VOLUME 1: RESEARCH COMPONENT

PREDICTORS AND OUTCOMES OF EATING DISORDERS

By

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A thesis submitted to the University of Birmingham in partial fulfilment for the degree of DOCTORATE OF CLINICAL PSYCHOLOGY

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THESIS OVERVIEW

This thesis is submitted in partial fulfilment of the requirements of the degree of Doctorate of Clinical Psychology at the University of Birmingham. It comprises both research and clinical components of the course.

Volume 1 is the research component which consists of two parts. Part I is a systematic review which aims to establish whether women with active or historic eating disorders are more vulnerable to the occurrence of pregnancy or delivery complications than women without eating disorders. Four electronic databases were searched and 15 studies were deemed relevant. Having reviewed the quality of the methodology of each study and compared the findings, there is currently no reliable evidence to suggest that women with eating disorders are at greater risk of pregnancy or delivery complications. This is in contrast to the arguments made by a multitude of non-systematic review papers that have formed conclusions on single or low quality papers. The review explains how the range of methods and outcomes utilised across the studies evaluated means that comparing the studies and forming reliable conclusions was difficult. The review recommends more consistent measures of birth cohort data and considers the implications of the findings as they currently stand.

Part II of Volume 1 is an empirical study that investigates the mediating role of emotion regulation in the association between early caregiver relationships and the development of eating disorders in adulthood. A non-clinical and a clinical sample were recruited via online media. Bootstrapped parameter estimators and confidence intervals were utilised in a mediator model. Specific emotion regulation deficits were

found to be important mediators in the relationship between early parental rearing styles and specific eating disorder symptomology. Emotion regulation deficits possess categorical roles that are distinctly different for clinical and non-clinical samples. Tentative explanations are hypothesised and clinical implications for the treatment focus of women with eating disorders are offered.

Volume 2 is the clinical component which consists of five clinical practice reports (CPR). Each CPR provides evidence of clinical work undertaken during the doctoral course. Although each CPR is an account of work completed, the names and identifiable information for clients or other individuals involved have been changed to provide anonymity and protect their confidentiality. CPR 1 presents the case of Megan, a 23 year old female referred to adult mental health services due to relationship difficulties and underlying low self-esteem. Her assessment and formulation is described from a cognitive-behavioural and a psychodynamic perspective respectively. CPR 2 reports on a service evaluation conducted to evaluate referrer satisfaction of a secondary care adult community mental health service. CPR 3 is the case study of John, a 76 year old male with post-stroke depression. A cognitive-behavioural approach was taken for his assessment and formulation, and recommendations for treatment were made prior to his disengagement. CPR 4 presents the case of Luke, a five-year old boy with learning disabilities and physical health difficulties. The behavioural intervention for his sleep problems is detailed. CPR 5 is presented as an abstract outlining the case of Joshua, an 11 year old boy with obsessive-compulsive disorder. In the oral presentation given for this assessment, the cognitive-behavioural approach utilised with Joshua was discussed.

DEDICATION

To my Mum, Dad, Charlie and Matt.

For always knowing exactly what to say and when to say it.

And for providing chocolate.

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I would like to express my gratitude to all the participants who took the time to take part in my study. It has been an inspiration to know that so many people support the development of a greater understanding of eating disorders and are willing to share their experiences to do so.

I would particularly like to thank B-Eat charity, whom enabled my study to be as far-reaching as it became. It is clear that so many people affected by eating disorders value the support they have received from such a compassionate charity.

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SYSTEMATIC REVIEW

ARE WOMEN WITH EATING DISORDERS AT GREATER RISK OF PREGNANCY AND DELIVERY COMPLICATIONS THAN THOSE WITHOUT? A SYSTEMATIC REVIEW OF THE LITERATURE.

Paper to be edited for submission to the British Journal of Obstetrics and Gynaecology

ABSTRACT

Background: Eating disorders are known to increase the risk of serious medical conditions. Pregnancy may pose additional health complications over and above those created by eating disorders or pregnancy alone.

Objectives: This systematic review aims to establish whether women with eating disorders are at greater risk of pregnancy and delivery complications than those without.

Data sources and study eligibility: Four databases were searched. Papers were included if they investigated eating disorders as risk factors for maternal complications during pregnancy or delivery compared to unexposed women. Papers were excluded, for example, if the focus was risks to the foetus.

Conclusions: The 15 studies evaluated, suggest that at present there is no reliable evidence to suggest women with eating disorders are at greater risk of physical complications during pregnancy and delivery. This is in contrast to conclusions made by a host of non-systematic review papers, which have made interpretations based on a limited number of study findings. However, the variation in quality of studies reviewed, along with the broad range of outcomes measured, made forming reliable conclusions difficult.

Implications: While non-systematic reviews press for increased screening of eating disorders there is little to suggest this is necessary and may be counterintuitive.

1.0 INTRODUCTION

1.1. Eating disorders and associated medical complications

The Diagnostic and Statistical Manual of Mental disorders (5th Ed.; DSM-V; The American Psychiatric Association, 2013) defines anorexia nervosa (AN) as a restriction of energy intake (less than is required) leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. In addition, one must have an intense fear of gaining weight or becoming fat, even though underweight. Finally, an individual must have a disturbance in the way in which they view their body weight or shape, the impact of body weight or shape unduly influencing self-evaluation, or denial of the seriousness of their current low body weight. For those with AN, risks are greatly increased for a vast range of debilitating and potentially life-threatening medical problems covering all major organs and including cardiovascular complications, osteoporosis and gastrointestinal disturbances (Hadley, & Walsh, 2003; Herzog, Deter, Fiehn, & Petzold, 1997; Katzman, 2005; Mitchell & Crow, 2006).

The DSM-V also provides a definition for bulimia nervosa (BN). For individuals to meet the criteria for a diagnosis of BN they must have *recurrent episodes of binge eating* characterised by eating large amounts of food within a 2 hour period *and* they must have a sense of a lack of control over eating during an episode. An individual with BN will also have recurrent inappropriate *compensatory behaviour*, known as purging, in order to prevent weight gain. These behaviours both occur, on average, at least once a week for three months. As with the characteristics of AN, their self-evaluation is also unduly influenced by body shape and weight. In order to differentiate the disorder from AN, BN must not occur exclusively during episodes of

AN. Similar to AN, BN also has inherent medical risks associated. Gastrointestinal problems and type I diabetes, for example are more likely in individuals with BN (Hadley & Walsh, 2003; Mitchell & Crow, 2006).

Binge eating disorder (BED) is also defined by the DSM-V. BED is characterised by recurrent episodes of binge eating. As with BN, each episode is characterized by eating, within any 2-hour period, an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances *and* a sense of lack of control over eating during the episode. Specific to BED, binge eating episodes must also include at least three of the following:

1. Eating much more rapidly than normal
2. Eating until feeling uncomfortably full
3. Eating large amounts of food when not feeling physically hungry
4. Eating alone because of feeling embarrassed by how much one is eating
5. Feeling disgusted with oneself, depressed, or very guilty after a binge eating episode.

Furthermore, BED is characterised by marked distress regarding the binge eating. It occurs, on average, at least once a week for three months, as with BN. However, unlike BN, the binge eating is *not* associated with the recurrent use of inappropriate compensatory behaviour. It does not occur exclusively during the course of AN or BN. As with AN and BN, BED is associated with adverse medical complications including type II diabetes and insomnia (Herpertz et al., 1998; Johnson, Spitzer & Williams, 2001).

The final eating disorder diagnosis is that of other specified feeding or eating disorder (OFSED), previously known in the 4th edition of the DSM as eating disorders not otherwise specified (EDNOS). As with the other eating disorder diagnoses, OFSED causes significant distress. For individuals with disordered eating who do not meet the criteria for other classifications of ED, a diagnosis of OFSED may be given. There are 5 examples offered by the DSM-V for individuals who may fit this classification:

1. *Atypical Anorexia Nervosa*: Meets the criteria for AN but is within a healthy weight range.
2. *Bulimia Nervosa of low frequency and/or limited duration:* Meets the criteria for BN but the binge eating and compensatory behaviors occur less than three times a week and/or has been occurring for less than 3 months.
3. *Binge Eating Disorder of low frequency and/or limited duration:* Meets the criteria for BED but the binge eating occurs less than three times a week and/or has been occurring for less than 3 months.
4. *Purging Disorder:* Recurrent purging of calories by self induced vomiting, misuse of laxatives and diuretics and/or excessive exercising but without the occurrence of binge eating.
5. *Night Eating Syndrome:* Recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating.

Although these examples are given, diagnosis is not limited to any one of them and other manifestations of disordered eating may be included here. Given the overlap with the other diagnoses, this classification also comes with inherent health risks. In fact, it is widely established that of all psychiatric disorders, EDs generally have the highest mortality rate (Chesney, Goodwin, & Fazel, 2014; Crow et al., 2009).

1.2. Eating disorders, pregnancy and medical complications

Although pregnancy was once considered rare in the eating disorder population, due to the impact upon ovulation (particularly in relation to anorexia nervosa) it is now evident that many women with eating disorders are becoming pregnant (Brinch, Isager, & Tolstrup, 1988; Crow, Keel, Thuras, & Mitchell, 2004). Turton, Hughes, Bolton and Sedgwick (1999) report that 1% of all pregnant women will have an eating disorder, most commonly anorexia nervosa. However, pregnancy itself comes with its own increased risk of health complications. Physical health difficulties prior to pregnancy can be triggered or exacerbated but pregnancy may also trigger new physical health complications such as hyperemesis gravidarum (HG), gestational diabetes mellitus (GDM) and pregnancy-induced hypertensive disorders, such as high blood pressure or associated pre-eclampsia. Given that it is well established that EDs are at significantly higher risk of physical health complications anyway, it is important to establish whether this population is at an increased risk of physical health complications in pregnancy, over and above the health concerns that EDs alone can create.

Early case studies and case series go some way to suggest women with ED are at risk of medical complications such as premature delivery during the perinatal period (Brinch et al., 1988; Mitchell, Seim, Glotter, Soll, & Pyle, 1991; Stewart, Raskin, Garfinkel, MacDonald, & Robinson, 1987). With that in mind, it is also important to consider not only the impact of these complications on the mothers, but the potential impact for the offspring too. For example, individuals with low birth weights (which commonly occur as a result of premature delivery) have been found to be more likely to experience educational problems and physical health difficulties later in life (Hack et al., 2002; Lemons et al., 2001). Moreover, it has been noted that individuals who develop an eating disorder are more likely to have experienced complications in utero or at birth (Cnattingius, Hultman, Dahl & Sparén, 1999; Favaro, Tenconi, Bosello, Degortes, & Santonastaso, 2011; Favaro, Tenconi, & Santonastaso, 2006; Favaro, Tenconi, & Santonastaso, 2008). If women with eating disorders experience more perinatal complications, and perinatal complications are found to be risk factors for the development of eating disorders in offspring, this poses intergenerational patterns of eating disorders or a so-called 'cycle of risk' (Bulik, Reba, Siega-Riz & Reichborn-Kjennerud, 2005) in which the occurrence of eating disorders is set to continue.

1.3. The current review

There has been a growing body of literature investigating perinatal outcomes in women with an active or historical eating disorder. Within the eating disorder population, pregnancy has been associated with changes in eating disorder symptomology (Mitchell-Gieleghem, Mittelstaedt, & Bulik, 2002; Newton &

Chizawsky, 2006) as well as postnatal depression (Mazzeo et al., 2006) in mothers. A number of articles have also set out to highlight and summarise the vast range of physical complications that can occur in women with eating disorders who become pregnant and their offspring (Anderson & Ryan, 2009; Cardwell, 2013; Hoffman, Zerwas & Bulik, 2011; Lowes, Kopeika, Micali & Ash, 2012). To date, the only paper to systematically review the maternal eating disorder-perinatal outcome literature has focussed on the impact of anorexia nervosa on low birth weight in offspring (Solmi, Sallis, Stahl, Treasure, & Micali, 2014). No reviews have yet investigated the *maternal health* impact of EDs for women during pregnancy and labour. Consequently, the objective of the present review was to determine whether having an active or historical eating disorder adversely affects perinatal complications for pregnant women. It is expected that mothers with remissive or active eating disorders will experience more perinatal complications than those who do not have remissive or active eating disorders.

2.0 METHOD

2.1 Search strategy

Four electronic databases were searched (PsycInfo, MEDLINE, Web of Science and CINAHL). Searches dated from the beginning of the database archives, to the fourth week of March 2015.

Studies of eating disorders were identified by searching for 'eating disorders' OR 'anorexia nervosa' OR 'bulimia' OR 'binge eating disorder' OR 'purging (eating disorders)' were exploded. 'Anorexi*' OR 'bulimi*' OR 'eating disorder*' OR 'binge' OR 'binging' were also searched as key words.

The results of the above search were then combined with a search for the term 'pregnan*'. Finally these results were then combined with the search terms 'outcome*' OR 'complicat*'.

The searches were limited to only journal articles written in the English language. The search was also refined to studies conducted on humans. This yielded 1410 results.

2.2 Inclusion and exclusion criteria

The inclusion and exclusion criteria are detailed in Table 1 along with the number of studies that were excluded on the basis of each of the criterion. Studies that focussed on body mass index (BMI) or on nutritional aspects of pregnancy were excluded as they weren't necessarily indicative of disordered eating. Studies that investigated the impact of complications on offspring were excluded, for example, because the focus of this review is the health complications for mothers only.

Table 1. Initial inclusion and exclusion criteria following database searches.

Inclusion criteria	Number of papers removed
Research studies investigating whether active or historical eating disorders are risk factors for physical complications during pregnancy or labour (for mother) compared to unexposed or referent women.	-
Research studies published in journal articles in the English language	-
Exclusion criteria	
Studies not conducted on human beings	80
Non-empirical papers (such as editorials, commentaries or review papers)	88
Focus on substance use/misuse	197
Focus on eating behaviours and attitudes (not eating disorders (ED))	4
Focus on nutritional deficiencies or food cravings/ aversions not specific to ED	47
Focus on body mass index (BMI) or obesity (not ED)	11
Focus on other eating disorders (e.g. Pica)	20
Focus on any other disorder or illness not related to an eating disorder (e.g. diabetes or appendicitis as well as mental health difficulties such as schizophrenia)	76
Focus on pre-pregnancy, prenatal or postnatal issues (i.e. amenorrhea, fertility, unplanned pregnancy, postnatal depression, breastfeeding- not related to course of pregnancy)	179
Focus on attitudes to pregnancy/ tokophobia in women with ED	2
Focus on outcomes not related to physical health complications (e.g. mental health outcomes of pregnant women with ED, sex ratio, treatment of pregnant women with ED)	25
Focus on feeding of offspring (and difficulties feeding) by mothers with ED	7
Focus on the course of ED during and after pregnancy	14
Focus on the development of ED in offspring following complications in pregnancy or other maternal factors	25
Focus on (psychological) factors of offspring of mothers with ED	9
Case reports and studies including ≤7 participants	33
Total papers removed	817

The overall procedures for the selection of studies are described in Figure 1 below. After the removal of duplicates, 856 results remained. Titles and abstracts were screened according to the inclusion and exclusion criteria (see table 1). The remaining 39 full text papers were read and those meeting the inclusion criteria were selected for the current review. Studies were retained if they;

- Were published in the last 20 years (post 1995). This was because studies predating 1995 were found to have poor methodological quality, with small sample sizes and limited or no statistical analysis, for example. It was also

decided that it would be important to apply this cut-off due to changes in diagnosis and medical and psychological care for women in the perinatal period and for the treatment of people with eating disorders.

- Only studies that statistically compared physical adversities during pregnancy and labour (for mother) across eating disorder (ED) subtypes or between ED and unexposed women were included.
- Only papers in which an interpretation could be made on the impact of a generic or specific ED on outcome measures were retained.
- Papers investigating remissive and/or active EDs were included in the review.

Following this, the references of the remaining 15 papers were searched for additional articles of relevance. No additional papers were identified from reference searches of the selected papers or recent review papers.

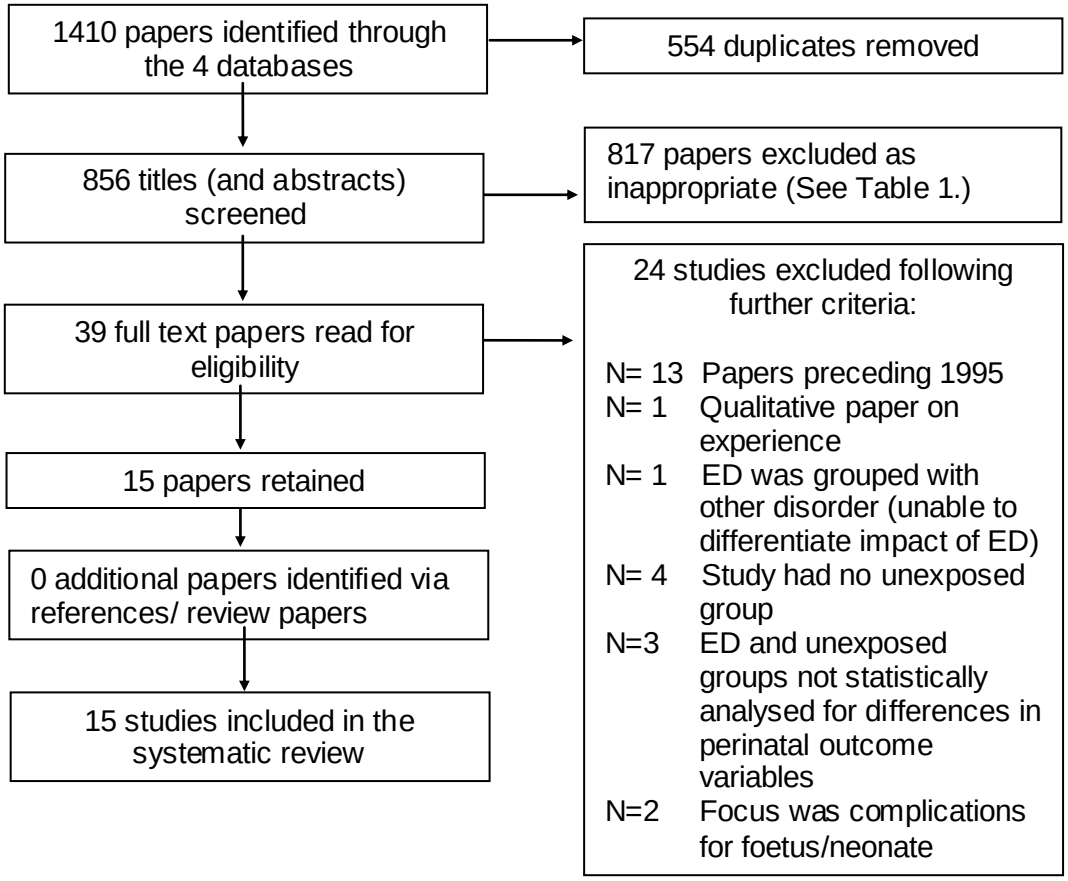


Figure 1. Flowchart of the literature searching

2.3 Evaluation of the quality of the literature

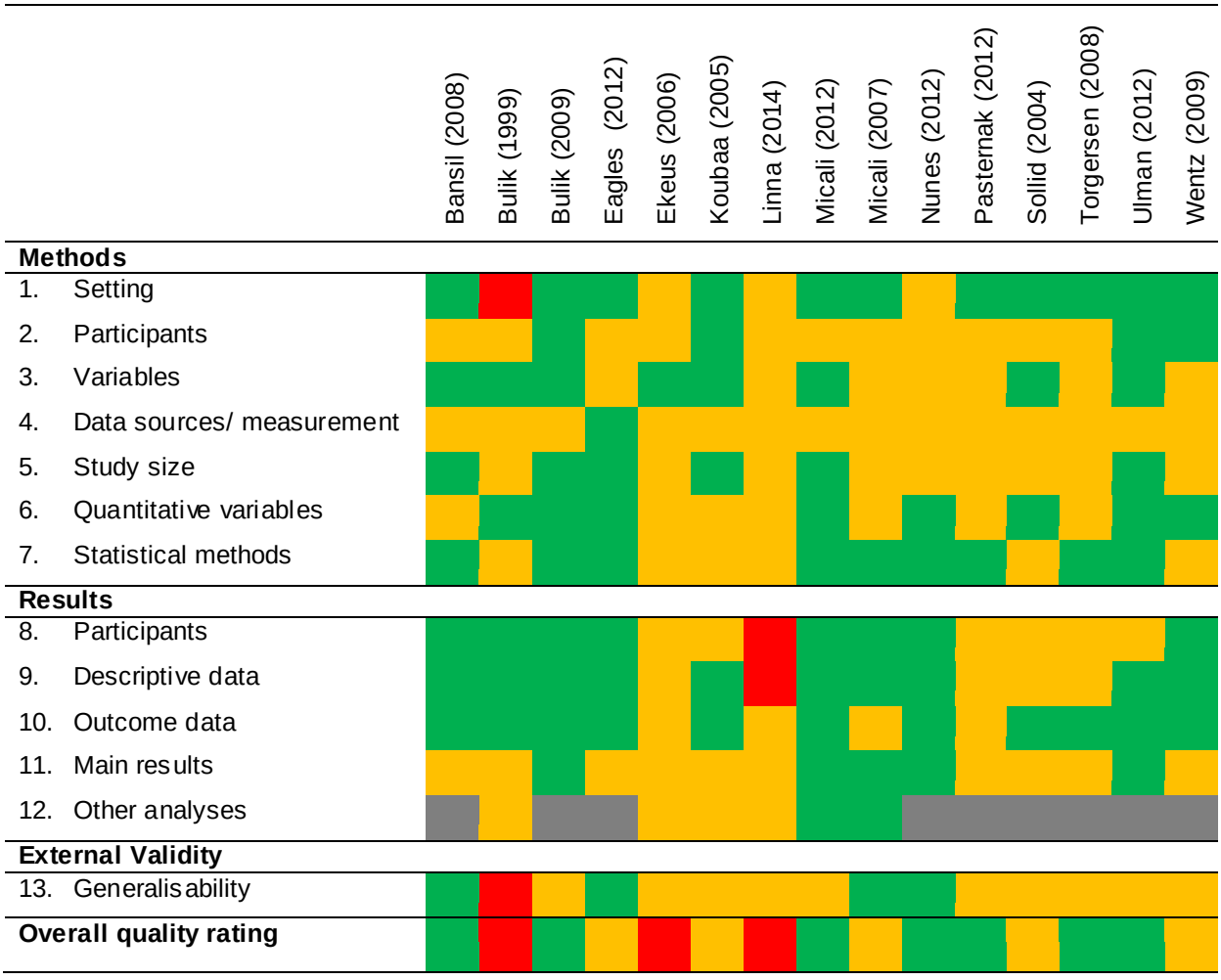
A final 15 articles were included in this review. Appendix 1 provides a summary of each of the study methodologies. Given that all of the papers were epidemiological in design, the STROBE Statement (see Appendix 2) devised by von Elm and colleagues (2007) was adapted and utilised as a means of consistently evaluating quality of the studies. Although the checklist was initially devised as a guide for authors on what and how to report their study, its structure was useful as it combined a review strategy for the three types of epidemiological, observational study designs under review here: cohort, case-control and cross-sectional designs. The STROBE Statement was adapted in order to accommodate the assessment of

the quality of the papers and the number of items was reduced from 22 to 13 (see Appendix 3) to eliminate aspects of a paper not related to quality of the methodology, for example sources of funding as well as sections covered by other sections, such as potential bias and limitations. Each criterion was assessed for each paper and allocated a colour representing the quality of that item. Good quality items were coded green, adequate items were coded amber and poor quality items were coded red. Based on how well each study met the quality criteria for each item, they were allocated an overall high, moderate or low quality rating. A summary of the methodological quality ratings of the 15 studies is shown in Table 2. Of the 15 studies 7 were considered high quality, 5 were considered moderate quality and 3 were considered of low quality. Two of those studies (Bulik et al., 1999; Linna et al., 2014) scored low ratings on elements of the quality review and an overall poor quality rating was issued because the limitations of those studies meant there was an inability to establish who the results applied to (because demographic data was not provided) and whether the conclusions drawn were reasonable (because important variables had not been accounted for). The claims made by the studies could not therefore be relied upon. The third study (Ekeus, Lindberg, Lindblad, and Hjern, 2006) scored moderately on all except one aspect of the quality review but it was felt that the volume of moderate ratings justified an overall low quality rating as the limitations over almost all areas cast serious doubt on the viability of the study overall.

When considering the importance of each point within the quality framework in contributing to the overall rating, it was felt that some points (such as the definition of the variables) would be more pertinent to the overall quality and interpretability of the study than others, such as how the quantitative variables were added to the analyses

(which included coding of continuous variables, for example). With this in mind, the overall quality of the study was considered, based on these weightings, rather than considering each quality point (or rating) as equal to each other. For example, while Eagles, Lee, Amalraj Raja, Millar and Bhattacharya (2012) included three amber ratings and was given an overall amber rating, Bansil et al. (2008) received four amber ratings but an overall green rating. This was because Eagles et al. (2006) scored poorer on a more heavily weighted quality point (definition of variables), while Bansil et al. (2008) scored poorer on lower weighted points (unclear source/ measurement and handling of quantitative variables).

Table 2. Methodological quality ratings for each article (by 1st author) included in the review



In terms of the methodology used to collect the required data, the distribution of green, amber and red ratings, indicate that the overall quality of the literature evaluated here is mixed. Most studies rated highly for setting factors because they were epidemiological studies utilising birth cohorts covering wide geographical areas. Those that coupled this data with a cross-reference of psychiatric case registers were able to eliminate the possibility of selection bias as they included the entire eligible population. While many studies utilised this methodology, others that utilised opportunistic clinical samples, such as from a caseload from the service within which an author worked (sometimes unclear about what this sample was i.e. inpatient or community samples), scored an amber rating, as is could not be established whether the sample was representative of the ED population. Other studies scoring amber ratings were those whose follow-up periods differed across individuals, making the likelihood of pregnancy and pregnancy complications different for each individual. The single study allocated a red (low quality) rating (Bulik et al., 1999) overlapped years of ED occurrence and years covering perinatal outcomes with 10 births occurring prior to the onset of ED that remained in the analysis.

The recruitment methods used to screen/ obtain a clinical and non-clinical sample revealed a range of limitations. While those rated highly for this criteria had accounted for co-morbidity across samples, matched cases and controls on important domains such as age and parity and had excluded multiple (e.g. twin) births, the less robust methods employed for other studies had failed to demonstrate exclusion of EDs from the control groups, had not stated how other ED subtypes not included in the study had been dealt with, and had not excluded women with more

than one pregnancy, counting them each time (therefore having a lack of independence between data sets).

The precision with which variables were selected and defined were wholly mixed. While no studies were rated poorly for this criterion, those that did not achieve a good rating typically grouped variables into wider categories (such as hypertensions and pre-eclampsia as one variable). Furthermore, moderately rated studies failed to report where and how definitions were established (i.e. if DSM criteria was utilised) and some did not provide definitions for variables included in analyses. While the quality of definitions across studies was mixed, the quality of tools used to measure variables was consistently moderate. While most studies consistently reported objective measures of outcomes or complications, the strength and strategy of measurement of EDs was variable and there was a lack of any consistency across studies for this. For example, studies that obtained a clinical sample from psychiatric registers retrospectively were unable to state how diagnoses of ED were made. Furthermore, such studies were less likely to utilise methods of excluding ED from controls. For studies taken from birth cohorts, many incorporated ED questions into a battery of assessments. However, the limitations of this were that ED samples were obtained based on retrospective, self-reports, thus leaving the sample vulnerable to recall bias.

The majority of the studies evaluated, obtained large samples of geographically defined birth cohort data, and these typically obtained a high quality rating because samples were non-selective and larger to enable better power to detect effects. A handful of studies sourced participants clinically by cases of ED and

then recruited a control group. These were comparatively much smaller in size with limited power to detect effects and were allocated an amber rating.

When adding variables into an analysis, studies were typically allocated a moderate rating because they merged separate variables into one overarching variable, unnecessarily categorised continuous variables or failed to incorporate potentially confounding variables that may have explained some of the variation in outcome occurrences across groups. To be rated highly in this criterion, studies typically included parity (where first pregnancies were not the inclusion criteria), maternal age, pre-pregnancy BMI and maternal weight gain in the analysis. Given the chosen variables for each study, most statistical analyses were appropriately chosen and justified in the majority of cases and were allocated a high quality rating. Those that did not meet full criteria and scored moderately were typically due to missing data unaccounted for, or because covariates previously chosen were not included and this was not justified.

The reported results for each study were then assessed for quality. Although there was variation in this across studies, each study rated reasonably consistently across different criteria for their statistical output. Those scoring highly for participant information gave detailed breakdowns of numbers of participants for each variable or set of analyses and provided numbers of missing data (if any) for each group separately (and possibly with reasons for this) with missing data being limited to ensure little impact upon overall conclusions. Those scoring moderately did not give detailed breakdowns of missing data or where they did, such data was sufficiently large to impact on the ability of the study to form representative conclusions. Only one paper scored poorly on this criterion (Linna et al., 2014). This was because there

was no indication of completeness or incompleteness of data and no differentiation in numbers for the two groups for each variable. Studies that scored highly for the reporting of descriptive data typically gave detailed breakdowns of numbers and percentages of demographic data for each group separately, and consequently conducted statistical analyses to establish any difference in these across groups that would need to be controlled for in the main analysis. For the single study rated poorly on this criterion (Linna et al., 2014) demographic data had not been provided for each group and no comparison could therefore be made to establish possible confounding variables. Without demographic breakdowns, the ability to form conclusions about who the results apply to is impossible.

The majority of studies typically achieved high ratings for their ability to meaningfully interpret outcome data. For the small proportion of those given amber ratings, this was because they had not provided number or percentages of occurrences of outcomes variables (or means and standard deviations (SD) where continuous data used) for each group. Studies were given moderate ratings if they reported no occurrences of particular outcome variables but still included them in the main analysis. When the main analysis data was investigated, a large number of studies rated moderately. This was because they had either failed to incorporate important confounding variables or where they had, they had only reported either adjusted or unadjusted models and so the influence of those variables could not be established. Other studies scoring moderately were given this rating as an indication to interpret significant results with caution. For example, where significance was found, on closer inspection confidence intervals were large, suggesting great variation, possibly explained by the impact of other variables left unaccounted for.

The only study to score poorly for the generalisability of the findings (Bulik et al., 1999) was given this rating due to the insufficient detail in the setting of the study. It was there impossible to establish who the study applied to within both the narrow study specific context but also within any wider context.

Table 3. Key findings of studies according to complication type

1st author (year)	Pregnancy complications	Delivery complications
Bansil (2008)	More anaemia in active ED after adjusting for age, insurance status, location, region. No differences found in UTIs or other infections during pregnancy. ED less likely to have hypertension (inc. pre-eclampsia).	Longer delivery hospitalisations for ED group. No differences between ED and unexposed for labour induction or c-section. More preterm labour in active ED.
Bulik (1999)	No differences between AN and unexposed for being confined to bed, pre-eclampsia or severe vomiting.	More unexposed women experienced uncomplicated deliveries than AN. More AN had caesareans and preterm delivery than unexposed. No differences between AN and unexposed for induced labour, forceps, breech, posterior presentation, retained placenta, postpartum haemorrhage and postpartum infection.
Bulik (2009)	No differences between BN or BED and unexposed women for placenta previa. No differences between BED and unexposed for HELLP syndrome. No differences between BN, EDNOS-P or BED and unexposed women for pre-eclampsia and GDM. There were insufficient numbers for statistical comparison for: Placenta previa in AN or EDNOS-P, HELLP syndrome in EDNOS-P, BN and AN, Pre-eclampsia in AN and GDM in AN.	No differences between AN, BN, BED or EDNOS-P subtypes and unexposed women for preterm births, non-vertex cephalic or slow progress. No differences between BN, EDNOS-P, BED and unexposed women for anaesthetics during labour. BED had more caesarean sections after controlling for all covariates except pre-pregnancy BMI and gestational weight gain, when they became no longer significant. No difference for AN, BN, EDNOS-P. AN more likely to have epidural after all covariates added. BED more likely to have epidural after all covariates added except pre-pregnancy BMI and gestational weight gain when it lost significance. No difference for BN or EDNOS-P. BN were more likely to have assistance at vaginal breech presentation after including all covariates. No differences found for BED or EDNOS-P. There were insufficient numbers for statistical comparison for assistance at vaginal breech presentation in AN and use of anaesthetics in AN.
Eagles (2012)	No differences between AN and unexposed group in adjusted and unadjusted models for pregnancy-induced hypertension (gestational hypertension/pre-eclampsia/other). AN more likely to have antepartum haemorrhage before and after adjustments.	No differences between AN and unexposed group in adjusted and unadjusted models for malpresentation, postpartum blood loss, induction of labour, preterm labour and caesarean section. AN had less instrumental deliveries but not after adjustment.
Ekeus (2006)	No differences were found before or after adjustments for pre-eclampsia or PROM.	No differences were found for babies born in week 32, babies born before 37 weeks or after 41 weeks, foetal distress, or forceps delivery. Instrumental delivery (caesarean section, forceps, vacuum extraction)

1st author (year)	Pregnancy complications	Delivery complications
		was less common in AN after adjustments. No differences in use of analgesia, except pudental blocks were used less by AN though not after adjustment for year of birth.
Koubaa (2005)	No differences for ED and non-ED for pregnancy-induced hypertension (including pre-eclampsia). ED had more anaemia and HG. When subgroups investigated, AN had higher blood pressure than non-ED. There were no occurrences of GDM to statistically test differences.	No differences for prematurity, epidural, induction of labour, vacuum extraction, forceps, breech presentation, planned caesarean section, acute caesarean section and postpartum bleeding.
Linna (2014)		No differences for premature birth for AN or BN, although very premature babies more common in AN. Unable to compare BED due to no occurrences for premature birth or very premature birth.
Micali (2012)	No difference between the presence of any pregnancy complications, pregnancy-induced hypertension (including pre-eclampsia and HELLP syndrome) or hospitalisation during pregnancy across groups. Differences in GDM could not be calculated due to no/ very low occurrences.	No difference for prematurity, instrumental delivery, elective caesarean or emergency caesarean. Differences in unknown caesarean sections or breech presentations could not be calculated due to no/ very low occurrences.
Micali (2007)		No differences across the groups for prematurity before and after adjustments.
Nunes (2012)		No differences for prematurity or caesarean section before or after adjustments.
Pasternak (2012)	No differences for hypertensive disorders, GDM, placenta previa, olioghydramnion, polyhydramnion or PROM.	ED more likely to have preterm birth and caesarean delivery after controlling for parity and maternal age. No differences for labour induction, malpresentation, slow progress of labour for first or second stage, placental abruption or postpartum haemorrhage.
Sollid (2004)		ED more likely to have preterm birth before and after adjusting for woman's age, parity, marital status and sex of the child.
Torgersen (2008)	BN-purge and BN-other more pregnancy-related nausea and nausea with vomiting in first month of pregnancy. No difference for BN-non-purge or EDNOS-P for nausea. EDNOS-P more nausea with vomiting but not for BN-non-purge.	

1st author (year)	Pregnancy complications	Delivery complications
	No differences for HG after controlling for time of gestation.	
Ulman (2012)	BED groups had more sleep problems before and after adjustments, across all the time points with no interaction between groups across time points. BED groups more likely to have more than 10 hours sleep than 6-9 hours sleep compared to unexposed. After adjustments, the difference was lost for the BED before and during pregnancy and the BED remission group but remained for the BED during pregnancy group. BED groups more likely to report less than 6 hours sleep rather than 6-9 hours sleep compared to unexposed. After adjustments, difference only remained for BED during pregnancy group.	
Wentz (2009)	No differences in numbers of AN and unexposed women with no complications during pregnancy.	No differences for planned caesarean delivery, numbers of women with no complications during delivery or mean gestational weeks at delivery between AN and unexposed women.

Note: Eating disorder (ED); binge-eating disorder (BED); anorexia nervosa (AN); bulimia nervosa (BN); Eating disorder not otherwise specified- purging subtype (EDNOS-P); urinary tract infection (UTI); hyperemesis gravidarum (HG); gestational diabetes mellitus (GDM); premature rupture of membranes (PROM); haemolysis, elevated liver enzymes and low platelet count (HELLP syndrome).

3.0 RESULTS

Fifteen studies, published between 1995 and 2015, were included in this review. A summary of the main results from these studies is provided in Table 3. As has been observed in previous summary articles, outcome results were divided into 2 categories or stages of the perinatal period; pregnancy complications and delivery complications. Where possible, these have been sub-divided according to ED subtype.

3.1 Pregnancy complications

Eleven studies compared pregnancy complications in women with and without EDs. Of those 11, a broad range of complications were assessed. The most commonly assessed category of complications (n=8) were hypertension related disorders (including blood pressure levels, pure hypertension, pre-eclampsia, and/ or haemolysis, elevated liver enzymes and low platelet count (HELLP syndrome)). This was followed by sickness related complications (n=3) including hyperemesis gravidarum (HG), severe vomiting, nausea and nausea with vomiting. Gestational diabetes mellitus (GDM), anaemia, premature rupture of the membranes (PROM) and placenta previa were examined by 2 studies. Urinary tract infections (UTIs) or other infections during pregnancy, sleep difficulties, olioghydramnion, polyhydramnion and antepartum haemorrhage were assessed in only single studies. Finally, 3 studies looked at broader representations of pregnancy complications

including being confined to bed, hospitalised during pregnancy, or the presence or absence of *any* pregnancy related complication.

Of the 11 studies comparing pregnancy complications in those with and without ED, 7 looked at the effects of AN on pregnancy complications, 3 looked at BN, 2 at BED, 2 at EDNOS-P, while 2 combined ED subtypes (AN, BN and EDNOS) to create a composite variable. Six of these papers were categorised as high quality, 3 as medium quality and 2 as low quality papers.

3.1.1 Studies investigating anorexia nervosa

The most commonly reported pregnancy complications compared for women with and without AN were those related to pregnancy-induced hypertensive disorders. Complications included here were gestational hypertension, pre-eclampsia and HELLP. On the whole no differences were found for these kind of pregnancy-related complications (Bulik et al., 1999; Eagles et al., 2012; Ekeus et al. 2006; Micali et al., 2012). However, there were some discrepancies in the findings. While Bansil et al. (2008) reported that those with AN were *less* likely to have a hypertensive disorder in a broad, diagnostic sense, Koubaa, Hallstrom, Lindholm, and Hirschberg (2005) found blood pressure was generally higher when measured on a continuous scale. Given the method of measurement for hypertension-related disorders varied to some degree, generalisation of conclusions made from these studies are tentative, though results do trend towards no differences.

For the remainder of pregnancy complications measured in those with AN, no two studies measured the same outcome. Single studies reported no differences between women with and without AN for UTIs or other infections (Bansil et al., 2008),

being confined to bed (Bulik et al., 1999) or hospitalised (Micali et al., 2012) during pregnancy, experiencing severe vomiting (Bulik et al., 1999) or PROM (Ekeus et al., 2006). The presence of AN was positively associated with anaemia (Bansil et al. (2008) and antepartum haemorrhage (Eagles et al., 2012) with more occurrences in women with AN following relevant adjustments in those single studies. Given the variation in the quality of these single studies as well as the measurement of pregnancy complications, conclusions cannot yet be made. However, two further studies, one of which was of good quality (Micali et al., 2012; Wentz, Gillberg, Anckarsater, Gillberg & Rastam, 2009), found no differences in the presence or absence of *any* pregnancy complications globally and their findings go some way to consolidate the general lack of findings for most of the specific complications described above.

3.1.2 Studies investigating bulimia nervosa

Only 3 studies have so far investigated the impact of BN on pregnancy complications. While the complications examined have been varied, the overall conclusions have been largely the same. Specifically, no difference between women with BN and unexposed women have been found for GDM (Micali et al., 2012), placenta previa (Bulik et al., 2009), pregnancy-induced hypertension (including pre-eclampsia, and HELLP; Bulik et al., 2009; Micali et al., 2012) or broader, undefined measures including hospitalisations during pregnancy or the presence of any complications during pregnancy (Micali et al., 2012). The only good quality study found differences in BN groups for a pregnancy complication was Torgersen et al. (2008). After accounting for the effects of ED purging behaviours (i.e. they

differentiated between pregnancy related nausea and vomiting and ED related nausea and vomiting), they reported more pregnancy-related nausea and nausea with vomiting in early stages of pregnancy in the subgroups BN-purge and BN-other. No differences were found, however, for the BN-non-purge subgroup for pregnancy-related nausea or nausea with vomiting, or in any of the BN subgroups for the potentially more problematic complication of HG after controlling for time of gestation.

3.1.3 Studies investigating binge eating disorder

Studies investigating the impact of BED on pregnancy complications are, to date, limited to two. While Bulik et al. (2009) found no differences between women with BED and unexposed women for occurrences of placenta previa, HELLP, pre-eclampsia or GDM, Ulman and colleagues (2012) found women with active or remissive BE symptoms reported more sleep difficulties throughout their pregnancy. Although both studies had high quality methodology, more studies are required to add weight to the currently limited findings.

3.1.4 Studies investigating EDNOS-P

As with the BED literature, there have been only two studies that have looked into pregnancy complications specifically in women with EDNOS-P. Types of complications examined have therefore been limited and there has been no overlap between those measured by the two studies. While Bulik et al. (2009) found no differences for GDM and pre-eclampsia, Torgersen et al. (2008) investigated pregnancy related sickness. While no differences were found between women with and without EDNOS-P for HG or less severe nausea, those with EDNOS-P were

found to experience more nausea with vomiting. While both studies were of a high methodological standard, the findings for the limited outcomes measured should not be relied upon to form conclusions at this early stage.

3.1.5 Studies investigating eating disorders as a composite variable

Two further studies investigated EDs as a single broad construct that included AN, BN and EDNOS combined. As with the studies investigating specific ED subgroups, no differences were found between ED and unexposed groups for GDM, placenta previa, PROM or pregnancy-induced hypertensive disorders (Koubaa et al., 2005; Pasternak et al., 2012). Also in line with the albeit limited findings to date, were the higher levels of anaemia in women with ED compared to those without (Koubaa et al., 2005). The only conflicting findings when looking at ED more generally were the higher rates of HG in women with ED compared to those without, where these had not been found in studies of BN or EDNOS-P specifically (Torgersen et al., 2008).

Pasternak et al. (2012) were the only authors to examine occurrences of olioghydramnion and polyhydramnion specifically and no differences were found between ED and non-ED women.

3.1.6 Summary of findings for pregnancy complications

Studies reporting on the impact of specific EDs on pregnancy complications were limited, making conclusions about their impact on particular outcomes difficult. However, the findings were predominantly indicative of no differences in the occurrence of pregnancy complications (GDM, PROM, placenta previa, UTIs or other

infections, olioghydramnion, polyhydramnion, confined to bed, hospitalised during pregnancy or the presence or absence of *any* pregnancy related complication) for women with EDs compared to those without. A minority of studies found greater number of antepartum haemorrhage, anaemia, and sleep difficulties in women with EDs. Studies investigating hypertensive disorders or sickness related disorders were slightly more inconsistent, although evidence showed a tendency towards no differences, having weighed up the quality of the studies included.

In sum, although most studies demonstrate no differences, there is some indication of possibly higher occurrences in certain complications. More studies would be required to consolidate the findings across all complications and across all ED subtypes.

3.2 Delivery complications

Thirteen studies included delivery complications when comparing ED and non-ED women in the perinatal period. Twelve considered prematurity as a complication of delivery, while one also compared post-term delivery rates. Nine investigated the use of assisted delivery, including the use of analgesics or anaesthetics (such as epidural) or instrumental delivery (including caesarean section, forceps, vacuum, induced labour). Malpresentation at labour was also investigated by 5 of the studies. This included breech, posterior and broader non-vertex cephalic presentations. Slow progress was also included by 2 studies. Retained placenta, placental abruption and postpartum bleeding or haemorrhage were features of 1, 1 and 4 studies respectively. Two studies investigated delivery complications in the broader sense of

the presence of any complications during delivery, while one further study measured length of delivery hospitalisation as an indication of complicated delivery.

Of the 13 studies comparing delivery complications in those with and without ED, 4 looked at the effects of AN alone on delivery complications and 1 at BED alone. Four studies looked at separate subtypes of ED, while a further 4 combined ED subtypes. Five of these papers were considered high quality, 5 as medium quality and 3 as low quality papers.

3.2.1 Studies investigating anorexia nervosa

In women with AN, most studies found consistently no differences in incidents of premature delivery at <37 weeks gestation compared to unexposed women (Bulik et al, 2009; Eagles et al., 2012; Ekeus et al., 2006; Linna et al., 2014; Micali et al., 2012; Micali, Simonoff, & Treasure, 2007). One low quality paper, Bulik et al. (1999) found more occurrences of prematurity in AN though the definition for prematurity was not clear for this study. Although Linna et al. (2014) found no differences in prematurity, when this was further investigated AN were found to have more very premature deliveries (<28 weeks gestation) than unexposed women. However no further studies have investigated this outcome measure. Similarly, no differences were found in the only study investigating post-term (>41 weeks gestation) deliveries (Ekeus et al., 2006).

Assisted delivery in AN was measured in several ways by six studies. Studies found no differences in rates of caesarean sections for women with AN compared to unexposed women (Bulik et al., 2009; Eagles et al., 2012; Micali et al., 2012; Wentz et al., 2009). This was irrespective of category of caesarean sections (elective or

emergency) where differentiated. In contrast, a single, low quality study found women with AN were more likely to have caesarean sections than unexposed women. For those studies that investigated other forms of instrumental delivery, including forceps and vacuum extraction (Bulik et al., 1999; Eagles et al., 2012; Ekeus et al., 2006; Micali et al., 2012), results were mixed though the more methodologically robust studies cited no differences after adjusting for relevant covariates. Assisted delivery through induction of labour was reported by two studies, both concluding there were no differences between AN and unexposed women. Limited conclusions can be made for differences in analgesia or anaesthetic in AN as these only have a single study evidence base, though no differences have yet been found for analgesia (Ekeus et al., 2006) while epidural has so far been found to be more common in women with AN, following adjustments, according to one high quality study (Bulik et al., 2012).

Although malpresentation during labour has been scarcely utilised as an outcome measure when comparing delivery complications in women with and without AN, studies so far concur that there are no differences, for example, with breech, posterior or broader non-vertex cephalic presentations (Bulik et al., 1999; Bulik et al., 2009; Eagles et al., 2012). Similarly, no differences have been found for complications immediately following delivery such as haemorrhage (Bulik et al., 1999; Eagles et al., 2012), or retained placenta (Bulik et al., 1999). Studies supporting this conclusion, however, are so far limited.

Bulik et al. (1999) and Wentz et al. (2009) grouped all the categories of delivery complications aforementioned to establish whether there were any differences between women experiencing *uncomplicated* deliveries. While Wentz et

al. (2009) found no differences, Bulik and colleagues (1999) suggested more unexposed women experienced uncomplicated deliveries, though this was a poor quality study. Both studies investigated a wide range of outcome measures which they incorporated into this broader outcome. Similarly, while Ekeus et al. (2006) argued no differences in numbers of pregnancies with foetal distress, Micali et al. (2012) found higher rates of *suspected* foetal distress in women with AN. Consequently, little can be deducted from these inconsistent results regarding these particular ways of measuring complicated delivery outcomes. Finally, Bulik et al. (2009) were the only authors to investigate slow progress. While no differences were found in this high quality study for women with AN compared to unexposed women, firm conclusions should not be based on single study findings.

3.2.2. Studies investigating Bulimia Nervosa

As with the outcomes found in AN, no differences emerged across the studies for prematurity in women with BN compared to unexposed women (Bulik et al., 2009; Linna et al., 2014; Micali et al., 2007; Micali et al., 2012) before or after adjustments, where they were made. For the two high quality studies addressing additional delivery outcomes for this population group (Bulik et al., 2009; Micali et al., 2012), no differences were found for instrumental delivery (including elective and emergency caesarean sections). While a further lack of differences were found for assisted delivery (namely anaesthetics or epidural) and slow progress, mal-presentation outcomes during labour were mixed in Bulik and colleagues' (2009) study. No differences were found for non-vertex cephalic presentations, while more women with BN had assisted vaginal breech deliveries after accounting for relevant covariates.

Micali et al. (2012) also reported no differences for suspected foetal distress for women with BN compared to unexposed women. Again conclusions should be tentative for those findings drawn from relatively few sources.

3.2.3 Studies investigating Binge Eating Disorder

While only two studies have explored delivery complications in BED (Bulik et al., 2009; Nunes, Pinheiro, Camey & Schmidt, 2012), both surmise that there are no significant differences for rates of prematurity or incidences of caesarean sections after making relevant adjustments, most pertinently for pre-pregnancy BMI and gestational weight gain. While findings are limited, the methodological quality of the two is sound enough to indicate that there may be foundation to these findings, though generalising should be tentative. Further to these outcomes, Bulik et al. (2009) measured additional delivery complications also. Women with BED were no more or less likely to utilise anaesthetic or an epidural (after controlling for pre-pregnancy BMI and gestational weight gain), non-vertex cephalic or assistance at breech presentation nor have a slow progress labour. While the design of the study merits a high quality rating, little should be taken from a single study finding, and further quality studies are needed consolidate these findings for women with BED.

3.2.4 Studies investigating EDNOS-P

Bulik et al. (2009) has been the only study to date to report separately on delivery outcomes in women with EDNOS-P. The authors found no significant differences across the delivery outcome measures, namely preterm births, assisted or instrumental deliveries (use of anaesthetics, epidural or incidences of caesarean

section), malpresentation (non-vertex cephalic and assistance at vaginal breech presentation) and slow progress of labour. As stated in the previous section, more studies are required for EDNOS-P delivery outcomes to enable firmer conclusions to be made.

3.2.5 Studies investigating eating disorders as a composite variable

While the majority (69%) of studies differentiated perinatal complications for specific eating disorder subtypes, the remaining 31% only reported outcomes for a composite eating disorder variable. Two studies (Micali et al., 2007; Micali et al., 2012) looked at specific subtypes, but also looked at individuals with a history of both AN and BN as a separate group.

All six studies with a composite ED group utilised prematurity (<37 weeks gestation) as an outcome measure for adverse perinatal complications. While specific subtypes of ED found consistently no differences between those and unexposed groups, results for the composite groups were largely mixed. While Bansil et al. (2008), Pasternak et al. (2012) and Sollid, Wisborg, Hjort and Secher (2004) found greater numbers of premature deliveries in women with ED, Koubaa et al. (2005), Micali et al. (2007) and Micali et al. (2012) did not. When taking the quality of the papers into consideration, those with more robust methodology were those who found significantly more prematurity in the ED groups.

In contrast, those that captured assisted deliveries in various forms found typically no differences for instrumental deliveries (including vacuum extraction and forceps; Koubaa et al., 2005; Micali et al., 2012), induction of labour (Bansil et al., 2008; Koubaa et al., 2005; Pasternak et al., 2012) or use of epidural (Koubaa et al.,

2005). Although Pasternak et al., (2012) found more caesarean sections (elective or emergency) in women with ED after controlling for maternal age and parity, the consensus was that there were no differences (Bansil et al., 2008; Koubaa et al., 2005 Micali et al., 2012).

In line with the lack of differences for assisted deliveries, no differences were found for malpresentation at delivery (Koubaa et al., 2005; Pasternak et al., 2012), slow progress of labour (Pasternak et al., 2012), placental abruption (Pasternak et al., 2012) or suspected foetal distress (Micali et al., 2012). These negative findings would make theoretical sense given the relationship between these complications and the need for assistance at delivery. Furthermore, no differences were found for postpartum haemorrhage across ED and unexposed groups (Koubaa et al., 2005; Pasternak et al., 2012). While the majority of delivery complications assessed indicated no differences across groups except prematurity, it should be noted that Bansil et al. (2012) reported longer delivery hospitalisations in women with ED than those without. This may be attributable to their finding of higher rates of prematurity in the same population. However, further investigations are required to strengthen these findings as well as to establish whether premature delivery is the reason for longer stays given no greater risk for other complications has been found or whether additional factors not yet known account for this difference.

3.2.6 Summary of findings for delivery complications

Given the lack of consistent findings for the impact of specific ED subtypes on delivery complications, even within the better quality papers, it is difficult to form a conclusive interpretation. The dearth of papers investigating specific delivery

complications among specific EDs points to a need for consistently measured studies in this area.

4.0 DISCUSSION

While the 15 studies reviewed investigated multiple measures of pregnancy and delivery complications, there seemed to be little evidence to suggest *overall* that women with EDs were at greater risk of either type of complication. Suggestions that women with ED are at increased risk of particular complications were limited to single studies. This is in contrast to the conclusions made by several non-systematic review papers (e.g. Cardwell, 2013; Lowes et al., 2012; Watson et al., 2014). This may be because they interpreted the conclusions made by the studies, some of which had over-interpreted 'trends', as meaningful and reliable positive findings without critiquing the evidence (e.g. Koubaa et al., 2005; Linna et al., 2014). Conclusions were also drawn from only one or two study findings. As a result, the non-systematic review papers have made recommendations for policy and procedures on findings that are not statistically accurate or robust. For example, recommendations have been made for early screening of ED in prenatal clinics. This would facilitate closer monitoring of pregnant women with a remissive or active ED. This in itself could cause unnecessary risks to be taken. If there is a perception that women with ED are more vulnerable during pregnancy and labour, steps may be taken proactively to reduce certain risks, while consequently increasing others. For example, a pregnant woman who has a known ED, may be perceived as less physically able to birth a child naturally, and so may decide a caesarean section is required, even though it may not actually be necessary in that case. While caesarean sections are themselves considered a complication, this is because risks of other medical complications (such as haemorrhage) are heightened. A further consequence of

screening for a history of ED is that the mother is alerted to potential pregnancy and delivery complications (where there is no evidence for this). Expectant mothers may then carry additional guilt or shame about their ED history and the impact this may have on them or their child. Anxieties may also rise at the knowledge of these increased risks, which will in turn increase their levels of cortisol, which will be exposed to the foetus. This in itself is known to increase pregnancy and delivery complications such as preterm birth (Mulder et al., 2002).

4.1 Limitations

Some tentative conclusions can be made on the basis of the findings in the aforementioned studies having taken into account the quality of the individual studies. While no differences have typically been found, meaningful comparisons across studies are inhibited by several wide-reaching methodological flaws or inconsistencies.

4.1.1 Sample size

While many of the studies utilised national birth cohort samples (e.g. Eagles et al., 2012; Ekeus et al., 2006), facilitating a large sample with which to compare ED and unexposed women, the prevalence of ED within the population is typically low, and when ED samples are grouped into subtypes, this further reduces the sample size, and increases the disparity between exposed and unexposed groups. Those with more robust methodology account for this by investigating risk ratios (e.g. Eagles et al., 2012; Nunes et al., 2012). However, the occurrence with which

complications occur in either group is relatively low. As a consequence, there may be limited power to be able to detect if there are any potentially small effects between the groups, despite overall large samples. Therefore those studies that have not found differences may be missing small, but present, effects of eating disorders on these outcomes. While national cohorts are large, it may be that a meta-analysis may provide a more accurate conclusion of the collection of data, though there is yet to be sufficient studies investigating one specific outcome that would facilitate this.

4.1.2 Measures

The method in which independent (ED) and dependent (complication) variables have been measured has varied widely. Given the time frame with which studies have been included in the review (1995-2015), and the even broader time frame with which data was collected (some dating back to 1965), the diagnostic classifications (e.g. the DSM editions) alone have changed several times. While there is large overlap in the evolving diagnostic criteria, difficulties in comparing subtypes with any certainty remain. Within that, the comparability of the measures and methods used to allocate individuals to case or control groups were also wide-ranging. While some studies cross-referenced pregnancies with psychiatric registers (e.g. Eagles et al., 2012; Ekeus et al., 2006; Pasternak et al., 2012), others utilised retrospective self-report tools (e.g. Bulik et al., 2009; Micali et al., 2012). Both have their advantages and disadvantages. While retrospective self-reports can lead to recall bias, the use of psychiatric registers typically meant the EDs were not excluded from the control group. The inherent difficulty with comparing studies using such

different methods of measurement is that there is potentially great difference in the degrees of severity of the women categorised into the ED group.

4.1.3 Participants

Given that many of the measures and methods used to differentiate ED and unexposed participants were recruited on the basis of historical data (e.g. Pasternak et al., 2012; Sollid et al., 2004), with only a few assessing ED at the time of or just prior to pregnancy (e.g. Bansil et al., 2008; Bulik et al., 2009) there was little reference to the status of the ED at the time of conception and pregnancy. Morgan, Lacey and Chung (2006) would suggest that those with active BN at the time of pregnancy were at higher risk of complications such as preterm delivery than those who were recovered from BN at the time of pregnancy. Such a claim would suggest there may be an inherent limitation in the comparability of studies assessing different statuses. Further evidence for this may lie in the occurrence of the pregnancy itself. For women with an ongoing severe ED, such as AN, who characteristically have a low BMI, their ability to conceive is likely to be impaired. Consequently, for the AN participants, the occurrence of a pregnancy may suggest that they have recovered to a sufficient degree to enable this and that their bodies are healthy enough to sustain a foetus.

4.1.4 Generalisability

Additionally, the wide range of countries in which the studies were conducted may impact on the ability to generalise the findings overall. One explanation may be the difference in medical practice across countries, for example.

In addition to this, the time in which the data was collected may also be affected by the state of the medical procedures in place at that time, and more recent studies may well report fewer occurrences as a result of improvements in recognition and treatment of ED as well as the outcomes, and early interventions for this may already be occurring more than they did 20 years ago.

Considering the limitations aforementioned, it would be important to consider methods of obtaining a more robust set of interpretable results. Future research should consider using national cohorts to obtain large samples (as many already have) but also utilising consistent measures of eating disorder symptomology. For example more studies should perhaps ascertain eating disorder status (active or remissive) at the time of pregnancy. Similarly, to add weight to the current dearth of studies, research should make attempts to measure outcomes consistently as at present only a handful, at best, measure the same outcomes. For example, more studies investigating caesarean sections would be particularly helpful, given the risk that this can pose further complications.

While there are certainly methodological limitations within and between studies, there is an overall consistency to the lack of findings. There may be a perception that women with ED are more physically vulnerable to complications during pregnancy, but the evidence here does not support that. It may in fact be that there are small differences not detected by the studies here, but if that is the case, the differences are smaller than we have previously been led to believe.

5.0 CONCLUSION

While 15 studies have investigated pregnancy and delivery complications in mothers with EDs, the variation in which such complications have been measured along with the variations in the quality of those studies prevents firm conclusions about whether pregnant women with ED are at increased risk during pregnancy and delivery. While most studies indicate that there are no differences in prevalence of maternal pregnancy and delivery complications, a small number of individual papers indicate an increased rate in women with eating disorders. In light of this, more population-based birth cohort studies that utilise consistent and clear methods of measurement of ED and pregnancy and delivery complications are required. While AN seems to be the most widely investigated ED subtype, the literature is still lacking in depth and quality and studies are therefore needed across all ED subtypes. Given the potential impact of recency of the ED on outcomes, this should also be investigated further, as should the inter-related impact of pre-pregnancy BMI and gestational weight gain already accounted for in the better quality studies already carried out. It is important to establish more reliably if any differences do or do not exist, though given what has so far been found, any new differences that emerge are likely to be small, indicating that complications during pregnancy and delivery in women with ED are less of a concern than previously hypothesised.

REFERENCES

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5* (5th Ed.). Arlington, VA: American Psychiatric Publishing.

Andersen, A. E.., & Ryan, G. L. (2009). Eating disorders in the obstetric and gynecologic patient population. *Obstetrics & Gynecology*, *114*(6), 1353-1367.

*Bansil, P., Kuklina, E. V., Whiteman, M. K., Kourtis, A. P., Posner, S. F., Johnson, C. H., & Jamieson, D. J. (2008). Eating disorders among delivery hospitalizations: Prevalence and outcomes. *Journal of Women's Health, 17*(9), 1523-1528.

Brinch, M., Isager, T., & Tolstrup, K. (1988). Anorexia nervosa and motherhood: reproduction pattern and mothering behavior of 50 women. *Acta Psychiatrica Scandinavia*, *77*(5), 611-617.

Bulik, C. M., Reba, L., Siega-Riz, A. M., & Reichborn-Kjennerud, T. (2005). Anorexia nervosa: definition, epidemiology, and cycle of risk. *International Journal of Eating Disorders*, *37*(S1), S2-S9.

*Bulik, C. M., Sullivan, P. F., Fear, J. L., Pickering, A., Dawn, A., & McCullin, M. (1999). Fertility and reproduction in women with anorexia nervosa: A controlled study. *Journal of Clinical Psychiatry, 60*(2), 130-135.

*Bulik, C. M., von Holle, A., Siega-Riz, A. M., Torgersen, L., Lie, K. K., Hamer, R. M., Berg, C. K., Sullivan, P., & Reichborn-Kjennerud, T. (2009). Birth outcomes in women with eating disorders in the Norwegian mother and child cohort study (MoBa). *International Journal of Eating Disorders, 42*(1), 9-18.

Cardwell, M. S. (2013). Eating disorders during pregnancy. *Obstetrical & gynecological survey*, *68*(4), 312-323.

Chesney, E., Goodwin, G. M., & Fazel, S. (2014). Risks of all-cause and suicide mortality in mental disorders: a meta-review. *World Psychiatry*, *13*(2), 153–160.

Cnattingius, S., Hultman, C. M., Dahl, M., & Sparén, P. (1999). Very preterm birth, birth trauma, and the risk of anorexia nervosa among girls. *Archives of General Psychiatry*, *56*(7), 634-638.

Conti, J., Abraham, S., & Taylor, A. (1998). Eating behavior and pregnancy outcome. *Journal of Psychosomatic Research, 44*(3-4), 465-477.

Crow, S. J., Keel, P. K., Thuras, P., & Mitchell, J. E. (2004). Bulimia symptoms and other risk behaviors during pregnancy in women with bulimia nervosa. *International Journal of Eating Disorders*, *36*(2), 220-223.

Crow, S. J., Peterson, C. B., Swanson, S. A., Raymond, N. C., Specker, S., Eckert, E. D., & Mitchell, J. E. (2009). Increased mortality in bulimia nervosa and other eating disorders. *The American journal of psychiatry*, *166*(12), 1342-1346.

*Eagles, J. M., Lee, A. J., Amalraj Raja, E., Millar, H. R., & Bhattacharya, S. (2012). Pregnancy outcomes of women with and without a history of anorexia nervosa. *Psychological Medicine, 42*(12), 2651-2660.

*Ekeus, C., Lindberg, L., Lindblad, F., & Hjern, A. (2006). Birth outcomes and pregnancy complications in women with a history of anorexia nervosa. *BJOG: An International Journal of Obstetrics & Gynaecology, 113*(8), 925-929.

Favaro, A., Tenconi, E., Bosello, R., Degortes, D., & Santonastaso, P. (2011). Perinatal complications in unaffected sisters of anorexia nervosa patients: testing a covariation model between genetic and environmental factors. *European archives of psychiatry and clinical neuroscience*, *261*(6), 391-396.

Favaro, A., Tenconi, E., & Santonastaso, P. (2006). Perinatal factors and the risk of developing anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, *63*(1), 82-88.

Favaro, A., Tenconi, E., & Santonastaso, P. (2008). The relationship between obstetric complications and temperament in eating disorders: a mediation hypothesis. *Psychosomatic medicine*, *70*(3), 372-377.

Hack, M., Flannery, D. J., Schluchter, M., Cartar, L., Borawski, E., & Klein, N. (2002). Outcomes in young adulthood for very-low-birth-weight infants. *New England Journal of Medicine*, *346*(3), 149-157.

Hadley, S., & Walsh, B. (2003). Gastrointestinal disturbances in anorexia nervosa and bulimia nervosa. *Current Drug Targets-CNS & Neurological Disorders*, *2*(1), 1-9.

Herpertz, S., Albus, C., Wagener, R., Kocnar, M., Wagner, R., Henning, A., Best, F., Foerster, H., Schlepinghoff, B. S., Thomas, W., Köhle, K., Mann, K., & Senf, W. (1998). Comorbidity of Diabetes and Eating Disorders: Does diabetes control reflect disturbed eating behavior? *Diabetes Care*, *21*(7), 1110-1116.

Herzog, W., Deter, H. C., Fiehn, W., & Petzold, E. (1997). Medical findings and predictors of long-term physical outcome in anorexia nervosa: a prospective, 12-year follow-up study. *Psychological Medicine*, *27*(02), 269-279.

Hoffman, E. R., Zerwas, S. C., & Bulik, C. M. (2011). Reproductive issues in anorexia nervosa. *Expert Review Obstetrics and Gynecology, 6(4)*: 403–414.

Johnson, J. G., Spitzer, R. L., & Williams, J. B. W. (2001). Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. *Psychological Medicine*, *31*(08), 1455-1466.

Katzman, D. K. (2005). Medical complications in adolescents with anorexia nervosa: a review of the literature. *International Journal of Eating Disorders*, *37*(S1), S52-S9.

*Koubaa, S., Hallstrom, T., Lindholm, C., & Hirschberg, A. L. (2005). Pregnancy and neonatal outcomes in women with eating disorders. *Obstetrics & Gynecology, 105*(2), 255-260.

Lemons, J. A., Bauer, C. R., Oh, W., Korones, S. B., Papile, L. A., Stoll, B. J., & Stevenson, D. K. (2001). Very low birth weight outcomes of the National Institute of Child health and human development neonatal research network, January 1995 through December 1996. *Pediatrics*, *107*(1), e1-e1.

*Linna, M. S., Raevuori, A., Haukka, J., Suvisaari, J.M., Suokas, J. T., & Gissler, M. (2014). Pregnancy, obstetric and perinatal health outcomes in eating disorders. *American Journal of Obstetrics & Gynecology, 211*(4), 392.e1-392.e8.

Lowes, H., Kopeika, J., Micali, N., & Ash, A. (2012). Anorexia nervosa in pregnancy. *The Obstetrician & Gynaecologist*, *14*(3), 179-187.

Mazzeo, S. E., Slof-Op't Landt, M. C., Jones, I., Mitchell, K., Kendler, K. S., Neale, M. C., Aggen, S. H., & Bulik, C. M. (2006). Associations among postpartum depression, eating disorders, and perfectionism in a population-based sample of adult women. *International Journal of Eating Disorders*, *39*(3), 202-211.

*Micali, N., De Stavola, B., dos-Santos-Silva, I., Steenweg-de Graaff, J., Jansen, P.W, Jaddoe, V. W. V., Hofman, A., Verhulst, F. C, Steegers, E. A. P., & Tiemeier, H. (2012). Perinatal outcomes and gestational weight gain in women with eating disorders: A population-based cohort study. *BJOG: An International Journal of Obstetrics & Gynaecology, 119*(12), 1493-1502.

*Micali, N., Simonoff, E., & Treasure, J. (2007). Risk of major adverse perinatal outcomes in women with eating disorders. *The British Journal of Psychiatry, 190*(3), 255-259.

Mitchell, J. E., & Crow, S. (2006). Medical complications of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, *19*(4), 438-443.

Mitchell, J. E., Seim, H. C., Glotter, D., Soll, E. A., & Pyle, R. L. (1991). A retrospective study of pregnancy in bulimia nervosa. *International Journal of Eating Disorders*, *10*(2), 209-214.

Mitchell-Gieleghem, A., Mittelstaedt, M. E., & Bulik, C. M. (2002). Eating disorders and childbearing: concealment and consequences. *Birth*, *29*(3), 182-191.

Morgan, J. F., Lacey, J. H., & Chung, E. (2006). Risk of postnatal depression, miscarriage, and preterm birth in bulimia nervosa: retrospective controlled study. *Psychosomatic medicine*, *68*(3), 487-492.

Mulder, E. J. H., De Medina, P. R., Huizink, A. C., van den Bergh, B. R. H., Buitelaar, J. K., & Visser, G. H. A. (2002). Prenatal maternal stress: effects on pregnancy and the (unborn) child. *Early human development*, *70*(1), 3-14.

Newton, M. S., & Chizawsky, L. L. (2006). Treating vulnerable populations: The case of eating disorders during pregnancy. *Journal of Psychosomatic Obstetrics & Gynecology*, *27*(1), 5-7.

*Nunes, M. A., Pinheiro, A. P., Camey, S.A., & Schmidt, M. I. (2012). Binge eating during pregnancy and birth outcomes: A cohort study in a disadvantaged population in Brazil. *International Journal of Eating Disorders, 45*(7), 827-831.

*Pasternak, Y., Weintraub, A. Y., Shoham-Vardi, I., Sergienko, R., Guez, J., Wiznitzer, A., Shalev, H., & Sheiner, E. (2012). Obstetric and perinatal outcomes in women with eating disorders. *Journal of Women's Health, 21*(1), 61-65.

*Sollid, C. P., Wisborg, K., Hjort, J., & Secher, N. J. (2004). Eating disorder that was diagnosed before pregnancy and pregnancy outcome. *American Journal of Obstetrics & Gynecology, 190*(1), 206-210.

Solmi, F., Sallis, H., Stahl, D., Treasure, J., & Micali, N. (2014). Low birth weight in the offspring of women with anorexia nervosa. *Epidemiologic* reviews, 36, 1, 49-56.

Stewart, D. E., Raskin, J., Garfinkel, P. E., MacDonald, O. L., & Robinson, G. E. (1987). Anorexia nervosa, bulimia, and pregnancy. *American journal of obstetrics and gynecology*, *157*(5), 1194-1198.

*Torgersen, L., von Holle, A., Reichborn-Kjennerud, T., Berg, C. K., Hamer, R., Sullivan, P., & Bulik, C. M. (2008). Nausea and vomiting of pregnancy in women with bulimia nervosa and eating disorders not otherwise specified. *International Journal of Eating Disorders, 41*(8), 722-727.

Turton, P., Hughes, P., Bolton, H., & Sedgwick, P. (1999). Incidence and demographic correlates of eating disorder symptoms in a pregnant population. *International Journal of eating disorders*, *26*(4), 448-452.

*Ulman, T. F., von Holle, A., Torgersen, L., Stoltenberg, C., Reichborn-Kjennerud, T., & Bulik, C. M. (2012). Sleep disturbances and binge eating disorder symptoms during and after pregnancy. *Sleep, 35*(10), 1403-1411.

von Elm, E., Altman, D. G., Egger, M., Pocock, S. J., Gøtzsche, P. C., Vandenbroucke, J. P. (2007). The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Preventive medicine*, *45*(4), 247-251.

Watson, H. J., Torgersen, L., Zerwas, S., Reichborn-Kjennerud, T., Knoph, C., Stoltenberg, C., Stoltenberg, C., Siega-Riz, A. M., von Holle, A., Hamer, R. M., Meltzer, H. M., Ferguson, E. H., Haugen, M., Magnus, P., Kuhns, R., & Bulik, C. M. (2014). Eating disorders, pregnancy, and the postpartum period: Findings from the Norwegian Mother and Child Cohort Study (MoBa).

*Wentz, E., Gillberg, I. C., Anckarsater, H., Gillberg, C., & Rastam, M. (2009). Reproduction and offspring status 18 years after teenage-onset anorexia nervosa-A controlled community-based study. *International Journal of Eating Disorders, 42*(6), 483-491.

*** Papers included in the systematic review.**

EMPIRICAL PAPER

EARLY PARENTAL RELATIONSHIPS, EMOTION REGULATION AND EATING PSYCHOPATHOLOGY IN ADULTHOOD

Paper to be submitted to the British Journal of Clinical Psychology

ABSTRACT

Objectives: The aim of this research is to understand the mediating role of specific emotion regulation deficits in the relationship between early parental rearing styles and later eating disorder symptomology.

Method: A non-clinical (n=114) and a clinical sample (n=130) were recruited via online media. Bootstrapped parameter estimators and confidence intervals were utilised in a mediator model.

Results: Non-acceptance of emotions, impulsivity in response to emotions, lack of clarity about emotions and limited strategies in response to emotions were each found to mediate the specific relationships between maternal rejection and eating concerns in the non-clinical sample. This was a distinctly different relationship than was found in the clinical sample. For those, non-acceptance of emotions and limited strategies in response to emotions were found to mediate the specific relationship between paternal rejection and shape concerns.

Conclusion: Specific emotion regulation deficits are important mediators in the relationship between early parental rearing styles and specific eating disorder symptomology. These do not, however, form a continuum and emotion regulation deficits possess categorical roles that are distinctly different for clinical and non-clinical samples. Tentative explanations are hypothesised and clinical implications for the treatment focus of women with eating disorders are offered.

1.0 INTRODUCTION

1.1 Early relationships

It is widely agreed that early parental attachment insecurity is related to the development of eating disorders (Broberg, Hjalmers & Nevonen, 2001; Latzer, Hochdorf, Bachar & Canetti, 2002; O'Kearney, 1996; O'Shaughnessy & Dallos, 2009; Ward, Ramsay & Treasure, 2000). However when looking at particular attachment styles, some studies have found anxious and avoidant attachment styles to be more prevalent in anorexia nervosa and bulimia nervosa (Latzer et al., 2002; Tereno, Soares, Martins, Celani & Sampaio, 2008), while Troisi, Massaroni and Cuzzolaro (2005) argue that both anorexia nervosa and bulimia nervosa were related to anxious but not avoidant attachment. Ward et al. (2001) found dismissive attachment was specifically related to anorexia nervosa. Others show ambivalent attachment to be related to anorexia nervosa and bulimia nervosa (Salzman, 1997). As a general consideration it is important to note that across the literature, a wide range of measures have been utilised, purporting to measure attachment. Furthermore, those who draw from attachment theory, utilise both 3-factor and 4-factor models. Certainly there are a number of studies that purport to measure attachment, but do not utilise an attachment framework or classification at all (Chassler, 1997; Dallos & Denford, 2008; Kenny & Hart, 1992; Orzolek-Kronner, 2002). It is therefore possible that this variation across studies may explain the differences found in relation to particular attachment styles related to eating disorders. Consequently, it has been difficult to form a strong conclusion about the specific relationship between attachment styles as a broad construct and eating disorders.

Outside of the attachment-eating disorder literature, a growing number of studies have chosen to focus on the relationship between parental rearing styles and eating disorders (Chassler, 1997; Jones, Leung & Harris, 2006; Kenny & Hart, 1992; Leung, Thomas & Waller, 2000; Meyer & Gillings, 2004; Orzolek-Kronner, 2002; Perry, Silvera, Neilands, Rosenvinge & Hanssen, 2008). These studies have found relationships between feeling rejected (Chassler, 1997; Jones et al., 2006), and emotionally unsupported (Kenny & Hart, 1992; Orzolek-Kronner, 2002) as a child and the presence of an eating disorder. Interestingly, feeling overprotected by parents (Jones et al., 2006; Leung et al., 2000; Meyer & Gillings, 2004; Perry et al., 2008) was also a theme among adults with an eating disorder. These opposing styles may be indicative of a push-pull parental rearing style.

1.2 Eating disorders

Eating disorders (ED) have been a topic of interest in psychology for decades. Over this course of time, the diagnostic criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders has evolved. It is noteworthy that despite this, and clear overlaps of behaviours across ED diagnoses (Fairburn, Cooper & Shafran, 2003; Garfinkel, Kennedy & Kaplan, 1995) as well as an individual's propensity to transition between them (Fairburn & Harrison, 2003; Garner & Garfinkel, 1997) many studies continue to cluster their samples into diagnostic groups (Harrison, Sullivan, Tchanturia & Treasure, 2010; Latzer et al., 2002; Ringer and Crittenden, 2007; Tereno et al., 2008). Others on the other hand measure severity of the eating disorder as a whole (McEwan & Flouri, 2009; Meyer & Gillings, 2004). It is difficult to

make comparisons across these studies, due to the varying diagnostic criteria and those that capture severity only, do not disentangle those features across the eating disorder spectrum, that are distinct from one another. It may be that these approaches are failing to detect specific relationships between early parental rearing styles and eating disorder symptomology. A transdiagnostic approach to eating disorders has been proposed (Dakanalis et al., 2014; Wade, Bergin, Martin, Gillespie & Fairburn, 2006).

1.3 Emotion regulation

In Zachrisson and Skarderud (2010)'s review of the attachment-eating disorder literature, they suggest that one explanation for such widely differing outcomes is that there are additional factors that mediate the relationship between attachment and eating disorders. Specific deficits in emotion regulation have previously been related to eating disorders (Harrison, Sullivan, Tchanturia & Treasure, 2009; Harrison et al., 2010; Racine & Wildes, 2013; Svaldi, Griepenstroh, Tuschen-Caffier & Ehring, 2012). Eating disorders may manifest from emotion regulation deficits as a means of self-soothing or distraction, for example (Overton, Selway, Strongman & Houston, 2005). Emotion regulation deficits have also been strongly linked to poor perceptions of early parental rearing (Baker & Hoerger, 2012; Cassidy, 1994; Kullik & Petermann, 2013). The influence of emotion regulation in mediating the relationship between early parental rearing and subsequent psychopathology, such as anxiety and depression, has also been established (Baker & Hoerger, 2012; Kullik & Petermann, 2013).

To date, there has been a paucity of studies that incorporate emotion regulation deficits into a mediator model with early parental relationships and eating disorders (McEwen & Flouri, 2009; Tasca et al., 2009; van Durme, Braet, & Goossens, 2014). McEwen & Flouri (2009) investigated the role of emotion regulation when considering fathers' parenting and eating disorders symptoms in adolescent males and females. While fathers' overprotection and psychological control predicted eating disorder symptom severity, emotion regulation deficits were not found to mediate this. van Durme et al. (2014) investigated similar relationships, though they focussed on the child or adolescent's current avoidant or anxious attachment to their mother. They also measured specific eating disorder symptomology. In this instance, both anxious and avoidant attachment styles were found to predict eating restraint, which was partially mediated by deficits in emotion regulation. Finally, Tasca et al. (2009) utilised an adult female sample to investigate role of emotional reactivity and deactivation on adult attachment and severity of eating disorder symptoms. Their findings suggest that emotional reactivity mediated the relationships between attachment anxiety and eating disorder symptom severity but not attachment avoidance and eating disorder symptom severity. Due to the limited number of studies, and the lack of consistent methodology between them, no real conclusions can be drawn between what specific characteristics of early parental rearing predict particular eating disorder symptomology and which emotion regulation difficulties may mediate this.

1.4 This study

This study aims to overcome the limitations drawn from each of the three previous studies that investigated all three constructs by including an adult sample of males and females that investigates specific elements of early parental rearing in relation to both mother and father that are predictive of later specific eating psychopathologies. It will investigate whether particular emotion regulation difficulties mediate any relationships that may be exposed in order to further the understanding of the aetiology of specific eating disorder symptoms. The study will aim to answer the following questions:

1. Do parental rearing styles predict later eating disorder symptomology?
2. Do parental rearing styles predict specific deficits in emotion regulation?
3. Do specific deficits in emotion regulation predict eating disorder symptomology?
4. Do specific deficits in emotion regulation mediate the relationship between parental rearing styles and eating disorder symptomology in adulthood?

2.0 METHOD

2.1 Design

The study was conducted utilising electronic advertisement and participation. Online media is an accessible, wide-reaching method for advertising research and, in this case, a webpage and global emails were generated to encourage greater numbers of participation and subsequently increase robustness of the study findings. Once prospective participants showed interest in taking part, they were directed via the aforementioned methods, to the online survey application LimeSurvey. The survey was split to differentiate two independent participant groups. The first group were those with significant eating disorder symptomology and the second were those from a non-clinical comparison group. From there, participants from both groups completed three quantitative questionnaires, each one covering a construct under investigation. The first was the independent variable which assessed parental rearing style. The second, the mediator variable, was the emotion regulation scale. The third questionnaire was the dependent variable and measured eating psychopathology. The responses of the questionnaires for the two groups were then statistically analysed separately to establish any relationships between the constructs as well as to compare the findings for clinical and non-clinical samples.

2.2 Participants

Participants were recruited via one of two means:

1. B-Eat (see Appendix 4)

B-Eat is an organisation that provides support and guidance to individuals with eating disorders and their families. Study information and a link to the online questionnaires were advertised on the B-Eat website and via email to those enlisted in the B-Eat research database. Consent was assumed if participants submitted the completed online questionnaires. Inclusion criteria were adults aged 18 years and above, who spoke fluent English and had had difficulties with eating in the four weeks prior to completion of the study.

2. University population (see Appendix 5)

In order to provide a comparison of relationships between variables, a non-clinical sample was recruited from undergraduate and postgraduate courses at a university in the West Midlands. All prospective students were sent the study information and a link to the online questionnaires via email. Consent was assumed if participants submitted the completed online questionnaires. Inclusion criteria were that they must be 18 years of age and over and speak fluent English.

Participants who had not completed the whole battery of assessments were excluded from analysis as this was taken to imply a withdrawal of consent to participation. Anyone who reached the clinical significance for an eating disorder

recruited through the university was excluded. Anyone who was recruited through the clinical route but did not reach clinical cut-off for significance was also excluded.

2.3 Measures

Each participant completed three self-report measures: The short form of the EMBU (Egna Minnen Betraffande Uppfostran: My Memories of Upbringing; Arrindell, et al., 1999); The Difficulties in Emotion Regulation Scale (DERS; Gratz and Roemer, 2004) and Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin, 1994).

1. The short form of the EMBU (s-EMBU)

The s-EMBU is comprised of 23 items split into three subscales: *rejection* (items include punitive and shaming elements as well as those that suggest a favouring of a sibling plus hostility from the parent), *emotional warmth* (which includes signs of love and affection) and *protection* (referring to the overprotection of the parent and the control exerted over the child's behaviour).The items distinguish between maternal and paternal responses to accommodate different parenting styles. This study will incorporate both of these and so six scores will be obtained. Arrindell et al. (1999) found the short form to have good convergent and divergent validity, while Deković et al. (2006) found cross-cultural equivalence. Test re-test reliability was also supported by Muris, van Brakel, Arntz and Schouten (2011) as was internal reliability Arrindell et al. (1999; 2001).

2. The Difficulties in Emotion Regulation Scale (DERS)

The DERS measures the multidimensional elements of emotion regulation and is comprised of 36 items, rated on a 5-point scale from 1 (almost never) to 5 (almost always), split across six subscales: *non-acceptance of emotional responses* (includes a tendency to experience secondary emotions to those primary emotions causing distress), *difficulties engaging in goal-directed behaviour* (includes poor concentration when experiencing difficult emotions)*, impulse control difficulties* (problems with controlling own behaviour when experiencing difficult emotions), *lack of emotional awareness* (difficulty attending to and acknowledging emotions), *limited access to emotion regulation strategies* (a belief that little can be done to manage difficult emotions) and *lack of emotional clarity* (how much the individual understands their own emotions). Gratz and Roemer (2004) have found high internal consistency, adequate construct and predictive validity and good test-retest reliability.

3. The Eating Disorder Examination Questionnaire (EDE-Q).

The EDE-Q has 36 questions requiring responses on a 7-point scale from 0-6; 0 being “not at all” and 6 being “markedly”. The EDE-Q has four subscales; eating concern, shape concern weight concern and dietary restraint. It also assesses for binge eating and compensatory behaviours such as laxative use, purging, and exercising hard to control weight or shape. Peterson et al. (2007) have assessed the psychometric properties of this measure to be fair in comparison to other eating disorder instruments. Mean global scores from the EDE-Q were utilised to establish

clinical cut-off for inclusion and exclusion from the clinical (must be equal to or above clinical cut-off; ≥4) and non-clinical groups (must be below clinical cut-off; <4).

2.4 Ethics

The procedures involved in this study were approved by the South Birmingham Research Ethics Committee (see Appendix 6). All responses were anonymous to the chief investigator. All prospective participants were provided with information about the study to enable them to make an informed decision about participation (see Appendices 7 and 8). For anyone who did not consent to taking part, they simply did not complete the questionnaires. For those who began, but wished to withdraw, they could simply discard their completed questions instead of submitting them. All participants were alerted to the potentially sensitive nature of the questions and were advised to seek the advice of the relevant professional involved in their care (the responsible clinician for the clinical group and the GP for the non-clinical group) should they become distressed regarding the questionnaires (see Appendices 9 and 10). Those who were receiving professional care for their eating disorder were advised to speak with a member of their care team before undertaking participation.

2.5 Data analysis

Bootstrapped parameter estimators and confidence intervals will be used wherever possible. Bias corrected confidence intervals resulting from bootstrapped

confidence intervals have been reported in preference to asymptotic probability values as the bootstrap estimates are more robust to the violation of parametric assumptions (Preacher and Hayes, 2004; 2008). Where bootstrapping has been used, the results are based on 1000 bootstrap samples.

Firstly, correlates between the subscale variables were examined and repeated for the clinical and non-clinical groups to establish any significant relationships. Secondly, the variables were tested to determine whether they met the criteria for mediator analysis. According to Baron and Kenny (1986), a variable may function as a mediator to the extent that it accounts for the relationship between the predictor and criterion. Specifically, a mediator must meet four criteria. Firstly, variations in the predictor variable must affect the mediator variable significantly. Secondly, the mediator variable must affect the dependent variable. Thirdly, the independent variable must affect the dependent variable. Finally, when the mediator variable is controlled, the prior relationship between independent and dependent variables is reduced.

Correlations were utilised to determine the presence or absence of each of these relationships:

1. Parental rearing styles predict later eating disorder symptomology
2. Parental rearing styles predict specific deficits in emotion regulation
3. Specific deficits in emotion regulation predict eating disorder symptomology.

In the presence of each of these three relationships, mediator analysis was then employed to answer the question:

4. Do specific deficits in emotion regulation mediate the relationship between early parental rearing styles and eating disorder symptomology in adulthood?

Due to the nature of data obtained from clinical samples, it is often not possible to assume a normal distribution of scores, resulting in the need to utilise non-parametric statistical tests. However, the use of standard nonparametric procedures is associated with a loss of statistical power for inferential tests (Howell, 2012) and there is no direct nonparametric equivalent test for mediation analysis. In smaller samples, the loss of statistical power may substantially increase the risk of type II errors. Consequently, the nonparametric bootstrapping procedure was employed to ensure adequate statistical inferences could be made for the sample size. Using bias corrected bootstrap estimates of mediation parameters and associated confidence intervals, Fritz and MacKinnon (2007) report that 71 participants (per group) are required to demonstrate for a moderate mediation effect (i.e. $\beta>0.389$). Bootstrapping also has the advantages of being independent of sample and population distribution and providing robust confidence intervals in smaller sample situations (Hardle, 1991; Moore & McCabe, 2005).

3.0 RESULTS

3.1 Characteristics of the sample

A total of 380 people responded to the invitation to participate. Of those, 65 did not complete the full set of questionnaires and so analysis was completed for these. A further 71 respondents did not meet the clinical criteria within the EDE-Q. Those in the B-Eat sample who fell below the clinical cut-off and those in the university sample who scored above the clinical cut-off were excluded from analysis.

The included participants were compared to the excluded participants. Using bootstrapping, an independent samples t-test revealed no differences in age, weight (kg), height (m) or BMI (see Appendix 11) between the two groups. When subscale scores were compared for those included and those excluded, the only differences that emerged were for the EMBU maternal over-protection and for the EDE-Q restraint subscales (see Appendix 12). In both instances, scores were significantly higher for the included group.

The remaining 244 participants were separated into clinical (B-Eat) and non-clinical (university) subgroups. The following analyses have been conducted on only these groups. There were a total of 114 participants in the university sample and 130 participants in the B-Eat sample. Table 1 indicates the mean and standard deviation (SD) for continuous demographic variables for the groups. A one-way analysis of variance indicated no significant difference in age across the two groups (p=0.47), nor in height (p=0.25). However, the non-clinical (university) sample weighed significantly more (p<0.01) and had significantly higher BMIs (p<0.05).

Table 1. Comparison of continuous demographic variables for the two groups.

Variable	University (n=114)	B-Eat (127)	One-way ANOVA
	Mean (SD)	Mean (SD)	*F* (Sig.)
Age in years	26.96 (8.61)	27.79 (9.10)	0.52 (0.47)
Current weight (Kg)	65.04 (13.38)	58.94 (19.10)	8.07 (<0.01)
Current height (m)	1.68 (0.07)	1.67 (0.07)	1.36 (0.25)
BMI	22.99 (4.18)	21.13 (6.63)	6.60 (<0.05)

Categorical demographic data for the two groups is presented in Table 2. A Pearson Chi-Square test found that there were significantly more men in the non-clinical (university) sample (p<0.01) than the clinical (B-Eat) sample. There was no differences in ethnicity (p=0.46) or marital status (p=0.38) across the groups. There were significant differences observed for employment status across the groups. Participants in the university sample were more likely to report being employed full-time or a student, while the B-Eat sample were more likely to be unemployed (p<0.01). The clinical (B-Eat) group also disclosed significantly more co-morbid mental illness than the non-clinical (University) sample (p<0.01).

Table 2. Comparison of categorical demographic variables for the two groups.

		Sample		Pearson Chi-Square (Exact Sig. 2-Sided)
		University (n=114)	B-Eat (n=130)	
Sex:	Male	14	3	
	Female	100	127	<0.01
Ethnicity:	White British	88	107	
	White Irish	2	3	
	Any Other White background	10	14	
	African	1	0	
	Indian	3	1	
	Pakistani	3	0	
	Any other Asian background	2	2	
	White and Black Caribbean	1	0	
	White and Asian	1	2	
	Any other Mixed background	1	1	
	Arab	2	0	0.46
Marital status:	Single	78	87	
	Married	25	23	
	Separated	1	2	
	Divorced	0	3	
	Other (in relationship/cohabiting/engaged)	9	15	0.38

		Sample		Pearson Chi-Square (Exact Sig. 2-Sided)
		University (n=114)	B-Eat (n=130)	
Employment:	Employed full time	48	33	
	Employed part time	7	14	
	Self-employed	5	6	
	Student	54	43	
	Retired	0	4	
	Unemployed	0	20	
	Not working or studying due to illness/disability	0	7	
	Full-time housewife/mother/carer	0	2	
	Other (home educator/ maternity leave/ volunteer)	0	1	<0.01
Missed periods (last 4 months):	No	79	71	
	Yes	21	56	
	Males	14	3	<0.01
Comorbidity	No	94	23	
	Yes	20	107	<0.01

As can be seen in Table 3 the clinical group reported significantly more problems on all of the subscales within the EMBU (parental rearing), DERS (emotion regulation) and EDE-Q (eating psychopathology) than the non-clinical group.

Table 3. Comparison of subscale scores for the samples.

Measure	Subscale	Score range	University (*n*=114)	B-Eat (*n*=130)	Bootstrap for independent samples t-test (95% CI)	
			Mean (S.D)	Mean (S.D)	Lower	Upper
EMBU	Rejection- mother	4	1.42 (0.45)	2.17 (0.85)	-0.92	-0.57
	Rejection- father	4	1.41 (0.51)	2.11 (0.87)	-0.89	-0.53
	Warmth- mother	4	3.19 (0.69)	2.44 (0.80)	0.56	0.94
	Warmth- father	4	2.98 (0.73)	2.26 (0.83)	0.52	0.02
	Overprotection- mother	4	2.15 (0.57)	2.68 (0.76)	-0.71	-0.36
	Overprotection-father	4	1.82 (0.55)	2.28 (0.73)	-0.62	-0.30
DERS	Non-acceptance	30	14.94 (6.43)	24.88 (5.57)	-11.54	-8.30
	Goals	25	14.63 (4.32)	19.33 (4.10)	-5.89	-3.64
	Impulsivity	30	11.76 (5.39)	21.83 (5.38)	-11.45	-8.61
	Awareness	30	14.54 (5.23)	21.63 (5.36)	-8.48	-5.65
	Strategies	40	17.34 (7.88)	30.06 (6.45)	-14.59	-10.83
	Clarity	25	10.81 (4.06)	17.94 (3.92)	-0.08	-6.02
	Total	180	84.02 (24.45)	135.68 (21.07)	-57.35	-45.47
EDE-Q	Restraint	6	1.44 (1.44)	4.70 (1.08)	-3.59	-2.93
	Eating concerns	6	0.86 (1.01)	4.74 (0.83)	-4.13	-3.64
	Shape concerns	6	2.31 (1.44)	5.60 (0.44)	-3.59	-3.01
	Weight concerns	6	1.84 (1.36)	5.30 (0.64)	-3.74	-3.18
	Global score	6	1.61 (1.13)	5.08 (0.50)	-3.71	-3.24

The university sample achieved comparable mean and standard deviation (S.D) scores across all subscale and global scores on the EDE-Q with normative data found in a large study of young, non-clinical women of the same mean age (Mond, Hay, Rodgers, & Owen, 2006). Mond et al. (2006) found mean scores of 1.34 (S.D=1.39) for restraint, 0.81 (S.D=1.10) for eating concerns, 1.84 (S.D=1.50) for weight concerns, 2.24 (S.D=1.61) for shape concerns and 1.56 (S.D=1.26) for the mean global score.

In comparison, studies that have recruited clinical samples opportunistically from a single eating disorder service have found mean scores of lower than was observed here (e.g. La Mela, Maglietta, Lucarelli, Mori & Sassaroli, 2013). La Mela et al. (2013) found mean scores of 3.20 for restraint, 3.80 for eating concerns, 3.00 for weight concerns, 3.75 for shape concerns and 3.21 for the mean global score. However, the decision was made in this study, to use the stringent cut-off of ≥4 for the mean global score and so all clinical respondent scores were above this, increasing the mean. As participants were recruited anonymously online, this cut-off was made to make some control for those people who might respond that did not have significant eating difficulties. For other studies whereby participants were already known to services, use of this cut-off may not have been necessary.

While there are no clinical cut-offs for scores on the DERS as it measures constructs rather than symptoms, scores obtained in the clinical and non-clinical samples of the DERS were found to be broadly comparable to

those found in a study of women with AN and BN and their healthy controls (Harrison et al., 2010).

As with the DERS, there are no clinical cut-off scores for the EMBU. However, when comparing scores to another study within the context of eating disorders, Jones et al. (2006) found comparable paternal scores in both clinical and non-clinical samples. Overall, the findings for both clinical and non-clinical groups are generalisable, given observed comparable scores across the subscales.

3.2 Bivariate analysis

3.2.1 Non-clinical (University) sample

Table 4 presents the correlation matrices for the three measures (parental rearing, emotion regulation and eating psychopathology) and their subscales for the non-clinical (university) sample.

3.2.1.1 Association of early parental rearing styles and eating disorder symptomology

Only one subscale for early parental rearing styles (EBMU) correlated significantly with one element of eating disorder symptomology (EDE-Q). Maternal rejection was positively correlated with eating concerns ($r = 0.27$, $p<0.01$).

3.2.1.2 Association of early parental rearing styles with emotion regulation

Maternal rejection was positively correlated with non-acceptance (r=0.23, p<0.05), impulsivity (r=0.36, p<0.01), strategies (r=0.34, p<0.01) and clarity (r=0.32, p<0.01). Similarly, paternal rejection was also correlated with impulsivity (r=0.29, p<0.01), strategies (r=0.38, p<0.01) and clarity (r=0.26, p<0.01) but also with goals (r=0.27, p<0.01). Maternal warmth was negatively correlated to impulsivity (r=-0.24, p<0.05), strategies (r=-0.20, p<0.05), and clarity (r=-0.26, p<0.01). Paternal warmth was only significantly negatively correlated with clarity (r=-0.22, p<0.05). Maternal overprotection was positively correlated with goals (r=0.27, p<0.01), impulsivity (r=0.29, p<0.01) and strategies (r=0.23, p<0.05). Paternal overprotection was also positively correlated with goals (r=0.28, p<0.01), impulsivity (r=0.30, p<0.01) and strategies (r=0.19, p<0.05) and was negatively correlated with awareness (r=-0.20, p<0.05).

3.2.1.3 Association of emotion regulation and eating disorder symptomology

Non-acceptance was significantly positively correlated to all EDE-Q subscales; restraint (r=0.26, p<0.01), eating concerns (r=0.30, p<0.01), shape concerns (r=0.31, p<0.01) and weight concerns (r=0.32, p<0.01). Goals was positively correlated with eating concerns (r=0.30, p<0.01), shape concerns (r=0.26, p<0.01) and weight concerns (r=0.19, p<0.05). Impulsivity was positively correlated with eating concerns (r=0.45, p<0.01), shape concerns (r=0.33, p<0.01) and weight concerns (r=0.32, p<0.01). Awareness was significantly and positively correlated to all EDE-Q subscales; restraint (r=0.36, p<0.01), eating concerns (r=0.24, p<0.05), shape concerns (r=0.26, p<0.01) and weight concerns (r=0.22, p<0.05). Strategies was significantly and positively correlated to eating concerns (r=0.42, p<0.01), shape

concerns (r=0.31, p<0.01) and weight concerns (r=0.27, p<0.01). Clarity was significantly and positively correlated to eating concerns (r=0.24, p<0.05) and shape concerns (r=0.23, p<0.05).

3.2.2 Clinical (B-Eat) sample

Table 5 presents the correlation matrices for the three measures and their subscales for the clinical (B-Eat) sample.

3.2.2.1 Association of early parental rearing styles and eating disorder symptomology

Maternal rejection was significantly positively correlated to weight concerns only (r=0.21, p<0.05). Paternal rejection was significantly positively correlated to shape concerns (r=0.20, p<0.05) and weight concerns(r=0.21, p<0.05). Maternal overprotection was positively correlated with restraint only (r=0.22, p<0.05). Finally, paternal overprotection was positively correlated with eating concerns only (r=0.21, p<0.05).

3.2.2.2 Association of early parental rearing styles with emotion regulation

Maternal rejection was positively correlated with non-acceptance (r=0.29, p<0.01), goals (r=0.18, p<0.05), impulsivity (r=0.28, p<0.01) and strategies (r=0.33, p<0.01). Paternal rejection was only correlated with non-acceptance (r=0.22, p<0.05), and strategies (r=0.33, p<0.01). Maternal warmth did not correlate significantly with any of the DERS subscales. Paternal warmth was only significantly negatively correlated with awareness (r=-0.20, p<0.05). Maternal overprotection was

positively correlated with non-acceptance (r=0.22, p<0.05) and strategies (r=0.23, p<0.05). Finally, paternal overprotection was only positively correlated with clarity (r=0.24, p<0.01).

3.2.2.3 Association of emotion regulation and eating disorder symptomology

Non-acceptance was significantly positively correlated to eating concerns (r=0.26, p<0.01) and shape concerns (r=0.32, p<0.01) only. Goals and impulsivity were not significantly correlated to any of the EDE-Q subscales. Awareness was only significantly positively correlated to shape concerns (r=0.19, p<0.05). Strategies was significantly and positively correlated to restraint (r=0.18, p<0.05) and shape concerns (r=0.27, p<0.01). Clarity was significantly and positively correlated to restraint (r=0.18, p<0.05) and eating concerns (r=0.23, p<0.05) only.

Table 4. Pearson correlations for the University sample (n=107)

		DERS							EMBU						EDE-Q				
		Non-acceptance	Goals	Impulsivity	Awareness	Strategies	Clarity	Total	Maternal rejection	Paternal rejection	Maternal warmth	Paternal warmth	Maternal overprotection	Paternal overprotection	Restraint	Eating concerns	Shape concerns	Weight concerns	Global
DERS	Non-acceptance	1	**.299****	**.514****	**.366****	**.612****	**.481****	**.784****	**.225***	.141	-.130	-.002	.161	.145	**.260****	**.301****	**.308****	**.250****	**.322****
	Goals		1	**.598****	-.147	**.614****	**.279****	**.600****	.161	**.268****	-.143	-.132	**.265****	**.284****	.100	**.304****	**.263****	**.194***	**.241***
	Impulsivity			1	.086	**.819****	**.453****	**.819****	**.358****	**.292****	**-.243***	-.108	**.290****	**.296****	.063	.451**	.331**	.315**	.320**
	Awareness				1	.148	**.534****	**.439****	.124	.020	-.168	-.149	-.179	**-.196***	**.357****	**.240***	**.258****	**.219***	**.314****
	Strategies					1	**.579****	**.900****	**.336****	**.375****	**-.195***	-.167	**.229***	**.194***	.085	**.420****	**.311****	**.273****	**.301****
	Clarity						1	**.742****	**.323****	**.261****	**-.259****	**-.221***	.077	.057	.123	**.242***	**.231***	.148	**.211***
	Total							1	**.355****	**.317****	**-.255****	-.170	**.202***	.183	**.224***	**.459****	**.394****	**.329****	**.397****
EMBU	Maternal rejection								1	**.581****	**-.601****	**-.409****	**.288****	**.194***	-.034	**.273****	.094	.106	.112
	Paternal rejection									1	**-.392****	**-.545****	.104	**.226***	.016	.178	.022	.013	.056
	Maternal warmth										1	**.719****	.009	-.004	-.079	-.178	.000	.024	-.057
	Paternal warmth											1	.070	.135	-.058	-.107	-.006	.009	-.042
	Maternal overprotection												1	**.670****	-.008	.123	.130	.150	.111
	Paternal overprotection													1	-.035	.095	.105	.114	.078
EDE-Q	Restraint														1	**.535****	**.578****	**.581****	**.794****
	Eating concerns															1	**.679****	**.686****	**.814****
	Shape concerns																1	**.887****	**.918****
	Weight concerns																	1	**.918****
	Global																		1

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 5. Pearson correlations for the B-Eat sample (n=120)

		DERS							EMBU							EDE-Q			
		Non-acceptance	Goals	Impulsivity	Awareness	Strategies	Clarity	Total	Maternal rejection	Paternal rejection	Maternal warmth	Paternal warmth	Maternal overprotection	Paternal overprotection	Restraint	Eating concerns	Shape concerns	Weight concerns	Global
DERS	Non-acceptance	1	**.223***	**.317****	**.355****	**.469****	**.419****	**.700****	**.293****	**.218***	-.021	-.023	**.215***	.137	.132	**.262****	**.316****	.090	**.280****
	Goals		1	**.605****	-.084	**.648****	**.238****	**.630****	**.180***	.102	-.111	-.046	.102	.122	-.023	.122	.143	.068	.092
	Impulsivity			1	.031	**.677****	**.355****	**.738****	**.279****	.127	-.139	-.009	.145	.149	.005	.138	.092	-.045	.066
	Awareness				1	.121	**.439****	**.459****	.003	.138	-.054	**-.200***	-.033	.018	.155	.141	**.186***	.030	**.194***
	Strategies					1	**.467****	**.847****	**.334****	**.184***	-.071	.050	**.227***	**.243****	**.182***	.171	**.272****	.139	**.275****
	Clarity						1	**.688****	.056	.086	.039	-.088	.145	.098	**.182***	**.226***	.170	-.003	**.229***
	Total							1	**.297****	**.217***	-.091	-.069	**.202***	**.195***	.161	**.258****	**.297****	.075	**.285****
EMBU	Maternal rejection								1	**.526****	**-.563****	**-.286****	**.533****	**.362****	.044	.178	.163	**.211***	**.202***
	Paternal rejection									1	**-.288****	**-.614****	**.210***	**.438****	.089	.179	**.197***	**.205***	**.232***
	Maternal warmth										1	**.620****	-.085	-.012	.035	-.048	-.017	-.140	-.050
	Paternal warmth											1	.026	-.032	.053	-.124	-.099	-.147	-.092
	Maternal overprotection												1	**.543****	**.215***	.170	.073	.048	**.219***
	Paternal overprotection													1	.106	**.214***	.027	.091	**.181***
EDE-Q	Restraint														1	.079	**.272****	.158	**.685****
	Eating concerns															1	**.368****	**.315****	**.641****
	Shape concerns																1	**.513****	**.688****
	Weight concerns																	1	**.652****
	Global																		1

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

3.3 Mediation Analysis

In order to explore the relationship between early parental rearing styles and eating disorder symptomology, the potential mediating effects of emotion regulation was examined using mediation analysis procedures described by Preacher and Hayes (2004; 2008). Prior to the mediation analysis, all of the variables were transformed into z scores in order to standardise the regression parameters.

3.3.1 Non-clinical (University) sample

In the unmediated model, the effect of maternal rejection on eating concerns was β = 0.55 (95CI 0.15 to 0.96). This was reduced to β = 0.44 (95CI 0.03 to 0.84) when the mediating effect of non-acceptance was included (Figure 1.). The total effect for the mediated pathway was β = 0.12 (95CI 0.01 to 0.37). Accordingly, a partial mediation effect was observed.

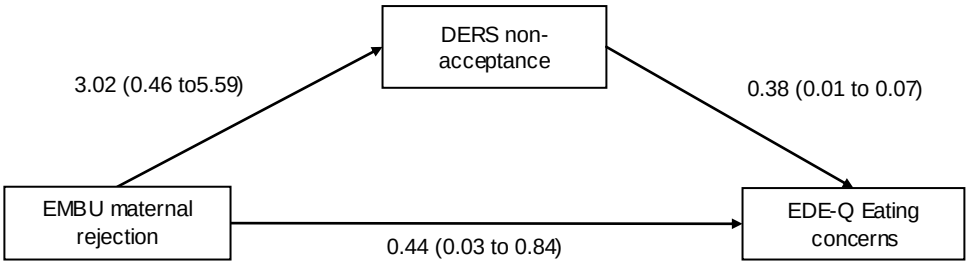


Figure 1. The mediating effect of non-acceptance upon maternal rejection and eating concerns.

The effect of maternal rejection on eating concerns was β = 0.55 (95CI 0.15 to 0.96) prior to mediation. This was reduced to β = 0.25 (95CI -0.14 to 0.65) when the mediating effect of impulsivity was included (Figure 2.). The total effect for the

mediated pathway was β = 0.30 (95CI 0.06 to 0.71). Consequently, a full mediation effect was observed.

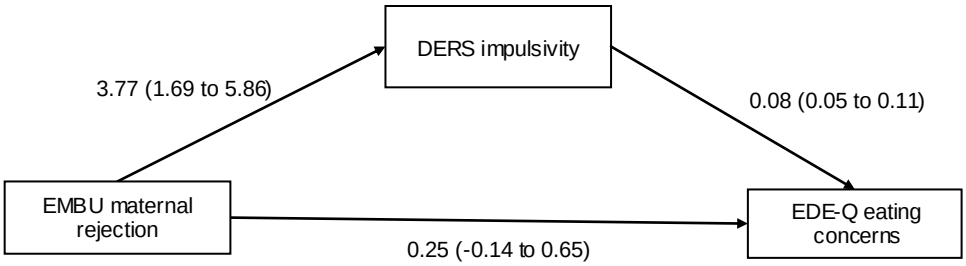


Figure 2. The mediating effect of impulsivity upon maternal rejection and eating concerns.

In the unmediated model, the effect of maternal rejection on eating concerns was β = 0.55 (95CI 0.15 to 0.96). This was reduced to β = 0.43 (95CI 0.00 to 0.84) when the mediating effect of clarity was included (Figure 3.). The total effect for the mediated pathway was β = 0.13 (95CI 0.00 to 0.39). A partial mediation effect was therefore revealed.

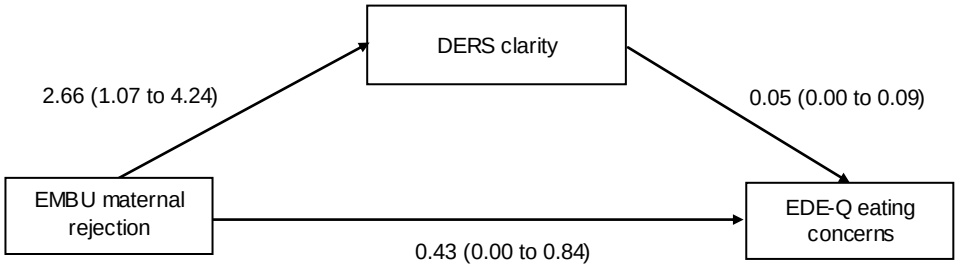


Figure 3. The mediating effect of clarity upon maternal rejection and eating concerns.

The effect of maternal rejection on eating concerns was β = 0.55 (95CI 0.15 to 0.96) in the unmediated model. This was reduced to β = 0.31 (95CI -0.09 to 0.70) when the mediating effect of strategies was included (see Figure 4.). The total effect

for the mediated pathway was β = 0.24 (95CI 0.06 to 0.58) and a full mediation effect was observed.

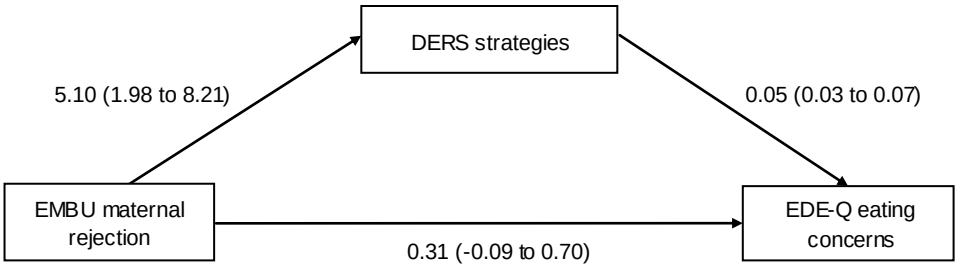


Figure 4. The mediating effect of strategies upon maternal rejection and eating concerns.

3.3.2 Clinical (B-Eat) sample

Prior to mediation, the effect of paternal rejection on shape concerns was β = 0.10 (95CI 0.01 to 0.19). This was reduced to β = 0.07 (95CI -0.02 to 0.16) when the mediating effect of non-acceptance was included (see Figure 5.). The total effect for the mediated pathway was β = 0.03 (95CI 0.01 to 0.07). Accordingly, a full mediation effect was observed.

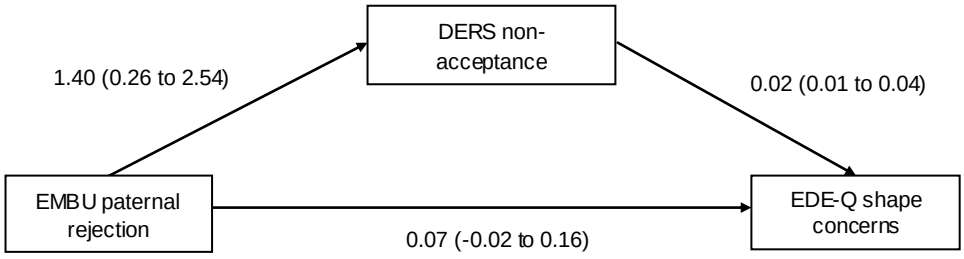


Figure 5. The mediating effect of non-acceptance upon paternal rejection and shape concerns.

In the unmediated model, the effect of paternal rejection on shape concerns was β = 0.10 (95CI 0.01 to 0.19). This was reduced to β = 0.08 (95CI -0.01 to 0.17)

when the mediating effect of strategies was included (see Figure 6.). The total effect for the mediated pathway was β = 0.02 (95CI 0.00 to 0.05). As such, a full mediation effect was indicated.

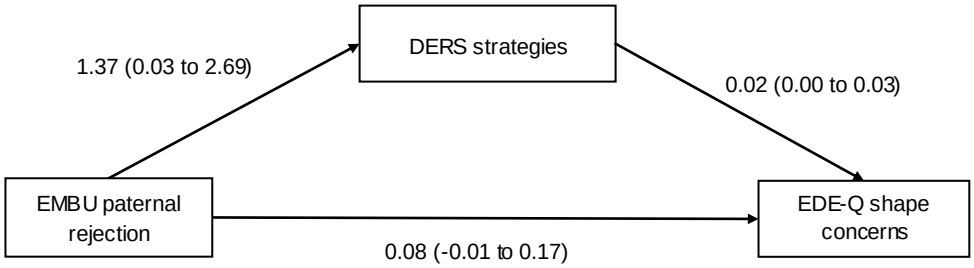


Figure 6. The mediating effect of strategies upon paternal rejection and shape concerns.

In the unmediated model, the effect of maternal overprotection on restrained eating was β = 0.24 (95CI -0.08 to 0.47). Given the non-significant findings, a mediation model to incorporate strategies was not pursued.

4.0 DISCUSSION

The aim of the present study was to examine the associations between parental rearing styles, emotion regulation and eating disorder symptomology. Moreover, the aim was to establish whether specific deficits in emotion regulation mediate the relationships between parental rearing and pathological eating behaviours and attitudes.

4.1 Non-clinical sample

Bivariate correlations did not find common relationships between any paternal parental rearing styles or maternal overprotection or warmth and any other DERS subscales and EDE-Q subscales, so they were not included in the multivariate mediation analyses for the non-clinical group. Maternal rejection was the only EMBU subscale to find common relationships with the DERS subscales and the EDE-Q eating concerns subscale. When entered into a regression model, maternal rejection was found to predict eating concerns. Given that maternal rejection also significantly predicted non-acceptance of emotional responses, impulse control difficulties, lack of emotional clarity and limited access to emotion regulation strategies and they subsequently predicted eating concerns, a mediation analysis was undertaken. Impulse control difficulties and limited access to emotional regulation strategies were both found to fully mediate the relationship between maternal rejection and eating concerns, while non-acceptance of emotional responses and lack of emotional clarity were found to partially mediate it.

4.2 Clinical sample

Bivariate correlations did not find any common relationships between any maternal parental rearing subscales or paternal warmth or overprotection and any other DERS subscales and EDE-Q subscales, so they were not included in the multivariate mediation analyses for the clinical group. Paternal rejection was the only EMBU subscale to find common relationships with the DERS subscales and the EDE-Q shape concerns subscale. When entered into a regression model, paternal rejection was found to predict shape concerns. Paternal rejection also significantly predicted non-acceptance of emotional responses and a limited access to emotion regulation strategies. Non-acceptance of emotional responses and a limited access to emotion regulation strategies also predicted shape concerns and consequently a mediation analysis was undertaken. Non-acceptance and limited access to emotion regulation strategies both fully mediated the relationship between paternal rejection and shape concerns.

Generally, these findings were in contrast to those of McEwan and Flouri (2009). Although they found no mediation effect of emotion regulation between paternal bonding and eating disorders, this may be because they failed to investigate specific features of emotion regulation and ED, and so important relationships may have been obscured by the compounding of lesser or unimportant relationships into single severity measures. The findings are, however, more congruent with those of Tasca et al. (2009) and van Durme et al. (2014), who conclude that specific emotion regulation deficits (Tasca et al., 2009) do in fact mediate the relationship between attachments and particular eating disorder symptomology (van Durme et al., 2014).

However, in these cases, emotional reactivity (Tasca et al., 2009) and eating restraint (van Durme et al., 2014) were found to be key features of the relationships.

Overall, specific deficits in emotions regulation skills were found to have varying influences as mediators. Difficulties in goal-directed behaviour and lack of emotional awareness were not found to be important factors in the mediation of the relationship between parental rearing and eating disorder symptomology for clinical or non-clinical samples.

While parental rejection appeared to be the most important predictor (of all the parental rearing styles) of eating disorder symptomology, this was specifically relevant to eating and shape concerns. Similarly, parental rejection was also found to be an important predictor of specific emotion regulation deficits, but not all. Those emotion regulation deficits were also found to significantly predict certain eating disorder symptomology. Again, this was specific to eating and shape concerns. When entered into a mediation analysis, four of the 6 emotion regulation skills either partially or fully mediated the relationship between early parental rearing and eating symptomology. As a result, the hypotheses raised in this paper are at least in part supported. Emotion regulation does appear to play an important role as a mediator, although this is specific to parental rejection and later eating and shape concerns.

The link between parental rejection and difficulties in emotion regulation has been well-established (e.g. Sheffield-Morris, Silk, Steinberg, Myers & Robinson, 2007). In this study, the presence of non-acceptance of emotion was an important mediating factor in both clinical and non-clinical groups between parental rejection and eating symptomology. In a rejecting parent, it is possible that they unable to tolerate the heightened emotions of the child, and subsequently push the child away.

The child may then learn that emotions are unacceptable and may reject their own emotions as their parents have, finding them intolerable.

Parental rejection appeared to lead to a deficiency of appropriate coping strategies in both samples indicating a lack of knowledge about how best to respond to emotions. If a parent is rejecting of the child and their emotions, it is likely that they have not been taught helpful strategies to help approach and manage them, thus leading the child to develop into an adult without effective emotion regulation strategies. This may also go some way to explain why individuals feel out of control, responding impulsively to emotions and feeling they have no way to manage them effectively as found in the non-clinical sample.

If an adult has not developed ways to acknowledge, accept and manage their emotions, certain eating disorder symptomology may ensue. Eating and shape concerns were two subscales that were important within the mediator model. It is possible that when individuals cannot accept their emotions, and do not have adaptive strategies to manage them, they may focus on their eating patterns and worry about their shape as cognitive strategies that may not only distract them from their emotions but also provide a way of self-soothing. These cognitions may then form the basis for modifying their eating habits in a more behavioural way. The theory behind maladaptive eating as a coping strategy has long been established (Svaldi et al., 2012).

One particularly interesting finding from the current study is the different patterns in parental influence in the mediating relationships. Paternal rejection was found to be a significant factor in the clinical population, whereas maternal rejection was a key component in the control group. The distinct differences in mediator

models between the clinical and non-clinical samples suggest categorical, rather than continuous relationships.

In terms of speculating why paternal rejection played a key role in the development of shape concerns in the clinical group it may be helpful to consider the father as being the first important male figure in the child's life. It is quite likely that the paternal figure has a strong influence on the psycho-sexual and psycho-social development of his daughter (Maine, 1993), her femininity and her identity as a woman. It is possible therefore that this shapes her beliefs about what men are likely to want in a woman with regards her appearance and shape. If the father is rejecting or absent, she may question her sense of self in terms of sexuality and body image, for example (Maine, 1993) and become preoccupied with her shape to ensure she remains attractive to men and is not rejected further. In addition, Lamb (2010) also postulates the presence of the father as playing an important role in supporting mothers in their role as care-giver, which may subsequently enhance the mother-child relationship.

Maternal rejection appeared to be the most important variable within the non-clinical sample, but not in the clinical sample. In attempting to understand this difference, the concept of enmeshment should be considered. Enmeshment has long been recognised as key to the understanding of ED development (e.g. Humphrey, 1989; Minuchin, Rosman & Baker, 1978), Enmeshment has been defined as a familial relationship within which psychological and emotional fusion between members is apparent (Barber & Buehler, 1996). This enmeshment can impede individuation of the offspring and prevent psychosocial maturity (Greenberger & Sorensen, 1974). Therefore, within the clinical sample, it is possible that

enmeshment with the mother was present, but not captured by the constructs in the EMBU parental rearing measure. If this was the case, this group will have experienced a 'closeness' to their mother, marked by an inability separate their own emotions from their mother's and vice versa along with an over-controlling and intrusive mother within the child's life. It is possible that within the context of an enmeshed maternal relationship, rejection goes undetected or unreported by the child because for all intents and purposes, the mother remains very physically 'present' even if she is critical and does not accept the child's emotions. The experience of enmeshment coupled with rejection can be confusing for the child. Conversely, if the child is not enmeshed with the father, they may be more attuned to his rejection. A physical absence, or unavailability, may be more apparent as rejection. This may therefore explain why, in the context of an eating disorder, paternal rejection is considered more detrimental, according to these findings.

4.3 Strengths and limitations

This study has important strengths and limitations. Very few studies have investigated emotion regulation as a mediating factor between early relationships and eating disorders. Of those, McEwan and Flouri (2009) utilised severity measures of emotion regulation and eating disorders, making clear links between constructs impossible. While Tasca et al. (2009) overcame this, in part, by differentiating emotional reactivity and emotional cut-off, they too utilised a severity measure of eating symptomology. In contrast, van Durme et al. (2014) investigated specific eating disorder symptoms, but utilised a severity scale for difficulties in emotion

regulation, and so were unable to ascertain which elements of emotion regulation contributed to the mediation model. This study overcame these limitations by investigating specific emotion regulation deficits as well as specific eating disorder symptomology (trans-diagnostically), and from the results this was clearly important.

A further advantage of this study is the incorporation of both clinical and non-clinical samples. As can be seen from the results, the types of relationships found across the two groups are distinctly different. This is an important preliminary finding that warrants further investigation. The on-line recruitment method produced larger sample sizes from which more robust conclusions could be made. This was further enhanced by the use of bootstrapping re-sampling methods that, again, were more resilient to error. While a large number of respondents were excluded due to being over or under the threshold for EDE-Q scores, the fact that there were no differences in their demographics goes some way to suggest the sample included were representative of a wider population.

There were, however, important limitations to be noted. As the EMBU looks at early life experiences, this is a retrospective measure and is therefore susceptible to recall bias. Similarly, as all measures were self-reported and completed anonymously online, and not corroborated by additional, objective sources, the issue of response bias was not addressed. It is possible that difficulties in each of the three areas were over or under-reported. Furthermore, given that the study was cross-sectional in design, causality between the variables could not be assumed. It is also important to note that other differences were found between the clinical and non-clinical sample. While most were expected, such as BMI and weight, attention should be paid to a significantly greater occurrence of co-morbidity of mental health problems in the

clinical group. Although co-morbidity is known to be high for this population (Hudson, Hiripi, Pope & Kessler, 2007), it is possible that this may have influenced the relationships between the variables, and may go some way to explain the different types of relationships found in the non-clinical sample. Further investigations should attempt to control for any influence of co-morbidity.

While differences were observed between the clinical and non-clinical samples, only speculative interpretations of those differences could be made at this early stage. Further investigations would need to explore any possible influence of enmeshment within maternal but also paternal relationships to establish the role this plays on the impact of parental rejection in those with and without eating disorder symptomology. In addition, new relationships have been observed in terms of the mediating role of specific elements of emotion regulation, predominantly non-acceptance and a lack of strategies. Given that not all elements were found to be important mediators, further research is needed to develop an understanding of whether these relationships are replicated, but also to establish why these specific elements play such a fundamental role.

4.4 Clinical implications

Given emotion regulation deficits were found to fully mediate the relationship between parental rejection and shape concerns in participants with ED, the findings support the importance of addressing emotion regulation in the treatment of pathological eating. Indeed, the mediator relationships emerging from this study also indicate a need for further investigation of the father-daughter role in the

development of ED, which has, in comparison to studies of the mother-daughter relationship, been overlooked. This would suggest that when considering appropriate treatment, any family therapy should certainly include the paternal caregiver, and any individual therapy should undoubtedly give consideration to the father-daughter relationship. In terms of considering preventative strategies, the impact of emotion regulation deficits on the development of ED would point to the need for greater emphasis on the development of the key components of emotion regulation perhaps within an educational environment. If children are able to develop an awareness of their emotions, along with an acceptance or tolerance for these emotions this will enable them to develop and utilise a range of adaptive strategies in response to these emotions. In turn, this will reduce the likelihood of developing pathological eating attitudes and behaviours as a maladaptive way of coping with emotions.

5.0 CONCLUSION

This was the first study to examine links between specific elements of early parental rearing styles, emotion regulation and ED attitudes and behaviours and to establish the role of emotion regulation as a mediator between early parental rearing styles and eating disorder symptomology. The findings indicate that emotion regulation deficits play an important mediating role in both clinical and non-clinical samples, though the specific nature of these relationships differs across groups. While these are preliminary findings, they are important in indicating very specific but key areas for treatment in an often difficult to treat population.

REFERENCES

Arrindell, W. A., Sanavio, E., Aguilar, G., Sica, C., Hatzichristou, C., Eisemann, M., Recinos, L. A., Gaszner, P., Peter, M., Battagliese, G., Kallai, J., & van der Ende, J. (1999). The development of a short form of the EMBU: Its appraisal with students in Greece, Guatemala, Hungary and Italy. *Personality and Individual Differences*, *27*(4), 613-628.

Arrindell, W., Richter, J., Eisemann, M., Gärling, T., Rydén, O., Hansson, S., Kasielke, E., Frindte, W., Gillholme, R., & Gustafsson, M. (2001). The short-EMBU* in East-Germany and Sweden: A cross-national factorial validity extension. *Scandinavian journal of psychology*, *42*(2), 157-160.

Baker, C. N., & Hoerger, M. (2012). Parental child-rearing strategies influence self-regulation, socio-emotional adjustment, and psychopathology in early adulthood: Evidence from a retrospective cohort study. *Personality and individual differences*, *52*(7), 800-805.

Barber, B. K., & Buehler, C. (1996). Family cohesion and enmeshment: Different constructs, different effects. *Journal of Marriage and the Family*, 433-441.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of personality and social psychology*, *51*(6), 1173.

Broberg, A. G., Hjalmers, I., & Nevonen, L. (2001). Eating disorders, attachment and interpersonal difficulties: a comparison between 18-to 24-year-old patients and normal controls. *European Eating Disorders Review*, *9*(6), 381-396.

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. *Monographs of the Society for Research in Child Development*, *59*(2-3), 228-249.

Chassler, L. (1997). Understanding anorexia nervosa and bulimia nervosa from an attachment perspective. *Clinical Social Work Journal*, *25*(4), 407-423.

Dallos, R., & Denford, S. (2008). A qualitative exploration of relationship and attachment themes in families with an eating disorder. *Clinical child psychology and psychiatry*, *13*(2), 305-322.

Dakanalis, A., Timko, C. A., Zanetti, M. A., Rinaldi, L., Prunas, A., Carrà, G., Riva, G., & Clerici, M. (2014). Attachment insecurities, maladaptive perfectionism, and eating disorder symptoms: A latent mediated and moderated structural equation modelling analysis across diagnostic groups. *Psychiatry research*, *215*(1), 176-184.

Deković, M., ten Have, M., Vollebergh, W. A., Pels, T., Oosterwegel, A., Wissink, I. B., De Winter, A. F., Verhulst, F. C., & Ormel, J. (2006). The cross-cultural equivalence of parental rearing measure: EMBU-C. *European Journal of Psychological Assessment*, *22*(2), 85-91.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363-370.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A “transdiagnostic” theory and treatment. *Behaviour research and therapy*, *41*(5), 509-528.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *The Lancet*, *361*(9355), 407-416.

Fritz, M. S., & MacKinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological science*, *18*(3), 233-239.

Garfinkel, P. E., Kennedy, S. H., & Kaplan, A. S. (1995). Views on classification and diagnosis of eating disorders. *The Canadian Journal of Psychiatry*, *40*(8), 445-456.

Garner, D. M., & Garfinkel, P. E. (Eds.). (1997). *Handbook of treatment for eating disorders*. New York: Guilford Press.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41-54.

Greenberger, E., & Sørensen, A. B. (1974). Toward a concept of psychosocial maturity. *Journal of Youth and Adolescence*, *3*(4), 329-358.

Hardle, W. (1991). Bootstrap Simultaneous Error Bars for Nonparametric Regression. *The Annals of Statistics*, *19*(2), 778–796.

Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2009). Emotion recognition and regulation in anorexia nervosa. *Clinical Psychology & Psychotherapy*, *16*(4), 348-356.

Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2010). Emotional functioning in eating disorders: attentional bias, emotion recognition and emotion regulation. *Psychological medicine*, *40*(11), 1887-1897.

Howell, D. (2012). *Statistical Methods for Psychology*. Cengage Learning.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological psychiatry*, *61*(3), 348-358.

Humphrey, L. L. (1989). Observed family interactions among subtypes of eating disorders using structural analysis of social behaviour. *Journal of Consulting and Clinical Psychology, 57*, 206-214.

Jones, C. J., Leung, N., & Harris, G. (2006). Father-daughter relationship and eating psychopathology: The mediating role of core beliefs. *British Journal of Clinical Psychology*, *45*(3), 319-330.

Kenny, M. E., & Hart, K. (1992). Relationship between parental attachment and eating disorders in an inpatient and a college sample. *Journal of Counselling Psychology*, *39*(4), 521.

Kullik, A., & Petermann, F. (2013). Attachment to Parents and Peers as a Risk Factor for Adolescent Depressive Disorders: The Mediating Role of Emotion Regulation. *Child Psychiatry & Human Development*, *44,* 537-548.

Lamb, M. E. (2010). How do fathers influence children's development? Let me count the ways. In M. E. Lamb (Ed.) *The role of the father in child development* (pp.1-26). New Jersey: John Wiley & Sons.

Latzer, Y., Hochdorf, Z., Bachar, E., & Canetti, L. (2002). Attachment style and family functioning as discriminating factors in eating disorders. *Contemporary Family Therapy*, *24*(4), 581-599.

Leung, N., Thomas, G., & Waller, G. (2000). The relationship between parental bonding and core beliefs in anorexic and bulimic women. *British Journal of Clinical Psychology*, *39*(2), 205-213.

Maine, M. (1993). *Father hunger.* London: Simon & Schuster.

McEwen, C., & Flouri, E. (2009). Fathers' parenting, adverse life events, and adolescents' emotional and eating disorder symptoms: The role of emotion regulation. *European child & adolescent psychiatry*, *18*(4), 206-216.

Meyer, C., & Gillings, K. (2004). Parental bonding and bulimic psychopathology: The mediating role of mistrust/abuse beliefs. *International Journal of Eating Disorders*, *35*(2), 229-233.

Minuchin, S., Rosman, B. L., & Baker, L. (1978). *Psychosomatic families: Anorexia nervosa in context*. Massachusetts: Harvard University Press.

Moore, D. S., & McCabe, G. P. (2005). Bootstrap methods and permutation tests. In *Introduction to the Practice of Statistics*. WH Freeman & Co.

Muris, P., van Brakel, A. M. L., Arntz, A., & Schouten, E. (2011). Behavioral inhibition as a risk factor for the development of childhood anxiety disorders: A longitudinal study. *Journal of Child and Family Studies, 20*(2), 157–170.

O'Kearney, R. (1996). Attachment disruption in anorexia nervosa and bulimia nervosa: A review of theory and empirical research. *International Journal of Eating Disorders*, *20*(2), 115-127.

O'Shaughnessy, R., & Dallos, R. (2009). Attachment research and eating disorders: A review of the literature. *Clinical child psychology and psychiatry*, *14*(4), 559-574.

Orzolek-Kronner, C. (2002). The effect of attachment theory in the development of eating disorders: Can symptoms be proximity-seeking? *Child and Adolescent Social Work Journal*, *19*(6), 421-435.

Overton, A., Selway, S., Strongman, K., & Houston, M. (2005). Eating disorders—The regulation of positive as well as negative emotion experience. *Journal of Clinical Psychology in Medical Settings*, *12*(1), 39-56.

Perry, J. A., Silvera, D. H., Neilands, T. B., Rosenvinge, J. H., & Hanssen, T. (2008). A study of the relationship between parental bonding, self-concept and eating disturbances in Norwegian and American college populations. *Eating Behaviors*, *9*(1), 13-24.

Peterson, C. B., Crosby, R. D., Wonderlich, S. A., Joiner, T., Crow, S. J., Mitchell, J. E., Bardone-Cone, A. M., Klein, M. K., & le Grange, D. (2007). Psychometric properties of the eating disorder examination-questionnaire: Factor structure and internal consistency. *International Journal of Eating Disorders*, *40*(4), 386-389.

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods*, *36*(4), 717–731.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*(3), 879–891. doi:10.3758/BRM.40.3.879

Racine, S. E., & Wildes, J. E. (2013). Emotion dysregulation and symptoms of anorexia nervosa: The unique roles of lack of emotional awareness and impulse control difficulties when upset. *International Journal of Eating Disorders*, *46*(7), 713-720.

Ringer, F., & Crittenden, P. M. (2007). Eating disorders and attachment: The effects of hidden family processes on eating disorders. *European Eating Disorders Review*, *15*(2), 119-130.

Salzman, J. P. (1997). Ambivalent attachment in female adolescents: Association with affective instability and eating disorders. *International Journal of Eating Disorders*, *21*(3), 251-259.

Sheffield-Morris, A., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotional regulation. *Social Development, 16*, 361-388.

Svaldi, J., Griepenstroh, J., Tuschen-Caffier, B., & Ehring, T. (2012). Emotion regulation deficits in eating disorders: A marker of eating pathology or general psychopathology? *Psychiatry Research*, *197*(1), 103-111.

Tasca, G. A., Szadkowski, L., Illing, V., Trinneer, A., Grenon, R., Demidenko, N., Krysanski, V., Balfour, L., & Bissada, H. (2009). Adult attachment, depression, and eating disorder symptoms: The mediating role of affect regulation strategies. *Personality and Individual Differences*, *47*(6), 662-667.

Tereno, S., Soares, I., Martins, C., Celani, M., & Sampaio, D. (2008). Attachment styles, memories of parental rearing and therapeutic bond: A study with eating disordered patients, their parents and therapists. *European Eating disorders review*, *16*(1), 49-58.

Troisi, A., Massaroni, P., & Cuzzolaro, M. (2005). Early separation anxiety and adult attachment style in women with eating disorders. *British Journal of Clinical Psychology*, *44*(1), 89-97.

van Durme, K., Braet, C., & Goossens, L. (2014). Insecure Attachment and Eating Pathology in Early Adolescence: Role of Emotion Regulation. *The Journal of Early Adolescence, 35*(1), **54-78.**

Wade, T. D., Bergin, J. L., Martin, N. G., Gillespie, N. A., & Fairburn, C. G. (2006). A transdiagnostic approach to understanding eating disorders. *The Journal of nervous and mental disease*, *194*(7), 510-517.

Ward, A., Ramsay, R., & Treasure, J. (2000). Attachment research in eating disorders. *British Journal of Medical Psychology*, *73*(1), 35-51.

Ward, A., Ramsay, R., Turnbull, S., Steele, M., Steele, H., & Treasure, J. (2001). Attachment in anorexia nervosa: A transgenerational perspective. *British Journal of Medical Psychology*, *74*(4), 497-505.

Zachrisson, H. D., & Skårderud, F. (2010). Feelings of insecurity: Review of attachment and eating disorders. *European Eating Disorders Review*, *18*(2), 97-106.

II PUBLIC DOMAIN DOCUMENT

PREDICTORS AND OUTCOMES OF EATING DISORDERS

This research was conducted by Alexandra Stanbury as part of the Doctorate of Clinical Psychology at the University of Birmingham. The research was supervised by Dr Chris Jones, Academic Tutor at the University of Birmingham and by Dr Newman Leung, Consultant Clinical Psychologist at the Birmingham Eating Disorder Service.

There are two parts of the research. The first part is a systematic review of existing literature aimed at establishing whether active or historic eating disorders increase the risk of maternal complications to health during pregnancy and delivery. In order to answer this question, four electronic databases were searched to find all relevant journal articles. The search yielded 15 relevant research articles, published between 1995 and 2015 that attempted to answer the question.

The 15 papers were evaluated against a set of quality criteria to determine how much weight could be given to their results. While lots of possible complications were investigated, ranging from hypertensive disorders (e.g. Koubaa et al., 2005; Micali et al., 2012; Pasternak et al., 2012) during pregnancy to premature labour (e.g. Bansil et al., 2008; Eagles et al., 2012; Ekeus et al., 2006), most results indicated that none of the different eating disorder diagnoses were at an increased risk. A handful of articles did find increased risks of certain complications, but because they were the only papers to investigate that complication (e.g. Torgerson et al., 2008), because they were poor quality papers (Bulik et al., 1999; Linna et al., 2014), or because the rest of the research found conflicting results, no real conclusions could be made about them. The review concluded that, at present, there is no substantial, reliable research to suggest women with eating disorders, past or present, are at

increased risk during pregnancy or labour. However, due to the fact that there was only a limited number of articles, and they all investigated different outcomes and in different ways, further research is required to confirm this. The review undertaken has important implications for maternity services. At present there is often concern amongst health professionals that women with an eating disorder who become pregnant are more at risk of health complications. This may lead to an increased monitoring or intervention where these are not necessarily required.

The second part to this research is an original project which aimed to investigate whether specific styles of early parenting play particular roles in eating disorder symptoms in offspring, and whether the amount of impact parenting has is affected by the individual's ability to understand and manage their emotions. A wealth of research has investigated each of these relationships separately (Barker & Hoerger, 2012; Harrison et al., 2009; Ward et al., 2000), though there is a lack of research investigating the relationships between early parenting, emotion regulation and eating symptomology (McEwan & Flouri, 2009; Tasca et al., 2009; van Durme et al., 2014). The study described here aimed to overcome the limitations of previous studies and add to the currently limited literature in this area.

Advertisements to take part were made via the University of Birmingham email system, and through B-Eat charity that supports people with eating disorders. Individuals who wished to take part did so using an online survey. This consisted of a list of questions asking about their relationship with their mother and father figures in early childhood, how they manage their emotions and the kinds of attitudes and behaviours they have around eating.

130 people with eating disorders completed the study along with 114 people without. Following statistical analysis, specific relationships were found. Feeling rejected by a parent was the most important aspect of parenting, according the findings, in terms of influencing particular shape concerns and worries about eating.

An overprotective style of parenting or parenting that lacks warmth did not appear to have an important influence over eating difficulties. Feeling rejected by a parent also influenced whether a person was able to develop a tolerance for their emotions as well as whether they learnt strategies to cope with them. This in turn was found to affect whether individuals then developed later concerns about eating patterns and concerns about their shape, possibly as a way of distracting them from their emotions or as a way of making themselves feel better.

This has important implications for the treatment and prevention of eating disorders, in that early education around emotion regulation at school may reduce the likelihood of developing an eating disorder as a coping strategy. Similarly, it seems that emotion regulation skills should perhaps be the target of intervention for those who have already developed an eating disorder.

APPENDICES

Appendix 1. Summary of methodology for studies

1st author (year), *location*	Sample *ED status*	Study design *Periods of data collection*	Method of recruitment *Measurement of variables*	Analysis *Covariates*
Bansil (2008) *USA*	N= 1,668 delivery hospitalisations with ED (AN and/or BN) aged 15-44 years - a rate of 0.39/10 000 to those without ED. *ED at time of delivery hospitalisation*	Retrospective cohort study. *Hospital delivery discharges between 1994 and 2004.*	National Inpatient Sample (all discharges from stratified sample of 20% of all U.S community hospitals). *Diagnosis of ED given using ICD-9-CM for AN and BN.* *Outcomes and covariates also used ICD-9-CM.* *Tool used to made diagnoses not identified.*	Chi-square for categorical data. Student t-test for continuous data. Multivariate logistic regression to estimate odds ratios and confidence intervals after adjustment *Depression, alcohol or substance abuse, age, insurance status, location, region.*
Bulik (1999) *New Zealand*	AN (n=66) Unexposed (n=98) matched on age banding. *Historical diagnosis of AN made between 1981 and 1984*	Retrospective cohort study. *Diagnosis of AN between 1981-1984, 'followed-up' 'over a decade' later.*	AN patients recruited through single ED Service in Christchurch. Unexposed randomly selected from electoral roll. *Original AN diagnosis according to DSM-III-R via ED service. Exclusion of caseness in unexposed group using same criteria.* *Outcomes measures defined according to Diagnostic Interview of Genetic Studies (DIGS)* *All information obtained via patient report through face-to-face or phone interview.*	Chi square or Fisher's Exact test for categorical data. Student t-test for continuous data. Nominal logistic regression and standard least squares regression. *Matched by age-banding. No covariates identified.*
Bulik (2009) *Norway*	AN (n=35) BN (n=304) BED (n=1,812) EDNOS-P (n=36) Unexposed (n=33,742). *ED 6 months prior to or during pregnancy*	Prospective cohort study. *Pregnancies in Norway from 1999-2006.*	All participants recruited via the Norwegian Mother and Child Cohort Study (MoBa). *ED and non-ED differentiated via self-report questionnaire broadly based on DSM-IV between 28 and 44 weeks of pregnancy.* *Outcome data from medical birth registry of Norway (MBRN) recorded by doctors and midwives.* *Covariate data was self-reported.*	Univariate Poisson regression for dichotomous variables. Planned models to estimate relative risk and include covariates. Quasi-likelihood approach with GEE to estimate variance of relative risk estimates. *Smoking during pregnancy, household income, education, parity, maternal age, gestational age, pre-pregnancy BMI, gestational weight gain.*

1st author (year), *location*	Sample *ED status*	Study design *Periods of data collection*	Method of recruitment *Measurement of variables*	Analysis *Covariates*
Eagles (2012) *Aberdeen, Scotland*	AN (n=134) matched to 5 unexposed by age, parity and year of first delivery Matched unexposed pregnant women (n=670). Total unexposed cohort (AMND) (n=54,100) *Historical diagnosis of AN made between 1965 and 2007.*	Retrospective cohort study. *First time diagnosis of AN in Aberdeen between1965-2007 and followed-up for occurrence of pregnancy during same time period.*	AN participants identified through the Aberdeen Psychiatric Case Register (APCR). Pregnant women (AN and unexposed) identified via Aberdeen Maternity and Neonatal Databank (AMND). *ED measurement unclear.* *Outcomes –from AMND. Unclear how collected.*	Multilevel logistic regression with GEE to estimate relative risk. *BMI, smoking, social class, marital status, baby gender, other pregnancy-related events that may have confounded the association (e.g. induction of labour, pre-eclampsia, for preterm labour).*
Ekeus (2006) *Sweden*	AN (n=1000) Unexposed (n=827,582). *Anyone having inpatient treatment between 1973 and 1996.*	Prospective cohort study. *Diagnosis of AN between 1973 and1996 with births between 1983 and 2002.*	AN patients identified through Swedish Hospital Discharge Register. Pregnant women (AN and unexposed) identified from the Swedish Medical Birth Register. *AN measured using ICD-8 or ICD-9 criteria but unclear how.* *Outcomes gleaned from Swedish Medical Birth Register. Unclear how collected.*	Logistic regression for categorical variables. Odds ratios calculated. Linear regression for birthweight. *Year of birth, maternal age, smoking.*
Koubaa (2005) *Stockholm, Sweden*	AN (n=24) BN (n=20) EDNOS (n=5) Unexposed (n=68) *ED Historical only (though 11 relapsed during pregnancy).*	Prospective cohort study. *Screened for current ED at 10 weeks gestation between August 1997 and June 2001.*	All participants recruited from 13 prenatal clinics in Stockholm. *ED (presence or absence) measured via self-report screening tool then interviewed by trained midwives according to DSM-IV criteria.* *Outcomes obtained from medical records and objectively measured by midwife.*	Chi-square for categorical data. 1-way ANOVA followed by Fisher's least significant difference test. ANCOVA to compare blood pressure. *BMI for blood pressure analysis in ANCOVA.*
Linna (2014) *Helsinki, Finland*	Total ED (n=2,257). Of these: AN (n=182 delivered 302 babies) BN (n=436 delivered 724 babies) BED (n=39 delivered 52 babies). Total unexposed (n=9,028) matched n=4 per case on age and geographical area. Of these: (n=3,642 delivered 6,319 babies). *ED treated in clinic between 1995 and 2010.*	Prospective cohort study. *Participants followed from admission to clinic between 1995 and Dec 2010 up to end of study in Dec 2010/death/moving abroad/age 50.*	ED patients were all those treated in ED clinic at Helsinki University Central Hospital who had live birth during the follow up period. Unexposed randomly selected from Central Population Register. *ED method of diagnosis not reported.* *Perinatal outcomes from the Medical Birth Register. Unclear how collected.*	Logistic regression. Estimated odds ratios and confidence intervals before and after adjustments. *Age, geographical area. Unclear what additional variables 'adjusted' for.*

1st author (year), *location*	Sample *ED status*	Study design *Periods of data collection*	Method of recruitment *Measurement of variables*	Analysis *Covariates*
Micali (2012) *Rotterdam, Netherlands*	AN (n=129) BN (n=209) AN+BN (n=100) Other psychiatric disorder (n=1,002) Unexposed (n=3,816) *ED in Lifetime*	Prospective cohort study. *All pregnant women with due delivery date between April 2002 and January 2006.*	All participants recruited via 'Generation R', a population-based birth cohort. *ED self-reported at 20 weeks (yes/no question).* *Perinatal outcomes obtained via midwifery and hospital registers, obstetric records, face-to-face assessment or self-report questionnaires* *Covariates obtained via self-report questionnaire at around 18 weeks.*	Chi-square or F-Test for categorical variables. Linear regression for continuous variables. Logistic regression for categorical and binary variables. Odds ratios and CIs calculated. *Maternal age, parity, ethnicity, maternal education, smoking, alcohol.*
Micali (2007) *Avon, England*	AN (n=171) BN (n=199) AN+BN= (n=82) Other psychiatric disorder (n=1166) Unexposed (n=10,636) *ED in Lifetime*	Prospective cohort study. *All pregnant women with due delivery date between April 1991 and December 1992.*	All participants recruited via ALSPAC, a population-based birth cohort. *ED self-reported at 12 and 18 weeks (yes/no question).* *Perinatal outcomes obtained via self-report (miscarriage) and obstetric records (birth weight, gestational age, prematurity). Unclear how collected.* *Covariates obtained via self-report.*	One-way ANOVA. Bivariate linear regression for continuous outcomes. Multi-nomial and binary logistic regression for categorical and binary outcomes. Covariates tested using bivariate analysis then included in multivariate where significant. *Pre-pregnancy BMI, gestational age, gender of baby, parity, maternal age, alcohol, smoking.*
Nunes (2012) *Southern Brazil*	BE (n=119) Unexposed (n=578). *Active during pregnancy.*	Prospective cohort study. *All women attending prenatal care clinics (between 16 and 36 weeks of pregnancy) from June 2006 to September 2007.*	All participants recruited via ECCAGE (The Study of Food Intake and Eating Behaviours in Pregnancy), a cohort study of pregnant women covering 18 primary care units. *BE (presence or absence) identified via EDE-Q for BED symptoms.* *Outcomes obtained from prenatal (GD, preeclampsia, anaemia, infection), and birth records (birth weight, SGA, LGA, Macrosomia, C-section, prematurity).* *Covariates obtained from self-report (maternal age, household income, maternal education, gestational weight gain, parity, BMI, smoking, alcohol use) and birth records (Gestational age).*	Poisson regression with robust variance to estimate relative risk and CIs with and without adjustments following systematic inclusion of covariates. *Maternal age, gestational age, parity, household income, maternal education, smoking, alcohol use, pre-pregnancy BMI, gestational weight gain.*
Pasternak (2012)	AN (n=41) BN (n=62) EDNOS (n=19)	Retrospective cohort study. *All Jewish women delivering babies at*	All participants recruited from sole hospital in Negev that serves whole population.	Chi-square or Fisher's Exact test for categorical variables. Student *t* test for continuous variables.

1st author (year), *location*	Sample *ED status*	Study design *Periods of data collection*	Method of recruitment *Measurement of variables*	Analysis *Covariates*
Israel	All EDs grouped together. Unexposed (n=117,875). *ED in lifetime (no differentiation between remissive/ active).*	*hospital between 1988 and 2009.*	*ED identified from outpatient records of ED clinic. Measured via interview by medical staff in accordance with DSM-IV. Tool not stated.* *Outcomes obtained from perinatal database (inputted to by obstetrician after birth).* *Covariates obtained from unknown source.*	Multivariate logistic regression with GEE to incorporate covariates. Odds ratios and CIs calculated. *Maternal age, parity.*
Sollid (2004) *Denmark*	ED (n=302 women, delivered 504 babies) EDs included AN and other specified and unspecified ED. Unexposed (n=900, delivered 1552 babies). *Those hospitalised for their eating disorder prior to pregnancy.*	Prospective cohort study. *Hospitalised for ED between 1973 and 1993 and had a subsequent birth within this time period.*	All participants recruited from Danish Database for Psychiatric Epidemiological Research linking two registers: Danish Psychiatric Central Register (DMBR; those admitted for treatment of ED) Danish Medical Birth Register. Unexposed were from a random sample of those without a prior hospitalisation for ED. *ED diagnosed according to ICD-8. Unclear what tool used.* *Outcomes obtained from DMBR inputted by midwives.* *Covariates obtained from unknown source.*	Student t-test for continuous variables. Logistic regression for categorical variables. Odds ratio and CIs calculated. Stratified analyses for interactions. Multiple linear regression adjusting for birth weight for gestational age. *Maternal age, parity, marital status, sex of child, birth weight for gestational age.* *Smoking not found to differ between groups so excluded.*
Torgersen (2008) *Norway*	BN-purge (n=118) BN-nonpurge (n=109) BN-other (n=63) EDNOS-P (n=42) Unexposed group (n=37,748). *ED over 6 month period prior to pregnancy and during pregnancy (up to 18 weeks).*	Prospective cohort study. *Pregnancies in Norway from 1999-unclear end point.*	All participants recruited via the Norwegian Mother and Child Cohort Study (MoBa) at 17 weeks of gestation. *ED and non-ED differentiated via self-report questionnaire broadly based on DSM-IV at 17 weeks of pregnancy.* *Outcomes obtained from self-report at 30 weeks retrospectively covering 8 epochs of gestation.* *Covariate data via self-report or MBRN.*	Odds ration from logistic regression *Age, stage of gestation.* *Marital status, education, income not found to relate to outcomes so excluded.*
Ulman (2012) *Norway*	BED before and during (n=1,495) BED remitted (n=921) BED during (n=1,235) Unexposed group (n=68,784).	Prospective cohort study. *Pregnancies in Norway from 1999-2008.*	All participants recruited via the Norwegian Mother and Child Cohort Study (MoBa) at 17 weeks of gestation. *BED and non-BED differentiated via self-*	Kruskal-Wallis test for continuous variables, Chi-square for categorical variables. Logistic regression with GEE to incorporate covariance of time. Odds

1st author (year), *location*	Sample *ED status*	Study design *Periods of data collection*	Method of recruitment *Measurement of variables*	Analysis *Covariates*
	ED status categorised as before pregnancy (6-month period) and during pregnancy (up to 18 weeks) vs. remitted during pregnancy vs. during pregnancy only.		*report questionnaire broadly based on DSM-IV.* *Outcomes obtained from self-reported covering time periods during pregnancy (Yes/no sleep problems and how much sleep).* *Covariate data via self-report or MBRN.*	ratios calculated. *Time, maternal age, gestational age at birth, sex of child, parity, parental income, smoking, education, anxiety and depression, interaction between anxiety and depression.*
Wentz (2009) *Göteborg, Sweden*	AN (n=48; 27 delivered 57 babies) Unexposed (n=48; 31 delivered 51 babies), matched for age and school. *Mean age of AN onset at 14. 3 had ED at time of conception.*	Prospective cohort study. *Children born between 1969 and 1977, selected at mean age of 14 years and followed up 18 years later at mean age 32 years.*	Children recruited from all schools in Göteborg by school nurses. *AN and non-AN differentiated according to DSM-III-R and DSM-IV criteria at age 14. Unclear what tool used. AN assessed again at age 32 by psychiatrist using SCID according to DSM-IV.* *Outcomes obtained from* face-to-face or telephone interview at age 32.	Mann-Whitney for continuous variables. Chi-square of Fisher's Exact test for dichotomous variables. *Matched for age and school. No covariates identified.*

Appendix 2. The STROBE Statement- checklist of items that should be addressed in reports of observational studies

	Item no.	Recommendation
TITLE and ABSTRACT		
	1	(a) Indicates study design with a commonly used term in the title or abstract (b) Provides in the abstract an informative and balanced summary of what was done and what was found
INTRODUCTION		
Background/ rationale	2	Explain the scientific background and rationale for the investigation being reported
Objectives	3	State specific objectives including any pre-specified hypotheses
METHODS		
Study design	4	Present key elements of study design early in the paper
Setting	5	Describe the setting, locations, and relevant dates, including period of recruitment, exposure, follow-up, and data collection
Participants	6	(a) *Cohort* - give eligibility criteria, and the sources and methods of selection of participants. Describes methods of follow-up. *Case-control*- give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for choice of cases and controls. *Cross-sectional*- give the eligibility criteria, and the sources and methods of selection of participants. (b) *Cohort study*- for matched studies, give matching criteria and number of exposed and unexposed *Case-control*- for matched studies, give matching criteria and number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable.
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group
Bias	9	Describe any efforts to address potential sources of bias
Study size	10	Explain how the study size was arrived at
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding (*b*) Describe any methods used to examine subgroups and interactions (*c*) Explain how missing data were addressed (*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy (*e*) Describe any sensitivity analyses
RESULTS		
Participants	13*	(a) Report numbers of individuals at each stage of study—e.g. numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage (c) Consider use of a flow diagram
Descriptive data	14*	(a) Give characteristics of study participants (e.g. demographic, clinical,

	Item no.	Recommendation
		social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) *Cohort study*—Summarise follow-up time (e.g., average and total amount)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time *Case-control study*—Report numbers in each exposure category, or summary measures of exposure *Cross-sectional study*—Report numbers of outcome events or summary measures
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (e.g., 95% confidence interval). Make clear which confounders were adjusted for and why they were included (*b*) Report category boundaries when continuous variables were categorized (*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period
Other analyses	17	Report other analyses done—e.g. analyses of subgroups and interactions, and sensitivity analyses
DISCUSSION		
Key results	18	Summarise key results with reference to study objectives
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence
Generalisability	21	Discuss the generalisability (external validity) of the study results
OTHER INFORMATION		
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based

*Give such information separately for cases and controls in case-control studies, and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Appendix 3. Adapted quality criteria based on the STROBE Statement checklist items

Quality subsections	Corresponding STROBE Item no.	Quality checklist elements derived from STROBE	Additional checklist items pertinent to review question
Methods			
1. Setting	5	Clear location, setting, and dates of recruitment/data collection/ follow-up?	Are cases identified according to inpatient/community sample? Are the comparison group taken from a comparable location? Are cases recruited according to date of first presentation or according to date of outcomes? Is it therefore possible outcomes predate ED? Does the study miss certain people from the population?
2. Participants	6	Was appropriate eligibility criteria utilised for participant groups? Were methods of follow up appropriate? Were methods of recruitment for cases and comparison groups appropriate? Were cases and controls selected/chosen appropriately? Were cases matched? If so, were the matching criteria appropriate? And were the number of controls justified?	Have multiple births been excluded? Are only first pregnancies included so as to eliminate dependence of variables? Have ED been excluded from the comparison group? Has co-morbidity been accounted for?
3. Variables	7	Are the exposure, outcome and potential confounder variables appropriately defined? (i.e. Diagnostic criteria)	Is ED status at time of pregnancy clear or recency to pregnancy considered? Was severity of ED considered? Were all subtypes of ED dealt with appropriately? Were cut offs for incidence of dichotomous data defined and appropriate (e.g. prematurity at <37 weeks)? Have the same definitions been applied to cases and controls?
4. Data sources/ measurement	8	Are the sources of data and methods of assessment appropriate and comparable for each group?	Were EDs, outcomes and covariates measured objectively?
5. Study size	10	Was the study size adequate for each of the groups?	
6. Quantitative variables	11	Were the variables handled appropriately in the analysis? If groupings were used were these justified?	Were any variables not included in analysis? If so, was this justified? Was grouping of ED subtypes, outcomes or covariates (where

Quality subsections	Corresponding STROBE Item no.	Quality checklist elements derived from STROBE	Additional checklist items pertinent to review question
			carried out) justified?
7. Statistical methods	12	Were statistical methods appropriate, including those used to control for confounding variables? Were statistical methods to examine subgroups and interactions appropriate? Were missing data addressed? Cohort-was loss to follow-up addressed? Case-control-was matching of cases and controls addressed? Cross-sectional-did the method account for sampling strategy?	
Results			
8. Participants	13	Was a detailed breakdown of individuals included at each stage provided? Was non-participation justified at each stage?	
9. Descriptive data	14	Were characteristics of participants (demographic, clinical, and social) and information on exposures and potential confounders clear? Was the number of missing data for each variable indicated? Cohort-was follow-up time summarised?	
10. Outcome data	15	Cohort- number of outcome events reported? Case-control-numbers for each exposure category reported? Cross-sectional-outcome events reported?	
11. Main results	16	Were unadjusted and, if applicable, confounder adjusted estimates and their precision (e.g. 95% CI) reported? Were the confounders adjusted for clear and justified? Were category boundaries reported for continuous variables that were categorised?	Did they account for pre-pregnancy BMI or maternal weight gain?
12. Other analyses	17	Where applicable, were other analyses (of subgroups, interactions etc.) conducted appropriately?	
Internal and External Validity			

Quality subsections	Corresponding STROBE Item no.	Quality checklist elements derived from STROBE	Additional checklist items pertinent to review question
13. Generalisability	21	Are the results of the study generalisable (i.e. External validity)?	Consider, demographics, severity, ED status, date of sampling (i.e. changes in diagnosis/treatment of ED or perinatal complications),

Appendix 4: B-Eat advertisement

EARLY PARENTAL RELATIONSHIPS, EMOTION REGULATION AND EATING PSYCHOPATHOLOGY IN ADULTHOOD

Principal Investigator

Alexandra Stanbury, School of Psychology, University of Birmingham.

The research

We are conducting a study that investigates a person's relationships with their parents/ caregivers during childhood, how they manage their emotions when they become an adult and the kinds of eating difficulties they may experience. By exploring these things, we hope that information obtained will offer better support for healthcare professionals to provide the most effective interventions for people with eating difficulties in the future.

Who can take part?

We are looking for males and females to participate who:

1. Are fluent in English language and;
2. Are aged 18 and above and;
3. Have had problems with eating within the last four weeks (you do not need to have a diagnosis) and;
4. May or may not have another known mental health problem such as anxiety or depression.

What does the study involve?

If you choose to take part in this study, you will be asked a series of online questions about you, your upbringing, your emotions, and your difficulties with eating including your current weight. Your responses will remain anonymous.

Your participation should take no more than 30 minutes to complete.

What should I do if I am interested?

If you think you might want to take part, the link below will take you to the website where you will be given further information. Simply follow the instructions if you wish to continue.

If you would like more details or have any questions, please contact **Alexandra Stanbury** (

Thank you for your time.

https://lesweb2.bham.ac.uk/surveys/index.php?r=survey/index/sid/352861/lang/en

Appendix 5: University advertisement

EARLY PARENTAL RELATIONSHIPS, EMOTION REGULATION AND EATING PSYCHOPATHOLOGY IN ADULTHOOD

My name is Alexandra Stanbury and I am a Trainee Clinical Psychologist at the University of Birmingham.

I would be grateful if you would read this email and consider taking part in my online research study.

The research

We are conducting a study that investigates a person's relationships with their parents/ caregivers during childhood, how they manage their emotions when they become an adult and the kinds of eating difficulties they may experience. By exploring these things, we hope that information obtained will offer better support for healthcare professionals to provide the most effective interventions for people with eating difficulties in the future.

Who can take part?

We are looking for males and females to participate who:

1. Are fluent in English language and;
2. Are aged 18 and above

What does the study involve?

If you choose to take part in this study, you will be asked a series of online questions about you, your upbringing, your emotions, and your eating. Your responses will remain anonymous.

Your participation should take no more than 30 minutes to complete.

What should I do if I am interested?

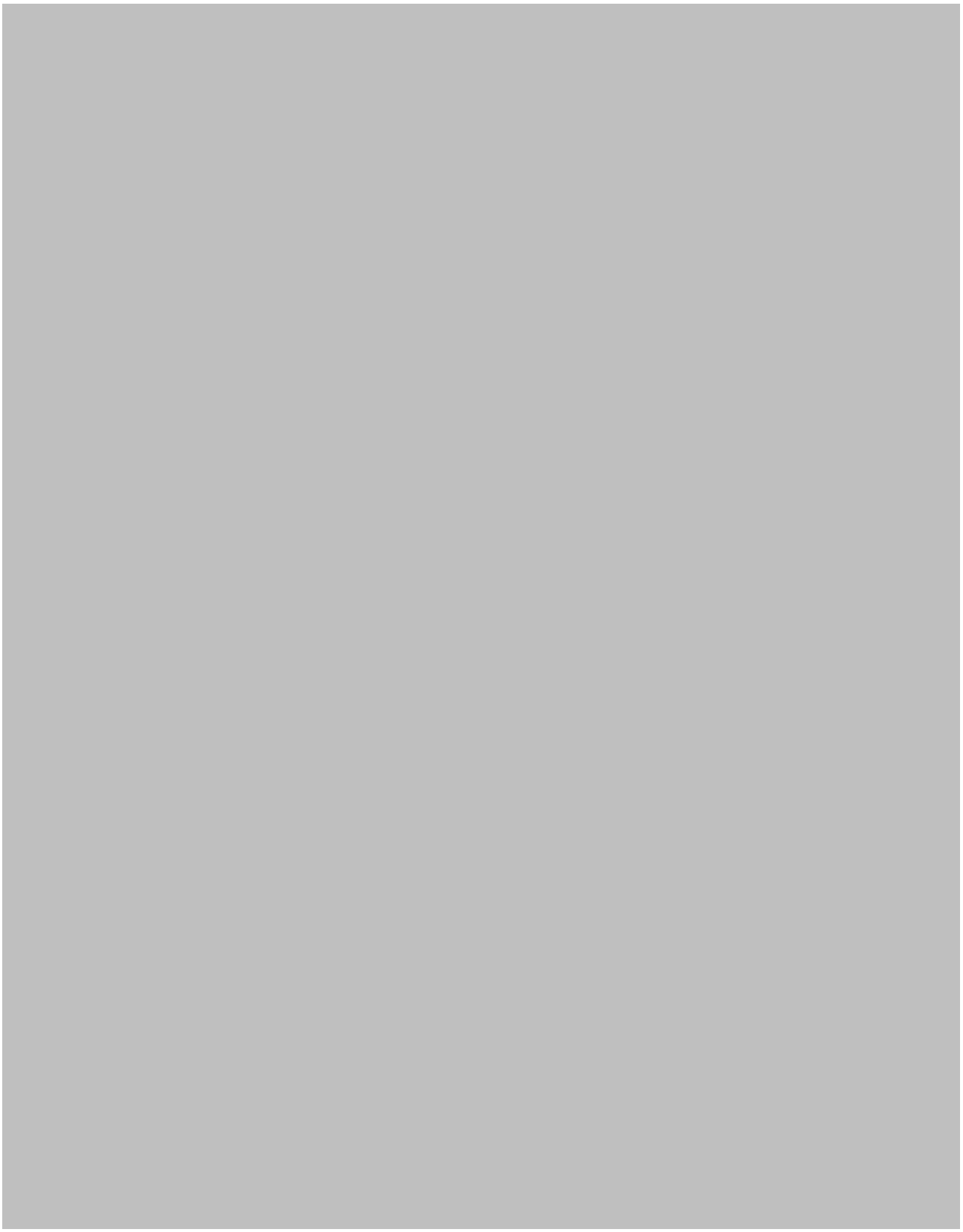
If you think you might want to take part, the link below will take you to the website where you will be given further information. Simply follow the instructions if you wish to continue.

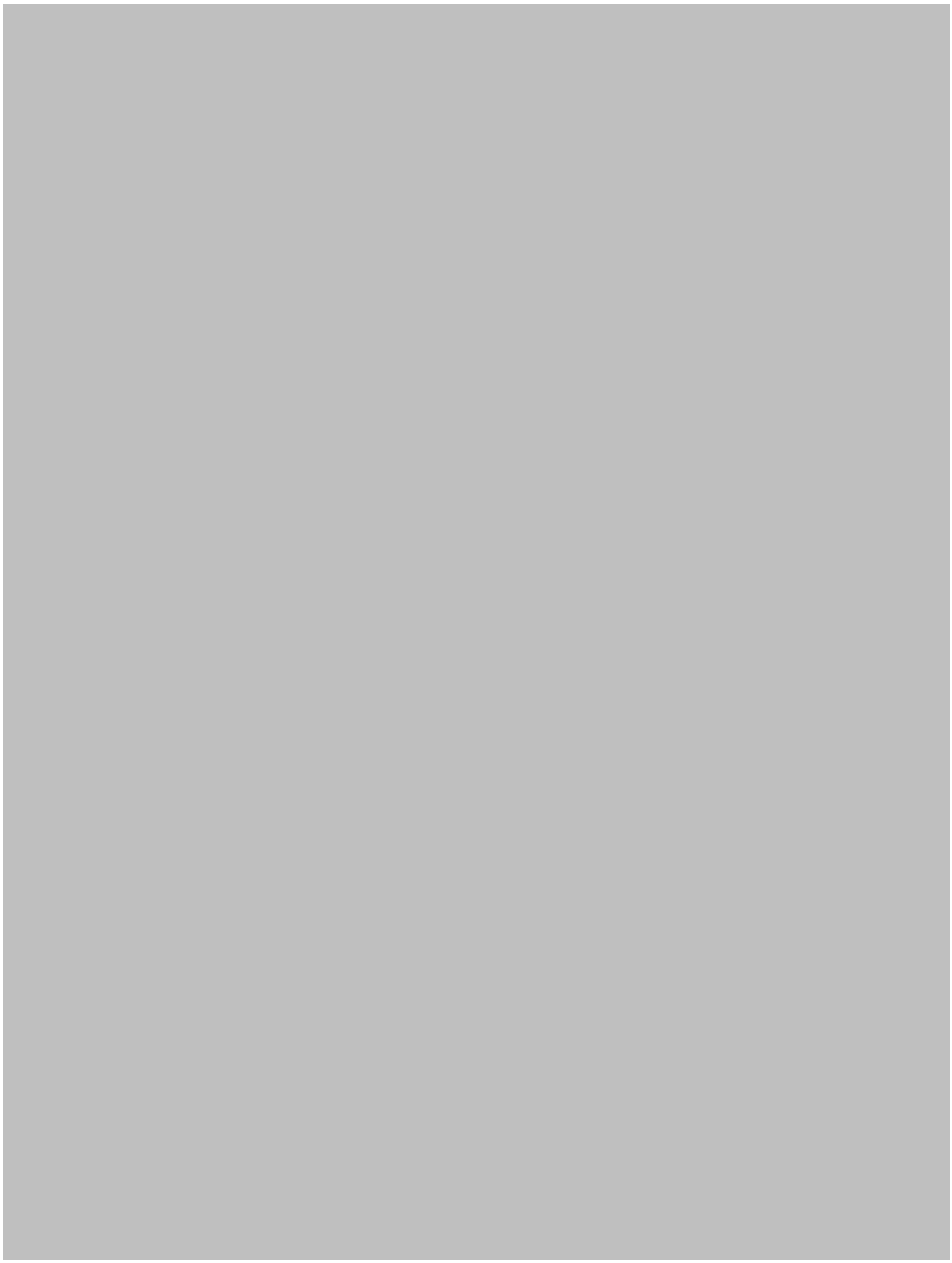
If you would like more details or have any questions, please contact **Alexandra Stanbury** (

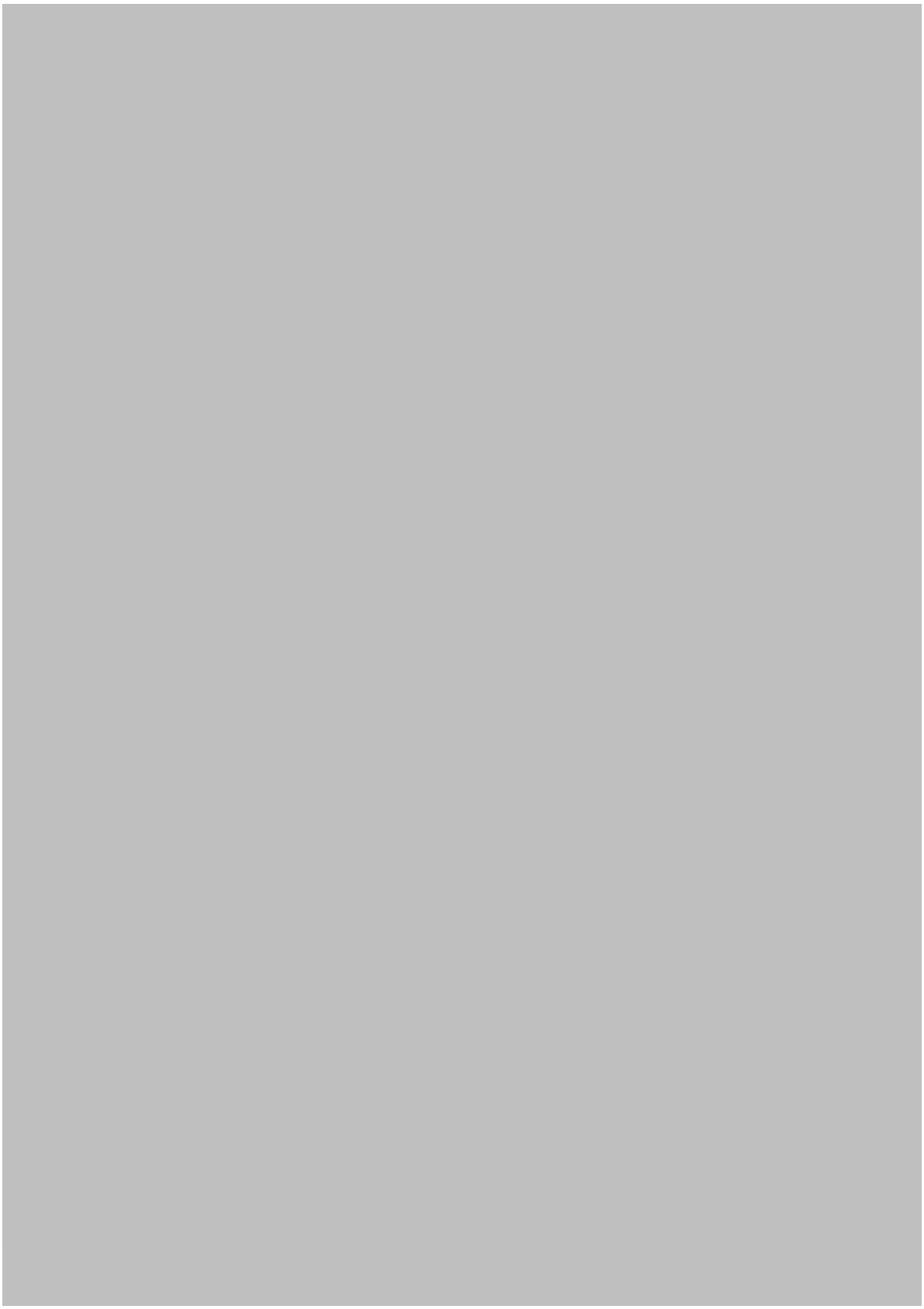
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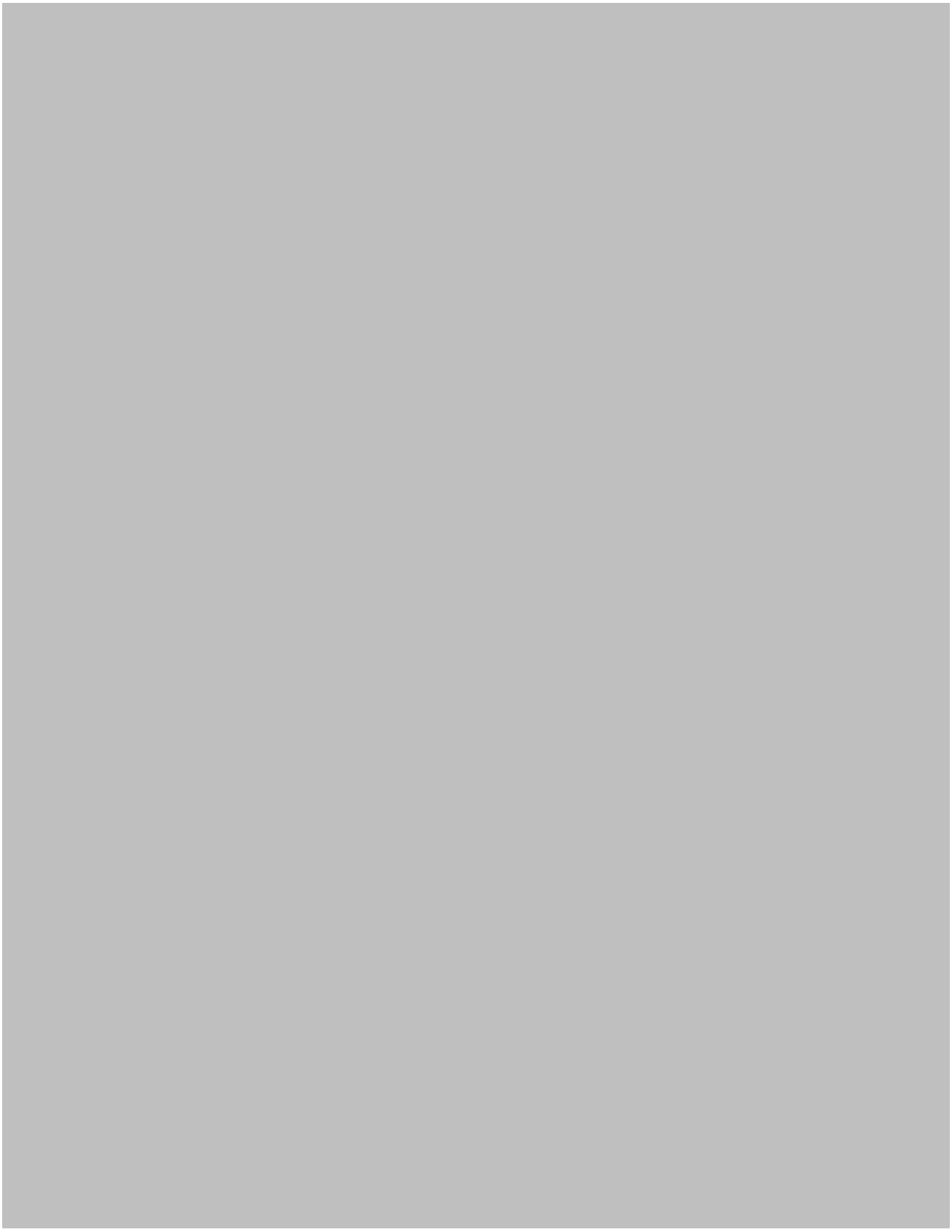
https://lesweb2.bham.ac.uk/surveys/index.php?r=survey/index/sid/716383/lang/en

Appendix 6: NHS Ethics approval









Appendix 7: B-Eat information and consent sheet

INFORMATION AND CONSENT SHEET

Title of Project: **Early parental relationships, emotion regulation and eating psychopathology in adulthood**

Name of Researcher: **Alexandra Stanbury, School of Psychology, University of Birmingham**

The research

We are conducting a study that investigates a person's relationships with their parents/ caregivers during childhood, how they manage their emotions when they become an adult and the kinds of eating difficulties they may experience. By exploring these things, we hope that information obtained will offer better support for healthcare professionals to provide the most effective interventions for people with eating difficulties in the future.

Who can take part?

We are looking for males and females to participate who:

1. Are fluent in English language and;
2. Are aged 18 and above and;
3. Have had problems with eating within the last four weeks (you do not need to have a diagnosis) and;
4. May or may not have another known mental health problem such as anxiety or depression.

What does the study involve?

If you choose to take part in this study, you will be asked a series of online questions about you, your upbringing, your emotions, and your difficulties with eating. The questions asked may be of a sensitive nature. Please make a note of the **B-Eat website: http://www.B-Eat.co.uk/support-services** and **helpline: 0845 634 1414**. B-Eat are able to provide support if you are experiencing distress related to your eating. If you are receiving professional support for this already, you are advised to discuss this study with your clinician before taking part. If you are not currently receiving support with your eating disorder and you become distressed when answering the online questions, seek the help of your GP who will be able to provide you with information and further guidance.

You can withdraw your participation at any time throughout your completion of the questionnaires. You do not have to give a reason for this. Just click on the "x" at the top right of the page. However, as we are unable to identify individual responses, once you press the "submit" button, you will be unable to withdraw.

Your participation is voluntary and does not affect any other care or treatment that you may receive/ are receiving.

Your responses will remain anonymous. We will not be asking you to provide your name, or any other information that may identify you.

Your participation should take no more than 30 minutes to complete.

What if I have more questions?

If you would like more details or have a question/s, please contact **Alexandra Stanbury**

What do I do now?

If you think you might want to take part, please read the consent information below. Having agreed to the conditions outlined, click the "next" icon to continue to the questionnaires.

Thank you for your time.

CONSENT

By clicking the link below, I am confirming that I:

1. Am 18 years of age or above
2. Have had problems with eating within the last four weeks
3. Am fluent in the English language
4. Have read, and understood, the preceding information sheet
5. Am willing to take part, but know that I can withdraw at any time prior to pressing the "submit" button, by clicking on the "x" in the top right hand corner.

LINK

Appendix 8: University information and consent sheet

INFORMATION AND CONSENT SHEET

Title of Project: **Early parental relationships, emotion regulation and eating psychopathology in adulthood**

Name of Researcher: **Alexandra Stanbury, School of Psychology, University of Birmingham**

The research

We are conducting a study that investigates a person's relationships with their parents/ caregivers during childhood, how they manage their emotions when they become an adult and the kinds of eating difficulties they may experience. By exploring these things, we hope that information obtained will offer better support for healthcare professionals to provide the most effective interventions for people with eating difficulties in the future.

Who can take part?

We are looking for any males and females to participate who:

1. Are fluent in English language and;
2. Are aged 18 or above

What does the study involve?

If you choose to take part in this study, you will be asked a series of online questions about you, your upbringing, your emotions, and any difficulties with eating. The questions asked may be of a sensitive nature. Please make a note of the **B-Eat website: http://www.B-Eat.co.uk/support-services** and **helpline: 0845 634 1414**. B-Eat are able to provide support if you are experiencing distress related to your eating. If you become distressed when answering the online questions, seek the help of your GP who will be able to provide you with information and further guidance.

You can withdraw your participation at any time throughout your completion of the questionnaires. You do not have to give a reason for this. Just click on the "x" at the top right of the page. However, as we are unable to identify individual responses, once you press the "submit" button, you will be unable to withdraw.

Your participation is voluntary and does not affect any other care or treatment that you may receive/ are receiving.

Your responses will remain anonymous. We will not be asking you to provide your name, or any other information that may identify you.

Your participation should take no more than 30 minutes to complete.

What if I have more questions?

If you would like more details or have a question/s, please contact **Alexandra Stanbury** ( ).

What do I do now?

If you think you might want to take part, please read the consent information below. Having agreed to the conditions outlined, click the "next" icon to continue to the questionnaires.

Thank you for your time.

CONSENT

By clicking the link below, I am confirming that I:

1. Am 18 years of age or above
2. Am fluent in the English language
3. Have read, and understood, the preceding information sheet
4. Am willing to take part, but know that I can withdraw at any time prior to pressing the "submit" button, by clicking on the "x" in the top right hand corner.

LINK

Appendix 9: B-Eat debrief

DEBRIEF

Title of Project: **Early parental relationships, emotion regulation and eating psychopathology in adulthood**

Name of Researcher: **Alexandra Stanbury, School of Psychology, University of Birmingham**

Thank you for taking part in this study. As outlined at the beginning, we aim to investigate early relationships with carers, ability to manage emotions and difficulties with eating. By exploring these areas we will be contributing to the literature that guides appropriate interventions for people who have difficulties with eating.

You may have found some of the questions in this study distressing. If you are experiencing distress, B-Eat are able to provide support via their **website**: **http://www.B-Eat.co.uk/support-services** and their **helpline: 0845 634 1414.** If you are already receiving professional support for your eating difficulties you are advised to speak with your clinician. Alternatively, if you are not currently receiving support with eating and you have become distressed by some of the questions, please seek the guidance of your GP.

You have now submitted your responses and we are unable to withdraw them from the study. This is because your answers are anonymous and we cannot identify which questionnaire is yours.

Should you wish to have a summary of the outcome of the study in general, this will be available on the B-Eat website (below) once the study has been completed:

http://www.B-Eat.co.uk/research/summary-of-completed-research

If you have any further questions about the study specifically, please contact **Alexandra Stanbury** [redacted]). Please refer to the above guidance for seeking clinical support. This email address is not intended for this purpose.

Thank you again for your time.

Appendix 10: University debrief

DEBRIEF

Title of Project: **Early parental relationships, emotion regulation and eating psychopathology in adulthood**

Name of Researcher: **Alexandra Stanbury, School of Psychology, University of Birmingham**

Thank you for taking part in this study. As outlined at the beginning, we aim to investigate early relationships with carers, ability to manage emotions and difficulties with eating. By exploring these areas we will be contributing to the literature that guides appropriate interventions for people who have difficulties with eating.

You may have found some of the questions in this study distressing. If you are experiencing distress regarding your eating, B-Eat is a national charity based in the UK. They provide information, help and support to people affected by eating disorders and can be accessed via their **website:** http://www.B-Eat.co.uk/support-services or via their **helpline: 0845 634 1414.** Alternatively, if you have become distressed by some of the questions, please seek the guidance of your GP.

You have now submitted your responses and we are unable to withdraw them from the study. This is because your answers are anonymous and we cannot identify which questionnaire is yours.

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If you any further questions about the study specifically, please contact **Alexandra Stanbury** [redacted] Please refer to the above guidance for seeking clinical support. This email address is not intended for this purpose.

Thank you again for your time.

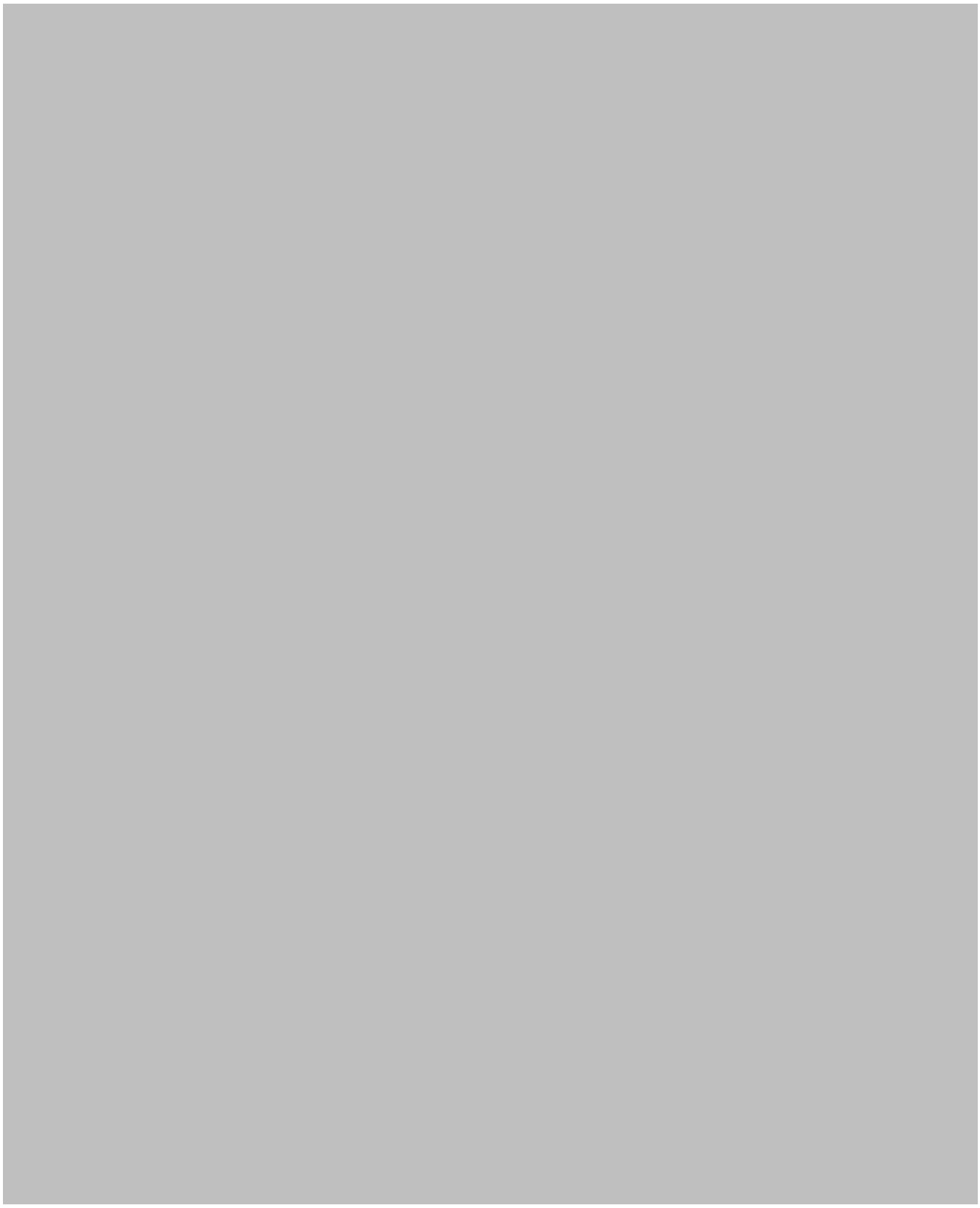
Appendix 11: Comparison of demographics for those included to those excluded

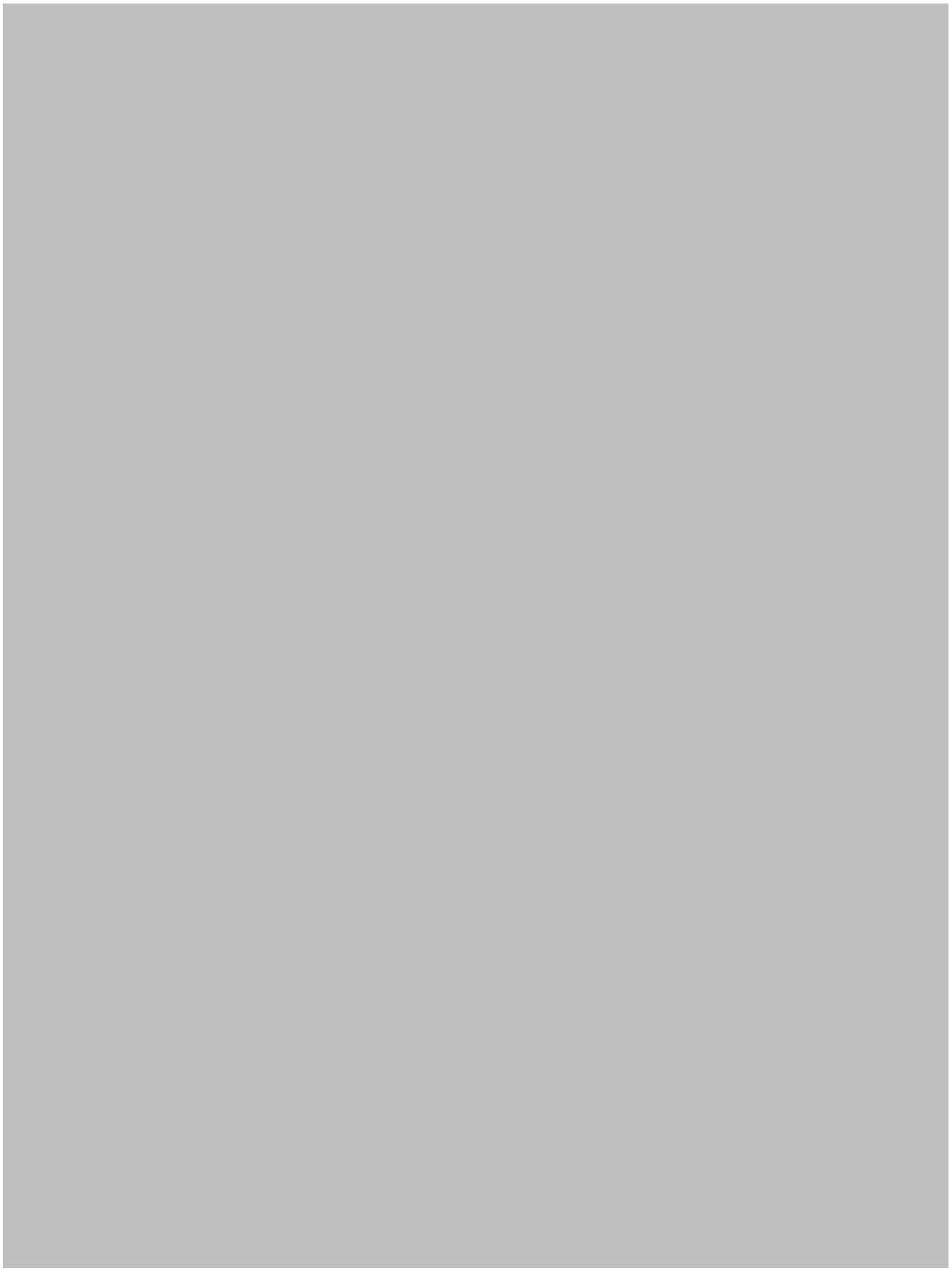
		Excluded (n=70)	Included (n=241)	Bootstrap for independent samples t-test (95% CI)	
		Mean (S.D)	Mean (S.D)	Lower	Upper
Demographics	Age in years	29.17 (11.83)	27.40 (8.86)	-1.10	4.80
	Current weight (Kg)	65.34 (24.27)	61.83 (16.89)	-2.38	9.46
	Current height (m)	1.66 (0.07)	1.67 (0.07)	-0.04	0.00
	BMI	23.71 (8.60)	22.01 (5.67)	-0.37	3.81

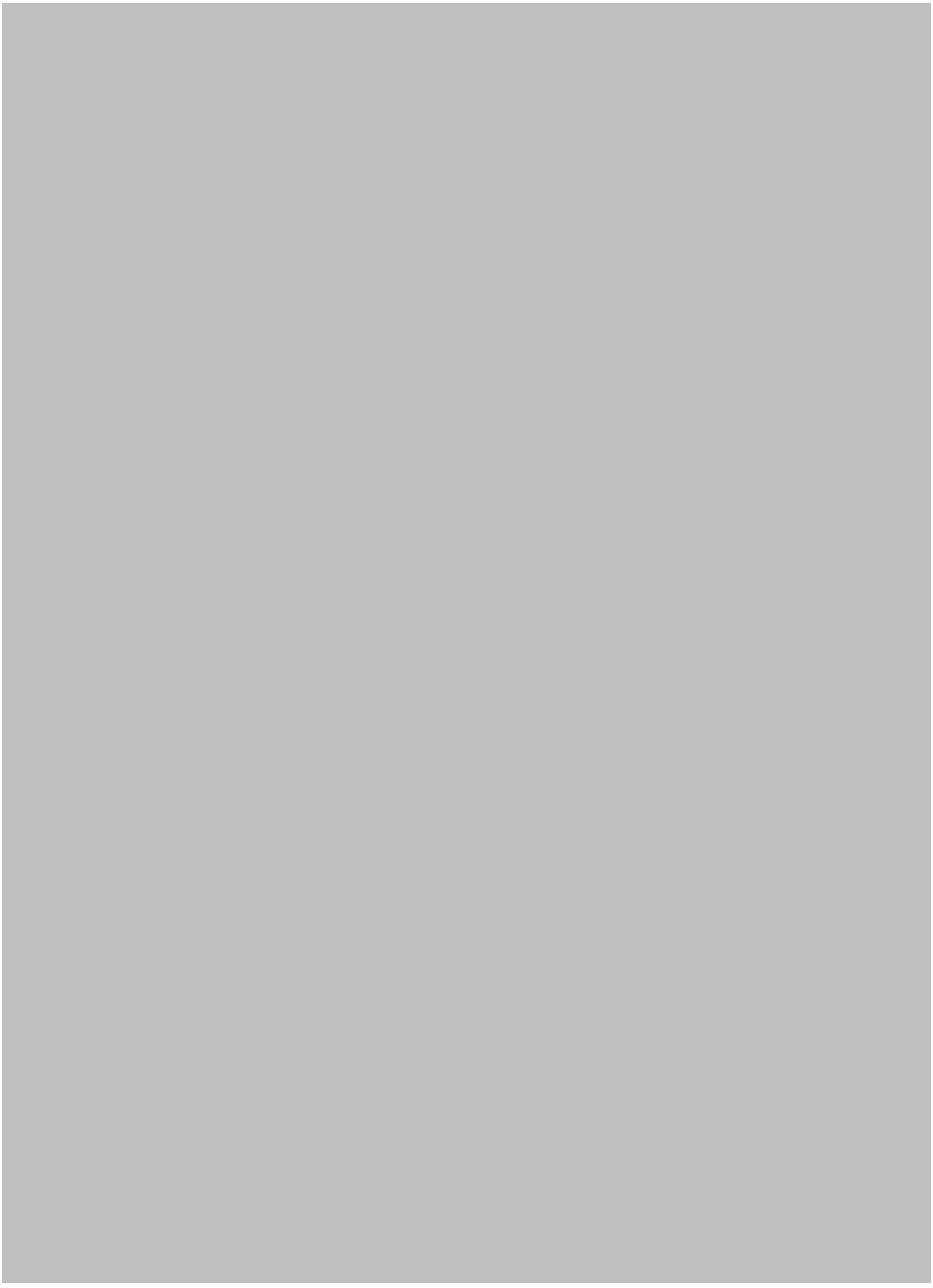
Appendix 12: Comparison of subscale scores for those included to those excluded

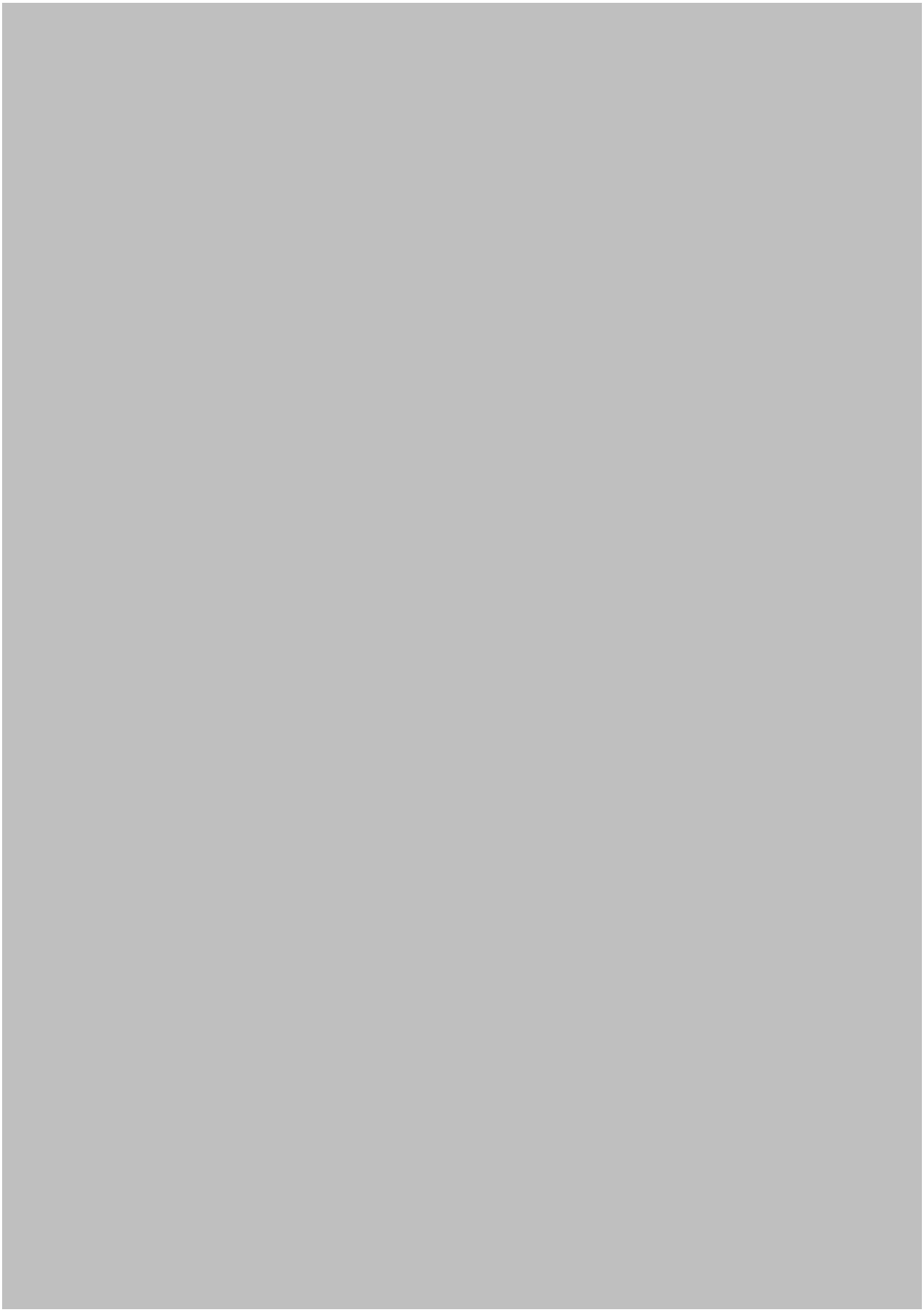
Measure	Subscale	Excluded (n=67)	Included (n=227)	Bootstrap for independent samples t-test (95% CI)	
		Mean (S.D)	Mean (S.D)	Lower	Upper
EMBU	Rejection- mother	1.93 (0.76)	1.81 (0.78)	-0.10	0.34
	Rejection- father	1.89 90.73)	1.78 (0.80)	-0.10	0.31
	Warmth- mother	2.64 (0.83)	2.79 (0.84)	-0.37	0.08
	Warmth- father	2.47 (0.87)	2.60 (0.86)	-0.37	0.12
	Overprotection- mother	2.19 (0.69)	2.43 (0.73)	-0.44	-0.03
	Overprotection-father	1.92 (0.65)	2.06 (0.69)	-0.32	0.04
DERS	Non-acceptance	20.63 (6.73)	2.20 (7.78)	-1.57	2.35
	Goals	17.03 (4.71)	17.11 (4.82)	-1.39	1.27
	Impulsivity	17.12 (6.04)	17.08 (7.37)	-1.72	1.71
	Awareness	18.13 (5.96)	18.29 (6.37)	-1.80	1.36
	Strategies	25.25 (7.73)	24.06 (9.57)	-2.16	2.36
	Clarity	15.07 (5.20)	14.58 (5.34)	-0.87	1.94
	Total	112.24 (27.49)	111.33 (34.38)	-7.41	9.13
EDE-Q	Restraint	2.58 (1.60)	3.16 (2.06)	-1.05	-0.13
	Eating concerns	2.75 (1.37)	2.91 (2.15)	-0.61	0.28
	Shape concerns	3.95 (1.27)	4.05 (1.95)	-0.52	0.28
	Weight concerns	3.39 (1.35)	3.66 (2.02)	-0.71	0.12
	Global score	3.17 (1.07)	3.44 (1.94)	-0.66	0.07

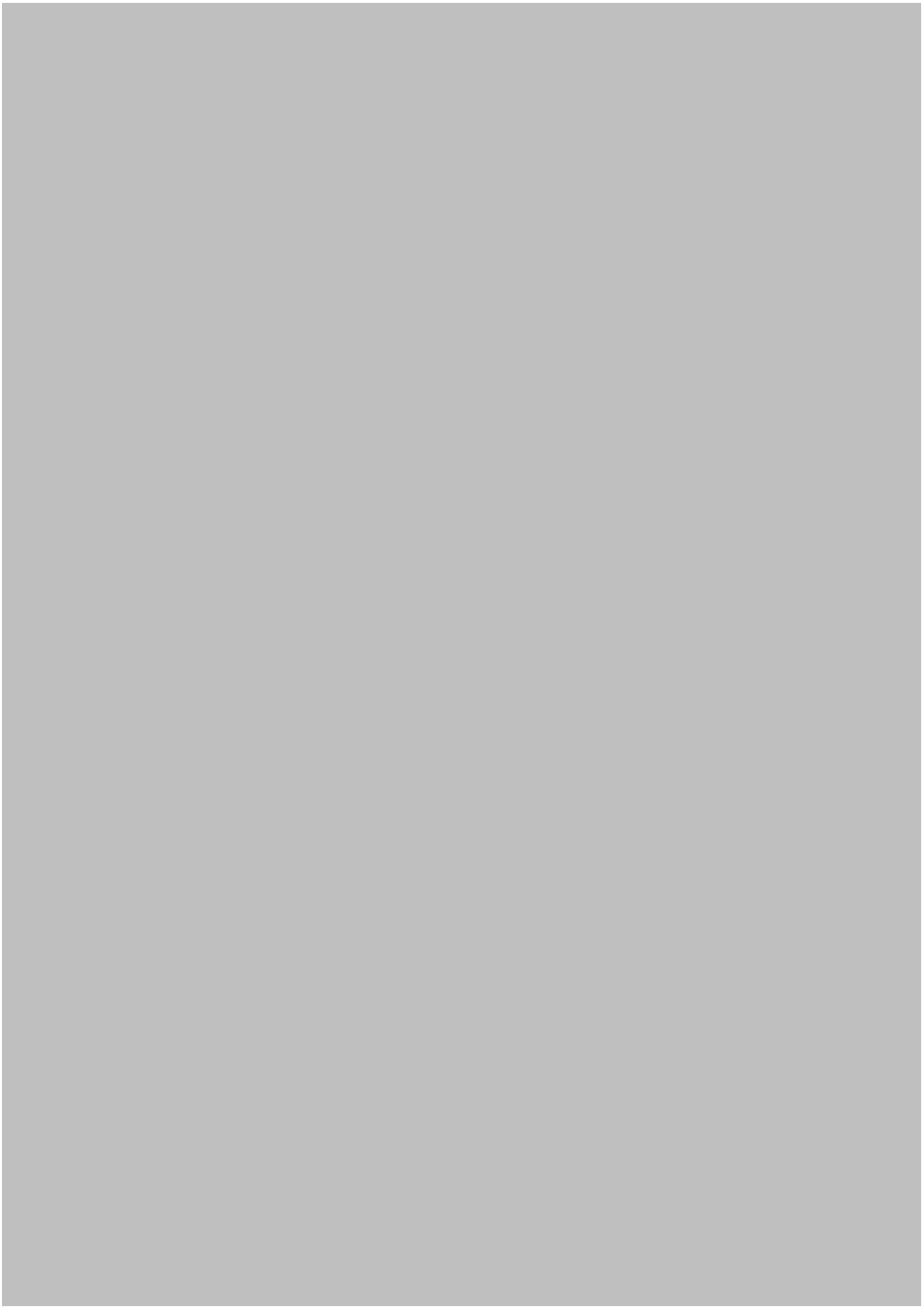
Appendix 13: Instruction for authors- British Journal of Obstetrics and Gynaecology

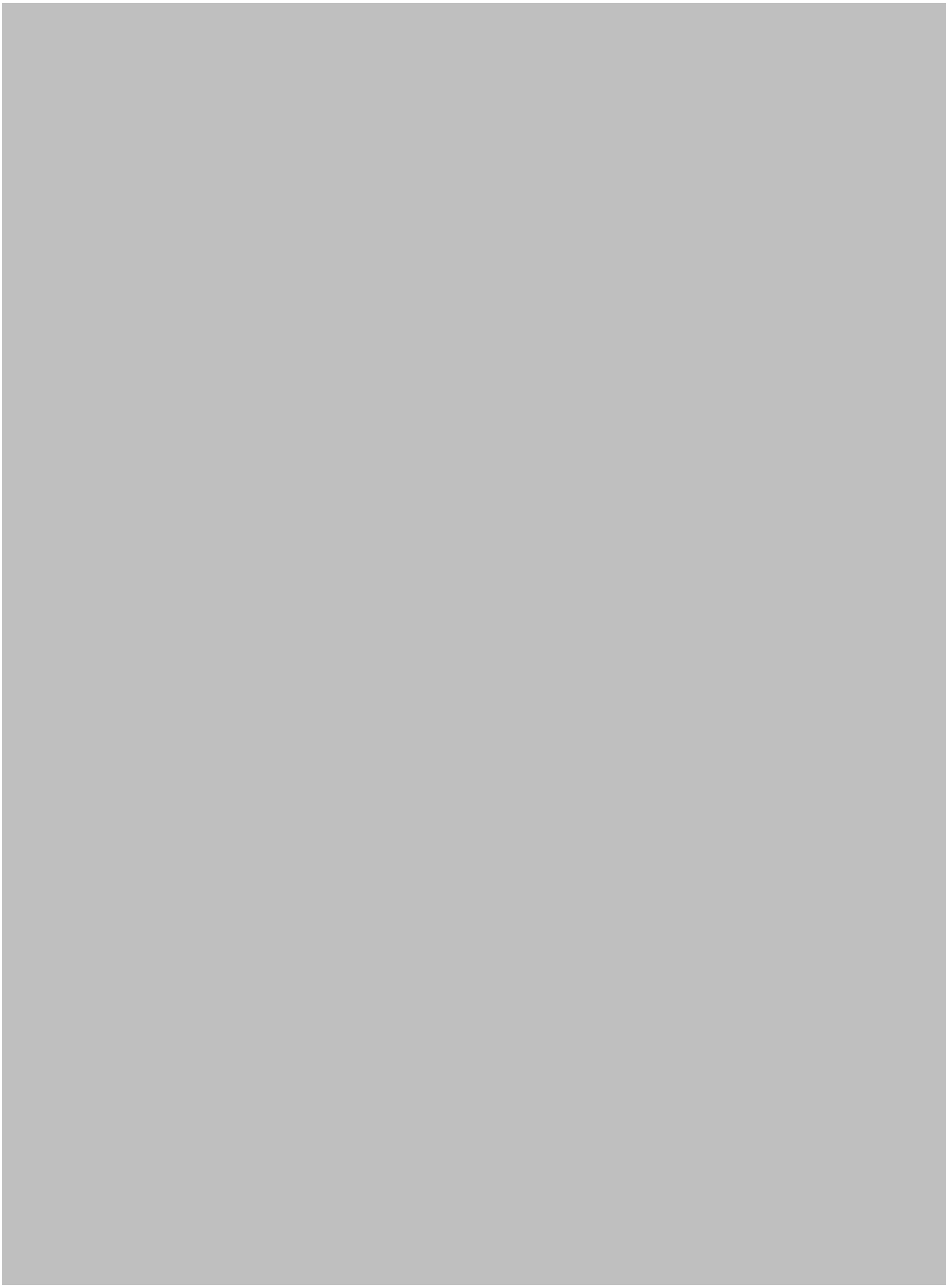


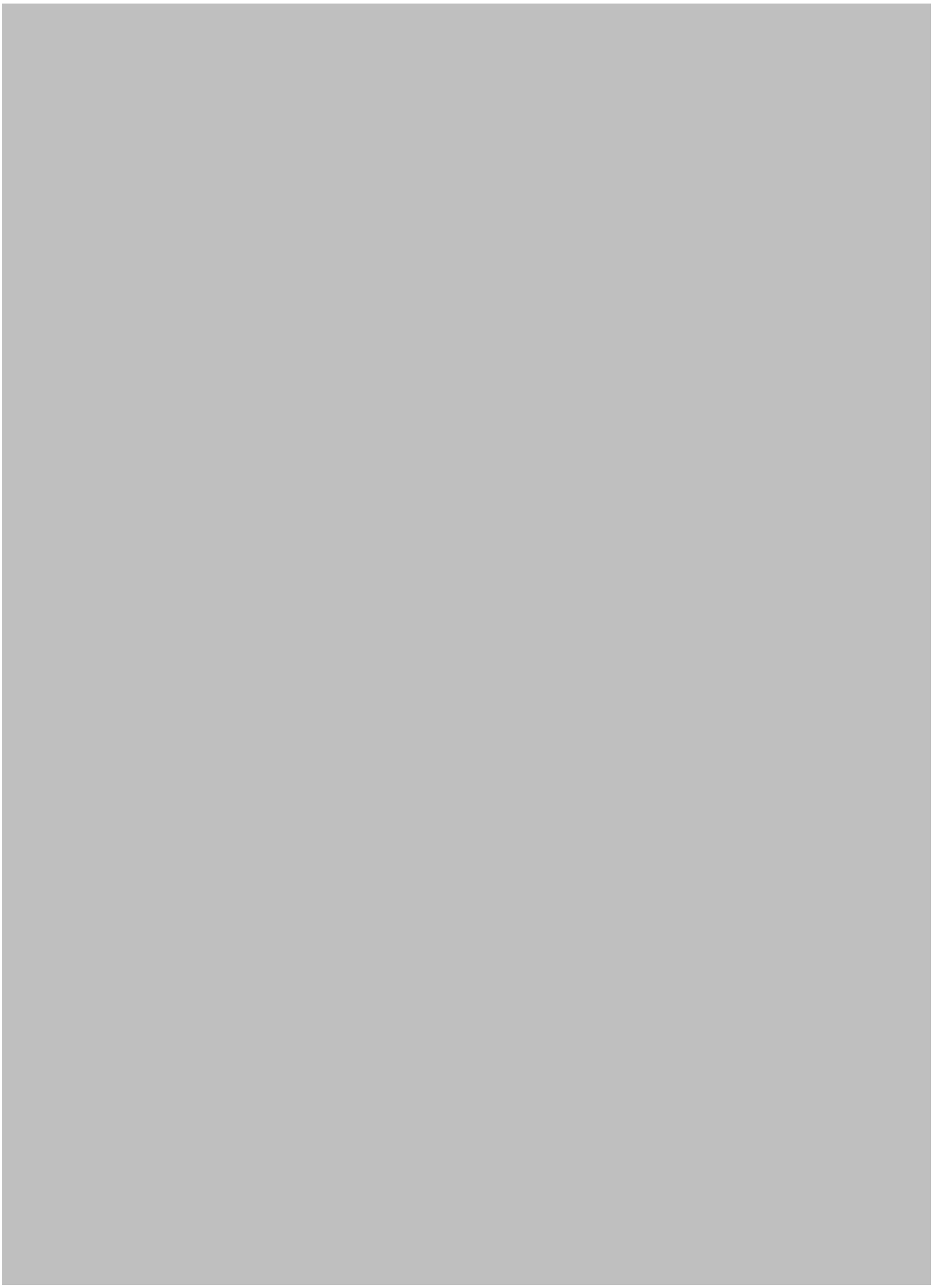


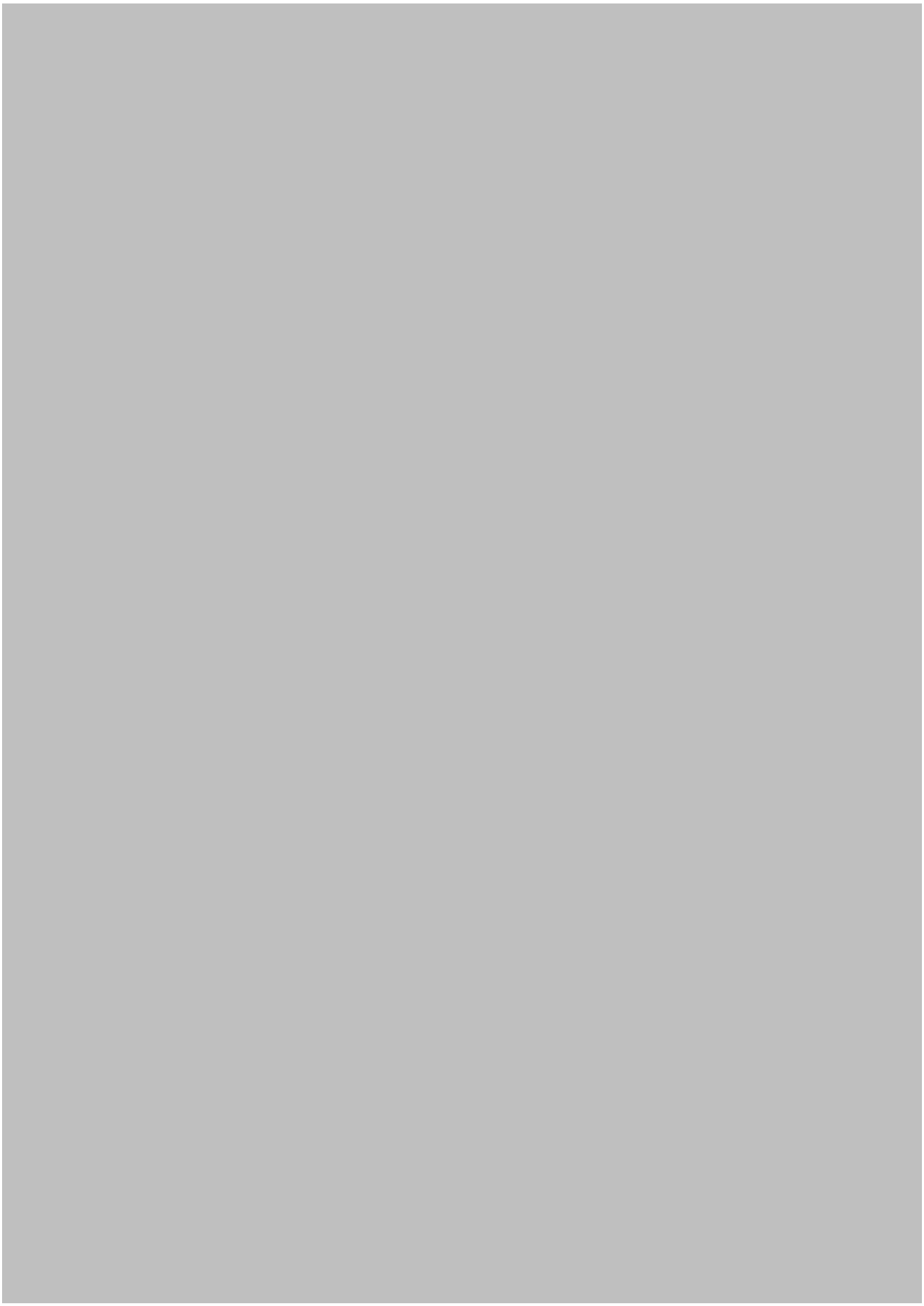


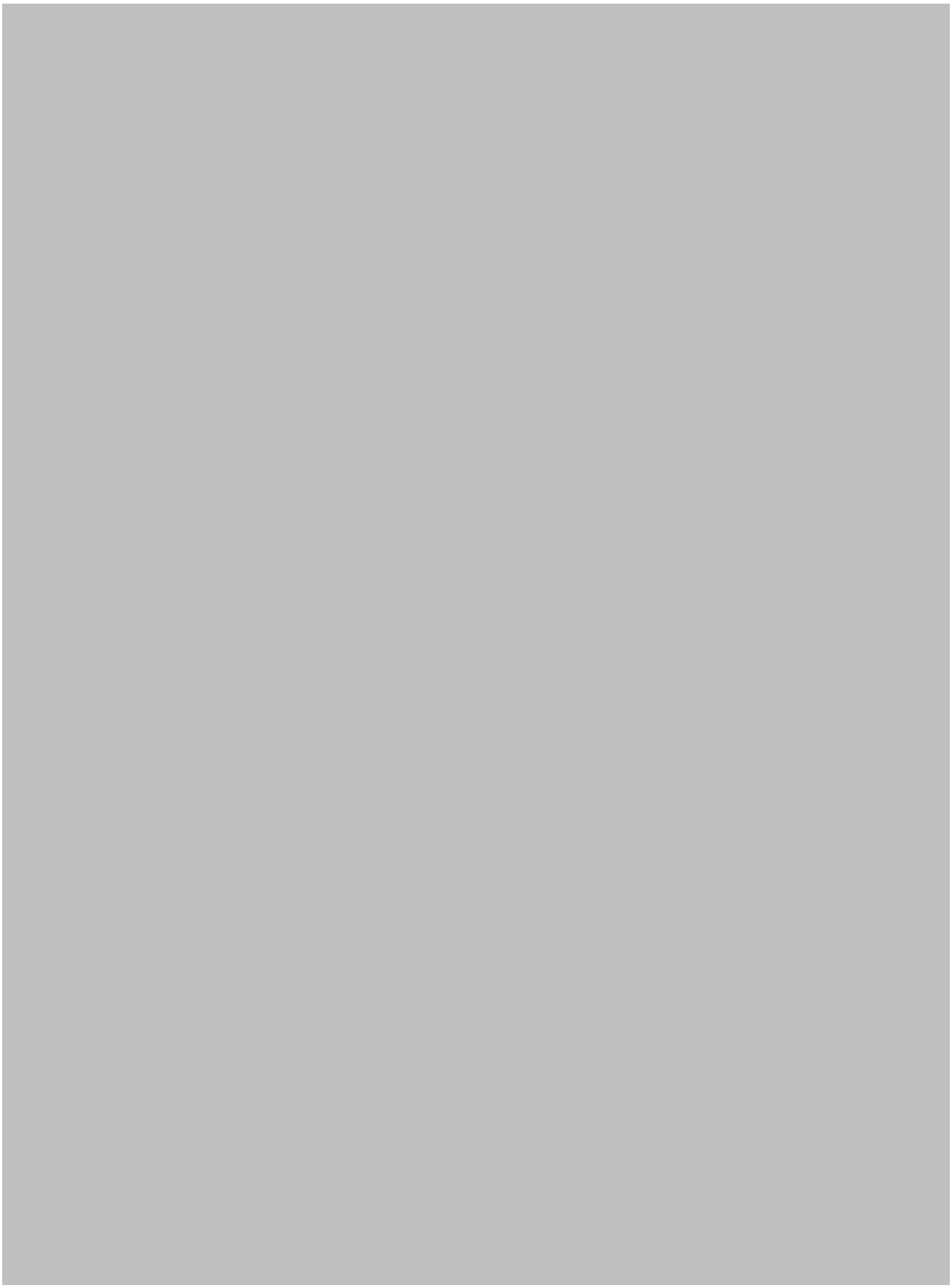














Appendix 14: Instruction for authors- British Journal of Clinical Psychology

