VOLUME 1

An Investigation into the Impact of Childhood Abuse and Care-giver Invalidation on Psychological Inflexibility in Clinical and Subclinical Eating Disorders

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1 ABSTRACT

As a whole, eating disorders have been characterised as having the following key features: a persistent over concern with body size and shape; and weight control behaviours such as fasting, exercise, and self-induced vomiting. However, there tends to be a blurred line between those that do and do not meet diagnostic thresholds as the level of psychological distress is comparably similar. This study examined whether psychological inflexibility (from an Acceptance and Commitment Therapy perspective) was associated with eating disorders and whether it mediated the relationship between childhood abuse and invalidation and eating disorders. This was considered to be important because high rates of abuse have consistently been found in this population, yet not everyone goes on to develop an eating disorder. In addition, the role of emotional abuse has been largely neglected. A clinical sample of 190 participants with a clinical or subclinical eating disorder were recruited from eating disorder charities and support forums; they completed a range of questionnaires measuring experiences of abuse and maternal/paternal emotional invalidation in childhood, current levels of cognitive fusion and experiential avoidance and current levels of eating pathology. The sample was split into three groups based on their Eating Disorder Risk Composite scores: elevated, typical and low clinical range. It was found that those in the elevated clinical range (most severe eating pathology) had the poorest emotional processing and significantly higher levels of psychological inflexibility, thought-shape-fusion, depression and anxiety than those in the low clinical range (least severe eating pathology). In terms of predicting current levels of eating pathology, three variables emerged as significant predictors: emotional processing, thought-shape fusion and depression. In terms of predicting current levels of psychological inflexibility, five variables emerged as significant predictors: childhood emotional abuse, emotional processing, thought-shape-fusion, depression and anxiety. The results add novel findings to the literature regarding the role of early experiences on the development of psychological inflexibility, and the role of psychological inflexibility in the maintenance of eating pathology and psychological distress. Clinical implications of these findings in relation to assessment, formulation, intervention and prevention are discussed.

2 INTRODUCTION

2.1 Overview

This study examined contextual factors associated with psychological inflexibility and distress in clinical and subclinical eating disorders (EDs). This introduction begins with an outline of clinical and subclinical EDs then orients the reader to the concepts of childhood abuse and invalidation. It then moves on to an examination of the theoretical and empirical relationships between these two topics, before outlining the theoretical and philosophical basis of Acceptance and Commitment Therapy (ACT; Hayes, Strosahl, & Wilson, 1999). An overview of the ACT (said as one word, not individual letters) approach is given along with a discussion of the concepts of experiential avoidance and cognitive fusion; two of six processes which make up psychological inflexibility. This is followed by a conceptualisation of how EDs and childhood abuse & invalidation might be understood from an ACT perspective. A brief review of the literature linking ACT and eating pathology is then presented, followed by a brief review of the literature linking ACT and childhood abuse and invalidation. This introduction concludes with the rationale for this study, its clinical relevance and aims and hypotheses.

2.2 Literature search strategy

A preliminary search for review papers was carried out using the Clinical Psychology review journal, the Annual Reviews database and the Cochrane database. The following search terms were used: eating disorders; eating pathology; anorexia nervosa; bulimia nervosa; eating disorder not otherwise specified; acceptance and commitment therapy (ACT); psychological inflexibility; experiential avoidance; cognitive-fusion; childhood abuse; emotional invalidation. Key references from relevant review articles were then obtained.

From the relevant papers, a second set of search terms were compiled. These included: eating disorders; anorexia nervosa; bulimia nervosa; eating disorder not otherwise specified; atypical eating disorder; binge-eating disorder; childhood abuse; sexual abuse; physical abuse; emotional abuse; abuse; neglect; invalidation; emotional invalidation;

emotional neglect; physical neglect; psychological inflexibility; acceptance and commitment therapy; ACT; cognitive fusion; experiential avoidance; avoidance; emotional regulation; cognitive flexibility; inflexibility; psychological distress. These terms were then used in different combinations to search the following databases: ScienceDirect, PsycINFO; APA PsycNET; Wiley Online Library, PubMed; Scopus. Studies were excluded if they were not reported in English.

The reference lists of relevant articles were also searched and additional papers that the author felt was most relevant were obtained. Searches on the World Wide Web were also conducted using internet search engines such as 'Google Scholar'. 'The Association for Contextual Behaviour Science' website was also used to find relevant articles.

2.3 Introduction to Clinical and Subclinical Eating Disorders

As a whole, EDs have been characterised as having the following key features (Garner & Myerholtz, 1998, p.592):

> "...a persistent overconcern with body size and shape indicated by behaviour such as prolonged fasting, strenuous exercise, and self-induced vomiting aimed at decreasing body weight and fat."

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association (APA) 2013) was released in May 2013 and it divides EDs into five diagnostic categories: Anorexia Nervosa (AN), Bulimia Nervosa (BN), Binge Eating Disorder (BED), Other Specified Feeding and Eating Disorder (OSFED) and Unspecified Feeding and Eating Disorder (UFED). The criteria in DSM-5 are different to the previous edition of the Diagnostic and Statistical Manual of Mental Disorders – the DSM-IV-TR (APA 2000) – with the key changes being: a lower threshold for a diagnosis of AN or BN, the inclusion of BED and the removal of the Eating Disorder Not Otherwise Specified (EDNOS) category. The rationale behind these changes was to reduce the reliance on EDNOS as it was more common than AN and BN (Fairburn & Cooper, 2011; Fairburn et al., 2007). This is discussed further in section 2.3.1.4.

If one viewed EDs on a continuum, then 'healthy' eating would be at one end of the spectrum and 'clinical' EDs (i.e. diagnosable EDs according to the DSM-5) would be at the other end of the spectrum. In between, there would be a range of eating and/or weight control practices some of which would include 'subclinical' EDs. A subclinical ED can be defined as a display of inappropriate eating behaviour or engagement in inappropriate eating practices without meeting the full diagnostic threshold for a specific ED. The continuum hypothesis is favoured by many within the literature (Lewinsohn, Striegel-Moore, & Seeley, 2000; Wildes & Marcus, 2013) because several people who fall within the subclinical category can go on to develop a clinical variant such as AN, BN and BED. However, there is much debate around how clinically useful a continuum approach is because clinicians may still place cut-off points along the dimensions in order to aid their clinical decisions. In turn, they could unintentionally convert the continuum into categories (Lavender, Crosby, & Wonderlich, 2013; Williamson, Gleaves, & Stewart, 2005).

In this study, the term 'eating pathology' will be used to refer to clinical and subclinical EDs. However, references will be made to clinical or subclinical EDs as and when required. Whilst a review of all the literature in relation to eating pathology is beyond the scope of this study, a brief summary of the most salient findings will be outlined below.

2.3.1 Clinical and Subclinical Eating Disorders: definition and brief overview

2.3.1.1 Anorexia Nervosa

AN most commonly emerges in adolescence or early adulthood (Fairburn & Harrison, 2003) and the lifetime prevalence rates for women tend to vary from 0.3% to 0.6% (Hoek & van Hoeken, 2003; Treasure, 2012). Prevalence rates in males have not been well studied, but the lifetime prevalence is estimated to be around 0.3% (Treasure, 2012). AN is a serious mental illness characterised by an avid pursuit of thinness through the use of dietary restraint and vigorous exercise which consequently leads to a failure to maintain a minimally normal weight. Those with AN often look emaciated and are

preoccupied with thoughts of food, body weight and shape, including a fear of being or becoming fat (Attia, 2010). Unsurprisingly, there are numerous physical consequences upon their body such as cardiac complications, low blood pressure, hypothermia and osteoporosis to name but a few (Meczekalski, Podfigurna-Stopa, & Katulski, 2013). A consistent finding within the literature is that the recovery rates are rather bleak (Herzog et al., 1999; Steinhausen, 2002). For example, Treasure (2012) suggests that over 50% of cases of AN have a protracted course over six years and a third of cases never make full recovery. Of those that do not recover, the unfortunate consequence is death. Indeed, the mortality rate for AN is extremely high – perhaps the highest of all psychiatric conditions (Guarda, 2008) – and approximately 5% will die prematurely from their illness (Arcelus, Mitchell, Wales, & Nielsen, 2011; Franko et al., 2013; Smink, van Hoeken, & Hoek, 2012). However, this figure must be interpreted with caution due to limitations of epidemiological studies such as sample sizes, diagnostic classifications, and duration of illness and length of follow-up.

2.3.1.2 Bulimia Nervosa

BN commonly emerges in adolescence or early adulthood (Fairburn & Harrison, 2003) and the lifetime prevalence rates vary from 1% to 1.5% for women and 0.1% to 0.5% for men (Hoek & van Hoeken, 2003; Treasure, 2012). Unlike those with AN, those with BN do not appear underweight and it is characterised by recurring episodes of binge-eating followed by intense weight-control behaviours in order to counteract the binge and avoid gaining weight (Walsh, 2011). These behaviours often take the form of self-induced vomiting, but it can also include laxative or diuretic use (Hay, Bacaltchuk, Stefano, & Kashyap, 2009). When not binge-eating, individuals with BN markedly restrict their food intake and a vicious cycle often ensues: binge-eating is followed by severe dietary restriction but this often leads to an increased appetite and then the stage is set for another episode of binge-eating (Fairburn, Cooper, & Shafran, 2003). This cycle of binging and purging inevitably takes its toll on the individual's body and some consequences include calluses on the back of the hand, Oesophagitis (inflammation of the throat), dental erosion, dehydration and cardiac problems (National Collaborating Centre for Mental Health; NCCMH; 2004).

There has been little research on the long-term outcome of BN. Fairburn, Cooper, Doll, Norman, and O'Connor (2000) prospectively studied 102 individuals with BN over 5 years. They found that whilst there were some improvements, the overall prognosis was relatively poor as 50% to 75% continued to have BN.

2.3.1.3 Binge Eating Disorder

BED appears to affect an older age group (Fairburn & Harrison, 2003) and the lifetime prevalence rates range from 1% to 3% (Hoek & van Hoeken, 2003; Treasure, 2012;(Kessler et al., 2013) for females. At the time of writing there is no information on males and this may be due to no published research on this population. BED is characterised by recurrent episodes of binge-eating and unlike BN, it is not followed by compensatory weight loss behaviours (Tanofsky-Kraff et al., 2013), so it is often associated with obesity. As a result of over-eating and weight gain, the physical complications of BED tend to be associated with obesity i.e. diabetes, heart disease, hypertension and stroke (NCCMH, 2004).

BED was only recognised as a clinical ED in DSM-5 as previous editions of the diagnostic and statistical manual placed it in the EDNOS category. Despite its recent acknowledgement, BED has actually been recognised in the literature and in clinical practice for several years, with the earliest mention being in 1959 (Stunkard, 1959). The literature to date suggests that it is potentially one of the most prevalent EDs (Kessler et al., 2013) and can persist over many years (Kessler et al., 2013).

2.3.1.4 Subclinical Eating Disorders

Within the literature, subclinical EDs go by many names such as atypical eating disorder, sub-threshold eating disorder or partial eating disorders. As of yet, there is no universally accepted definition (Chamay-Weber, Narring, & Michaud, 2005) and the relationship between subclinical and clinical EDs is not yet fully understood. What is clear, however, is that subclinical EDs form a category of individuals who suffer from serious cognitive, affective and behavioural eating-related symptoms but fail to meet the full diagnostic criteria for a clinical ED (Juarascio, Forman, & Herbert, 2010; Treasure, 2012).

EDNOS, OSFED and UFED can be classed as subclinical EDs because they are typically ascribed to those who do not *precisely* meet the diagnostic criteria for either AN, BN or BED. For example, all the features of AN may be present except that the individual's weight is slightly over the threshold: this results in them falling into the subclinical category. As mentioned previously, the general consensus amongst researchers was that there was an over-reliance on EDNOS; the majority of individuals tend to be given this diagnosis resulting in subclinical EDs being far more common than clinical EDs (Fairburn & Cooper, 2011; Fairburn et al., 2007; Tanofsky-Kraff et al., 2013; Zimmerman, Francione-Witt, Chelminski, Young, & Tortolani, 2008). Indeed, it has been suggested that more than 50% of cases in the community fall into the subclinical category (Treasure, 2012) and this is high compared to the prevalence rates for the clinical disorders. The DSM-V has attempted to address this issue by refining the diagnostic criteria, but, given the bulk of research has been carried out using the DSM-IV-TR classification, it remains to be seen as to what impact this amendment has.

Some researchers have suggested that individuals with subclinical eating pathology do not differ significantly from those with AN, BN or BED (Chamay-Weber et al., 2005; Fairburn & Bohn, 2005; Striegel-Moore et al., 2000) as there is often an extremely close resemblance between the two. Schmidt et al. (2008) looked at whether those with EDNOS differed from those with BN: they found no differences between the two in terms of demographic characteristics, duration of disorder or clinical severity. Overall, this suggests that clinical and subclinical EDs may have more similarities than differences and this is discussed further in the next section.

2.3.2 Transdiagnostic Model of Eating Disorders

Fairburn et al. (2003) developed the Transdiagnostic Model (TM) of eating pathology in order to recognise the common features of clinical and subclinical EDs. Given the high rate of diagnostic crossover (Fairburn & Cooper, 2011) this was believed to be more useful than looking at each ED (and its associated symptoms) separately. Additionally, some longitudinal studies indicate that many individuals move between AN and BN (Castellini et al., 2011) and that EDNOS is a common outcome of AN or BN and vice versa (Sullivan, Bulik, Fear, & Pickering, 1998). Eddy et al. (2002) carried out a

prospective study on 136 women with AN for 8 – 12 years and found that 62% with AN (restricting subtype) had crossed over to the binge-purge subtype within 8 years. However, it has also been argued that longitudinal stability is more common than crossover (Fichter & Quadflieg, 2007). Despite the lack of consensus, the fact that some crossover occurs suggests that clinical and subclinical EDs may share common maintaining mechanisms and that it is these, not the symptoms themselves, which should be targeted in treatment.

The crux of the TM is that there is an over-evaluation of weight and shape along with attempts to control them. Unfortunately, it is these attempts at control which manifest as a binge-purge cycle and/or restriction (Fairburn et al., 2003). The TM also proposes that the four common maintaining mechanisms which account for the persistence of clinical and subclinical EDs are: mood intolerance, low self-esteem, clinical perfectionism and interpersonal difficulties (Fairburn et al., 2003). This has some empirical support as Fairburn et al. (2009) offered a transdiagnostic intervention (based on the TM) to 154 individuals with an ED (regardless of their diagnosis) and found that many participants responded well regardless of diagnosis. In addition, their diagnosis made little difference to their short or longer-term outcome. A critique of the TM is that its applicability to BED is unknown. There is some research suggesting that the presentation of BED is unique in that it occurs with obesity and tends to be intermittent (Fairburn & Cooper, 2011; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). A further critique is that it is quite cross-sectional and does not appear to explicitly take into account the influence of contextual factors on interpersonal functioning which may then affect or maintain EDs.

The advantage of a transdiagnostic approach is that it may enable findings from the literature to be generalised to the understanding, prevention and treatment of *all* forms of eating pathology regardless of the actual diagnosis. This would encourage a greater transfer of the theoretical and treatment advances between the disorders. Furthermore, interventions could be targeted at the underlying psychological processes which in turn, could generalise to other symptoms (such as low mood and anxiety) as an intervention which reverses the maintaining processes in one ‘disorder’ could lead to an improvement in other ‘disorders’. This is particularly true given that depression and anxiety (for

example) are common comorbid diagnoses in individuals with EDs (APA workgroup on eating disorders, 2006).

2.3.3 What do we know about the aetiology of eating disorders?

The exact aetiology of eating pathology is unknown but the general consensus is that it is multifactorial. For example, research has highlighted the role of socio-cultural factors, family factors, individual factors (such as body dissatisfaction) and biological influences (Polivy & Herman, 2002). There is a vast breadth of literature covering these areas and it will not be discussed here due to space limitations. The reader is encouraged to refer to Polivy et al (2002) for further information as this provides a good starting point. Despite the vast literature, many questions still remain about how EDs develop and are maintained which suggests that other factors could be implicated. Of interest to this study is that of 'childhood contextual factors', as high rates of abuse have consistently been found in this population (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). It is worth mentioning that the picture is rather complicated as it is not yet clear as to whether different forms of childhood abuse are present in different types of EDs and this is discussed further in section 2.5.1. Before research linking abuse and eating pathology is outlined, an overview of childhood contextual factors shall be presented first.

2.4 Introduction to childhood contextual factors

In this study the term 'contextual factors' specifically refers to the experiences of abuse and emotional invalidation in childhood. Within the literature, these are often referred to as 'adverse early experiences' and these factors have been chosen for exploration as each have been linked to the experience of psychological distress and eating pathology in later life. It is acknowledged that there may be other relevant contextual factors relevant within ED aetiology.

2.4.1 Childhood abuse: definition and a brief overview

The term 'maltreatment' can be viewed as an umbrella term for the four main categories of abuse; physical, sexual, emotional and neglect. However, before the Children Act (1989) came into effect, definitions of maltreatment varied enormously (Giovanni, 1989) which led to difficulties in the identification and intervention of abuse. In the United

Kingdom (UK), The Children Act (1989, 2004) uses the concept of 'significant harm' to define abuse and this has been widely established as the legal threshold for the recognition of child abuse and neglect (Glaser, 2002). Significant harm is defined as:

> "…evidence of *either* ill-treatment of the child that has caused or is likely to cause significant harm to the child, *and*/*or* impairment of the child's health and development which is attributable to ill-treatment or to the care that the child has or has not received" (Glaser, 2000 p.98).

Despite this definition, there are no absolute criteria on which to rely when deciding what constitutes as significant harm. For example, it could be a single traumatic event or an on-going repeated event, all of which have the potential to impact on the child's health and development. Perhaps the only guideline specified in the Children Act (1989) is that the harm can be deemed significant if the child's health or development is not comparable to that of a similar child of a similar age. This can make it difficult to apply this concept in clinical practice as it is very much dependant on individual judgement. This is supported by Ayre (1998) who found that different approaches were used by professionals when attempting to work out if a child was at risk of significant harm or not.

Definitions of abuse for research purposes have generally tended to be even broader than the legal definitions, but the field of study has been complicated by similar difficulties in the definition of maltreatment and the methodology used to identify the occurrence of maltreatment (Baker, 2009; Shaffer, Huston, & Egeland, 2008). For example, some studies use official reports such as Child Protective Services (Manly, 2005), whereas others have relied on the victim's subjective account of the incident. A landmark study by Shaffer et al. (2008) examined the relationship between the incidents of maltreatment and the methodology used. They found large differences in prevalence depending on the methodology used e.g. prospective methods yielded higher rates of maltreatment than retrospective methods. From a clinical perspective, the meaning and interpretation an individual gives to an experience is highly relevant in determining what constitutes as abuse and neglect (Shaffer et al., 2008) but reporting on past experiences of maltreatment

can be affected by the accuracy of memory (Hardt & Rutter, 2004), the individuals relationship with the perpetrator, and current pathology (Briere, 1992).

In practice, this means that it is difficult to determine the prevalence rates of the different forms of abuse, along with the long-term consequences of such abuse. Indeed, even now there is still no agreement amongst researchers about the extent of the problem and whether the rates of maltreatment are increasing, declining or have remained the same (Gilbert et al., 2012). From 2006 to 2012, the number of children subject to child protection plans in England increased by 16,500 from 26,400 (Department for Education, 2012). However, this relates to official reports only and there may be more incidents which remain undetected due to non-disclosure of abuse by victims (NSPCC, 2011). This is hardly surprising given that abuse often occurs behind closed doors and at a time of dependency, i.e. within childhood. Nonetheless, in community samples it has been suggested that 53% of young people and young adults have experienced at least one incident of physical, sexual, or emotional abuse or neglect during childhood. Furthermore, 8.5% had experienced this more than once in the last year (Radford, Corral, Bradley, & Fisher, 2013) .

It is important to mention that different types of child abuse and neglect co-exist (Ney, Fung & Wickett, 1994) which means that it can be difficult to separate one form of abuse from another. Despite this, of the four main categories of abuse, childhood sexual abuse (CSA) is the most heavily researched within the literature. However, childhood emotional abuse (CEA) is becoming increasingly more recognised (Glaser, 2002; Shaffer, Yates, & Egeland, 2009). For example, in 2012, a total of 13,240 children were subject to a child protection plan under the category of emotional abuse, an increase of 40% from 2008 (NSPCC, 2012). However, similar issues regarding the definition of CEA plagues the literature as it has many different names in different countries, jurisdictions and the literature (Glaser, 2011). For example, some definitions refer to the *nature* of maltreatment only (Working Together, 2013) whereas others refer to *evidence* of actual harm or likely harm to the child (Baker, 2009). For the purpose of this study, CEA will be defined according to O'Hagan's (1995) definition because it takes into account the child's contextual environment: "the sustained, repetitive, inappropriate emotional response to the child's experience of emotion and its accompanying

expressive behaviour" (p.456). An implicit assumption within this definition is that abuse in childhood takes place in environments that are largely invalidating to the child (Follette, 1994) and this is discussed further in the next section.

2.4.2 Emotionally Invalidating Childhood Environments: definition and a brief overview

The concept of an invaliding environment was first proposed in the Dialectical Behaviour Therapy (DBT) model (Linehan, 1993) and was originally described as an environment in which the:

> …communication of private experiences is met by erratic, inappropriate and extreme responses. In other words the expression of private experiences is not validated; instead it is often punished, and/or trivialized (p.49).

The crucial experience in an invalidating environment is when the child's emotions are not recognised, appreciated or validated by the caregivers. For example, they are ignored, responded to negatively and displays of negative affect are not tolerated (Mountford, Corstorphine, Tomlinson, & Waller, 2007). In other words, there is a poor fit between the environment itself and the child's temperament. (Linehan, 1993) suggests that growing up in an invalidating environment gives the child the message that their view and experience of emotions is incorrect. This can result in difficulties in 'distress tolerance' – the skill to experience and accept negative affect such as sadness, guilt and shame. Those who struggle to tolerate such distress have a difficulty with identifying, labelling and regulating their emotional states (Waller, Corstorphine, & Mountford, 2007) so they either try and avoid potential triggers that may elicit their emotions or they use impulsive behaviours in order to manage them (Mountford et al., 2007). Emotional invalidation in its extreme can thus be construed as a form of emotional abuse (Krause, Mendelson, & Lynch, 2003) as invalidating environments have been associated with psychopathology in later life (Haslam, Mountford, Meyer, & Waller, 2008).

2.5 Links between childhood contextual factors and eating pathology

Within the literature, there is a vast amount of empirical evidence to show that any form of physical, sexual and emotional abuse or neglect during childhood is detrimental to the child's functioning and development in later life (Glaser, 2011; Swanston et al., 2003). In addition, this often extends to adulthood manifesting in psychological distress and a wide range of psychopathology, including eating pathology (Chirichella-Besemer & Motta, 2008; Hart, Binggeli, & Brassard, 1997). A review of all the literature is beyond the scope of this study but a brief summary of the relevant findings will be outlined below.

2.5.1 Abuse and eating pathology

As mentioned previously, elevated rates of childhood abuse have been consistently found in those who have clinical and subclinical eating pathology (Carter, Bewell, Blackmore, & Woodside, 2006; Castellini et al., 2013; Rayworth, Wise, & Harlow, 2004; Romans, Gendall, Martin, & Mullen, 2001). Some authors have found that CSA is more prevalent in those with BN (Jenkins, Meyer, & Blissett, 2013; Wonderlich et al., 2000). For example, Leonard, Steiger, and Kao (2003) found that those with BN had higher rates of CSA (N= 20, 39%) than those without BN (N=8, 32%). However, the difference between the groups' sizes in Leonard et al (2003) was large which can make comparisons difficult. A recent meta-analysis found substantial heterogeneity in the findings concerning CSA and eating pathology; the relationship varied depending on the methodology used and definitions used (Smolak & Murnen, 2002). Overall, the general consensus at the current time seems to be that CSA may be a non-specific risk factor for eating pathology and that it is associated with an increased risk of psychopathology *in general* (Thompson & Wonderlich, 2004; Cutajar et al., 2010; Jacobi et al., 2004).

Much of the focus has been on CSA, with few authors considering other forms of abuse within an ED population. Two exceptions to this are Fosse and Holen (2006) who looked at the relation between maltreatment and ED in later life in a sample of 107 female outpatients. They found that those with BN had more emotional, sexual and physical abuse than those without BN. Similarly, Kong and Bernstein (2009) had a sample of 73 people with AN, BN and EDNOS. They found that childhood emotional abuse, physical neglect and sexual abuse predicted current eating pathology.

Despite the wealth of research in this area over the last decade, there is still some uncertainty as to whether some forms of childhood abuse are unique in terms of their effect on eating pathology. This is particularly pertinent given that abuse usually occurs in the context of many other confounding variables such as family dysfunction (Noll, 2008). Moreover, as previously outlined, different forms of child abuse and neglect co-exist (Ney, Fung & Wickett, 1994) which means that it can be problematic separating out the specific effects of abuse. However, there is now some emerging literature which is only just beginning to focus on exploring whether there is a unique effect of childhood abuse on eating pathology (Fergusson 2008). One such finding is that CEA may have a unique impact on the development of eating pathology (Kennedy, Ip, Samra, & Gorzalka, 2007; Kent, Waller, & Dagnan, 1999; Kong & Bernstein, 2009). Burns, Fischer, Jackson, and Harding (2012) surveyed 1,254 college students and after controlling for the effects of physical and sexual abuse, they found that CEA was strongly associated with eating pathology. A major limitation of this study is that it focused on ED *symptoms* only and there is no mention as to whether the sample had different diagnoses of an ED. As discussed previously, there does appear to be differences in types of abuse between different ED groups. Whilst a further limitation of Burns et al (2012) is that the findings are limited by the characteristics of the sample (i.e. college students only and homogeneity), overall, it is an intriguing result which certainly merits further investigation.

2.5.2 Emotional invalidation and eating pathology

Considering CEA and emotional relational contexts further, whilst there is considerable evidence of links between childhood abuse and eating pathology within the literature, the exact nature of these links remains to be established. One possible link is the way in which the family contribute to the child's development and experience of emotions (Haslam et al., 2008). The literature looking explicitly at childhood invalidating environments and eating pathology is small, but there appears to be some evidence which supports the link between the two.

Mountford et al. (2007) suggested that: "poor distress tolerance, developed from an invalidating childhood environment, appears to be central to ED symptomatology"

(p.49). This is supported by Haslam et al. (2008) who studied fifty-eight patients with AN or BN and found that an invalidating childhood environment was associated with eating pathology. More specifically, an invalidating experience from the father was associated with bingeing, vomiting and excessive exercise whereas maternal invalidation was associated with less binging. This highlights how invalidation is not strictly causal; rather it there may be a relational component with it being influenced by the way in which the individual *perceived* their experience or their caregiver. It is also possible that these invalidating experiences stemmed from gender and cultural differences in parenting styles as an authorita*tive* parenting style (i.e. high warmth, nurturing and high control) is typically associated with greater emotional maturity than authorit*arian* (low warmth, punishment and high control) and permissive parenting styles (i.e. high warmth, lenient and low control) (Baumrind, 1991; Fabes, Leonard, Kupanoff, & Martin, 2001; Topham et al., 2011). A recent review by le Grange, Lock, Loeb, and Nicholls (2010) concluded that the research on parental behaviours and EDs is sparse or lacking and that further research is needed.

2.5.3 How might abuse and emotional invalidation increase the risk of eating pathology in later life?

Despite some evidence that adverse childhood experiences might increase the risk of eating pathology and psychological distress in later life, not everyone develops difficulties. This suggests that the relationships between the two are not direct and that a third factor may play a role. One way in which adverse experiences in childhood might increase the risk of eating pathology is through the way in which a person learns how to manage and process their internal experiences. These internal experiences refer to an individual's subjective experience and they include thoughts, emotions, memories and physical sensations.

Abuse and emotional invalidation may result in the individual developing a belief that emotions should never be experienced or expressed, regardless of the situation they may find themselves in. Over time, particular ways of managing and processing internal experiences develops until it becomes the default coping mechanism (Young & Klosko, 1998). One such coping mechanism related to eating pathology is that of avoidance. It could be hypothesised that an inability to tolerate extreme emotions (Mountford et al.

2007) may result in them attempting to avoid their emotional states via their eating behaviour i.e. binging, purging (Smyth et al., 2007) and starvation (Brockmeyer, Grosse Holtforth, Bents, Herzog, & Friederich, 2013). This avoidance can be conceptualised as *experiential avoidance* (Schmidt, Bone, Hems, Lessem, and Treasure, (2002). This is when an individual tries to avoid or control their internal experiences (Hayes, Strosahl, & Wilson, 1999), but although this process can bring short-term relief, it can end up maintaining psychological distress in the long-term.

The concept of experiential avoidance has been recognised within many systems of therapy (Blackledge & Hayes, 2001) but the one theoretical approach which stands out in offering the most comprehensive understanding of experiential avoidance and where this term originated within, is that of Acceptance and Commitment Therapy (ACT; Hayes et al., 1999). ACT is an intervention which is underpinned by Relational Frame Theory (RFT). It is hoped that by focusing on this approach, some of the underlying *processes* that might maintain eating pathology in individuals who have had adverse childhood experiences will be highlighted.

Despite focusing on one theoretical perspective here, it is acknowledged that this is not the only perspective which could explain or illuminate the underlying processes that may maintain eating pathology. For example, the TM by Fairburn et al (2003) considers 'mood intolerance' (which is conceptualised as an inability to cope appropriately with certain emotional states) to be one of four maintaining mechanisms in eating pathology (please refer back to section 2.3.2 for a discussion of this). They suggest that the individual is unable to cope adaptively with their mood, so they engage in maladaptive coping behaviour instead. This serves the function of reducing "their awareness of the triggering mood state" (Fairburn et al, 2003, p.517). However, the TM fails to explain *why* the individual cannot cope with their mood in the first place. In other words, there is a missing link connecting mood intolerance and eating pathology. This study focuses on the ACT model as it seems well placed to 'fill in' this missing link due to its focus on context and its underpinning theory – RFT.

Before research linking experiential avoidance and adverse childhood experiences in relation to eating pathology is outlined, an overview of ACT and RFT shall be presented.

2.6 Introduction to the ACT approach

ACT is a third-generation cognitive and behavioural therapy (Hayes et al., 1999) which "embraces a contextualistic philosophy, a basic theory of language and cognition and an applied theory of pathology and psychological change" (Hayes, Luoma, Bond, Masuda, & Lillis, 2006)p.4).

ACT is a transdiagnostic process-orientated approach which assumes that there are commonalities across a range of psychological disorders and that it is these commonalities which maintain psychological distress (Harvey, 2004). For example, ACT argues that experiential avoidance and cognitive-fusion are two key processes which maintain many different forms pathology as it affects the way that an individual processes their emotional experiences. This is in stark contrast to a diagnostic approach which takes the position that each psychological disorder has its own unique set of symptoms and processes, and that it only by focusing on one disorder at a time, that distress can be alleviated.

In order to outline ACT's model of pathology in greater detail, it will be helpful to start from the ground up. In other words, the theoretical and philosophical basis to this approach will be presented first as this is what underlies ACT. This will then be followed by an overview of the two transdiagnostic processes which ACT views as being the main origin of pathology in general.

2.6.1 The Theoretical and Philosophical Basis of ACT

2.6.1.1 Functional contextualism

ACT is a behavioural model of human suffering and it is rooted in a philosophical framework called functional contextualism. It takes the position that psychological events can only be understood in relation to historically and situationally defined contexts as there is an on-going interaction between the three (Hayes et al., 2006). Hayes et al. (1999) state that to look at psychological events in isolation would ignore important

contextual features to any action. From this perspective, no thought, feeling, memory or action is fundamentally problematic or leads to psychological distress, rather it all depends on the context and how it functions for the person. This leads to another position of functional contextualism: is what the individual doing working or not for them? For example, in a context of experiential avoidance or cognitive-fusion, an individual's thoughts, feelings, memories or actions often function in a way that increases their psychological distress because they are held back from the kind of life that they want to live. Yet in a context of no experiential avoidance or cognitive-fusion, those very same thoughts, feelings, memories or actions have far less of an impact. This is not to say that they are not painful, but they will not be harmful or hold someone back from valued living (Hayes, Levin, Plumb-Vilardaga, Villatte, & Pistorello, 2013). According to this philosophy, simply changing the content of thoughts and feelings in order to influence behaviour is unlikely to be effective because the contextual features are still present. Instead, the goal is to change the context in which these thoughts and feelings occur (Hayes et al., 2006). In other words, the focus is on changing the relationship that the individual has with their thoughts and feelings, thus reducing the psychological distress experienced.

2.6.1.2 Relational Frame Theory (RFT)

Underpinning ACT is relational-frame theory and in order to understand how ACT conceptualises and targets emotion regulation, it is important to outline how language and cognition operate from an RFT perspective (Kring & Sloan, 2010).

The basic premise of RFT is that human behaviour is governed largely through networks of mutual relations called 'relational frames' which form the core of human language and cognition, and allow us to learn without requiring direct experience (Hayes & Smith, 2005). For example, a child does not need to touch fire in order to be verbally taught that fire will burn them. Hayes (2004) gives the following example of how relational frames work:

> …Suppose a child has never before seen or played with a cat. After learning "C-A-T" animal, and C-A-T "cat", the child can derive four

> additional relations: animal C-A-T, “cat” C-A-T, “cat” animal, and animal “cat”. Now suppose that the child is scratched while playing with a cat, cries, and runs away. When the child later hears father saying, “Oh, look! A cat,” she may cry and run away even though scratches never occurred in the presence of the words “Oh, look! A cat” (pg. 649).

As seen in the above example, only two relationships were explicitly taught to the child (C-A-T = animal, C-A-T = cat), yet the child was able to derive four additional relationships without being explicitly taught. This highlights another unique ability that humans have: through the use of relational frames they can relate events mutually and in combination, as well as arbitrarily relate almost anything to anything (i.e. objects, thoughts, feelings, behaviour) in virtually any possible way (Hayes & Smith, 2005). Examples of relational frames are: coordination (i.e. ‘same as’, ‘different to’), temporal or causal (i.e. ‘if/then’ or ‘cause of’), comparative and evaluative (i.e. ‘bigger than’ or ‘thinner than’), perspective (i.e. ‘I/you’ or ‘here/there’) and spatial (i.e. ‘near/far’) (Hayes & Smith, 2005).

RFT suggests that whilst verbal and cognitive abilities are useful in that they allow humans to think about the future, problem-solve and evaluate and compare outcomes, they also have a dark side. That is, verbal relational frames can turn any event into a source of pain and distress (Hayes, Barnes-Holmes, & Roche, 2001). For example, with a temporal relation, people can predict bad events that may not happen such as death, illness or injury. With a comparative relation, people can compare themselves to others and think they are better than or worse than them (Hayes & Smith, 2005). Furthermore, humans are able to attach emotional content to their thoughts and memories which mean that describing a past painful experience or imagining a future one can be as distressing as the event itself.

Relational frames start to develop in infancy and are shaped by experience so over time, many events and words can be joined together to form vast relational networks. In returning to the example above, the word “cat” may now include: the smell of the cat, the feel and colour of the cat’s fur, the sound of a cat and the child’s feelings (i.e. fear) about the cat.

According to RFT, within a relational network the process of relational framing leads to all sorts of 'transformation of stimulus functions' in each stimulus. Simply put, the effect that each stimulus has upon the individual will transform or change depending on how it is related to other stimuli (Blackledge, 2003).

In a similar vein, words, thoughts, feelings and memories can also undergo a transformation of stimulus functions and spread across to every event within that vast relational network. If someone related the words 'awful' and 'meaningless' to the words 'my life', then some of the functions of those two words will not only transfer to the words 'my life', but also to everything else within that relational network. As a result, that individual would now view *everything* in their life as awful and meaningless (Harris, nd).

Overall, RFT highlights how humans suffer because they are verbal creatures (Hayes & Smith, 2005) and this is powerfully summarised by Wilson et al (2001):

> …a species [humans] that has by far the fewest contacts with direct sources of pain…through language is able to suffer with a degree of intensity, constancy and pervasiveness that is literally unimaginable in the nonhuman world…the aversive functions that humans avoid are now aspects of their own selves (p.215).

2.6.2 The ACT model of Pathology: the role of psychological inflexibility

According to ACT, 'psychological inflexibility' is the core of pathology due to two main processes: cognitive-fusion and experiential avoidance. These two processes will now be outlined in greater detail.

2.6.2.1 Cognitive fusion

Cognitive fusion is when an individual reacts to their thoughts as if they are literal truths because they have failed to distinguish the product of thinking (e.g. 'I am useless') from the process of thinking (e.g. 'I am having the thought that I am useless'). As a consequence, they react to the words used to describe and interpret experiences as if

those words *are* those very experiences (Blackledge & Hayes, 2001; Valdivia-Salas, Sheppard & Forsyth, 2010). For example, an individual having the thought 'I am useless' will react to the words as if they are truly useless and behave as one would if they were truly useless.

Cognitive-fusion can happen with a variety of thoughts and feelings related to the self, the past and the future which results in people "quite literally living in their heads, their pasts, or futures that have yet to be" (Valdivia-Salas et al., 2010, p.317). This gives rise to what ACT calls a 'conceptualised self' which is where an individual fuses with their self-description and behaves as if they truly are that description. For example those with eating pathology may fuse with descriptions such as 'I am fat/ugly/disgusting' and embody the self-description.

2.6.2.2 Experiential Avoidance

Experiential avoidance can be defined as the attempt to escape or avoid unwanted internal experiences and alter the form or frequency of these events and the contexts that occasion them, even when the attempt to do so causes psychological harm (Hayes, 2004; Hayes et al., 2006). It is important to mention that whilst experiential avoidance can be a useful strategy in some situations, it can become problematic when applied rigidly and inflexibly to the point where the individual is no longer pursuing their values and goals (Bond et al., 2011).

Boulanger, Hayes & Pistorello (2010) argue that the capacity for experiential avoidance is built into human language and cognition because humans are unable to control their pain or distress by purely situational means. Therefore, they naturally apply their verbal problem-solving skills to their internal experiences in an attempt to avoid or control the painful thoughts, feelings, memories and emotions themselves (Hayes, 2004). This may be achieved through the construction of verbal rules (i.e. 'don't think of X') in order to keep these unwanted experiences at bay. Unfortunately many of these rules contain the content that it is trying to get rid of and under these conditions, the rule will inadvertently create the very private event that the person is trying to avoid (Hayes et al., 1999). Experiential avoidance highlights this process in relation to thought-suppression

(Wegner, 1987) and emotional suppression (Gross, 2002) as deliberate attempts to keep unwanted thoughts and emotions out of mind can often lead to a rebound effect whereby there is a contradictory increase in these unwanted phenomena. It has also been documented that the rebound is greatest in contexts where the suppression took place (Abramowitz, Tolin, & Street, 2001; Gross, 2007; Magee, Harden, & Teachman, 2012). In linking this to RFT, Hayes et al., (1999) argue that the relational network inadvertently expands because new relations are made each time an individual unsuccessfully tries to get rid of their internal experiences.

It has been argued that many forms of pathology can be conceptualised as experiential avoidance (Hayes et al 1999) and this includes eating pathology. It has been hypothesised that engaging in disordered eating behaviours could serve as a desperate attempt to regulate the overwhelming negative affect that is being experienced (Polivy & Herman, 2002) and this is discussed further in section 2.7.1.

2.6.2.3 Psychological inflexibility

Psychological inflexibility then, comes from cognitive-fusion and experiential avoidance as this prevents people from moving forward in life in line with their chosen values (Bond et al., 2011). In turn, this leads to psychological distress because life is spent trying to eliminate content that cannot be eliminated.

Cognitive-fusion and experiential avoidance together give rise to four additional pathological processes which have been outlined in Figure 1. This is the ACT model of pathology. Fusion and avoidance can lead to individuals losing contact with their here-and-now experience as much time is spent dwelling on the remembered past or feared future instead of what is happening in the present moment (Harris, 2009). Furthermore, as an individual's behaviour becomes progressively driven by fusion or experiential avoidance, there can be a lack of values/clarity making it difficult for an individual to define what matters to them and to live the life that they truly want (Harris, 2009; Hayes & Smith, 2005).

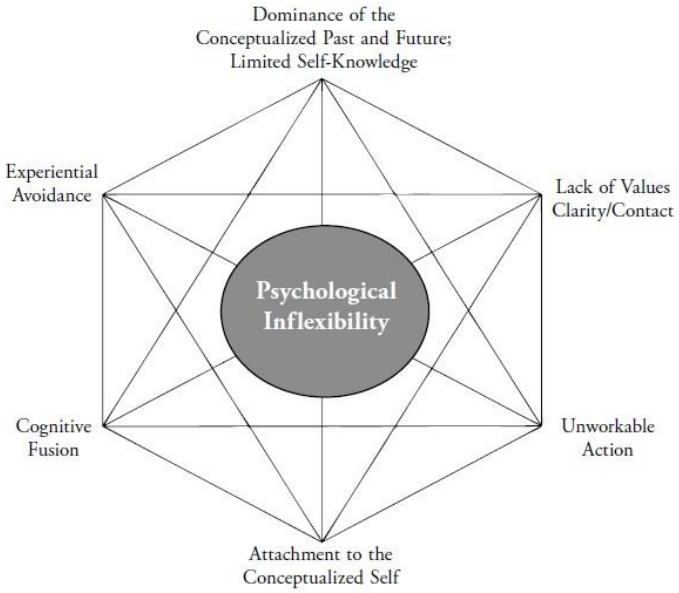

Figure 1: An ACT model of Pathology / psychological inflexibility from Hayes et al., (2013) p.5

2.6.2.4 Therapeutic aims of ACT

The overarching aim of ACT, then, is to target these six processes (see figure 2) in order to increase psychological flexibility and weaken the processes of cognitive-fusion and experiential avoidance. This is achieved through the use of experiential exercises and metaphors in order to encourage the individual to come into direct contact with their experiences and teach alternatives to cognitive-fusion (*de*fusion) and avoidance (acceptance). For example, through defusion, ACT focuses on changing the way an individual *relates to* or *interacts with* their thoughts so that they can be treated as just thoughts (Hayes et al., 2013; Hayes et al., 2006). Through acceptance, the individual is taught to simply be aware of, and embrace, their inner experiences instead of trying to get rid of them (Twohig, 2012). Ultimately, ACT works to develop a sense of 'self as context' whereby the individual has inner experiences but is not defined by them. Instead, they have a defused, non-judgemental stance and use language as a tool to notice and describe their on-going internal experiences (Hayes et al., 2006). Furthermore, the individual is encouraged to commit to their chosen values and to live the life that they truly want *in spite of* their thoughts and feelings. For example, an individual could have the thought that their stomach is too large and experience distress, but still choose to eat a balanced meal (rather than no meal at all) because this action is consistent with them valuing their physical health.

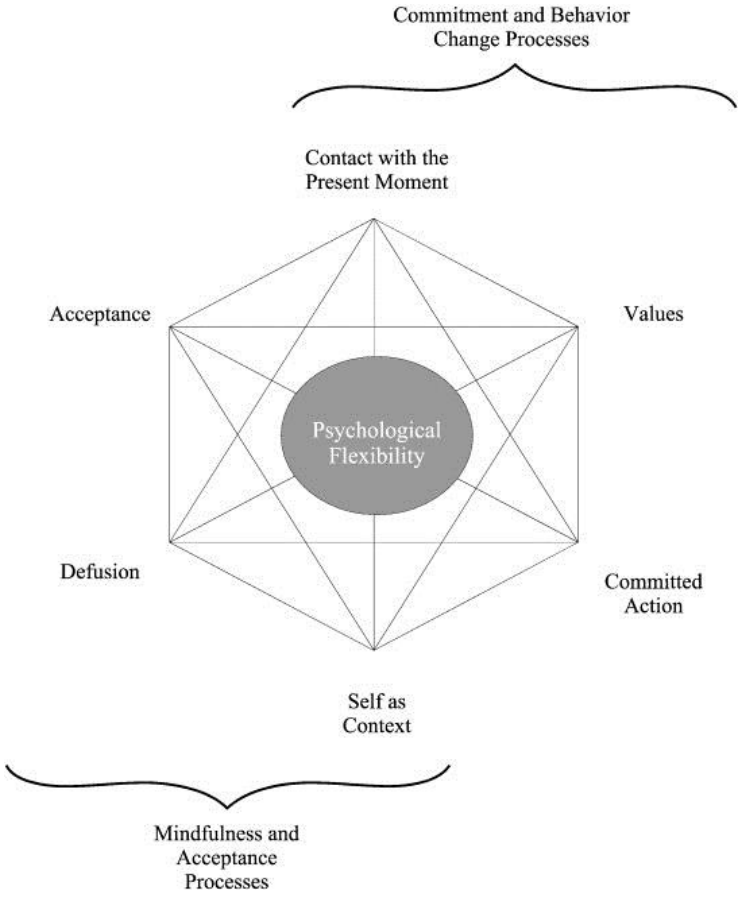

Figure 2: An ACT model of Psychological Flexibility from Hayes et al., (2013) p.6

2.6.2.5 Limitations of ACT

There is some debate in the literature as to whether ACT has anything new to offer and that it is no different to traditional CBT when one looks beyond the language used. For example, a commonly cited distinction between ACT and CBT is that CBT focuses on the *content* of cognitions whereas ACT focuses on the *process* of cognitions. However, Arch and Craske (2008) point out that ultimately, both approaches aim to reduce avoidance and enhance exposure to previously avoided internal experiences. Authors have also criticised ACT for 'getting ahead of the data' (Corrigan, 2001, 2002) and for using less stringent research methodology (Ost, 2008, 2009). In turn, this has cast some doubt on whether ACT is more effective than established treatments (Powers, Zum Vorde Sive Vording, & Emmelkamp, 2009). Despite these criticisms, ACT does appear distinct to CBT due to its philosophical assumptions and the way in which it

conceptualises and targets emotions in therapy (Hofmann, 2008; Hofmann & Asmundson, 2008).

2.7 Response to eating pathology: An ACT perspective

So far, it has been argued that there are relationships between adverse childhood experiences and emotional invalidation and psychological distress in later life. An overview of the ACT model has also been presented in order to illustrate the process of psychological flexibility i.e. cognitive-fusion and experiential avoidance. This section will now outline ACT in relation to eating pathology before going on to talk about child abuse and emotional invalidation.

2.7.1 Experiential avoidance, emotional processing and eating pathology

The role of emotion regulation in the development and maintenance of EDs has received considerable empirical attention (Sim & Zeman, 2006) with many authors suggesting that the EDs themselves may actually function as a way of regulating negative affect (Lampard, Byrne, McLean, & Fursland, 2011). One way in which affect could be regulated is via experiential avoidance as it allows the individual to avoid the experience and expression of negative affect such as depression and anxiety (Baer, Fischer, & Huss, 2005; Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007; Heatherton & Baumeister, 1991; Pearson, Follette, & Hayes, 2012; Schmidt & Treasure, 2006). Indeed, negative affective states have often been identified as common triggers for bingeing and purging and it has also been found to reduce following a binge (Polivy & Herman, 2002). It must be pointed out that much of the literature in relation to avoidance and negative affect has been carried out on those with BN with little consideration as to how it may play a role in those with restrictive attitudes and behaviours (e.g. AN). This may be due to some authors taking the view that negative affect has *no* influence on the eating attitudes and behaviours in AN (Fairburn, Cooper, & Shafran, 2003). However there is now some emerging literature which takes the opposite view (Haynos & Fruzzetti, 2011) and has found that those with AN do have difficulties with tolerating and regulating negative affect (Harrison, Sullivan, Tchanturia, & Treasure, 2010).

Linehan (1993; as cited in Baer et al 2005) and Greenberg & Paiviou (2003) described the idea of secondary emotions which are emotions that arise in response to another emotion i.e. feeling guilty (secondary) about feeling angry (primary). There is some literature to suggest that evaluating an emotion as 'bad' and the subsequent development of secondary emotional responses (e.g., fear or shame), may motivate the individual to avoid their emotions (Gratz, Tull, & Wagner, 2005). This suggests that those who have negative reactions to their own emotions are more likely to engage in experiential avoidance (Gratz, Bornovalova, Delany-Brumsey, Nick, & Lejuez, 2007). This is supported by Heatherton and Baumeister (1991) who put forward an 'escape avoidance hypothesis' which states that the act of binge-eating allows the individual to escape from their negative affect because their attention is focused on food and the physical act of eating. Although engaging in this type of behaviour appears to prevent the individual from feeling certain emotions in the short-term, the long-term consequences are that the emotions are far more intense (Safer, Telch, & Chen, 2009).

Experiential avoidance has been found to maintain eating pathology (Fulton et al., 2012; Lavender & Anderson, 2010; Rawal, Park, & Williams, 2010) and this is supported by Sim and Zeman (2004) who found that compared to a control group, girls with BN were more reluctant to express their emotions. A later study by the same authors found that those who reported high levels of disordered eating reported experiencing increased levels of negative affect, and more difficulty in coping constructively with negative affect (Sim & Zeman, 2006). It could be queried how experiential avoidance is possible in this population given that research has shown that they have specific deficits in identifying and communicating their emotions (alexithymia). How can one avoid an emotion if they cannot identify it? Whilst this is a valid point, it can be argued that if an individual does not have an ability to identify their emotions in the first place, then they may find it difficult to choose an appropriate strategy to manage (Barrett & Gross, 2001; as cited in Kring 2008), hence the extensive application of experiential avoidance via their eating behaviours. In addition, some qualitative studies have also highlighted how those with AN have stated that they value their disorder as it helps them to avoid aversive emotions (Serpell, Treasure, Teasdale, & Sullivan, 1999). This could be exacerbated by 'cognitive inflexibility' as some research has shown that those with AN, BN and EDNOS have difficulties with shifting their thinking and attention in response to

the environment (Tchanturia, Anderluh, et al., 2004; Tchanturia et al., 2011; Tchanturia, Morris, et al., 2004). However, this particular conceptualisation of 'cognitive inflexibility' is different to ACT as it is more informed by a neurological framework.

2.7.2 Cognitive-fusion and eating pathology

It has been proposed that those with eating pathology have their own unique variant of fusion called Thought-Shape Fusion (TSF; Shafran, Teachman, Kerry, & Rachman, 1999) wherein an individual believes they are fat because they think they are or that eating food will immediately make them fat. As with cognitive fusion, the individual reacts to their thoughts as if they are literal truths.

There is some support for this notion as TSF has been found to be significantly associated with eating pathology (Shafran et al., 1999) and those with clinical EDs have higher levels of TSF compared to non-clinical controls (Coelho, Carter, McFarlane, & Polivy, 2008; Radomsky, de Silva, Todd, Treasure, & Murphy, 2002; Shafran & Robinson, 2004). Some research has induced TSF in those with eating pathology by asking them to think about consuming fattening or high calorie foods and found that they exhibit higher levels of distress than the control group (Coelho, Baeyens, Purdon, Pitet, & Bouvard, 2012). More specifically, individuals with BN and AN reported increased anxiety, guilt, and body dissatisfaction after a TSF induction compared to those without eating pathology (Jauregui et al., 2011; Kostopoulou, Varsou, & Stalikas, 2011; Kostopoulou, Varsou, & Stalikas, 2013). This suggests that TSF is associated with negative affect and one could assume that this may lead to those with eating pathology engaging in efforts to escape from their emotions. This is particularly true when one refers to Section 2.3.2 which mentioned that a core pathology of EDs is an over-evaluation of body shape and weight; someone who fuses with such thoughts are likely to experience high levels of negative affect.

Whilst these findings lend support for the concept of cognitive-fusion, one must point out that the literature on TSF is rather small and that it has been carried out exclusively on women; it is possible that men may experience TSF differently. It is also important to highlight that as of yet, the underlying process in relation to TSF is still unclear and further research is needed.

2.7.3 Evidence Base of ACT and Eating Pathology

In relation to interventions, the literature looking explicitly at ACT and eating pathology is relatively new and emerging (Merwin et al., 2011; Merwin, Zucker, & Timko, 2013). The majority of research has been carried out on those with AN with the remainder of the EDs receiving little, if no attention.

Heffner, Sperry, Eifert, and Detweiler (2002) was one of the first published studies to look at the use of ACT for AN. This was a single case study with encouraging results as the anorexic symptoms remitted within ten sessions. However, the way in which ACT was used within this study was criticised by Orsillo and Batten (2002) as they believed that some of the techniques could have been applied in a better way. Later papers focused on the theoretical application of ACT to AN and concluded that ACT was a good fit for this population (Hayes & Pankey, 2002; Wilson & Roberts, 2002) due to its focus on changing how the individual *interacts* with their thoughts and feelings (as opposed to modifying the content).

Berman, Boutelle, and Crow (2009) used a case series methodology on three females with chronic and severe AN. They found that all participants experienced clinically significant improvement in their symptoms and that the treatment gains were maintained after 1 year. Similar findings have been reported in those with binge-eating (Lillis, Hayes, & Levin, 2011) and those with subclinical eating pathology wherein those in the ACT group showed reductions in body dissatisfaction and increases in acceptance compared to the control group (Pearson et al., 2012). A rather ambitious study has recently been carried out by Merwin et al. (2013) who offered an ACT-based family treatment to six adolescents with EDNOS and found significant improvements. However, it is difficult to disentangle these results and ascertain whether the improvements were due to ACT or the family work.

To the author's knowledge, only two Randomised Controlled Trials have been conducted. Juarascio et al. (2010) found that in those with subclinical eating pathology, ACT produced the largest decreases in eating pathology compared to cognitive therapy. This is supported by (Juarascio et al., in press) who studied 140 women with AN or BN and found that those in the ACT based group intervention *plus* treatment-as-usual (TAU)

showed greater improvements than TAU alone. However, there is no description of what TAU is; thus, it is difficult to elucidate whether the improvements were truly due to ACT. In addition, those with EDNOS were re-classified as AN or BN for this study which, again, makes it difficult to draw firm conclusions.

Whilst the majority of the earlier studies are case studies or have small samples with simple pre-post test designs, the data is encouraging and shows that ACT may be suited for the treatment of those with eating pathology due to its focus on reducing cognitive control and increasing acceptance. However, further research is clearly needed, particularly that which explores which underlying processes of ACT may be responsible for the positive outcomes. This is a gap that this study hopes to address as it is hypothesised that experiential avoidance and cognitive-fusion are the two key components which may be responsible in maintaining eating pathology.

2.8 Application of ACT to childhood abuse and emotional invalidation

Despite the link between emotions and eating pathology being well recognised within the literature, few authors have considered the *source* of the emotional issues experienced. One such source is that of 'childhood contextual factors' which will now be discussed using ACT as a framework.

According to the ACT model, psychological distress is more likely to occur when individuals become fused with the content of unwanted cognitions and engage in experiential avoidance in an attempt to avoid them. According to this view, the extent to which an individual fuses with their cognitions will be dependent upon their historical experience and verbal learning history (Flaxman, Blackledge, & Bond, 2011); different thoughts or words will mean different things based on how they are connected to events within a relational frame and the broader relational network. Therefore, the context in which a person grows up and how they experienced this context may be vital when considering how people might learn to respond to, and manage, internal events.

Growing up in what is perceived as an invalidating environment can mean that the child may not have been taught how to label their feelings or make sense of them, which fits with alexithymia (Haslam et al., 2008); thus, their ability to appropriately tolerate emotional distress is dramatically reduced. Experiences of abuse and emotional invalidation are particularly likely to give rise to a number of negative thoughts and feelings and it is not difficult to imagine how painful or intolerable this could be for the child. As mentioned previously, the ACT perspective proposes that relational frames start to develop in infancy and are shaped by experience so everything associated with the abuse would join together to form a vast relational network. Therefore, when the individual recalls a *past* traumatic event, words and images participating in that relational frame could be activated and the individual could feel as though they were literally reliving the abusive experience in the *present* moment (Blackledge, 2004).

It has been suggested that in order to manage the overwhelming negative affect experienced, the child may learn to engage in experiential avoidance in order to cope with the situation. This is particularly true if they are unable to physically remove themselves from the environment (Marx & Sloan, 2002). Whilst this is adaptive in the short-term, the habitual use of avoidant strategies (such bingeing, purging, starvation) could make them more readily available over time (Keville, Byrne, Tatham, & McCarron, 2008) because they are negatively reinforced by the instantaneous reduction of the intense negative thoughts and feelings associated with the abuse (Polusny & Follette, 1995).

It is has been well established within the field of trauma research that childhood abuse affects a child's developing sense of self (Phillips & Daniluk, 2004). Follette (1994) has argued that when an individual grows up in an abusive environment, their 'sense of self' is paired with a range of thoughts and feelings to the point where they cannot define themselves as being separate from their environment. In other words, they experience themselves 'as content' and believe that they truly *are* the thoughts and feelings experienced. Not surprisingly, with difficult to manage experiences, this could be frightening and so they may avoid it at all costs to avoid experiencing them. This pairing may occur because in an emotionally invalidating environment, the child's internal world may not be accurately described or validated for them by their parents and because they

have become overly reliant on experientially avoidant coping strategies (Blackledge 2004). This all underlies the acceptance of self within the ACT model.

From an ACT perspective, the consequence of the continued use of experiential avoidance is that the individual has less *flexibility* to manage their emotional experiences. In other words, avoidance is extensively applied as the individual may have no other immediate way of managing their emotional experiences (Keville et al., 2008). It is thought that this leads to greater psychological distress in the long-term.

2.8.1 Childhood abuse, emotional invalidation and psychological inflexibility: empirical studies

Experiential avoidance has been the primary focus within the literature in relation to abuse and emotional invalidation from an ACT perspective. However, empirical examinations of these relationships remains limited despite the theorised importance of experiential avoidance (Gratz et al., 2007). It is important to note that experiential avoidance has also been referred to as 'avoidant coping' and 'emotional non-acceptance' thus those studies using these terms have been included in the following section in order to provide a comprehensive, albeit brief overview of the most salient findings. This links with earlier sections (see 2.6.2.2 and 2.7.1).

2.8.1.1 Childhood abuse and experiential avoidance

A common finding within the literature is that childhood experiences of abuse are significantly associated with higher levels of experiential avoidance (Batten, Follette, & Aban, 2002; Brand & Alexander, 2003; Gratz et al., 2007; Rosenthal, Hall, Palm, Batten, & Follette, 2005; Sullivan, Meese, Swan, Mazure, & Snow, 2005), which in turn leads to greater psychological distress in later life. For example, Shenk, Putnam, and Noll (2012) examined 110 females who had experienced different types of maltreatment within the past year; they found that experiential avoidance mediated the relationship between childhood abuse and Post-Traumatic Stress (PTSD) symptoms. This is supported by (Marx & Sloan, 2002) who also found that experiential avoidance mediated the relationship between CSA and psychological distress in later life. This is further supported by Bal, Van Oost, De Bourdeaudhuij, and Crombez (2003) who found that

those who had been sexually abused in childhood used more avoidant coping strategies than those who reported a stressful event unrelated to sexual abuse, or no stressful event. Furthermore, avoidant coping mediated the relationship between the sexual abuse and the consequent stress-related symptoms.

Overall, these findings suggests that avoidance of unpleasant internal experiences is a common strategy adopted by those with abusive histories; some have argued that this may actually prevent the abusive experience from being processed in a way that is important to recovery (Cohen, Mannarino, & Deblinger, 2006; Kennerley, 1996). This is supported by Kimbrough, Magyari, Langenberg, Chesney, and Berman (2010) who carried out an 8-week mindfulness programme on 27 adult survivors of CSA. They found that overall their PTSD symptoms reduced, and that avoidance had the greatest reduction. In other words, sitting with here and now memories and experiences in a non-abusive context appeared to help participants process the traumatic thoughts, feelings or memories associated with the abuse.

These findings add weight to the relationship between childhood abuse, experiential avoidance and later psychological distress. However, many of the existing studies have small sample sizes, have been carried out on student populations and have no comparison group. Furthermore, most of the literature to date have has focused primarily on CSA which indicates that there is a need to focus on other forms of childhood abuse and explore what relationships there are with experiential avoidance.

2.8.1.2 Emotional invalidation and experiential avoidance

As stated previously, most of the literature to date has focused primarily on CSA which indicates that there is a need to focus on other forms of abuse. Some studies have only just begun to explore the concept of emotional invalidation and its relationship to experiential avoidance or avoidant coping. For example, Rosenthal et al. (2005) examined 141 undergraduate women and found that higher perceived criticism within their family of origin was associated with greater levels of experiential avoidance and distress. This is supported by (Eisenberg et al., 2001; Jones, Eisenberg, Fabes, & MacKinnon, 2002) who found that minimizing, punitive, and non-supportive parental

responses (that communicated to the child that their negative emotions were not acceptable) were associated with higher levels of avoidant coping. Furthermore, Krause et al. (2003) found, in a sample of 127 males and females, that invalidating childhood environments were associated with emotional inhibition. Furthermore, the relationship between childhood emotional invalidation and later psychological distress was mediated by emotional inhibition. Conceptually, the term emotional inhibition within this paper is similar to experiential avoidance as the authors defined it as a: 'conscious attempt to suppress emotional experience / escaping aversive emotional experiences' (Krause et al., 2003, p.200). However, Reddy, Pickett, and Orcutt (2006) criticised Krause et al. (2003) for not controlling for physical and sexual abuse within their sample and for being underpowered; so they expanded upon the study by using 987 participants. They found that experiential avoidance mediated the relationship between childhood psychological abuse and pathology in general.

A main criticism of the research looking into childhood abuse, emotional invalidation and experiential avoidance is that it has typically involved participants from non-clinical (i.e. student) backgrounds which means that it is difficult to generalise to clinical populations, particularly eating pathology, which is very specific. Furthermore, the heavy use of cross-sectional designs means that the relationships are correlational at a specific point in time. Research in this area would benefit from longitudinal designs as the long-term changes between variables could be monitored over time. Despite these criticisms, all these studies taken together do provide preliminary support for the idea that childhood abuse and emotional invalidation affects a person's ability to manage their internal experiences flexibly, which, in turn, may contribute to psychological distress in later life. Figure 3 is one way of conceptualising this (adapted from Keville et al., 2008).

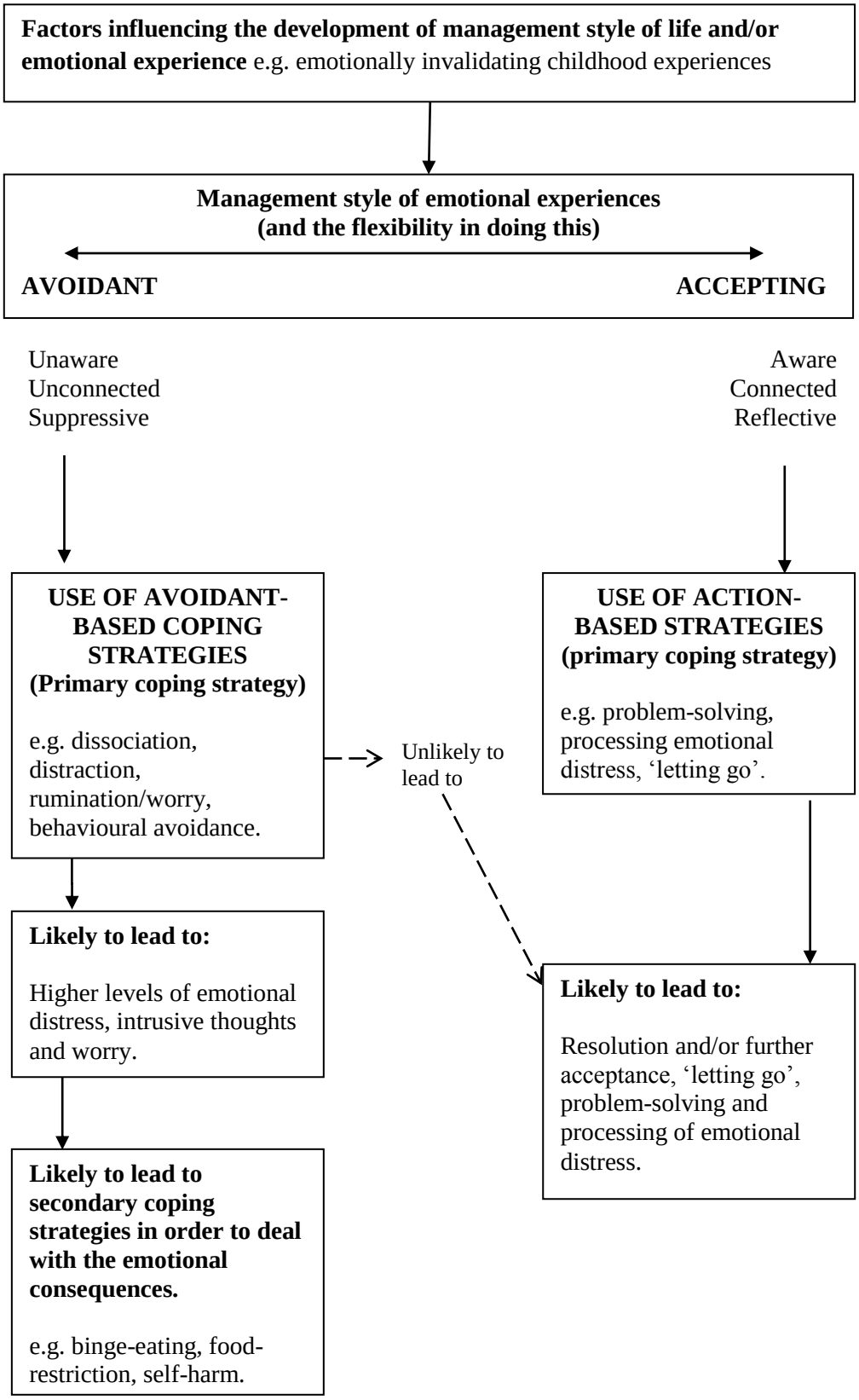

Figure 3: Conceptualising how early experiences might influence management style of life / emotional experiences. Adapted from Keville et al., (2008).

2.9 Rationale and clinical relevance

Although there has been a wealth of research into the potential risk factors for eating pathology, there has been little consideration of the 'contextual factors' such as childhood abuse and invalidation and how this may (if at all) impact on eating pathology. Of the literature that has considered these factors, it is not entirely clear why some people go on to develop an ED and others do not. For example, CEA has been neglected until recently and whilst there appears to be a unique link with eating pathology, it is not wholly clear why this may be and whether this link is present in clinical populations.

Understanding psychological distress and the ways in which one manages and processes their internal experiences is also very relevant in terms of clinical practice and the ways in which one may work with an individual. Although an individual may appear recovered externally (i.e. weight-restored), they may be on the brink of relapse if they experience psychological distress and engage in cognitive-fusion or experiential avoidance. In other words, their internal experience and the ways in which they manage affect are not 'recovered' and this could, therefore, be a potential target for intervention and assist the individual in maintaining their recovery. Whilst experiential avoidance has been looked at as a form of emotion regulation in eating pathology, it has often been subsumed under different names such as emotional inhibition and avoidant coping. Thus, its meaning could be very different between studies. Furthermore, little consideration has been given to cognitive-fusion, despite the importance of this construct within the ACT model of pathology.

Another area of clinical relevance is that increasing psychological flexibility (the opposite of inflexibility) may be a promising *preventative* intervention for those at risk of developing a full-blown clinical ED or as an intervention for those who have already been formally diagnosed. During the earlier stages of an ED, increasing psychological flexibility is likely to be perceived as less threatening than other interventions (i.e. thought-challenging in CBT), particularly as the individual may be reluctant to engage in treatment. As the individual progresses towards recovery, psychological flexibility can continue to be used by the clinician as the transdiagnostic stance of ACT means that

other difficulties associated with eating pathology (i.e. anxiety or depression) can be targeted simultaneously.

Further research is clearly needed in order to extend the existing findings and add greater depth to the knowledge base. This study hopes to address a gap within the literature by combining what is already known about childhood abuse and invalidation, psychological inflexibility and eating pathology. To the author's knowledge, each area has not been explored in combination before.

2.10 Aims and hypotheses

The aims of this study are to explore whether childhood experiences of abuse and emotional invalidation were long-term risk factors for the development of psychological inflexibility (i.e. cognitive-fusion and experiential avoidance) and eating pathology. This study hopes to explore and unravel these complex relationships in those with clinical and subclinical eating pathology. Based on the existing literature, it was hypothesised that:

1) There will be a positive relationship between eating pathology and psychological inflexibility (experiential avoidance and cognitive fusion)
2) There will be a positive relationship between eating pathology and Thought-Shape Fusion.
3) There will be a positive relationship between eating pathology and Emotional Processing.
4) There will be a positive relationship between maternal and/or paternal invalidation in childhood and eating pathology.
5) There will be a positive relationship between maternal and/or paternal invalidation in childhood and psychological inflexibility.
6) Experiences of abuse in childhood will be positively related to eating pathology.
7) Experiences of abuse in childhood will be positively related to psychological inflexibility.
8) There will be a positive relationship between Psychological Inflexibility and anxiety.

9) There will be a positive relationship between Psychological Inflexibility and depression.
10) There will be a positive relationship between eating pathology and anxiety and/or depression.

Additional hypotheses:

1a) the elevated and typical clinical groups will show higher psychological inflexibility than the low clinical group.
2a) the elevated and typical clinical groups will show higher levels of Thought-Shape-Fusion than the low clinical group.
3a) the elevated and typical clinical groups will show poorer levels of emotional processing than the low clinical group.
4a) the elevated and typical clinical groups will show higher levels of maternal and paternal invalidation than the low clinical group.
6a) the elevated and typical clinical groups will show higher levels of childhood abuse than the low clinical group.
10a) the elevated and typical clinical groups will show higher levels of anxiety and/or depression than the low clinical group.

3 METHOD

3.1 Design

A non-experimental, correlational design was used in order to examine the relationships between the study variables: childhood experiences of abuse and emotional invalidation; clinical and subclinical EDs and current psychological inflexibility. In order to investigate these study variables and collect the data, self-report questionnaires were made available online which meant that a web-based survey design was employed. This was deemed to be an advantageous method of data collection as it was low in cost and enabled a specific population to be targeted in a way that did not require a lot of their time. A major limitation of correlational research is that it is not possible to establish firm causal relationships. However, a causal hypothesis can be put forward.

Although some of the questionnaires were designed to measure the participants' past experiences, the study design was arguably cross-sectional because the questionnaires were completed by the participants at a specific point in time; thus their responses may have been influenced by their current life circumstances i.e. they may have under or over reported the severity of their difficulties. Furthermore, the nature of the self-report questionnaires means that the participant's retrospective accounts of their early experiences (particularly the difficult experiences such as childhood abuse), may have been affected by retrospective bias or distorted memory (Hardt & Rutter, 2004).

3.2 Participants

3.2.3 Intended target population and sampling

The target population for the study were males and females from the ages of sixteen onwards, who had been diagnosed with a clinical or subclinical ED and/or had some serious concerns about their eating habits. The participants were recruited from online advertisements posted on four ED support forums and three mental health charity websites within the UK: 'Beating Eating Disorders', 'No Bodies Perfect' and 'Men Get Eating Disorders Too'. Due to the nature in which the participants were recruited, the sample can be classified as a self-selected, opportunity sample.

3.2.2 Statistical power and sample size

In order to determine the minimum sample size required to detect an effect, a power calculation was carried out using GPower3.1. This revealed that a sample size of 111 would be sufficient to detect a medium effect size (r = .30) with an alpha error at .05 and power at 95%. This is displayed in figure 4. According to Cohen (1992) an effect size correlation of r = .20 is regarded as a small effect and r = .50 is detected as a medium effect.

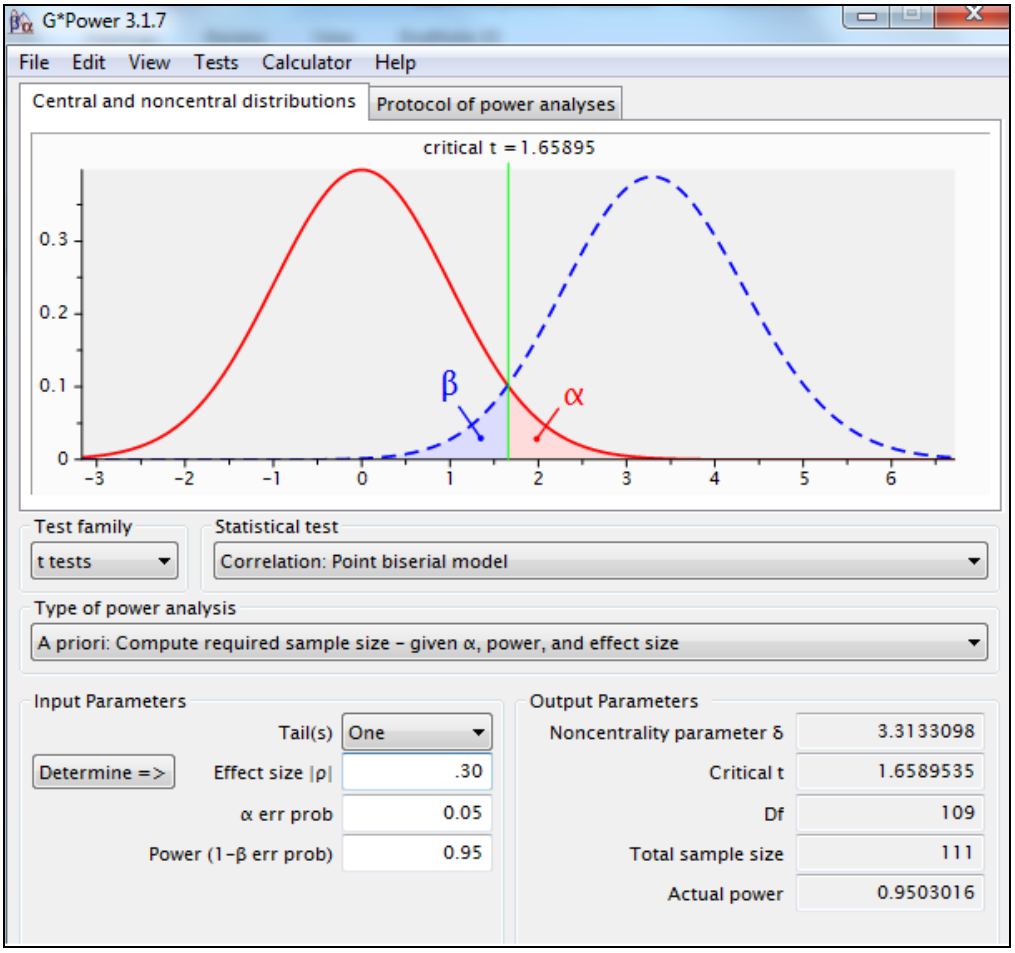

Figure 4: power calculation

3.2.3 Response rate, sample selection and final sample size

A total of 473 people entered the survey online. However, just under half of these (N = 222, 47%) did not complete the survey as they dropped out at various points throughout (see appendix 1 for a flow chart). This left 250 (53%) people who were eligible for inclusion into the study but an initial screen of the data revealed that 24% (N = 60) did not consider themselves to have an ED. These were therefore excluded from the analysis which left a final sample size of 190 people (40%) who fully completed the survey.

3.3 Measures

At the beginning of the survey, each participant was asked to complete a brief questionnaire (developed for this study) about relevant background information, including their age, gender, ethnicity and current employment and marital status. Additional questions asked about whether the participants had a current or historical

diagnosis of an ED and whether they were currently receiving input from services. Following this, eight standardised self-report questionnaires were administered in order to explore the key variables of this study. A copy of all the measures used can be found in appendices 2 – 10.

3.3.3 Childhood Trauma Questionnaire – short form (CTQ-SF; Bernstein et al., 2003)

This CTQ-SF was used as a measure of childhood abusive experiences. It is a 28-item retrospective self-report questionnaire which has been designed to provide quick screening for histories of abuse and neglect amongst clinical and non-clinical groups. It assesses five dimensions of childhood maltreatment: Emotional abuse, Emotional neglect, Sexual abuse, Physical abuse and Physical neglect. It also has a minimization/denial scale which consists of 3 items aimed at detecting the possible underreporting of maltreatment (false negatives). When completing the questionnaire, individuals are required to respond to a series of statements about childhood events on a 5-point Likert scale which ranges from 1 (*Never true)* to 5 *(Very often true).* Seven items are reverse scored and twenty-one items are summed to give a total score ranging from 28 to 128. The final score quantifies the severity of maltreatment in each area and the higher the score, the greater the severity of maltreatment.

3.3.1.1 Reliability and Validity

The psychometric properties of the CTQ-SF were studied on across seven samples of clinical and non-clinical individuals (N = 2,201). The CTQ-SF was found to have good levels of internal consistency on most scales apart from physical neglect: Emotional abuse (median = .89), physical abuse (median = .82), sexual abuse (median = .92) emotional neglect (median = .90) and physical neglect (median = .66). The CTQ-SF was also found to have good levels of test-retest reliability which suggests that respondent's trauma reports on the CTQ-SF are stable over time. Finally, it was found to have good content and construct validity (Bernstein & Fink, 1998; Bernstein et al., 2003). This is supported by more recent studies which have examined the psychometric characteristics of the CTQ-SF (Paivio & Cramer, 2004; Thombs, Bernstein, Lobbestael, & Arntz, 2009).

3.3.2 Invalidating Childhood Environments Scale (ICES; Mountford et al., 2007)

The ICES was used as a self-report, retrospective measure of childhood invalidation. It contains 14 items that examine specific maternal and paternal behaviours thought to reflect the eight themes that define an invalidating environment: ignore thoughts and judgements; ignore emotions; negate thoughts and judgements; negate emotions; over-react to emotions; overestimate problem solving; over-react to thoughts and judgements; and oversimplify problems (Linehan, 1993). Respondents are asked to rate their experience up to the age of 18 years and each item is rated on a Likert scale which ranges from 1 (*never*) to 5 (*all the time*) for each parent. Four items are reverse scored and ten items are summed to give a total score ranging from 0 to 70. Higher scores reflect a higher perception of emotional invalidation by each parent. Mountford et al. (2007) found that the ICES correlates with levels of eating pathology, as measured by the Eating Disorders Inventory and with measures of emotional processing, such as distress tolerance (Mountford et al., 2007). Mountford et al., (2007) also provides norms for the clinical eating disordered group (paternal scale M = 34.7; maternal scale M = 31.7) and the non-clinical group (paternal scale M = 27.8; maternal scale M = 28.2).

3.3.2.1 Reliability and Validity

The ICES has been validated on a clinical and non-clinical ED population. The ICES paternal invalidation and maternal invalidation scales have been shown to have good levels of internal consistency amongst the clinical group (paternal invalidation α= .79; maternal invalidation α = .77). However, the level of consistency was lower amongst the non-clinical group (paternal invalidation α = .58; maternal invalidation α= .66) (Mountford et al., 2007). In contrast, Robertson, Kimbrel, and Nelson-Gray (2013) found that the ICES demonstrated excellent internal consistency within a non-clinical sample of female women.

3.3.3 The Eating Disorders Inventory (EDI-3; Garner 2004)

The EDI-3 is a 91 item self-report questionnaire of the constructs which are clinically relevant to those with EDs and it has a six-choice response format. It is organised into

twelve primary scales and when the *T* score of two or more scales are added together, it produces one of six composite scores. The scales that will be used in this study are; Drive for Thinness (DT), Bulimia (B) and Body Dissatisfaction – a total of 25 items – where the total score of these three scales produce the Eating Disorder Risk Composite score (EDRC). The EDRC provides a global measure of eating and weight concerns and it is divided into the following three categories:

- Elevated clinical range (67^{th} to 99^{th} percentile) which is indicative of extreme eating and weight concerns.
- Typical clinical range (25^{th} to 66^{th} percentile) reflects significant eating concerns that characterise most individuals with a clinical ED. And;
- Low clinical range (below the 25^{th} percentile) which suggests that the individual does not have significant problems with eating and weight concerns. A score in this range is common amongst non-clinical samples.

In sum, the higher the EDRC score, the greater the level of eating and weight concerns. For the purpose of this study, the EDRC will be used to indicate the level of eating pathology for a given participant.

The remaining nine scales i.e. Low-Self Esteem, Personal Alienation, Interpersonal Insecurity, Interpersonal Alienation, Interoceptive Deficits, Emotional Dysregulation, Perfectionism, Ascetism and Maturity Fears assess psychological constructs that have conceptual relevance to the development of EDs. However, they were not administered as the EDI-3 manual indicates that the EDRC is sufficient to identify those with clinical and subclinical EDs.

A key deficit with the EDI-3 is that it contains no information about its utility and applicability to males (Cumella, 2006). There is limited and contradictory research on the EDI in males with some authors stating that it is valid (Keel, Baxter, Heatherton, & Joiner, 2007) and others stating that it is not (Rathner & Rumpold, 1994). Stanford and Lemberg (2012) concluded that, although the overall EDI-3 does not adequately detect symptoms in males with EDs, the EDRC is a far more reliable measure. To the author's knowledge, there are no existing measures for specifically detecting EDs in males. It

was, therefore, viewed that *cautiously* capturing some information using the EDRC would be better than excluding males altogether.

3.3.3.1 Reliability and Validity

The EDI-3 is the third version which was developed in response to the criticisms of its predecessor. It has been standardised in a US and International female sample and it includes normative and diagnostic groups. The EDRC reliability across all groups was .90 – .97 (median = .94). Furthermore, the test-retest stability coefficient for the EDRC is excellent at .98. Clausen, Rosenvinge, Friborg, and Rokkedal (2011) independently tested the psychometric properties of the EDI-3 by comparing 561 females with a clinical ED with 878 non-clinical controls. They found that all the EDI-3 subscales discriminated significantly ($p < .001$) and strongly (Cohen's d ranging from .71 to 2.0) between clinical and non-clinical controls. Furthermore, the internal consistency of each subscale was also found to be satisfactory for both clinical and non-clinical controls.

3.3.4 The Acceptance and Action Questionnaire (AAQ-II; Bond et al., 2011)

The AAQ-II was used as a measure of psychological inflexibility and experiential avoidance. This is a 7-item self-report questionnaire and the respondents are asked to rate the degree to which each statement applies to them using a 7-point Likert scale which ranges from 1 (*Never True)* to 7 (*Always True*). All seven items are summed to give a total score ranging from 7 to 49. The higher the score, the greater the level of experiential avoidance.

3.3.4.1 Reliability and Validity

Bond et al (2011) report that the results from 2,816 participants indicates that the AAQ-II is reliable and valid. For example, the mean alpha coefficient is .84 (.78 – .88) and the test-retest reliability is .81 and .79 over a 3 month and 12 month period respectively. They also found that the AAQ-II had similar concurrent, predictive and discriminate validity to that of the AAQ-I (r =.97), however, they also acknowledged that further research was needed to fully consider the psychometric properties of the AAQ-II.

3.3.5 The Cognitive Fusion Questionnaire (CFQ; Gillanders et al. (in press)

The CFQ was used as a measure of cognitive fusion. It was originally a 42-item self-report questionnaire and it has now been refined into a 7-item fusion only scale. The CFQ was developed to address the limitations of other published scales in the literature as cognitive-fusion was not adequately operationalised. For example, there tended to be a focus on specific aspects of fusion such as 'believability of thoughts' which meant that the broader aspects of cognitive fusion were missed i.e. inability to view cognitive events from a different perspective and reacting emotionally to events. Each item in the CFQ is rated on a 7-point Likert scale ranging from 1 (*Never True*) to 7 (*Always True*). All items are summed to give a total score ranging from 7 to 49. The higher the score, the greater the level of cognitive fusion.

3.3.5.1 Reliability and Validity

Based on a series of studies involving over 1,800 people, the CFQ has been shown to have excellent internal consistency and good test-retest reliability (r=.80). The CFQ also has adequate validity and can discriminate between clinical and nonclinical populations (Gillanders et al., in press)

3.3.6 Thought-Shape Fusion Scale (TSF; Coelho et al., 2013)

The TSF was used as a measure of cognitive-fusion in relation to body shape and/or weight. This is a shortened version of the original 34-item questionnaire (TSF; Shafran et al., 1999) as it contains 18 items regarding thought-shape fusion (TSF). Each item on this scale is rated on a 4-point Likert scale which ranges from 0 (*Not at all)* to 4 (*Totally/always*). All items are summed to give a total score ranging from 0 to 72 and the higher the score, the higher the level of TSF.

3.3.6.1 Reliability and Validity

The TSF-S was validated on a nonclinical population and an ED transdiagnostic sample. The TSF-S was shown to have good construct validity as it can distinguish between clinical and nonclinical samples. It was also shown to have high internal consistency (Coelho et al., 2013). However it is worth pointing out that the TSF-S was validated on a

limited sample size, thus, further research is needed to investigate its psychometric properties in greater detail. To the author's knowledge, there has been no further research on the TSF-S in the literature.

3.3.7 Emotional Processing Scale (ESP-25;(R. Baker et al., 2010)

The EPS-25 was used as a measure of general emotional processing. This is a 25 item self-report questionnaire which measures five emotional processing styles: Suppression, unregulated emotion, avoidance, signs of unprocessed emotion and impoverished emotional experience. Each item on the EPS-25 is measured on a 10-point Likert scale ranging from 0 (*completely disagree*) to 9 (*completely agree*). The total EPS-25 score is calculated by summing the means for each subscale and dividing by five. These can then be compared to the normative data which is as follows: Non-clinical group = 2.5 (25^{th} percentile) and 4.4 (75^{th} percentile) and the Clinical Group = 4.0 (25^{th} percentile) and 5.9 (75^{th} percentile).

3.3.7.1 Reliability and Validity

The EPS-25 has been shown to have good internal consistency (α = .92) and the test re-test reliability for the entire scale is good (r = .74). However, the test-retest reliability for the individual subscales were variable with impoverished emotional experience being the highest (r=.84) and unprocessed emotion being the lowest (r=.48) (R. Baker et al., 2010). Despite this, the EPS-25 was able to distinguish a healthy control group from the following clinical groups: mental health and pain.

3.3.8 Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith 1983)

The HADS was used as a measure of anxiety and depression due to their comorbidity with eating pathology (APA workgroup on eating disorders, 2006). This is a 14 item self-report questionnaire of depression and anxiety and it is quick to complete. Each item is rated on a 4-point Likert scale with each question and subsequent response being phrased slightly differently. The total score ranges from 0 – 21 for the depression subscale and 0 – 21 for the anxiety subscale. For both subscales the scoring is as follows: 0 – 7 is regarded as being in the normal/non-clinical range, 8 – 10 is regarded as being in

the borderline range (possible mood disorder is present) and a score of 11+ is regarded as being in the clinical range.

3.3.8.1 Reliability and Validity

The HADS has been shown to have a cronbach's coefficient of .80 for anxiety and .76 for depression in a sample of 69,648 adult participants. It was therefore concluded that the basic psychometric properties of the HADS was good in terms of factor structure, intercorrelation, homogeneity, and internal consistency (Mykleton, Stordal and Dahl, 2001). An updated literature review of the HADS was conducted by Bjelland (2002) who also found that the HADS was a valid and internally consistent measure of anxiety and depression. The author would like to acknowledge that, whilst there continues to be some debate within the literature as to how acceptable the HADS's psychometric properties are (Cosco, Doyle, Ward, & McGee, 2012; Coyne & van Sonderen, 2012a, 2012b), it was viewed that given its strengths this would be an appropriate measure to use in this study.

3.3.9 Body Mass Index (BMI)

A calculation of Body Mass Index (BMI) was undertaken. The BMI uses an individual's current weight and current height to calculate their body fatness and the following formula was used for those aged 20 and over: participants: *weight (kg) ÷ [height (m)]*^{2}. In this study, the BMI was used to indicate body fatness and whether the individual was underweight, overweight or at an ideal weight for their height. As the amount of body fat changes with age and differs between males and females, the BMI for those aged 16 – 19 was calculated by taking into account their height, weight, age and sex. This was then compared to the relevant BMI-for-age growth chart on the Centre for Disease Control (CDC) and Prevention website as these differences are taken into account. Once the BMI has been calculated, it is plotted on the BMI-for-age growth charts (for either girls or boys) in order to obtain a percentile ranking. The percentile then indicates the relative position of the individual's BMI compared to others of the same sex and age. The BMI-for-age weight status categories differ in the U.K from the U.S. and the corresponding percentiles are shown for each country in Table 1. The US classification system was used in this study in order to maintain consistency with the EDI-3 which is a US measure.

Table 1: US and UK BMI-for-age weight status categories

Weight Status Category	US BMI	US Percentile	UK BMI	UK Percentile
Underweight	Less than 18.5	Less than 5th percentile	Less than 18.5	Less than 2nd percentile
Healthy Weight	18.5 - 25	5th percentile to less than 85th percentile	18.5 - 25	3rd percentile to less than the 90th percentile
Overweight	25 – 30	85th to less than 95th percentile	25 – 30	91st to less than 98th percentile
Very Overweight	More than 30	Equal to or greater than the 95th percentile	More than 30	Equal to or greater than the 98th percentile

3.4 Procedure

Three charities and four ED support forums (see appendix 11 for a full list) were contacted via email outlining the study and asking for permission to place an advertisement on their website. When permission was granted, an advert was placed on the website (see appendix 12) and it contained a link which directed participants to the study website, Bristol Online Survey (BOS). Once the participants had clicked the link, they were presented with a welcome message which gave prior warning of the sensitive nature of the questions (appendix 13). The participants were requested to click continue in order to view the information sheet (appendix 14) and provide informed consent (appendix 15) to participate. The questionnaires were then presented in standard order with a reminder message about withdrawing from the study half-way through (appendix 16). All of the questions were compulsory with the exception of 'parent/caregiver two' on the ICES scale as some participants may have come from single parent families. The rationale behind the compulsory questions was to minimise missing data. Once the participants had completed the study, they were presented with a debrief sheet (appendix 17) which provided some more information about the study along with the contact details of the author along with external sources of support in the event of distress. All of the data contained in BOS was exported directly into IBM SPSS statistics database for analysis. The online dataset in BOS was then deleted.

3.5 Ethical Considerations

The author was mindful that some of the questionnaires were sensitive in nature and that some of the participants may experience distress. All of the participants were presented with a welcome message and an information sheet which made them aware that some of the questions may result in them feeling upset and distressed. All participants were informed that their participation was voluntary and that they could withdraw from the study at any time without giving a reason. They were also asked to confirm that they understood this when they provided informed consent.

It was acknowledged that some of the participants may not complete the study and, as such, may not be debriefed. The information sheet provided all participants with the contact details of relevant sources of support and they were encouraged to make a note of these so that they could refer to them if needed. Furthermore, all participants were given the contact details of the researcher and primary supervisor so that they could contact the researcher prior to, during or after participation. The debriefing sheet also contained information about sources of support such as 'The Samaritans', 'Beating Eating Disorders' and The National Association for People Abused in Childhood.

In order to ensure anonymity and confidentiality, none of the participants were asked to provide their names or any other identifying information when completing the questionnaires. BOS was the only survey programme recommended by the University of Hertfordshire due to its high security and encryption levels. This meant that any online data submitted by the participants was confidential and securely saved. All participants were assigned a unique ID number and the researcher was the only person who could access the online dataset as it could only be accessed by entering a username and password.

Ethics approval (appendix 18) was obtained from the University of Hertfordshire Research Ethics Committee and once ethics approval had been obtained (protocol number aLMS/PG/UH/00022), the survey website opened. Further ethics approval was not deemed necessary as the participants for this study were recruited from outside the National Health Service.

4 RESULTS

The results of the data analysis will be split into five sections. Section's 1 and 2 will begin with a description of the socio-demographic and clinical status of the final sample. Section 3 will include the testing of each hypothesis which will be defined and discussed in turn. Section 4 includes the findings obtained from the two multiple regression analyses conducted, then a series of mean group comparisons on the main study variables will be presented in section 5. Where there are outliers across the data set, these were checked to ensure they were not erroneous entries.

4.1 SECTION 1: Sample description

4.1.1 Age, gender and ethnicity

Table 2 shows that the sample was comprised of more females (91%) than males (9%), with the majority of participants classifying themselves as White British (48%). There was a broad range in age with the youngest being 16 and the eldest being 53. However the majority of the sample (55%) was between 20 – 29 years of age.

Table 2: Frequency and percentage of age, gender and ethnicity.

Variable	Category	Frequency	Percentage
Age	16 – 19	48	25
	20 – 29	104	55
	30 – 39	26	14
	40 – 49	9	5
	50 – 59	3	2
Total		190	100
Gender	Male	17	9
	Female	173	91
Total		190	100
Ethnicity	White British	91	48
	White Irish	17	9
	Any Other White Background	62	33
	Mixed White And Black Caribbean	1	0.5
	Mixed White And Asian	1	0.5
	Any Other Mixed Background	6	3
	Hispanic Or Latino	8	4
	Any Other Ethnic Groups	4	2
Total		190	100

4.1.2 Relationship and employment status

Table 3 shows that over half the sample were single (55%), followed by those in a long-term relationship (19%) and those who were married/in a civil partnership (13%). With regards to employment status, the majority of sample classified themselves as students (41%) and this was followed by 28% (N= 53) who were working either full or part-time.

Table 3: Frequency and percentage of relationship and employment status

Variable	Category	Frequency	Percentage
Relationship Status	Single	105	55
	In a long-term relationship	36	19
	In a new relationship	15	8
	Married/Civil Partnership	25	13
	Cohabiting	8	4
	Widowed	1	1
Total		**190**	**100**
Employment Status	Full-time	40	21
	Part-time	33	17
	Unemployed	18	10
	Looking for work	5	3
	Student	77	41
	Unemployed due to disability, physical or mental health	8	4
	Part-time student & part-time work	5	3
	Full time parent/carer	5	2
Total		**190**	**100**

4.2 SECTION 2: Clinical Status

4.2.1 Mental health difficulties and eating disorder diagnosis

Descriptive statistics pertaining to mental health status are presented in Table 4 which shows that the vast majority (92%) were currently reporting mental health difficulties, whereas 8% were not. Multiple diagnoses were common with depression being the most prevalent mental health difficulty (41%) followed by anxiety (26%) and Post-Traumatic Stress Disorder (8%).

Table 4: Frequency and percentage of mental health difficulties

Variable	Category	Frequency	Percentage
Current Mental Health Difficulties	Yes	174	92
	No	16	8
Total		**190**	**100**
Mental Health Diagnosis	Depression	101	41
	Anxiety	64	26
	Post-Traumatic Stress Disorder	20	8
	Obsessive Compulsive Disorder	15	6
	Bipolar Disorder	7	3
	Borderline Personality Disorder	16	7
	Body Dysmorphic Disorder	1	0.4
	Trichotillomania	3	1
	Psychosis	2	1
	Adjustment Disorder	2	1
	Other	15	6
Total		**246***	**100**

*** multiple diagnoses were common amongst the sample which is reflected in N being greater than the sample size of 190.**

An overview of ED diagnosis is presented in Table 5 which shows that the whole sample had been diagnosed with either a clinical or subclinical ED (100%). Of those who had been diagnosed, AN: Restricting-Subtype was the most common diagnosis (44%) followed by EDNOS (27%) and BN (25%).

Table 5: Frequency and percentage of eating disorder diagnosis

Variable	Category	Frequency	Percentage
Current Eating Disorder	Yes	190	100
Total		**190**	**100**
Eating Disorder diagnosis	Anorexia Nervosa: Restricting subtype	83	44
	Anorexia Nervosa: Binge-Purge subtype	6	3
	Bulimia Nervosa	48	25
	Eating Disorder Not Otherwise Specified (EDNOS)	53	28
Total		**190**	**100**

In order to explore whether those with a mental health difficulty also considered themselves to have an ED, a more detailed analysis was undertaken. This revealed that although 16 participants stated that they currently did not have a mental health difficulty, they did consider themselves to have an ED (as shown in Table 6).

Table 6: Frequency and percentage of those with a mental health difficulty and an eating disorder diagnosis

Current Mental Health Difficulties	Category	Frequency	Percentage
Mental Health Yes ***(N= 174)***	Eating Disorder Yes	174	92
Mental Health No ***(N= 16)***	Eating Disorder Yes	16	8
Total		**190**	**100**

4.2.2 Eating Pathology and treatment status

As mentioned in section 3.3.3, the Eating Disorder Risk Composite (EDRC) is being used to indicate the level of eating pathology for a given participant within this study (i.e. elevated, typical or low clinical range). The EDRC is obtained by summing the total score of the following three scales: Drive for Thinness (DT), Bulimia (B) and Body Dissatisfaction (BD) and comparing them to a normative diagnostic group. The descriptive statistics for these scales will be presented first, followed by the EDRC.

4.2.2.1 Drive for Thinness, Bulimia and Body Dissatisfaction scales

Descriptive statistics for participants' raw scores on the DT, B and BD scale, along with the three clinical ranges are presented in Table 7. It can be seen that overall, 49% of the sample were in the typical clinical range for all three scales followed by 34% who were in the elevated clinical range for all three scales and 17% who were in the low clinical range for all three scales.

Table 7: Frequency and percentage of eating pathology per scale

Eating Disorder Scale	Frequency	Percentage
Drive for Thinness *(elevated range)*	69	36
Drive for Thinness *(typical range)*	84	44
Drive for Thinness *(low range)*	37	20
Total	**190**	**100**
Bulimia *(elevated range)*	54	28
Bulimia *(typical range)*	105	56
Bulimia *(low range)*	31	16
Total	**190**	**100**
Body Dissatisfaction *(elevated range)*	70	37
Body Dissatisfaction *(typical range)*	93	49
Body Dissatisfaction *(low range)*	27	14
Total	**190**	**100**

4.2.2.2 Overall Eating Pathology (as measured by the EDRC)

The participants were then divided into three groups using the EDRC and this is shown in Table 8 below. It can be seen that 56% were classified as being in the elevated clinical range and this was followed by 27% in the typical clinical range and 17% in the low clinical range.

Table 8: Frequency and percentage of overall eating pathology

Eating pathology (EDRC percentiles)	Frequency	Percentage
Elevated clinical range (67th - 99th percentile)	107	56
Typical clinical range (25th – 66th percentile)	51	27
Low clinical range (1st – 24th percentile)	32	17
Total	**190**	**100**

A box-plot analysis of EDRC was also carried out (see figure 5) and it can be seen that overall, the median EDRC score was 70 (SD= 31) which falls in the elevated clinical range; this is suggestive of clinical levels of eating pathology.

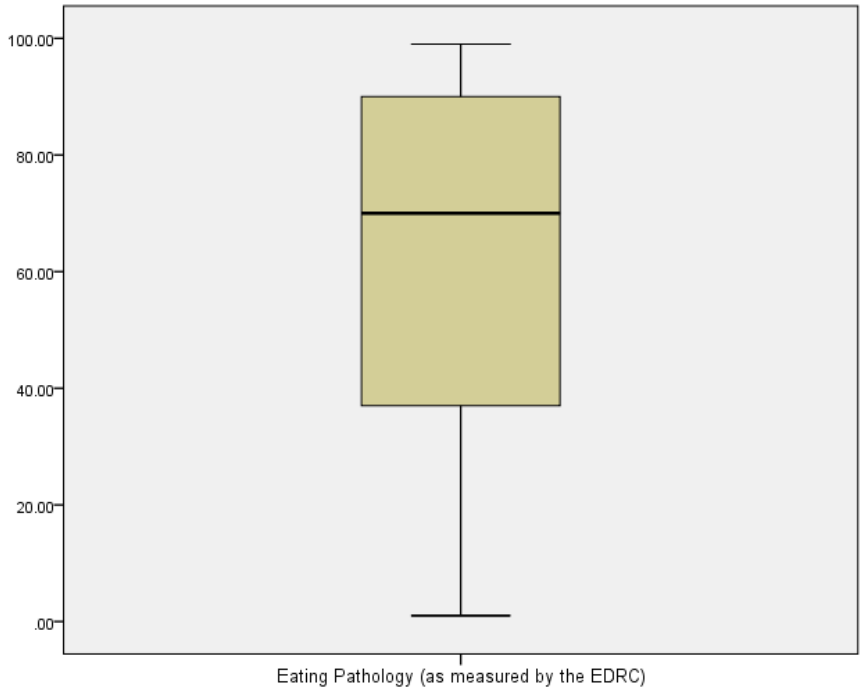

Figure 5: Boxplot of Eating Disorder Risk Composite scores

A more detailed analysis was undertaken to compare the proportion of each ED diagnostic group in relation to the EDRC and this is presented in Table 9. The elevated clinical range is the most prevalent classification for all four ED diagnoses (N= 107) with AN: Restricting Subtype (N= 64) being the highest, followed by EDNOS (N= 22).

Table 9: Overview of Eating Disorder diagnosis by EDRC

Eating Disorder Diagnosis	EDRC		
	Elevated clinical range	Typical clinical range	Low clinical range
Anorexia Nervosa: Restricting subtype	64	14	5
Bulimia Nervosa	18	15	15
EDNOS	22	18	13
Anorexia Nervosa: Binge-purge subtype	3	3	5
Total N	**107**	**50**	**33**

4.2.2.3 Eating pathology and treatment status

Data pertaining to the treatment and therapy status of the sample is presented in Table 10 and it can be seen that there were slightly more people in treatment for their ED (N= 97) than not in treatment (N= 93). This division between treatment received and not received (see appendix 20) can also be seen across all three EDRC groups with the most prevalent form of treatment being psychological therapy (47%) and outpatient treatment (18%).

Table 10: Frequency and percentage of treatment status in relation to EDRC

		EDRC			
		Elevated clinical range	**Typical clinical range**	**Low clinical range**	
Variable	**Category**				**Total N (%)**
Currently Receiving Treatment for eating disorder	Yes	53 (55%)	31 (32%)	13 (13%)	97 (100%)
	No	54 (58%)	19 (20%)	20 (22%)	93 (100%)

4.2.3 Body Mass Index (BMI) classification

An overview of the participants' BMI is presented in Table 11 and this shows that just under half of the sample was within the healthy range (47%) whereas a third was in the underweight range (34%). The data was missing for 3% as the participants declined to provide their current weight which meant that their BMI could not be calculated.

Table 11: Descriptive statistics of Body Mass Index (BMI)

	Frequency	**Percentage**	**Mean**	**Median**	**Range (min-max)**
Underweight (less than 18.5)	64	34	16.6	16.9	13 – 18
Healthy (18.5 – 25)	89	47	21.1	20.9	18 – 25
Overweight (25 – 30)	15	8	27.1	26.5	26 – 30
Very Overweight (30+)	16	8	35.7	34.1	36 – 42
Missing	*6*	*3*	*n/a*	*n/a*	*n/a*
Total	**190**	**100**	*n/a*	*n/a*	*n/a*

A more detailed analysis was undertaken to compare the proportion of each BMI group in relation to eating pathology and this is presented in Table 12. It can be seen that across all four BMI groups, the elevated clinical range was most prevalent (57%) followed by the typical clinical range (26%) and the low clinical range was low (17%). Although the majority of the sample had a healthy BMI, over a third of these (35%) were in the elevated to typical clinical range. The second most prevalent BMI amongst the sample was the underweight category (48%) and 31% of these were in the typical to elevated clinical range.

Table 12: Frequency and percentage of BMI in relation to eating pathology

	Eating pathology (EDRC)		
BMI	**Elevated clinical**	**Typical clinical**	**Low Clinical**
Underweight (less than 18.5)	43 (23%)	14 (8%)	7 (4%)
Healthy (18.5 – 25)	44 (24%)	25 (14%)	20 (11%)
Overweight (25 – 30)	8 (4%)	4 (2%)	3 (2%)
Very Overweight (30+)	10 (5%)	5 (3%)	1 (0.5%)
Total (%)	**105 (57%)**	**48 (26%)**	**31 (17%)**

4.2.4 Childhood Trauma Questionnaire (CTQ) scores

Table 13 shows that the most prevalent forms of childhood abuse experienced by the sample were emotional neglect (N= 176, 93%) and emotional abuse (N= 152, 80%). The least prevalent was physical abuse (N= 70, 37%). It can also be seen that N= 120 (63%) of the sample experienced low, moderate or severe abuse in one of three domains and that N= 47 (25%) experienced severe abuse across all five domains. Analysis of the CTQ minimisation / denial scale indicated that 13% (N= 21) of the sample may have been underreporting maltreatment. This meant that 87% (N= 169) were likely to be accurately reporting their experiences of childhood abuse.

Table 13: Frequency and percentage of CTQ raw scores (N= 190)

	Classification				
Type of Abuse	**None**	**Low**	**Moderate**	**Severe**	**Total N (%)**
Emotional Abuse	38 (20%)	38 (20%)	40 (21%)	74 (39%)	190 (100%)
Physical Abuse	120 (63%)	22 (12%)	25 (13%)	23 (12%)	190 (100%)
Sexual Abuse	100 (53%)	11 (6%)	18 (10%)	61 (32%)	190 (100%)
Emotional Neglect	14 (7%)	74 (39%)	54 (28%)	48 (25%)	190 (100%)
Physical Neglect	79 (42%)	39 (21%)	43 (23%)	29 (15%)	190 (100%)

Descriptive statistics for participants' raw scores on the CTQ are presented in Table 14 which shows that the highest mean scores were for Emotional Abuse (M= 14.4) and Emotional Neglect (M= 14.9) which is classified as moderate levels of abuse. The lowest mean score was for Physical Abuse (M= 8.0) which is classified as no abuse experienced.

Table 14: Descriptive statistics for the five CTQ subscales (N= 190).

Scale	Min	Max	Mean	Median	SD	Skewness	Kurtosis
Emotional Abuse	5	25	14.4	14.0	5.9	.10	-1.1
Physical Abuse	5	25	8.0	6.0	4.5	1.9	3.7
Sexual Abuse	5	25	10.2	5.0	7.1	1.9	-.45
Emotional Neglect	5	24	14.9	15.0	3.5	0.6	1.5
Physical Neglect	5	24	8.9	8.0	3.8	1.2	1.5

4.2.5 Invalidating Childhood Environments Scale (ICES) scores

Descriptive statistics for participants' raw scores on the ICES are presented in Table 15 and as a boxplot in figure 6. Not all participants had a maternal and paternal figure, hence the differing frequency counts and percentages. It can be seen that 98% of the sample had a maternal caregiver, 82% had a paternal caregiver and 5% (N=9) had a carer. A visual inspection of the raw data revealed that 'carers' were comprised of a

foster parent or a family member such as a grandparent. No further analyses were carried out on 'carers' as it was a very small sample size.

Table 15: Descriptive of Invalidating Childhood Environments Scale raw scores (Maternal and Paternal behaviours only)

	Frequency	Percentage	Mean ICES score	Median ICES score	SD	Skewness	Kurtosis
Maternal behaviours	186	98	35.3	34.5	13.8	.40	-.90
Paternal behaviours	156	82	39.6	40.0	14.7	.21	-.84

Paternal behaviours were the most prevalent form of invalidation (M= 39.6) and this was followed by maternal invalidation (M= 35.3). Both scores for each parent are higher than the norms presented by Mountford et al., (2007) (paternal scale *M*= 34.7; maternal scale *M*= 31.7) which suggests that the levels of invalidation experienced by the sample were high.

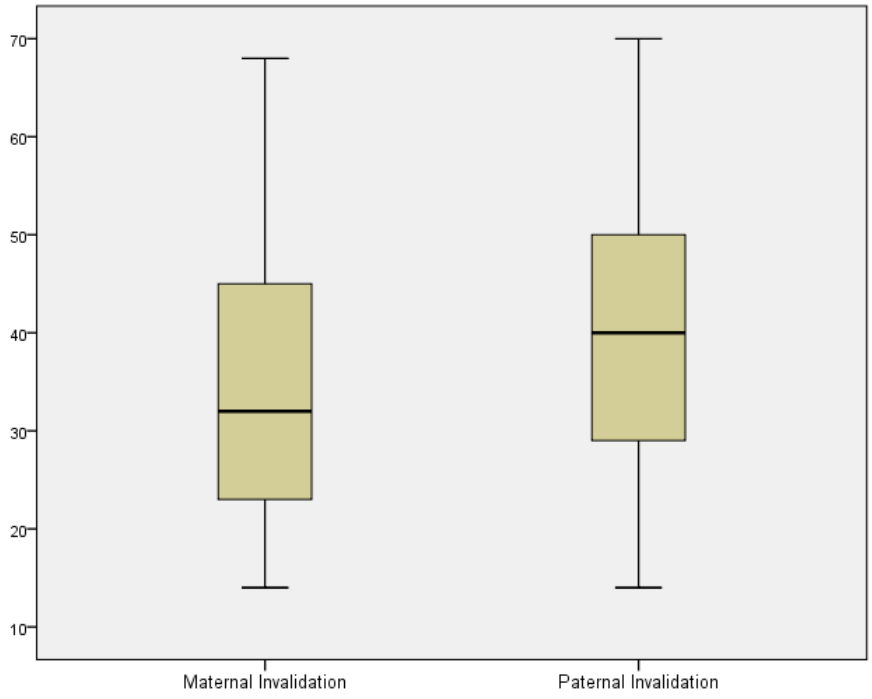

Figure 6: Boxplot of ICES maternal and paternal behaviours

4.2.6 Acceptance and Action Questionnaire (AAQ-II) scores

A summary of the participants scores on the AAQ-II are presented in Table 16 and as a boxplot in figure 7. It can be seen that the median score on the AAQ-II was 37.0 (SD= 8.1).

Table 16: Summary of the participants scores on the AAQ-II

	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min – Maximum
AAQ-II	190	100	36.0	37.0	8.1	-.97	1.08	7 – 49

Figure 7: Boxplot of AAQ-II scores

4.2.7 Cognitive Fusion Questionnaire (CFQ) scores

A summary of the participants scores on the CFQ are presented in Table 17 and as a boxplot in figure 8. It can be seen that the median score on the CFQ was 39.0 (SD= 7.7) which are higher than the norms found in a mixed mental health sample (M= 34) (Gillanders et al., in press).

Table 17: Summary of the participants scores on the CFQ

	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Maximum
CFQ	190	100	38.0	39.0	7.7	-1.05	1.91	7 – 49

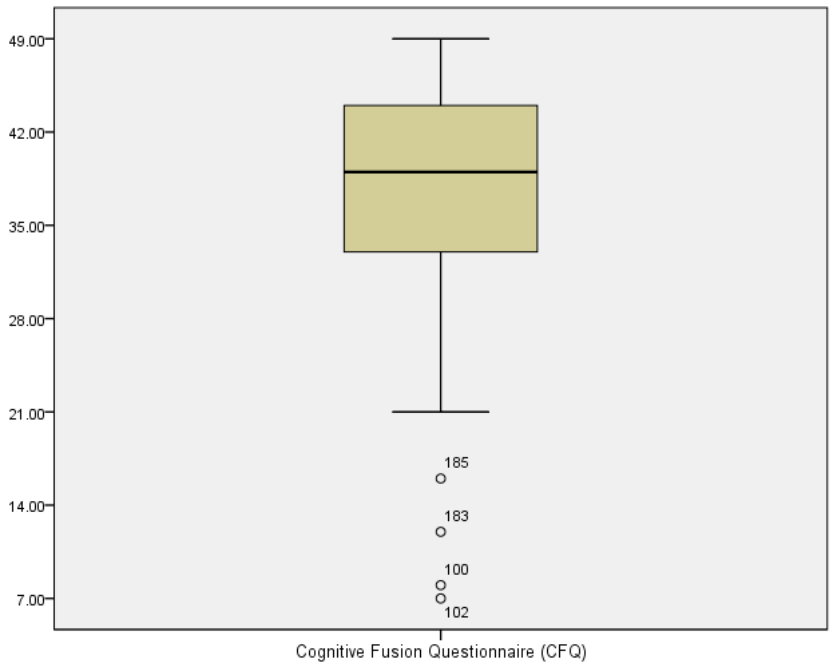

Figure 8: Boxplot of CFQ scores

4.2.8 Thought-Shape Fusion (TSF) scores

A summary of the participants scores on the TSF are presented in Table 18 and as a boxplot in figure 9. It can be seen that the mean score on the TSF was 35.7 (SD= 19.2) and that there are no outliers present.

Table 18: Summary of the participants scores on the TSF

	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min – Maximum
TSF	190	100	35.7	36.5	19.2	-.03	-.99	0 – 72

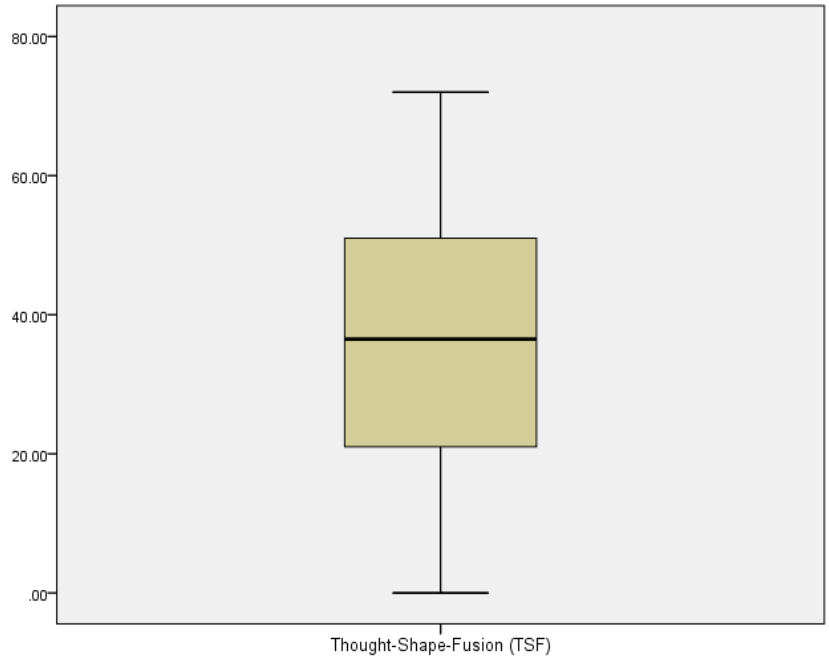

Figure 9: Boxplot of TSF scores

4.2.9 Emotional Processing (EPS-25) scores

Descriptive statistics for participants' raw scores on the EPS-25 are presented in Table 19 (see appendix 21 for a boxplot). Higher scores are indicative of poorer emotional processing and it can be seen that the most commonly used forms of emotional processing were Unprocessed emotion (M= 6.7) and Suppression (M= 6.3). The least commonly used form of emotional processing was Impoverished Emotional Experience (M= 4.9). When all five emotional processing styles are combined, it gives rise to an overall emotional processing score. It can be seen that the median levels of emotional processing for the sample was 6.0 (SD= 1.4) which suggests that the sample were in the clinical range (75th percentile) according to the norms reported by Baker et al (2010).

Table 19: Summary of the participants scores on Emotional Processing (EPS-25) N=190

	Suppression	Unprocessed	Unregulated	Avoidance	Impoverished	Total Emotional Processing
Mean (SD)	6.3 (1.9)	6.7 (1.8)	5.5 (2.1)	5.4 (1.7)	4.9 (1.9)	5.8 (1.5)
Median	6.6	7.0	5.6	5.6	5.1	6.0
Skew	-.96	-1.2	-.42	-.49	-.39	-1.0
Kurtosis	.62	1.8	-.47	.17	-.15	1.8
Min-Max	0-9	0-9	0-9	0-9	0-9	0-9

4.2.10 Hospital Anxiety and Depression Scale (HADS) scores

Descriptive statistics for participants' raw scores on the HADS is presented in Table 20 and as a boxplot in figure 10. It can be seen that over three quarters of the sample were experiencing clinical levels of anxiety (83%) and that over half were experiencing clinical levels of depression (56%).

Table 20: Frequency and percentage of Hospital Anxiety and Depression scale raw scores

	Anxiety	**Depression**
Non-Clinical Range	11 (6%)	42 (22%)
Borderline Range	21 (12%)	41 (23%)
Clinical Range	158 (83%)	107 (56%)
Total	**190 (100%)**	**190 (100%)**

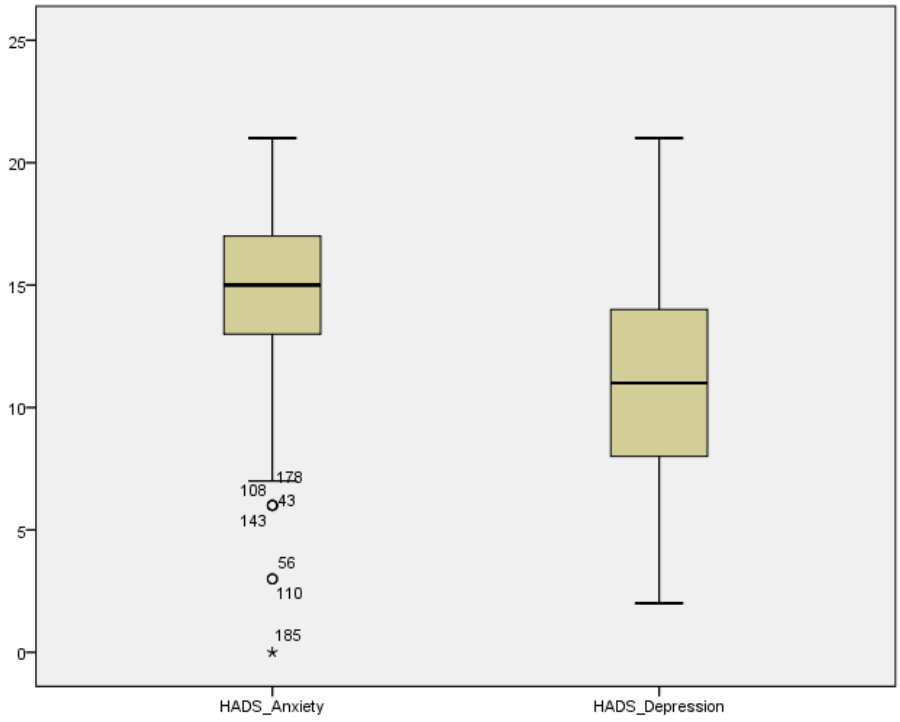

Figure 10: Boxplot of Anxiety and Depression scores

4.3 SECTION 3: Testing the hypotheses of the study

This section will now outline the hypotheses of the study and each hypothesis will be stated and tested in turn. The primary hypothesis of the study will be considered first.

Hypothesis 1: There will be a positive relationship between eating pathology and psychological inflexibility (experiential avoidance and cognitive fusion).

In order to explore the relationship between experiential avoidance and eating pathology, a scatterplot was constructed (figure 11). There appears to be a positive correlation because when eating pathology increases, experiential avoidance also increases, which is indicative of a linear relation. There was a significant and positive correlation (Pearson's r = .45, p <.01, N= 190) between experiential avoidance and eating pathology. As there were some outliers, a sensitivity analysis was undertaken and this revealed that there was a significant and positive correlation (Spearman's Rho = .42, p <.01, N= 190).

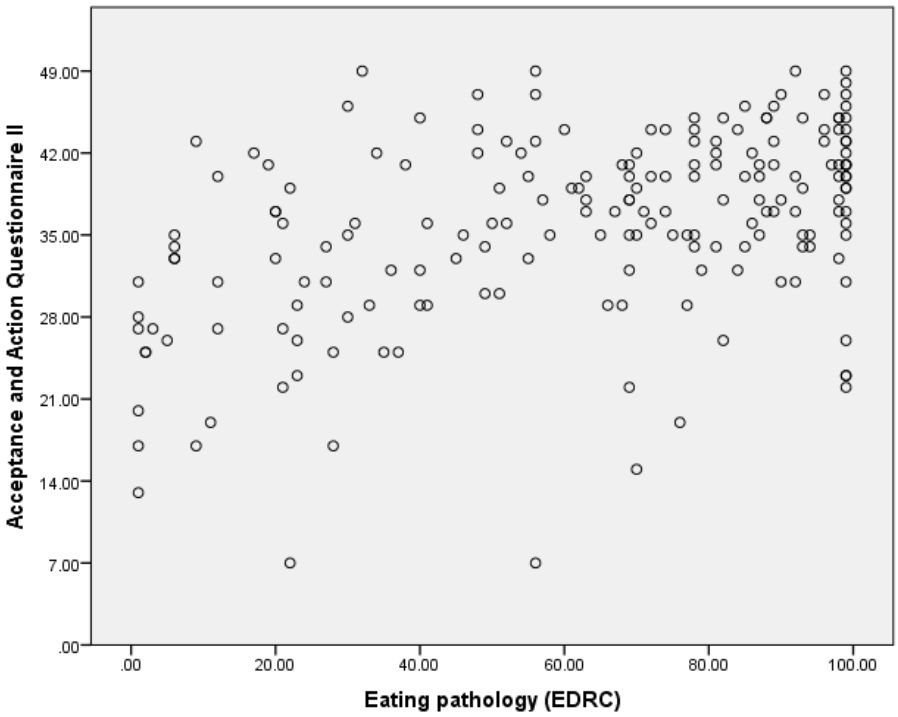

Figure 11: A scatterplot of Experiential Avoidance (AAQ-ll) and eating pathology (EDRC)

In order to explore the relationship between cognitive fusion and eating pathology, a second scatterplot was constructed (figure 12). There appears to be a positive correlation because when eating pathology increases, cognitive fusion also increases, which is indicative of a liner relation. There was a significant and positive correlation (Pearson's r = .45, p <.01, N= 190) between cognitive fusion and eating pathology. As there were

some outliers, a sensitivity analysis was undertaken and this also revealed a significant and positive correlation (Spearman's Rho = .42, p <.01, N= 190).

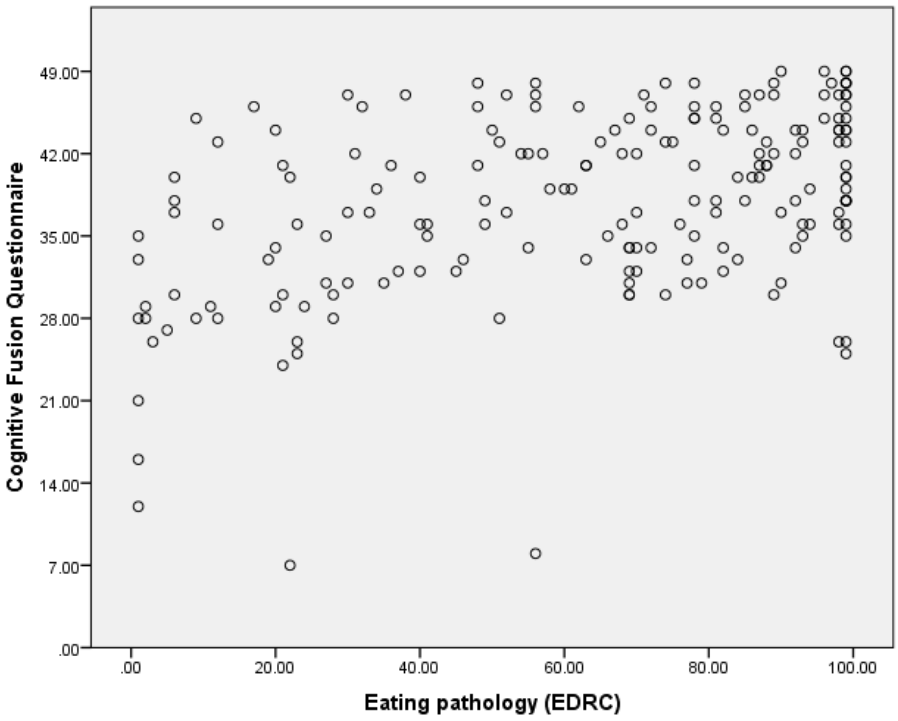

Figure 12: A scatterplot of Cognitive Fusion (CFQ) and eating pathology (EDRC)

Hypothesis 2: There will be a positive relationship between eating pathology and Thought-Shape Fusion (TSF).

In order to explore the relationship between TSF and eating pathology, a scatterplot was constructed. Figure 13 shows that there appears to be a positive correlation because when eating pathology increases, TSF also increases. There was a significant and positive correlation (Pearson's r = .58, p <.01, N= 190) between TSF and eating pathology.

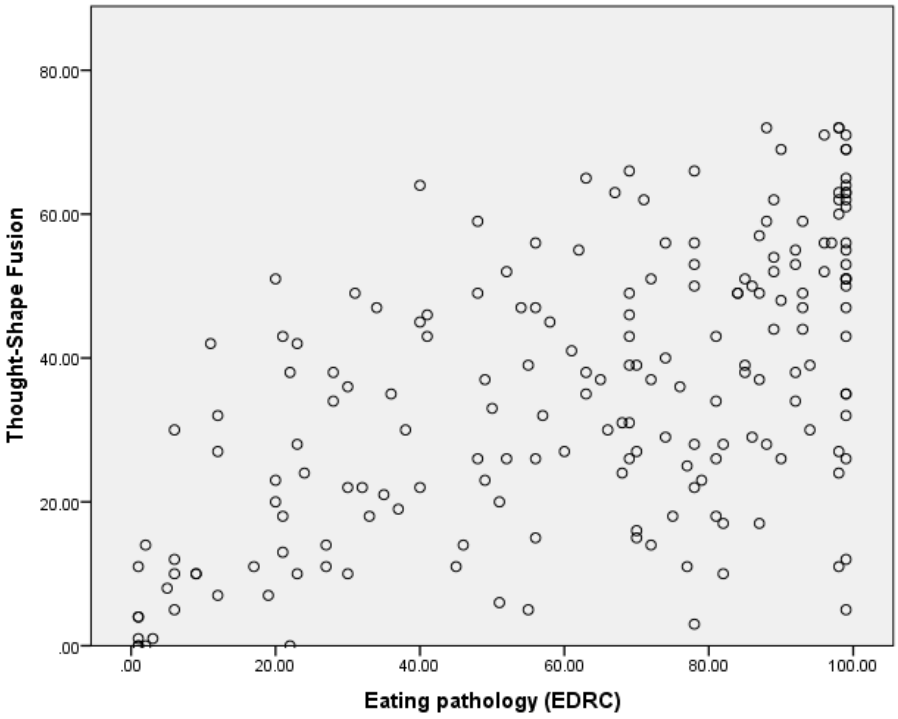

Figure 13: Scatterplot of Thought-Shape Fusion (TSF) and eating pathology (EDRC)

Hypothesis 3: There will be a positive relationship between eating pathology and Emotional Processing.

In order to explore the relationship between emotional processing and eating pathology, a scatterplot was constructed below. Figure 14 shows a positive correlation because when eating pathology increases, emotional processing increases. There was a significant and positive correlation (Pearson's r = .52, p <.01, N= 190), with higher levels of eating pathology being associated with poorer emotional processing. As there were some outliers, a sensitivity analysis was undertaken and this also confirmed that there was a significant and positive relationship (Spearman's Rho = .45, p <.01, N= 190).

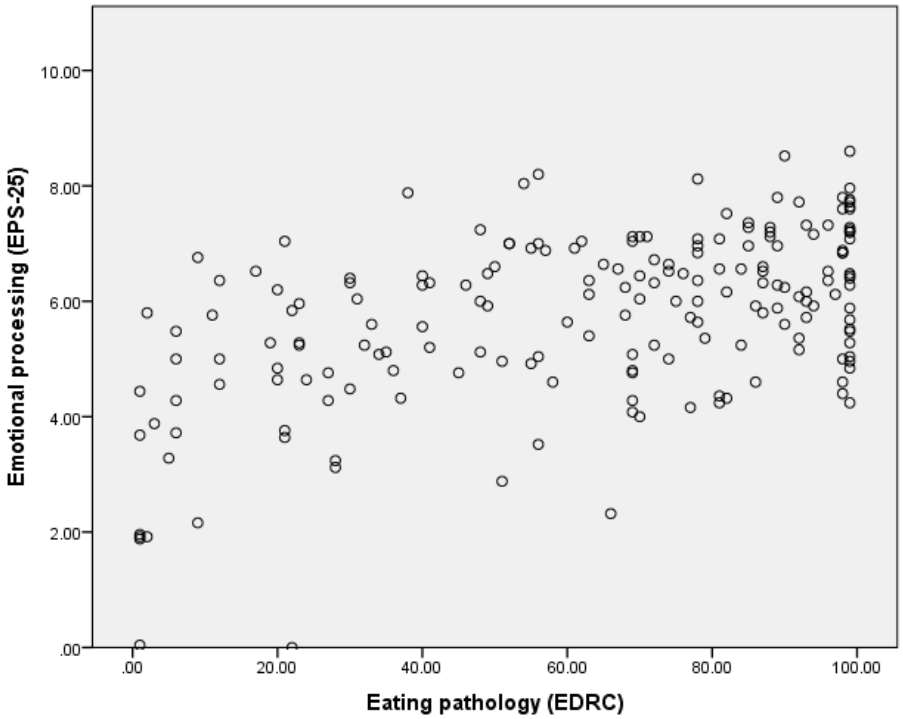

Figure 14: Scatterplot of Emotional Processing (EPS-25) and eating pathology (EDRC)

Due to some of the similarities in items on the measures of emotional processing and psychological inflexibility, it was decided to investigate whether controlling for psychological inflexibility (as measured by the AAQ-II and CFQ) had any effect on the relationship between emotional processing and eating pathology. There was a significant and positive partial correlation between emotional processing and eating pathology whilst controlling for psychological inflexibility (r = .28, p <.01, N= 190). An inspection of the zero order correlation (r = .52) suggested that controlling for psychological inflexibility had a strong effect on the strength of the relationship between these two variables.

Hypothesis 4: There will be a positive relationship between maternal and/or paternal invalidation in childhood and eating pathology.

A preliminary analysis of each scatterplot revealed that there were no linear relationships between maternal or paternal invalidation and eating pathology. A Spearman's Rho correlation coefficient was carried out and this is summarised in Table 21 below.

Table 21: Spearmans Rho correlation of the ICES and eating pathology

Scale	Invalidating environment classification	
	Maternal Invalidation (N=186)	**Paternal invalidation (N=156)**
Eating pathology	.06	.08

There were no significant relationships between eating pathology and maternal invalidation (Spearman's Rho = .06, p= .40, N= 186). There were also no significant relationships between eating pathology and paternal invalidation (Spearman's Rho = .08, p= .30, N= 156).

Hypothesis 5: There will be a positive relationship between maternal and/or paternal invalidation in childhood and psychological inflexibility.

In order to explore the relationship between psychological inflexibility and maternal invalidation, two scatterplots were constructed. With regards to experiential avoidance (figure 15), there appears to be a weak but positive correlation because when experiential avoidance increases, maternal invalidation also increases. However, there are some unusual outliers present as they show high levels of maternal invalidation and low levels of experiential avoidance, but these are not erroneous data entries. There was a significant and positive correlation (Spearman's Rho = .23, p <.01, N= 186) between Experiential Avoidance and Maternal Invalidation.

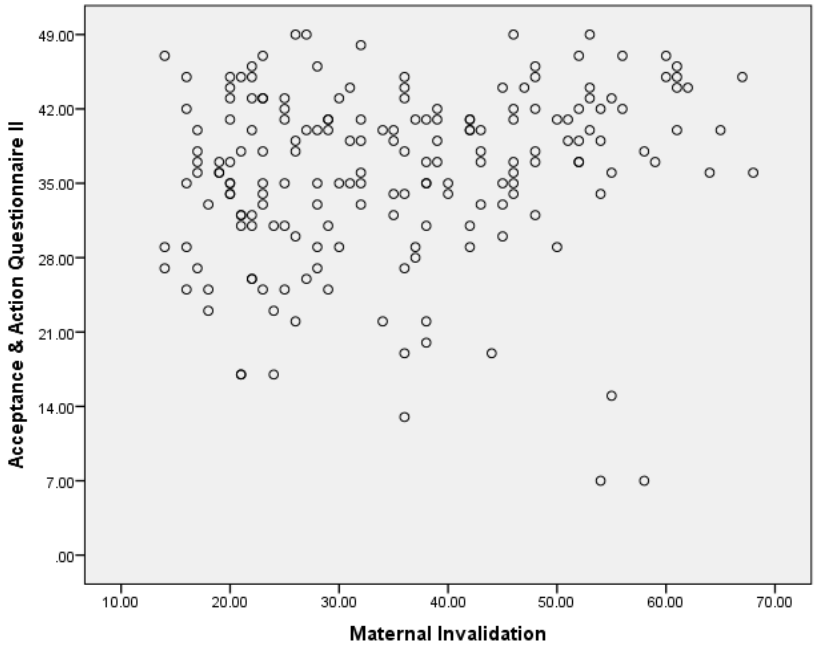

Figure 15: Scatterplot of Experiential Avoidance (AAQ-II) and Maternal Invalidation

With regards to cognitive fusion (figure 16), there does not appear to be any liner relationship between cognitive fusion and maternal invalidation. There are some unusual outliers present as they show high levels of maternal invalidation and low levels of cognitive fusion. There was no significant relationship (Spearman's Rho = .12, p= .06, N= 186) between cognitive fusion and Maternal Invalidation.

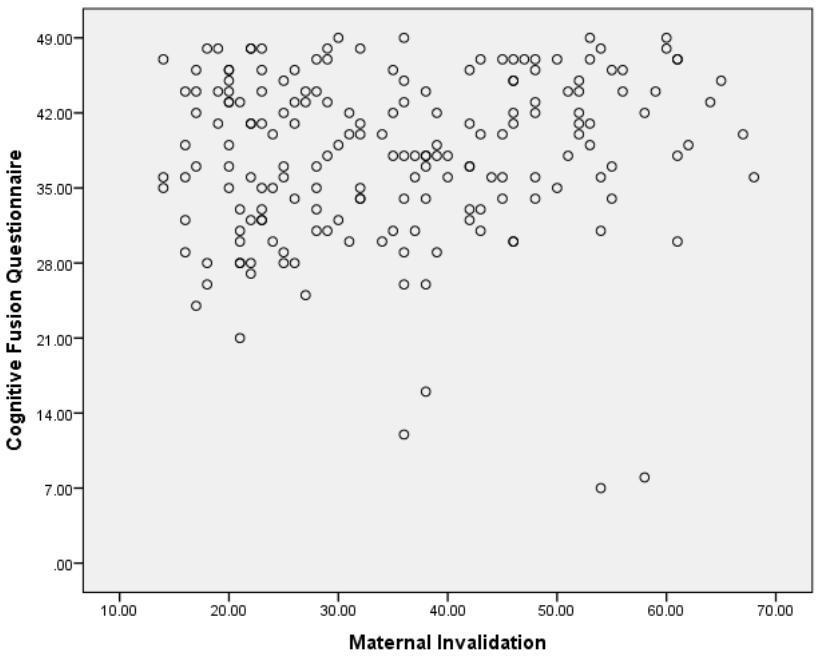

Figure 16: Scatterplot of Cognitive Fusion (CFQ) and Maternal Invalidation

In order to explore the relationship between psychological inflexibility and paternal invalidation, another two scatterplots were constructed. With regards to experiential avoidance (figure 17), there appears to be a weak but positive correlation because when experiential avoidance increases, paternal invalidation also increases. This is indicative of a small linear relation but there are some unusual outliers. Some of these could be classed as 'extreme' because they show high levels of paternal invalidation and low levels of experiential avoidance. However, these were not erroneous data entries. Despite this, a significant and positive correlation (Spearman's Rho = .24, p<.01, N= 156) was found between experiential avoidance and paternal invalidation.

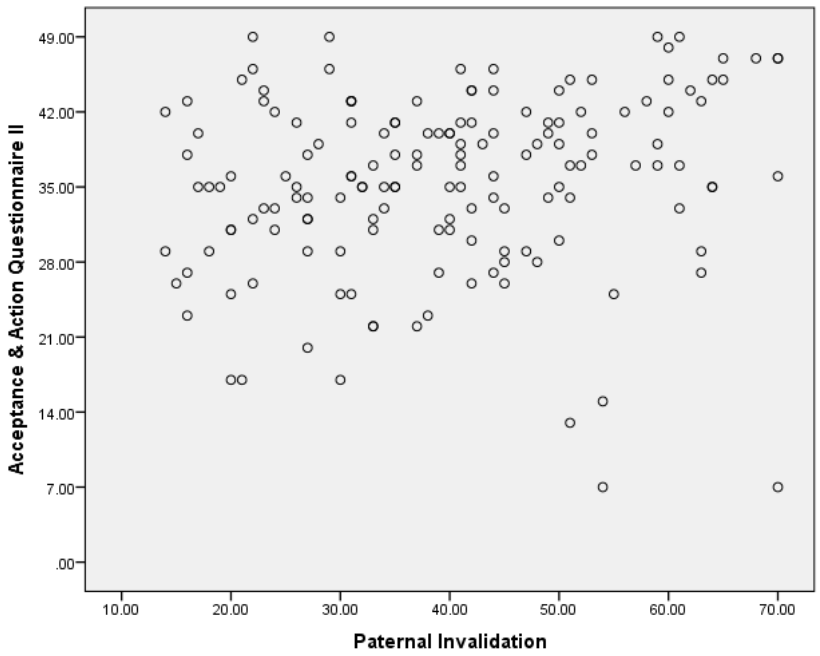

Figure 17: Scatterplot of Experiential Avoidance (AAQ-II) and Paternal Invalidation

With regards to cognitive fusion (figure 18), there appears to be a weak but positive correlation because when cognitive fusion increases, Paternal Invalidation also increases. There are some unusual outliers; some of which could be classed as 'extreme' because they show high levels of paternal invalidation and low levels of cognitive fusion. However they are not erroneous data entries. There was a significant and positive correlation (Spearman's Rho = .17, p= .03, N= 156) between cognitive fusion and Paternal Invalidation.

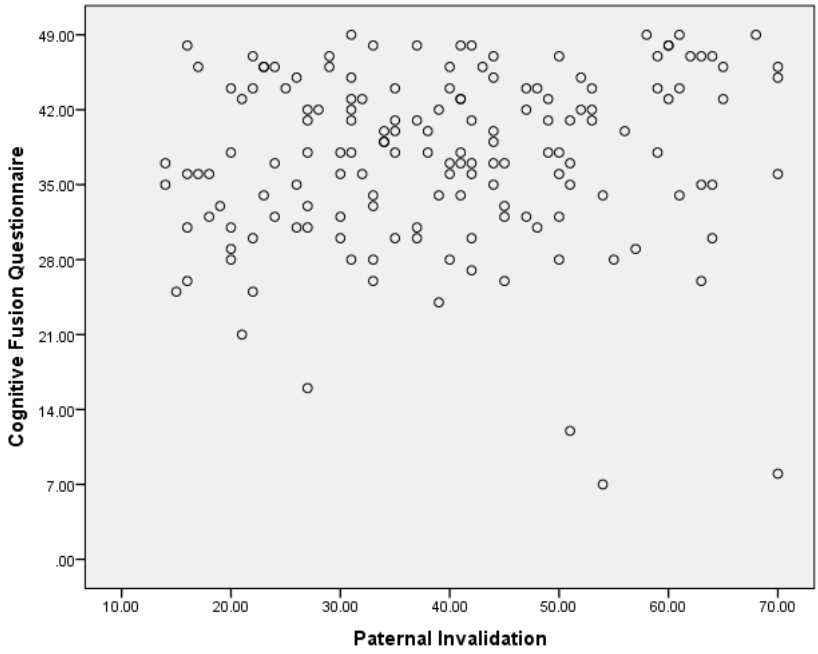

Figure 18: Scatterplot of Cognitive Fusion (CFQ) and Paternal Invalidation

Hypothesis 6: Experiences of abuse in childhood will be positively related to eating pathology.

As the five scales on the CTQ could not be combined to create an overall score, a decision was made to run separate analyses on each subscale. A preliminary analysis of each scatterplot revealed that there were no linear relationships between any of the CTQ subscales and eating pathology. A Spearman's Rho correlation coefficient was carried out on all of the childhood abuse subscales and this is summarised in Table 22.

Table 22: Spearmans Rho correlation of the CTQ subscales and eating pathology

Scale	Childhood Abuse classification				
(N= 190)	**Emotional Abuse**	**Physical Abuse**	**Sexual Abuse**	**Emotional Neglect**	**Physical Neglect**
Eating pathology	.09	-.00	.08	-.13	-.14

There were no significant relationship between eating pathology and the following five subscales: Emotional Abuse (Spearmans Rho = .09, p= .23), Physical abuse (Spearman's Rho = -.00, p= .97, N= 190), Sexual Abuse (Spearmans Rho = .08, p= .23), Emotional neglect (Spearmans Rho = .13, p= .06) and Physical Neglect (Spearmans Rho = -.15, p= .06).

Hypothesis 7: Experiences of abuse in childhood will be positively related to psychological inflexibility.

A Pearson's product-moment correlation coefficient was carried out on all of the childhood abuse subscales and this is summarised below.

Table 23: Pearsons Correlation of the CTQ subscales and AAQ-II

Scale	Childhood Abuse classification				
N= 190	**Emotional Abuse**	**Physical Abuse**	**Sexual Abuse**	**Emotional Neglect**	**Physical Neglect**
Experiential Avoidance (AAQ-II)	.27**	.11	.22**	.12	.07

** P = .01

Table 23 shows that there was a significant and positive correlation (Pearson's r = .27, p <.01) between Experiential Avoidance and Emotional Abuse. There was also a significant and positive correlation (Pearson's r = .22, p <.01) between Experiential Avoidance and Sexual abuse. There were no significant relationships between experiential avoidance and the following three subscales: Physical abuse (Pearson's r = .11, p= .14), Emotional neglect (Pearson's r = .12, p= .11) and Physical Neglect (Pearson's r = .07, p= .33).

With regards to Cognitive Fusion, only the Emotional Abuse subscale appeared to show a positive correlation because when the severity of emotional abuse increased, cognitive fusion also increased. The rest of the CTQ subscales had widely dispersed data points with no clear linear relationship. A Pearson's product-moment correlation coefficient was carried out on all of the childhood abuse subscales and this is summarised in Table 24 below.

Table 24: Pearson Correlation of the CTQ subscales and CFQ

Scale	Childhood Abuse classification				
(N= 190)	**Emotional Abuse**	**Physical Abuse**	**Sexual Abuse**	**Emotional Neglect**	**Physical Neglect**
Cognitive Fusion (CFQ)	.21**	.09	.14	.01	.03

** p = .01

There was a significant and positive correlation (Pearson's r = .21, p <.01) between Cognitive Fusion and Emotional Abuse only. There were no significant relationships between experiential avoidance and the following four subscales: Physical abuse (Pearson's r = .09, p= .20), Sexual Abuse (Pearson's r = .14, p= .06), Emotional neglect (Pearson's r = .01, p= .93) and Physical Neglect (Pearson's r = .03, p= .73).

Hypothesis 8: There will be a positive relationship between Psychological Inflexibility and anxiety.

In order to explore the relationship between Psychological Inflexibility and anxiety, two scatterplots were constructed. With regards to experiential avoidance (figure 19), there appears to be a strong, positive correlation because when anxiety increases, experiential avoidance also increases. There is a linear relationship and when a Pearson's product-moment correlation coefficient was carried out, it revealed a significant and positive correlation (Pearson's r = .65, p <.01, N= 190) between anxiety and experiential avoidance.

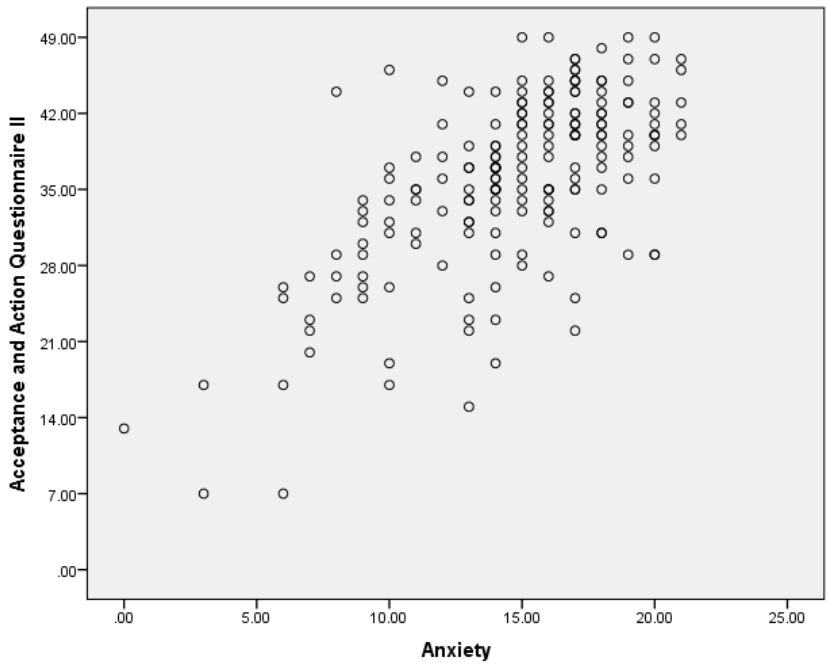

Figure 19: Scatterplot of Experiential Avoidance (AAQ-II) and Anxiety

With regards to cognitive fusion (figure 20), there appears to be a strong, positive correlation because when anxiety increases, cognitive fusion also increases. There is a linear relationship and when a Pearson's product-moment correlation coefficient was carried out, it revealed a significant and positive correlation (Pearson's r = .65, p <.01, N= 190) between anxiety and cognitive fusion.

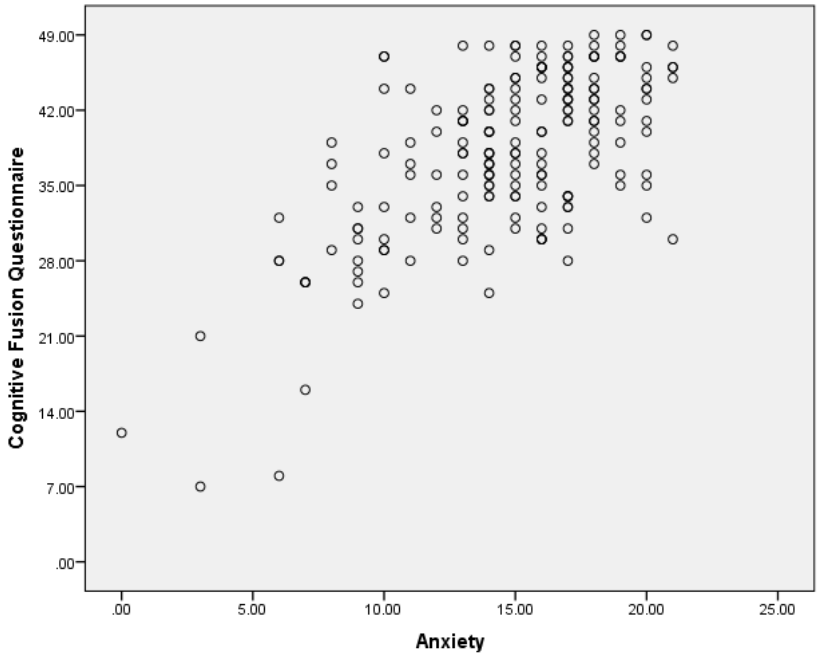

Figure 20: Scatterplot of Cognitive Fusion (CFQ) and Anxiety

A partial correlation was used to explore the relationship between psychological inflexibility and anxiety, while controlling for depression. There was a significant and positive partial correlation between experiential avoidance and anxiety whilst controlling for depression (r = .56, p <.01, N= 190). An inspection of the zero order correlation (r = .65) suggested that controlling for depression had some effect on the strength of the relationship between these two variables. There was also a significant and positive partial correlation between cognitive fusion and anxiety whilst controlling for depression (r = .57, p <.01, N= 190). An inspection of the zero order correlation (r = .65) suggested that controlling for depression had some effect on the strength of the relationship between these two variables.

Hypothesis 9: There will be a positive relationship between Psychological Inflexibility and depression.

In order to explore the relationship between Psychological Inflexibility and depression, two scatterplots were constructed. With regards to experiential avoidance (figure 21), there appears to be a positive correlation because when depression increases, Experiential Avoidance also increases which is indicative of a linear relation. There was a significant and positive correlation (Pearson's r = .50, p <.01, N= 190) between depression and Experiential Avoidance. As there were some outliers, a sensitivity analysis was undertaken and this also revealed that there was a significant and positive correlation (Spearman's Rho = .52, p <.01, N= 190).

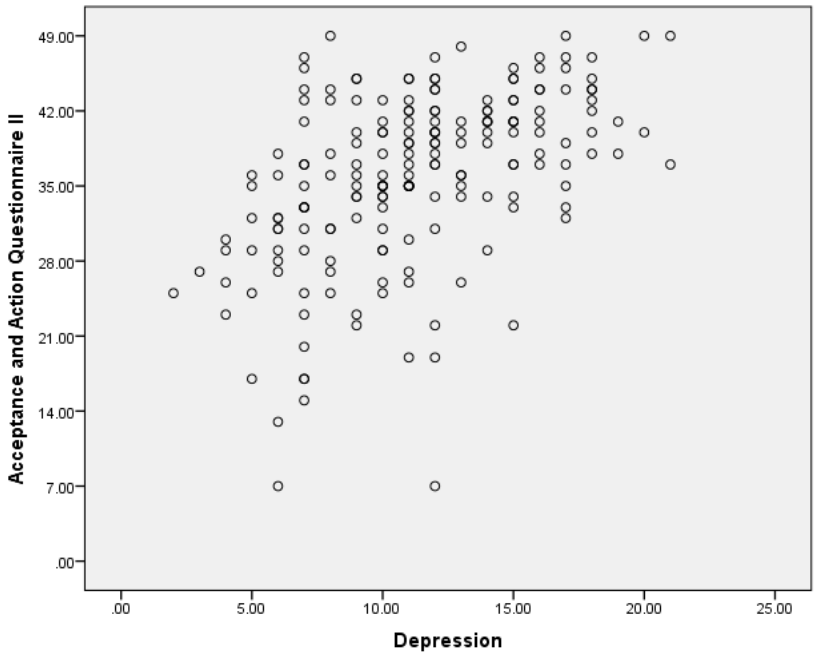

Figure 21: Scatterplot of Experiential Avoidance (AAQ-II) and Depression

With regards to Cognitive Fusion (figure 22), there appears to be a positive correlation because when depression increases, Cognitive Fusion also increases. There was a significant and positive correlation (Pearson's r = .44, p <.01, N= 190) between depression and Cognitive Fusion. As there were some outliers, a sensitivity analysis was undertaken and this also revealed that there was a significant and positive correlation (Spearman's Rho = .46, p <.01, N= 190).

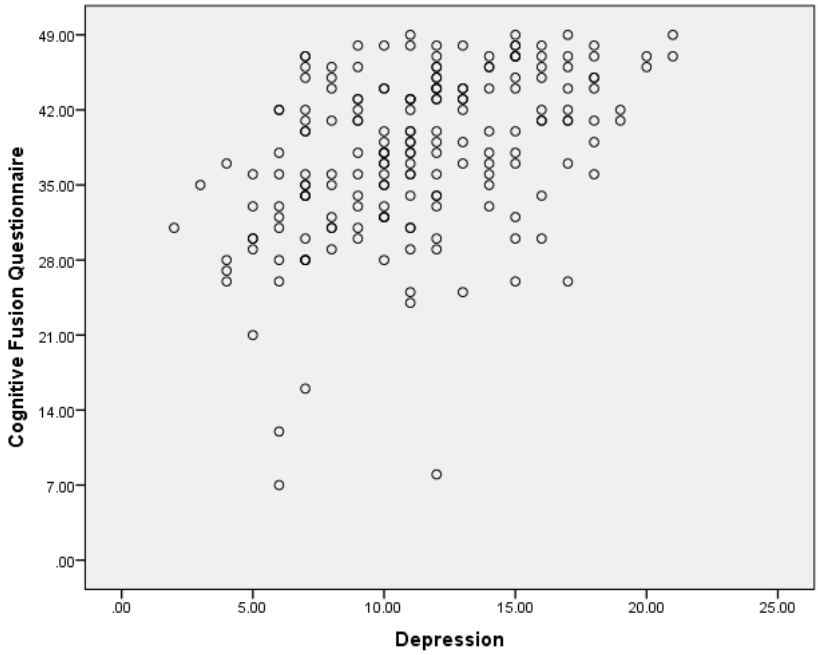

Figure 22: Scatterplot of Cognitive Fusion (CFQ) and Depression

A partial correlation was used to explore the relationship between psychological inflexibility and depression, while controlling for anxiety. There was a significant and positive partial correlation between experiential avoidance and depression whilst controlling for anxiety (r = .32, p <.01, N= 190). An inspection of the zero order correlation (r = .50) suggested that controlling for anxiety had some effect on the strength of the relationship between these two variables. There was also a significant and positive partial correlation between cognitive fusion and depression whilst controlling for anxiety (r = .24, p <.01, N= 190). An inspection of the zero order correlation (r = .44) suggested that controlling for anxiety had a strong effect on the strength of the relationship between these two variables.

Hypothesis 10: There will be a positive relationship between eating pathology and anxiety and/or depression.

In order to explore the relationship between eating pathology and anxiety, a scatterplot was constructed (appendix 22). There appeared to be a weak but positive correlation because when anxiety increases, eating pathology also increases slightly. There was a significant and positive correlation (Pearson's r = .44, p <.01, N= 190) between anxiety

and eating pathology. As there were some outliers, a sensitivity analysis was undertaken and this also revealed a significant and positive correlation (Spearman's Rho = .39, p <.01, N= 190).

A partial correlation was used to explore the relationship between eating pathology and anxiety, while controlling for depression. There was a significant and positive partial correlation between eating pathology and anxiety whilst controlling for depression (r = .32, p <.01, N= 190). An inspection of the zero order correlation (r = .44) suggested that controlling for depression had some effect on the strength of the relationship between these two variables.

A second scatterplot was constructed to explore the relationship between eating pathology and depression (appendix 23). There appeared to be a weak but positive correlation because when depression increases, eating pathology also increases slightly. There was a significant and positive correlation (Pearson's r = .42, p <.01, N= 190) between depression and eating pathology. As there were some outliers, a sensitivity analysis was undertaken and this also revealed a significant and positive correlation (Spearman's Rho = .39, p <.01, N= 190).

A partial correlation was used to explore the relationship between eating pathology and depression, while controlling for anxiety. There was a significant and positive partial correlation between eating pathology and depression whilst controlling for anxiety (r = .29, p <.01, N= 190). An inspection of the zero order correlation (r = .42) suggested that controlling for anxiety had a strong effect on the strength of the relationship between these two variables.

4.3.1 Additional analyses in relation to the hypotheses

Further analyses were carried out to explore the distribution of scores for the differing severity levels of eating pathology in relation to the hypotheses above.

(1a) the elevated and typical clinical groups will show higher psychological inflexibility than the low clinical group.

The following boxplots display the distributions, spread of scores and outliers of the AAQ-II for each group.

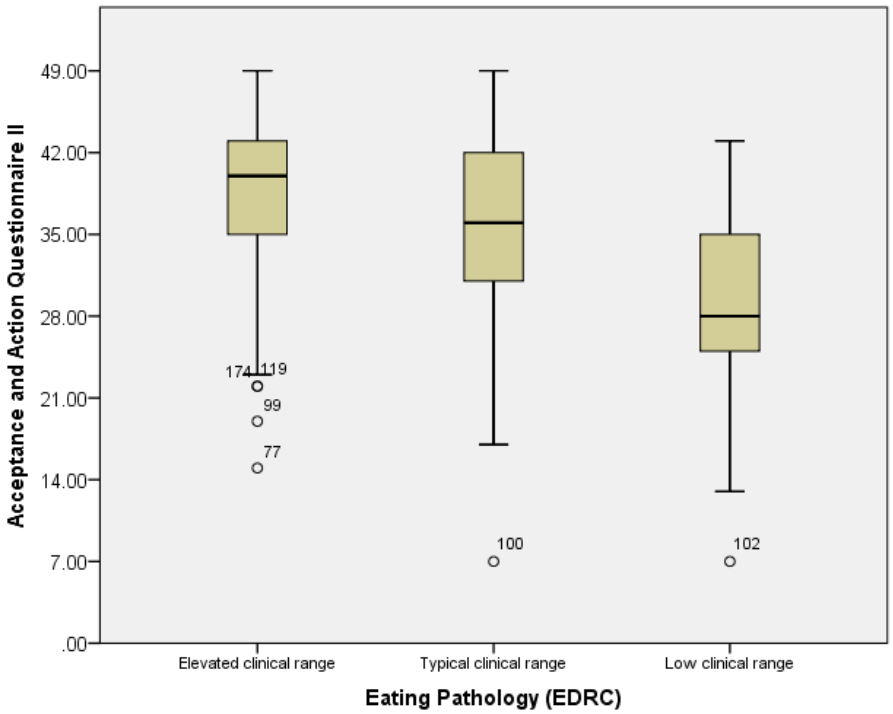

Figure 23: Boxplot of Experiential Avoidance (AAQ-II) for each eating pathology group

This shows that those in the elevated clinical group appear to have the highest levels of experiential avoidance (thus, higher levels of inflexibility) which lends some initial support to the hypothesis that inflexibility will be associated with higher levels of eating pathology. The relevant descriptive statistics are presented below.

Table 25: Descriptive statistics of Experiential Avoidance per eating pathology group

Eating pathology group			**Experiential Avoidance**					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical range	107	56	38.0	40.0	6.5	-1.1	1.6	15 – 49
Typical clinical range	50	26	35.6	36.0	8.1	-.93	2.2	7 – 49
Low clinical range	33	17	28.8	28.0	8.6	-.43	-.04	7 – 43

Table 25 shows that the elevated clinical group has the highest mean experiential avoidance score (M= 38.0), followed by the typical clinical group (M= 35.6) and the low clinical group (M= 28.8). A one-way ANOVA with eating pathology as the independent variable and AAQ-II as the dependent variable revealed significant mean differences between the three groups (F (2, 187) = 21.2, p <.01, η^2 = .18).

Table 26: Results of the ANOVA exploring mean difference between eating pathology groups in relation to experiential avoidance

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	2.7	1.3	.09	0.2
Typical clinical compared to Low clinical	6.8*	1.7	.00	0.8
Low clinical compared to Elevated clinical	-9.5*	1.5	.00	1.2

* p = .05

Post-hoc comparisons (see Table 26) indicated that: (a) the typical clinical group was significantly different from the low clinical group and (b) that the low clinical group was significantly different from the elevated clinical group. In other words, the elevated and typical clinical groups have higher experiential avoidance than the low clinical group.

The following boxplot displays the distributions, spread of scores and outliers of the CFQ for each eating pathology group.

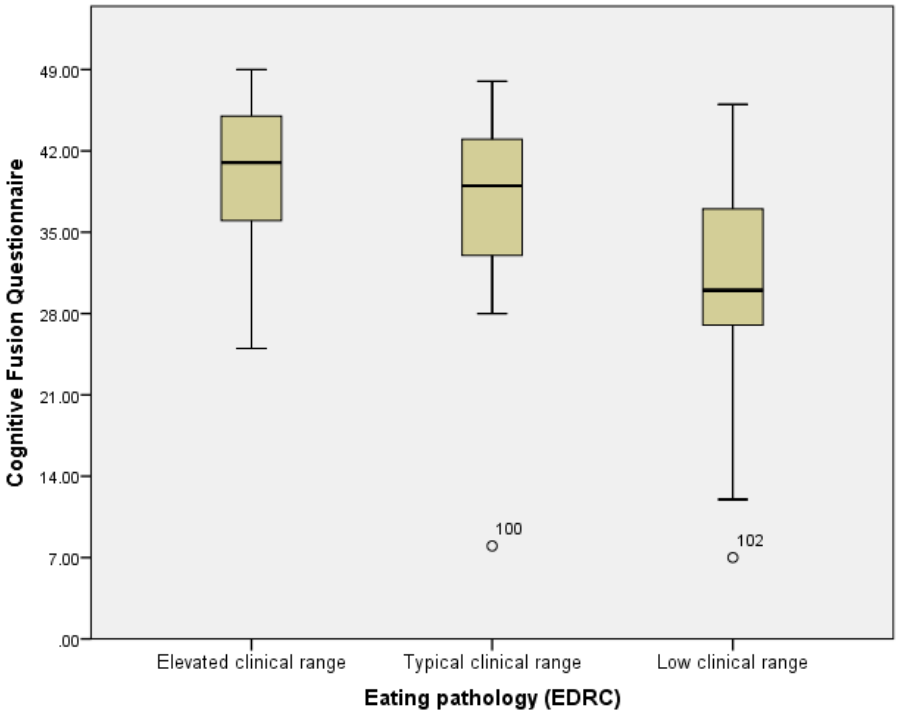

Figure 24: Boxplot of Cognitive Fusion (CFQ) for each eating pathology group

This shows that those in the elevated clinical group appear to have the highest levels of cognitive fusion (thus, higher levels of a component that may underlie inflexibility) which lends some initial support to the hypothesis that inflexibility will be associated with higher levels of eating pathology. The relevant descriptive statistics are presented below.

Table 27: Descriptive statistics of Cognitive Fusion per eating pathology group

Eating pathology group			**Cognitive Fusion**					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical range	107	56	40.2	41.0	6.0	-.48	-.66	25 – 49
Typical clinical range	50	26	38.0	39.0	7.2	-1.4	4.5	8 – 48
Low clinical range	33	17	31.0	30.0	9.0	-.58	.59	7 – 46

Table 27 shows that the Elevated clinical group have the highest mean cognitive fusion (M= 40.2), followed by the Typical clinical group (M= 38.0) and the Low clinical group (M= 31.0). In order to examine the differences between the three groups, a one-way

ANOVA was carried out with eating pathology as the independent variable and CFQ as the dependent variable and this revealed that there were significant mean differences between the three groups (F (2, 187) = 22.0, p <.01, η^2 = .19).

Table 28: Results of the ANOVA exploring mean difference between eating pathology groups in relation to cognitive fusion

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	2.2	1.2	.18	0.3
Typical clinical compared to Low clinical	6.9*	1.6	.00	0.9
Low clinical compared to Elevated clinical	-9.2*	1.4	.00	1.2

* p = .05

Post-hoc comparisons (see Table 28) indicated that: (a) the Typical clinical group was significantly different from the Low clinical group, (b) that the Low clinical group was significantly different from the Elevated clinical group and (c) there was no significant difference between the Elevated clinical and Typical clinical groups. In other words, the elevated and typical clinical groups had higher cognitive fusion than the low clinical group.

(2a) the elevated and typical clinical groups will show higher levels of Thought-Shape Fusion than the low clinical group.

Figure 25 and Table 29 shows that those in the elevated clinical group appear to have the highest levels of thought-shape fusion (thus, higher levels of inflexibility) (M= 43.1), followed by the Typical clinical group (M= 32.9) and the Low clinical group (M= 16.1).

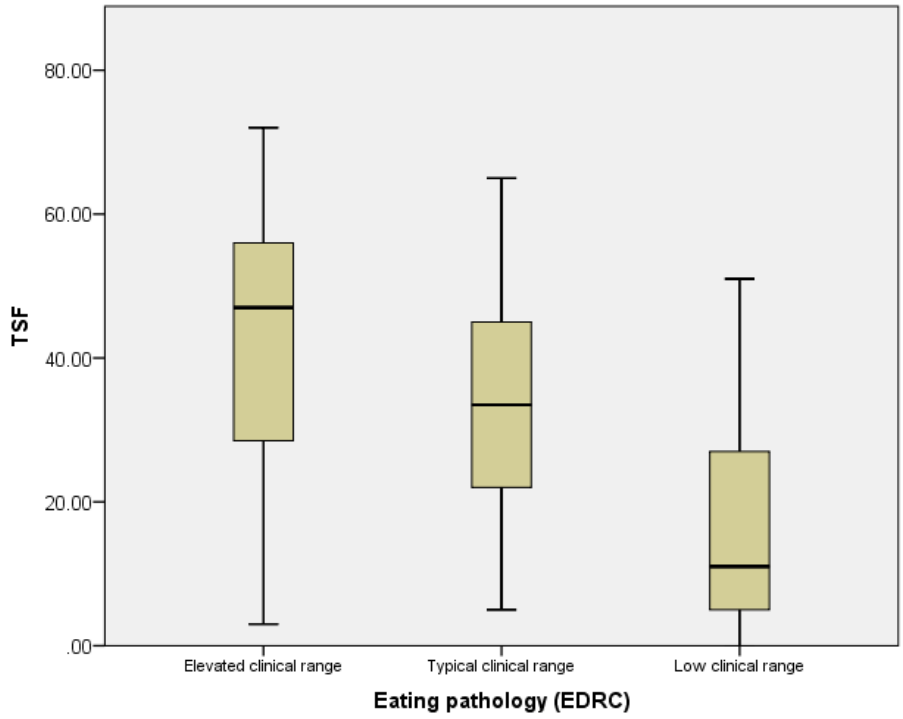

Figure 25: Boxplot of the TSF for each eating pathology group

Table 29: Descriptive statistics of Thought-Shape Fusion per EDRC

Eating pathology group			**Thought-Shape Fusion**					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical	107	56	43.1	47.0	17.6	-.29	-.84	3 – 72
Typical clinical	50	26	32.9	33.5	15.3	.15	-.69	5 – 65
Low clinical	33	17	16.1	11.0	14.7	.89	-.29	0 – 51

In order to examine the differences between the three groups, a one-way ANOVA was carried out with eating pathology as the independent variable and TSF as the dependant variable and this revealed that there were significant mean differences between the three groups ($F (2, 187) = 34.6$, $p < .01$, $\eta^2 = .27$).

Table 30: Results of the ANOVA exploring mean difference between eating pathology groups in relation to thought-shape fusion

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	10*	2.8	.01	0.6
Typical clinical compared to Low clinical	17*	3.7	.00	1.1
Low clinical compared to Elevated clinical	-27*	3.2	.00	1.7

*p = .05

Post-hoc comparisons (see Table 30) indicated that: (a) the Elevated clinical group was significantly different from the Typical clinical group; (b) the Typical clinical group was significantly different from the Low clinical group and; (c) the Low clinical group was significantly different from the Elevated clinical group. This suggests that the elevated and typical clinical groups had higher thought-shape fusion than the low clinical group.

(3a) the elevated and typical clinical groups will show poorer levels of emotional processing than the low clinical group.

Figure 26 shows the distributions and spread of scores of the EPS-25 for each eating pathology group and it can be seen that those in the elevated and typical clinical groups appear to have the highest levels of emotional processing (thus, poorer processing of emotions) which lends some initial support to the hypothesis that poorer emotional processing will be associated with higher levels of eating pathology.

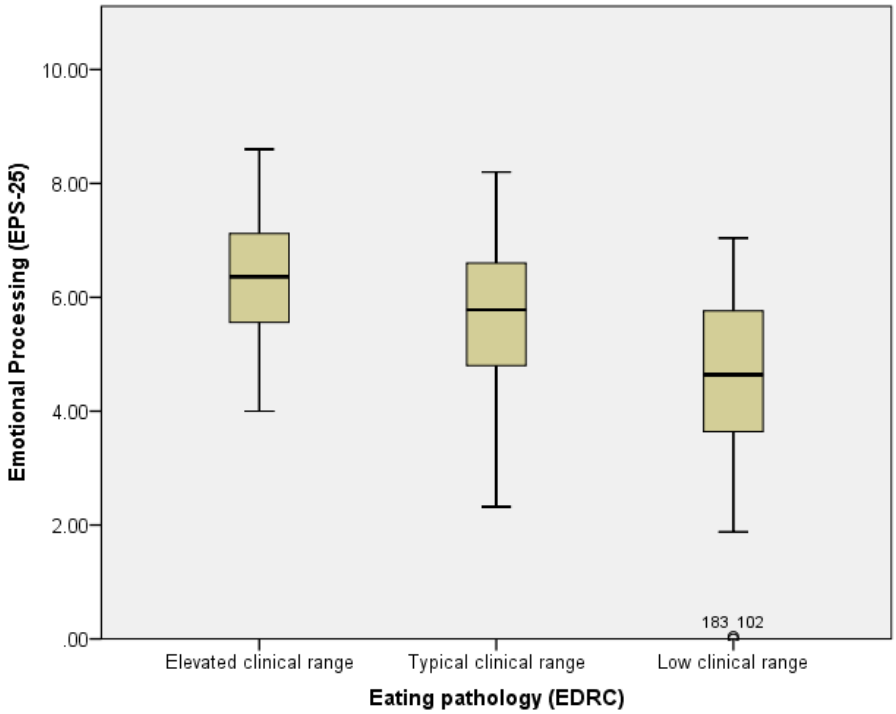

Figure 26: Boxplot of the Emotional Processing Scale per eating pathology group

Table 31: Descriptive statistics of Emotional Processing per eating pathology group

Eating pathology group			**Emotional Processing (EPS-25)**					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical range	107	56	6.3	6.4	1.0	-.27	.59	4 – 8
Typical clinical range	50	26	5.7	5.8	1.3	-.42	-.02	2 – 8
Low clinical range	33	17	4.3	4.6	1.8	-.77	.01	0 – 7

Table 31 shows that the Elevated clinical group had the poorest emotional processing (M= 6.3), followed by the Typical clinical group (M= 5.7) and the Low clinical group (M= 4.3). In order to examine the differences between the three groups, a one-way ANOVA was carried out with eating pathology as the independent variable and EPS-25 as the dependant variable. The assumption of homogeneity of variance was violated; therefore the Welch F ratio is reported. There were significant mean differences between the three groups (F (2, 187) = 18.8, p <.01, η^2 = .25). Post-hoc comparisons were carried out and the results are presented below.

Table 32: Results of the ANOVA exploring mean difference between eating pathology groups in relation to Emotional Processing

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	.61*	.22	.00	0.5
Typical clinical compared to Low clinical	1.4*	.29	.00	0.9
Low clinical compared to Elevated clinical	-1.9*	.26	.00	1.4

* p = .05

Table 32 indicated that: (a) the Elevated clinical group was significantly different from the Typical clinical group; (b) the Typical clinical group was significantly different from the Low clinical group and; (c) the Low clinical group was significantly different from the Elevated clinical group. This suggests that the elevated and typical clinical groups had poorer emotional processing than the low clinical group.

(4a) the elevated and typical clinical groups will show higher levels of maternal and paternal invalidation than the low clinical group

Descriptive statistics for maternal and paternal invalidation per eating pathology group are presented below.

Table 33: Descriptive statistics of maternal and paternal invalidation per eating pathology group

Eating pathology group			Maternal Invalidation					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical	107	56	35.7	35.0	13.9	.34	-1.0	14 – 65
Typical clinical	50	26	36.7	35.0	14.5	.39	-.94	14 – 68
Low clinical	33	17	31.0	32.0	11.7	.45	-.48	14 – 59
			Paternal Invalidation					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical	107	56	39.8	39.5	14.0	.15	-.84	14 – 70
Typical clinical	50	26	40.5	41.0	15.9	.29	-.82	14 – 70
Low clinical	33	17	37.9	37.0	15.0	.24	-.98	15 – 64

Table 33 shows that Paternal Invalidation was the highest for all three groups. The Typical clinical group had the highest levels of Maternal (M= 36.7) and Paternal invalidation (M= 40.5) and this was followed by the Elevated clinical group which had similar, but slightly lower levels of Maternal (M= 35.7) and Paternal (M= 39.8) Invalidation. The low clinical group had the lowest levels of maternal (M= 31.0) and paternal invalidation (M= 37.9). In order to explore the impact of Maternal Invalidation on eating pathology, a one-way ANOVA was conducted which revealed no significant differences between the three groups (F (2, 183) = 1.31, p= .27). Another ANOVA was conducted to explore the impact of Paternal Invalidation on eating pathology and this revealed no significant differences between the three groups (F (2, 153) = .259, p= .77).

(5a) the elevated and typical clinical groups will show higher levels of childhood abuse than the low clinical group.

In order to examine the impact the different forms of childhood abuse on the severity of eating pathology, a series of one-way ANOVA's were carried out. With regards to

Childhood Emotional Abuse, there were no significant differences between the three groups (F (2, 187) = .689, p= .50). With regards to Childhood Physical Abuse, there were no significant differences between the three groups (F (2, 187) = .339, p= .71). With regards to Childhood Sexual Abuse, there were no significant differences between the three groups (F (2, 187) = 1.28, p= .23). With regards to Childhood Emotional Neglect, there were no significant differences between the three groups (F (2, 187) = 2.40, p= .09). With regards to Childhood Physical Neglect, there were no significant differences between the three groups (F (2, 187) = 2.39, p= .09). This hypothesis was therefore disconfirmed.

(6a) the elevated and typical clinical groups will show higher levels of anxiety and/or depression than the low clinical group.

Descriptive statistics for anxiety per group are presented in Table 34 which shows that the Elevated clinical group have the highest levels of anxiety (M= 15.8) and that the Low clinical group have the lowest levels of anxiety (M= 11.7).

Table 34: Descriptive statistics of anxiety levels per eating pathology group

Eating Pathology group			**Anxiety**					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical range	107	56	15.8	16.0	3.2	-.63	.29	6 – 21
Typical clinical range	50	26	14.1	14.5	3.7	-.38	-.61	6 – 20
Low clinical range	33	17	11.7	13.0	4.7	-.68	-.28	0 – 19

In order to examine the differences in anxiety between the three groups, a one-way ANOVA was carried out with eating pathology as the independent variable and anxiety as the dependant variable. The assumption of homogeneity of variance was violated; therefore the Welch F ratio is reported. There were significant mean differences between the three groups (F (2, 187) = 12.5, p <.01, η^2 = .15).

Table 35: Results of the ANOVA exploring mean difference per eating pathology group in relation to anxiety

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	1.7*	.62	.02	0.6
Typical clinical compared to Low clinical	2.4*	.82	.01	0.5
Low clinical compared to Elevated clinical	-4.1*	.73	.00	1.0

* p = .05

Post-hoc comparisons (see Table 35) indicated that: (a) the Elevated clinical group was significantly different from the Typical clinical group; (b) the Typical clinical group was significantly different from the Low clinical group and; (c) the Low clinical group was significantly different from the Elevated clinical group. This suggests that the elevated and typical clinical groups had higher levels of anxiety than the low clinical group.

Descriptive statistics for depression per group are presented in Table 36 which shows that the Elevated clinical group have the highest levels of depression (M= 12.5) and that the Low clinical group have the lowest levels of depression (M= 8.7).

Table 36: Descriptive statistics of Depression per eating pathology group

Eating Pathology group			Depression					
	Frequency	Percentage	Mean	Median	SD	Skewness	Kurtosis	Min–Max
Elevated clinical	107	56	12.5	12.0	3.7	.18	-.56	5 – 21
Typical clinical	50	26	10.2	10.0	4.1	.42	-.30	2 – 20
Low clinical	33	17	8.7	7.0	3.6	.52	-.75	3 – 16

In order to examine the differences in depression between the three groups, a one-way ANOVA was carried out with eating pathology as the independent variable and depression as the dependant variable. There were significant mean differences between the three groups (F (2, 187) = 15.6, p <.01, η^2 = .14).

Table 37: Results of the ANOVA exploring mean difference between eating pathology groups in relation to depression

	Mean Difference	Standard Error	Sig.	Cohen's d
Elevated clinical compared to Typical clinical	2.3*	.64	.00	0.8
Typical clinical compared to Low clinical	1.5	.84	.20	0.3
Low clinical compared to Elevated clinical	-3.8*	.75	.00	1.2

* p = .05

Post-hoc comparisons (see Table 37) indicated that: (a) the Elevated clinical group was significantly different from the Typical clinical group and; (b) the Low clinical group was significantly different from the Elevated clinical group. However, the Typical clinical group was not significantly different from the Low clinical group. This suggests that the elevated clinical groups had higher levels of depression than the low clinical group.

4.3.2 Summary of hypothesis testing

For a summary of hypothesis testing, please refer to appendix 19.

4.4 SECTION 4: predicting current levels of cognitive fusion and experiential avoidance

Two standard multiple regression analyses were carried out to explore how well each of the variables predicted current levels of psychological inflexibility within the sample, and how much of the variance could be explained by these variables. A summary of the Pearson's correlations (unless otherwise indicated) between potential predictor and criterion variables are presented in Table 38.

Table 38: Summary of the correlations between potential predictor variables and criterion variables (cognitive fusion and experiential avoidance)

Scale	Cognitive Fusion	Experiential Avoidance
Emotional Processing (N= 190)	.65**	.65**
Thought-Shape-Fusion (N= 190)	.55**	.51**
Maternal Invalidation (N= 186) *(Rho)*	.12	.23**
Paternal Invalidation (N= 156) *(Rho)*	.17*	.24**
Childhood Emotional Abuse (N= 190)	.21**	.27**
Childhood Sexual Abuse (N= 190)	.14	.22**
Anxiety (N= 190)	.57**	.56**
Depression (N= 190)	.24**	.32**
Eating disorder pathology (N= 190)	.45**	.45**

* p = .05 ** p = .01

4.4.1 Cognitive fusion as the criterion variable

The first standard multiple regression revealed that the model was significant and as a whole it explained 58.7% of the variance on the cognitive fusion scale (R^2 = .587, (F (7, 148) = 30.1, p<.05). Table 39 shows that three of the independent variables – emotional processing, thought-shape-fusion and anxiety – made a uniquely statistically significant contribution to the model.

Table 39: Summary of multiple regression analysis for variables predicting cognitive fusion (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Emotional Processing	1.97	.42	.380	4.7	.00	1.1	2.8
Thought-Shape-Fusion	.06	.03	.159	2.2	.03	.01	.12
Paternal Invalidation	-.05	.04	-.085	-1.1	.27	-.13	.04
Childhood Emotional Abuse	.14	.10	.111	1.4	.15	-.05	.34
Anxiety	.54	.14	.273	3.7	.00	.25	.81
Depression	.16	.12	.082	1.3	.19	-.08	.39
Eating disorder pathology	.001	.02	.004	.05	.96	-.03	.04

A second multiple regression was run using only emotional processing, thought-shape-fusion and anxiety. This revealed that the model was significant and as a whole, it

explained 57% of the variance on the cognitive fusion scale (R^2 = .577, (F (3, 186) = 84.4, p<.05). Table 40 shows that these three variables still made a uniquely statistically significant contribution to the model.

Table 40: Results of the second multiple regression analysis for variables predicting cognitive fusion (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Emotional Processing	2.2	.36	.42	6.0	.000	1.5	2.9
Thought-Shape-Fusion	.07	.02	.17	2.9	.005	.02	.12
Anxiety	.56	.13	.29	4.4	.000	.31	.81

4.4.2 Experiential avoidance as the criterion variable

The next stage of the analysis involved a standard multiple regression analysis with experiential avoidance as the criterion variable. This revealed that the model was significant and as a whole it explained 58% of the variance on the experiential avoidance scale (R^2 = .581, (F (9, 143) = 22.3, p<.05). Table 41 shows that three of the independent variables – emotional processing, depression and anxiety – made a uniquely statistically significant contribution to the model.

Table 41: Summary of multiple regression analysis for variables predicting experiential avoidance (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Emotional Processing	1.8	.45	.33	4.0	.00	.93	2.7
Thought-Shape-Fusion	.01	.03	.02	.30	.77	-.05	.07
Maternal Invalidation	-.05	.04	-.09	-1.2	.20	-.14	.03
Paternal Invalidation	.00	.04	.00	.00	.99	-.09	.09
Childhood Emotional Abuse	.23	.12	.17	1.9	.06	-.01	.46
Childhood Sexual Abuse	.06	.07	.05	.86	.39	-.08	.19
Anxiety	.62	.16	.30	3.9	.00	.31	.93
Depression	.37	.13	.18	2.8	.01	.11	.62
Eating disorder pathology	.01	.02	.05	.64	.52	-.02	.05

Given that childhood emotional abuse was so close to .05, it was decided to take this variable forward into the next multiple regression analysis. Therefore, the second analysis was run using emotional processing, depression, anxiety and childhood emotional abuse. This revealed that the model was significant and as a whole, it explained 57% of the variance on the experiential avoidance scale (R^2 = .574, (F (4, 185) = 62.3, p<.05). Table 42 shows that these four variables still made a uniquely statistically significant contribution to the model.

Table 42: Result of second multiple regression analysis for variables predicting experiential avoidance (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Emotional Processing	1.9	.37	.36	5.2	.000	1.2	2.7
Depression	.66	.14	.32	4.9	.000	.39	.93
Anxiety	.34	.11	.18	3.2	.001	.14	.57
Childhood Emotional Abuse	.18	.07	.13	2.7	.008	.05	.31

4.5 Predicting current levels of eating pathology

A final standard multiple regression was carried out to explore how well each of the variables predicted current levels of eating pathology (dependant variable). The model was significant and as a whole it explained 41% of the variance in eating pathology (R^2 = .411, (F (6, 183) = 21.3, p<.05). Table 43 shows that only two of the independent variables made a uniquely statistically significant contribution to the model with the strongest predictors being Thought-Shape Fusion and depression.

Table 43: Summary of multiple regression analysis for variables predicting current eating pathology (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Experiential Avoidance	.29	.38	.08	.77	.44	-.46	1.0
Cognitive Fusion	-.16	.41	-.04	-.39	.69	-.98	.65
Thought-Shape Fusion	.62	.12	.38	5.3	.00	.39	.86
Emotional Processing Scale	3.6	1.9	.17	1.9	.06	-.23	7.5
Anxiety	.35	.66	.04	.53	.59	-.96	1.7
Depression	1.2	.52	.15	2.3	.03	.15	2.2

Given that emotional processing was so close to .05, it was decided to take this variable forward into the next multiple regression analysis. Therefore, the second analysis was run using thought-shape fusion, depression and emotional processing as the independent variables and eating pathology as the dependant variable. This revealed that the model was significant and as a whole, it explained 40% of the variance in eating pathology (R^2 = .408, (F (3, 186) = 42.6, p<.05). Table 44 shows that these three variables made a uniquely statistically significant contribution to the model.

Table 44: Result of second multiple regression analysis for variables predicting current eating pathology (N = 190).

Model	B	SE (B)	β	t	sig	95% *CI* for B	
						Lower	Upper
Thought-Shape Fusion	.63	.11	.39	5.5	.000	.40	.86
Depression	1.3	.50	.17	2.6	.010	.32	2.3
Emotional Processing Scale	4.5	1.5	.21	2.9	.004	1.5	7.5

4.6 SECTION 5: group differences

In section 4.2.2.3, almost half of the sample reported that they were currently receiving treatment for their ED and the other half reported that they were not. It was therefore decided to carry out a series of mean comparisons in order to investigate whether any group differences existed on the main study variables based on treatment status.

4.6.1 Current treatment status

A series of independent samples t-tests were carried out with 'treatment status' as the independent variable and the main variables as the dependant variables. The group means and standard deviations are presented in Table 45.

Table 45: Group means, medians and standard deviations of the main variables by treatment status

		Exp. Avoid.	Cog. Fusion	Thought -Shape Fusion	Emo. Proc.	Mat. Inval.	Pat. Inval.	Anx.	Dep.	Eat. Path.
Curr. Rec. Treatm.	*N*	*97*	*97*	*97*	*97*	*97*	*82*	*97*	*97*	*97*
	M	37.2	39.5	37.1	5.9	36.0	40.9	15.2	11.7	63.8
	SD	6.6	6.4	17.9	1.2	13.7	13.7	3.2	4.0	28.8
Not Rec. Treatm	*N*	*93*	*93*	*93*	*93*	*89*	*74*	*93*	*93*	*93*
	M	34.6	36.5	34.3	5.6	34.6	38.2	14.0	10.8	62.0
	SD	9.2	8.6	20.6	1.7	13.9	15.6	4.5	4.1	33.4

An independent-samples t-test revealed significant group differences in experiential avoidance (t (167) = 2.25, p= .03, two-tailed, d= .24) with those currently receiving treatment exhibiting higher levels of experiential avoidance. Similarly, there were significant group differences in cognitive fusion (t (188) = 2.82, p= .005, two-tailed, d= .52) with those currently receiving treatment exhibiting higher levels of cognitive fusion than those who were not receiving treatment. There were also significant group differences in anxiety (t (166) = 2.19, p= .03, two-tailed, d= .25) with those currently receiving treatment exhibiting higher levels of anxiety than those who were not receiving treatment.

An independent-samples t-test revealed no significant group differences in thought-shape fusion (t (188) = .996, p= .321, two-tailed), Emotional Processing (t (188) = 1.69, p= .09, two-tailed), ED pathology (t (188) = .378, p= .706, two-tailed) or Depression (t (188) = 1.605, p= .110, two-tailed).

Finally, a series of independent-samples t-tests were carried out on all of the childhood abuse subscales which revealed that there were no significant group differences in:

childhood emotional abuse (*t* (188) = .857, p= .393, two-tailed), childhood physical abuse (*t* (188) = .115, p= .908, two-tailed), childhood sexual abuse (*t* (188) = 1.25, p= .213, two-tailed), childhood emotional neglect (*t* (188) = -.545, p= 586, two-tailed) or childhood physical neglect (*t* (188) = -.397, p= .692, two-tailed). There were also no significant group differences in maternal invalidation (*t* (184) = .709, p= .479, two-tailed) or paternal invalidation (*t* (154) = 1.16, p= .247, two-tailed).

5 DISCUSSION

5.1 Overview

The main aim of this study was to test the hypotheses that psychological inflexibility (cognitive fusion and experiential avoidance) would be associated with eating pathology within a clinical population. Other variables such as Thought-Shape Fusion, depression and anxiety were also explored in relation to psychological inflexibility and eating pathology. Furthermore, it examined whether childhood experiences of abuse and emotional invalidation were long-term risk factors for the development of psychological inflexibility and eating pathology. In this study, the Eating Disorder Risk Composite (EDRC) was used to divide the sample into three groups according to the severity of their eating pathology (elevated, typical and low) so that comparisons could be made where appropriate.

A series of scatterplots, ANOVA's and multiple regression analyses were used in order to investigate the multitude of relationships between these variables. This section will begin with an overview of the main findings in relation to the hypotheses and links will be made to previous research and theory. There will be a discussion of the external validity and clinical implications of the study, followed by a consideration to the strengths and limitations of the study along with the conclusions and suggestions for future research.

5.4 Discussion of the results of the hypotheses (main findings)

5.2.1 Eating pathology and psychological inflexibility

It was predicted that psychological inflexibility would be associated with eating pathology and this was supported by the findings of this study as there was a medium correlation (r = .45) with the AAQ-II (a measure of psychological inflexibility and experiential avoidance) and the CFQ (a measure of cognitive fusion). Furthermore, those in the elevated and typical clinical ranges had significantly higher levels of psychological inflexibility than those in the low clinical range.

These results suggest that those with eating pathology have difficulties with flexibility (the opposite of inflexibility) when it comes to managing their internal experiences. For example, as the severity of their eating pathology increases, the level of experiential avoidance also increases which implies that this allows them to escape or avoid unwanted internal experiences. This lends support to the conceptualisation that it is the ED behaviours *themselves* which may serve the function of helping the individual avoid distressing thoughts, feelings and sensations (Heffner et al., 2002; Sandoz, Wilson, & Dufrene, 2011). This is further supported by others such as Heatherton & Baumeister (1991) who highlighted the role of avoidance in those with EDs. This suggests that experiential avoidance, as a process, is important. However, it could be queried whether the mere presence of experiential avoidance within this sample really is indicative of this. Whilst this is a valid point, the picture becomes clearer when one is reminded of the other aspect to psychological inflexibility; cognitive fusion.

As stated in section 2.6.2.1 cognitive fusion refers to the process of an individual reacting to their thoughts as if they are literal truths. This study found a medium correlation (r = .45) with cognitive fusion and eating pathology and a medium correlation (r = .58) with TSF and eating pathology. In addition, those in the elevated clinical range had significantly higher levels of cognitive fusion and TSF than those in the low clinical range and it supports previous findings such as Coelho et al (2008). This suggests that those with more severe eating pathology react to their thoughts more strongly and/or are more distressed by them.

A possible explanation for this stems from Fairburn's (2003) argument that a core feature common to all EDs is an overvaluation of body shape and weight. It is not difficult to imagine that for someone in the throes of an eating pathology, a multitude of thoughts (e.g. "I am fat", "I hate my stomach") may cross their mind and give rise to high levels of negative affect and psychological distress. ACT appears to be well placed to highlight how the underlying processes leading to psychological distress may work. ACT would argue that this distress is indicative of cognitive fusion and TSF because the thoughts themselves are being treated as literal truths (Hayes et al., 1999) and are attached to the conceptualized self (Hayes & Smith, 2005). Therefore, an individual who *truly* believes they are fat will be highly distressed and may attempt to get rid of their unpleasant thoughts and feelings about their body via their ED behaviours (i.e. engaging in starvation in order to achieve a thin ideal). Given that those in the elevated clinical range were found to have significantly higher levels of Cognitive Fusion, TSF and experiential avoidance than those in the low clinical range, lends support to this notion.

If psychological inflexibility is associated with more severe eating pathology and that the reverse (i.e. flexibility) is associated with less eating pathology, then an intervention aimed at increasing psychological flexibility by reducing experiential avoidance and cognitive fusion may be important. This will be discussed later in section 5.8.

5.2.2 Eating pathology and childhood abuse

The rates of childhood abuse and emotional invalidation in this sample were high; over half scored in the moderate to severe range on at least one domain of abuse or neglect. This fits with the existing literature which has also found elevated rates of childhood abuse (Fosse & Holen, 2006; Leonard et al., 2003) and emotional invalidation (Mountford et al., 2007) in this population. Given previous research it was, therefore, expected that reports of abuse and emotional invalidation in childhood would be associated with current levels of eating pathology but no evidence was found in support of this hypothesis as there were no significant correlations. Furthermore, there were no significant differences in the rates of abuse and the severity of eating pathology.

These results appear to suggest that early experiences of abuse and invalidation do not directly impact on current levels of eating pathology. If this is taken at face value, this is

surprising and at odds with the wealth of research showing strong links between early abuse and later psychological distress (Glaser, 2002, 2011). It also fails to support the literature which suggests that childhood emotional abuse has a unique impact on the development of eating pathology (Burns et al., 2012; A. Kent & Waller, 2000). One possible explanation for this finding is that the impact of abuse is not long-lasting. Another possible explanation is that the link between early experiences of abuse and invalidation and current levels of eating pathology may be an *indirect* one. In other words, early abuse may set the *context* for eating pathology and render the individual susceptible; other patterns of managing life experiences may then take precedence and these may then become maintaining factors. One potential variable which may mediate the relationship between childhood abuse and eating pathology is how these earlier aversive experiences are managed, including how psychologically flexible an individual is. Thus, psychological inflexibility will be discussed next (section 5.2.3).

5.2.3 Childhood abuse and psychological inflexibility

It was expected that reports of abuse and emotional invalidation in childhood would be associated with psychological inflexibility and there was some evidence to support this hypothesis.

With regards to psychological inflexibility and experiential avoidance (as measured by the AAQ-II) three correlations were found for sexual abuse, emotional abuse and emotional invalidation. Although the correlations were weak, it is impressive when one considers the distance in time between experiences of childhood abuse and reporting it in this study; it suggests that these early experiences can have a long-lasting impact. This is in line with existing literature documenting the significance of early abuse and emotional invalidation and the impact it has on later emotional functioning (Krause et al., 2003; Linehan, 1993; Mountford et al., 2007). It seems that negative experiences at an early age may result in a decreased ability to manage difficult experiences effectively and flexibly (Hayes et al., 1999; Keville et al., 2008), due to a habitual use of avoidant-based coping strategies (which are rapidly effective in the short-term). This makes sense when one considers that abuse can be perceived as intolerable and life-threatening for the child (Kennerley, 1996).

Another finding of this study was that childhood emotional abuse and paternal emotional invalidation were the only variables which were associated with cognitive fusion (as measured by the CFQ) in this sample. This seems to suggest that father figures are more likely to be perceived as emotionally abusive. However, some methodological considerations are worth mentioning when interpreting this finding. The ICES makes the assumption that an individual grew up with heterosexual parents as it asks about mother and father figures only. Some of the sample may have grown up in either single parent or same-sex parent families which meant that they could not accurately report their experiences. This explanation is supported by the fact that this study found that N=9 of the sample were not raised by their parents at all. Another possible explanation is due to culturally prescribed gender roles. 90% of the sample was white and were from the UK. Typically, western cultures share the stereotypical belief that women are more emotionally expressive than men (Brody & Hall, 2008). Therefore, it is possible that the fathers in this particular sample were less emotionally expressive or poorer at communicating emotions, hence being perceived as more emotionally invalidating by the individual. However, further research is needed to explore this and to perhaps unpick why paternal (and not maternal) invalidation was associated with cognitive fusion.

5.3 Discussion of additional findings

5.3.1 Eating pathology, psychological inflexibility, depression and anxiety

The rates of anxiety and depression in this sample were high with over half experiencing clinical levels of anxiety and depression. The hypothesis that eating pathology would be associated with anxiety and/or depression was supported and there was a stronger relationship with anxiety (r = .32) than depression (r = .29). A mean difference was also found between the severities of eating pathology: those with more severe levels of eating pathology had higher levels of anxiety and/or depression. This is in line with the extant literature which has highlighted that anxiety and/or depression are common comorbid disorders with eating pathology (Bulik 2002; Pallister & Waller, 2008). However, no causal links can be made as it is unclear whether anxiety and/or depression are risk factors for eating pathology or are secondary to eating pathology. This will be discussed further in section 5.4.2.

This study also found that higher levels of psychological inflexibility were associated with higher levels of anxiety and depression. This suggests that the sample were currently experiencing high levels of psychological distress *and* that those with the most severe eating pathology were experiencing the highest levels of psychological inflexibility. This links to section 5.2.1 and lends support to the ACT conceptualisation that the ED behaviours themselves may serve the function of helping the individual avoid distressing thoughts, feelings and sensations (Heffner et al., 2002; Sandoz, Wilson, & Dufrene, 2011).

Despite this association, no causal links can be made as it is unclear what is happening within these relationships. Psychological flexibility relates to how experiences are viewed and/or managed, and here, it is unclear how anxiety and/or depression fit into this picture. This will be discussed further in section 5.4.1, but it is worth mentioning that the relationships are likely to be complex and bidirectional.

5.4 Discussion of the multiple regression analyses

5.4.1 Predicting current psychological inflexibility

Based on the simple correlations, it was expected that childhood emotional abuse and invalidation would significantly predict current levels of psychological inflexibility. The findings will now be discussed in relation to each process of the ACT model of pathology.

5.4.1.1 Experiential avoidance

The findings showed that childhood emotional abuse, emotional processing, depression and anxiety made significant and unique contributions to a regression model that explained 57% of the variance in experiential avoidance. The hypothesis that childhood emotional abuse would predict psychological inflexibility in those with eating pathology was supported and adds to the literature which highlights the impact of early experiences on the way an individual learns to tolerate and manages their internal experiences (Haslam et al., 2008). It also was anticipated that emotional invalidation would emerge as a significant predictor of current levels of psychological inflexibility but this was not

the case. One possible explanation for this is that the weak association between maternal and paternal invalidation (as measured by the ICES) and experiential avoidance was diminished to the point of insignificance when controlled for by the other predictors of the multiple regression analyses.

Another finding of this study was that anxiety and depression predicted experiential avoidance, with anxiety being a stronger predictor than depression. This could be due to anxiety being a more immediate emotional response due to the neurological and physiological context around it i.e. perceived threats trigger a fight/flight response. With this in mind, anxiety could be experienced as a more powerful and distressing emotion by the individual, thus they may be more likely to utilise experiential avoidance in order to manage it. Another possibility is that the relationship between anxiety and/or depression and experiential avoidance may be more complex, i.e. some contextual factors may serve as a mediator or moderator variable. For example, if an individual already uses an inflexible way of coping due to their aversive childhood experiences, then their anxiety and/or depression levels may be exacerbated because ACT argues that psychological inflexibility increases psychological distress (Hayes et al., 1999). This may be a partial explanation because this study observed a small correlation between childhood emotional abuse and psychological inflexibility.

Thus far, these findings appear to fit with Keville et al's (2008) conceptualisation (figure 3) of the importance of flexibility with regards to managing internal experiences. Childhood emotional abuse and invalidation may trigger a range of painful private events for the individual, some of which may include feelings of anxiety and depression amongst other intolerable emotions. When the individual comes into contact with these painful private events, they may use a primary and immediate avoidant-based coping strategy such as experiential avoidance. However, this is likely to exacerbate and maintain their emotional distress (Keville et al., 2008). Instead of flexibly altering their coping strategy, the individual continues to use avoidance. This, arguably, gives rise to secondary ways of managing distress, e.g. via their eating disorder behaviours, in order to try and reduce the unpleasantness of the experience (Hayes & Pankey, 2002). Indeed, if one refocuses attention onto weight, shape and eating, then there is little space to consider feelings. This is supported by some literature which has found that EDs do

serve to deal with continuing emotional problems because the eating behaviours themselves help an individual re-gain some emotional control (Polivy & Herman, 2002). However, experiential avoidance can increase psychological distress because it may prevent the individual from experiencing and processing (a) the abusive experience (which is often central to recovery) (Schauer, Neuner, & Elbert, 2005) and (b) the unpleasant private events. This conceptualisation is supported by another finding of the multiple regression analysis: emotional processing also emerged as a strong predictor of psychological inflexibility. This particular finding implies that someone who is unable to accept and work-through their aversive experiences may avoid them instead. It is interesting to note that in this study, those currently in treatment had higher levels of experiential avoidance. There is a suggestion here that the potential for re-experiencing and processing experiences may be greater within therapeutic environments, and thus the need to manage this via experiential avoidance may be greater. Conversely, those not within treatment may have processed their distress and may be managing this more effectively, more flexibly.

5.4.1.2 Cognitive Fusion

The findings showed that emotional processing, TSF and anxiety made significant and unique contributions to a regression model that explained 57% of the variance in cognitive fusion. The hypothesis that childhood emotional abuse and invalidation would predict cognitive fusion was not supported as childhood emotional abuse and invalidation did not emerge as significant predictors. Although this was surprising, perhaps childhood abuse and invalidation has less of an impact on this particular *process* of psychological inflexibility in those with eating pathology. This is a feasible explanation given the findings of the previous section (5.4.1.1). Abuse, particularly in early childhood, may induce more intolerable *feelings* (as opposed to cognitions) in the individual, thus, experiential avoidance could serve as a more effective escape in the familial context. It is also plausible that the abuse occurred in a pre-verbal context, i.e. prior to the development of language.

Another finding was that TSF emerged as a strong predictor of cognitive-fusion and this was not surprising given that it is arguably measuring the same process. Further, it is

likely to be an issue specific to ED. Indeed the CFQ and TSF were highly correlated (r = .55) and this could be considered as a criticism because the only difference between the two is that 'pure' cognitive fusion is broad (e.g. 'I feel upset when I have negative thoughts about myself') whereas Thought-Shape-Fusion is specific to food and shape (e.g. 'I feel huge if I imagine not exercising for a month'). Despite this, the finding does highlight that those who display inflexibility in terms of body and food-specific internal experiences, may also display inflexibility in terms of broader thoughts and feelings. For example, the individual may initially fuse with 'I'm fat,' then fuse with a non-body experience such as 'I'm a failure". This is likely to be a distressing experience for the individual and this is supported by the finding that anxiety was a predictor of cognitive-fusion: the more someone fuses with their thoughts, the greater their anxiety levels and vice versa.

If the individual remains inflexible because they consider their thoughts to be true (Hayes et al., 2001) then they may experience psychological distress and be unable to process their emotional experience. This is a potential explanation as this study found that Emotional processing also emerged as a strong predictor of cognitive fusion. This is interesting as within the scale there is a strong experiential avoidance component, and this may be about an unwillingness to connect with emotional experiences (Keville et al., 2008). However, further research would be needed to explore how emotional processing impacts on psychological inflexibility as the relationship between the two is not entirely clear. For example, does the experience of emotion and consequential emotional processing mediate the relationship between childhood abuse and psychological inflexibility? Or is it an entirely different phenomenon altogether? This could be a focus of future research.

5.4.2 Predicting current eating pathology

Based on the simple correlations, it was expected that psychological inflexibility (as measured by the AAQ-II and CFQ) would significantly predict current eating pathology; but no evidence was found in support of this hypothesis as experiential avoidance and cognitive fusion did not emerge as significant predictors. This was a surprise and one possible explanation is that there were methodological limitations in terms of the questionnaires used. The AAQ-II is very short and some of the questions appear to have

debatable relevance to the construct of psychological inflexibility. For example, the statement 'worries get in the way of my successes' could be considered as rather vague and it is unclear which process of psychological inflexibility is being measured.

Another possibility is that the AAQ-II was not a good measure of experiential avoidance in those with eating pathology. This is a plausible explanation because the EPS-25 (a measure of emotional processing) has a strong experiential avoidance component, and it was one of the strongest unique predictors of current levels of eating pathology. The EPS-25 suggests that the way in which an individual currently manages their emotions *is* important and that the poorer someone is at managing their emotions, the more likely they may be to develop some form of eating pathology later on. When one refers to section 4.2, the sample were in the clinical range for emotional processing and exhibited high levels of psychological inflexibility which implies that those with eating pathology do have some degree of difficulty in managing their internal experiences. In addition, this difficulty appears to be associated with the severity of eating pathology as there were mean differences amongst the three groups with the most severe eating pathology (i.e. elevated clinical range) being associated with poorest emotional processing and highest levels of psychological inflexibility. Therefore, the EPS-25 may be a better measure of experiential avoidance. However, further research would be needed to investigate this.

Another explanation is that there is multicollinearity in the model. Experiential avoidance may no longer make a unique and significant contribution to the prediction of eating pathology when controlled for by emotional processing in the multiple regression analysis. This is a plausible explanation because the AAQ-II and the EPS-25 both have a strong avoidance component. In addition, they were significantly related to eating pathology on their own.

A final explanation is that there may be additional variables which have not been captured or accounted for in this study as 60% of the variance in eating pathology remains unexplained.

Despite psychological inflexibility failing to emerge as a predictor of eating pathology, Thought-Shape Fusion did emerge as the strongest unique and significant predictor

which suggests that an inflexible stance towards managing ones internal experiences *in relation to body shape and weight* has an impact on current levels of eating pathology. This is in line with the existing literature which has documented the importance of psychological inflexibility in EDs (Hill, Masuda & Latzman, 2013) and the over evaluation of shape and weight within EDs (Fairburn et al, 2003).

5.5 Findings from group comparison data

This study also explored whether any mean differences existed on any of the main study variables based on treatment status. This was explored because despite the sample being a clinical population, almost half were not receiving treatment which could have implications for clinical practice. However, these findings need to be interpreted cautiously as the results below do not differentiate between the treatments (i.e. psychological input or medication), the *nature* of treatment (i.e. type of psychological therapy or medication) received or, whether the sample were *previously* in treatment, due to limitations with the study design.

5.5.1 Treatment status

Small to medium mean differences existed between the two groups with those currently receiving treatment scoring significantly higher on measures of anxiety and psychological inflexibility than those who were not currently in treatment. There was a small effect size for experiential avoidance and a medium effect size for cognitive fusion. At first, this seems paradoxical as one would expect to see lower scores for those in treatment. However, as discussed in section 5.4.1, it is possible that being in treatment brings a range of issues to the forefront which may result in people engaging in more avoidance or cognitive-fusion in order to manage them. It is also possible that those in treatment happen to be those who had more functional impairments than those not in treatment. However, this would merely be speculation as this study did not measure functional impairment in this sample. A final explanation is that the non-treatment group may have received treatment in the past and this impacted on the results. This fits with some scant literature which has found that negative affect such as anxiety can be lower depending on what stage of recovery the individual is at (Harney, Fitzsimmons-Craft,

Maldonado, & Bardone-Cone, 2014). Again, this is speculation as this study did not capture any information on historical treatment.

5.6 External validity and generalisability of the findings

The sample used in this study was predominantly female (91%), white (90%) and aged between 20 and 29 (55%). This may indicate a particular bias in the sample and reflect people who are more likely to access the internet (i.e. web-based forums and charities) for information or support related to their ED. It is also possible that it reflects those who are more motivated to access help as those with eating pathology are typically a difficult to engage group (Jurascio et al., in press). This means that the external validity of this study may be compromised as the findings can not necessarily be generalised to the wider population, particularly with regards to males and those of other ages and ethnic and cultural backgrounds.

This study aimed to target a clinical population, i.e. they were currently experiencing clinical or sub-clinical EDs. The results from the relevant scales confirm that this was achieved as 100% of the sample used within this analysis reported that they had been diagnosed with either AN-R, AN-BP, BN or EDNOS. On the EDRC, 83% of participants were identified as being in the typical or elevated clinical range which is indicative of a clinical ED. On the HADS, 83% of the sample was in the clinical range for anxiety and 56% were in the clinical range for depression. The clinical status of the sample was further supported by their maternal (M= 35.3) and paternal invalidation (M= 39.6) scores as they exceeded the norms found in an ED sample (M= 31.7 & 34.7) and a non-clinical sample (M= 28.4 & 27.8) in Mountford et al's (2007) study. Finally, the sample's cognitive fusion (M= 38.0) scores exceeded the norms found in a mixed mental health sample (M= 34) (Gillanders et al., in press), but their TSF scores were slightly lower than the clinical sample in Coelho et al's (2012) study. There are no norms available for experiential avoidance but given that the highest possible score on the AAQ-II is 49, the sample's score does appear to be comparably high (M= 36.0).

5.7 Clinical implications

5.7.1 Assessment and formulation

The findings of this study have important implications for the conceptualisation of psychological distress and eating pathology. It seems that this population has high rates of childhood abuse and invalidation, with emotional abuse being especially prevalent. In clinical work, it may be important to routinely include questions about abusive experiences as part of the assessment process so these issues can be brought to light. In addition, these issues can be included as part of the formulation process so that the client's current difficulties can be understood as part of a broader context. This is of particular importance when one considers that childhood emotional abuse and emotional processing were predictors of psychological inflexibility. A childhood which induces intolerable emotions can, for some individuals, lead to a rigid and inflexible way of coping with intolerable experiences. This may well impact on the therapeutic relationship if the individual has difficulties with relating to a therapist who, by the very nature of the therapeutic work, may trigger intolerable emotions and subsequently inducing such emotions during therapy.

Although psychological inflexibility (i.e. experiential avoidance and cognitive fusion) did not predict current levels of eating pathology in this study, the fact that it was present (medium correlation) suggests that there is something important about the way in which those with eating pathology manage their psychological distress. This is further supported by poor emotional processing being predictive of current eating pathology. This could also be a focus of assessment and formulation because it could then inform treatment, for example, to be explicitly highlighted as an issue that may be a) maintaining distress, and b) maintaining a reluctance to engage in therapy.

5.7.2 Intervention

The findings of this study imply that a transdiagnostic approach could be important in the treatment of those with eating pathology. Rather than focusing on the ED symptoms associated with a particular diagnostic label (i.e. AN, BN, EDNOS) or the comorbid symptoms (i.e. anxiety or depression), the underlying processes can be targeted instead. This is a similar stance to Fairburn et al's (2003) transdiagnostic model as they also

emphasise four key maintaining processes across disorders rather than specifying different treatments for different disorders. However, a unique contribution of this study is that it has highlighted some potentially different maintaining processes which may drive eating pathology i.e. emotional processing and psychological inflexibility. A clinical intervention which targets the underlying language process of thought-shape-fusion and fosters a willingness to experience discomfort (in order to promote flexibility) could be promising as it may help those with more severe eating pathology increase their ability to tolerate the distress caused by treatment i.e. weight gain or connecting with and processing emotional experiences. Furthermore, this could reduce relapse rates.

One type of therapy which has been designed for this very purpose is ACT (Hayes et al., 1999). ACT is an intervention that fosters cognitive *de*fusion (to target fusion), *acceptance* (to target experiential avoidance) and four other processes (see figure 2 in section 2.6.2.4) in order to increase psychological flexibility and reduce psychological suffering. It is experientially orientated and uses a variety of metaphors, stories, and role-plays. With regards to defusion, the individual is encouraged to change the way that they relate to their thoughts in order to reduce the literal meaning of their inner experiences; thoughts are just thoughts, feelings are just feelings and bodily sensations are just bodily sensations (Hayes et al., 1999; Twohig, 2012). This is then thought to reduce the symbolic impact of thoughts, with the individual learning to 'step back' from their thoughts and look *at* them rather than *from* them. With time, they would come to realise that their thoughts do not necessitate specific behaviours (Baer, 2006) such as bingeing and starvation. Examples of cognitive defusion techniques include labelling the process of thinking (e.g. *"I am having the thought that I am fat", "I am having the feeling of anxiety"*) or repeatedly stating the negative thought aloud in a musical tone of voice (Harris, 2008). Other techniques include a 'leaves on the stream' exercise where the individual is asked to simply notice the thoughts that come into their mind, put each one on a leaf and watch that leaf float down the stream without reacting to it (Hayes & Smith, 2005).

Other interventions in ACT aim to foster acceptance (rather than avoidance). However, Hayes et al. (1999) argues that acceptance is different to 'tolerance' because it's viewed as a *choice* and involves a more welcoming stance towards the inner experience. Rather

than engaging in inner dialogue about how awful they may feel when/after bingeing and whether they should purge, the individual would be encouraged to simply observe their thoughts and feelings without acting upon them (i.e. purging, criticising self for having such thoughts) which could increase their willingness to experience discomfort (Hayes et al., 2006). The notion of choice is important because ACT emphasises how the individual can choose how they respond to their thoughts and internal experiences (rather than being dictated by their eating pathology). As well as encouraging acceptance, the individual is also encouraged to identify what they value in life and to take steps to act in accordance with them (Hayes & Smith, 2005). In other words, the individual's desire for thinness could be replaced with healthier values. This could be important as values may help an individual begin to face life with those values that are important to them without using their ED behaviours.

The findings from the mean differences data which showed that those with more severe eating pathology had the poorest levels of emotional processing highlights how an intervention which targets emotions and/or management of internal experiences could be important. Again, ACT could be well suited for this as the techniques could help the individual become more aware of, and begin to accept, their emotionally provocative thoughts and feelings (Kater, 2010). If acceptance fosters an ability to actually experience a range of emotions then arguably this could also aid emotional processing as the individual would no longer be avoiding their negative affect. This is supported by the trauma literature which argues that exposure to distressing content is key to recovery (Schauer et al., 2005).

Although CBT is currently the treatment of choice in the UK and is effective in 60% of cases (NICE, 2010; Fairburn et al, 2008), there is room for improvement because there are still a number of people who do not recover following treatment (Treasure, 2012; Fairburn et al., 2000). ACT can potentially fill this gap as it focuses on altering the underlying cognitive processes (Hayes et al., 1999) instead of the content of thoughts. Indeed, whilst the evidence base for ACT and EDs is still in its infancy, the initial findings have been promising (Berman et al., 2009; Heffner et al., 2002; Merwin et al.,

2011; Merwin et al., 2013). However, these studies have focused almost exclusively on AN.

It has been suggested that ACT may benefit those with more severe eating pathology as the ego-syntonic nature of their ED means that many of them are ambivalent towards treatment (Jurascio et al., in press). Rather than challenging the content of their thoughts, ACT can offer a new and less threatening way intervene with an individual who may be consumed and restricted by, their thoughts (Kater, 2010). As stated earlier, increasing psychological flexibility may help to decrease fusion. Finally, when one considers the finding that these individuals may come from emotionally abusive and invalidating backgrounds, ACT could be perceived as a kinder and perhaps more validating intervention due to its non-pathological and non-blaming stance (Biglan, Hayes, & Pistorello, 2008). In turn, this could aid engagement and a more compassion based focus.

5.7.3 Prevention

The findings from the mean differences data showed that those with the lowest eating pathology (i.e. low clinical range) had better emotional processing and the lowest levels of psychological inflexibility, depression and anxiety. This has clinical implications with regards to prevention as all of these variables worsen as the severity of eating pathology increases.

Preventative interventions which teach cognitive defusion and acceptance might be effective because the individual would be given strategies to manage their internal experiences *flexibly*. Indeed, if those who exhibit subclinical levels of eating pathology become more open to their experiences (due to a preventative intervention), then they may be more likely to manage adaptively. Examples of adaptive coping may include the processing of their thoughts, feelings and emotions and 'letting go' of their distressing thoughts. In turn, this may reduce the likelihood of them resorting to increasingly severe disordered eating behaviours in order to manage their internal experiences.

A unique contribution of ACT is that it differs from traditional coping methods (such as problem solving or distraction) because it focuses on a *willingness* to experience private events rather than regulating or controlling private events (Hayes et al., 1999). Indeed, if

one is not experiencing something, then one cannot process it. The evidence for ACT as a preventative intervention is limited, especially for those with eating pathology. However, Pearson et al. (2012) found that a one-day ACT workshop led to reductions in body-related anxiety and increases in acceptance for those with 'disordered eating attitudes'.

5.8 Strengths and limitations

There were many strengths of this study. First, the large sample size resulted in adequate power to detect relationships between the variables. Second, this study was successful in recruiting those with clinical EDs which is in contrast to other studies which have focused on non-clinical or student populations. This may have been aided by the online survey design as it meant that it was easier for people to take part. However, a limitation of this design was that only those participants who wanted to take part did so. Therefore, given the high number of participants that dropped out, the results of this study may be a reflection of a self-selection bias; perhaps those who took part completing the survey tended to be those who were most motivated or were experiencing the most psychological distress. Indeed, this self-selected opportunity sampling does raise issues regarding external validity because as mentioned in section 5.7, there was an over-representation of young, white, female respondents. The study could have been improved by the inclusion of older people and those from a more diverse ethnic and cultural background. Perhaps future research could focus more specifically on these issues within an ED population.

A further strength of this study was that it looked at different forms of abuse within an ED population. CSA has been extensively studied with other forms of abuse, such as CEA, receiving less attention. Indeed this study highlighted how emotional abuse and neglect were the most prevalent forms of abuse and adds to the emerging literature which is exploring whether CEA has a unique impact on eating pathology (Burns et al., 2012). However, it is accepted that there may well have been overlaps between the different domains of abuse and that it is difficult to disentangle one form from another.

One limitation relates to the accuracy of the information gathered as this could have been compromised by factors such as retrospective bias, distorted memory or social desirability (Hardt & Rutter, 2004). Although the participants were classified as a clinical population in terms of the measures used, one cannot be 100% certain that they truly had clinical or subclinical EDs because the study was dependent upon self-report. Future research could use a more rigorously identified and selected clinical sample and use a comparative group.

Another limitation of this study was its cross-section design; it only offers a snap-shot of a sample at a particular point in time. Therefore, the findings cannot offer a long-term perspective or conclude on casual or maintaining factors. Future research could use a longitudinal study design so that the relationships between the variables and the possible direction of causality can be tested more reliably.

With regards to the measures used, there was a limitation with the AAQ-II which was used as a measure of experiential avoidance as it has been described as a measure of experiential avoidance *and* psychological inflexibility (Bond et al., 2011). This implies that the AAQ-II is a measure of several ACT processes, not just experiential avoidance. Given that it is difficult to ascertain which question within the AAQ-II is measuring which process; further evaluation of this measure may be warranted. This is particularly important when one considers that it was very highly correlated with the CFQ in this study ($r = .78$) which specifically measures one process of inflexibility. The high correlation means that it is difficult to disentangle the two from each other.

5.9 Conclusion and suggestion for future research

In sum, the current study found that psychological inflexibility (from an ACT perspective) was significantly higher in the elevated clinical range group (i.e. most severe eating pathology) compared to those in the low clinical range groups (i.e. lowest eating pathology). In addition, those in the elevated clinical range for eating pathology had the poorest levels of emotional processing and the highest levels of anxiety and depression. However, there were no significant differences between the groups in terms of childhood abuse and invalidation which suggests that there may be other variables that

have not been accounted for in this study. Indeed, it is acknowledged that there are likely to be many different variables which combine over time and trigger eating pathology.

The current study also found that childhood emotional abuse, emotional processing, depression and anxiety predicted experiential avoidance (57% of the variance) and that emotional processing, thought-shape fusion and anxiety predicted cognitive fusion (57% of the variance). With regards to predicting current levels of eating pathology, emotional processing, thought-shape fusion and depression were the only predictors (40% of the variance). Overall, this was impressive and contributes towards the understanding of how contextual factors may be related to or implicated in the development of psychological inflexibility and eating pathology. The results do provide some support for the ACT model and the study made a unique contribution in that it added to literature on EDs and ACT as this research is still in its infancy. However, further research is clearly needed in order to determine whether ACT as a whole is a viable and effective treatment for a clinical and subclinical ED population. More specifically: longitudinal studies are needed to prospectively track trajectories between relevant predictive variables; More rigorous studies are needed to assess whether ACT is a viable intervention for EDs by evaluating it in comparison to other approaches and; further clarification regarding the two constructs of cognitive fusion and experiential avoidance and how they are measured would be useful and advance research in this area. It may also be useful for future research to look more closely at emotional processing and its relationship to inflexibility.

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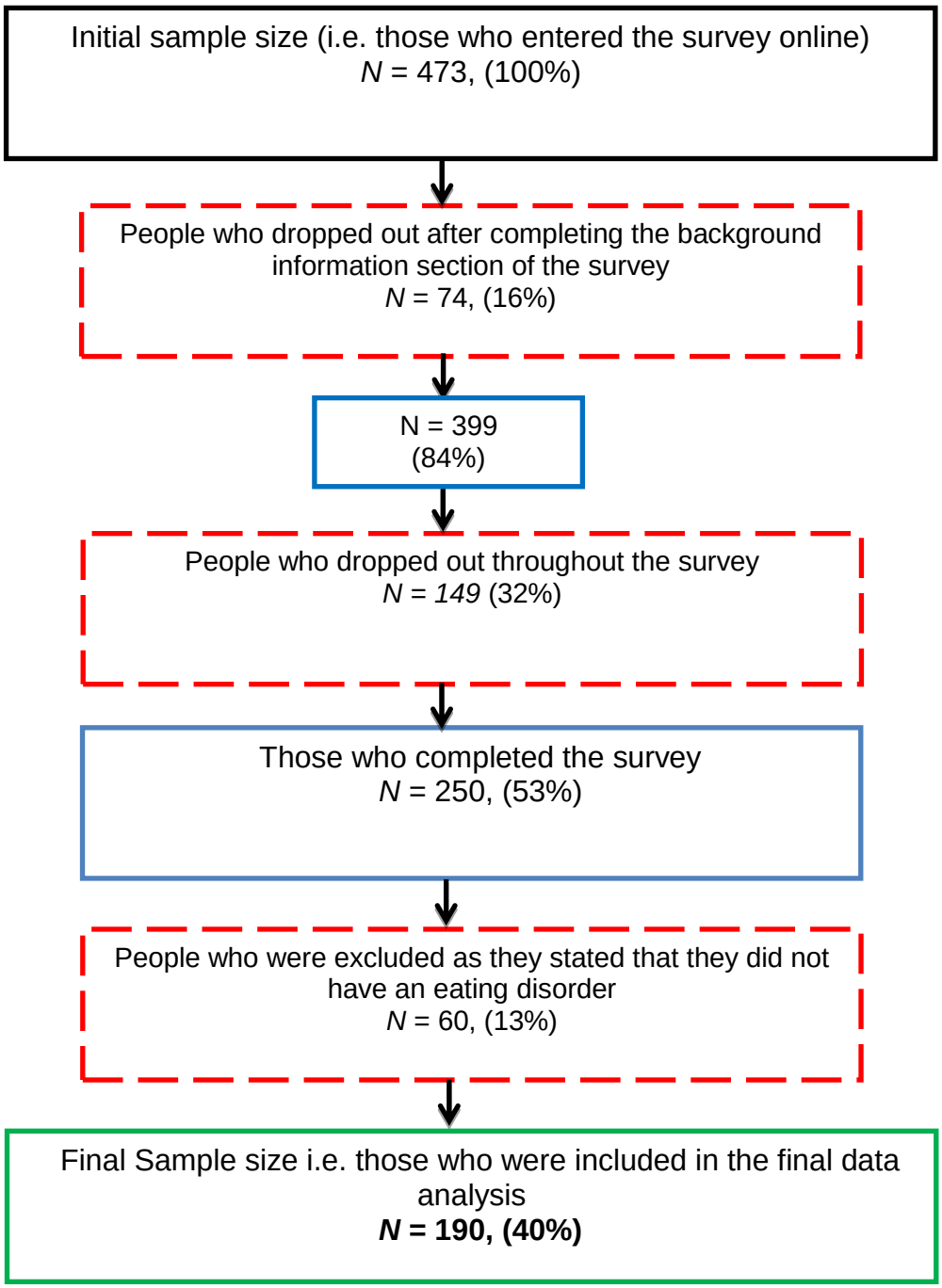
7 APPENDICES

Appendix 1: Flow chart of recruitment



Appendix 2: Background information

Background information

Please answer all of this information on this questionnaire.

1. What is your age? _____ years

2. What is your gender?
   a) Male
   b) Female

3. How would you describe your ethnicity?

   a) White

   British

   Irish

   Any other White background

   b) Mixed

   White and Black Caribbean

   White and Black African

   White and Asian

   Any other mixed background

   c) Asian or Asian British

   Indian

   Pakistani

   Bangladeshi

   Any other Asian background

   d) Black or Black British

   Caribbean

   African

   Any other Black background

   e) Other Ethnic Groups

   Chinese

Any other ethnic group

f) **Other** (please specify)_____

4. What is your current employment status?

a) Full-time
b) Part-time
c) Unemployed
d) Looking for work
e) Student
f) Other

5. What is your relationship status?

Single
In a long-term relationship
In a new relationship
Married/civil partnership
Cohabiting
Widowed

6. Have you been diagnosed with a mental health problem?

a) Yes (please specify your diagnosis_____)
b) No

Appendix 3: The Eating Disorders Inventory (EDI-3)

1. What is your current weight? _____ pounds

2. What is your height? _____ feet _____ inches

3. What has been your highest weight in the past (as a young person or adult) (not including pregnancy) _____ pounds
   I. How long ago did you reach this weight? _____ months
   II. How long did you weigh this weight? _____ months

4. What has been your lowest weight as a young person or adult? _____ pounds
   a) How long ago did you reach this weight? _____ months
   b) How long did you weigh this weight? _____ months

5. What weight have you been at for the longest period of time? _____ pounds
   a) At what age did you first reach this weight?

6. Have you ever been diagnosed with an eating disorder? _____ yes _____ no
   a) If yes, what were you diagnosed with?
      i. Anorexia nervosa
      ii. Bulimia nervosa
      iii. Binge-eating disorder
      iv. Other (please specify) _____
   b) When were you diagnosed? _____ (dd/mm/yy)

7. Are you currently receiving any treatment for your eating disorder? _____ yes _____ no
   a) If yes, what treatment are you receiving? _____

Instructions

The items ask about your attitudes, feelings and behaviours. Some of the items relate to food or eating; other items ask about your feelings about yourself. For each item, decide if an item is true about you **Always (A), Usually (U), Often (O), Sometimes (S), Rarely (R)** or **Never (N).**

A	U	O	S	R	N
Always	**Usually**	**Often**	**Sometimes**	**Rarely**	**Never**

1. I eat sweets and carbohydrates without feeling nervous
2. I think my stomach is too big
3. I eat when I am upset
4. I stuff myself with food
5. I think about dieting
6. I think my thighs are too large
7. I feel extremely guilty after overeating
8. I think my stomach is just the right size
9. I am terrified of gaining weight
10. I feel satisfied with the shape of my body
11. I exaggerate or magnify the importance of weight
12. I have gone on eating binges where I felt that I could not stop
13. I like the shape of my buttocks
14. I am preoccupied with the desire to be thinner
15. I think about bingeing (overeating)
16. I think my hips are too big
17. I eat moderately in front of others and stuff myself when they are gone
18. I feel bloated after eating a normal meal
19. If I gain a pound, I worry that I will keep gaining
20. I have the thought of trying to vomit to lose weight
21. I think that my thighs are just the right size
22. I think my buttocks are too large
23. I eat or drink in secrecy
24. I think my hips are just the right size
25. When I am upset, I worry that I will start eating

Appendix 4: Thought Shape Fusion Scale (TSF)

Below you will find a list of statements. Please rate how true each statement is for you by circling a number next to it. Use the scale below to make your choice.

1	2	3	4	5
Not at all	**Slightly**	**Moderately**	**Very Much**	**Totally / Always**

1. I feel fatter after thinking about eating fattening / “forbidden” foods (e.g. chocolate).
2. If I think about gaining weight, I want to check that my clothes are fitting more tightly.
3. Thinking about gaining weight is almost as immoral to me as actually gaining weight.
4. Just picturing myself gaining weight can really make me gain weight.
5. I feel huge if I imagine not exercising for a month.
6. Just thinking about “pigging out” makes me want to weigh myself.
7. Thinking about breaking my diet makes me want to check in the mirror that I don’t look any fatter.
8. Just thinking about not exercising can change the way I really look.
9. I feel fatter if I just think about “pigging out”.
10. Just thinking about not exercising for a month makes me want to cut down on what I eat.
11. If I think about breaking my diet, it is almost as unacceptable as really breaking my diet.
12. My shape can actually change, just by me planning to eat fattening food.
13. I feel fatter just by thinking about gaining weight.
14. Picturing myself eating fattening/”forbidden” foods (e.g. chocolate) makes me want to check my body to make sure I haven’t gained any weight.
15. How often do you have thoughts about the effects of eating fattening/”forbidden” foods?
16. Generally, to what extent do thoughts about “forbidden” foods affect you, or interfere with your daily life?
17. When you have thoughts about “forbidden” foods, to what extent is it *important* for you to get them out your mind?
18. When you have thoughts about “forbidden” foods, to what extent is it *difficult* to get them out of your mind?

Appendix 5: Cognitive Fusion Questionnaire (CFQ)

Below you will find a list of statements. Please rate how true each statement is for you by circling a number next to it. Use the scale below to make your choice.

1	2	3	4	5	6	7
Never true	**Very seldom true**	**Seldom true**	**Sometimes true**	**Frequently true**	**Almost always true**	**Always true**

1. My thoughts cause me distress or emotional pain.
2. I tell myself that I shouldn't be thinking the way I am thinking.
3. Even when I am having distressing thoughts, I know that they may become less important eventually.
4. I find myself preoccupied with the future or the past.
5. I make judgements about whether my thoughts are good or bad.
6. Even when I am having upsetting thoughts, I can see that those thoughts may not be literally true.
7. I get upset with myself for having certain thoughts.
8. I feel like my thoughts need to change before I can have a good life.
9. I find it easy to view my thoughts from a different perspective.
10. I tend to get very entangled in my thoughts.
11. I think some of my thoughts are bad or inappropriate.
12. I feel upset when I have negative thoughts about myself.
13. I get very focussed on distressing thoughts.
14. It's such a struggle to let go of upsetting thoughts even when I know that letting go would be helpful.
15. My thoughts distract me from what I am actually doing.
16. I get so caught up in my thoughts that I am unable to do the things that I most want to do.
17. I over-analyse situations to the point where it's unhelpful to me.
18. I can watch my thoughts from a distance without getting caught up in them.
19. It's OK to have inconsistent thoughts on the same subject.
20. It's possible for me to have negative thoughts about myself and still know that I am an OK person.
21. I am able to do what's important in life even when I have upsetting thoughts.
22. I struggle with my thoughts.

23. I can do difficult things even if my thoughts say they are impossible to do.

24. I can be aware of my thoughts without necessarily reacting to them.

25. Once I've thought about something upsetting it's difficult for me to focus on anything else.

26. I need to control the thoughts that come into my head.

27. I tend to react very strongly to my thoughts.

28. I get so caught up in my thoughts that I forget what I'm actually doing.

Appendix 6: Acceptance and Action Questionnaire II (AAQ-II)

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following scale to make your choice.

1	2	3	4	5	6	7
Never true	Very seldom true	Seldom true	Sometimes true	Frequently true	Almost always true	Always true

1. My painful experiences and memories make it difficult for me to live a life that I would value
2. I'm afraid of my feelings
3. I worry about not being able to control my worries and feelings
4. My painful memories prevent me from having a fulfilling life
5. Emotions cause problems in my life
6. It seems like most people are handling their lives better than I am
7. Worries get in the way of my successes

Appendix 7: Childhood Trauma Questionnaire (CTQ)

These questions ask about some of your experiences growing up as a child and a teenager. Although some of these questions are of a personal nature, please try to answer as honestly as you can. For each question, write the number that best describes how you feel.

1	2	3	4	5
Never true	**Rarely true**	**Sometimes true**	**Often true**	**Very often true**

When I was growing up:

1. I didn't have enough to eat

2. I knew that there was someone to take care of me and protect me

3. People in my family called me things like "stupid", "lazy" or "ugly"

4. My parent/carers were too drunk or high to take care of the family

5. There was someone in my family who helped me feel that I was important or special

6. I had to wear dirty clothes

7. I felt loved

8. I thought that my parent/carers wished I had never been born

9. I got hit so hard by someone in my family that I had to see a doctor or go to the hospital

10. There was nothing I wanted to change about my family

11. People in my family hit me so hard that it left me with bruises or marks

12. I was punished with a belt, a board, a cord or some other hard object

13. People in my family looked out for each other

14. People in my family said hurtful or insulting things to me

15.I believe that I was physically abused

16. I had the perfect childhood

17. I got hit or beaten so badly that it was noticed by someone like a teacher, neighbour or doctor

18. I felt that someone in my family hated me

19. People in my family felt close to each other

20. Someone tried to touch me in a sexual way, or tried to make me touch them

21. Someone threatened to hurt me or tell lies about me unless I did something sexual with them

22. I had the best family in the world

23. Someone tried to make me do sexual things or watch sexual things

24. Someone molested me

25. I believe I was emotionally abused

26. There was someone to take me to the doctor if I needed it

27. I believe that I was sexually abused

28. My family was a source of strength and support.

Appendix 8: Invalidating Childhood Environments Scale (ICES)

The following questions address your experiences of how your parent(s)/carer(s) responded to your emotions when you were young. For each item, please choose the rating from 1 to 5 that most closely reflects your experience up to the age of 18years. Because your parent(s)/carer(s) may have been very different, please rate them separately. *Space given for this*

1	2	3	4	5
Never	**Rarely**	**Some of the time**	**Most of the time**	**All of the time**

1. My parent/carers would become angry if I disagreed with them.

2. When I was anxious, my parent/carers ignored this.

3. If I was happy, my parent/carers would be sarcastic and say things like: “What are you smiling at?”

4. If I was upset, my parent/carers said things like: “I'll give you something to really cry about!”

5. My parent/carers made me feel OK if I told them I didn't understand something difficult the first time.

6. If I was pleased because I had done well at school, my parent/carers would say things like: “Don't get too confident”.

7. If I said I couldn't do something, my parent/carers would say things like: “You're being difficult on purpose”.

8. My parent/carers would understand and help me if I couldn't do something straight away.

9. My parent/carers used to say things like: “Talking about worries just makes them worse”.

10.If I couldn't do something however hard I tried, my parent/carers told me I was lazy.

11. My parent/carers would explode with anger if I made decisions without asking them first.

12. When I was miserable, my parent/carers asked me what was upsetting me, so that they could help me.

13. If I couldn't solve a problem, my parent/carers would say things like: “Don't be so stupid — even an idiot could do that!”

14. When I talked about my plans for the future, my parent/carers listened to me and encouraged me.

Appendix 9: Emotional Processing Scale (EPS-25)

Please note that this appendix has been removed due to copyright

Appendix 10: Hospital Anxiety and Depression Scale (HADS)

Tick the box beside the reply that is closest to how you have been feeling in the past week. Don't take too long over you replies: your immediate is best.

1. I feel tense or 'wound up':
Most of the time
A lot of the time
From time to time, occasionally
Not at all

2. I still enjoy the things I used to enjoy:

Definitely as much
Not quite so much
Only a little
Hardly at all

3. I get a sort of frightened feeling as if something awful is about to happen
Very definitely and quite badly
Yes, but not too badly
should
A little, but it doesn't worry me
Not at all

4. I can laugh and see the funny side

of things
As much as I always could
Not quite so much now
Definitely not so much now
Not at all

5. Worrying thoughts go through my to
Mind
A great deal of the time
A lot of the time
From time to time, but not too often
Only occasionally

6. I feel cheerful
Not at all
Not often
Sometimes
Most of the time

8. I feel as if I am slowed down:
Nearly all the time
Very often
Sometimes
Not at all

9. I get a sort of frightened feeling like 'butterflies' in the stomach:
Not at all
Occasionally
Quite Often
Very Often

10. I have lost interest in my appearance:
Definitely
I don't take as much care as I

I may not take quite as much care
I take just as much care as ever

11. I feel restless as I have to be on the
move:
Very much indeed
Quite a lot
Not very much
Not at all

12. I look forward with enjoyment

things:
As much as I ever did
Rather less than I used to
Definitely less than I used to
Hardly at all

13. I get sudden feelings of panic
Very often indeed
Quite often
Not very often
Not at all

7. I can sit at ease and feel relaxed:

Definitely
Usually
Not Often
Not at all

14. I can enjoy a good book or radio or TV program:

Often
Sometimes
Not often
Very seldom

Appendix 11: List of charities and eating disorder support forums

1) Beating Eating Disorders: http://www.b-eat.co.uk/support-us/get-involved/research/take-part/

2) Men Get Eating Disorders Too: http://mengetedstoo.co.uk

3) No Bodies Perfect: http://www.nbp-eating-disorders.co.uk/

4) http://prettythinagain.webs.com/

5) http://www.eatingdisorderselfhelp.com/forum

6) http://webiteback.com/forum/

7) http://www.recoveryboat.com/

Appendix 12: Advertisement used

ADVERTISEMENT

Title: Contextual factors associated with psychological inflexibility and distress in clinical and sub-clinical eating disorders

Hello there!

My name is Maria Tucknott and I am a Trainee Clinical Psychologist. As part of my doctoral qualification in clinical psychology, I am investigating what background factors may be associated with how people manage their emotions and the amount of psychological distress they experience. The results will help us get a better understanding of how we can support people who have concerns about their eating

Can you help?
I'm looking for males and females aged 16 and over to take part and this will involve you filling in some questionnaires online.

- Do you have some concerns about your eating?
- Do you **have** a diagnosis of an eating disorder now?

If you answered **YES** to *any* of the above then please continue reading as you are suitable to take part!

Interested?
Taking part in this study will involve you clicking the following link *[web link here]* and answering a series of questionnaires **once**. Then that's it, all done..!

If you would like to take part, then please click the link to get started.

Thank you for taking the time to read this. If you have any further questions then please click the link to read the information sheet or contact me at: m.tucknott@herts.ac.uk

Appendix 13: Warning of sensitive nature of study

Thank you for clicking through to this study. Please click the **continue** button for more information. Once you have read this and are happy to take part, you will then be able to begin.

Please make sure that you read the information sheet carefully. The questionnaires within this study will ask sensitive questions about lots of things such as difficult life experiences and questions about your body shape and weight. There will also be questions about how thoughts and feelings are managed and processed, as well as questions about your mood and any concerns you may have about eating and/or your weight.

Please note that this survey will need to be completed in one go. This means that you cannot exit and finish it later on.

Appendix 14: Information sheet

Information Sheet for Participants

Research Title: Contextual factors associated with psychological inflexibility and distress in clinical and sub-clinical eating disorders

Introduction

You are invited to take part in a research study exploring factors that may be associated with how people manage their emotions and the amount of psychological distress they experience. Before you decide whether you would like to give consent to take part, please take the time to read the following information which I have written to help you understand why the research is being carried out and what it will involve.

The researchers

The study is being carried out by Maria Tucknott, Trainee Clinical Psychologist, as part of a Doctoral qualification in Clinical Psychology. The study is supervised by Dr Saskia Keville, Academic Tutor and Chartered Clinical Psychologist.

What is the purpose of the study?

This research is looking at whether contextual factors, such as difficult life experiences and relationships, are associated with how people internally manage and process their emotional experiences and the amount of psychological distress they subsequently experience. This study is therefore looking at 'normal', everyday ways of managing experiences.

What is involved?

Following reading this information sheet, participants will be asked to spend approximately 25 minutes filling in a range of questionnaires. These will ask about difficult life experiences, difficulties experienced as a child and how you feel about your body. There will also be questions about how thoughts and feelings are managed and processed, as well as questions about your mood and any concerns you may have about eating and/or your weight.

Who is taking part?

This study will invite young people and adults (aged 16 years and over) to take part. They may have concerns about eating and / or their weight and they may have a current diagnosis of an eating disorder or have had an eating disorder in the past. Participants will be recruited from online support forums for people with an eating disorder.

Do I have to take part?

No. If you do not want to take part, or you change your mind, you are free to withdraw at *any time*, without giving a reason.

Will taking part be confidential?

Yes. I will not ask for your name. The guidelines in the Data Protection Act (1998) will be followed, meaning that all information about you will be handled in confidence. All of your responses to the questionnaires will go onto a database using numbers (not names). This means that your data will be anonymous and these responses will only be made available to the researchers.

What are the benefits of taking part?

It is possible that you will not experience any direct benefits as a result of taking part in this research. However, it is hoped that you will find it a positive experience and the knowledge gained from this study will help to develop our understanding of how best to support and help people with eating disorders and mental health difficulties in the future.

What are the possible disadvantages of taking part?

Given the sensitive nature of the topic area it is possible that some of the questions you are asked may make you feel upset after doing this study. You are provided with some sources of support below, so that you can contact them in the event that you do become upset. You will also be given further information about where to get support from at the end of the study. If you continue to feel upset then you can contact me. There are no other disadvantages or risks inherent in this study and you are free to withdraw from the study at any point, without giving a reason.

What will happen to the results of this research study?

The results of this study (anonymised) will be reported in a thesis for the purpose of gaining a qualification in Clinical Psychology. The thesis will be held in the University of Hertfordshire Learning Resource Centre which will be accessible to interested parties. Further to this, a summary of the main research findings may be published in a research paper but you will not be identifiable.

What if I have questions or concerns?

If you have any concerns or further questions about the research, please feel free to contact me, details of which are below. Alternatively, you can contact Dr Saskia Keville (research supervisor) on the email below

Who has approved this study?

This study was reviewed by University of Hertfordshire Research Ethics Committee and was given ethical approval (aLMS/PG/UH/00022).

I've read everything and would like to take part

Thank you for taking time to read this. If you would like to take part in this study then please click *[insert web link here]* to start the survey.

I've decided not to take part

Thank you for taking the time to read this. Please close your browser window if you do not wish to take part in the study.

Sources of support

If answering these questions makes you upset or worried, you can find advice and support from:

Beating Eating Disorders		**NHS Direct**
Website: www.b-eat.co.uk	or	Website: www.nhsdirect.nhs.uk
Helpline: 0845 634 1414		Helpline: 0845 4647

Contact details of the researcher:

Maria Tucknott (Trainee Clinical Psychologist), Email address: m.tucknott@herts.ac.uk

Dr Saskia Keville (Clinical Psychologist). Email address: s.keville@herts.ac.uk

Postal address: Doctorate in Clinical Psychology Training Course , University of Hertfordshire, Hatfield, Herts, AL10 9AB

Appendix 15: Informed Consent sheet

Consent Form

Title of Project: Contextual factors associated with psychological inflexibility and distress in clinical and sub-clinical eating disorders

Researcher: Maria Tucknott, Trainee Clinical Psychologist

Please tick box

1) I confirm that I have read and understand the information explaining what the research entails and what will be expected from me. I have been given the opportunity to consider the information and ask questions if required. ☐

2) I understand that participation is voluntary and that I am free to withdraw at any time, without giving any reason. I understand that if I withdraw from the study, the data that I have submitted will also be withdrawn at my request. ☐

3) I understand that the information that I will submit will be confidential and anonymised, and used only for this study. I understand that my information will be filed in a locked cabinet or encrypted and stored electronically on password protected computers. ☐

4) I agree to participate in the study. ☐

Appendix 16: Reminder about withdrawing from the study

Thank you so much for answering the questions so far, you are almost at the end. Just a few more questions to go!

Just a quick reminder: If you decide that you no longer want to continue with this study, please email me at m.tucknott@herts.ac.uk with "withdrawal" in the subject line. You do not have to give me a reason and I will not ask you any questions. All I will do is send you some further information about the study and confirm that your data will not be included.

Appendix 17: Debriefing sheets

Debriefing Sheet for those who completed the study

Thank you very much for taking part in this study, it is greatly appreciated!

This study aimed to explore the relationships between contextual factors, such as difficult early experiences, psychological inflexibility (how people manage and process their internal thoughts and feelings) and psychological distress in people who have difficulties with eating. Research has shown that psychological inflexibility is associated with psychological distress in eating disorders, however it is unclear what factors or experiences may influence how flexibly one can process internal events. It is hoped that this research will (a) begin to address this question and; (b) help us improve our understanding of how we can help and support people in future.

In the event that participation in this research has raised any issues or concerns for you, please do not hesitate to contact me, or my supervisor, using the details below.

Researcher: Mrs Maria Tucknott
Trainee Clinical Psychologist
University of Hertfordshire
m.tucknott@herts.ac.uk

Supervisor: Dr Saskia Keville
Clinical Psychologist
University of Hertfordshire
s.keville@herts.ac.uk

SOURCES OF SUPPORT

Thinking about your experiences may have left you feeling low or upset, this is quite normal and often passes after a few days. However, if these feelings persist there are local sources of support and comfort which may already be familiar to you. The most immediate sources of comfort and help are likely to be your own family and friends or someone that you trust. If you are concerned about your psychological wellbeing or are worried that you may have an eating disorder, then your GP may be able to refer you to more specialised local support services such as counsellors.

The following national organisations also offer support:

- **The Samaritans**
  Telephone: 08457 909090
  Web address: www.samaritans.org

The Samaritans is a helpline which is open 24 hours a day for anyone in need. It is staffed by trained volunteers who will listen sympathetically.

- **ChildLine**
  Telephone: 0800 1111
  Web address: www.childline.org.uk

ChildLine is a registered charity for children and young people. You can call them at any time to speak to a counsellor and the calls are free and confidential. The counsellors are trained and have experience of listening and talking to children and young people.

- **Beating Eating Disorders (B-EAT)**
  Telephone: 0845 634 1414 (for adults aged 18+) and 0845 634 7650 (for young people under 18).
  Web address: www.b-eat.co.uk

B-EAT is a registered charity for everyone of all ages and it offers support and information for people who have an eating disorder or are worried that they might have an eating disorder.

- **The National Association for People Abused in Childhood**
  Telephone: 0800 0853330
  Web address: www.napac.org.uk

The National Association for People Abused in Childhood is a registered charity that offers support and information for people abused in childhood.

Debriefing Sheet for those who withdrew from the study

As you have decided that you no longer wish to continue with this study, I would like to confirm that your data (i.e. any information you have provided) will **not** be included in the project. This means that your data will be permanently and securely destroyed.

This study aimed to explore the relationships between contextual factors, such as difficult early experiences, psychological inflexibility (how people manage and process their internal thoughts and feelings) and psychological distress in people who have difficulties with eating. Research has shown that psychological inflexibility is associated with psychological distress in eating disorders, however it is unclear what factors or experiences may influence how flexibly one can process internal events. It is hoped that this research will (a) begin to address this question and; (b) help us improve our understanding of how we can help and support people in future.

In the event that participation in this research has raised any issues or concerns for you, please do not hesitate to contact me, or my supervisor, using the details below.

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Trainee Clinical Psychologist
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m.tucknott@herts.ac.uk

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The National Association for People Abused in Childhood is a registered charity that offers support and information for people abused in childhood.

Appendix 18: Ethics Approval form

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University of Hertfordshire

UNIVERSITY OF HERTFORDSHIRE
HEALTH AND HUMAN SCIENCES

MEMORANDUM

University of Hertfordshire
Higher Education Corporation
Hatfield, Hertfordshire
AL10 9AB

Telephone +44 (0) 1707 284000
Fax +44 (0) 1707 284115
Website www.herts.ac.uk

TO **Maria Tucknott**

CC **Saskia Keville**

FROM Dr Richard Southern, Health and Human Sciences, ECDA Chairman

DATE **28/06/13**

Protocol number: aLMS/PG/UH/00022

Title of study: Contextual Factors Associated With Psychological Inflexibility and Distress in Clinical and Sub-Clinical eating Disorders

Your Minor Modification request has been approved.

This approval is valid:

From: 25/04/13

To: 30/12/14

On behalf of the Health and Human Sciences Ethics Committee with Delegated Authority,

Dr Richard Southern
HHS ECDA Chairman

Please note:

Approval applies specifically to the research study/methodology and timings as detailed in your Form EC1. Should you amend any aspect of your research, or wish to apply for an extension to your study, you will need your supervisor's approval and must complete and submit form EC2. In cases where the amendments to the original study are deemed to be substantial, a new Form EC1 may need to be completed prior to the study being undertaken.

Appendix 19:

Hypotheses	Finding	Effect size
1) There will be a positive relationship between eating pathology and psychological inflexibility (experiential avoidance and cognitive fusion)	**Confirmed** (p <.01)	*r* = .45
2) There will be a positive relationship between eating pathology and Thought-Shape Fusion	**Confirmed** (p <.01)	*r* = .58
3) There will be a positive relationship between eating pathology and Emotional Processing.	**Confirmed** (p <.01)	*r* = .51
4) There will be a positive relationship between maternal invalidation and/or paternal invalidation in childhood and eating pathology	**Not confirmed** (all p >.05)	
5 i) There will be a positive relationship between maternal invalidation in childhood and psychological inflexibility	**Partially Confirmed** (only experiential avoidance was significant at p <.01)	**Experiential avoidance:** Rho = .23
5 ii) There will be a positive relationship between paternal invalidation in childhood and psychological inflexibility	**Confirmed** (p <.01) (p <.05)	**Experiential avoidance:** Rho = .24 **Cognitive fusion:** Rho = .17
6) Experiences of abuse in childhood would be positively related to eating pathology	**Not confirmed** (all subscales were p >.05)	
7 i) Experiences of abuse in childhood would be positively related to psychological inflexibility (**experiential avoidance**)	**Partially confirmed** (only emotional abuse & sexual abuse were significant at p <.01)	**Emotional abuse** *r* = .27 **Sexual abuse** *r* = .22

7 ii) Experiences of abuse in childhood would be positively related to psychological inflexibility (**Cognitive Fusion**)	**Partially confirmed** (only emotional abuse was significant at p <.01)	**Emotional abuse** *r* = .21
8) There will be a positive relationship between Psychological Inflexibility and anxiety. *(partial correlation is reported here)*	**Confirmed** (p <.01) (p <0.1)	**Experiential avoidance** *r* = .56 **Cognitive fusion:** *r* = .57
9) There will be a positive relationship between Psychological Inflexibility and depression *(partial correlation is reported here)*	**Confirmed** (p <.01) (p <.01)	**Experiential avoidance:** *r* = .32 **Cognitive fusion:** *r* = .24
10) There will be a positive relationship between eating pathology and anxiety and/or depression. *(partial correlation is reported here)*	**Confirmed** (p <.01) (p <.01)	**Experiential avoidance:** *r* = .32 **Cognitive fusion:** *r* = .29
1a) The typical and elevated clinical groups will show higher psychological inflexibility than the low clinical group.	**Confirmed** (p <.05)	**Experiential avoidance:** η^2 = .18 **Cognitive fusion:** η^2 = .19
2a) The typical and elevated clinical groups will show higher levels of Thought-Shape Fusion than the low clinical group	**Confirmed** (p <.05)	η^2 = .27

3a) The typical and elevated clinical groups will show higher levels of emotional processing than the low clinical group	**Confirmed** ($p < .05$)	$\eta^2 = .25$
4a) The typical and elevated clinical groups will show higher levels of maternal and paternal invalidation than the low clinical group	**Not confirmed** ($p > .05$)	N/A
5a) The typical and elevated clinical groups will show higher levels of childhood abuse than the low clinical group	**Not confirmed** ($p > .05$)	N/A
6a) The typical and elevated clinical groups will show higher levels of anxiety than the low clinical group	**Confirmed** ($p < .05$)	$\eta^2 = .15$
7a) The typical and elevated clinical groups will show higher levels of depression than the low clinical group	**Partially confirmed** (only the elevated clinical group had significantly higher levels of depression at ($p < .05$)	$\eta^2 = .14$

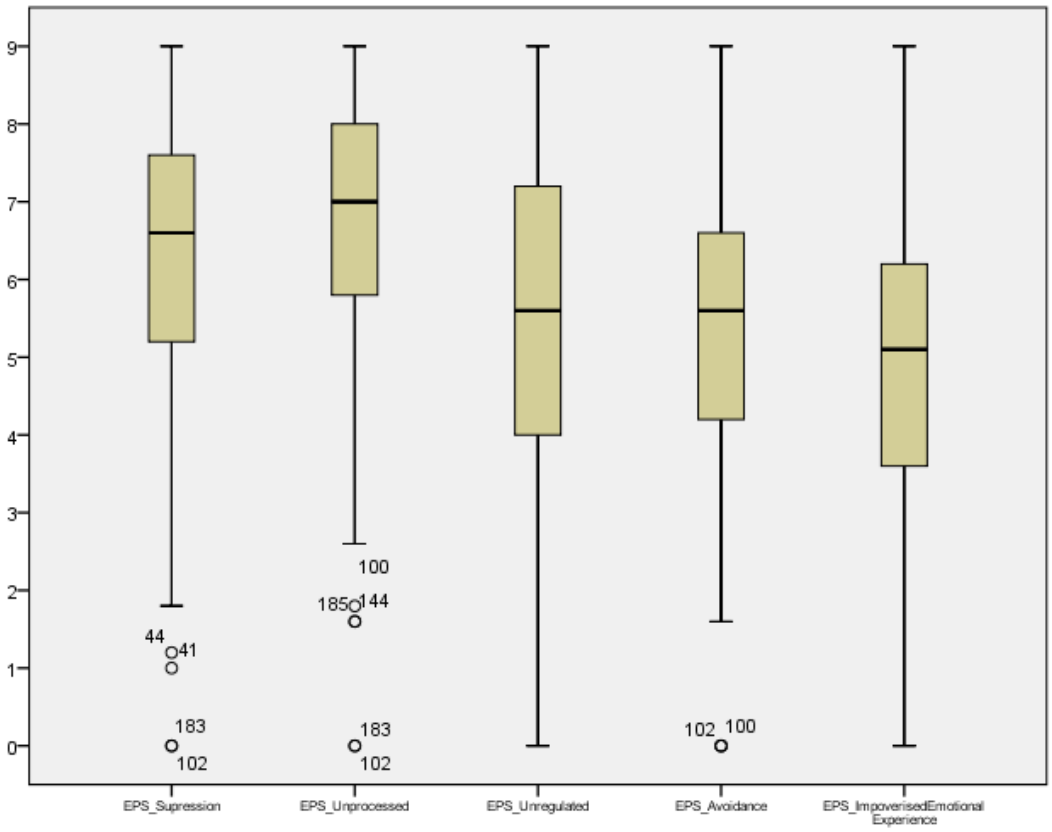
Appendix 20: Detailed Table of Treatment Status

Frequency and percentage of treatment status in each Eating Disorder Risk group

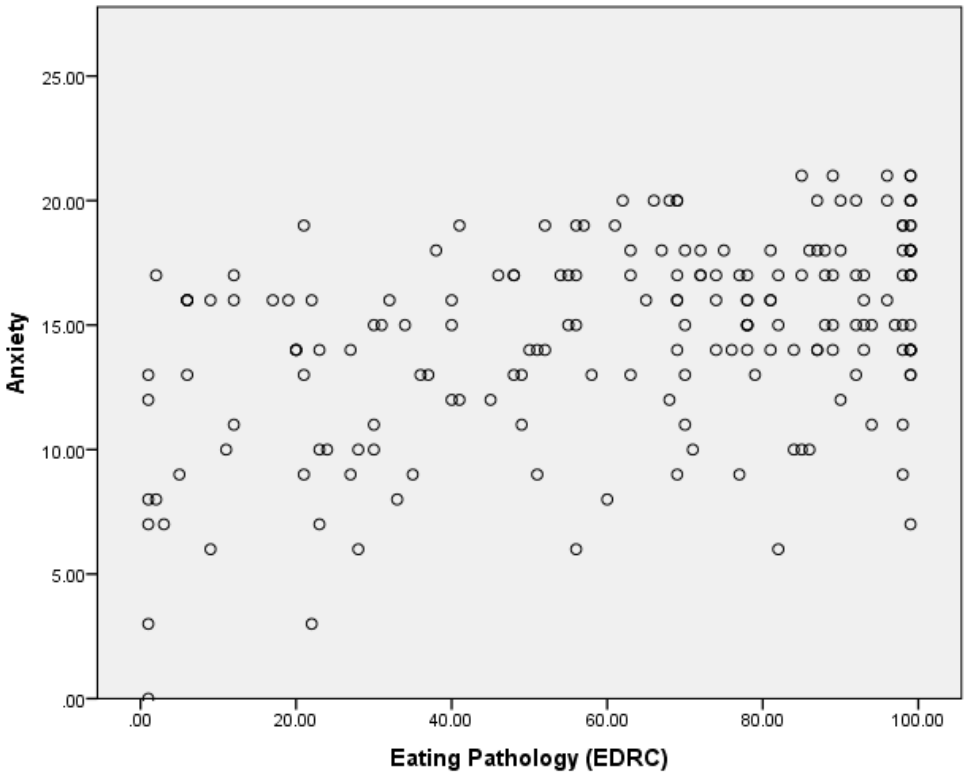
		EDRC Classification			
		Elevated Clinical Range	**Typical clinical Range**	**Low clinical Range**	
Variable	**Category**				**Total N**
Currently Receiving Treatment	Yes	53	31	13	97
	No	54	19	20	93
Total N (%)		**107**	**50**	**33**	**190 (100%)**
Nature of Treatment Received	Inpatient treatment	3	0	0	**3**
	Due to start inpatient treatment	3	0	0	**3**
	Outpatient treatment	15	8	3	**26 (18%)**
	Psychological Therapy	34	22	10	**66 (47%)**
	Psychiatrist	4	1	2	**7 (5%)**
	Dietician / Nutritionist	7	10	0	**17 (12%)**
	Supervision (of meals & snacks)	1	1	0	**2 (1%)**
	Support Group	1	3	1	**5 (4%)**
	Self help	0	0	1	**1 (1%)**
	Medication	2	5	0	**7 (5%)**
	GP	0	3 (2%)	1 (1%)	**4 (3%)**
Total N (%)		**70 (50%)**	**53 (38%)**	**18 (13%)**	**141* (100%)**

* multiple interventions were common amongst the sample which is reflected in N being greater than the sample size of 97 (51%) currently receiving treatment.

Appendix 21: Box-plot of Emotional Processing scores



Appendix 22: Scatterplot of anxiety and eating pathology



Appendix 23: Scatterplot of depression and eating pathology