal risk factor.
Implications for theory

The findings of this review, and other developments in the fields of attachment and mentalization, suggest a need to revise theory regarding the relationships between attachment, mentalization and eating pathology. Firstly, infant attachment has been considered along with other perinatal factors, such as obstetric difficulties, in developmental models of risk for anorexia nervosa.\(^{11,99}\) However, the prospective studies examined in this review\(^ {56,73,76}\) suggest that it is the pre-pubertal and pubertal phases of development in which attachment may come to play a more important role in the development of eating pathology.

Secondly, the meaning of adolescent attachment needs to be reconsidered as more than a marker of parent-child relationships, since it appears to be partly heritable\(^ {15}\) and more closely associated with qualities of peer relationships than with parent-child relationships.\(^ {100}\) Theory needs to include a role for peer attachment in the development of eating pathology, as suggested by the findings of Le Grange et al.’s\(^ {55}\) study. The potential role of affect regulation needs expanding in theoretical models, as supported by findings from both the child\(^ {70}\) and adult\(^ {29}\) literature. Moreover, it has been suggested that adolescent attachment interviews are better viewed as measuring affect regulation in the context of social interaction, rather than measuring parent-child relationships \textit{per se}.\(^ {100}\)

Thirdly, theoretical links between attachment and mentalization need development. Mentalizing ability is a dynamic capacity that is influenced by stress and arousal, particularly in the context of specific attachment relationships.\(^ {101}\) Under conditions of high arousal, patterns of brain activity ‘switch’ from flexibility to automaticity, resulting in the loss of mentalizing.\(^ {102}\) The threshold for this ‘switch’ to automatic, non-mentalizing modes of thought varies between individuals. However, the ability to tolerate strong negative affects is a marker for secure attachment.\(^ {102}\) Individuals with insecure attachment representations are thus more likely to experience mentalizing failures in the context of affective arousal.

This could have implications for treatment, particularly when using eating disorders focused family therapy (FT-AN) - sometimes referred to as family based treatment or FBT - where an early focus is on parents helping the young person to eat.\(^ {103,104}\) If adolescents and/or other family members become highly emotionally aroused during therapy sessions, they may be more likely to interpret material arising from the session in negative terms and feel criticised.
or blamed. This could potentially reduce family cohesion and damage the therapeutic alliance, thereby moderating the effectiveness of treatment. This may be a particular risk for adolescents and adults categorised as having preoccupied attachments on interview measures of attachment. Individuals assigned this category display on-going anger towards attachment figures, often seeming ‘caught up’ in past grievances. It is possible that adolescents displaying attachment preoccupation may benefit less from FT-AN, since they may experience their parents’ attempts to help them manage their eating as controlling or even punitive. By contrast, adolescents with secure attachment representations may be more likely to experience parental attempts to help them manage their eating as an act of care and support.

Implications for research

Stice et al have called for methodologically rigorous prospective risk factor studies that include a broad range of variables to investigate the possibility of interaction between risk factors. On the basis of this review, attachment, mentalization and affect regulation should be included as variables within such studies. Other known risk factors for eating disorders, such as genetic vulnerability and perfectionism will also need inclusion, as should potential mediators such as depression and self-esteem. Given the relative rarity of clinical cases of eating disorders, prospective studies of at-risk community samples, drawn from large population studies, will be needed to further understanding regarding the interaction of risk factors. Case-control study designs, in which participants are matched for level of eating pathology, but differ on attachment and mentalization, could help to shed light on whether, and how, these variables are implicated in the development of eating pathology for some individuals.

With regard to measurement of attachment and mentalization, both are complex constructs and it will be necessary to use a range of measures to investigate them. It is known that self-report and interview measures tap different aspects of attachment. Studies of adolescent eating disorder samples using interview-based measures such as the Child Attachment Interview would help to elucidate aspects of attachment representations that are not available to conscious appraisal, such as idealisation of attachment figures. Similarly, mentalizing is also a multi-faceted concept and requires a range of assessment measures to explore fully. Importantly, well-validated measures of mentalization for children and
adolescents have now been developed, and their use in future studies would represent an advance on the measures used in the studies included in this review. Future studies should also adopt more rigorous approaches to the measurement of eating pathology in children and adolescents, for instance by combining self-report instruments with interview measures or clinical diagnosis as given by specialist eating disorder clinicians.

Furthermore, future research should investigate attachment and mentalization using clinical samples of children and adolescents, and should explore the impact of these variables on the process and outcome of treatment in child and adolescent eating disorders. Studies in the adult field have shown differential responsiveness to treatment approach based on attachment style and mentalizing. In this review, the study by Rothschild-Yakar et al suggests there is variance in mentalization within clinical adolescent eating disorder samples. Future research should investigate whether variance in mentalization moderates treatment response in adolescent populations.

**LIMITATIONS**

A limitation of this review is that we could not rule out the possibility of publication bias, given that positive findings are more likely to be published than negative findings. The apparent relationship between attachment, mentalization and eating pathology may therefore be weaker than it appears.

**CONCLUSION**

The finding of an association between insecure attachment and eating pathology has been replicated several times in child and adolescent samples. The findings of the longitudinal studies in this review suggest that insecure attachment in early infancy is a distal risk factor with a relatively weak effect on adolescent eating pathology, whereas insecure attachment in pre-adolescence may predict increases in eating pathology more reliably. Currently, few studies have examined associations between mentalization and eating pathology in children and adolescents. The available evidence suggests that poor mentalization may be associated with eating pathology in both clinical and non-clinical samples. Future research in non-clinical samples should include prospective studies and experimental designs, to elucidate potential etiological pathways. Studies using clinical samples should investigate the roles played by attachment and mentalization in the process and outcome of treatment. Given
the multi-factorial causation of eating disorders, any single variable is likely to have a complex relationship with eventual eating disorder pathology, in interaction with multiple other variables. The utility of the constructs of attachment and mentalization will therefore not be in providing a definitive account of etiology. By contrast, the value of these constructs to the eating disorder field will be in the extent to which they can inform innovations to treatment programs that lead to improved outcomes. On this basis, whilst the evidence base is currently very limited, attachment and mentalization represent potentially promising areas for further research.
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