Abstract

Background

Research indicates that eating disturbances are twice as prevalent among adolescents with type 1 diabetes compared to their healthy peers; comparisons with other chronic illness groups are inconclusive. Adolescent self-esteem and parenting factors have been found to be associated with eating disturbances in type 1 diabetes. However, to date the literature is methodologically limited by a lack of comparison group, and has failed to consider the role of parent care and overprotection.

Aims

This study aimed to explore the relationship between adolescent-perceived parent care and overprotection, self-esteem and eating disturbances in a group of adolescents with type 1 diabetes compared to a group with asthma.

Method

Participants were 16 – 18 year old males and females with a diagnosis of type 1 diabetes ($n = 65$) or asthma ($n = 37$) recruited through NHS clinics. The Eating Disorder Examination-Questionnaire was used along with the Parental Bonding Instrument, as a measure of parent overprotection and care, and the Rosenberg Self-Esteem Scale. A cross-sectional, self-report questionnaire design was utilised.

Results

Unexpectedly, the asthma group reported significantly higher levels of eating psychopathology than the type 1 diabetes group. Significant negative associations were found between parent care and eating disturbances in both illness groups. Additionally, significant
positive associations were found between parent overprotection and eating disturbances in the type 1 diabetes group, but not the asthma group. As predicted, self-esteem was strongly negatively correlated with eating disturbances in both groups.

**Conclusion**

Overall, the results suggest that adolescents with both asthma and type 1 diabetes may be vulnerable to higher levels of eating psychopathology, which in turn is associated with poorer self-esteem and less adaptive parenting. The findings are considered in relation to illness-related weight gain and the broader impact of chronic illness. The theoretical and clinical implications are discussed.
Eating Disturbances in Chronic Illness  

J. Hatton

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CHAPTER ONE

1. Introduction

1.1 General Overview

The psychological and emotional well-being of children and adolescents with chronic illnesses has increasingly been on the agenda of healthcare providers in the UK in recent years (Department of Health, 2009). Evidence suggests that this group are at an increased risk of psychopathology compared to their healthy peers (Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995). Of particular interest has been the increased prevalence of eating disorders (EDs) and sub threshold levels of eating disturbances in young people with type 1 diabetes. The current study is concerned with better understanding these phenomena. Disordered attitudes and behaviours towards food, eating and weight have been shown to be associated with poor treatment adherence in this group (Kichler, Foster & Opipari-Arrigan, 2008). Non-adherence to treatment can have serious physical consequences for individuals with diabetes, which can be life-threatening. Therefore, it is important that the development and maintenance of eating disturbances in adolescents with type 1 diabetes is understood better. This will enable appropriately targeted preventative strategies and interventions to be developed.

Daneman, Olmsted, Rydall, Maharaj and Rodin (1998) propose that the observed comorbidity between type 1 diabetes and EDs can be explained by a combination of general risk factors for eating disturbances, and diabetes-specific factors including: 1) treatment related weight gain; 2) an emphasis on diet in illness management; and 3) availability of insulin manipulation as a weight loss strategy. Psychological models of EDs in the general population emphasise the role of self-esteem and the parent-child relationship, amongst other factors, in the development and maintenance of EDs (Fairburn, Cooper & Shafran, 2003). These factors may be amplified in adolescents with a chronic illness as evidence indicates
that they may be more vulnerable to developing low self-esteem (Vitulano, 2003). In addition, chronic illness can have a significant impact on the functionality of families, with evidence to suggest that the parent-child relationship, in particular, may be negatively affected (Streisand, Swift, Wickmark, Chen, & Holmes, 2005).

Indeed, previous research indicates that both low self-esteem and difficulties in the parent-child relationship are associated with higher levels of eating disturbances in young people with type 1 diabetes (Colton, Olmsted, Daneman, Rydall, & Rodin, 2007). However, the evidence base is methodologically limited by a consistent lack of a comparison group in the study designs. This means that conclusions cannot be drawn about whether or not the observed associations are unique to a type 1 diabetes population, or whether they would also be observed in other chronic illness groups. This is an important distinction as, if low self-esteem and problematic parent-child relationships are similarly associated with higher levels of eating disturbances in other chronic illness groups, then these associations do not further our understanding about the increased prevalence of eating disturbances seen in type 1 diabetes. In order to address this, the current study employs a comparison group of young people with a diagnosis of chronic asthma. There is very little research to date looking at the psychological well-being of young people with asthma.

Therefore, the aim of this investigation is to explore the relationship between both individual and interpersonal factors and eating disturbances among young people with type 1 diabetes compared with those with chronic asthma. More specifically, the study is designed to explore associations between adolescent-perceived parent care and overprotection, adolescent self-esteem and eating disturbances in the two groups.

1.2 Chapter Overview

This chapter begins with an explanation of the pathology, prevalence and impact of type 1 diabetes in adolescence. The relationship between type 1 diabetes and eating disturbances...
disturbances is explored in light of relevant theoretical models. A review of the relevant
literature is presented and the limitations of previous studies are outlined. The inclusion of a
comparison group of young people with asthma is introduced and the rationale provided.
Finally, the research questions and a priori hypotheses are outlined.

1.3 Type 1 Diabetes

1.3.1 Pathology, prevalence and treatment of type 1 diabetes.

Type 1 diabetes is a life-long endocrine disorder that is characterised by impaired
insulin production. It is thought to be caused by a combination of genetic factors and
environmental triggers that compromise the immune system (Devendra, Liu, & Eisenbarth,
2004). In type 1 diabetes the autoimmune system destroys beta cells in the pancreas, which
are needed for the production of insulin. Insulin is a hormone that facilitates the movement of
glucose from the blood into body cells to be used as energy (Daneman, 2006). Therefore, a
lack of insulin production impacts upon healthy regulation of blood glucose levels and, if left
untreated, means that glucose levels remain at a dangerously high level in the blood; the
consequences of which are outlined below. Early signs and symptoms of the disorder include
increased thirst and need to urinate, weight loss and excessive tiredness. Onset is typically in
childhood and affects around 1 in every 700-1000 adolescents in the UK (Diabetes UK,
2010). Prevalence rates in adolescents do not appear to differ by gender (DIAMOND Project
Group, 2006), although evidence suggests that females are more vulnerable to certain
complications, such as diabetic ketoacidosis (Rewers et al., 2002).

The treatment of type 1 diabetes is focused on maintaining metabolic control (i.e.
healthy blood glucose levels) through a combination of intensive daily interventions. These
include self-monitoring of blood glucose levels, multiple insulin injections, meal planning
and nutritional control (Carr, 2002). Several approaches to meal planning are available,
although currently a carbohydrate counting approach is the most common (Bui & Daneman,
2006). This involves close monitoring of carbohydrate intake and calculation of the required insulin dose in response. Recent developments have also led to an increasing number of young people receiving insulin pump therapy. This involves continuous infusion of insulin into the blood rather than multiple injections throughout the day. Research suggests that quality of life improves for young people when transitioned onto pump therapy (McMahon et al., 2005). People with type 1 diabetes are at risk of developing both hyperglycaemia and hypoglycaemia, which are caused by high and low blood glucose levels respectively. Failure to maintain healthy metabolic control can lead to severe medical complications. These include retinopathy, which leads to blindness, kidney failure, peripheral vascular problems, which can lead to amputations, and cardiovascular disease (Melendez-Ramirez, Richards, & Cefalu, 2010).

### 1.3.2 Type 1 diabetes in adolescence.

The management of type 1 diabetes requires considerable daily attention, control and responsibility on the part of the diagnosed individual. For children and adolescents, this places additional demands on the family unit as the diagnosed child progresses through childhood and adolescence, and finally into adulthood. In normal child development the majority of a child’s care needs are met by their parent(s), with increasing responsibility being granted to the child as they approach and enter into adolescence (Bui & Daneman, 2006; Daneman & Frank, 1996). Similarly, during adolescence those with type 1 diabetes have to learn to manage their illness more independently. During this time metabolic control is often very poor (Dashiff & Bartolucci, 2002; Wills et al., 2003) and is associated with increased risk of serious physical complications (Bryden, Dunger, Mayou, Peveler, & Neil, 2003). It is likely that poor metabolic control during this period is due to several normal developmental changes that occur in adolescence, each of which shall be discussed below.
1.3.2.1 Physical and biological changes.

During puberty the body experiences many physical and biological changes. Certain hormonal changes increase the body’s resistance to insulin production. This makes achieving good metabolic control even more difficult (Amiel, Sherwin, Simonson, Lauritano, & Tamborlane, 1986; Cameron, 2006).

1.3.2.2 Changes in responsibility and independence.

Throughout adolescence there are changes to the amount of autonomy and responsibility entrusted to a young person. It is widely accepted that the primary developmental challenge of this phase is to develop increasing individuation and autonomy. Erikson (1968) describes the process as one of negotiating independence and of experimenting with different roles to be able to emerge from adolescence with a clearer sense of identity as an autonomous individual. The added demands of chronic illness during adolescence add to the normative challenges presented by these important developmental tasks (Yeo & Sawyer, 2005). In particular, in diabetes this is a period during which primary responsibility for diabetes self-care is transitioned from parent(s) to adolescent (Anderson et al., 2009). The challenge of negotiating this transition may explain the difficulties with metabolic control for some young people.

1.3.2.3 Increasing body dissatisfaction.

There is a rapid increase in levels of body dissatisfaction and disturbed eating behaviour in males and females in the general population post-puberty (Field et al., 2001; Rosenblum & Lewis, 1999; Thompson, 1990). Factors associated with such increases in body dissatisfaction include physical maturation, history of being teased about weight and/or shape, negative affect and changes in Body Mass Index (BMI; Bearman, Martinez, & Stice, 2006; Thompson, Coovert, Richards, Johnson, & Cattarin, 1995). Eating disturbances, including body dissatisfaction, in groups of young people with type 1 diabetes have been
shown to be associated with poorer metabolic control. For example, Neumark-Sztainer et al. (2002) found a significant correlation between disturbed eating behaviour and blood glucose levels in a sample of adolescent males ($r = .26, p < .05$) and females ($r = .33, p < .01$). This association between eating disturbances and type 1 diabetes will be discussed further below.

### 1.4 Type 1 Diabetes and Eating Disturbances

#### 1.4.1 Defining eating disturbances.

Eating disordered thoughts, feeling and attitudes are best understood on a spectrum, ranging from sub-threshold eating disturbances through to clinically significant EDs (Patton, 1988). The Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-V; American Psychiatric Association, 2013) describes four types of EDs. The diagnostic criteria for each are outlined in Table 1. The core psychopathology in Anorexia Nervosa (AN) and Bulimia Nervosa (BN) is an over-evaluation of, and extreme concern with, one’s own weight and shape. In AN this core feature is accompanied by control and restriction over calorie intake in order to maintain a low body weight. In BN the core psychopathology presents with episodes of binge eating and compensatory purging strategies. Both disorders are accompanied by emotional disturbance (Harrison, Sullivan, Tchanturia, & Treasure, 2009; Markey & Vander Wal, 2007) and low self-esteem (Williams et al., 1993). Full syndrome EDs are relatively rare in the general population. Review papers have reported prevalence rates for AN and BN ranging from 0.3% - 0.9% and 1% - 1.5% respectively among females (Hudson, Hiripi, Pope, & Kessler, 2012; Smink, Hoeken, & Hoek, 2012). Prevalence rates reported in these review papers for males are considerably lower, ranging from 0.1% - 0.5% for BN and estimates of around 0.3% for AN.
### DSM-V Diagnostic Criteria for Eating Disorders

<table>
<thead>
<tr>
<th>Eating disorder</th>
<th>Diagnostic criteria</th>
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<tr>
<td><strong>Anorexia nervosa (AN)</strong></td>
<td>- Maintaining significantly low weight.</td>
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<td></td>
<td>- Fearful of gaining weight or becoming fat.</td>
</tr>
<tr>
<td></td>
<td>- Over-evaluation of shape and weight.</td>
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<tr>
<td><strong>Bulimia nervosa (BN)</strong></td>
<td>- Frequent binge eating episodes (consumption of an unusually large amount of food accompanied by a sense of lack of control).</td>
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<tr>
<td></td>
<td>- Compensatory behaviours to prevent weight gain, including self-induced vomiting, extreme exercising and laxative abuse.</td>
</tr>
<tr>
<td></td>
<td>- Over-evaluation of shape and weight.</td>
</tr>
<tr>
<td><strong>Binge eating disorder (BED)</strong></td>
<td>- Absence of AN.</td>
</tr>
<tr>
<td></td>
<td>- Frequent binge eating episodes accompanied by distress.</td>
</tr>
<tr>
<td></td>
<td>- Absence of compensatory behaviours.</td>
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<tr>
<td><strong>Other specified eating disorder</strong></td>
<td>- All criteria are met for AN apart from significantly low weight, despite weight loss.</td>
</tr>
<tr>
<td></td>
<td>- Criteria for BN are met but symptoms occur less frequently.</td>
</tr>
<tr>
<td></td>
<td>- Frequent use of compensatory behaviour to influence weight and/or shape after normal food consumption (e.g. vomiting after eating a bag of crisps).</td>
</tr>
<tr>
<td></td>
<td>- Criteria for BED are met but symptoms occur less frequently.</td>
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Sub-threshold eating disturbances are defined as milder disturbances in eating attitudes and behaviours that, due to lower frequency and/or severity, do not meet the threshold for diagnosis of a clinical ED (Maharaj, Rodin, Olmsted, Connolly, & Daneman, 2003). Accurate prevalence rates for this group in the general population are more difficult to define. In a sample of 919 females aged 13-19 years of age, 10.7% were found to report sub-threshold eating disturbances, as defined above (Cotrufio, Barretta, Monteleone, & Maj, 1998).

Research has shown that young people with eating disturbances are at an increased risk of developing clinical levels of EDs in the future. A sample of 14-15 year old Australian female adolescents completed measures of ED symptoms at six time points over three years. The results showed that 8% of the sample reported extreme dieting and 60% reported moderate dieting at baseline. Those who reported extreme dieting had a 20% chance of developing a clinical ED over the next year, compared to a 2.5% chance for those disclosing moderate dieting and 0.2% chance for the non-dieters (Patton, Seizer, Coffey, Carlin, & Wolfe, 1999). Furthermore, research has demonstrated that eating disturbances in adolescent females are significantly associated with functional impairment and emotional distress (Stice, Marti, Shaw, & Jaconis, 2010).

1.4.2 Prevalence rates for EDs and eating disturbances in type 1 diabetes.

There are many studies reporting prevalence rates of eating disturbances in samples of people with type 1 diabetes. Historically there has been disagreement about whether or not the incidence rates for EDs are elevated in this population. Some studies have demonstrated increased levels (Lloyd, Steel, & Young, 1987; Rodin, Craven, Littlefield, Murray, & Daneman, 1991), while others have found no differences, when comparing eating disturbances in samples with type 1 diabetes with either healthy controls or standardisation samples (Marcus, Wing, Jawad, & Orchard, 1992; Robertson & Rosenvinge, 1990). This may
be because earlier studies were hampered by small sample sizes, variability in the definitions used to operationalize EDs, and lack of comparison groups.

Daneman (2002) reviewed eight studies that reported prevalence rates for EDs and/or subthreshold eating disturbances in samples of adolescent and young adult females with type 1 diabetes. Studies that employed control groups and used standardised interview-based measures for assessing the presence of EDs were included. Of these eight studies, five reported prevalence rates of both EDs, as defined by DSM-IV (American Psychiatric Association, 2000), and subthreshold eating disturbances (e.g. Jones, Lawson, Daneman, Olmsted, & Rodin, 2000; Vila et al, 1995); the remaining three studies only reported on EDs (e.g. Engstrom et al., 1999; Fairburn, Peveler, Davies, Mann, & Mayou, 1991). Rates for EDs in the type 1 diabetes groups across the studies ranged from 0% to 11% compared to 0% to 7.5% in the control groups. Not surprisingly, prevalence rates for subthreshold eating disturbances were considerably higher and ranged from 6.5% to 35% for those with type 1 diabetes compared to 4% to 22% in the control groups. Daneman combined the findings from across these studies and reported an odds ratio (OR) of 2:0; indicating that EDs and subthreshold eating disturbances are twice as prevalent in young females with type 1 diabetes compared to their age-matched healthy peers. In a similar meta-analysis, Nielson (2002) mirrored this finding, reporting an OR of 2:0 for subthreshold EDs in young females with type 1 diabetes. Interestingly, Neilson did not find increased prevalence rates of AN in groups with type 1 diabetes. Similarly, Mannucci et al. (2005) reported significantly higher levels of BN, but not AN, in females with type 1 diabetes compared to non-diabetics. The majority of research in this area has been carried out with mid- to late- adolescent females. It is at this age that young people, both with and without type 1 diabetes, are at the greatest risk of developing both sub-clinical and diagnosable EDs (Daneman, 2002; Goldbloom & Garfinkel, 1993).
The occurrence of EDs and subthreshold eating disturbances in males with type 1 diabetes has received considerably less attention. This is likely due to the fact that the incidence of eating disturbances is lower in males than in females among the general population (Striegel-Moore et al., 2009; Woodside, & Kennedy, 1995). Estimates suggest that about 10% of those diagnosed with a clinical ED in the general population are male, and that bulimic symptoms are more common in males than restricting symptoms (Carlat, Camargo, & Herzog, 1997; Weltzin et al., 2005). However, there is some evidence to suggest that this gender gap is narrower in adolescent samples compared with adult samples (Colton, Rodin, Bergenstal, & Parkin, 2009). In many ways EDs present similarly in men and women, although men tend to present with higher levels of compulsive exercising compared to females, who are more likely to use purging strategies such as laxative use and self-induced vomiting (Smolak & Striegel-Moore, 2004).

There is growing evidence to suggest that males with type 1 diabetes are more likely to develop eating disturbances than males without type 1 diabetes. A large state-wide survey carried out in America compared eating disturbances in a group of 310 adolescents who reported having a diagnosis of type 1 diabetes, with a group of 850 demographically matched controls (Neumark-Sztainer et al., 1996). Significantly more males in the type 1 diabetes group reported engaging in binge eating, purging and dieting to control weight, than males in the non-diabetes group. However, these findings must be treated cautiously as the study did not make use of an established questionnaire to measure ED symptoms. Ad-hoc measures were used to ask about certain ED symptoms, e.g. “Do you use any of the following to lose weight? : Laxative or diuretics (water pills)”. Additionally, the study design relied on self-reported diagnosis of type 1 diabetes and therefore, the reliability and validity of the sample is unknown. However, more recent research making use of standardised measures has found similar patterns of results. Svensson, Engstrom and Aman (2003) found that Swedish males
with a diagnosis of type 1 diabetes had significantly higher scores on the Drive for Thinness subscale of the Children’s Eating Disorder Inventory (C-EDI; Garner, 2004) than their healthy peers. Although no participants reported clinical levels of ED, the outcomes suggest that males with type 1 diabetes are at an increased risk of developing ED symptoms. Taken together, these findings indicate that the investigation of eating disturbances is warranted in males with type 1 diabetes as well as females.

**1.4.3 The impact of eating disturbances in type 1 diabetes.**

People with type 1 diabetes have available to them a unique method for weight loss. The deliberate omission or under-dosing of insulin injections leads to weight loss, as the body lacks the insulin required to make use of glucose. Insulin-related compensatory behaviours are more common in this population than other purging behaviours seen in the general population, such as self-induced vomiting and excessive exercise (Jones et al., 2000); they are also included in the DSM-V criteria for EDs (APA, 2013). As discussed in section 1.3.2.3, eating disturbances in young people with type 1 diabetes have been found to be associated with poorer glycaemic control (Mannucci et al., 2005), which may reflect the use of insulin manipulation for weight control among other factors. Research in this area is inconclusive, with some studies finding significant associations between eating disturbances and poor glycaemic control (e.g. Kichler et al., 2008), and others not (e.g. Colton et al., 2007). Additionally, there is variability in the outcomes of studies that have investigated the presence of insulin-omitting behaviour for the purpose of weight control. This is a complex behaviour to investigate as participants may be reluctant to disclose insulin omission. This is particularly pertinent if anonymity is not included as part of the study design and disclosure of such behaviour will be made known to the participant’s clinical team.

In a review paper, Daneman (2002) reported female prevalence rates of 10-15% for insulin omission in early adolescence, increasing to around 30-39% by late adolescence. A
longitudinal study of 91 females with type 1 diabetes found that 14% of participants disclosed omitting their insulin for the purpose of weight control at baseline (Rydall, Rodin, Olmsted, Devenyi, & Daneman, 1997). At four year follow-up this number had increased to 34%. The same study found that blood glucose levels were higher (indicating poor metabolic control) in the group reporting more significant levels of ED symptoms than in the non-disordered group. At follow-up, 86% of participants who had reported high levels of eating disturbances at baseline, and 43% with moderate levels, had developed retinopathy compared to only 23% in the non-disordered group. Retinopathy is caused by high blood glucose levels damaging the retina, and if not treated can lead to blindness. There are few studies investigating insulin manipulation for weight loss in males. Those that are available indicate that generally males do not report omitting their insulin for weight loss purposes (Bryden et al., 1999; Neumark-Sztainer et al., 2002). For example, in an investigation of eating behaviour in males with type 1 diabetes, only three participants (N = 141) reported weight-related insulin omission on a self-report questionnaire (Svensson et al., 2003). Interestingly, these participants then denied this behaviour when asked about it in a face-to-face interview. This highlights the benefit of using anonymous self-report assessment methods for assessing ED behaviours.

In summary, both EDs and subthreshold eating disturbances in this population may be associated with insulin omission and poor glycaemic control for females, which can lead to potentially life threatening diabetes-related complications (Young-Hyman & Davis, 2010). Given this, it is important that the complex interplay between type 1 diabetes and eating disturbances is further understood.

1.4.4 Eating disturbances in chronic illness.

Chronic health conditions, particularly those affecting appearance and/or diet, may increase the risk of eating disturbances during adolescence (Thompson, Heinberg, Altable, & Tantleff-Dunn, 1999). Little research to date has investigated eating disturbances in
populations with heterogeneous chronic health conditions. In a population study in America, a research team investigated the prevalence of self-reported body dissatisfaction and weight-control practices in adolescents with and without chronic illness (Neumark-Sztainer et al., 1995). They recruited a sample of 2149 young people with self-reported illnesses, including diabetes, asthma, seizure disorder, physical disabilities and attention-deficit disorder; the study also employed a comparison group comprising 1381 healthy controls. The findings indicated that the chronic illness group reported both significantly higher levels of body dissatisfaction and more severe weight-loss behaviours than their healthy peers after controlling for demographic characteristics, including BMI and age. Interestingly, analysis revealed that this pattern was consistent across the entire sample of young people with chronic health conditions and was not limited to the conditions with a dietary focus (i.e. diabetes), as was predicted. This study is limited by its reliance on self-report data regarding the presence of chronic illness and its failure to make use of a standardised measure of eating pathology.

Gross, Ireys and Kinsman (2000) investigated the presence of ED symptomology in 71 adult women with either spina bifida or rheumatological disorders, using the Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983). The authors reported that 11 participants (8%) reported clinically significant levels of ED symptoms and that at least 20% reported subthreshold levels of eating disturbances. They found that higher scores on the EDI were correlated with certain illness characteristics, including the impact of the condition on participants’ life and uncertainty around illness trajectory. There has also been interest in investigating eating disturbances in people with cystic fibrosis, which involves dietary management as part of its treatment. However, research in this area has found mixed results, with some findings indicating increased rates of eating disturbances (Shearer & Bryon, 2004), and others indicating no differences between cystic fibrosis groups and healthy controls.
(Raymond et al., 2000). Smith, Latchford, Hall and Dickson (2008) compared eating disturbances in adolescents with type 1 diabetes ($n = 40$), scoliosis ($n = 76$) and healthy controls ($n = 76$). They found that the diabetes group were significantly more likely to have an ED than either of the other two groups ($p < .05$); in the diabetes group 27.5% were classified as having either BN or binge eating disorder. Therefore, whether or not the higher prevalence of subthreshold eating disturbances and EDs is exclusive to type 1 diabetes, or is also observable in other chronic illnesses, remains unclear.

**1.4.5 Summary.**

Taken together the evidence suggests that both EDs and subthreshold eating disturbances are more prevalent in young females with a diagnosis of type 1 diabetes than healthy peers. Symptoms of BN, at both a clinical and sub-clinical level, are more common in this group than symptoms related to AN. Although less research has been carried out using male samples, similar patterns are indicated as in the female groups. This population has available to them a unique compensatory method for weight loss, namely manipulation or omission of insulin treatment. On balance, the evidence suggests that higher levels of eating disturbances are related to poorer glycaemic control in females. It is well documented that poor treatment adherence and lack of metabolic control can lead to serious short and long term physical consequences for the individual. The prevalence of EDs in other chronic illness groups is inconclusive. It is therefore important that further investigations of eating disturbances are carried out in both groups with type 1 diabetes and those with other chronic health conditions. The theoretical context will now be considered.

**1.5 Theoretical Context**

**1.5.1 Diabetes-specific vulnerabilities.**

An explanatory model for the increased prevalence of EDs in type 1 diabetes has been developed by the Eating Disorders and Diabetes Research Group based at the University of
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Toronto (Figure 1), which is one of the most prominent research groups investigating EDs in this illness group (Daneman et al., 1998). The model draws on literature from the general population in proposing that individual, interpersonal, familial and societal risk factors for the development and maintenance of EDs are amplified by certain diabetes-specific factors. Daneman et al. propose that the threshold for the expression of ED symptomology is lower in people with type 1 diabetes. This is due to an interaction between the known risk factors for EDs in the general population (Polivy & Herman, 2002) and vulnerabilities that are unique to people with type 1 diabetes, namely, insulin-related weight gain, focus in treatment on diet and weight, and availability of insulin manipulation for weight loss. Each of these shall be discussed below.

*Figure 1.* Model of interactions between diabetes-specific vulnerabilities, risk factors for eating disturbances in the general population, and diabetes-specific outcomes (Daneman et al., 1998).
1.5.1.1 Insulin-related weight gain.

Young people with type 1 diabetes often experience significant weight-loss pre-diagnosis due to the biological effects of impaired insulin production, which they may find desirable. Post-diagnosis, and once treatment is established, they often then gain weight due to the effectiveness of insulin in feeding their body cells with glucose (Russell-Jones & Khan, 2007; The Diabetes Control and Complications Trial Research Group, 1994). It is well documented in the diabetes literature that adolescents and young adults with type 1 diabetes, both male and female, tend to have higher BMIs compared to healthy matched controls due to insulin treatment (Bryden et al., 1999; Peveler, Fairburn, Boller, & Dunger, 1992). This weight gain may lead to elevated body dissatisfaction and attempts to lose weight or prevent further weight gain.

Methodologically robust prospective studies with adolescents in the general population have found higher BMI and being overweight to be associated with the onset of ED symptoms (Calzo et al., 2012; Cattarin & Thompson, 1994; Graber, Brooks-Gunn, Paikoff, & Warren, 1994). In particular, this has been demonstrated in adolescent females where BMI has been shown to partly predict the onset of binge eating episodes (Stice, Presnell, & Spangler, 2002). In a meta-analysis, Stice (2002) concludes that higher BMI leads to increased body dissatisfaction, perceived pressure to be thin and subsequent dieting behaviour, which are all features of EDs. Therefore, weight gain associated with the treatment of type 1 diabetes may mean that young people are at a greater risk of developing eating disturbances and EDs due to increases in body dissatisfaction and associated attitudes and behaviours.

1.5.1.2 Nutritional counselling.

Close monitoring of food intake and dietary restraint are important aspects of treatment for type 1 diabetes. Individuals are required to pay close attention to the food that
they eat and are advised to limit intake of certain types of high sugar foods in order to prevent blood glucose levels becoming too high (Franz et al., 2002). Additionally, at clinic appointments there is a focus on discussing weight and diet, and young people are often advised to meet with a dietician for dietary education and food planning. Daneman et al. (1998) propose that this focus on food and eating heightens an individual’s awareness of their own body weight and the influence that eating has on it. This may make them more vulnerable, firstly, to developing a preoccupation with eating, weight and shape, and secondly, to placing higher value on the importance of these things in evaluating their sense of worth.

1.5.1.3 Insulin omission.

The availability of insulin manipulation and omission as a unique weight loss strategy for those with type 1 diabetes has been discussed in section 1.4.3. Daneman et al. (1998) emphasise the importance of this in understanding the elevated levels of eating disturbances in this population. They propose that access to this method of weight control increases young people’s vulnerability to developing eating disturbances and employing inappropriate behaviours to influence their weight and/or shape.

1.5.1.4 Summary.

There are several factors related to the treatment of type 1 diabetes that may heighten vulnerability to developing subthreshold eating disturbances and EDs in this group. As outlined in the model described by Daneman et al. (1998), these factors must be considered in combination with other individual and systemic factors that are known to be associated with the development of ED symptomology in the general population. This is particularly important for the development of preventative and treatment approaches for eating disturbances in those with type 1 diabetes. A focus on food and eating, and management of this, is inherent in the pathology and treatment of the disorder and is therefore not currently a
changeable factor. Therefore, it is important to investigate other individual and interpersonal variables that may be amplified in this population in relation to ED symptoms, in order to guide future treatment approaches.

1.5.2 Cognitive-behavioural theories of eating disorders.

There are many psychological theories that attempt to explain the development and maintenance of EDs in the general population. It is widely accepted that the developmental pathway involves a complex interaction of biological, psychological, systemic and societal factors (Polivy & Herman, 2002; Stice, 2002). Cognitive-behavioural theories dominate current understanding of the psychopathological processes involved in the development and maintenance of BN and AN (Fairburn, Marcus, & Wilson, 1993). Theories have tended to focus on maintenance factors, although many of the identified processes are also thought to apply to the development of EDs (Fairburn et al., 2003).

Disorder specific models for AN and BN state that the core process involved in each disorder relates to an individual’s system for evaluating their self-worth (Fairburn, Cooper, & Cooper, 1986). The theories propose that ED symptoms develop, and are maintained by, an over-evaluation of the importance that weight, shape and eating have on self-worth. Therefore, individuals become preoccupied by a compulsive need to control their weight and subsequently develop a fear of losing control over eating and gaining weight. This results in the development of behavioural strategies, including restriction of food, body checking and the use of additional compensatory actions, e.g. excessive exercising, self-induced vomiting and laxative use. Those with AN present as significantly underweight as they engage in more restrictive behavioural strategies rather than episodes of over-eating followed by compensatory strategies (Section 1.4.1). Alternatively, the theory of BN describes a cycle of perceived failure and loss of control following a period of restriction, which triggers binge eating episodes and subsequent purging strategies (Hilbert & Tuschen-Caffier, 2007). Both
restrictive behaviours and binge-purge cycles further perpetuate the negative cycle as they “magnify patients’ concerns about their ability to control their eating, shape and weight” (Fairburn et al., 2003, p. 511).

The theory of BN is well supported by empirical and treatment-based evidence (Bryne & Mclean, 2002; Cooper & Steere, 1995; Fairburn, Peveler, Jones, Hope, & Doll, 1993; Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). However, treatment for BN underpinned by this theory is, at best, only effective for 50% of clients (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). In addition, research has demonstrated that outcomes for the treatment of AN using cognitive behavioural therapy (CBT) are poor in both adults and adolescents (Fairburn, 2005; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003). Fairburn et al. (2003) propose that this is not because the theory is invalid, but because it fails to account for additional crucial mechanisms that are integral in the development and maintenance of EDs. In response to this they developed a revised, and extended, conceptualisation of the cognitive behavioural theory of BN. This is known as the transdiagnostic theory of EDs (Fairburn et al., 2003).

1.5.3 Transdiagnostic theory of eating disorders.

The transdiagnostic theory extends disorder-specific models by making an assumption that all ED presentations are maintained by common underlying mechanisms (see Figure 2). They identified four mechanisms, namely, dysfunctional perfectionism, intolerance of strong emotions, a universal and unconditional low self-esteem, and interpersonal difficulties (labelled “life” in Figure 2). Each shall be briefly described below.

1.5.3.1 Clinical perfectionism.

Clinical perfectionism is defined as an unceasing, and all consuming, striving for achievement in a variety of life domains, but particularly in relation to weight and eating for individuals with an ED. This mechanism is closely associated with, and perpetuates, the core
psychopathology of an over-evaluation of eating, shape and weight. Individuals high in clinical perfectionism also have extreme fears of failing and tend to be highly self-critical (Shafran, Cooper, & Fairburn, 2002).

1.5.3.2 Mood intolerance.

Individuals with an ED may have difficulties in recognising and regulating mood states that are experienced as intense and intolerable. In order to manage the intensity of these feelings individuals engage in what Fairburn et al. (2003) term “dysfunctional mood modulatory behaviour”. These may include deliberate self-harm (Claes, Vandereycken, & Vertommen, 2001), use of psychoactive substances (Holderness, Brooks-Gunn, & Warren, 1994), and most commonly, binge eating, excessive exercising and self-induced vomiting. As these behaviours serve an important purpose in regulating mood, they can be very difficult to change.

Figure 2. Transdiagnostic theory of eating disorders (Fairburn et al., 2003).
1.5.3.3 Self-esteem.

Fairburn et al. (2003) differentiate between the core self-esteem described in their theory and negative evaluation based on failure to achieve weight related goals. They describe core self-esteem as global and pervasive, and an integral part of the individual’s self-identity. It is problematic as it creates a sense of hopelessness in individuals, and further propels them toward striving for control over their weight and shape, both of which make change difficult (Fairburn, Kirk, O’Connor, Anastasiades, & Cooper, 1987). The association between self-esteem and EDs in type 1 diabetes will be discussed in section 1.7.

1.5.3.4 Interpersonal difficulties.

Within the interpersonal difficulties category there is a particular emphasis on family and peer relationships. Research carried out in the general population suggests that familial influence on the development and maintenance of EDs can happen on two levels (Polivy & Herman, 2002), as described by the dual process model (Leung, Schwartzman, & Steiger, 1996). Firstly, through positive reinforcement of an individual’s slenderness (Branch & Eurman, 1980) and modelling of weight/shape concern (Smolak, Levine, & Schermer, 1999). Secondly, evidence links certain family dynamics to the presence of eating disturbances in individuals, including insecure attachment with parents (Ward, Ramsay, & Treasure, 2000), a critical and controlling family environment (Haworth-Hoeppner, 2000) and perceived low levels of parent caring (Haudek, Rorty, & Henker, 1999). Discussion of these factors in relation to ED in type 1 diabetes populations is provided in section 1.6.

1.5.3.5 Evaluation of the transdiagnostic theory.

A strength of the transdiagnostic theory is that it provides an explanation for the diagnostic migration and overlap often seen in people with ED symptoms (Eddy et al., 2002). CBT based on this approach has been found to significantly reduce ED symptoms for those with a diagnosis of an ED in a robust single-blind randomised controlled trial (Fairburn et al.,
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2009). The treatment was more effective for those with significant difficulties in one of the four domains, i.e. clinical perfectionism, mood intolerance, core low self-esteem or interpersonal difficulties, than for those with less complex ED presentations. In addition, the validity of the theory has been demonstrated by research groups separate from the original authors. For example, Hoiles, Egan and Kane (2012) recruited a non-clinical community sample of 224 females to examine whether the four mechanisms described by Fairburn et al. impacted, either directly or indirectly, on dietary restraint. Structural equation modelling revealed that the mechanisms did impact on dietary restraint indirectly through eating, shape and weight concern, as described in the theory.

However, this theory has been challenged by some who believe that there is sufficient empirical evidence to support the existence of separate EDs. For example, Birmingham, Touyz and Harbottle (2009) propose that a transdiagnostic approach is not warranted as evidence suggests significant differences between AN and BN in prevalence across age and geographical location, outcome and prognosis, and the biological expression of each disorder. Despite this, the theory seems to offer a helpful explanation of how certain individual and interpersonal factors may interact in the development and maintenance of EDs. As the transdiagnostic theory is based on research carried out in the general population and clinical ED samples, little is known about its applicability to type 1 diabetes populations; further exploration of this theory is warranted.

1.5.3.6 Summary.

Taken together, the diabetes-specific vulnerabilities described by Daneman et al. (1998) and the transdiagnostic theory of EDs offer a helpful insight into developmental and maintenance pathways for the presence of eating disturbances in those with type 1 diabetes. This current study is concerned specifically with investigating self-esteem and relationships
within the family system in relation to eating disturbances. Each of these factors shall be considered below.

1.6 The Role of the Family

1.6.1 Introduction.

Singular causal factors for EDs, both in the general population and in type 1 diabetes, cannot be identified (Walsh & Devlin, 1998). Amongst other factors, family environment and familial relationships play an important part in the development and maintenance of EDs in the general population (Polivy & Herman, 2002). This section begins with a description of the dual process family model as a framework for considering familial influence in relation to EDs (Section 1.5.3.4). The model describes two pathways through which family factors can influence the development of eating disturbances, both of which are considered in turn. For each pathway a brief overview of the relationship between family factors and EDs in the general population is presented. This is followed by a critical analysis of relevant investigations from the type 1 diabetes literature.

1.6.2 Dual process family model.

The dual process family model (Leung et al., 1996) outlines two key inter-related pathways through which an individual’s family context may increase their vulnerability to developing ED symptoms. The first pathway is concerned with a preoccupation with weight, shape and eating within the family. When high value is placed on these factors in a family system, associated behaviours, such as dieting, weight related self-criticism and celebration of thinness, may be modelled by parents to children and adolescents. This can lead to internalisation of these beliefs and behaviours by the young person, and a sense of inadequacy in relation to weight, shape and eating, which in turn can lead to development of body dissatisfaction and other ED symptoms (Field et al., 2001; Stice, 1998).
The second pathway is more subtle, and relates to problematic interpersonal relationships within the family, particularly in the parent-child relationship. Leung et al. (1996) propose that unhelpful ways of interacting within the family lead to lower child self-esteem, which is in turn a risk factor for the development of EDs; thus the influence of this pathway is indirect. The term problematic interpersonal relationships as described in the dual process model, is an umbrella term encompassing a range of difficulties. Lack of parent care, high levels of overprotection, and developmentally inappropriate expectations from parents are all examples of difficulties in the parent-child relationship (Le Grange, Lock, Loeb, & Nicholls, 2010). Both pathways have been investigated in the general population and in samples of adolescents with type 1 diabetes; the relevant literature shall be reviewed below.

1.6.3 Evidence for the dual process family model in the general population.

The impact of parental beliefs about their own weight and shape on adolescent expression of eating disturbances in the general population has been well documented. In Western society slenderness tends to be culturally desirable and is widely celebrated, idealised and internalised (Groesz, Levine, & Murnen, 2002; Stice, 2002). Investment in these cultural beliefs can be seen within family systems. Modelling is a process described in social learning theory, which refers to the process by which behaviours are learnt through observation of significant others (Bandura, 1969). It is hypothesised that parents who are invested in their own slimness and report higher levels of weight and shape concern, may model unhelpful eating attitudes and behaviours. However, there is inconsistent evidence in support of this hypothesis (Pike & Rodin, 1991; Thelen & Cormier, 1995). In a meta-analysis Stice (2002) proposed that these inconsistencies may be related to the operational definition of eating disturbances utilised in individual studies. Significant effects of modelling on child and adolescent eating disturbances have been demonstrated most consistently in prospective studies when eating disturbances have been defined in behavioural terms (e.g. binge eating;
Stice et al., 2002), compared to when attitudinal aspects have been investigated (e.g. body dissatisfaction; Byely, Archibald, Graber, & Brooks-Gunn, 2000).

In addition to modelling, research has also shown an association between parents’ direct negative communication about weight and shape and higher levels of adolescent eating disturbances (Cooley, Toray, Wang, & Valdez, 2008; Kluck, 2010). Smolak et al. (1999) recruited a sample of mothers \((n = 131)\), fathers \((n = 89)\) and adolescent children \((n = 131)\) to investigate the relative contribution of modelling of parents’ weight concerns and direct negative communication about their child’s weight. They found that the latter was more predictive of child weight concern and dieting than the former. A strength of this study was that gender differences were reported. The only significant predictor of frequency of weight loss attempts for boys was maternal negative comments \((R^2 = .55, F[1, 25] = 32.69, p < .0001)\). On the contrary, frequency of weight loss attempts for daughters was predicted by maternal negative weight comments, paternal complaints about his weight and paternal belief in dieting; together these factors explained 41% of the variance for daughters. However, the cross-sectional study design does not allow for conclusions to be drawn about the direction of the observed relationships. Furthermore, this study utilised non-standardised ad-hoc measures of parent weight concern and negative communication for which the reliability and validity is unknown.

In line with the second pathway described by Leung et al. (1996), there is evidence in the general population to indicate an association between problematic family functioning (e.g. high conflict, low cohesion, low expressiveness) and the emergence of ED symptoms (Brookings & Wilson, 1994; Felker & Stivers, 1994). For example, in a population study Berge et al. (2012) investigated the association between family factors and self-reported eating disturbances in a large sample of non-clinical adolescents \((N = 2793)\). Logistic regression analysis revealed that higher levels of family functioning, parental connectedness
and knowledge of adolescents’ whereabouts were associated with lower odds of adolescents engaging in ED behaviour. Interestingly, significant interaction effects revealed that the association between better family functioning and lower levels of ED behaviour was moderated by parental psychological control, where higher levels were more problematic. Psychological control refers to intrusive attempts to control the inner psychological and emotional world of another (Barber, 1996). Similar patterns have been found in clinical samples with AN and BN (Stern et al., 1989; Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003). However, the studies described are all limited by cross-sectional study designs. Meta-analysis has revealed that more robust prospective studies, e.g. Ball and Lee (2002), have failed to show significant causal pathways for general family dynamics in the development of EDs (Stice, 2002). Stice proposes that this may be because they increase vulnerability to eating disturbances in a more subtle and indirect manner. This explanation fits with Leung et al.’s hypothesis that the mechanism by which problematic family dynamics influence the development of eating disturbances happens indirectly through development of a poor sense of identity and self-esteem. This in turn may increase their vulnerability to developing eating disturbances, especially if combined with higher levels of parental weight and shape concern.

In addition to general family dynamics, specific types of parenting have been shown to be related to eating disturbances. Low levels of parent care (Swanson et al., 2010), high levels of parent overprotection (Deas, Power, Collin, Yellowlees, & Grierson, 2011) and critical parenting styles (Haworth-Hoeppner, 2000) have all been found to be associated with higher levels of AN and BN symptoms. For example, Turner, Rose and Cooper (2005) investigated the relationship between parent care and overprotection, as measured by the Parental Bonding Instrument (PBI; Parker, Tupling & Brown, 1979), and eating disturbances, as measured by the Eating Attitudes Test (EAT; Garner & Garfinkel, 1979), in a self-selected non-clinical sample of 367 adolescent females. Multiple regression analysis revealed that
both maternal and paternal care and maternal overprotection significantly predicted scores on the EAT ($F = 10.4, p < .0001, \beta = -.169/-1.43/1.150$). However, this predictor model only explained 10% of the variance and the contribution of individual factors is not reported. A strength of this study was that it made use of self-report measures evidenced to have good reliability and validity estimates (e.g. Garfinkel & Newman, 2001; Safford, Alloy, & Pieracci, 2007). Furthermore, it also considered an explanatory mechanism for the observed relationship, finding that core beliefs of defectiveness/shame and dependence/incompetence mediated the relationship between parent care and overprotection and eating disturbances.

Similar findings have been replicated in a clinical ED sample of 66 females (Jones, Leung, & Harris, 2006). Although the findings of this study cannot be generalised to male adolescents, it did consider the father-daughter dyad, in contrast to previous research that has traditionally focused on mother-daughter dyads.

Tata, Fox and Cooper (2001) investigated the associations between parent care and overprotection, as measured by the PBI, and eating disturbances in a non-clinical sample of males and females. In agreement with the studies described above, higher levels of parent overprotection were found to be associated with higher levels of eating disturbances in females. On the contrary, high parent overprotection was found to be significantly associated with low body dissatisfaction, but not eating disturbances in males. Tata et al. (2001) propose that this is because gender differences in the presentation of EDs mean that males are more likely to report engaging in excessive exercise rather than other disordered eating attitudes and behaviours.

In summary, evidence from the general population indicates that the potential influence of the family on increasing vulnerability to developing eating disturbances is multifaceted. Parents’ weight, shape and eating concerns play a role in the development of adolescent eating disturbances. This may occur through both modelling of unhelpful eating
behaviours and direct negative comments about weight; the evidence is stronger and more conclusive for the latter. In addition, both the general family climate and specific aspects of parenting style also appear to play a role. Gender of child and parent both appear to influence the relationships between these factors.

1.6.4 Pathway one in type 1 diabetes: Preoccupation with weight, shape and eating in the family.

Researchers have investigated the association between preoccupation with weight, shape and eating within the family and the presence of eating disturbances in adolescents with type 1 diabetes. Kichler et al. (2008) investigated negative parent communication directed at female adolescents about their weight and appearance using a questionnaire design. Negative communication (e.g. encouragement to diet) was found to be a significant predictor of eating disturbances in this group; hierarchical regression analysis revealed that adolescent body dissatisfaction moderated this relationship. However, these findings must be treated cautiously as a non-standardised ad-hoc questionnaire was used to measure negative parental communication (Negative Communication Form; Kichler & Crowther, 2003). This measure did not demonstrate good reliability data; an internal consistency estimate as low as .49 was reported by the authors. However, the measure is reported to have adequate convergent validity (Kichler et al., 2008). The Drive for Thinness, Bulimia and Body Dissatisfaction subscales of the Eating Disorder Inventory (EDI; Garner et al., 1983) were used as a measure of ED symptoms. The EDI is a 64-item likert style, standardised, self-report questionnaire measuring the attitudinal and behavioural characteristics of AN and BN; it has been shown to have good psychometric properties (Garner, 2004; Weber, Davis, & Mcphie, 2006). However, the psychometric properties for use in a type 1 diabetes adolescent population remain unknown. This is particularly important to consider as some of the items
may yield positive responses due to the type 1 diabetes treatment regime rather than underlying disordered eating attitudes and behaviours.

In addition to negative weight-related communication, maternal weight/shape concern has been found to significantly predict eating disturbances in adolescent daughters with type 1 diabetes (Maharaj et al., 2003). Adolescent participants were divided into non-disordered, moderately-disordered and highly-disordered eating groups based on their responses on the EDI and Diagnostic Survey for Eating Disorders self-report questionnaires (DSEDS; Johnson, 1985). Mothers of daughters in the moderate and highly disordered groups were more likely to binge eat \( (p = .04) \) and be on a diet \( (p = .05) \) than mothers of those in the non-disordered group. A strength of this study is that it considered the process by which maternal and adolescent eating disturbances were related. Regression analysis revealed that the relationship was mediated by poorer adolescent self-concept and impairment in the quality of the mother-daughter relationship, as measured by the Inventory of Parent and Peer Attachment (IPPA; Armsden & Greenberg, 1987). This indicates that, in this sample, the mechanism by which maternal eating disturbances influenced daughters’ eating disturbances may not be sufficiently explained by the process of modelling. The findings suggest that both the quality of relationship between mother and daughter, and daughters’ self-esteem, play an important role in explaining the observed association.

These findings were replicated by O’Brien, Dempster, Doherty, Carson and Bell (2011) in a sample of 60 mother-daughter dyads recruited through type 1 diabetes clinics. They found that 18% of the variance in daughters’ reported bulimia symptoms were predicted by their mothers’ own dieting and encouragement to diet \( (F[2, 57] = 6.138; p = .004) \). Interestingly, they also compared maternal reported eating disturbances with adolescent-perceived maternal eating disturbances. They found that the latter explained a greater proportion of the variance in adolescents’ body dissatisfaction (27%) compared to the former
(20%). This indicates that it may be the young person’s perception of their family context that is important, rather than other family members’ perceptions. Studies that rely solely on parental report may underestimate the association between parental and adolescent eating disturbances.

The studies described above are all limited by their cross-sectional designs and therefore no conclusions can be made about the direction of causality with regards to the reported associations. Colton et al. (2007) employed a one-year longitudinal design to investigate predictors of eating disturbances in a sample of 106 adolescent females with type 1 diabetes. Eating disturbances were assessed using the Child Eating Disorder Examination (cEDE; Bryant-Waugh, Cooper, Taylor, & Lask, 1996), which is a semi-structured interview that has good psychometric properties (Watkins, Frampton, Lask, & Bryant-Waugh, 2005). The Eating Attitudes Test self-report questionnaire was used as a measure of parental eating disturbance (EAT; Garner, Olmsted, Bohr, & Garfinkel, 1982). Stepwise linear regression analysis revealed a three factor model in which variables from Time One were found to predict eating disturbances in participants at Time Two. Adolescent self-worth, BMI and maternal attitudes toward eating together explained 35% of the variance in participants’ scores on the cEDE ($R^2 = .35; F = 18; p < .001$). Paternal eating attitudes did not significantly predict eating disturbances at one year. Interestingly, these findings are in contrast to those of a one year longitudinal study in the general population, which found dieting behaviour in adolescent males ($n = 5287$) and females ($n = 6770$) to be predicted by greater importance placed on thinness by fathers (Field et al., 2001). This contrast may be due to a lack of statistical power due to the relatively small sample size employed by Colton et al.

It is important to consider whether these findings can be generalised beyond parent-daughter dyads, as eating disturbances appear to be elevated in males with type 1 diabetes as well as females (Svensson et al., 2003). Currently there is only one published quantitative
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investigation to include both male and female participants (Smith et al., 2010). Correlation analysis revealed primary caregivers' weight ($r = .27, p < .05$) and shape ($r = .34, p < .01$) concern to be the only significant correlates of adolescent eating disturbances. However, the correlation between primary caregiver and adolescent weight concerns did not remain significant after controlling for parent and adolescent BMI. Furthermore, gender was not controlled for in this instance, so conclusions cannot be drawn about gender differences in the relationships.

In a qualitative study semi-structured interviews were carried out with 30 young females with type 1 diabetes, and their families (Mellin, Neumark-Sztainer, Patterson, & Sockaloskly, 2004). The sample was divided into those who reported eating disturbances ($n = 15$), and those who did not ($n = 15$). Content analysis revealed themes around meal time structure and family weight concern, with the latter being present in over 75% of the families. Parental negative comments about appearance and weight were more commonly described by participants reporting eating disordered behaviour than their matched comparisons. The authors made attempts to ensure reliability by checking for inter-rater reliability and co-rating 5/30 transcripts; agreement for broad themes was 90%. This qualitative analysis provides additional evidence that weight concern within the family system may be related to the presence of eating disturbances in adolescents with type 1 diabetes.

1.6.4.1 Summary of findings.

These results indicate that pathway one, as described by Leung et al. (1996) in the dual process family model, has some utility when applied to samples with type 1 diabetes. Familial interactions around eating, weight and shape appear to play an important role in the expression of eating disturbances. Some research has indicated a strong relationship between maternal eating disturbances and ED symptoms in daughters with type 1 diabetes. Additional interpersonal and individual factors, including the quality of the parent-child relationship and
adolescent self-concept, have been found to mediate the observed associations. To date, the
generalizability of these findings to male adolescents and fathers is unclear.

1.6.5 Pathway two in type 1 diabetes: Problematic familial relationships.

The second pathway of the dual process model described by Leung et al. (1996) is
less concerned with direct parental influences on young peoples’ vulnerability to developing
EDs, and is focused more broadly on the quality of relationships within the family system;
this can be conceptualised in many different ways. Studies have looked both broadly at the
association between family environment and eating disturbances, and more specifically at the
quality of the parent-adolescent relationship. Relevant literature shall be described and
evaluated below.

1.6.5.1 Family environment.

To date, three published papers have considered the role of the family environment as
either a correlate or a predictor of eating disturbances in young people with type 1 diabetes
(Maharaj, Rodin, Olmsted, & Daneman, 1998; Neumark-Sztainer et al., 2002; O’Brien et al.,
2011). All studies used the Family Environment Scale (FES; Moos & Moos, 1981), which is
a widely used self-report measure. The FES comprises 10 subscales, which measure three
underlying dimensions. These are: 1) interpersonal relationships (family cohesion,
expressiveness and conflict subscales); 2) system maintenance (organisation and control
subscales); and 3) personal growth (independence, activity-recreation, achievement,
intellectual-cultural orientation and moral-religious orientation subscales). Although the
authors report the FES to have good reliability and validity (Grotevant & Carlson, 1989),
there is disagreement about the internal consistency of the scale, with alpha coefficients
ranging from .31 to .72 in a large adolescent sample (Boyd, Gullone, Needleman, & Burt,
1997). Therefore, this must be taken into consideration when interpreting the literature
described below.
Maharaj et al. (1998) recruited 113 mother-daughter dyads to investigate the association between family functioning and eating disturbances at a single time-point. Self-report questionnaires were used to measure eating attitudes and behaviours and participants were subsequently divided into none, mildly or highly disturbed eating groups. The study yielded a significant multivariate group effect of both adolescent-perceived \( F(20, 192) = 1.65, p = .04 \) and mother-perceived \( F(20, 192) = 1.70, p = .04 \) family environment on level of eating disturbances (high, medium or none). Contrasts between the groups revealed a linear pattern of perceived family dysfunction. As the level of eating disturbance increased so did reports of poorer interpersonal relationships marked by low cohesion and high conflict, impaired personal growth and poorer system maintenance defined by less family organisation. However, although the authors did control for age of diabetes onset, which has been shown to be associated with eating disturbances in this population, they failed to account for BMI and weight perception, which are also significantly associated with eating disturbances (Neumark-Sztainer et al., 2002).

Neumark-Sztainer et al. (2002) extended these findings by investigating adolescent perceived family cohesion (subscale of the FES) in a cross-sectional study of males \((n = 73)\) and females \((n = 70)\) with type 1 diabetes. They found cohesion to be significantly negatively correlated with eating disturbances in both females \((r = -.52, p < .001)\) and males \((r = -.41, p < 0.001)\). No significant associations were found with other subscales of the FES. These findings partly agree with those reported by Maharaj et al. (1998). This mixed picture might be explained by the relatively small sample size and subsequent lack of power to detect more subtle effects in Neumark-Sztainer et al. Furthermore, the sample was self-selecting which may have introduced a response bias that skewed the data in the latter study. The authors reported that higher blood glucose levels were found in non-responders compared to those that participated in the study. This may indicate that those with more significant eating
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disturbances chose not to participate in the study. However, a strength of this study is that it included both male and female participants and that separate analyses were carried out based on gender, in order that gender-specific patterns could be analysed. The study by O’Brien et al. (2011) found a similar mixed picture in their correlational analysis of the associations between subscales of the FES and self-reported eating disturbances in 60 mother-daughter dyads recruited from an adolescent diabetes clinic. However, these outcomes should be treated with caution due to the large number of correlations made in this study and the subsequent increased risk of a type 1 error occurring (Cook & Campbell, 1979).

**1.6.5.2 Parent-adolescent relationship.**

More attention in the literature has been given to investigating the role of the parent-adolescent relationship in relation to eating disturbances in adolescents with type 1 diabetes, than other familial influences. Several studies have investigated specific dimensions of the parent-adolescent relationship. The domains studied include attachment to parents, autonomy and intimacy in the mother-daughter relationship, negative communication and parental criticism. Relevant literature in relation to this shall be considered below.

The role of parent-child attachment has been investigated in relation to eating disturbances in adolescent females with type 1 diabetes (Colton et al., 2007; Maharaj et al., 1998; Maharaj et al., 2003; Olmsted, Rydall, Colton, Rodin, & Daneman, 2008). All studies used the Inventory of Parent and Peer Attachment to measure attachment (IPPA; Armsden & Greenberg, 1987), which has demonstrated good psychometric data (Gullone & Robinson, 2005). Each study used only the scales that related to adolescent report of current attachment to parents. Maharaj et al. (1998) found significant group effects when comparing reports of parent attachment across groups of female adolescents reporting no eating disturbances ($n = 56$), mild eating disturbances ($n = 37$) and high levels of eating disturbances ($n = 20$). The results indicated a linear pattern, as females with mild and high levels of eating disturbances
reported less trust ($F_{[2, 105]} = 4.29; p = .01$), greater alienation ($F_{[2, 105]} = 7.39; p = .001$),
less acceptance ($F_{[2, 105]} = 3.25; p = .04$) and poorer communication ($F_{[2, 105]} = 9.16; p = .0001$) in their relationship with their mother; similar patterns were found for attachment to fathers. These findings have been replicated by Maharaj et al. (2003) in a sample of 88 mother-daughter dyads. This suggests that females who perceive their attachment relationship with their parents as lacking in closeness, warmth, trust and positive communication may be more vulnerable to developing eating disturbances. Longitudinal studies are required to confirm these directional hypotheses.

Colton et al. (2007) used a longitudinal design to investigate whether adolescent-perceived parent attachment, amongst other factors, predicted eating disturbances in females with type 1 diabetes. They found that attachment to mother ($r = -.29, p = .003$) and father ($r = -.39, p < .001$) at Time One, as measured on the IPPA, was significantly associated with eating disturbances one year later. Additionally, attachment to mother at baseline explained 15% of the variance in predicting new-onset eating disturbances at Time Two. This sample was followed up at five years (Olmsted et al., 2008), at which point adolescent-perceived attachment to mothers ($X^2 = 6.03 , 1 \text{ d.f.}, p < .02$) and to fathers ($X^2 = 3.91 , 1 \text{ d.f.}, p < .05$) were found to be significantly associated with onset of eating disturbances in this group. However, neither added any additional predictive power to the regression model after BMI, weight and shape concern, physical appearance, self-worth and depression had been added. The lack of predictive value of parent attachment on eating disturbances shown here is consistent with findings in the general population (Jacobi, Hayward, Zwaan, Kraemer, & Agras, 2004). This indicates that in this sample, poorer adolescent-perceived attachment to parents predicted new onset of eating disturbances over time, but that this predictive power was lost when other individual factors were entered into the model alongside this variable. This may be a result of the high level of shared variance between adolescent attachment to
parents and individual factors such as self-worth and depression. Furthermore, the IPPA is a measure of general attachment to parents as perceived by the adolescent at the time of assessment, measures of more specific parenting dimensions may yield more predictive power; further investigation is warranted.

In addition to attachment, the negotiation of successful individuation between mother and daughter has been investigated in relation to the expression of eating disturbances in type 1 diabetes. Individuation is a term used to describe the developmental challenge of reducing dependency on parents and increasing independence throughout adolescence (Section 1.3.2.2.). Two cross-sectional studies have found eating disturbances in female adolescents to be negatively associated with autonomy and intimacy (individuation) in the mother-daughter relationship (Maharaj, Rodin, Connolly, Olmsted, & Daneman, 2001; Maharaj et al., 2003). This was measured using the Autonomy and Intimacy Rating System (AIRS; Maharaj, Connolly, & Becker, 1996), which is an observational method designed to evaluate parent-adolescent communication in the negotiation of autonomy and intimacy around a problem solving task. The authors reported acceptable levels of inter-rater reliability (average Cohen's $K = .80$). Both studies found significant differences in mothers’ facilitation of, and adolescent expression of, autonomy and intimacy between none, mildly and highly eating disordered groups of females with type 1 diabetes. Mothers of daughters in the highly eating disordered group were observed to be less supportive of their daughters’ own views ($p = .0005$), less open to their daughter ($p = .002$) and more constraining ($p = .0005$). This effect was more pronounced when the problem solving task was diabetes-related rather than focused on more general issues. The authors propose that maternal difficulties in supporting their daughters to successfully negotiate the developmental task of individuation hinders their emerging sense of self, which may make them more vulnerable to developing ED symptoms. Currently the
generalisability of these findings to fathers and male adolescents is unknown, as is the direction of causality.

One published paper has reported the outcomes of a study that considered the parent-child relationship in both male and females with type 1 diabetes. Associations between parental criticism, as measured by the Five-Minute Speech Sample (Daley, Sonuga-Barke, & Thompson, 2003), and eating disturbances, measured by the Eating Disorder Examination (EDE; Cooper & Fairburn, 1987), were investigated in a sample of 50 adolescent-parent dyads recruited through a type 1 diabetes clinic in the UK (Smith et al., 2009). No significant associations were found between the variables. However, this study was looking at general parental criticism as opposed to weight-related criticism, which may be more closely associated with the expression of eating disturbances. Furthermore, the small sample size meant that this study had only 50% statistical power to detect medium effects.

1.6.5.3 The role of parenting style.

Research in type 1 diabetes populations has been limited to considering attachment style, parental criticism and parent facilitation of autonomy and intimacy. Another helpful way to conceptualise the parent-child relationship is to consider the role of parenting style. In the general population there is a strong body of research that points to a relationship between parenting style and the development of eating disturbances (Section 1.6.3).

There are many ways to conceptualise parenting style, including dimensional and categorical approaches (e.g. Baumrind, 1966), as well as focusing predominantly on behavioural, attitudinal or emotional aspects. Parker et al. (1979) offer a helpful dimensional perspective concerned with the emotional context surrounding the parent-child bond. Based on early factor analytic investigations of parenting attributes they proposed parent care, as opposed to rejection, and parent overprotection, as opposed to autonomy, to be two key parenting dimensions implicated in the development and maintenance of a healthy parent-
child relationship (Parker et al., 1979). There is a wealth of research pointing to the important role that these parenting dimensions play in enabling children and adolescents to become well-adjusted individuals (Kashani, Ezpeleta, Dandoy & Reid, 1991; Lamb & Lewis, 2011). For example, in a community sample of over 2000 adolescents low paternal care was found to be a significant predictor of poor body image and higher levels of neuroticism (Cubis, Lewin & Dawes, 1989). This literature has been extended to ED populations and findings suggest that young people with EDs perceive their parents to be less caring and more controlling/overprotective (Deas et al., 2011). To date there is no published research looking at parent care or overprotection in relation to eating disturbances in a type 1 diabetes sample.

This is an important area to investigate as research suggests that parenting dimensions, such as care and overprotection, may be impacted by the additional challenges faced in parenting children with chronic illnesses (Spurrier et al., 2000). A recent meta-analysis of 325 published and unpublished cross-sectional studies was carried out to compare certain dimensions of the parent-child relationship in children with and without chronic illnesses (Pinquart, 2013). The findings suggest a small-medium effect size ($g = .39$, $z = 7.24$, $p < .001$) for higher levels of parent overprotection and a small effect size ($g = -.22$, $z = -6.55$, $p < .001$) for lower levels of parent care and warmth in children and adolescents with chronic illnesses compared to healthy controls. These findings may indicate that while most families adapt well to the challenge of childhood chronic illness, some parents may struggle to negotiate healthy levels of warmth and autonomy/protection in their parenting. Pinquart suggests that this may be due to a number of factors including stress related to increased parenting responsibilities (Drotar, 1992), distress related to feelings of being overwhelmed, disappointed, depressed and angry (Power & Franck, 2008), and the demanding nature of behavioural problems associated with childhood chronic illness (Pinquart & Shen, 2011). However, it is important to note that this was a meta-analysis of cross-sectional studies and
therefore conclusions about causal relationships between childhood chronic illness and changes in parent care and overprotection cannot be made. Furthermore, it is likely that the association between the variables is reciprocal in nature, with parenting style potentially influencing adaptation to childhood chronic illness as well as being influenced by onset of chronic illness. Despite this, the investigation of childhood psychopathologies associated with lower levels of parent care and higher levels of parent overprotection, including eating disturbances, is warranted.

Parent care and overprotection have been investigated specifically in samples of young people with type 1 diabetes. The two dimensions were compared in a questionnaire-based study between a sample of adolescents with type 1 diabetes (n = 115), physical disabilities (n = 291) and healthy controls (n = 9345; Graue, Wentzel-Larsen, Hanestad, & Sovik, 2005). They found that the type 1 diabetes group reported higher levels of parent overprotection compared to both the healthy controls and those with a physical disability. Graue et al. (2005) propose that this is likely related to the intensive daily management required to prevent ill health in type 1 diabetes. It may be that parent overprotection is influenced, in part, by chronicity and intensity of the illness. No significant between group differences were found for level of parent care. However, contrary to these findings, Hullmann et al. (2010) found no differences in levels of parent overprotection in parents of children with type 1 diabetes (n = 149), asthma (n = 100), cancer (n = 115) and cystic fibrosis (n = 61). These differences may be accounted for by the measure utilised to assess overprotection. Graue et al. used the Parental Bonding Instrument (PBI; Parker et al., 1979), which is an adolescent-report of parent overprotection, whereas Hullmann et al. made use of the Parent Protection Scale (PPS; Thomasgard, Metz, Edelbrock, & Shonkoff, 1995), which is a parent-rated measure. Further, the lack of a healthy comparison group in the latter study
precludes conclusions being drawn about whether or not parent overprotection was elevated across all illness groups.

Cameron, Young and Wiebe (2007) found maternal overprotection to be significantly correlated ($r = .35$, $p < .01$) with maternal anxiety in a sample of adolescents with type 1 diabetes ($n = 59$) and their mothers ($n = 47$). However, although this study utilised a well standardised and psychometrically evidenced measure of anxiety (State-Trait Anxiety Inventory; Spielberger, Gorusch, & Lushene, 1974), an ad-hoc measure of adolescent-perceived parent overprotection was utilised and the reliability and validity of the measure remain unknown. However, the results do tentatively indicate an association between maternal anxiety and adolescent perceived maternal overprotectiveness in a sample of young people with type 1 diabetes. It is possible that heightened levels of overprotection and control may partly explain the elevated levels of eating disturbances seen in type 1 diabetes populations. Further research in this area is needed.

1.6.5.4 Evaluation and summary.

In line with findings from the general population, certain aspects of the family environment appear to be related to eating disturbances in both male and female adolescents with type 1 diabetes. However, mixed results have been found regarding the significance of each individual family dimension measured by the Family Environment Scale. Family cohesion is the only factor to show a consistent association with eating disturbances across the three studies. Furthermore, the investigations are limited by a lack of longitudinal design. No conclusions can be drawn about the causal nature of the association between aspects of the family environment and eating disturbances in this group. When considering the influence of the parent-child relationship, research has predominantly focused on mother-daughter dyads. Poorer parental attachment, a lack of intimacy and lower levels of autonomy-granting
have been shown to be significantly associated with eating disturbances in females with type 1 diabetes.

The studies reported here are limited by a consistent failure to employ a comparison group in the study design. Given this methodological limitation, it is not possible to draw conclusions about whether the associations identified between family factors and eating disturbances are unique to type 1 diabetes populations, or whether such associations would also be present in other chronic illness populations. It is important to determine the specificity of such associations for two reasons. Firstly, evidence suggests that EDs and subthreshold eating disturbances may be more prevalent among young people with type 1 diabetes than other chronic illness groups. Therefore, in order to make sense of this higher prevalence rate it is necessary to identify factors that may increase vulnerability to eating disturbances specifically in young people with type 1 diabetes. Without a comparison group it is not possible to draw conclusions about this. Secondly, a lack of specificity makes the challenge of developing appropriate, effectively targeted interventions more difficult.

1.7 The Role of Self-Esteem

1.7.1 Introduction and overview.

Low self-esteem has consistently been shown to be associated with, and predictive of, the development of EDs in the general population (Polivy & Herman, 2002). Fairburn et al. (2003) identify self-esteem as one of the key factors in their transdiagnostic theory of EDs (Section 1.5.3). Research has shown that adolescents with type 1 diabetes have lower levels of self-esteem in comparison to their healthy peers (Seigel, Golden, Gough, Lashley, & Sacker, 1990). Therefore, an exploration of the role of self-esteem in relation to eating disturbances in young people with type 1 diabetes is warranted. This section will begin by defining self-esteem and will then describe and evaluate relevant literature.

1.7.2 Definition of self-esteem.
Self-esteem is a concept that has been widely researched and theorised about in psychological literature. There are many approaches to defining and understanding it as a human phenomenon (Mruk, 2006). One of the most useful organisations of the differing definitions was proposed by Wells and Marwell (1976). They noticed that self-esteem definitions broadly fitted into one of two key psychological processes. The first is cognitive in nature, and describes the positive and negative evaluative thoughts people have about themselves; this is now often referred to as self-perception. The second relies more strongly on affective components, and refers to the positive and negative feelings humans have in association with their own self-worth.

Rosenberg (1965) was an early pioneer in the measurement and study of self-esteem. He defined it as one’s perception of one’s own feelings about one’s worthiness. Some have criticised the simplicity and uni-dimensional nature of Rosenberg’s definition (Mruk, 2006). Despite this, it remains one of the most commonly used operational definitions. This is likely a result of the availability of Rosenberg’s simple, brief, self-report questionnaire for measuring global self-esteem. More recent developments in this area propose that the most acceptable current definition of self-esteem is one that combines both an affective worthiness component (i.e. feeling good about one’s self) and more cognitive-based evaluation of competency (Mruk, 2006). However, for the purposes of this study, Rosenberg’s definition of self-esteem will be utilised. This is because the core self-esteem component outlined in the transdiagnostic theory is predominantly concerned with a global sense of one’s own worth and value, which is congruent with Rosenberg’s definition. Additionally, the availability of the Rosenberg Self Esteem Scale (RSES; Rosenberg, 1965) as a brief, free measure of self-esteem addresses the practical limitations of the current study. However, the limitations of such an approach will be taken into consideration in interpreting the findings.

1.7.3 Self-esteem and eating disorders: Evidence from the general population.
Low self-esteem has consistently been found to be associated with, and predictive of, ED symptomology in both male and females in the general population (Courtney, Gamboz, & Johnson, 2008; Polivy & Herman, 2002; Olivardia, Pope, Borowiecki, & Cohane, 2004; Ricciardelli & McCabe, 2004; Stice et al., 2002). For example, a prospective study found self-esteem, as measured by the RSES, at age 11-12 years significantly predicted onset of ED symptoms at age 15-16 years in schoolgirls \( n = 594 \) (Button, Sonuga-Barke, Davies, & Thompson, 1996).

As described in the transdiagnostic model of EDs (Fairburn et al., 2003), low self-esteem interacts with other individual and interpersonal factors in the development and maintenance of ED symptomology. It has been shown to moderate the predictive relationship between perfectionism and BN (Bardone, Vohs, Abramson, Heatherton, & Joiner, 2000). Further support for the importance of self-esteem in the developmental pathway for EDs is found in the evaluation of ED prevention programs. For example, O’Dea and Abraham (2000) describe a preventative approach that focused on increasing self-esteem in adolescents identified as being at risk of developing an ED in the future. They found the approach to be effective in reducing ED symptoms at 1 year follow-up for both male and females. This provides further evidence for the role of low self-esteem not only in the maintenance of the disorder, but also in its development.

1.7.4 Self-esteem, eating disturbances and type 1 diabetes.

The literature concerned with investigating self-esteem as a factor associated with, and predictive of, eating disturbances in young people with type 1 diabetes is inconclusive. Maharaj et al. (2003) found a significant association between low self-esteem and eating disturbances in a sample of 88 adolescent females with type 1 diabetes. This study employed the Self-Perception Profile for Adolescents (SPPA; Harter, 1988) as a multidimensional measure of both the cognitive and affective components of self-esteem. This measure has
demonstrated good internal consistency and test re-test reliability estimates (Muris, Meesters, & Fijen, 2003). A further strength of the questionnaire is that it was designed specifically for use with adolescents, and asks about domains of life that are particularly pertinent during adolescence, e.g. physical appearance and social competence. The results of this cross-sectional study found a significant association between self-esteem and eating disturbances. More specifically, a significant multivariate effect of self-esteem was found for presence of eating disturbances ($F[18, 124] = 3.08; p < .000$). Those who reported either mild or high levels of eating disturbances reported poorer self-esteem in relation to their physical appearance ($p < .000$), behaviour conduct ($p = .001$) and global self-worth ($p < .000$). These findings are congruent with evidence from the general population (Polivy & Herman, 2002) and psychological theories of the development and maintenance of EDs (e.g. Fairburn et al., 2003).

Vila et al. (1995) recruited two groups of adolescent females, those with type 1 diabetes ($n = 52$) and those without ($n = 46$). In contrast to the above study, they found no significant associations between self-esteem and eating disturbances for either group. For this study the Coopersmith Self-Esteem Inventory was used (Coopersmith, 1967). It is a 50 item self-report questionnaire designed to measure attitudes about the self, both globally and in different domains of life, e.g. *I am pretty sure of myself*. The questionnaire has demonstrated good internal consistency (Blascovich & Tomaka, 1991), but the construct validity of the measure has been questioned (Ahmed, Valliant, & Swindle, 1985). The lack of agreement between these studies may be accounted for by the different self-esteem measures used. Given the breadth of operational definitions available in the measurement of self-esteem, it may be that the SPPA and the Coopersmith Self-esteem inventory measure different dimensions of self-esteem, which are differentially associated with EDs.
The research studies described above are limited by their cross-sectional designs and subsequent inability to infer about the direction of causality in the relationship. More methodologically robust longitudinal research provides support for the role of low self-esteem in the development of eating disturbances in young females with type 1 diabetes. Colton et al. (2007) found that baseline self-esteem, as measured by the global self-worth subscale of the SPPA, significantly correlated with scores on the ED measure used (cEDE) at one year follow up ($r = -.47, p < .001$). Baseline global self-worth was shown to explain 15% of the variance in eating disturbances at Time Two when entered into a linear regression one-factor model ($R^2 = .15; F = 23; p = < .001$). In addition, Olmsted et al. (2008) investigated predictors of new onset eating disturbances in the same sample. In order to attain this, those who reported EDs or subthreshold ED symptoms at baseline were excluded from the data set. Regression analysis found global self-worth and self-esteem related to physical appearance significantly predicted the development of onset of new eating disturbances. Combined with BMI, weight and shape concern and scores on a depression measure, self-worth and physical appearance accounted for 48.2% of the variance in new onset eating disturbances.

This longitudinal data provides a strong case for the role of self-esteem, both globally and in relation to physical appearance, in the development of eating disturbances in females with type 1 diabetes. However, research to date has failed to look at the role of self-esteem in predicting eating disturbances in adolescent males with type 1 diabetes. In addition, other than Vila et al. (1995), there is a lack of comparison groups within the study designs to enable more robust conclusions to be drawn about the proposed risk factors for eating disturbances in this population. Therefore, the current study aims to employ a comparison group of young people with a diagnosis of asthma. The rationale and supporting evidence for this will now be described.

1.8 Asthma: A Comparison Group
1.8.1 Prevalence, pathology and treatment of asthma.

Asthma is a chronic upper respiratory disease that is characterised by chronic inflammation of the airways (Gibson, Henry, Vimpani, & Halliday, 1995). Despite being one of the most common illnesses present in children and adolescents in Western society (Ortega, Huertas, Canino, Ramirez, & Rubio-Stipec, 2002), it can be a severe illness that significantly impacts on individuals’ functioning (Rabe, Vermeire, Soriano, & Maier, 2000). In the UK recent estimates indicate that around one in ten children in the UK are affected, which amounts to around 800,000 adolescents (Asthma UK, 2006; Couriel, 2003). The disorder is more common in early childhood, and about one third experience spontaneous remission from their asthma symptoms during puberty (Seiffge-Krenke, 2001). This means that two thirds of those with a diagnosis of asthma continue to experience symptoms beyond puberty and into adulthood. Adolescents experience more severe exacerbations of their asthma symptoms and more subsequent hospital admissions than their younger peers (Bruzzese et al., 2011).

Symptoms of asthma include wheezing, coughing, experiencing a tight feeling in the chest and shortness of breath. They are triggered by contact with an irritant (e.g. smoke, dust, cold air) that causes the muscles in the airway to constrict and become inflamed, which makes it more difficult for air to pass through to the lungs. The causes of asthma in childhood remain relatively unknown, although genetic factors and biological vulnerability to allergies are thought to play a part (Gibson et al., 1995). There is currently no cure for asthma, and therefore the condition requires regular monitoring and management of symptoms. The focus of asthma treatment is on preventing and suppressing inflammation through the use of inhaled steroid medications. Individuals with asthma are often required to take preventative medication on a daily basis as well as monitoring their symptoms and responding with the appropriate medication when symptoms are exacerbated (Asthma UK, 2009).
1.8.2 Impact of asthma in childhood and adolescence.

Asthma can significantly impact on the physical, social and emotional well-being of children and adolescents (Snipes, 2013). Research carried out in the United States has found that young people with asthma have more days absent from school due to ill health than their healthy peers (Fowler, Davenport, & Garg, 1992; Snipes, 2013). This absenteeism in turn seems to negatively impact on academic performance (Moonie, Sterling, Figgs, & Castro, 2008). A community survey has shown that adolescents with asthma report a poorer quality of life than those without (Gibson et al., 1995). Further, research indicates that parents of children with asthma may also be negatively affected, with some studies reporting higher levels of parenting stress compared to other chronic illness groups (Hullmann et al., 2010). Higher levels of parent overprotection have also been demonstrated in asthma groups. For example, Parker and Lipscombe (1979) found significantly higher levels of child-perceived parent overprotection, as measured using the PBI, in a group of adult children with asthma (n = 50), compared to gender matched controls (n = 50). This indicates that childhood asthma not only impacts on the individual but may also impact on their parent(s).

Furthermore, those with the condition may find their ability to engage in physical activity to be limited. Evidence suggests that young people with asthma engage in significantly fewer physical activities than their healthy peers (Glazebrook et al., 2006). Additionally, on average they tend to have a higher BMI than their healthy peers (Tantisira & Weiss, 2001). Research suggests that higher BMI may both be a precursor to asthma onset (Beuther, 2010) and a consequence related to limited ability to engage in physical activity and weight-gain side effects of steroid medication, which is the treatment of choice to assist opening of the airways (National Heart, Lung & Blood Institute, 2002). The impact of asthma on emotional well-being will now be discussed.
1.8.3 Psychopathology in asthma.

The emotional well-being of adolescents with asthma is a relatively under-researched area. However, research does suggest that there may be an increased risk of psychiatric disorder in asthma populations. For example, a community based study in America found that young people aged 9 – 17 years old with a diagnosis of asthma \( n = 199 \) were more likely to experience significant levels of anxiety than their healthy peers \( n = 1096 \); Ortega et al., 2002). Anxiety was measured using the Diagnostic Interview Schedule for Children (DISC 2.3; Shaffer et al., 1996), which is a robust interview-based measure that has been found to be reliable and valid for use with adolescents (Shaffer et al., 1996). Interestingly, the study showed that those with other chronic illness diagnoses were more likely to report affective symptoms than anxiety symptoms, compared with the asthma group. Additional findings within the literature are mixed, with some replicating this association between asthma and anxiety (e.g. Silverglade, Tosi, Wise, & D’Costa, 1994), and others not (e.g. Gupta, Mitchell, Giuffre, & Crawford, 2002).

Further evidence comes from a cross-sectional study which recruited a sample of 82 adolescents with asthma and 82 healthy controls, who were matched based on gender, age and socio-economic status (Vila, Nollet-Clemencon, Blic, Mouren-Simeoni, & Scheinmann, 2000). Each participant completed a battery of self-report questionnaires and the Kiddie Schedule for Affective Disorders and Schizophrenia (Birmingham et al., 2009), which was used to diagnosis the presence of psychiatric disorders. The authors found that 36\% \( n = 29 \) of the asthma group reported symptoms that indicated the presence of an anxiety disorder, with Generalized Anxiety Disorder being the most common diagnosis \( n = 24 \). Participants within this subgroup of adolescents with asthma and a co-morbid anxiety disorder also reported lower self-esteem in comparison to those with asthma but no anxiety disorder and healthy controls. There were no differences found between males and females with asthma.
on the measures of anxiety and self-esteem. These results indicate that, in this sample, participants with a diagnosis of asthma were more likely to report clinical levels of anxiety and low self-esteem. Further weight is added to this argument as a recent meta-analysis revealed significantly higher levels of depression and anxiety amongst adolescents with asthma compared to healthy peers (Lu et al., 2012).

Evidence suggests that it is not only young people with asthma that are vulnerable to developing low self-esteem or poor self-concept, but young people with chronic illnesses more broadly. Seigel et al. (1990) compared a group of 80 adolescents with chronic illness with 100 healthy controls matched by age and socio-economic status. The chronic illness group was made up of sickle cell disease \( (n = 20) \), asthma \( (n = 40) \) and diabetes \( (n = 20) \). Those with chronic illness had lower self-esteem \( (p < .001) \), as measured by the RSES, and higher depression \( (p < .001) \) compared to their healthy peers. No differences were observed between the three illness groups. Similarly, a large community sample of adolescents with heterogeneous chronic illness \( (n = 1683) \) have been shown to report lower emotional well-being and poorer body image than healthy controls \( (n = 1650; \) Wolman, Resnick, Harris, & Blum, 1994). Interestingly, within the chronic illness group no differences in emotional well-being and body image were found for those with and without visible conditions. A recent meta-analysis of 621 studies added substantial weight to these findings by concluding that young people with chronic illnesses do have reduced self-esteem compared to their healthy peers, and that this is most pronounced for females, rather than males, and adolescents, rather than children (Pinquart, 2012). Taken together these findings indicate that vulnerability to psychopathology may be related to common aspects of heterogeneous chronic illnesses, such as the psychosocial and emotional burden of living with a chronic illness, as opposed to something specifically related to asthma or type 1 diabetes. Therefore, this supports the rationale for including this group as a comparison group in the current study.
1.8.3.1 Eating disorders in adolescents with asthma.

There are no published studies to date that have investigated the presence of eating disturbances in young people with a diagnosis of asthma using standardised measures. Eating behaviours and obesity have been investigated in a population-based sample of French adolescents ($n = 11,700$), of which 11.7% reported having had a diagnosis of asthma at some point over their lifetime (Moreau, Kalaboka, Choquet, & Annesi-Maesano, 2009). Disturbances in eating behaviours were assessed using five questions derived from the DSM-IV (American Psychiatric Association, 2000) criteria for EDs, e.g. Are you afraid of gaining weight? Participants were given four response options ranging from never to quite often. The findings showed that the asthma group reported significantly more eating disturbances, including being less likely to eat for pleasure, more likely to skip meals and diet, and more concerned about their weight, than those without asthma. The study also showed that adolescents with asthma were more likely to be overweight and to have experienced weight changes over the past year; those who were both asthmatic and overweight were the most likely to report eating disturbances on the ad-hoc measure. Moreau et al. attribute the association between asthma and eating disturbances to a combination of biological vulnerabilities and the high comorbidity of anxiety and depression evidenced in those with asthma. However, these findings must be treated cautiously as the reliability of the measure of eating disturbances is unknown.

Kelsay, Nicholas and Wamboldt (2005) investigated predictors of body dissatisfaction in a sample of male ($n = 60$) and female ($n = 63$) adolescents with asthma. They used the Colour-a-Person Body Dissatisfaction Test, which is has been shown to have good psychometric properties (Wooley & Roll, 1991). They found BMI to be a significant predictor of body dissatisfaction along with anxiety and depression. In combination these three predictors accounted for 21% of the variance in body dissatisfaction in females, and
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15% in males. Furthermore, after accounting for BMI, lack of physical activity was also significantly associated with body dissatisfaction in females but not males. Therefore, higher BMI and limited physical activity appear to increase vulnerability to developing body dissatisfaction in this group. Body dissatisfaction has been shown to be one of the most significant risk factors for the development of further ED symptoms in the general population (Stice, Marti, & Durant, 2011). However, the lack of comparison group in this study means that it is not possible to draw conclusions about whether or not body dissatisfaction in this sample of adolescents with asthma was elevated in comparison to either their healthy peers or other chronic illness groups.

1.8.4 Summary.

In summary, preliminary evidence indicates that young people with asthma are at a heightened risk of having low self-esteem, a high BMI, and experiencing anxiety. All of these factors have been shown to be implicated in the development of eating disturbances in the general population. Further, preliminary evidence indicates that they may also be at an increased risk of eating disordered behaviours and body dissatisfaction. However, this research is methodologically weak due to failure to either use a standardised, reliable measure of ED symptomology or make use of a comparison group. The presence of eating disturbances in this group remains an under researched area, and therefore, the investigation of eating disturbances in this group would appear to be logical and is warranted.

1.9 Rationale for Proposed Study

“The chronicity of the illness and the impact that it has on the child, his parents, and his siblings is more significant than the specific character of the disorders, be it diabetes, cerebral palsy, haemophilia, etc. In other words there are certain problems common to all illnesses over and above particular changes posed by individual need.” (Pless & Pinkerton, 1975, pp. 52)
There is general consensus within the literature that eating disturbances are more prevalent in adolescents with type 1 diabetes compared to their healthy peers. Research about prevalence rates in comparison to other chronic illness groups remain inconclusive. In addition to the impact on emotional wellbeing, ED symptoms are particularly problematic in those with type 1 diabetes due to the impact of such symptoms on treatment adherence and physical wellbeing. Research in this area has found lower self-esteem and more problematic relationships with parents to be associated with, and predictive of, eating disturbances. However, the literature is methodologically limited due to a persistent lack of a comparison group. This means that it is unclear whether the associations found between individual and familial factors and eating disturbances are unique to type 1 diabetes, or are a result of vulnerabilities common to a broader range of childhood chronic illnesses.

Childhood and adolescent chronic illness creates exceptional circumstances for young people and their families to navigate through and adapt to. As such, negotiation of normal developmental tasks for the individual and their family can be impaired. Lubkin and Larsen (2006) list stigma, adaptation, social isolation, body image, quality of life, sexuality, family relationships, powerlessness and impact on the self, amongst other factors, as domains that may be affected by the presence of a chronic illness in childhood. Research has shown that adolescents experiencing chronic illness, across diagnoses, may be vulnerable to difficulties in these domains (Cadman, Boyle, Sztamari, & Offord, 1987; Hamlett, Pellegrini, & Katz, 1992; Neumark-Sztainer et al., 1995; Sawyer, et al., 2004; Zeltzer, Kellerman, Ellenber, Dash, & Rigler, 1980). In particular, adolescents with heterogenous chronic illnesses have been found to have poorer self-esteem (e.g. Pinquart, 2012). Furthermore, parents of children with chronic illnesses report high levels of parenting stress and more overprotective parenting styles (e.g. Hullmann et al., 2010; Streisand, et al., 2005). Therefore, evidence suggests that low self-esteem and difficulties in the parent-child relationship may be amplified in
heterogenous chronic illness groups, which may in turn increase vulnerability to eating disturbances.

In light of this, the study reported here aims to investigate the role of self-esteem and parenting factors in relation to eating disturbances in those with type 1 diabetes compared to those with asthma. A comparison group of young people with asthma has been chosen for two reasons. Firstly, consideration was given to including a comparison group with similar illness characteristics to those of type 1 diabetes (e.g. onset in childhood, requires daily management), in order to reduce the potential for additional bias in the groups. Secondly, although methodologically limited, preliminary evidence suggests that young people with asthma may be more likely to experience ED symptoms than their healthy peers. Therefore, exploration of eating disturbances in adolescents with asthma is warranted.

In addition to the lack of controlled investigations, the concept of parenting styles is under researched in this area, specifically in relation to the dimension of overprotection. This is particularly pertinent given that parent overprotection has been found to be associated with eating disturbances in the general population (Deas et al., 2011) and has been found to be heightened in parents of children with both type 1 diabetes and asthma groups (e.g. Graue et al., 2005; Parker & Lipscombe, 1979). Further, the role of self-esteem in relation to eating disturbances in males with type 1 diabetes has not been investigated previously in the literature. Both parenting styles and self-esteem may provide useful insight into factors associated with eating disturbances in certain chronic illness groups.

Therefore, this study aims to investigate the relationships between 1) parent care and overprotection, 2) self-esteem, and 3) eating disturbances, in male and female adolescents with type 1 diabetes and chronic asthma. This study is interested in a broader definition of eating disturbances than diagnosis of ED and will focus on subthreshold eating disturbances, as defined in Section 1.4.1. In addition, much of the research in this area has been conducted
by the Eating Disorders and Diabetes Research Group based at the University of Toronto.

Further investigations carried out by a variety of researchers in different contexts and settings are required to improve the robustness of the literature base.

1.10 Research Questions and Hypotheses

The primary and secondary research questions are as follows:

1. Are adolescent reports of parent care and overprotection associated with eating disturbances in each group?
2. Is adolescent self-esteem associated with eating disturbances in each group?

The secondary research questions are:

3. Are levels of eating disturbances higher in young people with type 1 diabetes compared to those with asthma?
4. Are there differences in adolescent reported parent care and overprotection and adolescent self-esteem between those with type 1 diabetes and asthma?

1.10.1 Research hypotheses.

1. Adolescent-perceived parent care will be negatively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.
2. Adolescent-perceived parent overprotection will be positively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.
3. Adolescent self-esteem will be negatively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.
4. Level of eating disturbances will be higher in the type 1 diabetes group than the asthma group.
5. There will be differences in the level of adolescent-perceived parent care, parent overprotection and adolescent self-esteem between the type 1 diabetes group and the asthma group.
CHAPTER TWO

2. Method

2.1 Chapter Overview

This chapter begins with an outline of the current study design, participants and recruitment procedure. Each measure is then described in terms of suitability for the study, application and psychometric data. Identified ethical issues are addressed before a detailed account of the study procedure and plan for analysis are provided.

2.2 Design

A between-groups design was used in the current study to explore factors associated with eating disturbances in two groups. These were defined by type of chronic illness: Participants in Group 1 had a diagnosis of type 1 diabetes and participants in Group 2 a diagnosis of chronic asthma. Group 2 acted as a comparison group for the investigation of eating disturbances in type 1 diabetes, as well as providing a novel opportunity to explore risk factors for eating disturbances in this population. Employing a comparison group afforded the opportunity to explore whether observed relationships are unique to a type 1 diabetes sample, or whether they are also present in another chronic illness group.

The design required participants to complete a battery of self-report questionnaires at one time point. A cross-sectional design was an appropriate methodology for investigating associations between self-esteem, parenting style and eating disturbances in each group. Additionally, participation in the study was anonymous. Anonymity was an important aspect of the design as it was anticipated that participants would be less likely to report the presence of eating disturbances if they were identifiable. This follows from a previous investigation of adolescents with type 1 diabetes that reported lack of anonymity supressed reporting of symptoms (Smith et al., 2009). In addition, this is supported by the literature which indicates
that face to face interviews for assessing eating disturbances yield lower scores than self-report methods (Fairburn & Beglin, 1994).

2.3 Participants

Participants were males and females aged 16 to 18 years old with a diagnosis of either type 1 diabetes or chronic asthma. This age range was chosen as research suggests that eating disturbances emerge over time and are more prominent in later adolescence, both in the general population (Hudson et al., 2007; Stice, Killen, Hayward, & Taylor, 1998) and in those with type 1 diabetes (Daneman et al., 2002). Additionally, as this study is concerned, in part, with participants’ perceptions of their parents’ levels of care and overprotection, consideration was given to maximizing the validity of participants’ reports. A review paper looking at the literature on retrospective reports of negative childhood experiences found reports to be partly biased, with significant levels of false negative reports (Hardt & Rutter, 2004). The study reported here used the Parental Bonding Instrument (PBI; Parker et al., 1979) to assess participants’ perceptions of their parents. The PBI requires individuals to rate their parents’ attitudes and behaviours based on how they remember their parents over the first 16 years of their life. Participants in the study reported here were close in age to the period of time being reported on and it was anticipated that this proximity in time would improve the reliability of the retrospective report.

2.3.1 Inclusion and exclusion criteria.

The inclusion criteria included:

1. Diagnosis of type 1 diabetes and receiving insulin treatment (either through multiple injections or insulin pump therapy) or chronic asthma. Chronic asthma was defined as those needing to attend either hospital or specialist community-based clinics, including asthma clinics within general practices, as part of their asthma monitoring and/or treatment.
2. Diagnosis to be present for a minimum of 6 months. This criterion is in line with previous research in this area (Kichler et al., 2008; Olmsted et al., 2008) where it was included to ensure that participants had time to begin to adjust to the diagnosis and treatment regime.

The exclusion criteria included:

1. Participants reporting a diagnosis of both type 1 diabetes and chronic asthma on the demographic information sheet.

2. Those who did not speak fluent English, as the questionnaires included in the study required a good understanding of the English language.

2.3.2 Recruitment procedure.

Participants were recruited from NHS services in Norfolk and Cambridgeshire. Consultant Paediatricians or GP Managers within potential services were initially approached by the researcher, either by email or telephone, and introduced to the study. If interested, further information was provided and a meeting arranged with the researcher to discuss the possibility of the service facilitating recruitment. Once a service had agreed to support the study they were provided with a study pack. This included the study protocol, proof of ethical approval and NHS Trust research and development approval, and contact details for the researcher. Information about the recruitment procedure for each group is provided below.

2.3.2.1 Recruitment of the type 1 diabetes group.

Four outpatient diabetes clinics in Norfolk and Cambridgeshire were approached regarding the study; all agreed to take part in the study. Over a five month period the researcher attended weekly clinics at two hospitals and monthly clinics at another. Recruitment was carried out by a specialist diabetes nurse at monthly clinics held at the fourth hospital on behalf of the researcher.
Young people meeting the inclusion criteria were told about the study by their clinician during a routine clinic appointment. If they were interested in participating then they were introduced to the researcher, who provided them with an information sheet (Appendix A) and gave them an opportunity to ask questions. If they agreed to participate then they were given the option of (a) taking away a questionnaire pack to complete in their own time and return in a stamped, addressed envelope to the researcher, or (b) to take away a web address to access an online version of the questionnaire pack. If the researcher was unable to attend the clinic then the clinician carried out the above steps on behalf of the researcher. In total 115 young people with type 1 diabetes were told about the study and invited to take part. Of these, 65 participated in the study, which indicates a 56% response rate. Seven young people opted for the online participation method (11%) and the remaining 58 completed paper questionnaires (89%); no significant differences were found in level of eating disturbances between those who participated online and by post. One participant was excluded from the analyses as they reported having both type 1 diabetes and asthma. It is not possible to report on the percentage of participants recruited from each clinic as this information was not collected to maximise participant anonymity.

2.3.2.2 Recruitment of the asthma group.

Initially ethical approval was granted to recruit young people with asthma using the same recruitment procedure as outlined above for the type 1 diabetes group. Respiratory clinics at four hospitals across East Anglia and 16 general practices within NHS Norfolk Primary Care Trust were approached and invited to participate in the study. All four hospital clinics declined the invitation due to having low numbers of 16-18 year olds with asthma on their clinic registers. Five of the GPs approached (31%) agreed to support the study with recruitment. Each surgery had a specialist respiratory nurse who was responsible for running asthma clinics within the practice. However, clinic times were not regular and there were no
clinics specifically targeting adolescents and young people. Due to practical limitations it was not possible for the researcher to be present at the participating asthma clinics. Therefore, recruitment was carried out by the respiratory nurses on behalf of the researcher. Between the five clinics it was anticipated that 241 potential participants would be seen in clinic over a six month period, based on estimates provided by each surgery.

Young people meeting the inclusion criteria were introduced to the study by their nurse during a routine asthma clinic appointment. If they agreed to participate then they were given the same postal and online participation options as the type 1 diabetes group (Section 2.3.2.1). Over a three month period only three young people were invited to participate in the study, and of those one participated. Feedback provided by the nurses revealed that few 16-18 year olds were attending their asthma clinic appointments, and when they did it was difficult for the nurses to remember to invite them to participate in the study, despite regular contact with the researcher.

The recruitment procedure for the asthma group was reviewed. Ethical approval was granted for a revised procedure, which involved inviting young people to take part in the study through a mailshot. Invitation letters and information sheets were posted to all 16-18 year olds registered at participating GP surgeries as having a diagnoses of asthma (Appendix A). The letter was sent jointly from the participating GP surgery and the researcher on GP headed paper. In the letter the young person was introduced to the researcher, the purpose of study and what participation would involve. If interested in participating they were directed to the online battery of questionnaires. In total 37 GP surgeries within NHS Norfolk Primary Care Trust were contacted and invited to facilitate recruitment for the study; this included re-contacting surgeries that had been invited to participate in the study using the original recruitment procedure. Of these 37, ten GP surgeries (27%) agreed to take part and facilitate
a mailshot. In total 411 letters were posted and 37 young people participated through the online participation pathway; this is a response rate of 9.0%.

2.3.3 Sample size.

In order to determine the required sample size for this study power calculations were carried out using GPower (Erdfelder, Faul & Buchner, 1996). For the primary research questions (Section 1.10) 67 participants were required in each group to achieve 80% power, using a one tailed correlational analysis, with $\alpha = .05$ and assuming a moderate size relationship. For the secondary research questions 64 participants were required in each group to achieve 80% power when carrying out a between groups analysis ($\alpha = .05, d = .05$). Therefore, the study aimed to recruit a total of 134 participants, with 67 participants in each group (type 1 diabetes and chronic asthma).

2.4 Measures

Six self-report questionnaires were used to collect data from participants. Each is described below, along with an evaluation of the psychometric properties of the questionnaire, where appropriate, and suitability for this study.

2.4.1 Demographic information.

An idiographic demographic information sheet was designed specifically for use in this study (Appendix B). This included questions about participants’ age, gender and current education/employment, as well as previous/current diagnosis of an ED, age at diagnosis of type 1 diabetes/chronic asthma, frequency of visits to the clinic and steroid use (for those with asthma). Participants were also asked about whom they live with, if they have children and parent marital status. This information was used to describe the sample characteristics and contextualise the sample.

Information provided by participants about their height and weight was used to calculate BMI using the formula (weight [kg]/(height[m])$^2$). BMI (Maharaj et al., 2003;
Eating disturbances in young people with type 1 diabetes. Similar associations have been found between BMI, gender and body dissatisfaction in adolescents with asthma (Kelsay et al., 2005). Therefore, data on participants’ BMI, gender and age of illness onset were collected in order that it could be controlled for during the analysis stage if required.

2.4.2 Measure of eating disturbances.

Eating disturbances were measured using the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 2008; Appendix B). The questionnaire has been developed from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), which is a clinician rated, semi-structured interview designed to measure specific ED psychopathology. The EDE has been widely used in the literature and is generally considered to be the gold standard measure of ED psychopathology (Garner, 1995). There has been much debate amongst researchers about whether interview-based or self-report measures are most appropriate when assessing ED symptoms. For the purpose of this study the EDE-Q was chosen because maintaining anonymity was an important aspect of the study design. In addition, the EDE is considerably more time-consuming to administer than the EDE-Q and requires the interviewer to be trained in administering the assessment, which was not within the scope of the study.

2.4.2.1 Description of measure and scoring.

The EDE-Q is a 28 item self-report questionnaire that measures restraint, eating concern, weight concern and shape concern, as well as global levels of eating disturbances. The EDE-Q requires respondents to report on the presence and frequency over the past 28 days of certain behaviours, cognitions and emotions related to ED psychopathology. Questions 1 to 12 and 19 to 21 use a seven point forced-choice scale ranging from No days
(0) to *Every day* (6), while questions 22 to 28 use a seven point scale ranging from *Not at all* (0) through to *Markedly* (6). Questions 13 to 18 do not make use of a forced-choice scale and require respondents to report openly on the frequency of certain behaviours. In addition, the EDE-Q asks for participants’ weight, height and, if female, whether they have missed any menstrual periods in the past 3-4 months.

Two types of data are produced by the EDE-Q. Firstly, the questionnaire yields four subscale scores for restraint, eating concern, weight concern and shape concern. The subscale scores are calculated by summing the subscale items and dividing by the number of items; they are reported as means and standard deviations. Similarly, the global score is determined by calculating the average score across the four subscales. Higher scores indicate greater severity of ED psychopathology. Scores of four or higher on the individual items, subscale scores and global score are considered to be in the clinical range. Secondly, questions 13 to 18 yield information regarding the frequency of certain behavioural aspects of ED psychopathology over the past 28 days, e.g. frequency of self-induced vomiting.

**Table 2**

*Normative Data for the EDE-Q for Males and Females in the General Population*

<table>
<thead>
<tr>
<th>EDE-Q subscale</th>
<th>Female (SD)</th>
<th>Male (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>1.25 (1.32)</td>
<td>1.04 (1.19)</td>
</tr>
<tr>
<td>Eating concern</td>
<td>0.62 (0.86)</td>
<td>0.43 (0.77)</td>
</tr>
<tr>
<td>Weight concern</td>
<td>1.59 (1.37)</td>
<td>1.59 (1.38)</td>
</tr>
<tr>
<td>Shape concern</td>
<td>2.15 (1.60)</td>
<td>1.29 (1.27)</td>
</tr>
<tr>
<td>Global score</td>
<td>1.55 (1.21)</td>
<td>1.09 (1.00)</td>
</tr>
</tbody>
</table>

*a*Fairburn and Beglin (1994); *b*Lavender et al. (2010).
This data does not contribute to the subscale or global scores. Normative data for community samples of adult females aged 16-35 years old (Fairburn & Beglin, 1994) and adult males aged 18-26 (Lavender, De Young & Anderson, 2010) are provided (Table 2). The questionnaire takes approximately 10 minutes to complete.

2.4.2.2 Psychometric data.

The current study used the EDE-Q version 6 (Fairburn & Beglin, 2008). Much of the published psychometric data for the EDE-Q is based on an earlier version of the questionnaire (Fairburn & Cooper, 1993). However, as there are only “minor changes” between the earlier version and the current sixth edition, psychometric data for both versions will be described here (Anderson, De Young, & Walker, 2009, p. 420).

The EDE-Q has been shown to have good internal consistency. Cronbach's alpha estimates for each subscale have been equal to or above the accepted level of .70 (Cicchetti, 1994). Estimates range from .78 to .93 and .70 to .83 in community and clinical samples respectively (Luce & Crowther, 1999; Peterson et al., 2007). A recent review indicated that, to date, there are no published data pertaining to the internal consistency of the EDE-Q when used with a male sample (Berg, Peterson, Frazier, & Crow, 2011). Despite the EDE-Q having been used with samples of young people with type 1 diabetes samples (Schwartz, Weissberg-Benchell, & Perlmutter, 2002; Smith et al., 2008), there are currently no published psychometric data available for this specific group. However, D’Emden et al. (2012) recently reported good internal consistency for the Youth EDE-Q in a sample of adolescents with type 1 diabetes; The subscale internal consistency coefficients ranged from .78 to .95. This is an adapted version of the original questionnaire for use with 12-17 year olds and those with a younger reading age (Goldschmidt, Doyle, & Wilfey, 2007). The EDE-Q has not been used previously with young people with asthma. In the current study Cronbach’s alpha coefficient for the Global EDE-Q scale was .96. Coefficients for the individual subscales in the current
study were also good (Restraint = .83, Eating Concern = .82, Weight Concern = .89, Shape Concern = .93).

Significant two week test re-test correlation coefficients have been found for all EDE-Q subscales and behaviour frequency questions (Luce & Crowther, 1999; Reas, Grilo, & Masheb, 2006). Correlations across these two studies ranged from .66 to .94 and .51 to .92 for the subscales and behavioural frequencies respectively. Over longer test re-test periods coefficients for the dietary restraint subscale dropped from .81 at a two week test re-test period, to .57 over 14 months (Mond, Hay, Rodgers, Owen, & Beumont, 2004). Other subscale coefficients remained similar to those yielded in the two week test re-test analysis, and still greater than .70. This indicates that the EDE-Q has adequate temporal stability.

Psychometric data indicate that the EDE-Q has high agreeability with other prominent measures of eating disturbances, demonstrating good concurrent validity. Significant correlations have been found between scores of the EDE-Q and the EDE (Fairburn & Beglin, 1994; Kalarchian, Wilson, Brolin, & Bradkey, 2000; Mond et al., 2004; Wilfley, Schwartz, Spurrell, & Fairburn, 1997). The EDE-Q has also been shown to have good predictive validity in identifying those with and without clinical levels of ED psychopathology and in detecting change in level of ED following treatment (Hilbert, Tuschen-Caffier, Karwautz, Niederhofer, & Munsch, 2007; Mond et al., 2004)

2.4.2.3 Adaptations.

Insulin under-dosing and omission are unique methods for controlling weight and shape for individuals with type 1 diabetes (Daneman et al., 1998). Therefore, it was important to take this into consideration when assessing eating disturbances in this group. Adaptations have been made to the EDE for use with adults with type 1 diabetes (Fairburn et al., 1991). This group reported including additional questions asking about the "under use or omission of insulin for the purpose of weight control" (p. 18). For the purpose of the current study two
questions were added to the EDE-Q for the type 1 diabetes group asking about insulin manipulation in relation to weight control (Questions D1 and D2 on the EDE-Q; Appendix B). These data were analysed separately from the standardised questionnaire.

2.4.3 Measure of parenting style.

The Parental Bonding Instrument (PBI; Parker et al., 1979) was used to measure parenting style (Appendix B). The PBI is a self-report questionnaire that was developed with adults aged 16 years and over. It is a retrospective report of an individual’s perception of their parents’ parenting behaviours and attitudes during the first 16 years of their life. The PBI is a well established measure of retrospective parenting that has has been widely used in research settings with adolescents (Cubis et al., 1989) and with clinical and non-clinical samples, including those with EDs (Deas et al., 2011; Fichter, Quadflieg, & Brandl, 1993; McEwen & Flouri, 2009). Additionally, it has been used to investigate the associations between parent factors and the psychosocial well-being of young people with chronic illness (Agostini et al., 2010; Fisher & Chalder, 2003), including in those with type 1 diabetes (Graue et al., 2005).

2.4.3.1 Description of measure and scoring.

The PBI is a 25 item self-report questionnaire. Factor analysis during the original development of the PBI revealed two interdependent dimensions of parenting, namely care and overprotection (Parker et al., 1979). Care refers to parent warmth and affection, which is contrasted by coldness and rejection at the other end of the dimension. Overprotection in this context is used to describe parenting behaviour and attitudes that are controlling and intrusive and are contrasted by psychological autonomy. Subsequent analyses of the factorial structure of the PBI has been mixed, with some supporting a two-factor model (Kazarian, Baker, & Helmes, 1987; Mackinnon, Henderson, Scott, & Duncan-Jones, 1989) and others proposing alternative three factor models with the addition of a behavioural freedom factor (Murphy, Brewin, & Silka, 1997). Despite this disagreement Parker’s original two factor model
remains the most widely used in the literature and will therefore be utilised for the purposes of this study.

The questionnaire is made up of two scales, which ask about maternal and paternal parenting separately; it takes approximately 7 minutes to complete. For the purposes of this study participants were asked to complete both the mother and father scales if applicable. Respondents are asked to rate the 25 items on a forced-choice four point scale (ranging from very like to very unlike) based on how they remember their parents during the first 16 years of their life. The questionnaire yields a score for level of care and overprotection for each parent; higher scores reflect higher levels of each dimension. In addition, based on normative data reported by Parker (1983), responses can be categorised as either high or low levels of care and overprotection. The cut off scores for the care category are 27.0 and 24.0 and for the overprotection category are 13.5 and 12.5 for mothers and fathers respectively. Scores equal to and above the cut off score fall into the high category, those below into the low category. Parker proposed that based on these categories parents could be assigned to one of four types of parenting, which are detailed in Table 3.

<table>
<thead>
<tr>
<th></th>
<th>High overprotection</th>
<th>Low overprotection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High care</strong></td>
<td>Affectionate control</td>
<td>Optimal parenting</td>
</tr>
<tr>
<td><strong>Low care</strong></td>
<td>Affectionless control</td>
<td>Neglectful parenting</td>
</tr>
</tbody>
</table>

2.4.3.2 Psychometric data.

The PBI has demonstrated good psychometric data in both clinical and non-clinical samples. Alpha coefficients ranging from .85 to .94 have been achieved for the care and
overprotection dimensions (Brewin et al., 1992; Safford et al., 2007). In a review of the psychometric properties of the PBI Parker (1989) reported a mean alpha coefficient of .88, which indicates good internal consistency. For the current study cronbach’s alpha coefficients were .89 and .93 for mother and father care subscales, and .86 and .86 for mother and father overprotection subscales respectively.

Test-retest reliability over seven months has been reported as ranging from .79 to .81 (Richman & Flaherty, 1990). Murphy, Wickramaratne and Weissman (2010) demonstrated no significant changes in male and female responses on the maternal care and overprotection dimensions of the PBI across 20 years. Although there were no significant changes for reports of paternal care, paternal overprotection was found to reduce by an average of 1.6 points over the 20 year period. Evidence in support of the long term temporal stability of the PBI is also reported in Wilhelm, Niven, Parker & Hadzi-Pavlovic (2005). The questionnaire has adequate concurrent and predictive validity (Safford et al., 2007). In addition, research suggests that retrospective reports of parenting as measured by the PBI are not significantly influenced by mood (Gerlsma, Kramer, Scholing, & Emmelkamp, 1994). Additionally, high agreement has been found between siblings’ reports on the PBI and between parent and child reports (Mackinnon, Henderson, & Andrews, 1991; Parker, 1983). Taken together this indicates that the questionnaire is a valid measure of perceptions of parenting.

2.4.4 Measure of self-esteem.

The Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965) was used to measure global self-esteem (Appendix B). The questionnaire is a 10-item, uni-dimensional, self-report measure that assesses self-esteem defined as an attitude towards one’s self based on feelings about one’s own worth (Mruk, 2006). The RSES is the most commonly used measure of self-esteem and has been chosen in this instance due to its brevity, accessibility and ease of use for participants; it takes approximately 3 minutes to complete. It has been extensively used in
clinical and non-clinical populations as well as with adolescents with chronic health conditions (Birkeland, Melkevik, Holsen, & Wold, 2012; Brockmeyer et al., 2012; Powers et al., 2012).

2.4.4.1 Description of measure and scoring.

The RSES consists of 10 statements that are rated on a four point likert scale ranging from strongly agree to strongly disagree. The questionnaire yields a single global self-esteem score, with higher scores reflecting higher self-esteem. Normative data are available for British adolescents aged 12-19 (Bagley & Mallick, 2001; Appendix D).

2.4.4.2 Psychometric data.

The scale was initially developed for American adolescents and yielded excellent internal consistency coefficients of $\alpha = .92$ and test-retest reliability of .85-.88 over two weeks and .77 over one year (Rosenberg, 1979). The internal consistency estimate for the study reported here was .92. Griffiths et al. (1997) investigated the predictive validity of the RSES and found it to be significantly inversely correlated with measures of depression and ineffectiveness in a sample of young people with EDs. Strong associations have been found between scores on the RSES and on the Global Self Worth scale of the Harter’s Self-Perception Profile (Harter, 1988); this indicates good concurrent validity (Hagborg, 1993).

2.4.5 Measure of psychological distress.

In order to be able to report on levels of general psychological distress within the sample, the Depression Anxiety Stress Scale - 21 (DASS-21; Henry & Crawford, 2005) was administered. The DASS-21 is a 21 item self-report questionnaire that has three subscales measuring levels of anxiety, depression and stress, the latter of which is characterised by nervous tension, difficulty relaxing and irritability (Appendix B). The subscale scores give an indication of the severity of distress a participant has experienced over the past week ranging from normal to extremely severe. This is a shortened version of the widely used Depression
Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995) and was chosen due to its brevity and free accessibility; it takes approximately 5 minutes to complete. In addition, it has been validated with samples of young people aged 16, 17 and 18 (Tully, Zajac, & Venning, 2009) and with participants with EDs (Harrison et al., 2010) and chronic illnesses (Moore et al., 2009).

2.4.5.1 Description of measure and scoring.

The DASS-21 comprises 21 statements which measure negative emotional symptoms. Respondents are asked to rate how much each statement has applied to them over the past week on a four point likert scale ranging from did not apply to me to applied to me very much. Subscale scores are calculated by summing respondents’ scores on each individual scale. A global score for negative emotional symptoms can be calculated by finding the average of the subscale scores. Higher scores on all scales reflect higher levels of distress. Normative data and percentile ranks are available for the DASS-21 (Henry & Crawford, 2005).

2.4.5.2 Psychometric properties.

The DASS-21 has shown excellent internal consistency with alpha coefficients of .93, .90, .88, and .82 for the global negative distress scale and stress, depression and anxiety subscales respectively (Henry & Crawford, 2005). Coefficients ranging between .82 and .96 have been reported for a sample of adults with EDs (Harrison et al., 2010). Two week test re-test correlations of .71 for depression, .79 for anxiety and .81 for stress indicate that the measure has adequate short-term temporal stability (Brown, Chorpita, Korotitsch, & Barlow, 1997). Cronbach alpha coefficients for the current study were .94, .86, .86 and .88 for the global score, stress, depression and anxiety scales respectively. In addition, Henry and Crawford (2005) report concurrent validity coefficients that are comparable to those reported
for the longer version of the questionnaire (DASS; Antony, Bieling, Cox, Enns, & Swinson, 1998).

2.4.6 Measure of illness severity.

In order to be able to compare severity of illness reported by participants in each group, participants were asked to complete two visual analogue scales (VASs) which were designed to measure this for the purpose of the current study (Appendix B). An idiosyncratic measure of illness severity was chosen due to a lack of suitable self-report questionnaires measuring this construct. Current measures of illness severity either rely on clinician or parent report (Knaus, Draper, Wagner, & Zimmerman, 1985; Leung et al., 1997; Parkerson, Broadhead & Chiu-Kit, 1993), are illness-specific (Bishop, Carlin, & Nolan, 1992; DCCT Research Group, 1989;) or focus on related constructs, such as quality of life (Stewart et al., 1989; Varni, Seid, & Roder, 1999).

The VASs used are based on those that are reported in the arthritis literature (Cella et al., 2005) and the widely used general health status scales (e.g. EQ5-D; The EuroQol Group, 1990). The first VAS required participants to rate the severity of their illness symptoms over the past seven days, and the second to rate the severity of their illness symptoms in the past when they had been at their worst. Each VAS was numbered from 0 to 100 and was anchored at one extreme with 0 = Not at all Severe, in the middle with 50 = Moderately Severe and at the opposite extreme with 100 = Extremely Severe. Participants were asked to choose a number on the line which best represented the severity of their illness symptoms, as perceived by them.

2.5 Ethical Considerations

2.5.1 Consent.

Informed consent to take part in the study was sought from all participants. If the young person was interested in finding out about the study either the researcher or their
clinician provided them with a clearly written study information sheet (Appendix A). In addition, either the researcher or the young person’s clinician talked through the information sheet with them and gave them an opportunity to ask questions. Participants were encouraged to take some time to consider whether or not they would like to participate. Despite being over the age of 16 they were encouraged to discuss the study with their parents where appropriate. A parent information sheet was available for participants to give to their parents if they thought that would be helpful (Appendix A). It was explained that completing and returning the questionnaires implied consent to take part. Written consent was not required in order to maintain participant anonymity, which is an important aspect of the study design (Section 2.2).

### 2.5.2 Risks and benefits.

It was not anticipated that participating in the study would cause distress. However, participants were told that should they become distressed by taking part they should stop completing the questionnaires and contact the clinician who first introduced them to the study. The researcher ensured that clinicians involved with the study felt able to signpost participants appropriately should they need to. There was potential for some participants to report significant levels of ED symptomology. However, due to the study design it was not possible to feed this back to either the young person or their clinician. At the point of recruitment it was made clear to participants that some of the questions may be highly relevant to them and that should they be concerned about their own well-being they should speak to either their clinician or another responsible adult whom they trust.

There were no direct benefits to taking part for the young person other than having the opportunity to contribute to the literature on what is known about the emotional well-being of young people with type 1 diabetes and asthma. Participants were able, however, to opt into a prize draw to win one of two £20 high street clothing vouchers.
2.5.3 **Confidentiality.**

All data collected remained anonymous, with each participant being assigned a participant number. The procedures used for handling data were in accordance with the Data Protection Act (1998). Paper-based data were kept securely in a locked cabinet at the researcher’s home while the study was on going. Data submitted via the online questionnaire battery were kept on a password protected computer and only transferred using an encrypted memory stick. On completion of the investigation data will be archived at the University of East Anglia for 5 years.

2.5.4 **Ethical approval.**

Ethical approval was obtained from an NHS Research Ethics Committee (Appendix C). Additionally, NHS permission was obtained from the five participating NHS trusts (Appendix C).

2.6 **Procedure**

Potential participants were invited to take part in the study through the recruitment procedure described in Section 2.3.2. Participants in the type 1 diabetes group were given the option of either completing the questionnaire pack through an online website or in paper format. Those choosing the paper option were given a pack of questionnaires to take away and complete in their own time, along with a stamped addressed envelope. Participants were informed that they may either return the questionnaires by post or by placing them in a specially designed post box in their clinic. Participants choosing the online option were given a link for the website hosting the questionnaires, which they could access online in their own time to complete and submit their responses. They were also sent an email from the researcher containing a hyperlink for the website (www.eatingstudy.co.uk). Following difficulties with the initial recruitment strategy, potential participants for the asthma group were invited to participate through an invitation letter and information sheet posted from the
Eating Disturbances in Chronic Illness

GP surgery (Section 2.3.2.2). If interested in taking part in the study then participants were directed to the online battery of questionnaires accessible via the hyperlink described above. The letter clearly stated that participants must read the information sheet before participating in the study.

Those participating online were given a unique code to enter along with their questionnaire responses to ensure that those taking part had been introduced to the study at a participating clinic or through an invitation letter. The online questionnaires were hosted by the free survey provider SurveyExpression, which states that surveys hosted by them are secure and that data submitted online by respondents are safe and only accessible to the researcher. The website included a welcome page, on which participants were thanked for taking part in the study. The questionnaires were loaded onto the website using the exact wording and format of the paper-based questionnaires.

All participants were offered the opportunity to enter into a prize draw to win one of two £20 high street clothing vouchers. In order to preserve anonymity they were required to seal their name and preferred contact details inside a prize draw envelope, which they returned with their questionnaire packs. For those completing the questionnaires online, they were asked to email contact details to a designated email address upon completion of the questionnaires, if they wanted to be entered into the draw. Once participants’ contact details were received, they were printed and stored in a sealed envelope. All prize draw entries were stored separately from the data and could not be linked to corresponding participant data. They were not opened unless drawn from the prize draw at the end of the study. The winners were contacted and arrangements made to send them their voucher.

2.6.1 Use of internet mediated research.

As described above participants were given the option of completing and submitting the self-report questionnaires through either the traditional paper and postal method, or
through the use of an online website. Internet mediated approaches to collecting data for research purposes are becoming increasingly popular (Whitehead, 2007). This method was chosen for the current study as research indicates that it may have benefits over postal methods. In particular, it has been shown to be more effective in reaching difficult to access groups, which may include adolescents, and reduces potential practical barriers to participation (Mann & Stewart, 2000; Stewart, Eckermann, & Zhou, 1998). Participants were given the option of either postal or internet-mediated methods in order to maximise accessibility of the study and not exclude those without access to the internet.

2.7 Plan of Analysis

This section describes the analysis plan based on the research questions; any deviations from the plan based on the collected data are described in Chapter 3. All data were analysed using Predictive Analytics Software Version Statistics 18 (PASW Statistics 18). The data set was hand-searched for missing data and anomalous data points. Descriptive statistics were used to describe the sample characteristics and to ensure that the two groups did not differ significantly on key demographic variables. In addition, the distribution of the data was explored using normality plots and Kolmogorov-Smirnov statistical tests for normality. If the data sets did not follow a normal distribution attempts were made to transform the data to resemble a normal distribution. The data analysis plans for the primary and secondary research questions are described below.

Pearson’s Product Moment Correlation Coefficients were used to answer the primary research questions. Firstly, correlations between paternal and maternal care and overprotection, as measured by the PBI, and eating disturbances, as measured by the global scale of the EDE-Q, were explored. Secondly, correlation coefficients for self-esteem, as measured by the RSES, and eating disturbances, as measured by the global scale of the EDE-Q, were calculated. These statistical tests were carried out separately for the type 1 diabetes
group and the chronic asthma group. The secondary research questions were concerned with between group comparisons. Analysis of Variance tests were used to explore differences in the group means for eating disturbances (EDE-Q global scale), paternal and maternal care and overprotection (PBI) and self-esteem (RSES).
CHAPTER THREE

3. Results

3.1 Chapter Overview

This chapter begins with an outline of the data treatment process and a summary of the demographic information and illness characteristics for each group. Descriptive data analyses are provided for all variables, including explorations of data distribution. The results of statistical testing are presented for each hypothesis in turn. Additional exploratory analyses investigating the moderating effect of diagnosis on the observed relationships are described. Finally, a summary of the results is provided.

3.2 Treatment of Data

All data were entered into a spreadsheet using the PASW Statistics 18 software package. The data screening process was twofold: Firstly, checking for missing data and secondly, correcting errors in the data set. One or more items (maximum of two) were missing from the Parental Bonding Instrument (PBI) for five participants, from the Depression, Anxiety and Stress Scale (DASS-21) for four participants, and from the Rosenberg Self Esteem Scale (RSES) for two participants. These missing data were replaced with the mean as a measure of central tendency (Tabachnick & Fidell, 2001). Therefore, missing item-level data were imputed with the average rating on that scale for each individual participant. Cases which had data missing for an entire questionnaire were excluded pairwise from analysis (Tabachnick & Fidell, 2001). This was relevant on two occasions, for one participant who did not complete the PBI and for another who did not complete the DASS-21. Some demographic data were also missing, which will be detailed in Section 3.3.1. It is likely that missing data are the result of participants being asked to respond to a large number of consecutive questions without the researcher having the opportunity to ensure all questions had been answered. The data set was checked for errors of data entry by scanning frequency.
and descriptive data for the categorical and continuous variables respectively. Suspected errors were checked against the raw data and changes made where necessary. In total five errors were identified and corrected.

3.3 Sample Description

In total 102 participants were recruited into the study. Of these, 65 were in the type 1 diabetes group and 37 were in the chronic asthma group. Difficulties with recruitment in the latter group are discussed in Section 4.5.2.2. Across the two groups 62.7% of participants were female ($n = 64$) and 37.3% were male ($n = 38$). A description of the demographic information for each group is presented below.

3.3.1 Sample characteristics.

The age range of participants in both groups was 16 years and 0 months to 18 years and 11 months (192 to 227 months). The mean age was 17 years and 8 months ($SD = 9.50$) for the type 1 diabetes group and 17 years and 5 months ($SD = 9.36$) for the asthma group. There was no statistically significant difference between groups with respect to age ($Z = -1.88$, $p = .059$). No participants reported having any children of their own in either group. Additional demographic information for each group is presented in Table 4. A Chi-Square test revealed significant differences in the proportion of males and females in each group, $\chi^2 (1, n = 102) = 6.07$, $p = .030$. There was a higher proportion of males in the type 1 diabetes group than in the asthma group; this will be further discussed in Chapter Four in relation to the study findings. No other significant between group differences were found for the demographic data (Table 4).
Table 4

Demographic Information for the Type 1 Diabetes and Asthma Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter</th>
<th>T1D</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (% )</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Gender*</td>
<td>Male</td>
<td>30 (46.2%)</td>
<td>8 (21.6%)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>35 (53.8%)</td>
<td>29 (78.4%)</td>
</tr>
<tr>
<td>Education/employment</td>
<td>Student</td>
<td>55 (84.7%)</td>
<td>31 (83.8%)</td>
</tr>
<tr>
<td></td>
<td>Employed</td>
<td>8 (12.4%)</td>
<td>5 (13.5%)</td>
</tr>
<tr>
<td></td>
<td>Unemployed</td>
<td>2 (3.1%)</td>
<td>1 (2.7%)</td>
</tr>
<tr>
<td>Living situation</td>
<td>With parents</td>
<td>50 (76.9%)</td>
<td>28 (75.7%)</td>
</tr>
<tr>
<td></td>
<td>With Mum only</td>
<td>14 (21.5%)</td>
<td>7 (18.9%)</td>
</tr>
<tr>
<td></td>
<td>With Dad only</td>
<td>1 (1.5%)</td>
<td>1 (2.7%)</td>
</tr>
<tr>
<td></td>
<td>Missing data</td>
<td>0</td>
<td>1 (2.7%)</td>
</tr>
<tr>
<td>Parents’ marital status</td>
<td>Married/cohabiting</td>
<td>38 (58.5%)</td>
<td>25 (67.6%)</td>
</tr>
<tr>
<td></td>
<td>Divorced/separated</td>
<td>25 (38.5%)</td>
<td>12 (32.4%)</td>
</tr>
<tr>
<td></td>
<td>Widowed</td>
<td>1 (1.5%)</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Missing data</td>
<td>1 (1.5%)</td>
<td>0</td>
</tr>
<tr>
<td>Primary caregiver</td>
<td>Mum</td>
<td>42 (64.6%)</td>
<td>23 (62.2%)</td>
</tr>
<tr>
<td></td>
<td>Dad</td>
<td>2 (3.1%)</td>
<td>2 (5.4%)</td>
</tr>
<tr>
<td></td>
<td>Mum &amp; Dad</td>
<td>16 (24.6%)</td>
<td>6 (16.2%)</td>
</tr>
<tr>
<td></td>
<td>Missing data</td>
<td>5 (7.7%)</td>
<td>6 (16.2%)</td>
</tr>
<tr>
<td>ED diagnosis</td>
<td>Yes – current</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Yes – past</td>
<td>1 (1.5%)</td>
<td>4 (10.8%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>61 (93.8%)</td>
<td>32 (86.5%)</td>
</tr>
<tr>
<td></td>
<td>Missing data</td>
<td>3 (4.6%)</td>
<td>1 (2.7%)</td>
</tr>
</tbody>
</table>

* Significant between group difference at $p < .05$.  

Note. T1D = Type 1 diabetes; ED = Eating Disorder Diagnosis.
The mean BMI across the whole sample was 22.54 ($n = 98$; $SD = 4.55$) and ranged from 13.10 to 37.10; a reported BMI of 59.90 was considered an outlier and excluded from the analysis. For the asthma and type 1 diabetes groups the mean BMI scores were 21.75 ($n = 35$, $SD = 5.18$, minimum = 13.10, maximum = 37.10) and 22.98 ($n = 63$, $SD = 4.15$, minimum = 17.00, maximum = 35.80) respectively; this difference was not statistically significant. The outcome of a Mann Whitney U test revealed a significant difference between BMI for males and females across the whole sample ($Z = -2.07, p = .038$), with females having a higher BMI on average than males.

Steroid use for treatment was linked to BMI in the asthma group ($\chi^2 [2, n = 35] = 7.86, p = .020$) with those reporting no steroid use having a higher BMI ($Mdn = 21.3$) than those with current ($Mdn = 20.15$) or past ($Mdn = 18.75$) steroid use. This is in contrast to previous research that has linked steroid treatment to higher BMI (Wickens et al., 2005). The reasons for this contrast are unclear. It may be that the reliability of the data was compromised by the reliance on self-report and lack of clarity in the question (e.g. participants may not be aware that their current medication contains steroids and may not have known about past steroid use from their childhood).

Table 5

<table>
<thead>
<tr>
<th>Weight category</th>
<th>Type 1 diabetes ($n = 63$)</th>
<th>Asthma ($n = 35$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>%</td>
<td>Frequency</td>
</tr>
<tr>
<td>Underweight</td>
<td>3</td>
<td>4.8%</td>
</tr>
<tr>
<td>Healthy weight</td>
<td>44</td>
<td>69.8%</td>
</tr>
<tr>
<td>Overweight</td>
<td>10</td>
<td>15.9%</td>
</tr>
<tr>
<td>Obese</td>
<td>6</td>
<td>9.5%</td>
</tr>
</tbody>
</table>
Participants were classified into categories of *underweight*, *healthy weight*, *overweight* and *obese* based on their BMI, age and gender. The category definitions described by Mei et al. (2002) were used. The healthy weight category was defined by BMI between the 5th and 85th percentile, the underweight category by BMI below the 5th percentile, the overweight category by BMI between the 85th and 95th percentile, and the obese category by BMI above the 95th percentile. Age and gender specific growth charts were used to determine participants’ BMI percentiles (National Centre for Health Statistics & National Centre for Chronic Disease Prevention and Health Promotion, 2000). The majority of participants in both groups fell into the healthy weight category (Table 5). It was not possible to explore associations between type of illness and weight classification due to small cell sizes for some categories. Those classified as overweight or obese across both groups reported significantly higher levels of eating disturbances, as measured by the EDE-Q global scale ($Z = -2.44$, $p = .015$).

### 3.3.1.1 Descriptive illness-related data.

The mean age of illness onset in the type 1 diabetes group was 9.6 years ($n = 63$, $SD = 3.71$, minimum = 1 year, maximum = 17 years). In contrast, the average age at illness onset reported by participants in the asthma group was 5.75 years ($n = 36$; $SD = 4.38$; minimum = 0 years, maximum = 15 years). Mean illness duration was 7.94 years ($n = 63$; $SD = 3.77$) and 11.56 ($n = 36$; $SD = 4.30$) for the type 1 diabetes and asthma groups respectively. Both average age of illness onset ($Z = -3.85$, $p < .000$) and average duration of illness ($Z = -3.74$, $p < .000$) significantly differed between the two groups when compared using a Mann Whitney U test. Those in the asthma group had, on average, been diagnosed at an earlier age and therefore, had been living with their illness for a longer period of time. However, contrary to previous research (Meltzer et al., 2001), age of illness onset was not found to be significantly
associated with scores on the global EDE-Q scale in the type 1 diabetes group ($n = 63, r = - .18, p = .153$) or the asthma group ($n = 36, r = .13, p = .434$).

The two groups were compared according to the frequency at which participants reported attending appointments regarding their illness (Table 6). As expected, the type 1 diabetes group reported more frequent appointments than the asthma group. This is likely due to the complexity of the type 1 diabetes treatment regime and importance of close monitoring of blood glucose levels to prevent life-threatening consequences of poor treatment adherence.

Table 6

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter</th>
<th>T1D</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appointment Frequency</td>
<td>More than 3 monthly</td>
<td>5 (7.7%)</td>
<td>2 (5.4%)</td>
</tr>
<tr>
<td></td>
<td>3 monthly</td>
<td>49 (75.4%)</td>
<td>5 (13.5%)</td>
</tr>
<tr>
<td></td>
<td>6 monthly</td>
<td>9 (13.8%)</td>
<td>7 (18.9%)</td>
</tr>
<tr>
<td></td>
<td>Yearly or less</td>
<td>0</td>
<td>21 (56.7%)</td>
</tr>
<tr>
<td></td>
<td>Missing data</td>
<td>2 (3.1%)</td>
<td>2 (5.4%)</td>
</tr>
</tbody>
</table>

*Note.* T1D = Type 1 Diabetes.

3.4 Descriptive Data Analysis

In this section data are presented for the study variables, including the EDE-Q, PBI, RSES, DASS-21 and Illness Severity VAS. Descriptive data will be provided for each variable, along with an exploration of the distribution of the data.

3.4.1 Descriptive statistics for the Eating Disorder Examination – Questionnaire.

The descriptive statistics for the restraint, eating concern, weight concern, shape concern and global scales of the EDE-Q are presented in Tables 7 and 8.
Table 7

*Descriptive Statistics for the EDE-Q Subscales and Global Scale for the Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>n</th>
<th>Min - Max</th>
<th>M (SD)</th>
<th>Skewness (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>65</td>
<td>0-6</td>
<td>.97 (1.35)</td>
<td>1.58 (.30)</td>
</tr>
<tr>
<td>Eating concern</td>
<td>65</td>
<td>0-4.8</td>
<td>.85 (1.22)</td>
<td>1.64 (.30)</td>
</tr>
<tr>
<td>Weight concern</td>
<td>65</td>
<td>0-6</td>
<td>1.48 (1.69)</td>
<td>1.10 (.30)</td>
</tr>
<tr>
<td>Shape concern</td>
<td>65</td>
<td>0-6</td>
<td>1.82 (1.71)</td>
<td>.83 (.30)</td>
</tr>
<tr>
<td><strong>Global score</strong></td>
<td><strong>65</strong></td>
<td><strong>0-5.7</strong></td>
<td><strong>1.28 (1.37)</strong></td>
<td><strong>1.25 (.30)</strong></td>
</tr>
</tbody>
</table>

Table 8

*Descriptive Statistics for the EDE-Q Subscales and Global Scale for the Asthma Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>n</th>
<th>Min - Max</th>
<th>M (SD)</th>
<th>Skewness (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>37</td>
<td>0-6</td>
<td>1.78 (1.73)</td>
<td>.85 (.39)</td>
</tr>
<tr>
<td>Eating concern</td>
<td>37</td>
<td>0-5.6</td>
<td>1.50 (1.59)</td>
<td>.97 (.39)</td>
</tr>
<tr>
<td>Weight concern</td>
<td>37</td>
<td>0-5.4</td>
<td>2.49 (1.65)</td>
<td>.19 (.39)</td>
</tr>
<tr>
<td>Shape concern</td>
<td>37</td>
<td>0-5.5</td>
<td>2.97 (1.67)</td>
<td>-.07 (.39)</td>
</tr>
<tr>
<td><strong>Global score</strong></td>
<td><strong>37</strong></td>
<td><strong>0-5.63</strong></td>
<td><strong>2.18 (1.52)</strong></td>
<td><strong>.47 (.39)</strong></td>
</tr>
</tbody>
</table>

Scores on the individual EDE-Q subscales that are equal to, or greater than, four are generally considered to have clinical significance (Mond, Hay, Rodgers, & Owen, 2006). Ten participants (15.4%) with type 1 diabetes reported ED symptoms that reached clinical significance on at least one subscale, compared to 15 in the asthma group (40.5%). The shape concern subscale had the highest number of participants reaching clinical significance, with 13.8% \( (n = 9) \) in the type 1 diabetes group and 35.10% \( (n = 13) \) in the asthma group.
Additionally, females in both the type 1 diabetes and asthma groups scored higher on average across all subscales compared to normative data reported by Fairburn and Beglin (1994) for a community sample of 16-35 year old women (Appendix D). In comparison to normative data available for men aged 18 – 26 years old (Lavender et al., 2010), males in the type 1 diabetes group scored lower across all subscales. Males in the asthma group scored lower on the restraint and weight concern scales and higher on the eating concern, shape concern and global scales (Appendix D).

3.4.1.1 Behavioural items on the EDE-Q.

In addition to the continuous scale scores (Section 3.4.1), the EDE-Q includes questions about the frequency of certain eating disordered behavioural items over the past 28 days. The frequency of participants with type 1 diabetes and asthma who reported engaging in each of these behaviours at least once and on a regular basis is provided (Table 9).

Participants in the type 1 diabetes group were also asked about frequency of insulin misuse and omission for the purpose of weight control over the past 28 days. Two participants with type 1 diabetes reported insulin misuse. One male participant reported having under-dosed on his insulin on three occasions and omitted it entirely on two occasions as a means of controlling his weight and shape. The other participant was female and disclosed having under-dosed once and omitted taking her insulin entirely on another occasion for the purpose of weight control.
Table 9

*Frequency of Disordered Eating Behaviours as Reported on the EDE-Q*

<table>
<thead>
<tr>
<th>ED behaviours</th>
<th>Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Regular&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Binge eating episode</td>
<td>26.2%</td>
<td>18.5%</td>
</tr>
<tr>
<td></td>
<td>(n = 17)</td>
<td>(n = 12)</td>
</tr>
<tr>
<td>Self-induced vomiting</td>
<td>4.5%</td>
<td>1.5%</td>
</tr>
<tr>
<td></td>
<td>(n = 3)</td>
<td>(n = 1)</td>
</tr>
<tr>
<td>Laxative use</td>
<td>1.5%</td>
<td>1.5%</td>
</tr>
<tr>
<td></td>
<td>(n = 1)</td>
<td>(n = 1)</td>
</tr>
<tr>
<td>Excessive exercise</td>
<td>33.8%</td>
<td>27.7%</td>
</tr>
<tr>
<td></td>
<td>(n = 22)</td>
<td>(n = 18)</td>
</tr>
</tbody>
</table>

Note. Regular occurrence was defined as 4 or more episodes over the past 28 days, as described by Lavender et al. (2010)

<sup>a</sup>Any = Any occurrence; <sup>b</sup>Regular = Regular occurrence.

3.4.2 Overview of descriptive statistics for the independent study variables.

Descriptive statistics, including the minimum/maximum, mean and skewness values for each variable are provided for both groups (Tables 10 & 11). Participants in both groups reported lower levels of self-esteem compared to normative data for males and females aged 16-18 years old (Bagley & Mallick, 2001; Appendix D). Statistical analyses of between group differences for the PBI and RSES are explored in Section 3.5.5 as these relate to study Hypothesis 5. Significant differences were found between scores on all subscales of the DASS-21 apart from the stress subscale (Appendix E). Participants in the asthma group reported higher levels of depression, anxiety and global negative emotion than the type 1
diabetes group. No significant differences were found between reports of illness severity (Appendix E).

Table 10

*Descriptive Statistics for the Study Variables for the Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Min</th>
<th>Max</th>
<th>M (SD)</th>
<th>Skewness (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PBI - Care</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>64</td>
<td>12</td>
<td>36</td>
<td>28.98 (6.15)</td>
<td>-1.19 (.30)</td>
</tr>
<tr>
<td>Paternal</td>
<td>64</td>
<td>0</td>
<td>36</td>
<td>24.42 (9.14)</td>
<td>-.881 (.30)</td>
</tr>
<tr>
<td>PBI - Overprotection</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>64</td>
<td>0</td>
<td>31</td>
<td>9.70 (6.31)</td>
<td>.810 (.30)</td>
</tr>
<tr>
<td>Paternal</td>
<td>64</td>
<td>0</td>
<td>27</td>
<td>9.12 (6.65)</td>
<td>.782 (.30)</td>
</tr>
<tr>
<td>RSES</td>
<td>65</td>
<td>6</td>
<td>30</td>
<td>19.29 (6.58)</td>
<td>-.32 (.30)</td>
</tr>
<tr>
<td>DASS-21</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>64</td>
<td>0</td>
<td>42</td>
<td>10.90 (11.26)</td>
<td>1.10 (.30)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>64</td>
<td>0</td>
<td>42</td>
<td>9.60 (10.00)</td>
<td>1.33 (30)</td>
</tr>
<tr>
<td>Stress</td>
<td>64</td>
<td>0</td>
<td>42</td>
<td>12.72 (10.79)</td>
<td>.84 (.30)</td>
</tr>
<tr>
<td>Global</td>
<td>64</td>
<td>0</td>
<td>42</td>
<td>11.08 (9.82)</td>
<td>1.13 (.30)</td>
</tr>
<tr>
<td>Illness severity VAS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>63</td>
<td>0</td>
<td>80</td>
<td>28.52 (23.12)</td>
<td>.718 (.30)</td>
</tr>
<tr>
<td>Past worst</td>
<td>63</td>
<td>0</td>
<td>100</td>
<td>62.60 (28.53)</td>
<td>-.70 (.30)</td>
</tr>
</tbody>
</table>

*Note.* PBI = Parental Bonding Instrument; RSES = Rosenberg Self Esteem Scale; DASS-21 = Depression, Anxiety, Stress Scale 21; VAS = Visual Analogue Scale.
Table 11

*Descriptive Statistics for the Study Variables for the Asthma Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Min</th>
<th>Max</th>
<th>M (SD)</th>
<th>Skewness (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PBI - Care</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>37</td>
<td>6</td>
<td>36</td>
<td>27.44 (7.52)</td>
<td>-1.12 (.39)</td>
</tr>
<tr>
<td>Paternal</td>
<td>37</td>
<td>3</td>
<td>36</td>
<td>23.08 (9.28)</td>
<td>-.53 (.39)</td>
</tr>
<tr>
<td><strong>PBI - Overprotection</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>37</td>
<td>2</td>
<td>32</td>
<td>13.62 (7.39)</td>
<td>.63 (.39)</td>
</tr>
<tr>
<td>Paternal</td>
<td>37</td>
<td>2</td>
<td>30</td>
<td>13.77 (7.84)</td>
<td>.31 (.39)</td>
</tr>
<tr>
<td><strong>RSES</strong></td>
<td>37</td>
<td>1</td>
<td>29</td>
<td>14.84 (6.69)</td>
<td>.01 (.39)</td>
</tr>
<tr>
<td><strong>DASS-21</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>37</td>
<td>0</td>
<td>42</td>
<td>16.49 (11.53)</td>
<td>.39 (.39)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>37</td>
<td>0</td>
<td>38</td>
<td>14.04 (11.46)</td>
<td>.75 (.39)</td>
</tr>
<tr>
<td>Stress</td>
<td>37</td>
<td>0</td>
<td>42</td>
<td>16.17 (10.37)</td>
<td>.37 (.39)</td>
</tr>
<tr>
<td>Global</td>
<td>37</td>
<td>1.33</td>
<td>40.67</td>
<td>15.56 (9.73)</td>
<td>.74 (.39)</td>
</tr>
<tr>
<td><strong>Illness severity VAS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>37</td>
<td>0</td>
<td>85</td>
<td>25.82 (24.13)</td>
<td>.83 (.39)</td>
</tr>
<tr>
<td>Past worst</td>
<td>37</td>
<td>10</td>
<td>100</td>
<td>66.86 (25.64)</td>
<td>-.65 (.39)</td>
</tr>
</tbody>
</table>

*Note.* PBI = Parental Bonding Instrument; RSES = Rosenberg Self Esteem Scale; DASS-21 = Depression, Anxiety, Stress Scale 21; VAS = Visual Analogue Scale.

### 3.4.3 Distribution of the data.

The distribution of the scores for each independent and dependent variable were examined using skewness values, histograms and normality tests. Skewness values (Tables 10 & 11) and observation of the histograms indicated that, in general, the scores for the type 1
diabetes group were more skewed than for the asthma group. Overall, scores were positively skewed for the diabetes group, with the exception of the PBI care subscales and self-esteem, which were negatively skewed.

Table 12

*Kolmogorov-Smirnov Statistical Test for Normality for the Independent and Dependent Study*

*Variables: Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Kolmogorov-Smirnov Statistic</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.17</td>
<td>65</td>
<td>.000**</td>
</tr>
<tr>
<td></td>
<td>PBI - Care</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.17</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>Paternal</td>
<td>.11</td>
<td>64</td>
<td>.068</td>
</tr>
<tr>
<td></td>
<td>PBI - Overprotection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.09</td>
<td>64</td>
<td>.200</td>
</tr>
<tr>
<td>Paternal</td>
<td>.14</td>
<td>64</td>
<td>.003**</td>
</tr>
<tr>
<td></td>
<td>RSES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>.11</td>
<td>65</td>
<td>.038*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DASS-21 global scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>.17</td>
<td>63</td>
<td>.000**</td>
</tr>
<tr>
<td>Past worst</td>
<td>.13</td>
<td>63</td>
<td>.014**</td>
</tr>
</tbody>
</table>

*p < .05; p** < .01.
Table 13

Kolmogorov-Smirnov Statistical Test for Normality for the Independent and Dependent Study

Variables: Asthma Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Kolmogorov-Smirnov</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.14</td>
<td>37</td>
<td>.063</td>
</tr>
<tr>
<td>PBI – Care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.15</td>
<td>37</td>
<td>.036*</td>
</tr>
<tr>
<td>Paternal</td>
<td>.11</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>PBI – Overprotection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.12</td>
<td>37</td>
<td>.194</td>
</tr>
<tr>
<td>Paternal</td>
<td>.11</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>RSES</td>
<td>.11</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>DASS-21 global scale</td>
<td>.15</td>
<td>37</td>
<td>.026*</td>
</tr>
<tr>
<td>Illness severity VAS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>.14</td>
<td>37</td>
<td>.057</td>
</tr>
<tr>
<td>Past worst</td>
<td>.13</td>
<td>37</td>
<td>.130</td>
</tr>
</tbody>
</table>

*p < .05.

The results of the Kolmogorov-Smirnov statistical test for normality for each variable indicated that scores on the EDE-Q and RSES significantly deviated from a normal distribution curve for the type 1 diabetes group (Table 12). Test statistics and significance values were mixed for the scores on the PBI subscales. On the contrary, the outcome for the asthma group showed that, with the exception of the maternal care subscale of the PBI and
the DASS-21 global scale, scores on each of the variables did not significantly deviate from a normal distribution (Table 13). As the scores did not consistently follow a normal distribution across both groups, the assumption of normality has not been met. The data were resistant to transformation attempts using logarithm and square-root transformations (Appendix F). Therefore, for the purposes of this study non-parametric statistical tests were utilised.

3.4.4 Confounding Variables.

For the purposes of this study, a confounding variable was defined as one that significantly correlated with both the independent and dependent variables and was considered to be theoretically independent of the association between the variables (Field, 2013). Therefore, Spearman’s Rho Correlation Coefficient matrices were reviewed for the type 1 diabetes group and asthma groups individually. The global negative emotions scale (DASS-21) was found to be correlated with the independent and dependent variables in both groups. However, as this could be considered to contribute to the relationships between parenting style, self-esteem and eating disturbances, it was not controlled for statistically.

3.5 Hypotheses Testing

In this section statistical analyses relating to each hypothesis will be described. Outcomes of statistical tests were deemed significant if \( p < .05 \), as is conventional for exploratory studies (Field, 2013). For all hypotheses, level of eating disturbances was determined by the global EDE-Q scale. Individual subscales of this measure are also included in the analyses to look for patterns of ED symptomology in the findings. Consideration as to whether or not the findings support each hypothesis will be provided.

3.5.1 Hypothesis one: Adolescent-perceived parental care will be negatively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.

Spearman’s Rho one-tailed correlations were utilised in order to test the prediction
that maternal and paternal care were negatively associated with level of eating disturbances (global EDE-Q scale) in the type 1 diabetes group and the asthma group (Tables 14 & 15). The outcome shows highly significant negative correlations between paternal care and all subscales of the EDE-Q for the type 1 diabetes group. Therefore, as level of perceived paternal care reduces the level of eating disturbances increases. Other than for the restraint subscale, moderate strength correlation coefficients were demonstrated between paternal care and eating disturbances (Cohen, 1992). For the diabetes group, maternal care was also found to be significantly negatively correlated with all subscales of the EDE-Q, apart from the restraint subscale. However, despite reaching statistical significance at \( p < .05 \), the coefficients should be treated cautiously as they are weak (range from \( r = -.219 \) to \( -.252 \)) and indicate only 5% - 6% shared variance between the variables (calculated using the formula \( r^2 \times 100 \); Field, 2013).

Table 14

*Spearman’s Rho Correlations Between the EDE-Q Scales and Parent Care Subscales of the PBI for the Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Type 1 diabetes group (n = 65)</th>
<th>Maternal care (r)</th>
<th>Paternal care (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td></td>
<td>-.114</td>
<td>-.252*</td>
</tr>
<tr>
<td>Eating concern</td>
<td></td>
<td>-.252*</td>
<td>-.442**</td>
</tr>
<tr>
<td>Weight concern</td>
<td></td>
<td>-.219*</td>
<td>-.331**</td>
</tr>
<tr>
<td>Shape concern</td>
<td></td>
<td>-.244*</td>
<td>-.395**</td>
</tr>
<tr>
<td>Global EDE-Q</td>
<td></td>
<td>-.233*</td>
<td>-.401***</td>
</tr>
</tbody>
</table>

* Significant at \( p < .05 \); **Significant at \( p < .01 \); ***Significant at \( p < .001 \).
Eating Disturbances in Chronic Illness

Table 15

*Spearman’s Rho Correlations Between the EDE-Q Subscales and Parent Care Subscales of the PBI for the Asthma Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Asthma group ($n = 37$)</th>
<th>Maternal care ($r$)</th>
<th>Paternal care ($r$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td></td>
<td>-.285</td>
<td>-.154</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-.479**</td>
<td>-.162</td>
<td></td>
</tr>
<tr>
<td>Weight concern</td>
<td>-.516**</td>
<td>-.180</td>
<td></td>
</tr>
<tr>
<td>Shape concern</td>
<td>-.494**</td>
<td>-.188</td>
<td></td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-.449</strong></td>
<td><strong>-.169</strong></td>
<td></td>
</tr>
</tbody>
</table>

** Significant at $p < .01$.

For the asthma group maternal care was significantly associated with eating disturbances across the subscales of the EDE-Q, excluding the restraint subscale. This suggests that global level of eating disturbances increased as perceived level of maternal care reduced. The coefficients ranged from $r = -.449$ to -.516, indicating moderate – strong correlation coefficients. No other significant associations were found for the asthma group.

Taken together, the results indicate that hypothesis one, which predicted that parent care would be negatively associated with level of eating disturbances in both groups, was mainly supported. Higher levels of global eating disturbances were associated with lower maternal care in the asthma group, and lower maternal and paternal care in the type 1 diabetes group.

**3.5.2 Hypothesis two: Adolescent-perceived parent overprotection will be positively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.**

Hypothesis two predicted that adolescent reports of parent overprotection would be
positively associated with level of eating disturbances, measured by the global EDE-Q scale, in both groups. This hypothesis was tested using one-tailed Spearman’s Rho Correlation Coefficients (Tables 16 & 17).

Table 16

Spearman’s Rho Correlations Between the EDE-Q Subscales and Parent Overprotection

Subscales of the PBI for the Type 1 Diabetes Group

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Type 1 diabetes group (n = 64)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maternal overprotection (r)</td>
</tr>
<tr>
<td>Restraint</td>
<td>.312**</td>
</tr>
<tr>
<td>Eating concern</td>
<td>.359**</td>
</tr>
<tr>
<td>Weight concern</td>
<td>.416**</td>
</tr>
<tr>
<td>Shape concern</td>
<td>.367**</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>.395</strong></td>
</tr>
</tbody>
</table>

*Significant at p < .05; **Significant at p < .01.

In the type 1 diabetes group both maternal and paternal overprotection were significantly positively correlated with all scales of the EDE-Q; Higher levels of parent overprotection were associated with higher levels of eating disturbances. The coefficients for the global EDE-Q scale were $r = .395$ and $ .433$, indicating moderate strength coefficients. On the contrary, in the asthma group no significant relationships were found between either paternal or maternal overprotection and scores on the global EDE-Q scale. Maternal overprotection was found to be significantly correlated with eating concern and weight concern in the asthma group; however, only weak correlation coefficients were found. Therefore, hypothesis two, which predicted that adolescent reports of maternal and paternal overprotection would
be positively associated with level of eating disturbances, as measured by the global EDE-Q scale, is partially supported. Adolescent reports of maternal and paternal overprotection were significantly positively associated with global level of eating disturbances in the type 1 diabetes group, but not in the asthma group.

Table 17

Spearman’s Rho Correlations Between the EDE-Q Subscales and Parent Overprotection

Subscales of the PBI for the Asthma Group

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Asthma group (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maternal overprotection ($r$)</td>
</tr>
<tr>
<td>Restraint</td>
<td>.179</td>
</tr>
<tr>
<td>Eating concern</td>
<td>.288*</td>
</tr>
<tr>
<td>Weight concern</td>
<td>.282*</td>
</tr>
<tr>
<td>Shape concern</td>
<td>.253</td>
</tr>
<tr>
<td>Global EDE-Q</td>
<td><strong>.265</strong></td>
</tr>
</tbody>
</table>

*Ssignificant at $p < .05.$

3.5.2.1 Parenting style and eating disturbances.

Participants’ responses on the PBI were categorised into high or low levels of parent care and overprotection, and then into parenting style, as described by Parker (1983; Table 18). For the type 1 diabetes group Kruskal-Wallis tests revealed a statistically significant difference in global EDE-Q scores across the four different parenting styles for mothers ($\chi^2 [3, n = 64] = 16.95, p < .001$) and fathers ($\chi^2 [3, n = 64] = 8.78, p < .032$). The optimal parenting group reported a lower mean score for eating disturbances compared to the
affectionate control, affectionless control and neglectful parenting groups for mothers and fathers alike (Appendix G). No significant differences were found in EDE-Q global score across the four parenting categories for the asthma group.

Table 18

<table>
<thead>
<tr>
<th>Variable Type</th>
<th>Type 1 diabetes (n = 64)</th>
<th>Asthma (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maternal</td>
<td>Paternal</td>
</tr>
<tr>
<td>PBI category</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low care</td>
<td>26.6 %</td>
<td>43.8%</td>
</tr>
<tr>
<td>High overprotection</td>
<td>24.6 %</td>
<td>28.1%</td>
</tr>
<tr>
<td>Parenting style</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affectionate control</td>
<td>18.8%</td>
<td>10.9%</td>
</tr>
<tr>
<td>Affectionless control</td>
<td>6.3%</td>
<td>17.2%</td>
</tr>
<tr>
<td>Optimal parenting</td>
<td>56.3%</td>
<td>51.6%</td>
</tr>
<tr>
<td>Neglectful parenting</td>
<td>18.8%</td>
<td>20.3%</td>
</tr>
</tbody>
</table>

Note. PBI category taken from Parker (1983).

3.5.3 Hypothesis three: Adolescent self-esteem will be negatively associated with level of eating disturbances in the type 1 diabetes group and the asthma group.

In order to test the prediction that self-esteem would be negatively associated with scores on the global EDE-Q scale in each group, one-tailed Spearman’s Rho correlation coefficients were used (Table 19). Strong negative associations were found between self-esteem and all scales of the EDE-Q for both groups, indicating that self-reported lower levels
of self-esteem were associated with higher levels of eating disturbances regardless of illness diagnosis.

Table 19

Spearman’s Rho Correlations Between the EDE-Q Subscales and Rosenberg Self Esteem Scale for the Type 1 Diabetes Group and the Asthma Group

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-.479**</td>
<td>-.340*</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-.697**</td>
<td>-.596**</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-.655**</td>
<td>-.636**</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-.721**</td>
<td>-.639**</td>
</tr>
<tr>
<td>Global EDE-Q</td>
<td>-.698**</td>
<td>-.574**</td>
</tr>
</tbody>
</table>

*Significant at $p < .05$; ** Significant at $p < .01$.

3.5.4 Hypothesis four: Level of eating disturbances will be higher in the type 1 diabetes group than the asthma group.

Between groups analysis tested the hypothesis that the type 1 diabetes group would report higher levels of eating disturbances, as measured by the global EDE-Q scale, than the asthma group. Two-tailed Mann Whitney U tests revealed significant differences between the two groups for all scales of the EDE-Q apart from the eating concern scale, which was approaching significance at $p < .05$ (Table 20). The median scores for all scales of the EDE-Q were higher in the asthma group than in the type 1 diabetes group, indicating that the asthma group reported significantly higher levels of eating disturbances. The outcome of this analysis indicates that hypothesis four, which predicted that global levels of eating disturbances would
be higher in the type 1 diabetes group, was not supported.

Table 20

*Mann Whitney U Test for Differences in the Median Value of EDE-Q Subscales Between the Type 1 Diabetes Group and the Asthma Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Z statistic</th>
<th>p value</th>
<th>Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-2.85</td>
<td>.004**</td>
<td>.20</td>
<td>1.20</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-1.93</td>
<td>.053</td>
<td>.20</td>
<td>1.20</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-3.14</td>
<td>.002**</td>
<td>.80</td>
<td>2.60</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-3.27</td>
<td>.001**</td>
<td>1.50</td>
<td>2.75</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-3.24</strong></td>
<td><strong>.001</strong></td>
<td><strong>.76</strong></td>
<td><strong>2.26</strong></td>
</tr>
</tbody>
</table>

**p < .01.

3.5.5 Hypothesis five: There will be differences in the level of adolescent-perceived parent care, parent overprotection and adolescent self-esteem between the type 1 diabetes group and the asthma group.

Hypothesis five stated that differences would be observed between the type 1 diabetes group and the asthma group in the level of parent care, parent overprotection and self-esteem reported. Two-tailed Mann Whitney U tests were used in order to test this hypothesis (Table 21). Contrary to the prediction, the outcomes suggest no difference in the level of maternal or paternal care between the two illness groups. However, significant differences were demonstrated for maternal and paternal overprotection, with the asthma group reporting higher levels of overprotection than the type 1 diabetes group. Similarly, the asthma group had a significantly lower median value for self-esteem, indicating lower levels of self-esteem.
in this group than in the type 1 diabetes group. Therefore, this hypothesis is partially supported.

Table 21

*Mann Whitney U Test Statistics for Differences in Parent Care, Parent Overprotection and Self-Esteem Between the Type 1 Diabetes Group and Asthma Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Z statistic</th>
<th>p value</th>
<th>Mdn value Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal care</td>
<td>-.72</td>
<td>.471</td>
<td>31.00</td>
<td>29.45</td>
</tr>
<tr>
<td>Paternal care</td>
<td>-.73</td>
<td>.467</td>
<td>26.00</td>
<td>25.00</td>
</tr>
<tr>
<td>Maternal overprotection</td>
<td>-2.60</td>
<td>.009**</td>
<td>9.00</td>
<td>13.00</td>
</tr>
<tr>
<td>Paternal overprotection</td>
<td>-2.89</td>
<td>.004**</td>
<td>7.00</td>
<td>14.00</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>-3.00</td>
<td>.003**</td>
<td>20.00</td>
<td>16.00</td>
</tr>
</tbody>
</table>

**p < .01.

3.6 Additional Analyses

Additional exploratory analyses were carried out to determine whether type of chronic illness moderated the associations observed between self-esteem, parent care and overprotection, and eating disturbances. Predictor variables were chosen based on whether or not they significantly predicted the outcome variable in a simple regression analysis. Based on this self-esteem, as measured by the RSES, and parent care and overprotection, as measured by the PBI, were selected for further analyses; the outcome variable was global score on the EDE-Q. The moderating effect of chronic illness was considered individually for each of the selected predictor variables. This approach to analysis was selected as the sample
size was insufficient to carry out a regression analysis with multiple variables and interactions (Field, 2013). The moderation analysis was carried out using the PROCESS Procedure for SPSS (Hayes, 2013).

3.6.1 Moderation Analyses.

Simple regression analysis was used to determine the predictive ability of paternal overprotection in relation to global scores on the EDE-Q for the whole sample. In the simple regression model paternal overprotection explained 12% ($R^2$ adjusted) of the variance, where $F (1, 101) = 15.15, p < .000$. The assumption of linearity was met and inspection of the regression residual histograms for the regression model suggested that the distributions of the residuals were positively skewed. This deviation from normality was confirmed by Kolmogorov-Smirnov tests of normality. However, as this was an exploratory analysis and the model parameters were of more interest than significance testing, it was deemed appropriate to carry out the regression analysis, as described in Field (2013).

### Table 22

<table>
<thead>
<tr>
<th>Variable</th>
<th>b</th>
<th>SE B</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-1.27</td>
<td>.73</td>
<td>-1.73</td>
<td>.087</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>1.63</td>
<td>.54</td>
<td>3.05</td>
<td>.003**</td>
</tr>
<tr>
<td>Paternal overprotection</td>
<td>.19</td>
<td>.06</td>
<td>3.17</td>
<td>.002**</td>
</tr>
<tr>
<td>Paternal overprotection X diagnosis</td>
<td>-.09</td>
<td>.04</td>
<td>-2.25</td>
<td>.026*</td>
</tr>
</tbody>
</table>

Note. $R^2 = .21$.

*Significant at $p < .05$; ** Significant at $p < .01$.  

The paternal overprotection interaction model (Table 22) was found to be significant \((b = -0.09, t = -2.25, p = .026)\). This indicates that type of diagnosis significantly moderated the relationship observed between parent overprotection and global EDE-Q. The association between the two variables was stronger for the type 1 diabetes group compared to the asthma group (Figure 3).

*Figure 3.* Regression of eating disturbances on the interaction between paternal overprotection and chronic illness diagnosis.
Type of chronic illness was not found to significantly moderate the relationships observed between global EDE-Q scores and self-esteem, maternal and paternal care, and maternal overprotection (Appendix I).

3.7 Summary of Results

In summary, the outcome of the correlation analyses indicated that hypotheses one and two were partially supported, and hypothesis three was fully supported. Significant relationships were found between the variables of interest, namely eating disturbances (measured by the global EDE-Q scale) and parent care, parent overprotection and self-esteem. The exact nature of these relationships differed between illness groups. There was a pattern of significant moderate-sized negative associations between adolescent-perceived paternal care and global eating disturbances in the type 1 diabetes group. In contrast, for the asthma group there was a pattern of significant moderate-sized negative associations between maternal care and global eating disturbances.

Within the type 1 diabetes group the outcomes of the analysis indicated that higher levels of parent overprotection, both maternal and paternal, were significantly associated with higher levels of global eating disturbances. Maternal and paternal overprotection were not found to be significantly correlated with global EDE-Q scores in the asthma group. Tentative supplementary analyses suggested that the relationship between paternal overprotection and global eating disturbances was significantly moderated by type of chronic illness, with the relationship being stronger for those with type 1 diabetes than asthma. Self-esteem was found to be significantly negatively associated with scores on the global EDE-Q scale in both illness groups; moderate to strong correlation coefficients were shown for this relationship.

Hypothesis four was not supported as the asthma group reported significantly higher levels of global eating disturbances than the type 1 diabetes group. Interestingly, supplementary analysis revealed no significant between group differences when comparing
females in each illness group. Between group analysis revealed significant differences in level of parent overprotection and self-esteem. The asthma group reported significantly higher levels of parent overprotection, both maternal and paternal, and lower levels of self-esteem, compared with the type 1 diabetes group. No significant differences were found between groups for parent care. Therefore, hypothesis five is partially supported.
CHAPTER FOUR

4. Discussion

4.1 Chapter Overview

The primary aim of the current study was to explore the relationship between individual and interpersonal factors and eating disturbances in adolescents with type 1 diabetes and asthma. More specifically, the study was designed to investigate relationships between: 1) adolescent-perceived parent care, 2) adolescent-perceived parent overprotection, and 3) adolescent self-esteem, and eating disturbances within each group. The associations between parenting factors, self-esteem and eating disturbances have been investigated in adolescents with type 1 diabetes in previous studies. However, these studies have been methodologically limited due to a persistent lack of comparison group. It has remained unclear whether associations found between the factors are common to other chronic illness groups or are unique to type 1 diabetes. Chronic illness creates difficult circumstances for young people and their families to navigate and may increase vulnerability to psychopathology (Lubkin & Larsen, 2006; Neumark-Sztainer et al., 1995). Therefore, the use of a chronic illness comparison group and the investigation of parenting styles, as defined by Parker (1979), are novel elements of this study.

In this chapter the outcomes of the study will be considered for each hypothesis in turn and in relation to previous literature. Theoretical and clinical implications will be discussed and suggestions made for future research. A methodological critique of the strengths and weaknesses of the study will be provided. Finally, the chapter will close with a summary and conclusion.

4.2 Evaluation of the Findings in Relation to each Hypothesis

4.2.1 Hypothesis one: Adolescent-perceived parent care will be negatively associated with eating disturbances in the type 1 diabetes and the asthma group.
The findings indicated that adolescent reports of both paternal and maternal care were significantly negatively associated with scores on the global EDE-Q scale for those with type 1 diabetes. This means that in this group lower levels of parent care were associated with higher levels of global eating disturbances; the effect was stronger for paternal care than maternal care. Within the asthma group maternal care was also significantly negatively correlated with scores on the global EDE-Q scale. This indicates that in this group lower levels of maternal care were associated with higher levels of eating disturbances. No significant relationships were found between paternal care and level of global eating disturbances in the asthma group. Overall the results indicate that hypothesis one was largely supported, as significant negative associations were found between eating disturbances and both maternal and paternal care in the type 1 diabetes group, and maternal care in the asthma group. Taken together these findings are largely consistent with research in the general population, which has found both maternal and paternal care to be negatively associated with eating disturbances in females (Jones et al., 2006; Swanson et al., 2010; Turner et al., 2005).

At the time of writing the only published study to investigate parent care in relation to eating disturbances in chronic illness found significant associations between the variables (Neumark-Sztainer et al., 1998), though this did not make use of standardised measures and did not differentiate between different types of chronic illness in the sample. Therefore, the current study is the first to investigate these variables in adolescents with type 1 diabetes and asthma using standardised measures. The findings reported here are in agreement with previous studies that have more broadly found the quality of parent-child relationship to be negatively associated with eating disturbances in adolescent females with type 1 diabetes (e.g. Colton et al., 2007; Maharaj et al., 2003). To date no studies have investigated the relationship between parenting dimensions and eating disturbances in males with type 1 diabetes.
4.2.2 Hypothesis two: Adolescent-perceived parent overprotection will be positively associated with level of eating disturbances in the type 1 diabetes and asthma group.

In this study both maternal and paternal overprotection were significantly positively correlated with scores on the EDE-Q global scale in the type 1 diabetes group. Therefore, higher levels of parent overprotection were associated with higher levels of eating disturbances for participants with type 1 diabetes. This is the first study to investigate these variables in a type 1 diabetes sample, however, the findings are consistent with literature from the general population which has found greater parent overprotection to be associated with eating disturbances in females (Deas et al., 2011; Meyer & Gillings, 2003; Turner et al., 2005) and lower body satisfaction in males (Tata et al., 2001).

This pattern was not replicated in the asthma group as global levels of eating disturbances were not significantly associated with either maternal or paternal overprotection. In line with these between-group differences, supplementary analyses tentatively suggested that the relationship between paternal overprotection and eating disturbances was significantly moderated by type of chronic illness; the relationship was stronger for those with type 1 diabetes. Taken together therefore, hypothesis two was partially supported as significant positive correlations between the variables were found for the type 1 diabetes group but not the asthma group.

This was the first study to investigate the role of parent overprotection in eating disturbances in chronic illness groups. The results indicate that, for this sample, parent overprotection was related to eating disturbances for adolescents with type 1 diabetes but not for those with asthma. The difference between the two groups in relation to these variables may be explained in part by methodological limitations, such as gender proportions in the
two groups and small sample size in the asthma group (Section 4.5). This is discussed further in Section 4.3.2.2.

4.2.3 Hypothesis three: Adolescent self-esteem will be negatively associated with level of eating disturbances in the type 1 diabetes and asthma group.

The findings of this study indicate that self-esteem was significantly negatively associated with eating disturbances in both groups. Moderate to strong correlation coefficients were shown for this relationship across all subscales and the global scale of the EDE-Q. This indicates that lower levels of self-esteem were related to higher levels of eating disturbances as predicted, and therefore hypothesis three is supported. These findings are consistent with previous research carried out in the general population which has found lower self-esteem to be associated with, and predictive of, eating disturbances in male and female adolescents (Button et al., 1996; Olivardia et al., 2004; Polivy & Herman, 2002). They are also in agreement with cross-sectional and longitudinal investigations in samples of female adolescents with type 1 diabetes, which have found self-esteem to be related to, and predict new onset of, eating disturbances (Colton et al., 2007; Maharaj et al., 2003; Olmsted et al., 2008).

The comparable associations between self-esteem and eating disturbances in adolescents with type 1 diabetes and asthma is similar to that reported by Vila et al. (1995). Using a between-groups study design they divided a sample of female adolescents with and without type 1 diabetes into groups of those who were, and were not, obese. Self-esteem was not associated with eating disturbances in females with or without type 1 diabetes, unless they were also categorised as being obese. This suggests that rather than presence of chronic illness, it may be weight status that increases vulnerability to low self-esteem and eating psychopathology. It was not within the scope of the study reported here to investigate the associations between weight status, self-esteem and eating disturbances. However, given that
25.4% and 17.1% of the type 1 diabetes and asthma groups respectively were classified as being overweight or obese, and that those with this weight status reported significantly higher levels of global eating disturbances, further research in this area is warranted.

4.2.4 Hypothesis four: Level of eating disturbances will be higher in the type 1 diabetes group than the asthma group.

Hypothesis four was not supported by the findings in this study as, unexpectedly, the asthma group reported significantly higher levels of eating disturbances on the global EDE-Q scale than the type 1 diabetes group. Additionally, four participants in the asthma group reported having a past diagnosis of ED, compared to one participant in the type 1 diabetes group. It is also important to note that a minority of participants in each group reported engaging in self-induced vomiting and laxative use, which are behaviours rarely seen outside of ED populations (Grave, Calugi, & Marchesini, 2009). This indicates that some participants in both groups reported clinically relevant ED symptoms. This is further supported by the finding that mean scores on the EDE-Q for females in both groups were higher than those reported in normative data for a community sample of 16-35 year old females (Fairburn & Beglin, 1994). In this study, males with type 1 diabetes reported lower levels of eating disturbances than indicated by normative data for adult males (Lavender et al., 2010). This is in contrast with a small body of evidence that indicates males, as well as females, with type 1 diabetes are at an increased risk of developing eating disturbances and EDs (Neumark-Sztainer et al., 1996; Svensson et al., 2003). However, these studies have been hampered by use of non-standardised measures and small sample sizes and the findings must be treated cautiously. For males with asthma the pattern was inconclusive, as mean scores were higher than normative data for the eating concern, shape concern, and global scale and lower for the restraint and weight scale. This inconsistent pattern is likely due to the small number of males with asthma in the sample (n = 8).
This is the first study to compare eating disturbances in young people specifically with type 1 diabetes and asthma. Previous findings have indicated either no difference in eating disturbances between chronic illness groups, or higher levels in type 1 diabetes. For example, in a population study of American adolescents, those with a self-reported chronic illness ($n = 2149$) reported higher levels of eating disturbances, measured using an ad-hoc questionnaire, compared to their healthy peers ($n = 1381$; Neumark-Sztainer et al., 1995). The authors reported that this finding was consistent across all types of chronic illness, including type 1 diabetes and asthma, and was not limited to those with a dietary element to treatment, as had been predicted. Contrary to this, Smith et al. (2008) found that those with type 1 diabetes were significantly more likely to report ED symptoms, measured using the EDE-Q, than those with scoliosis or healthy controls. Given this, the finding that eating disturbances in the asthma group were not just comparable to those in the type 1 diabetes group, but higher, is an interesting and novel finding.

The elevated level of eating disturbances evidenced in the asthma group is partly supported by a population-based study carried out by Moreau et al. (2009). They investigated ED symptoms in 11,700 adolescents, of which 11.7% reported a diagnosis of asthma. Using an ad-hoc measure of ED symptoms, which was based on DSM-IV criteria for EDs (American Psychiatric Association, 2000), they found that the asthma group reported significantly higher levels of eating disturbances than their healthy peers. More specifically, the asthma group reported being more likely to miss meals and diet, having greater weight concern, and being less likely to eat for pleasure, compared to those without asthma. The outcomes of the study also indicated that those with asthma were more likely to be overweight compared to their healthy peers, and that those who were both asthmatic and overweight were the most likely to report the presence of eating disturbances. Taken together, the findings from the study reported here and those reported by Moreau et al. indicate that
young people with asthma may be more vulnerable to developing ED symptoms than their healthy peers, as are young people with type 1 diabetes (Daneman, 2002).

This finding could be understood in light of the impact of illness-related weight gain evidenced in young people with both type 1 diabetes and asthma (Bryden et al., 1999; Moreau et al., 2009). Weight gain and higher BMI are known risk factors for the development of ED symptoms in the general population (Calzo et al., 2012). This hypothesis is explored further in Section 4.3.1. The higher level of eating psychopathology reported by the asthma group may also be understood, in part, by methodological limitations of the study. Firstly, the scores may be partly elevated by an increased likelihood of self-selection bias in the asthma group compared to the type 1 diabetes group (Section 4.5.2.2). Secondly, there was a significantly higher proportion of females (78.4%) in the asthma group compared to the type 1 diabetes group (53.8%). Research clearly shows that females report higher levels of ED symptoms than males (Smink et al., 2012). Therefore, having more females in the asthma group may have increased the mean scores on the EDE-Q. This hypothesis is supported by the fact that no significant differences were found when comparing EDE-Q scores for females only within the type 1 diabetes and asthma groups. This could tentatively be interpreted as indicating no difference in eating psychopathology between the groups when gender is taken into consideration. However, even if this hypothesis has validity the outcomes remain of interest, as this is the first study to compare eating disturbances in young people with asthma and find levels of eating psychopathology that are at least comparable to a sample of young people with type 1 diabetes.

4.2.5 Hypothesis five: There will be differences in the level of adolescent-perceived parent care, parent overprotection and self-esteem between the type 1 diabetes and asthma group.
Hypothesis five was partially supported, as significant between group differences were found for parent overprotection and self-esteem, but not for parent care. Less attention has been given to the investigation of levels of parent care in chronic illness populations than parent overprotection. However, previous research has found no significant differences in parent care when comparing adolescents with chronic illnesses, including those with type 1 diabetes (e.g. Graue et al., 2005). Further, a recent meta-analysis revealed a significant, but small, effect size for lower levels of parent warmth/care in children with chronic illnesses compared to healthy controls (Pinquart, 2013). Pinquart proposed that this may be due to the additional burden associated with parenting children with chronic illnesses, which may lead to higher levels of stress, depression and feelings of being overwhelmed (e.g. Power & Franck, 2008). These difficulties may in turn reduce parents’ capacity to offer sensitive and containing care for their children. For example, parental depression has been found to be associated with lower levels of warmth and nurturance in parents of children with asthma (Lim, Wood & Miller, 2008).

In support of hypothesis five, significant differences were found in adolescent reports of maternal and paternal overprotection, with the asthma group reporting higher levels of overprotection than the type 1 diabetes group. This indicates that participants with type 1 diabetes perceived their parents to be less controlling, restrictive and invasive than those in the asthma group. Higher levels of parent overprotection, as measured by the PBI, have been found in previous investigations of children and adolescents with type 1 diabetes and asthma, when compared with healthy controls (Graue et al., 2005; Parker & Lipscombe, 1979; Pinquart, 2013). Interestingly, Graue et al. (2005) found levels of parent overprotection to be higher in adolescents with type 1 diabetes compared to both healthy controls and adolescents with physical disabilities. They proposed that parents of those with type 1 diabetes may be invited into a position of greater overprotection due to both the higher daily demand of
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diabetes management and parental anxiety potentially caused by a fear of serious physical consequences related to poor diabetes management (Patton, Dolan, Henry, & Powers, 2008). Parental anxiety has also been found in samples of children with asthma, particularly in relation to parents’ perceptions of their own ability to manage the unpredictable nature of asthma symptoms, their child’s ability to manage their symptoms and the long term consequences of asthma medication (Cashin, Small, & Solberf, 2008; Syndor-Greenberg & Dokken, 2000). Therefore, parental anxiety caused by treatment demands, responsibility of asthma and type 1 diabetes, and fear of serious physical illness, may lead to higher levels of parent overprotection as evidenced in these groups (Cameron et al., 2007).

No previous studies have compared the two illness groups reported here in terms of levels of parent overprotection. Hypotheses have been made about causes of heightened overprotection in both the asthma and type 1 diabetes groups, as described above. However, potential reasons for higher levels of overprotection in the asthma group compared to the type 1 diabetes group are unclear. It may be that the more unpredictable nature of asthma symptoms leads to greater parent anxiety and subsequent overprotection, as hypothesised by Parker and Lipscombe (1979). In addition, the findings may be explained in part by methodological limitations of the study. It may be that the results were influenced by the likelihood of higher levels of self-selecting bias in the asthma group given the nature of recruitment for this group (Section 4.5.2.1). Further research is needed to explore these differences further and to enable conclusions to be drawn about levels of overprotection in the two groups.

Significant differences in levels of self-esteem were found between the type 1 diabetes group and the asthma group, as predicted by hypothesis five. Participants in the asthma group reported, on average, lower self-esteem than those with type 1 diabetes. This is inconsistent with a previous study that found no differences in self-esteem, as measured by
the RSES, between groups of adolescents with sickle cell disease, asthma and type 1 diabetes (Seigel et al., 1990). This discrepancy may be explained by the limitations of the sample in this study. The lower self-esteem levels in the asthma group may be a result of this group having a significantly higher proportion of females compared to the diabetes group. This hypothesis is supported by there being no significant differences in self-esteem between females only with type 1 diabetes and asthma.

4.2.6 Summary of findings.

In summary, the findings of this study suggest that in this sample higher levels of parent overprotection were associated with higher levels of eating disturbances in the type 1 diabetes group but not in the asthma group. The observed relationships between parent care and eating disturbances were mixed, but overall patterns suggested a significant negative association between the variables. Lower self-esteem was found to be strongly related to higher eating disturbances in both groups. Overall, participants in the asthma group reported greater impairment on all measures, compared to the diabetes group. Most notably, the asthma group reported significantly higher levels of eating psychopathology than the type 1 diabetes group. The findings have been discussed in the context of previous research and the theoretical and clinical implications will now be considered.

4.3 Theoretical Implications of Findings

4.3.1 Eating disturbances in type 1 diabetes and asthma.

There is strong evidence within the literature pointing to an increased prevalence of eating disturbances in young people with type 1 diabetes. Following a meta-analysis Daneman (2002) reported an odds ratio of 2:0, indicating that those with type 1 diabetes were twice as likely to report both sub-threshold eating disturbances and threshold EDs compared to their healthy peers. Definitive conclusions about level of eating disturbances in the current sample compared to healthy peers cannot be made as a healthy comparison group was not
used. However, mean scores on all scales of the EDE-Q were higher than mean scores reported by normative data for female participants in both the asthma and type 1 diabetes groups (Appendix D); the picture was more mixed for male participants.

There have been relatively few investigations that have compared level of eating disturbances in adolescents with different chronic illnesses. Previous research has found mixed results and remains inconclusive (Section 4.2.4). This study is the first to compare eating disturbances in adolescents with type 1 diabetes and asthma and found that participants with asthma reported higher levels of eating psychopathology. However, methodological limitations of the study, in terms of the higher proportion of females in the asthma group, mean that the reliability of this finding remains unknown. Comparisons of females only indicate no differences in eating disturbances between the two groups. This may suggest that females with either type 1 diabetes or asthma are at an increased risk of developing ED symptoms.

Daneman et al. (1998) have proposed a model to explain the increased prevalence of eating disturbances seen in young people with type 1 diabetes compared to their healthy peers. This model suggests that the threshold for the expression of ED symptomology in people with type 1 diabetes is lowered compared to those without diabetes. They proposed that individual, familial and sociocultural risk factors for ED symptoms are amplified in those with type 1 diabetes due to illness-specific vulnerabilities. These include insulin-related weight gain, focus in treatment on diet and weight, and availability of insulin manipulation for weight control. However, this explanation is not sufficient if the risk is also increased in young people with asthma. Adolescents with asthma may be more likely to develop eating disturbances due to having, on average, a higher BMI (Tantisira & Weiss, 2001), which has been found to be associated with greater body dissatisfaction in male and female adolescents with asthma (Kelsay et al., 2005).
Vulnerability to eating disturbances in young people with both type 1 diabetes and asthma may be a result of illness related weight gain, and subsequent increased BMI, as has been evidenced in both groups (Bryden et al., 1999; Moreau et al., 2009). More specifically, weight gain has been found to be related to insulin treatment in type 1 diabetes (Russell-Jones & Khan, 2007), while in those with asthma it is attributed to a combination of steroid treatment (National Heart, Lung & Blood Institute, 2002) and reduced ability to participate in physical activity (Glazebrook et al., 2006). The uncontrollable nature of this weight gain, related to the necessity of treatment, may further amplify its impact on the individual. Higher BMI may lead to increases in weight/shape concern and body dissatisfaction at a particularly challenging time for adolescents both developmentally and culturally. Research has shown that levels of body dissatisfaction peak during adolescence due to physical maturation and associated changes in BMI, and hormonal changes and associated negative affect (Bearman et al., 2006; Thompson et al., 1995). Furthermore, adolescents in Western society are faced with a cultural preference for low weight and thin body type (Groesz et al., 2002). Therefore combined together, cultural pressures to be thin, developmental changes, and illness-related weight gain, may increase body dissatisfaction leading to a fear of gaining weight, as described in the transdiagnostic theory of EDs (Fairburn et al., 2003). Body dissatisfaction has been shown to be one of the most significant risk factors for the development of further ED symptoms in the general population due to its influence on both dieting behaviour and negative affect (Stice et al., 2011). Body dissatisfaction may in turn heighten vulnerability to engaging in excessive weight-control strategies, binge-eating and compensatory behaviours, such as restriction, self-induced vomiting and excessive exercise. This hypothesis is supported by the finding in this study that being overweight or obese was associated with higher levels of eating disturbances. The implications of the findings in relation to the transdiagnostic theory of EDs will now be discussed.
4.3.2 Transdiagnostic theory of eating disorders.

This study was partly concerned with investigating associations between eating disturbances and key mechanisms involved in the development and maintenance of EDs, as described in the transdiagnostic theory of EDs (Fairburn et al., 2003). The theory extends cognitive-behavioural disorder specific models of EDs (Fairburn et al., 1986) by proposing that ED symptoms are maintained by four key underlying mechanisms. The current study was concerned specifically with adolescent self-esteem and adolescent-perceived parent care and overprotection. These factors were of particular interest as evidence suggests that both may be more impaired in adolescents with chronic illnesses than those without (Pinquart, 2012; Pinquart, 2013); Each shall be considered below.

4.3.2.1 The role of self-esteem.

In this study the asthma group reported significantly lower levels of self-esteem than the type 1 diabetes group. Both groups reported lower self-esteem compared to available normative data for healthy adolescents (Appendix D; Bagley & Mallick, 2001). This may be understood in light of research into the impact of childhood chronic illness on self-esteem, which shows it to be negatively affected (Ferro & Boyle, 2012; Vila et al., 2000). Sense of self may be threatened for those with chronic illness due to multiple factors. These may include feelings of difference and inadequacy, impact of illness on achieving social and educational goals and interruption in development of autonomy and identity (Garrett & Weisman, 2001; Lubkin & Larsen, 2006; Ryden et al., 1994).

Additionally, in this study self-esteem was found to be significantly negatively associated with eating disturbances across males and females in both groups. The nature of the relationship was not influenced by type of chronic illness or gender, which provides evidence for the relevance of self-esteem in understanding eating psychopathology. It could by hypothesised that the threshold for the expression of ED symptoms may be lowered in
young people with asthma and type 1 diabetes due to the potential negative impact of chronic illness on self-esteem. The cross sectional nature of this study precludes conclusions being drawn about the direction of this relationship. However, prospective studies have found self-esteem in the general population, and in young people with type 1 diabetes, to be a significant predictor of onset of ED symptoms (Courtney et al., 2008). Fairburn et al. (1987) propose that global and pervasive low self-esteem creates a sense of hopelessness and inadequacy in individuals, which then propels them toward striving for control over weight and shape in an attempt to increase their worthiness and esteem. This focus on using weight control as a means of increasing self-esteem may be heightened by higher levels of BMI and potential body dissatisfaction seen in these groups (Section 4.3.1). Research also indicates that the relationship between low self-esteem and development of eating disturbances may be partly mediated by negative affect and depression (Courtney et al., 2008). It was not within the scope of this study to investigate the applicability of these pathways between self-esteem and ED symptoms for adolescents with asthma and type 1 diabetes, but represents a potential fruitful area of investigation for future research.

4.3.2.2 The role of parenting style.

The transdiagnostic theory outlines the importance of interpersonal life in the development and maintenance of ED symptoms. Fairburn et al. (2003) broadly define interpersonal life as referring to the quality of interpersonal relationships in an individual’s life, placing particular emphasis on relationships with parents. One way to conceptualise the adolescent-parent relationship is to consider adolescents’ perceptions of their parents’ levels of care (positive dimension) and overprotection (negative dimension) throughout their childhood, as described by Parker et al. (1979). The results of this study found lower parent care to be related to eating disturbances across both groups. Higher levels of overprotection were associated with eating disturbances in the type 1 diabetes group but not the asthma...
group. It is difficult to interpret the finding that parent overprotection was associated with eating disturbances in the diabetes group but not the asthma group. This is particularly ambiguous given that the latter group reported higher levels of both eating disturbances and parent overprotection. It may be that certain parenting dimensions, including low care and high overprotection, do increase vulnerability to eating disturbances in these groups, but that due to the small sample size this was not evidenced in the asthma group on this occasion. Alternatively, parent overprotection may be associated with eating disturbances specifically in this group due to the potential for parent overprotection to be expressed in relation to supporting their child in managing the dietary element to treatment for type 1 diabetes. Further replicative research is required to bring some clarity to this finding.

Taken together these outcomes provide some support for the importance placed on parenting factors in the transdiagnostic model. In the dual process model Leung et al. (1996) propose that the pathway between less than optimal parenting and adolescent vulnerability to developing ED symptoms is indirect and may be mediated by additional factors, including poor sense of identity, self-esteem and maladaptive schemas. Maladaptive schemas are internal patterns of distorted thinking that are developed in childhood and influence the way an individual perceives themselves, others and the world into adulthood (Young, 1994). Research has found retrospective reports of negative parenting style, most notably low levels of parent care, to be associated with maladaptive schemas (Muris, 2006). Specifically, schemas of defectiveness/shame, mistrust/abuse and dependence/incompetence have been found to mediate the relationship between parent care and overprotection and eating disturbances in the general population (Meyer & Gillings, 2004; Turner et al., 2004). These schemas relate to beliefs about the self as inferior and unattractive, others as mistreating and a lack of self-efficacy in managing daily life. Meyer and Gillings (2004) propose that overprotective parenting may foster a suspiciousness of others in children, which then leads
to difficulties in developing satisfying, intimate relationships with others. This may result in feelings of loneliness, inadequacy and worthlessness, which may lead to eating disordered symptoms, such as restraint and binge eating, in some individuals. Further exploration of the applicability of these hypotheses to samples of young people with type 1 diabetes and asthma is warranted.

4.3.3 Summary of theoretical implications.

In summary, the findings of this study have been considered in light of relevant theoretical contexts and existing literature. It remains unclear to what extent differences in eating disturbances observed between the groups were a product of methodological limitations of the study, or were representative of wider populations, indicating previously unobserved higher levels of ED symptoms in adolescents with asthma. This is an important distinction as the latter suggestion would challenge dominant explanations for the increased prevalence seen in type 1 diabetes compared with other chronic illness populations, such as the diabetes-specific vulnerabilities model (Daneman et al., 1998). Tentative hypotheses have been explored in relation to treatment-related weight gain increasing vulnerability to eating disturbances in both adolescents with type 1 diabetes and asthma.

Additionally, support is provided for the salience of self-esteem in relation to eating disturbances, as described in the transdiagnostic theory of EDs (Fairburn et al., 2003). This was considered in light of the impact of chronic illness on a young person’s self-esteem and subsequent additional vulnerability to developing ED symptoms. Parent care and overprotection were also found to be related to eating disturbances in line with the transdiagnostic theory, although these relationships were not evidenced consistently across the two groups. Potential mechanisms relating parent care and overprotection to the development of maladaptive cognitive schemas and subsequent expression of ED symptoms were considered.
4.4 Clinical Implications of Findings

In light of the findings of this study, two key clinical implications are explored. The first is in relation to assessment and awareness of eating disturbances in type 1 diabetes and asthma, and the second is in relation to intervention and management.

4.4.1 Awareness and assessment.

As described, the prevalence of eating disturbances in adolescents with type 1 diabetes is well documented. Furthermore, attending to the broader psychological and emotional wellbeing of this group has increasingly become an integral part of paediatric diabetes services in the UK. This is most evident in the introduction of the paediatric diabetes best practice tariff, which makes access to clinical psychology a requirement for young people with type 1 diabetes (Department of Health, 2011). The outcomes of the study reported here provide support for clinical practices such as these, which promote awareness of adolescents’ emotional wellbeing. The results from the current study suggest that clinicians working with young people with type 1 diabetes should be aware of the potential presentation of EDs. It may be appropriate to place additional focus on those where there appear to be notable difficulties in the family, particularly in enabling adolescent independence and autonomy, or with self-esteem. Interestingly, a qualitative study carried out with medical clinicians working with young people with type 1 diabetes in the UK revealed themes related to a lack of confidence in assessing eating disturbances in this population (Tierney, Deaton, & Whitehead, 2008). Given this, further training may be beneficial in increasing staff confidence and competence in assessing disordered eating attitudes and behaviours in young people with health conditions, as well as in noticing difficulties with self-esteem and family dynamics.

Cameron et al. (2007) considered the appropriateness of introducing routine screening for psychosocial difficulties, including eating psychopathology, in adolescents with type 1
diabetes. They applied the criteria outlined by Morrison (1998) for appropriate routine psychosocial screening, which included the need for high prevalence rates, suitable measures, availability of adequate intervention strategies and cost effectiveness. Cameron et al. concluded that it is both appropriate and important to screen for psychological difficulties in this population. They proposed that this is important not only because it is desirable to reduce distress experienced by adolescents, but also because adaptive mental health outcomes are associated with improved physical health (Jones et al., 2000).

In contrast, relatively little attention has been paid to either the emotional wellbeing, or potential presence of eating disturbances, in young people with asthma. Although the findings of this study are limited in their generalizability, they do indicate that some young people with asthma appear to be experiencing high levels of ED psychopathology, low self-esteem and difficulties in their relationships with their parent(s). In addition, given that a minority of the asthma group reported self-induced vomiting and laxative use as compensatory behaviours, which are rarely seen outside of ED populations, awareness and monitoring of such behaviours in this group is warranted. However, in this study 56.7% of participants in the asthma group reported attending appointments at their asthma clinic less than yearly. It may in fact be important for routine asthma review appointments to occur on a more frequent basis and for professionals working with them to have an awareness of potential distress that their patients may be experiencing, particularly in terms of low self-esteem and disordered eating attitudes and behaviours. This is particularly pertinent given that young people with chronic illnesses, including diabetes and asthma, do not report accessing mental health services more frequently than those without chronic illnesses despite reporting higher levels of emotional distress (Suris, Parera, & Puiq, 1996).

The current study made use of an anonymous design following reports in a previous investigation of EDs in a sample of adolescents with type 1 diabetes that lack of anonymity
suppressed reporting of symptoms (Smith et al., 2009). Given the prevalence of ED symptoms reported by participants in the current study, the outcomes indicate that this anonymous study design enabled at least some participants to report the presence of eating disturbances. Therefore, it is important that particular consideration is given to the way in which ED symptoms are assessed. This is especially important given that they are often accompanied by shame, which has also been found to inhibit disclosure (Swan & Andrews, 2003). Therapeutic relationships characterised by warmth, acceptance, genuineness and trust have been shown to create the most successful environment for disclosure and treatment of EDs to occur (McGilley & Szablewski, 2010). It is important, therefore, for paediatric services to prioritise the development of therapeutic relationships within a medical system that is often limited in resources. Furthermore, young people may find the use of self-report measures, such as the EDE-Q, offer a less shame-inducing experience than face-to-face assessments. They may also provide a useful platform from which clinicians are able to carry out further assessment that is less threatening and more containing for the young person.

4.4.2 Management and intervention.

The diabetes-specific model (Daneman et al., 1998) proposes that young people with type 1 diabetes are at an increased risk of eating disturbances due to factors related to their illness and its treatment. Similarly, it has been hypothesised that illness-related weight gain in young people with asthma may also lower threshold for the expression of eating disturbances. Currently these factors, namely illness-related weight gain, insulin use, and focus on food and eating, are not changeable as they are part of the expression and management of both asthma and type 1 diabetes. Therefore, it is important to consider other factors that may be contributing to the presence of eating disturbances in these groups, which are amenable to intervention and change.
The results of the current study suggest that for adolescents with type 1 diabetes in particular, preventative and intervention strategies focusing on the adolescent-parent relationship may be beneficial. More specifically, interventions that target discrete parenting variables such as tendency toward overprotection, are indicated. Previous research has found an association between parent anxiety and subsequent overprotection in parents dealing with childhood chronic illness (Cameron et al., 2007). Parker and Lipscombe (1979) propose that overprotection is best comprehended as an understandable adaptational response to the uncertainty of chronic illness in childhood. This is a helpful conceptualisation as it distances blame from the parent and focuses on understanding the difficult position parents find themselves in when dealing with childhood chronic illness. Preventative strategies, such as provision of parent support groups, may be beneficial in reducing anxiety and subsequent overprotective parenting. Support for this suggestion comes from results from a randomised controlled trial of a community-based support program for mothers and children with various chronic illnesses, which found improved psychological well-being for children receiving treatment compared to those in the control group (Chernoff, Ireys, DeVet, & Kim, 2002). However, the impact of the support group specifically on maternal anxiety and overprotection was not measured.

The associations found between parent care and overprotection and eating disturbances in this study also provide some support for the utility of family-based approaches in treating ED symptoms in adolescents with type 1 diabetes and asthma. Research has found family therapy to be a moderately effective treatment for EDs, particularly when focusing on building on families’ inherent resources (Gowers & Bryant-Waugh, 2004). Research investigating treatment for ED symptoms in samples of young people with type 1 diabetes has, to date, been limited to cognitive-behavioural approaches (Peveler & Fairburn, 1992) and psycho-educational interventions (Olmsted, Daneman,
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Rydall, Lawson, & Rodin, 2002), both of which have found mixed outcomes. Further research is needed to evaluate the effectiveness of family based approaches in this population and specific interventions targeting parenting styles.

In the current study lower self-esteem was significantly associated with higher levels of eating disturbances in both groups. Although the direction of causality within this sample remains unknown, previous research points strongly to the predictive power of poorer self-esteem in the onset of eating disturbances (e.g. Colton et al., 2007; Polivy & Herman, 2002) The clinical implications of such a finding indicate the importance of intervention strategies that focus on improving self-esteem in adolescents with type 1 diabetes and asthma. O’Dea and Abraham (2000) trialled an educational programme aimed at improving self-esteem to prevent onset of eating disturbances with adolescents in the general population. Topics covered included building a positive sense of self, relationship skills, positive self-evaluation and dealing with stress. They found that both males and females in the intervention group reported higher self-esteem and body satisfaction following the programme compared to the control group; this finding was maintained at one year follow-up. A recent systematic review of preventative programmes aimed at improving body satisfaction and self-esteem in adolescents found gender specific programmes (e.g. Richardson & Paxton, 2010; Stanford & McCabe, 2005) were more effective than those that targeted both male and female adolescents (Yager, Diedrichs, Ricciardelli, & Halliwell, 2013). Taken together, the evidence described suggests that there may be scope for the development of similar gender-specific programmes for adolescents with chronic health conditions in order to reduce the likelihood of them experiencing emotional difficulties, including eating disturbances.

4.5 Methodological Critique

4.5.1 Strengths of the study.
Four key strengths have been identified, including the use of a comparison group, anonymity of the study, use of well-validated measures and the sample demographics; each shall be considered in turn.

The inclusion of a comparison group of young people with asthma is the primary strength of this study. Previous literature in this area has failed to make use of a comparison group when investigating factors associated with eating disturbances in type 1 diabetes (e.g. Colton et al., 2007). As has been noted, causal pathways and maintaining factors for EDs are hugely complex and involve a combination of individual, familial and societal factors (Stice, 2002). This complexity makes it difficult to delineate the relative contribution of discrete variables in explaining the increased prevalence of EDs evidenced in this group. Utilising a comparison group of participants with a chronic illness helps to address the question of whether there is something different about the relationships between these variables observed in the type 1 diabetes group (Boyd, 2002). It also enables the potential impact of chronic illness on vulnerability to eating disturbances to tentatively be explored. Consideration was given, where possible, to including a comparison group with similar illness characteristics to reduce the potential for additional bias in the groups. For example, as in type 1 diabetes, onset of asthma is typically in childhood (Seiffge-Krenke, 2001), it usually involves daily management (Asthma UK, 2009) and can at times be a severe illness requiring hospitalisation (Bruzzese et al., 2011). In addition, the robustness of the between group comparisons is further strengthened by the fact that no significant differences were found in participants’ ratings of their illness severity (according to visual analogue scales), either past or current, between the two illness groups. Taken together these factors indicate the appropriateness of asthma as a comparison group.

Secondly, the anonymity is another strength of the study design. As noted, eating disordered thoughts, attitudes and behaviours are often associated with high levels of shame,
which have been linked to non-disclosure of symptoms in a clinical sample of females with EDs (Swan & Andrews, 2003). In a recent study investigating ED symptoms in adolescents with type 1 diabetes, Smith (2009) found unexpectedly low levels of eating disturbances reported using interview measures (Eating Disorder Examination Interview; Fairburn & Cooper, 1993) and concluded that this was likely due to a reluctance to disclose because of a lack of anonymity.

Thirdly, this study benefited from the use of reliable and valid measures. The self-report questionnaires used to measure the independent and dependent variables have all been shown to be reliable and valid for use with young people aged 16-18 years old (e.g. Peterson et al., 2007; Powers et al., 2012; Safford et al., 2007). There is a lack of published EDE-Q reliability data for adolescent males and for young people specifically with type 1 diabetes and asthma. However, the internal consistency estimates for this sample across all measures were excellent, indicating good internal reliability.

A limitation of previous literature has been that it has largely focused on investigating the association between parenting factors and eating disturbances in female samples of adolescents with type 1 diabetes (e.g. O’Brien et al., 2011). This is the first study to investigate eating disturbances in both male and female adolescents and associations with both maternal and paternal parenting styles. Therefore, the applicability of this study to both males and females, and mothers and fathers, is a further strength.

4.5.2 Limitations of the study.

4.5.2.1 Study Design.

The cross-sectional design of this study is a limitation. Cross-sectional research designs are helpful for establishing associations between variables and generating hypotheses for further exploration, but do not allow for conclusions to be drawn about causality. It is for this reason that it can also be more difficult to interpret results from a cross-sectional design.
A longitudinal study would have been a more robust method for investigating relationships between the variables of interest. However, this was not possible due to practical limits on time and resources.

A further limitation of the study design is that, again due to practical constraints, it was not possible to make use of a matched-pairs design. When it is not possible to randomly assign participants to groups a matched-pairs design is considered to be a credible alternative for reducing bias (Rubin, 1973). Matched-pairs designs enable the researcher to control for potential confounding variables by matching participants across groups based on these identified variables. In this study the groups significantly differed in proportion of males and females, age of illness onset and frequency of hospital/clinic appointments. Contrary to previous findings (Meltzer et al., 2001), age of illness onset was not found to be significantly associated with eating disturbances in either group. However, the significant difference in proportion of males and females in each group is considered to be a substantial source of bias in the current study. The asthma group had a significantly higher proportion of females (78.4%) to males compared to the diabetes group (53.8%). The recruitment method for the asthma group may have attracted more female respondents due the nature of the research topic and self-selection bias. Further, it was not possible to control for gender statistically as non-parametric tests were utilised due to the distribution of the data. Therefore, this limitation has been taken into consideration in interpreting the findings of this study.

In an attempt to maximise recruitment two methods for data collection, either postal or internet, were offered to participants. In the type 1 diabetes group 11% of participants used the internet participation method, compared to 97% of the asthma group. It is possible that the different methods for data collection may have introduced additional bias to the study findings. For example, the findings of a randomised population study found that participants who chose to respond to an asthma health survey online, rather than by post, were more likely
to be male and have access to the internet at home, which may be an indication of socio-economic status (Brogger, Nystad, Cappelen & Bakke, 2007). In addition, they also found that those who responded online were more likely to report having asthma than those who responded using traditional postal methods. This indicates that data collected through internet methods may be susceptible to gender and non-response biases. In addition, in a review of 29 papers Hoonakker and Carayon (2009) report that online responders appear to be more willing to provide information about themselves in comparison to those who use postal methods. Internet participation may allow participants to have a greater sense of privacy and anonymity, which in turn may enable them to respond with greater honesty and less social-desirability than postal methods (Joinson, 1999). The potential for biases such as these are an additional limitation of the study reported here. However, it is important to note that no significant differences were found in level of eating disturbances between those who participated online and by post across both illness groups (Section 2.3.2.1).

4.5.2.2 Sample.

One of the main limitations of the study is the use of different recruitment strategies to recruit participants into the each group. Participants in the type 1 diabetes group were invited to take part in the study through face-to-face conversations with the researcher, which facilitated recruitment of the desired number of participants in this group. However, significant and persistent difficulties were experienced in recruiting asthma participants in this way (Appendix A). The main difficulty was in identifying locations through which adolescents with asthma could be approached directly and invited to participate in the study, as routine asthma review appointments occurred on an unpredictable and ad hoc basis in the GP practices approached to assist with the study. Therefore, in order to recruit a sample within the study time frame the strategy was changed and participants with asthma were invited to take part through an invitation letter posted to them from their GP surgery.
Attempts were made to reduce selection bias by stating in the invitation letter that the researcher was interested in hearing from both males and females, regardless of their experience. Although both groups were recruited using self-selecting methods, postal research invitations are more susceptible to this bias than face-to-face strategies and have a much lower response rate (Picavet, 2001). Further, participants in the asthma group may have been more likely to take part in the study if the area of eating disturbances had relevance to them.

Despite significant efforts to overcome the barriers in recruitment of the asthma group, only 37 participants were able to be recruited into this group. This means that for the primary research questions the asthma group \((n = 37)\) had only 15% power to detect weak correlation coefficients \((.10)\), 57% power to detect moderation correlations \((.30)\), and 95% power to detect strong correlations \((.50)\), when using a one tailed correlation analysis (GPower; Erdfelder et al., 1996). The type 1 diabetes group \((n = 65)\) had 20% power to detect weak correlation coefficients \((.10)\), 79% power to detect moderate correlations \((.30)\), and 99% power to detect strong correlations \((.50)\). For the secondary research questions, with two groups \((n_1 = 65, n_2 = 37)\), using two tailed between-group tests, the study had 16% power to detect small effect sizes \((.20)\), 67% power to detect medium effect sizes \((.50)\) and 97% power to detect large effect sizes \((.80)\). This means that the asthma group did not have sufficient power to detect weak or moderate correlation coefficients, though the type 1 diabetes group was sufficiently powered to detect moderate correlations. The study had adequate power to detect large effect sizes in the between groups analysis. Taken together this means that there was an increased risk of a Type II error occurring in the statistical analyses, i.e. accepting the null hypothesis when it should be rejected, within the asthma group (Cohen, 1992). In an attempt to reduce the influence of this on the study outcomes, correlation coefficients and
model parameters were considered alongside statistical significance testing to identify potential trends that had not reached significance.

A further potential limitation of the sample was the requirement that illness diagnosis had been present for a minimum of 6 months. This timeframe was chosen as it is widely accepted as an appropriate length of time for young people to adapt to their illness diagnosis (e.g. Kichler et al., 2008). However, those with type 1 diabetes may experience a phase commonly known as the *honeymoon period*, during which insulin treatment may enable the beta cells to continue to secrete some insulin (Daneman, 2006). This period may last for up to a year, and therefore it is not until after this phase has passed that young people experience the full effect of their diabetes diagnosis. However, the minimum length of illness reported by participants with type 1 diabetes in this study was two years. Therefore, it is unlikely that any participants were in the honeymoon phase during participation in the study. Future research should take into consideration the diabetes honeymoon period and extend the required period of time since diagnosis to at least one year.

4.5.2.3 Measures.

There is some debate within the literature about whether self-report methods, as used in this study, or clinician rated interviews are most suitable for investigating eating disturbances. Research suggests that self-report methods yield higher scores than face-to-face interviews (Fairburn & Beglin, 1994). This may indicate either under reporting of eating disturbances when assessed through face-to-face interview, or over reporting through self-report methods. One of the most prominent arguments for the use of interview-based methods is that some ED features, particularly binge eating episodes, are difficult to define and require a trained interviewer to be able to communicate the concept and accurately assess level of severity (Cooper & Fairburn, 1987). For example, Fairburn et al. (1994) found frequency of binge eating and severity of shape concern to be higher when reported using the EDE-Q
compared to the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993). They propose that this difference is the product of over-reporting on the EDE-Q due to ambiguity of the concepts and difficulty in communicating definitions through a self-report measure.

In order to overcome these potential limitations, the most recent version of the EDE-Q, as used in this study, includes descriptive prompts to aid accurate completion of the questionnaire given the potential lack of clarity in defining certain symptoms. For example, the following description is given alongside the question about binge eating episodes, “i.e. you have eaten an unusually large amount of food and have had a sense of loss of control at the time”. Celio, Wifley, Crow, Mitchell and Walsh (2004) found the frequency of reported binge eating days on the version of the EDE-Q used in the current study to be significantly correlated with the frequencies reported on the EDE ($r = .65$, $p < .001$). Furthermore, the pattern of the EDE-Q yielding higher scores has not been consistent in the literature, with other studies showing the EDE yielding higher scores (Carter, Aime, & Mills, 2001) or no difference in scores between the two measures (Binford, Le Grange, & Jellar, 2005). In addition to the debate about the validity of self-report measures of eating disturbances, there is also question about the validity of dietary restraint scales as measures of dietary restriction (Stice, Sysko, Roberto, & Allison, 2010). For example, Stice, Fisher and Lowe (2004) found that scores on the EDE-Q restraint subscale did not correlate with observed calorie intake. In the current study the restriction subscale of the EDE-Q often did not follow patterns of associations evidenced with the global scale and weight, eating, and shape concern subscales; this finding could be understood in light of the uncertainty about the validity of self-report measures of dietary restriction.

A further limitation was that the measures used relied solely on participant self-report for measure of height and weight in order to calculate BMI. Given the range of scores reported it is possible there was some misreporting of this data. This is particularly pertinent
given that biases in self-reported height and weight have been found in males and females in the general population, as well as in those reporting eating disturbances. A systematic review into the accuracy of subjectively measured weight and height in adult populations reported a trend of under-reporting of weight and over-reporting of height (Gorber, Tremblay & Gorber, 2007); the magnitude of this finding was dependent upon the population being studied and participants’ gender. Most notably males may be more susceptible to over-reporting their height (Bogaert & McCreary, 2011), and females under-reporting their weight (Dhaliwal, Howat, Bejoy & Welborn, 2010), due to Western ideals about male and female body image. In addition, eating psychopathology has been linked with both over- and under-reporting of weight. For example, a recent study investigating biases in self-reported height and weight in adult females found higher levels of eating concern, as measured by the EDE-Q, were associated with overestimated weight. On the contrary, higher levels of weight concern were associated with underestimated weight (Meyer, McPartlan, Sines & Waller, 2009). Taken together these findings suggest that the results reported here with regard to BMI may have been influenced by a variety of biases in the reporting of weight and height. In particular, the finding that higher BMI was significantly associated with greater eating psychopathology may have been influenced by these self-report biases and should be interpreted cautiously.

4.5.2.4 Statistical analysis.

In this study multiple hypotheses were tested in order to identify associations between variables. Carrying out multiple testing can increase the risk of a Type I error occurring, i.e. of rejecting the null hypothesis when it should be accepted (Shaffer, 1995). This possibility can be corrected for by using Bonferroni Corrections, which reduce the level at which statistical significance is achieved. However, as this was an exploratory investigation that aimed to identify relationship trends to direct future research Bonferroni Corrections were not utilised (Field, 2013).
4.5.3 Summary.

In summary, the strengths of the current study enable it to make an interesting contribution to the literature. Most importantly the study made use of a comparison group, which enabled the potential influence of type of chronic illness on vulnerability to ED psychopathology, and associated factors, to be tentatively explored. Participant anonymity is considered to have added to the robustness of the design along with the use of reliable and valid measures. Furthermore, the inclusion of male and female participants and the investigation of maternal and paternal factors add to the generalizability of the findings. The limitations of the study have also been considered and most prominently include the different recruitment strategies for each group and subsequent influences on sample size and potential bias. The use of self-report methods for measuring eating disturbances have also been discussed alongside the possibility for Type I errors due to multiple statistical testing.

4.6 Future Research

The findings of this study suggest a need for further research into the presence of eating disturbances in adolescents with asthma. This study was limited, in part, by differences in sampling technique between the two groups and the small size of the asthma group. Future investigations should aim to recruit a larger and more representative sample of adolescents with asthma to improve the generalizability of the study findings. Recruitment of a larger sample would also enable multiple regression analyses to be carried out to determine the relative contribution of BMI, self-esteem and parenting factors in interaction with type of chronic illness in predicting eating disturbances. However, to enable this to happen, further consideration is needed to overcome some of the barriers to recruitment of asthma participants faced in the study reported here. Investigations in the future might consider recruiting participants through schools and colleges rather than health settings to overcome some of the difficulties described. In addition, use of a matched comparison group would
increase the validity of study findings by minimising the effect of individual differences within the group (Rubin, 1973). At a minimum, future studies would benefit from matching participants based on gender.

Further research is needed to test the hypothesis that illness related weight gain may increase vulnerability to eating disturbances through a pathway of heightened body dissatisfaction, eating and weight concern and subsequent weight control strategies. In order to achieve this, inclusion of comparison groups of young people with chronic illnesses, potentially without treatments or symptoms that impact upon diet and weight, and healthy controls will be important. This will enable the relative contribution of illness-related weight gain on subsequent ED symptoms to be investigated more robustly. Longitudinal research designs will also provide useful insight into the validity of these hypotheses.

Additionally, given that many young people with type 1 diabetes and asthma have higher BMIs than their healthy peers but not all develop eating disturbances, it will be important to investigate additional vulnerability factors in these two groups. Of particular interest may be factors that are hypothesised to be both associated with eating disturbances and potentially influenced by the presence of chronic illness. Further research investigating the influence of chronic illness on parenting factors and subsequent impact on eating psychopathology in groups considered at risk of developing eating disturbances, e.g. those with higher BMI, is warranted. The study of potential mediating factors, including self-schemas and self-esteem, will strengthen current understanding of ED symptoms in young people with type 1 diabetes and asthma.

Alongside environmental factors, further investigation of genetic contributions to the development of eating disturbances, and possible gene-environment interactions, is required. Preliminary evidence indicates that certain early developmental contexts, such as high maternal stress, pre- and post-natal stress, and impaired nutrition, may stimulate epigenetic
changes that increase future risk of developing eating disorder symptoms (Campbell, Mill, Uher & Schmidt, 2010); this may occur through changes to both metabolic and neuropsychological processes. In the cognitive-interpersonal model of AN, Treasure and Schmidt (2013) propose that genetic vulnerabilities predispose certain individuals to a particular cognitive phenotype, which when triggered by environmental factors, such as high stress and weight-related teasing, may lead to the development of EDs. This cognitive phenotype is typically characterised by both anxious-avoidant and obsessive-compulsive traits. More specifically, research has found that individuals with these traits have less cognitive flexibility, weaker central coherence, more expert detail processing and poorer global integration (Harrison, Tchanturia, Naumann, & Treasure, 2012). This means that they have a tendency toward noticing and fixating on detail, and find it harder to see the bigger picture. Given this, further research into the interaction between presence of chronic illness, cognitive style and genetic vulnerabilities is warranted. Significant individual differences in the neuropsychological profiles of those with AN have been demonstrated and should also be taken into consideration in any future research (Rose, Frampton & Lask, 2012).

4.7 Final Summary and Conclusion

The primary aim of the study reported here was to explore the relationship between adolescent self-esteem, adolescent-perceived parent care and overprotection, and eating disturbances among male and female adolescents with type 1 diabetes compared with those with asthma. In this chapter the outcomes of the study have been considered in relation to each hypothesis and previous literature. Overall, the findings of this study tentatively suggest that the relationship between higher levels of parent overprotection and higher levels of eating disturbances were moderated by chronic illness. The observed relationships between parent care and eating disturbances were more mixed, but taken together the pattern of results suggested a significant association between the variables. Lower self-esteem was consistently
found to be related to higher eating disturbances in both groups. Unexpectedly, participants in the asthma group reported greater impairment on measures of eating disturbances, parent overprotection and self-esteem compared to the type 1 diabetes group.

The findings of this study have been considered in light of relevant theoretical contexts and existing literature. The outcomes are tentatively interpreted as suggesting ED scores were higher for females in both illness groups in comparison to normative data for the general population. It remains unclear whether differences in eating disturbances observed between the type 1 diabetes and asthma groups are representative of wider populations, indicating higher levels ED symptoms in adolescents with asthma. Tentative hypotheses have been made about illness-related weight gain and low self-esteem creating vulnerability to ED psychopathology in young people with both type 1 diabetes and asthma. Although less clear, this vulnerability may be further heightened by lower levels of parent care and higher levels of overprotection, which may be impacted by the presence of childhood chronic illness.

In light of the outcomes of this study, two key clinical implications have been explored. The first is in relation to assessment and awareness of eating psychopathology in type 1 diabetes and asthma populations, and the second is in relation to focusing interventions on factors amenable to change. In particular a case is made for increasing awareness of potential ED symptoms and distress experienced by adolescents with asthma, and prevention and intervention strategies focused on improving self-esteem and reducing parent overprotection in both groups.

The strengths of the investigation have been outlined and are focused on the use of a comparison group, participant anonymity, use of well-validated measures and the inclusion of males and females and mothers and fathers. The main limitations of the study have been discussed. Particular consideration was given to the different sampling techniques for each group and subsequent impact on self-selection bias, along with the small size of the asthma
group. Recommendations for future research have been made and particularly emphasise the need for further between group comparisons making use of matched pair samples, longitudinal designs and investigations of mediating factors. Despite the limitations of the study, this investigation has provided a novel exploration into the experience of adolescents with type 1 diabetes and asthma in relation to eating disturbances. The outcomes have provided direction for future research in this area. Furthermore, the findings have clinical relevance for the prevention and treatment of eating disturbances in type 1 diabetes populations, and at the very least indicate the need for greater awareness and consideration of EDs in adolescents with a diagnosis of asthma.
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Eating Disturbances in Chronic Illness  J.Hatton


Eating Disturbances in Chronic Illness


834.


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J. Hatton


Eating Disturbances in Chronic Illness  J.Hatton


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Eating Disturbances in Chronic Illness


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APPENDICES

Appendix A  Recruitment Documents

Appendix B  Study Questionnaires

Appendix C  Ethical Approval

Appendix D  Normative Data for Measures

Appendix E  Results of Mann Whitney U Tests: DASS-21 and Illness Severity

Visual Analogue Scales

Appendix F  Tests of Normality for Transformed Data

Appendix G  Results of Kruskal Wallis Tests: Differences in EDE-Q scores across Parenting Style Categories

Appendix H  Mann Whitney U Tests: Gender Comparisons on the EDE-Q
Appendix A

Recruitment Documents

A1 Participant Information Sheet 1
A2 Parent Information Sheet
A3 Recruitment Flowchart
A4 Participant Invitation Letter: Asthma Group
A5 Participant Information Sheet 2
A6 Details of Recruitment Attempts
Participant Information Sheet 1

**Research study title:** Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

This project is being carried out as a thesis research project for Judith Hatton (Trainee Clinical Psychologist) and is being supervised by Drs Imogen Hobbis and Sian Coker (Clinical Lecturers in Clinical Psychology, University of East Anglia). This study has been reviewed and approved by the NRES Committee East of England – Cambridge South REC.

Before you decide whether you wish to take part or not, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

Take time to decide whether or not you wish to take part.

**What is the purpose of the study?**

This research is interested in finding out more about young people with type 1 diabetes and asthma and about whether the way they perceive their parent(s) and the way they feel about themselves is related to their thoughts and behaviours toward food and eating. We know a little bit about the relationship between these factors in the general population but do not know much about them in young people with type 1 diabetes and asthma and are interested in finding out more.

**Why have I been invited?**

You are a young person aged 16, 17 or 18 who is living at home with at least one parent and are attending either a diabetes or asthma clinic and therefore can take part in the study.

**Do I have to take part?**

No. It is up to you to decide whether or not to take part. Even after you have agreed to take part and taken away a questionnaire pack or the web address, you are free to change your mind and not return the questionnaires. You do not have to give a reason for this. If you decide that you do not wish to take part in the research this will not affect your current or future treatment in the clinic.

**What will happen to me if I take part?**

You will be given either a paper questionnaire pack or the web address for an online version of the questionnaires (depending on your preference) by Judith Hatton (Trainee Clinical Psychologist) or the clinician you are seeing in the clinic. The paper questionnaire pack contains a copy of this information sheet giving details about the study and some questionnaires. If you choose to complete the questionnaires online you will be given a unique code, along with the web
address, to enter with your questionnaire responses. If you do not enter the code, or the same code is entered more than once, your responses will not be able to be used in the study.

You will be asked to complete a few questionnaires in your own time when the researcher is not present. If you have a paper pack of the questionnaires, once you have completed them you will then be asked to return them by post in the stamped addressed envelope provided or by posting them into the specially designed post box in your clinic. In total the questionnaires will take about 25 minutes to complete. By completing and returning the questionnaires you are consenting (agreeing) to participate in the study. Before you decide to take part you can discuss this with the researcher and your parents if you wish to. You may want to take a copy of the parent information sheet to share with them.

For those who do wish to take part in this study there will be a prize draw to win one of two available £20 vouchers for Outfit (Topshop, Topman, Dorothy Perkins, Burtons), as a way of saying thank you for your time. If you would like to participate in the prize draw then there is a short form for you to fill in asking for your name and contact details. Before you return your questionnaire pack you need to seal the prize draw form in the provided envelope and return along with your questionnaires in the larger envelope. If you are completing the questionnaires online your contact details will be copied onto a slip of paper by the researcher and sealed in an envelope to go with the other prize draw entries. Your sealed envelope will not be opened unless it is selected out of the prize draw. If you do not wish to take part in the prize draw you do not need to return the prize draw form.

**What do I have to do?**

You will be asked to complete some questionnaires as honestly as you can. There are five questionnaires that ask about different areas; your thoughts and behaviours toward food and eating, your perceptions of your parents as you were growing up, the way you feel about yourself, your emotional well-being and the severity of your illness. There are also a few general information questions (e.g age, height, etc) for you to answer.

**What are the possible disadvantages and risks of taking part?**

Some of the questions ask about your emotional well-being and your thoughts and behaviours toward food. It is possible that these questions may raise concerns for you. If this happens you are advised to discuss this with the clinician who first introduced you to this study (either your diabetes doctor or your asthma nurse). If you become distressed when filling out the questionnaires please stop filling them in immediately.

**What are the possible benefits of taking part?**

Other than opportunity to be entered into a prize draw there are no direct personal benefits to taking part in this study. However, you will be contributing to what we know about how young people with type 1 diabetes and asthma think about feel about food and eating.

**What if there is a problem?**
It is not anticipated that anyone will be harmed by taking part in this study, but if you have a complaint about the way you have been treated during the study or suffer from any unforeseen harm, this will be addressed.

Complaints

If you have a concern about any aspect of this study, you should ask to speak with Judith Hatton (Trainee Clinical Psychologist), Dr Imogen Hobbis or Dr Sian Coker (Research Supervisors) at:

Doctoral Programme in Clinical Psychology  
Department of Psychological Sciences  
Norwich Medical School  
University of East Anglia  
NORWICH  
NR4 7TJ  
Tel 01603 593076

Will my taking part in this study be kept confidential?

Our procedures for handling, processing, storage and destruction of data are in line with the Data Protection Act 1998. All information about your taking part in this study will be anonymous and will be kept confidential.

This means that we will not ask you to put your name on your questionnaires, so that nobody will be able to identify which questionnaires you filled in. We hope that by not asking you to give us your name you will feel able to be as honest as possible in your answers. Instead of having your name on, your questionnaires will be given a number.

All of your documents (questionnaires and prize draw envelopes) will be kept in a locked cabinet and at the end of the study they will then be kept safely at the University of East Anglia for 5 years and then destroyed. The information you provide us with will be put onto a spreadsheet on the computer. This spreadsheet will be password protected so that nobody other than Judith Hatton (Trainee Clinical Psychologist) or Drs Imogen Hobbis and Sian Coker (Academic Supervisors) will be able to look at it.

What will happen to the results of the research study?

The results of the research will be used for part of Judith Hatton’s Doctorate in clinical psychology. A summary of the overall results will be sent to your clinic so that you can have a look at them next time you are there, if you would like to. This summary, and any future reports and publications, will not include any information which could personally identify you but will be reporting on general trends seen in the group as whole.

If you choose to take part in this study you will receive a copy of the information sheet to keep. Thank you for considering taking part in this study and taking the time to read this sheet.

CONTACT DETAILS

If you would like any further information please contact:
Parent Information Sheet

Research study title: Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

This project is being carried out as a thesis research project for Judith Hatton (Trainee Clinical Psychologist) and is being supervised by Drs Imogen Hobbis and Sian Coker (Clinical Lecturers in Clinical Psychology, University of East Anglia). The study has been reviewed and approved by the NRES Committee East of England – Cambridge South REC.

Your child has been invited to participate in this study whilst at a routine clinic appointment for their asthma or type 1 diabetes. As they are 16 years or over they are legally able to decide whether or not they would like to participate in the study. They have been provided with an information sheet to help them decide whether they would like to take part or not. However, we have encouraged them to share this Parent Information Sheet with you as part of their decision making.

What is the purpose of the study?

This research is interested in finding out more about young people with type 1 diabetes and asthma and about whether the way they perceive their relationship with their parent(s) and the way they feel about themselves is related to their thoughts and behaviours toward food and eating. We know a little bit about the relationship between these factors in the general population but do not know much about them in young people with type 1 diabetes and asthma and are interested in finding out more.

Why has my child been invited to take part?

Your child has been invited to participate because they are a young person aged 16, 17 or 18 who is living at home with at least one parent and are attending either a diabetes or asthma clinic.

Do they have to take part?

No. It is up to your child to decide whether or not to take part. Even after they have agreed to take part and taken away a questionnaire pack or the web address, they are free to change their mind and not return the questionnaires. They do not have to give a reason for this. If they decide that they do not wish to take part in the research this will not affect their current or future treatment in the clinic.

What will happen to my child if they take part?

They will be given either a paper questionnaire pack or the web address for an online version of the questionnaires (depending on their preference) by Judith Hatton (Trainee Clinical Psychologist) or the clinician they are seeing in the clinic. The paper questionnaire pack contains
For those who do wish to take part in this study there will be a prize draw to win one of two available £20 vouchers for Outfit (Topshop, Topman, Dorothy Perkins, Burtons), as a way of saying thank you for their time. If your child would like to participate in the prize draw then there is a short form for them to fill in asking for their name and contact details. If they do not wish to take part in the prize draw then they do not need to return the prize draw form.

What do they have to do?

Your child will be asked to complete some questionnaires as honestly as they can. There are five questionnaires that ask about different areas: their thoughts and behaviours toward food and eating, their perceptions of their relationship with their parents as they were growing up, the way they feel about themselves, their emotional well-being and the severity of their illness. There are also a few general information questions (e.g., age, height, etc) for them to answer.

What are the possible disadvantages and risks of taking part?

Some of the questions ask about your child’s emotional well-being and their thoughts and behaviours toward food. It is possible that these questions may raise concerns for them. If this happens they have been advised to discuss this with their clinician.

What are the possible benefits of taking part?

Other than opportunity to be entered into a prize draw there are no direct personal benefits to taking part in this study. However, your child will be contributing to what we know about how young people with type 1 diabetes and asthma think and feel about food and eating.

What if there is a problem?

It is not anticipated that anyone will be harmed by taking part in this study, but if you, or your child, has a complaint about the way they have been treated during the study or suffer from any unforeseen harm, this will be addressed.

Complaints

If you, or your child, have a concern about any aspect of this study, you should ask to speak with Judith Hatton (Trainee Clinical Psychologist), Dr Imogen Hobbis or Dr Sian Coker (Research Supervisors) at:

Doctoral Programme in Clinical Psychology
Department of Psychological Sciences
Will my child’s taking part in this study be kept confidential?

Our procedures for handling, processing, storage and destruction of data are in line with the Data Protection Act 1998. All information about your child’s taking part in this study will be anonymous and will be kept confidential.

This means that we will not ask them to put their name on their questionnaires, so that nobody will be able to identify which questionnaires they filled in. We hope that by not asking your child to give us their name they will feel able to be as honest as possible in their answers. Instead of having their name on their questionnaires will be given a number.

All of your child’s documents (questionnaires and prize draw envelopes) will be kept in a locked cabinet and at the end of the study they will then be kept safely at the University of East Anglia for 5 years and then destroyed. The information your child provides us with will be put onto a spreadsheet on the computer. This spreadsheet will be password protected so that nobody other than Judith Hatton (Trainee Clinical Psychologist) or Drs Imogen Hobbis and Sian Coker (Academic Supervisors) will be able to look at it.

What will happen to the results of the research study?

The results of the research will be used for part of Judith Hatton’s Doctorate in Clinical Psychology. A summary of the overall results will be sent to your child’s clinic so that you, and your child, can have a look at them next time you are there, if you would like to. This summary, and any future reports and publications, will not include any information which could personally identify your child but will be reporting on general trends seen in the group as whole.

CONTACT DETAILS

If you would like any further information please contact:
Judith Hatton, Trainee Clinical Psychologist
Postgraduate Office,
Elizabeth Fry Building,
University of East Anglia,
Norwich NR4 7TJ
Telephone number: 01603 59358, Email address: judith.hatton@uea.ac.uk
Recruitment Flowchart

Has this person had a diagnosis of **asthma** for at least 6 months?

- YES
  - Do they also have type 1 diabetes?
    - NO
      - Are they aged **over 16?** (16, 17 or 18 years of age?)
        - NO
          - Invite them to participate in the study!
        - YES
          - NOT SUITABLE
    - YES
      - NOT SUITABLE

Invite them to participate in the study!

1. Please introduce the study to the young person and give them a participant information sheet.
2. If they are interested in taking part please give them the option of completing the questionnaires in paper format or online.
3. Give them either a paper questionnaire pack or the web address for the online questionnaires.
Hi,

Our practice is currently supporting a student research project looking into the well-being of young people with chronic illness. We are writing to you to introduce you to the project and the researcher, and to invite you to take part.

Who is carrying out this research and why are they writing to you?

The researchers name is Jude Hatton, and she is a Trainee Clinical Psychologist from the University of East Anglia in Norwich. Together with Jude, we are writing to you to invite you to take part in a study that she is carrying out. You have been invited because you are aged 16-18 years, have a diagnosis of asthma and are registered at (name of GP surgery).

What is the study about?

We are hoping to find out more about how young people with chronic illnesses (including asthma) feel about food, eating and the way they look. In particular we are interested in whether this is related to how they feel about themselves in general and their relationship with their Mum and/or Dad. We are interested in hearing from both males and females, whatever your experience. The information you tell us will help us to help other people who are in similar situations to you in the future.

What does taking part involve?

If you would like to take part then you will be asked to complete five questionnaires that ask about how you feel about food and eating, yourself and your relationship with your Mum and/or Dad. This will take about 20-25 minutes. You can complete the questionnaires online by going to:

www.eatingstudy.co.uk

Unique code: XXX

If you do not have access to the internet, please contact me on 01603 593581 and I will arrange for a paper questionnaire pack to be sent to you.

What do you really need to know?

1. As a thank you, you will be able to take part in a prize draw to win one of two £20 vouchers for Outfit (which can be spent at Topshop, Topman, Burton, Miss Selfridge etc).

2. You do not have to take part if you do not want to – please take some time to think about whether or not you would like to take part.
3. Your responses to the questionnaires are **anonymous** – this means we will not ask you to put your name on your questionnaires. Also, we won’t feedback anything you tell us to anybody who knows you, like your parents or your GP.

4. It is important that you **read the information sheet** attached to this letter if you would like to take part

Thank you for taking the time to think about taking part in this study. If you have questions please contact either your asthma nurse at your surgery or myself, judith.hatton@uea.ac.uk.

Thank you again!

(name of GP surgery)

&

Jude Hatton

(Trainee Clinical Psychologist, UEA)
Research study title: Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

This project is being carried out as a thesis research project for Judith Hatton (Trainee Clinical Psychologist) and is being supervised by Drs Imogen Hobbis and Sian Coker (Clinical Lecturers in Clinical Psychology, University of East Anglia). This study has been reviewed and approved by the NRES Committee East of England – Cambridge South REC.

Before you decide whether you wish to take part or not, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

Take time to decide whether or not you wish to take part.

What is the purpose of the study?

This research is interested in finding out more about young people with type 1 diabetes and asthma and about whether the way they perceive their parent(s) and the way they feel about themselves is related to their thoughts and behaviours toward food and eating. We know a little bit about the relationship between these factors in the general population but do not know much about them in young people with type 1 diabetes and asthma and are interested in finding out more.

Why have I been invited?

You are a young person aged 16, 17 or 18 who is living at home with at least one parent and attend either a diabetes or asthma clinic and therefore can take part in the study.

Do I have to take part?

No. It is up to you to decide whether or not to take part. If you decide that you do not wish to take part in the research this will not affect your current or future treatment in the clinic.

What will happen to me if I take part?

You have been sent the web address for an online version of the questionnaires. As mentioned in the introductory invitation letter, if you would like to complete a paper questionnaire pack, please contact Judith Hatton (Trainee Clinical Psychologist) to access this. If you choose to complete the questionnaires online you will be given a unique code, along with the web address, to enter with your questionnaire responses. If you do not enter the code, or the same code is entered more than once, your responses will not be able to be used in the study.
You will be asked to complete a few questionnaires in your own time. If you have a paper pack of the questionnaires, once you have completed them you will then be asked to return them by post in the stamped addressed envelope provided. In total the questionnaires will take about 25 minutes to complete. By completing and returning the questionnaires you are consenting (agreeing) to participate in the study. Before you decide to take part you can discuss this with your parents if you wish to.

For those who do wish to take part in this study there will be a prize draw to win one of two available £20 vouchers for Outfit (Topshop, Topman, Dorothy Perkins, Burtons), as a way of saying thank you for your time. Once you have completed the online questionnaires you will be given an email address to send your contact details to, in order to be entered into the prize draw. If you are completing a paper questionnaire pack, then there is a short form for you to fill out asking for your name and contact details. Before you return your questionnaire pack you need to seal the prize draw form in the provided envelope and return along with your questionnaires in a larger envelope. Your sealed envelope will not be opened unless it is selected out of the prize draw. If you do not wish to take part in the prize draw you do not need to return the prize draw form.

What do I have to do?

You will be asked to complete some questionnaires as honestly as you can. There are five questionnaires that ask about different areas; your thoughts and behaviours toward food and eating, your perceptions of your parents as you were growing up, the way you feel about yourself, your emotional well-being and the severity of your illness. There are also a few general information questions (e.g. age, height, etc) for you to answer.

What are the possible disadvantages and risks of taking part?

Some of the questions ask about your emotional well-being and your thoughts and behaviours toward food. It is possible that these questions may raise concerns for you. If this happens you are advised to discuss this with the clinician named in the invitation letter (either your asthma doctor or nurse). If you become distressed when filling out the questionnaires please stop filling them in immediately.

What are the possible benefits of taking part?

Other than opportunity to be entered into a prize draw there are no direct personal benefits to taking part in this study. However, you will be contributing to what we know about how young people with type 1 diabetes and asthma think about feel about food and eating.

What if there is a problem?

It is not anticipated that anyone will be harmed by taking part in this study, but if you have a complaint about the way you have been treated during the study or suffer from any unforeseen harm, this will be addressed.

Complaints
If you have a concern about any aspect of this study, you should ask to speak with Judith Hatton (Trainee Clinical Psychologist), Dr Imogen Hobbis or Dr Sian Coker (Research Supervisors) at:

Doctoral Programme in Clinical Psychology
Department of Psychological Sciences
Norwich Medical School
University of East Anglia
NORWICH
NR4 7TJ
Tel 01603 593076

**Will my taking part in this study be kept confidential?**

Our procedures for handling, processing, storage and destruction of data are in line with the Data Protection Act 1998. All information about your taking part in this study will be anonymous and will be kept confidential.

This means that we will not ask you to put your name on your questionnaires, so that nobody will be able to identify which questionnaires you filled in. We hope that by not asking you to give us your name you will feel able to be as honest as possible in your answers. Instead of having your name on, your questionnaires will be given a number.

All of your documents (questionnaires and prize draw envelopes) will be kept in a locked cabinet and at the end of the study they will then be kept safely at the University of East Anglia for 5 years and then destroyed. The information you provide us with will be put onto a spreadsheet on the computer. This spreadsheet will be password protected so that nobody other than Judith Hatton (Trainee Clinical Psychologist) or Drs Imogen Hobbis and Sian Coker (Academic Supervisors) will be able to look at it.

**What will happen to the results of the research study?**

The results of the research will be used for part of Judith Hatton's Doctorate in clinical psychology. A summary of the overall results will be sent to your clinic so that you can have a look at them next time you are there, if you would like to. This summary, and any future reports and publications, will not include any information which could personally identify you but will be reporting on general trends seen in the group as whole.

*If you choose to take part in this study you will receive a copy of the information sheet to keep. Thank you for considering taking part in this study and taking the time to read this sheet.*

**CONTACT DETAILS**

If you would like any further information please contact:

Judith Hatton, Trainee Clinical Psychologist
Postgraduate Office,
Elizabeth Fry Building,
University of East Anglia,
Norwich NR4 7TJ

Telephone: 01603 593581, Email address: judith.hatton@uea.ac.uk
Details of Recruitment Attempts

Table A1.  

Recruitment Attempts for the Type 1 Diabetes Group

<table>
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<tr>
<th>Outpatient Clinic</th>
<th>Frequency of clinics</th>
<th>Number of clinics attended</th>
<th>Number of people invited to participate</th>
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<tbody>
<tr>
<td>NNUH</td>
<td>Weekly</td>
<td>15</td>
<td>36</td>
</tr>
<tr>
<td>AHC</td>
<td>Weekly</td>
<td>17</td>
<td>48</td>
</tr>
<tr>
<td>JPHGY*</td>
<td>Monthly</td>
<td>4</td>
<td>15</td>
</tr>
<tr>
<td>QEHKL</td>
<td>monthly</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>40</strong></td>
<td><strong>115</strong></td>
</tr>
</tbody>
</table>

*Recruitment at this site was carried out by a Diabetes Specialist Nurse on behalf of the researcher as the clinic happened at a time the researcher was unable to attend.

Note. NNUH = Norfolk and Norwich University Hospital, AHC = Addenbrookes Hospital Cambridge, JPHGY = James Paget Hospital Great Yarmouth, QEHKL = Queen Elizabeth Hospital Kings Lynn.

Table A2  

Recruitment Attempts for the Asthma Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of clinics (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital and/or community clinics approached</td>
<td>37</td>
</tr>
<tr>
<td>Clinics participating</td>
<td>10 (27%)</td>
</tr>
<tr>
<td>Clinics not participating</td>
<td>27 (73%)</td>
</tr>
</tbody>
</table>

Reasons for not participating:

- Small number of patients in target group: 3 (11%)
- Did not want to take part: 8 (30%)
- Could not contact despite several attempts: 16 (59%)
<table>
<thead>
<tr>
<th>GP Surgery</th>
<th>Number of invitation letters sent</th>
<th>Response rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>St Stephens Gate Medical Practice</td>
<td>22</td>
<td>2 (9%)</td>
</tr>
<tr>
<td>Magdalen Medical Practice</td>
<td>26</td>
<td>6 (23%)</td>
</tr>
<tr>
<td>Bowthorpe and Trinity Medical Practice</td>
<td>19</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Timberhill Medical Practice</td>
<td>20</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Hellesdon Medical Practice</td>
<td>31</td>
<td>4 (13%)</td>
</tr>
<tr>
<td>Taverham Surgery Practice</td>
<td>30</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>Acle Medical Practice</td>
<td>101</td>
<td>11 (11%)</td>
</tr>
<tr>
<td>Gayton Road Surgery</td>
<td>75</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Thetford Surgery</td>
<td>63</td>
<td>6 (9%)</td>
</tr>
<tr>
<td>Heathgate Medical Practice</td>
<td>24</td>
<td>2 (8%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>411</strong></td>
<td><strong>37 (9%)</strong></td>
</tr>
</tbody>
</table>
Appendix B

Study Questionnaires

B1  Demographic Information Sheet
B2  Eating Disorder Examination - Questionnaire
B3  Parental Bonding Instrument
B4  Rosenberg Self-Esteem Scale
B5  Depression Anxiety Stress Scale - 21
B6  Illness Severity Visual Analogue Scales
Demographic Information

Please complete the following questions:

What is your diagnosis (Please circle): Type 1 Diabetes  Asthma

Your age: ___years ___ months

Your gender: Male  Female

Current education/employment:

Full-time student  Part-time student  Full-time employment

Part-time employment  Unemployed

Who do you live with? _________________________________________

Who would you describe as your primary caregiver: ________________________

Do you have any children?  Yes  No

If yes, how many and how old are they? ________________________________

What is your parents’ marital status?

Married  Divorced  Separated  Cohabiting  Mum remarried  Dad remarried

How old were you when you were diagnosed with type 1 diabetes/asthma?
___________________________________

How frequently do you attend appointments regarding your type 1 diabetes/asthma?
___________________________________

If you have asthma, do you take steroids as part of your treatment?

Yes - currently  Yes - in the past  No  N/A

Have you ever had a diagnosis of an eating disorder (Please circle)

Yes - currently  Yes - in the past  No

Thank you for taking the time to complete these questions.
**Eating Disorder Examination Questionnaire**

**Instructions:** The following questions are concerned with the past four weeks (28 days) only. Please read each question carefully. Please answer all the questions. Thank you.

**Questions 1 to 12:** Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days) only.

<table>
<thead>
<tr>
<th>Question</th>
<th>No days</th>
<th>1-5 days</th>
<th>6-12 days</th>
<th>13-15 days</th>
<th>16-22 days</th>
<th>23-27 days</th>
<th>Every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2. Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3. Have you tried to exclude from your diet any foods that you like in order to influence your shape or weight (whether or not you have succeeded)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4. Have you tried to follow definite rules regarding your eating (for example, a calorie limit) in order to influence your shape or weight (whether or not you have succeeded)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5. Have you had a definite desire to have an empty stomach with the aim of influencing your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6. Have you had a definite desire to have a totally flat stomach?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7. Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>8. Has thinking about shape or weight made it very difficult to concentrate on things you are interested in (for example, working, following a conversation or reading)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>9. Have you had a definite fear of losing control over eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>10. Have you had a definite fear that you might gain weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>11. Have you felt fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>12. Have you had a strong desire to lose weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
Questions 13-18: Please fill in the appropriate number on the line to the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past four weeks (28 days) ......

13. Over the past 28 days, how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances)? ..............................................

14. ..... On how many of these times did you have a sense of having lost control over your eating (at the time that you were eating)? ..........................................................

15. Over the past 28 days, how many days have such episodes of overeating occurred (i.e., you have eaten an unusually large amount of food and have had a sense of loss of control at the time)? ..........................................................

16. Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight? ..........................................................

17. Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight? ..........................................................

18. Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat, or to burn off calories? ..........................................................

D1. IF YOU HAVE DIABETES, over the past 28 days, how many times have you under dosed on your insulin as a means of controlling your shape or weight? ..........................................................

D2. IF YOU HAVE DIABETES, over the past 28 days, how many times have you omitted using your insulin all together as a means of controlling your shape or weight? ..........................................................

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term "binge eating" means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

19. Over the past 28 days, on how many days have you eaten in secret (i.e. furtively)? ..... Do not count episodes of binge eating.

   | Days | 0   | 1-5  | 6-12 | 13-15 | 16-22 | 23-27 | Every day |
---|------|-----|------|------|-------|-------|-------|-----------|
   | 1    | 2   | 3    | 4    | 5     | 6     |

   None of the times: A few of the times: Less than half: Half of the times: More than half: Most of the time: Every time:

0 1 2 3 4 5 6

20. On what proportion of the times that you have eaten have you felt guilty (felt that you've done wrong) because of its effect on your shape or weight? ..... Do not count episodes of binge eating.

   Not at all: Slightly: Moderately: Markedly

0 1 2 3 4 5 6
Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th>Slightly</th>
<th>Moderately</th>
<th>Markedly</th>
</tr>
</thead>
<tbody>
<tr>
<td>22. Has your <strong>weight</strong> influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>23. Has your <strong>shape</strong> influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>24. How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less, often) for the next four weeks?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>25. How dissatisfied have you been with your <strong>weight</strong>?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>26. How dissatisfied have you been with your <strong>shape</strong>?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>27. How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>28. How uncomfortable have you felt about others seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

What is your weight at present? (Please give your best estimate).

..........................................................................................

What is your height? (Please give your best estimate)

..........................................................................................

If female: Over the past three-to-four months have you missed any menstrual periods?

..........................................................................................

If so, how many? ..........................................

Have you been taking the "pill"? .........................

THANK YOU
**MOTHER FORM**

This questionnaire lists various attitudes and behaviours of parents. Based on how you remember your MOTHER in your first 16 years of life, place a tick in the most appropriate box next to each question.

<table>
<thead>
<tr>
<th>Question</th>
<th>Very like</th>
<th>Moderately like</th>
<th>Moderately unlike</th>
<th>Very unlike</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Spoke to me in a warm and friendly voice.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Did not help me as much as I needed.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Let me do those things I liked doing.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Seemed emotionally cold to me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Appeared to understand my problems and worries.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Was affectionate to me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Liked me to make my own decisions.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Did not want me to grow up.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Tried to control everything I did.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Invaded my privacy.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Enjoyed talking things over with me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Frequently smiled at me.</td>
<td></td>
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<td>14. Did not seem to understand what I needed or wanted.</td>
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<td></td>
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<tr>
<td>15. Let me decide things for myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Made me feel I wasn't wanted.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Very like</td>
<td>Moderately like</td>
<td>Moderately unlike</td>
<td>Very unlike</td>
</tr>
<tr>
<td>---</td>
<td>-----------</td>
<td>----------------</td>
<td>------------------</td>
<td>------------</td>
</tr>
<tr>
<td>17. Could make me feel better when I was upset.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Did not talk with me very much.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Tried to make me feel dependent on her.</td>
<td></td>
<td></td>
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<td>20. Felt I could not look after myself unless she was around.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>21. Gave me as much freedom as I wanted.</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>22. Let me go out as often as I wanted.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. Was overprotective of me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>24. Did not praise me.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>25. Let me dress in any way I pleased.</td>
<td></td>
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</tr>
</tbody>
</table>
FATHER FORM

This questionnaire lists various attitudes and behaviours of parents. Based on how you remember your FATHER in your first 16 years of life, place a tick in the most appropriate box next to each question.

<table>
<thead>
<tr>
<th>Question</th>
<th>Very like</th>
<th>Moderately like</th>
<th>Moderately unlike</th>
<th>Very unlike</th>
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</thead>
<tbody>
<tr>
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<td>7. Liked me to make my own decisions.</td>
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<td>8. Did not want me to grow up.</td>
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<td>9. Tried to control everything I did.</td>
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<td>12. Frequently smiled at me.</td>
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<td>14. Did not seem to understand what I needed or wanted.</td>
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<td>15. Let me decide things for myself.</td>
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<td>16. Made me feel I wasn't wanted.</td>
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<td></td>
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<tr>
<td>17. Could make me feel better when I was upset.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Very like</td>
<td>Moderately like</td>
<td>Moderately unlike</td>
<td>Very unlike</td>
</tr>
<tr>
<td>---</td>
<td>-----------</td>
<td>-----------------</td>
<td>-------------------</td>
<td>------------</td>
</tr>
<tr>
<td>18. Did not talk with me very much.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>19. Tried to make me feel dependent on him.</td>
<td></td>
<td></td>
<td></td>
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<td>20. Felt I could not look after myself unless he was around.</td>
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<td></td>
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<tr>
<td>25. Let me dress in any way I pleased.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Rosenberg Self-esteem Scale

Instructions

Below is a list of statements dealing with your general feelings about yourself. If you strongly agree, circle SA. If you agree with the statement, circle A. If you disagree, circle D. If you strongly disagree, circle SD.

<table>
<thead>
<tr>
<th>Statement</th>
<th>SA</th>
<th>A</th>
<th>D</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. On the whole, I am satisfied with myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. At times, I think I am no good at all.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. I feel that I have a number of good qualities.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. I am able to do things as well as most other people.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. I feel I do not have much to be proud of.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. I certainly feel useless at times.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. I feel that I'm a person of worth, at least on an equal plane with others.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. I wish I could have more respect for myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. All in all, I am inclined to feel that I am a failure.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. I take a positive attitude toward myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of the time
3 Applied to me very much, or most of the time

<table>
<thead>
<tr>
<th>Statement</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>I found it hard to wind down</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I was aware of dryness of my mouth</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I couldn't seem to experience any positive feeling at all</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I experienced breathing difficulty (eg, excessively rapid breathing,</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>breathlessness in the absence of physical exertion)</td>
<td></td>
</tr>
<tr>
<td>I found it difficult to work up the initiative to do things</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I tended to over-react to situations</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I experienced trembling (eg, in the hands)</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I felt that I was using a lot of nervous energy</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I was worried about situations in which I might panic and make a fool of</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>myself</td>
<td></td>
</tr>
<tr>
<td>I felt that I had nothing to look forward to</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I found myself getting agitated</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I found it difficult to relax</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I felt down-hearted and blue</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I was intolerant of anything that kept me from getting on with what I</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>was doing</td>
<td></td>
</tr>
<tr>
<td>I felt I was close to panic</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I was unable to become enthusiastic about anything</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I felt I wasn't worth much as a person</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I felt that I was rather touchy</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I was aware of the action of my heart in the absence of physical</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>exertion (eg, sense of heart rate increase, heart missing a beat)</td>
<td></td>
</tr>
<tr>
<td>I felt scared without any good reason</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>I felt that life was meaningless</td>
<td>0 1 2 3</td>
</tr>
</tbody>
</table>
Visual Analogue Scales – Illness Severity

To help people say how severe their illness is (either asthma or diabetes), we have a drawn a scale (rather like a thermometer) on which the most severe you can imagine the symptoms of your illness being is marked 100, and the least severe you can imagine the symptoms of your illness being is marked 0.

1. We would like you to indicate on this scale how severe the symptoms of your own illness (either asthma or diabetes) have been on average over the past 7 days…

2. We would like you to indicate on this scale how severe the symptoms of your own illness (either asthma or diabetes) have been in the past when they have been at their worst…
Appendix C
Ethical Approval

C1  Original Ethical Approval Letter
C2  Ethical Approval Letter for Major Amendment
C3  Trust Research & Development Approval Letters
31 October 2012

Mrs Judith Hatton
Trainee Clinical Psychologist
University of East Anglia
Cambridge and Peterborough NHS Foundation Trust
PGR Hub Elizabeth Fry Building (2.30)
UEA
Norwich  NR4 7TJ

Dear Mrs Hatton

Study title: Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

REC reference: 12/EE/0411

Thank you for your letter of 12 October 2012 responding to the Committee’s request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair in consultation with another member of the REC.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see “Conditions of the favourable opinion” below).

Non-NHS sites

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.


1. The parent PIS has ‘Will my taking part...’ rather than ‘Will my child’s taking part...’; you are asked to amend this statement.

2. The Committee believe that it would be beneficial to include a telephone number in the contact details (in both PISs) should further information be required.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission (‘R&D approval’) should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at [http://www.rfforum.nhs.uk](http://www.rfforum.nhs.uk).

Where a NHS organisation’s role in the study is limited to identifying and referring potential participants to research sites (“participant identification centre”), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

You should notify the REC in writing once all conditions have been met (except for site approvals from host organisations) and provide copies of any revised documentation with updated version numbers. Confirmation should also be provided to host organisations together with relevant documentation.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

<table>
<thead>
<tr>
<th>Document</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Governing Letter from Judith Hatton</td>
<td></td>
<td>03 August 2012</td>
</tr>
<tr>
<td>Covering Letter email attaching response from Judith Hatton</td>
<td></td>
<td>16 October 2012</td>
</tr>
<tr>
<td>Evidence of insurance or indemnity - Zurich Municipal</td>
<td></td>
<td>15 May 2012</td>
</tr>
<tr>
<td>Investigator CV - Judith Hatton</td>
<td></td>
<td>June 2012</td>
</tr>
<tr>
<td>Letter from Sponsor - UEA</td>
<td></td>
<td>03 August 2012</td>
</tr>
<tr>
<td>Other: CV - Academic Supervisor - Sian Coker</td>
<td></td>
<td>June 2012</td>
</tr>
<tr>
<td>Other: Flowchart for recruitment - asthma group</td>
<td>Version 1.0</td>
<td>10 July 2012</td>
</tr>
<tr>
<td>Other: Flowchart for recruitment - type 1 diabetes group</td>
<td>Version 1.0</td>
<td>10 July 2012</td>
</tr>
<tr>
<td>Other: CV - Academic supervisor - Imogen Hobbs</td>
<td></td>
<td>June 2012</td>
</tr>
<tr>
<td>Participant Information Sheet: - Participant</td>
<td>Version 1.1</td>
<td>October 2012</td>
</tr>
<tr>
<td>Participant Information Sheet: - Parent</td>
<td>Version 1.0</td>
<td>October 2012</td>
</tr>
<tr>
<td>Protocol</td>
<td>Version 1.1</td>
<td>October 2012</td>
</tr>
<tr>
<td>Questionnaire: Eating Questionnaire</td>
<td></td>
<td>October 2012</td>
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</table>
Eating Disturbances in Chronic Illness  J.Hatton

<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosenberg Self-esteem Scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perception of parents questionnaire (mother form)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression, Anxiety and Stress Scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual Analogue Scale - Illness Severity</td>
<td>Version 1.0</td>
<td>June 2012</td>
</tr>
<tr>
<td>Demographic Information Sheet</td>
<td>Version 1.0</td>
<td>June 2012</td>
</tr>
<tr>
<td>Perception of parents questionnaire (father form)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>REC application</td>
<td>Submission code: 105028/337200/1/92</td>
<td>21 June 2012</td>
</tr>
<tr>
<td>Response to Request for Further Information from Judith Hatton, Chief Investigator</td>
<td></td>
<td>12 October 2012</td>
</tr>
</tbody>
</table>

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document “After ethical review – guidance for researchers” gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

12/EE/0411 Please quote this number on all correspondence

With the Committee’s best wishes for the success of this project
Yours sincerely,

Dr Leslie Gelling
Chair

Email: susan.davies@eoe.nhs.uk

Enclosures: "After ethical review – guidance for researchers" [SL-AR2]

Emailed to: Mrs Judith Hatton judith.hatton@uea.ac.uk
Yvonne Kirkham Y.kirkham@uea.ac.uk
Dr Imogen I lobbis j.worlds@uea.ac.uk
Dr Stan Coker s.coker@uea.ac.uk
Mark O'Callaghan mark.ocallaghan@nuh.nhs.uk
Health Research Authority

NRES Committee East of England - Cambridge South
Victoria House
Capital Park
Fulbourn
Cambridge
CB21 5XB
Tel: 01223 597733
Fax: 01223 597645

27 February 2013

Mrs Judith Hatton
Trainee Clinical Psychologist
University of East Anglia/Cambridge and Peterborough NHS Foundation Trust
P&GR Hub Elizabeth Fry Building (2.30)
UEA, Norwich
NR47TJ

Dear Mrs Hatton

Study title: Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

REC reference: 12/EE/0411
Amendment number: Amendment #3 (New recruitment plan)
Amendment date: 11 February 2013
IRAS project ID: 105028
Amendment summary: Changes to recruitment strategy:

Currently, the approved procedure states that young people with type 1 diabetes and asthma will be recruited through hospital clinics and specialist community-based clinics (i.e. primary care respiratory clinics). Potential participants will be invited to take part in the study by either the researcher or their clinician when attending routine clinic appointments.

This approach is proving to be effective for recruiting those with type 1 diabetes, but not for those with asthma because:

1. The majority of 16-18 year olds (target group) with asthma are managed by specialist respiratory nurses based in GP surgeries. However, young people are often not able to attend specific clinic times and are therefore offered ad-hoc appointments throughout the week. This means that the researcher cannot attend clinics to support nurses with recruitment.

2. Although hospital clinics and GP surgeries running specialist asthma clinics do have many patients aged 16-18 years on their asthma registers, very few attend the offered routine appointments. The target population is a difficult to reach group and tend to only attend
appointments in the respiratory clinics if they are having particular difficulties with managing their asthma. Furthermore, routine appointments tend to be yearly. The deadline for recruitment for this study is April 2013 (dictated by the deadline for thesis submission), this means that many routine appointments will be missed. Therefore, in order to be able to recruit a group of young people with asthma, the following recruitment strategy is proposed:

People aged 16-18 years old and on the asthma register at participating hospital clinics and specialist community-based clinics will be identified by a member of their clinical care team. They will be sent a letter inviting them to take part in the study and a copy of the participant information sheet. The mailshot will be carried out by a member of the clinical care team, and therefore the researcher will not have access to confidential patient information. Potential participants will be invited to participate on-line and will be able to contact the researcher to access a paper questionnaire pack, if they wish. The recruitment of the type 1 diabetes group will continue as per the original protocol.

The above amendment was reviewed prior to 28 February 2013 by the Sub-Committee in correspondence.

Ethical opinion

You have found that half of the potential trial subjects are not as accessible as you thought they would be, because the asthma patients are more independent than the diabetic patients. In order to recruit the right number of young people with asthma, you are trying to reach them on-line.

An invitation mail-shot will be sent out to potential study subjects by the asthma clinical team, so you will not have access to personal clinical details of potential subjects, although you will have written the letter.

The member of the Committee taking part in the review feel the letter submitted is appropriate and the revised PIS (as the solution) is considered practical.

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

Approved documents

The documents reviewed and approved at the meeting were:

<table>
<thead>
<tr>
<th>Document</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Letter of invitation to participant</td>
<td>1.0</td>
<td>Jan 2013</td>
</tr>
<tr>
<td>Participant Information Sheet</td>
<td>1.3</td>
<td>Jan 2013</td>
</tr>
<tr>
<td>Protocol</td>
<td>1.3</td>
<td>Jan 2013</td>
</tr>
<tr>
<td>Notice of Substantial Amendment (non-CTIMPs)</td>
<td>Amendment #3 (New recruitment plan)</td>
<td>11 February 2013</td>
</tr>
<tr>
<td>Covering Letter</td>
<td>Email from Judith Hatton</td>
<td>14 February 2013</td>
</tr>
</tbody>
</table>
Membership of the Committee

The members of the Committee who took part in the review are listed on the attached sheet.

R&D approval

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

We are pleased to welcome researchers and R & D staff at our NRES committee members’ training days – see details at http://www.hra.nhs.uk/hra-training/

12/EE/0411: Please quote this number on all correspondence

Yours sincerely

[Signature]

Dr. Leslie Gelling
Chair

E-mail: nrescommittee.eastofengland-cambridgesouth@nhs.net

Enclosures: List of names and professions of members who took part in the review

Email to:

Mrs Judith Hatton judith.hatton@uea.ac.uk

Mark O'Callaghan, Norfolk and Norwich University Hospitals NHS Foundation Trust mark.o'callaghan@nnuh.nhs.uk

Yvonne Kirkham, University of East Anglia y.kirkham@uea.ac.uk
### NRES Committee East of England - Cambridge South

**Attendance at Sub-Committee of the REC meeting on 28 February 2013**

<table>
<thead>
<tr>
<th>Name</th>
<th>Profession</th>
<th>Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dr Leslie Gelling</td>
<td>(Chair) Reader in Research Ethics</td>
<td>Expert</td>
</tr>
<tr>
<td>Dr Frank Wells</td>
<td>(Vice-Chair) Retired Pharmaceutical Physician</td>
<td>Expert</td>
</tr>
</tbody>
</table>
Dear Mrs Judith Hatton

Re: 2012PAED05 Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

REC Number: 12/EE/0411
Chief Investigator: Mrs Judith Hatton
Sponsor: University of East Anglia

Further to your submission of the above project to the R&D office at NHS Norfolk your project has now been reviewed and all the mandatory research governance checks for Participant Identification Centres¹ (PICs) have now been satisfied. I am therefore pleased to inform you on behalf of Norfolk PCT that that agreement was granted on 1st November 2012 for the following Participant Identification Centres to refer patients for this study:

- Norfolk PCT

You may now begin your study at the above PICs.

PIC Authorisation is granted on the basis of the information supplied in the application form, protocol and supporting documentation, if anything subsequently comes to light that would cast doubts upon, or alter in any material way, any information contained in the original application, or a later amendment application there may be implications for continued Authorisation.

Authorisation is granted on the understanding that the study is conducted in accordance with the Research Governance Framework and the terms of REC favourable opinion.

¹ Where potential participants will be identified through NHS organisations other than the research sites themselves, these organisations are termed “Participant Identification Centres” (PIC) NIHR Question Specific Guidance - Part C Version 2.2 dated April 2009.
If you have any queries regarding this or any other project please contact Paul Mills, R&D Officer, at
the above address. Please note, the reference number for this study is 2012PAED05 and this should
be quoted on all correspondence.

Yours sincerely

Dr Augustine Pereira
Consultant in Public Health Medicine, and Research & Development Lead
NHS Norfolk & Waveney

cc: Dr Imogen Hobbs, Academic supervisor, UEA
    Dr Sian Coker, Academic supervisor, UEA
    Yvonne Kirkham, Sponsor representative, UEA

Enc

Conditions of Authorisation
Please note the following conditions of NHS Permission - it is your responsibility to ensure that these
conditions are disseminated to all parties involved in this project at the above sites.

You must notify the R&D Office at NHS Norfolk & Waveney of:
- All practices within Norfolk & Suffolk that are participating in the study
- All proposed changes to this study, whether minor or substantial
- All Serious Adverse Events relevant to the above PICs
- Any deviations from the protocol or protocol breaches including any urgent safety measures
  that are required to be taken in order to protect research participants against any immediate
  hazard to their health or safety
- Any other conditions or complaints in relation to the research project at the above PICs
- Any Sponsor or funder initiated audits, or any regulatory inspections to be conducted in relation
to this study at the above PICs
- The study conclusion and/or termination of the study; where smartcards have been issued, this
  notification must be made on a PIC by PIC basis to allow deactivation of smartcards at that
  PIC.
- All publications relating to the study

Documentation:
You are required to provide all participating PICs with all relevant study information to enable them to
fulfil their role within the research. This will include as a minimum:
  (a) Final approved protocol
  (b) Copies of REC favourable opinion, NHS Permission letter covering that PIC, any other
      approvals necessary (e.g. MHRA)
  (c) Participant information sheets, consent forms, invitation letters, posters/adverts and any other
      documentation given to the participant

2 An incident is defined as any event or circumstance that could have, or did, lead to harm, loss or damage and
includes loss of data, confidentiality breaches, harm to researchers or staff or damage to property.

2012PAED05S (117-08-12)
It is your responsibility to update the information held at each PIC with any amendments made to this
documentation and all approval letters applicable to those amendments and to ensure that all
essential documents held at each PIC are maintained, stored and archived as appropriate.

**Transfer of data**
- Transfer of patient identifiable or confidential data must be in accordance with PCT policies.

**Scope of permission**
- Please note that the above permission applies only to research activity on NHS staff or
  premises or involving NHS Patients and/or their tissues, data or samples. Separate
  agreements and permissions will be required for research involving private patients or those
  under the care of social services.

**Documents Reviewed**
The following documents were reviewed:

- Evidence of Insurance or Indemnity, 15th May 2012
- Investigator CV, 21st June 2012
- Flowchart of Recruitment—asthma group, Version 1, 10th July 2012
- Flowchart for recruitment-type 1 diabetes group, Version 1, 10th July 2012
- Participant Information Sheet, Version 1.1, October 2012
- Study proposal Version 1.1, October 2012
- Eating Questionnaire
- Rosenberg Self-Esteem Scale
- Perception of Parents Questionnaire
- Depression, Anxiety and Stress Scale
- Visual Analogue Scale—Illness Severity, Version 1, 21st June 2012
- Demographic Information Sheet, Version 1, 21st June 2012
- Parent Information Sheet, Version 1.0, October 2012

**Other Documents reviewed**
- Signed NHS R&D Form, Look code 105028/337204/14/889
- Response to Ethics, 12th October 2012
Dear Dr Evans

Re: 12/EE/0411 Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem

In accordance with the Department of Health's Research Governance Framework for Health and Social Care, all research projects taking place within the Trust must receive a favourable opinion from an ethics committee and approval from the Department of Research and Development (R&D) prior to commencement.

I am pleased to confirm that Cambridge University Hospitals NHS Foundation Trust has reviewed the above study and agree to act as a Participant Identification Centre (PIC) referring potential participants to the relevant research teams based in University of East Anglia.

Please note that as a PIC the Trust does not provide indemnity for this study.

Sponsor: University of East Anglia
Funder: PhD project, no external funding
End date: 01/06/2013
Protocol: version 1.1, dated October 2012

The project must follow the agreed protocol and be conducted in accordance with all Trust Policies and Procedures especially those relating to research and data management.

Please ensure that you are aware of your responsibilities in relation to The Data Protection Act 1998, NHS Confidentiality Code of Practice, NHS Caldicott Report and Caldicott Guardians, the Human Tissue Act 2004, Good Clinical Practice, the NHS Research Governance Framework for Health and Social Care, Second Edition April 2005 and any further legislation released during the time of this study.

Members of the research team must have appropriate substantive or honorary contracts with the Trust prior to the study commencing. Any additional researchers who join the study at a later stage must also hold a suitable contract.

Innovation and excellence in health and care
Addenbrooke's Hospital | Rosie Hospital
NIHR – Cambridge Biomedical Research Centre | Academic Health Science Centre – Cambridge University Health Partners
If the project is a clinical trial under the European Union Clinical Trials Directive the following must also be complied with:


Amendments

Please ensure that you submit a copy of any amendments made to this study to the R&D Department.

Annual Report

It is obligatory that an annual report is submitted by the Chief Investigator to the research ethics committee, and we ask that a copy is sent to the R&D Department. The yearly period commences from the date of receiving a favourable opinion from the ethics committee.

Please refer to our website www.cuh.org.uk/research for all information relating to R&D including honorary contract forms, policies and procedures and data protection.

Should you require any further information please do not hesitate to contact us.

Yours sincerely

Louise Stockley
Research Governance Manager
The Queen Elizabeth Hospital
King’s Lynn
NHS Foundation Trust

Dr Parvez Moondi
Chair of the Research Governance Committee
R&D Co-ordinator – Karen Lupton
E-mail: karen.lupton@qeht.nhs.uk
Tel: 01553 214571

Mrs Judith Hatton
PGR Hub Elizabeth Fry Building (2.A30)
University of East Anglia, Norwich
NR4 7TJ

Dear Ms Hatton,

The Queen Elizabeth Hospital
Gayton Road
Kings Lynn
Norfolk
PE30 4ET
www.qehki.nhs.uk

Research and Development

R&D ID 31/12
REC: 12/EE/0411
Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self esteem

Thank you for sending the following documentation relating to the above study:

- Protocol version 1.1 dated October 2012
- R&D form - 10328/557294/14/9/99

This study has been reviewed by the Trust’s Research Governance team and we can confirm that the Trust is willing for this work to take place.

A letter of access will be issued allowing you to conduct these research activities within our Trust.

I would like to take this opportunity to remind you that the Trust manages all research in accordance with the requirements of the Research Governance Framework.

In order to comply with the above, if the study is not completed within one year from the date of this letter, a report summarising the progress of the study should be submitted to the R&D Office. In the case of multi-centre studies this is usually provided by the Chief Investigator/Clinical Trials Unit. Alternatively, we can supply a blank form for you to complete: please contact us for a copy.

YOU ARE REQUIRED TO NOTIFY OUR R&D OFFICE THE DATE OF THE FIRST PARTICIPANT YOU RECRUIT TO THE STUDY – IF APPLICABLE

On completion of the project, please forward to the R&D Office any “final report” relating to the project – e.g. report from Chief Investigator/Clinical Trials Unit, copy of any published articles, etc. Any reports resulting from the study, which may be produced at a later date, should also be forwarded, to ensure a complete record is held here.

If our department can be of any further assistance please do not hesitate to contact me.

Yours sincerely,

Dr Parvez Moondi
Chair of the Research Governance Committee

Dated: 3 December 2012

CC: Mrs Allison Piper, Clinical Health Psychology
Dr A Pawlowicz, Consultant Physician

Chair, Kate Gordon OBE, CEO; Executive; Patricia Wright
Patron, Her Majesty The Queen
The Preferred Hospital for Local People

218
Mrs Judith Hatton  
University of East Anglia  
PGR Hub Elizabeth Fry Building (2.30)  
Norwich Research Park  
Norwich  
NR4 7TJ

02 November 2012

Dear Mrs Hatton,

Re:  R&D Reference Number: 2012PAED005G (117-08-12)  
Project Title: Risk factors for eating disturbances in young people with type 1 diabetes and chronic asthma: The role of parenting style and self-esteem.

I am pleased to inform you that the above project has been given full NHS permission for research at Norfolk & Norwich University Hospitals NHS Foundation Trust as a Participant Identification Centre.

This NHS permission for research has been granted on the basis described in the application form, protocol and supporting documentation as listed below:

<table>
<thead>
<tr>
<th>Governance Documents</th>
<th>Version No</th>
<th>Date</th>
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</thead>
<tbody>
<tr>
<td>Protocol</td>
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<td>01/01/2012</td>
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<tr>
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<td>1.1</td>
<td>01/10/2012</td>
</tr>
<tr>
<td>PIS: parent</td>
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<td>01/10/2012</td>
</tr>
<tr>
<td>REC favourable opinion letter</td>
<td></td>
<td>31/10/2012</td>
</tr>
<tr>
<td>R&amp;D form</td>
<td>105/026/337200/14/889</td>
<td>10/07/2012</td>
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<td>Flowchart for recruitment: T1DM</td>
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</tr>
<tr>
<td>Flowchart for recruitment: asthma</td>
<td>1.0</td>
<td>10/07/2012</td>
</tr>
</tbody>
</table>

I have enclosed two copies of the Standard Terms and Conditions of Approval. Please sign both copies and return one copy to the Research & Development Department at the above address and keep the other in your study file. Failure to return the standard terms and conditions may affect the conditions of approval.

Please note, under the agreed Standard Terms and Conditions of Approval you must inform the R&D department of any proposed changes to this study and submit annual progress reports to the R&D department.

If you have any queries regarding this or any other project please contact Clare Collum, Research Facilitator, at the above address. Please note, the reference number for this study is 2012PAED005G (117-08-12) and this should be quoted on all correspondence.

Yours sincerely,

[Signature]

Professor Krishna Sethia  
Medical Director

[Signature]

Professor Marcus Flather  
R&D Director
Eating Disturbances in Adolescents with Chronic Illness

R&D Ref No: 2012/STU/03
REC Ref: 12/EE/0411
CSP Ref: N/A

Thank you for submitting your research application for NHS Permission to undertake the above study. This is to confirm that your study has been reviewed by the Research Support and Governance Group and this site has been approved as a Participant Identification Centre.

The following documents were reviewed:

<table>
<thead>
<tr>
<th>Document</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>R&amp;D Application Form</td>
<td>105028/337204/14/089</td>
<td>21/06/12</td>
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<tr>
<td>Protocol</td>
<td>1.1</td>
<td>October 2012</td>
</tr>
<tr>
<td>Participant Information Sheet</td>
<td>1.2</td>
<td>October 2012</td>
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<td>Parent Information Sheet</td>
<td>1.1</td>
<td>October 2012</td>
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<tr>
<td>Flowchart</td>
<td>1.0</td>
<td>10/07/12</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>VAS – Illness Severity</td>
<td>1.0</td>
<td>June 2012</td>
</tr>
<tr>
<td>Rosenberg Self Esteem Scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Demographic Information Sheet</td>
<td>1.0</td>
<td>June 2012</td>
</tr>
<tr>
<td>Parental Binding Instrument (Mother &amp; Father)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating Disorder Examination Questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td>UEA Ethics</td>
<td>Dr J Watson</td>
<td></td>
</tr>
<tr>
<td>CV (Chief Investigator)</td>
<td>Judith Hatton</td>
<td></td>
</tr>
<tr>
<td>Evidence of Insurance</td>
<td>Zurich</td>
<td>15/05/12</td>
</tr>
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<td>REC Correspondence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confirmation of Ethical Approval</td>
<td>Cambridge South – REC</td>
<td>31/10/12</td>
</tr>
<tr>
<td>Covering Letter – amended documents</td>
<td>Judith Hatton</td>
<td>31/10/12</td>
</tr>
</tbody>
</table>
The National Institute for Health Research (NIHR) has implemented a 70 day benchmark relating to the time taken to recruit the first patient to each study undertaken and is a metric against which we, as a trust are measured. Since future research funding will be determined on our overall performance it is imperative that, where feasible, at least one participant is recruited to this study within 70 days of the date that the application was submitted for NHS Permission.

The 70 Day period for this study started on: 08/11/12
Your Day 70 recruitment target date is: 08/02/13

You must advise the R&D Department (see email address above) as soon as the first patient is consented – if the study is a randomised study you must also advise the randomisation date, when available. If you fail to recruit within this timeframe you must advise the R&D Department of the reasons why recruitment was not possible.

By July 2013 Trusts will be obliged to make public data relating to recruitment to all research studies being undertaken so notification of accurate data to the R&D Department is essential.

That the investigator will refer proposed amendments and updates to trial documentation to the Group and obtain the Group's approval prior to implementation (except in cases of emergency where the welfare of the subject is paramount).

It is the investigator's responsibility to notify the Group immediately of any information received or of which they become aware, which would cast doubts upon, or alter, any information contained in the original application, or a later amendment application, submitted to the Group which would raise questions about the safety and / or continued conduct of the research. Such information will be reviewed by the Chair of the Group and appropriate action taken.

The investigator must comply with the requirements to furnish the Research Support and Governance Group with details of the progress of the research project submitted on the Research Progress Monitoring form at a frequency specified in the letter of approval received from the Research Ethico Committee (usually annually). The report should include the conclusion, outcomes of the research and any publications arising from the study, and inform the Research Support and Governance Group if the research has been discontinued. The investigator accepts the responsibility to comply with the requirement to complete and submit a Research Project Completion report.

The Group may require certain projects to produce more regular progress reports depending on the risk assessment of the research. The progress/final monitoring report forms will be received and reviewed by the R&D Manager and reported to the Group. If no report is received the R&D Manager will take steps to ascertain whether the research is still being conducted.

It is expected that all necessary reporting procedures for adverse event reporting will be in place (eg to MHRA, etc). The Group will expect to be advised of any adverse events, failure to complete research or any other information that may be considered of interest to the Group in the conduct of its work. In the case of serious adverse events to local participants, these must be reported to the Group within 24 hours of such events.
happening or the investigator becoming aware of them. Any subsequent updates to
incident reports must also be reported. The researcher is also required to advise the
Group if the research is withdrawn or if difficulties are experienced in recruiting research
subjects.

A member of the RSGG will review all local serious adverse event reports, the Principal
Investigator is expected to advise the Group of all adverse events that may affect the
running of the study as well as provide Investigator Brochure updates.

- All research may be subject to internal audit (either as part of ‘routine’ or ‘for cause’ audit
  activity) on behalf of the Research Support and Governance Group.

- Good Clinical Practice (GCP) training is mandatory for Chief and Principal Investigators
  and desirable for all other researchers involved in clinical trials (eg trials involving drugs,
  medical devices, implants etc) or other research studies. GCP training should be
  refreshed 2 yearly for those researchers actively involved in research projects. Anyone
  trained in GCP who has not actively taken part in any recent research should refresh
  their GCP training prior to the start of a research project. All researchers are expected
  to attend refresher training in the event of major changes to the GCP guidelines.

Yours sincerely,

Dr WG Notcutt
Chairman
Research Support and Governance Group

CC:
Appendix D:

Normative Data for Measures
Table D1.

Descriptive Statistics and Normative Data for the EDE-Q Subscales and Global Scale for the Type 1 Diabetes Group

<table>
<thead>
<tr>
<th>EDE-Q subscale</th>
<th>Females ($n = 35$)</th>
<th>Males ($n = 30$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ($SD$)</td>
<td>Normative data$^a$</td>
</tr>
<tr>
<td>Restraint</td>
<td>1.45 (1.56)</td>
<td>1.25 (1.32)</td>
</tr>
<tr>
<td>Eating concern</td>
<td>1.43 (1.41)</td>
<td>0.62 (0.86)</td>
</tr>
<tr>
<td>Weight concern</td>
<td>2.35 (1.82)</td>
<td>1.59 (1.37)</td>
</tr>
<tr>
<td>Shape concern</td>
<td>2.68 (1.75)</td>
<td>2.15 (1.60)</td>
</tr>
<tr>
<td>Global score</td>
<td>1.97 (1.48)</td>
<td>1.55 (1.21)</td>
</tr>
</tbody>
</table>

$^a$Fairburn and Beglin (1994); $^b$Lavender, De Young and Anderson (2010).

Table D2.

Descriptive Statistics and Normative Data for the EDE-Q Subscales and Global Scale for the Asthma Group

<table>
<thead>
<tr>
<th>EDE-Q subscale</th>
<th>Females ($n = 29$)</th>
<th>Males ($n = 8$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ($SD$)</td>
<td>Normative data$^a$</td>
</tr>
<tr>
<td>Restraint</td>
<td>2.03 (1.80)</td>
<td>1.25 (1.32)</td>
</tr>
<tr>
<td>Eating concern</td>
<td>1.64 (1.60)</td>
<td>0.62 (0.86)</td>
</tr>
<tr>
<td>Weight concern</td>
<td>2.83 (1.59)</td>
<td>1.59 (1.37)</td>
</tr>
<tr>
<td>Shape concern</td>
<td>3.30 (1.57)</td>
<td>2.15 (1.60)</td>
</tr>
<tr>
<td>Global score</td>
<td>2.45 (1.51)</td>
<td>1.55 (1.21)</td>
</tr>
</tbody>
</table>

$^a$Fairburn and Beglin (1994); $^b$Lavender, De Young and Anderson (2010).
## Table D3.

Descriptive Statistics and Normative Data for the RSES for Males and Females in Each Group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Rosenberg self-esteem scale</th>
<th>Normative data&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td></td>
</tr>
<tr>
<td><strong>Type 1 diabetes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n = 30)</td>
<td>21.83 (6.04)</td>
<td>30.54 (5.72)</td>
</tr>
<tr>
<td>Female (n = 35)</td>
<td>17.11 (6.30)</td>
<td>28.37 (5.36)</td>
</tr>
<tr>
<td><strong>Asthma</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n = 8)</td>
<td>18.47 (7.13)</td>
<td>30.54 (5.72)</td>
</tr>
<tr>
<td>Female (n = 29)</td>
<td>13.83 (6.32)</td>
<td>28.37 (5.36)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Bagley and Mallick (2001)
Appendix E:

Results of Mann Whitney U Tests:

DASS-21 and Illness Severity Visual Analogue Scales
### Table E1.

**Mann Whitney U Tests for Differences in the DASS-21 Subscales Between the Type 1 Diabetes Group and the Asthma Group**

<table>
<thead>
<tr>
<th>DASS-21 subscale</th>
<th>Z statistic</th>
<th>p value</th>
<th>Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mdn value</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>-2.59</td>
<td>.010**</td>
<td>4.00</td>
<td>8.00</td>
</tr>
<tr>
<td>Anxiety</td>
<td>-2.15</td>
<td>.031*</td>
<td>3.00</td>
<td>5.00</td>
</tr>
<tr>
<td>Stress</td>
<td>-1.72</td>
<td>.086</td>
<td>5.00</td>
<td>8.00</td>
</tr>
<tr>
<td>Global negative</td>
<td>-2.45</td>
<td>.014*</td>
<td>4.83</td>
<td>6.33</td>
</tr>
</tbody>
</table>

* *p < .05, **p < .01

### Table E2.

**Mann Whitney U Tests for Differences in the Past and Current Illness Severity Visual Analogue Scale (VAS) Ratings Between the Type 1 Diabetes and the Asthma Group**

<table>
<thead>
<tr>
<th>Illness severity</th>
<th>Z statistic</th>
<th>p value</th>
<th>Type 1 diabetes</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mdn value</td>
<td></td>
</tr>
<tr>
<td>Current severity</td>
<td>-.63</td>
<td>.530</td>
<td>20.00</td>
<td>20.00</td>
</tr>
<tr>
<td>Past severity</td>
<td>-.644</td>
<td>.519</td>
<td>70.00</td>
<td>69.00</td>
</tr>
</tbody>
</table>
Appendix F:

Tests of Normality for Transformed data.
Table F1.

Tests of Normality for Log Transformed EDE-Q, PBI and RSES Data for the Type 1 Diabetes Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Kolmogorov-Smirnov Statistic</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.12</td>
<td>65</td>
<td>.024*</td>
</tr>
<tr>
<td>PBI care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.22</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>Paternal</td>
<td>.28</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>PBI overprotection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.18</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>Paternal</td>
<td>.22</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>RSES</td>
<td>.18</td>
<td>65</td>
<td>.000**</td>
</tr>
</tbody>
</table>

*p < .05, p** < .01
### Table F2.

*Tests of Normality for Log Transformed EDE-Q, PBI and RSES Data for the Asthma Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Kolmogorov-Smirnov Statistic</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.16</td>
<td>37</td>
<td>.018**</td>
</tr>
<tr>
<td>PBI care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.20</td>
<td>37</td>
<td>.001**</td>
</tr>
<tr>
<td>Paternal</td>
<td>.20</td>
<td>37</td>
<td>.001**</td>
</tr>
<tr>
<td>PBI overprotection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.11</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>Paternal</td>
<td>.14</td>
<td>37</td>
<td>.062</td>
</tr>
<tr>
<td>RSES</td>
<td>.16</td>
<td>37</td>
<td>.021*</td>
</tr>
</tbody>
</table>

*p < .05, p** < .01
Table F3.

*Tests of Normality for Square Root Transformed EDE-Q, PBI and RSES Data for the Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Kolmogorov-Smirnov Statistic</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.08</td>
<td>65</td>
<td>.200</td>
</tr>
<tr>
<td>PBI care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal PBI care</td>
<td>.20</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>Paternal PBI care</td>
<td>.17</td>
<td>64</td>
<td>.000**</td>
</tr>
<tr>
<td>PBI overprotection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal PBI overprotection</td>
<td>.09</td>
<td>64</td>
<td>.200</td>
</tr>
<tr>
<td>Paternal PBI overprotection</td>
<td>.09</td>
<td>64</td>
<td>.200</td>
</tr>
<tr>
<td>RSES</td>
<td>.15</td>
<td>65</td>
<td>.001**</td>
</tr>
</tbody>
</table>

*p < .05, p** < .01
### Table F4

*Tests of Normality for Square Root Transformed EDE-Q, PBI and RSES Data for the Asthma Group*

<table>
<thead>
<tr>
<th></th>
<th>Kolmogorov-Smirnov Statistic</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q global scale</td>
<td>.11</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>PBI care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.17</td>
<td>37</td>
<td>.01**</td>
</tr>
<tr>
<td>Paternal</td>
<td>.14</td>
<td>37</td>
<td>.08</td>
</tr>
<tr>
<td>PBI overprotection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td>.08</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>Paternal</td>
<td>.08</td>
<td>37</td>
<td>.200</td>
</tr>
<tr>
<td>RSES</td>
<td>.14</td>
<td>37</td>
<td>.05*</td>
</tr>
</tbody>
</table>

*p < .05, p** < .01
Appendix G:

Results of Kruskal Wallis Tests:
Differences in EDE-Q scores across Parenting Style Categories
Table G1.

*Kruskal Wallis Test Statistics and Median Values for Differences in the EDE-Q Global Scale Between Parenting Styles in Each Group*

<table>
<thead>
<tr>
<th>Parenting style</th>
<th>Type 1 diabetes ($n=64$)</th>
<th>Asthma ($n=37$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maternal</td>
<td>Paternal</td>
</tr>
<tr>
<td>Affectionate control</td>
<td>1.99</td>
<td>0.90</td>
</tr>
<tr>
<td>Affectionless control</td>
<td>2.92</td>
<td>2.34</td>
</tr>
<tr>
<td>Optimal parenting</td>
<td>0.34</td>
<td>0.46</td>
</tr>
<tr>
<td>Neglectful parenting</td>
<td>1.31</td>
<td>0.67</td>
</tr>
</tbody>
</table>

**Kruskall-Wallis Test - $\chi^2$ (df) 16.95 (3) $p = .001^{**}$

$p = .001^{**}$

$p = .032^{*}$

$p = .342$

$p = .837$

*p < .05, **p < .01*
Appendix H:

Mann Whitney U Tests:

Gender Comparisons on the EDE-Q.
Table H1.

*Mann Whitney U Tests for Differences in the EDE-Q Subscales Between Males and Females in the Type 1 Diabetes Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Z statistic</th>
<th>p value</th>
<th>Female (n = 35)</th>
<th>Male (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-3.66</td>
<td>.000*</td>
<td>.80</td>
<td>.00</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-4.13</td>
<td>.000*</td>
<td>1.20</td>
<td>.10</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-4.52</td>
<td>.000*</td>
<td>2.40</td>
<td>.50</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-4.61</td>
<td>.000*</td>
<td>2.75</td>
<td>.10</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-4.70</strong></td>
<td><strong>.000</strong>*</td>
<td><strong>1.84</strong></td>
<td><strong>.28</strong></td>
</tr>
</tbody>
</table>

*p < .000

Table H2.

*Mann Whitney U Test for Differences in the EDE-Q Subscales Between Males and Females in the Asthma Group*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Z statistic</th>
<th>p value</th>
<th>Female (n = 29)</th>
<th>Male (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-1.67</td>
<td>.095</td>
<td>2.20</td>
<td>.60</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-1.18</td>
<td>.237</td>
<td>1.20</td>
<td>.10</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-2.46</td>
<td>.014*</td>
<td>2.60</td>
<td>.70</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-2.31</td>
<td>.021*</td>
<td>3.37</td>
<td>1.12</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-2.03</strong></td>
<td><strong>.042</strong>*</td>
<td><strong>2.37</strong></td>
<td><strong>.70</strong></td>
</tr>
</tbody>
</table>

*p < .05
Table H3.

*Mann Whitney U Test for Differences in the EDE-Q Subscales Between Females with Type 1 Diabetes and Asthma*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Z statistic</th>
<th>p value</th>
<th>T1D(^a) (n = 35)</th>
<th>Asthma (n = 29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-1.51</td>
<td>.131</td>
<td>.80</td>
<td>2.20</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-.25</td>
<td>.801</td>
<td>1.20</td>
<td>1.20</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-1.14</td>
<td>.253</td>
<td>2.40</td>
<td>2.60</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-1.42</td>
<td>.154</td>
<td>2.75</td>
<td>3.37</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-1.31</strong></td>
<td><strong>.188</strong></td>
<td><strong>1.84</strong></td>
<td><strong>2.37</strong></td>
</tr>
</tbody>
</table>

\(^a\)T1D = Type 1 diabetes

Table H4.

*Mann Whitney U Test for Differences in the EDE-Q Subscales Between Males with Type 1 Diabetes and Asthma*

<table>
<thead>
<tr>
<th>EDE-Q scale</th>
<th>Z statistic</th>
<th>p value</th>
<th>T1D(^a) (n = 30)</th>
<th>Male (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-1.47</td>
<td>.142</td>
<td>.00</td>
<td>.60</td>
</tr>
<tr>
<td>Eating concern</td>
<td>-1.16</td>
<td>.246</td>
<td>.00</td>
<td>.10</td>
</tr>
<tr>
<td>Weight concern</td>
<td>-1.94</td>
<td>.052</td>
<td>.10</td>
<td>.70</td>
</tr>
<tr>
<td>Shape concern</td>
<td>-1.74</td>
<td>.082</td>
<td>.50</td>
<td>1.12</td>
</tr>
<tr>
<td><strong>Global EDE-Q</strong></td>
<td><strong>-1.91</strong></td>
<td><strong>.056</strong></td>
<td><strong>.28</strong></td>
<td><strong>.70</strong></td>
</tr>
</tbody>
</table>

\(^a\)T1D = Type 1 diabetes
### Table H5.

*Mann Whitney U Test for Differences in the PBI Subscales and RSES Between Males and Females with Type 1 Diabetes*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$Z$ statistic</th>
<th>$p$ value</th>
<th>Female ($n = 34$)</th>
<th>Males ($n = 30$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal care</td>
<td>-.15</td>
<td>.882</td>
<td>30.00</td>
<td>31.00</td>
</tr>
<tr>
<td>Paternal care</td>
<td>-.77</td>
<td>.952</td>
<td>25.50</td>
<td>27.00</td>
</tr>
<tr>
<td>Maternal overprotection</td>
<td>-.06</td>
<td>.439</td>
<td>9.00</td>
<td>10.00</td>
</tr>
<tr>
<td>Paternal overprotection</td>
<td>-2.21</td>
<td>.027*</td>
<td>10.00</td>
<td>5.50</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>-2.94</td>
<td>.003**</td>
<td>17.00</td>
<td>22.00</td>
</tr>
</tbody>
</table>

*p < .05, ** p < .01

### Table H6

*Mann Whitney U Test for Differences in the PBI Subscales and RSES Between Males and Females with Asthma*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$Z$ statistic</th>
<th>$p$ value</th>
<th>Female ($n = 29$)</th>
<th>Males ($n = 8$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal care</td>
<td>-1.15</td>
<td>.252</td>
<td>29.00</td>
<td>31.50</td>
</tr>
<tr>
<td>Paternal care</td>
<td>-.63</td>
<td>.530</td>
<td>25.00</td>
<td>22.00</td>
</tr>
<tr>
<td>Maternal overprotection</td>
<td>-1.09</td>
<td>.275</td>
<td>14.00</td>
<td>9.00</td>
</tr>
<tr>
<td>Paternal overprotection</td>
<td>-1.85</td>
<td>.853</td>
<td>14.00</td>
<td>11.00</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>-1.59</td>
<td>.112</td>
<td>12.00</td>
<td>18.39</td>
</tr>
</tbody>
</table>
Appendix I:

Moderation Analyses.
Moderation Analysis: Self-esteem and Eating Disturbances

Simple regression analysis was used to determine the predictive ability of self-esteem in relation to global eating disturbances for the whole sample. The assumptions of normality and linearity were met. The total variance explained by self-esteem was 51% \( (R^2 \text{ adjusted}) \), \( F(1, 102) = 104.60, p < .000 \). The interaction effect was not significant \( (b = .01, t = .17, p = .865) \) indicating that the relationship between self-esteem and global eating disturbances was not moderated by illness diagnosis (Table II). This effect can be seen in Figure II, which indicates a strong negative relationship between self-esteem and global eating disturbances for both groups.

Table II

<table>
<thead>
<tr>
<th>Variable</th>
<th>b</th>
<th>SE B</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>4.04</td>
<td>0.92</td>
<td>4.40</td>
<td>( p &lt; .000^{***} )</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>0.15</td>
<td>0.59</td>
<td>0.25</td>
<td>( p = .803 )</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>-0.16</td>
<td>0.05</td>
<td>-3.27</td>
<td>( p &lt; .000^{***} )</td>
</tr>
<tr>
<td>Self-esteem X diagnosis</td>
<td>0.01</td>
<td>0.03</td>
<td>0.17</td>
<td>( p = .865 )</td>
</tr>
</tbody>
</table>

Note. \( R^2 = .52 \)

*** Significant at \( p < .000 \)
Moderation Analysis: Parent Care and Eating Disturbances

Two regression analyses were used to determine separately the ability of maternal and paternal care in predicting global eating disturbances for the whole sample. In the first regression model the total variance explained by maternal care was 15% ($R^2$ adjusted), $F(1, 101) = 18.22, p < .000$. In the second model paternal care explained 12% ($R^2$ adjusted) of the variance, where $F(1, 101) = 14.11, p < .000$. The assumption of linearity was met. Inspection of the regression residual histograms suggested that the residual distributions approximated a normal distribution. However, Kolmogorov-Smirnov tests of normality revealed that the residuals for both the maternal care regression model ($d = .17, p < .000$) and the paternal care...
regression model ($d = .10$, $p = .009$) significantly deviated from a normal distribution. However, as this was an exploratory analysis and the model parameters were of more interest than significance testing, it was deemed appropriate to carry out the regression analysis, as described in Field (2013).

There were no significant interaction effects for either the maternal care ($b = -.02$, $t = -.50$, $p = .616$) or paternal care ($b = .05$, $t = 1.73$, $p = .085$) interaction models (Tables I2 & I3). This suggests that the relationships between both maternal care and eating disturbances and paternal care and eating disturbances are not significantly moderated by diagnosis.

**Table I2**

*Linear Model of Maternal Care, Diagnosis and Maternal Care x Diagnosis as Predictors of Global EDE-Q Score*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$b$</th>
<th>$SE$</th>
<th>$t$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>2.02</td>
<td>1.83</td>
<td>1.10</td>
<td>.273</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>1.35</td>
<td>1.17</td>
<td>1.15</td>
<td>.251</td>
</tr>
<tr>
<td>Maternal care</td>
<td>-0.05</td>
<td>0.06</td>
<td>-0.83</td>
<td>.406</td>
</tr>
<tr>
<td>Maternal care X diagnosis</td>
<td>-0.02</td>
<td>0.04</td>
<td>-0.50</td>
<td>.616</td>
</tr>
</tbody>
</table>

*Note. $R^2 = .22$*
Table I3

Linear Model of Paternal Care, Diagnosis and Paternal Care x Diagnosis as Predictors of Global EDE-Q Score

<table>
<thead>
<tr>
<th>Variable</th>
<th>b</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>3.49</td>
<td>1.13</td>
<td>3.08</td>
<td>.002**</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>-.41</td>
<td>.77</td>
<td>-.53</td>
<td>.596</td>
</tr>
<tr>
<td>Paternal care</td>
<td>-.13</td>
<td>.04</td>
<td>-2.87</td>
<td>.005**</td>
</tr>
<tr>
<td>Paternal care X diagnosis</td>
<td>.05</td>
<td>.03</td>
<td>1.73</td>
<td>.0855</td>
</tr>
</tbody>
</table>

Note. $R^2 = .22$

** Significant at $p < .01$

Figure I2. Regression of eating disturbances on the interaction between maternal care and
diagnosis.

Figure I2 indicates that there is a negative relationship between maternal care and eating disturbances for both groups. On the contrary, Figure I3 suggests that there may be a trend toward the negative relationship between paternal care and eating disturbances being moderated by diagnosis, although this does not reach significance here. The graph suggests that there may be a stronger negative association between paternal care and eating disturbances for the type 1 diabetes group compared to the asthma group.

*Figure I3.* Regression of eating disturbances on the interaction between paternal care and diagnosis.
Moderation Analysis: Maternal Overprotection and Eating Disturbances

As before, a simple regression analysis was carried out to determine the predictive ability of maternal overprotection on global eating disturbances for the whole sample. In this model the total variance explained by maternal overprotection was 15% ($R^2$ adjusted), $F$ (1, 101) = 19.10, $p < .000$. The assumption of linearity was met. Inspection of the regression residual histogram for the regression model suggested that the distribution of the residuals was positively skewed. This deviation from normality was confirmed by Kolmogorov-Smirnov tests of normality. Despite this, regression analyses were utilised in order to identify possible interaction trends.

Table I4

Linear Model of Maternal Overprotection, Diagnosis and Maternal Overprotection $\times$ Diagnosis as Predictors of Global EDE-Q Score

<table>
<thead>
<tr>
<th>Variable</th>
<th>b</th>
<th>SE</th>
<th>T</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-.41</td>
<td>.78</td>
<td>-.52</td>
<td>.603</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>.87</td>
<td>.57</td>
<td>1.53</td>
<td>.129</td>
</tr>
<tr>
<td>Maternal overprotection</td>
<td>.11</td>
<td>.06</td>
<td>1.70</td>
<td>.093</td>
</tr>
<tr>
<td>Maternal overprotection $\times$ diagnosis</td>
<td>-.02</td>
<td>.04</td>
<td>-.52</td>
<td>.602</td>
</tr>
</tbody>
</table>

Note. $R^2 = .20$

No significant interaction effects were found for the maternal overprotection ($b = -.02$, $t = -.52$, $p = .602$) interaction model (Table I4, Figure I4). This indicates that type of diagnosis did not significantly moderate the relationship between maternal overprotection and eating disturbances.
Figure I4. Regression of eating disturbances on the interaction between maternal overprotection and diagnosis.