DISORDERED EATING ATTITUDES AND BEHAVIOURS IN COELIAC DISEASE

ROSE-MARIE SATHERLEY

A thesis submitted to the University of Birmingham for the degree of DOCTOR OF PHILOSOPHY

University of Birmingham
School of Psychology
June 2017

Abstract

This thesis examines the relationship between coeliac disease (CD) and disordered eating attitudes and behaviours. The literature review describes the development of a theoretical model of disordered eating in CD that will be evaluated throughout this thesis. Chapter Three reports the results of a study that found a high prevalence of disordered eating in CD. Chapter Four reports the results of a qualitative study; participants in this study discussed an increased concern around food that affected their eating patterns. Chapter Five describes the development and validation of the CD Food Attitudes and Behaviours Scale (CD-FAB), which was designed to assess the increased food concerns reported in CD. Chapter Six reports the results of an online survey that explored the correlates of this tool; participants with increased food concerns were more psychologically distressed and had an impaired quality of life. Chapter Seven reports the results of a laboratory study that explored the relationship between food concerns, food intake and cognitive processes related to eating. Overall, this thesis provides novel experimental and theoretical insights into the relationship between CD and disordered eating. The findings have implications for the management and treatment of people with CD.

Acknowledgements

Both my supervisors, Ruth and Suzanne, have provided me with fantastic academic and personal support throughout the last three years. Their supervision has improved the quality of my work and I am amazed they managed to put up with me. I would also like to thank all of the participants who took part in my research; I wouldn't have been able to complete this without them!

Fiona Lerigo, my partner in crime, is less deserving of an acknowledgement but without you distracting me with swing dancing and coffee shop adventures, I would have gone stir-crazy in our quiet office. I would also like to thank Caroline Hetherington for listening to all my whinging and moaning over the last three years worth of lunch breaks and for encouraging me to take well-deserved rock climbing breaks (as well as this thesis, I can now climb V4 routes)! The Wayfarers have made my time at Birmingham thoroughly enjoyable, and I can't thank them enough for being there over the last three years. Katherine Smith also exists.

Finally, I would like to thank my parents and sisters for supporting me in this journey. The never-ending supply of gluten-free food means I can now actually consume a gluten-free kit-kat without gagging. Issy and Krissy, sometimes spooning on the sofa or watching stupid films was exactly what I needed. Ps. Dad, I promise I won't do another degree!

Disseminations

The contents of this thesis have been presented at the Midlands Health Psychology Symposium (2014), the Coeliac UK Research Symposium (2014-2016), the International Coeliac Disease Symposium (2015) and the British Feeding and Drinking Group (2016).

Experimental work in has been published in peer-reviewed journals:

Chapter 2

Satherley, R., Howard, R., & Higgs, S. (2014). Disordered eating in gastrointestinal disorders. Appetite, *84,* 240-250. R.S. conceptualised the review, screened the search results, conducted the quality appraisal, analysed and extracted data, wrote the manuscript and approved the final manuscript as submitted. R.H. and S.H. screened the search results against eligibility criteria, provided support in the interpretation of results, contributed to and approved the final manuscript as submitted.

Chapter 3

Satherley, R., Howard, R., & Higgs, S. (2016). The prevalence and predictors of disordered eating in women with coeliac disease. Appetite, *107*, 260-267. R.S. conceptualised and designed the study, collected and analysed the data, wrote the manuscript and approved the final manuscript as submitted. R.H. and S.H. provided supervision, provided support in study design, reviewed analyses, support in the interpretation of results and approved the final manuscript as submitted.

Experimental work is under peer-review at:

Chapter 4

Satherley, R., Higgs, S., & Howard, R. (under review). The experiences of living with disordered eating and coeliac disease. Journal of Human Nutrition and Dietetics. R.S. conceptualised and designed the study, collected and analysed the data, wrote the manuscript and approved the final manuscript as submitted. R.H. and S.H. provided supervision, took part on discussions in identifying qualitative codes and themes, and approved the final manuscript as submitted.

Chapter 5

Satherley, R., Howard, R., & Higgs, S. (under review). The development and validation of the coeliac disease food attitudes and behaviours scale. Alimentary Pharmacology and Therapeutics. R.S. conceptualised and designed the study, collected and analysed the data, wrote the manuscript and approved the final manuscript as submitted. R.H. and S.H. provided supervision, support in the study design, interpretation of data and analyses, and approved the final manuscript as submitted.

Tables and Figures

Chapter One

Chapter Two

Chapter Three

Chapter Four

Chapter Five

Chapter Six

Chapter Seven

Chapter Eight

Appendix

CHAPTER ONE: DISORDERED EATING AND CD: WHAT IS CURRENTLY KNOWN

1.0. Chapter Rationale

Coeliac Disease (CD) is an autoimmune condition of the small intestine that affects approximately 1 in 100 individuals across the UK (World Gastroenterology Organisation, 2013). The condition is managed by maintaining a strict gluten-free diet (GFD) that requires individuals to attend to what and how they are eating. This increased focus on food may place some individuals with CD at risk for disordered eating patterns.

The purpose of this chapter is to introduce and provide an overview of CD and disordered eating. The chapter will begin with an examination of the pathology and treatment of CD and explore risk factors that may place individuals diagnosed with CD at risk for disordered eating attitudes and behaviours. This chapter will also introduce and describe the phenomenon of disordered eating and provide the rationale for the overall thesis.

1.1. Coeliac Disease

1.1.1. Definition

CD results from a genetic variant that creates an autoimmune reaction that causes the body's cells to show a heightened response to gluten (NICE, 2015). Gluten is the protein that is found in wheat, rye and barley (Green & Jabri, 2003). Some individuals are also sensitive to oats (La Vieille et al., 2016). In a healthy individual, the finger-like projections called villi increase the surface area of the small intestine, which allows nutrients passing through the intestine to be reabsorbed back into the blood stream (NICE, 2015). In individuals with CD, the consumption of gluten causes the villi to flatten (see Figure One), which is known as villous atrophy. Villous atrophy results in a reduced ability to absorb nutrients into the bloodstream (Sollid, 2002).

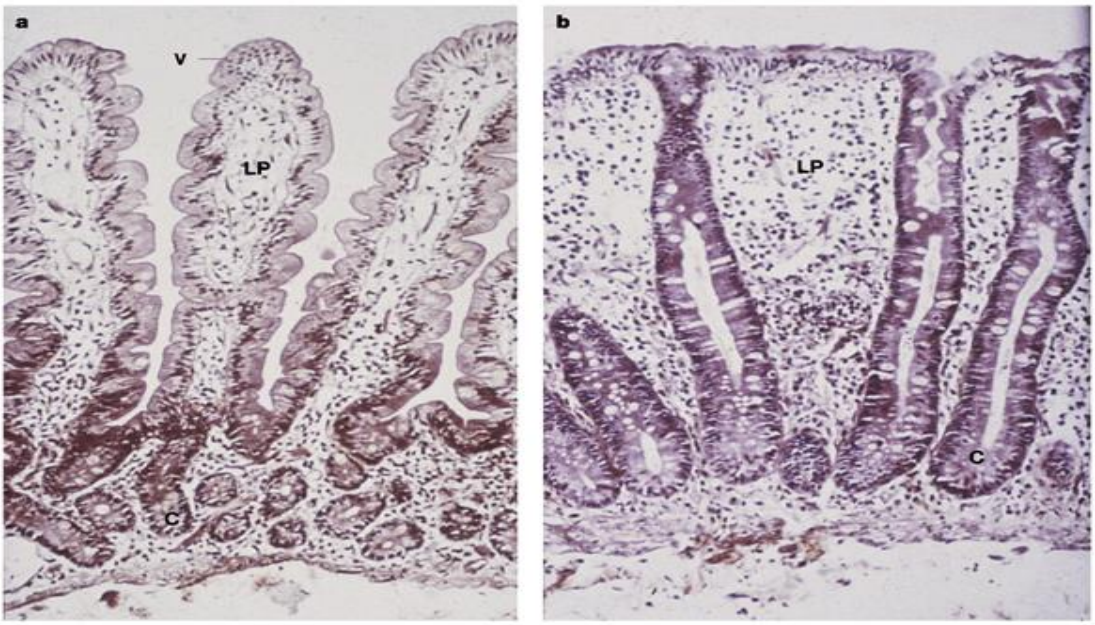

Figure 1: Normal villi in healthy individuals (left); villous atrophy in those with untreated coeliac disease (right; Figure taken with permission from Sollid, 2002, *Nature Reviews Immunology*).

1.1.2. Prevalence

CD is an autoimmune condition that affects approximately 0.5-1% individuals globally (Gjral et al., 2012). The prevalence of CD varies internationally, with diagnosis being less prevalent in the Orient and sub-Saharan Africa, where the diet is largely based on gluten-free foods (Kang et al., 2013). However, these prevalence rates are confounded by the availability of diagnostic facilities. In the UK, CD is present in approximately 1 in 100 individuals (West et al., 2014). Diagnosis is twice as common in females than males (Fasano & Catassi, 2012; West et al., 2014; World Gastroenterology Organisation, 2013). Unlike some other chronic health conditions, CD can present at any point across the life span, but there is an increase in diagnosis between 40 and 60 years (Rashtak & Murray, 2009).

1.1.3. Presentation

There is no typical presentation of CD but symptoms can be divided into gastrointestinal and non-gastrointestinal (NICE, 2015). Gastrointestinal symptoms include diarrhoea, abdominal pain, constipation and bloating (Bao et al., 2012). Non-gastrointestinal symptoms include anaemia, fatigue and osteoporosis (Ludvigsson et al., 2013). This collection of symptoms is common to other health conditions, such as irritable bowel syndrome and other food intolerances, making CD diagnosis challenging (Chowdhury & Osmani, 2016).

To further complicate diagnosis, approximately 36% of individuals present as asymptomatic, meaning there are no clear symptoms associated with their CD (Whyte & Jenkins, 2013). CD also occurs alongside other autoimmune conditions that can mask the symptoms of CD. 10% of individuals with CD have type one diabetes and 7% have autoimmune thyroid disease (Elfstrom et al., 2008; Ludvigsson et al., 2006; Ludvigsson et al., 2013; NICE, 2015).

1.1.4. Current Diagnostic Guidelines for Coeliac Disease

CD is diagnosed via a two-stage process, during which the individual must continue to consume a gluten-containing diet. When CD is suspected, a serological blood test is offered (NICE, 2015). The most sensitive blood tests for CD detect endomysial and tissue transglutaminase antibodies (Tortora et al., 2014). If antibodies are detected, or CD is suspected, a biopsy of the small intestine is taken to assess the presence of villous atrophy, which confirms CD diagnosis (Lee & Green, 2005). There are four guidelines that influence the UK CD diagnostic procedure, all of which recommend the combined use of an intestinal biopsy and serological blood tests (ESPGHAN, 2012; British Society of Gastroenterology, 2014; NICE, 2015; World Gastroenterology Organisation, 2013).

The ESPGHAN guidelines are specific to children with CD, and recommend using only non-invasive methods (serological blood tests) in children who have: 1) transglutaminase antibodies greater than ten-fold above the upper limit of normal; 2) positive endomysial antibodies in a separate blood sample and; 3) carry the genes responsible for CD. The World Gastroenterology Organisation guidelines for adults recommend serological testing and intestinal biopsy, but the biopsy is not mandatory for diagnosis. However, the British Society of Gastroenterology (BSG) guidelines for adults state that the use of intestinal biopsy is essential for CD diagnosis.

Evidence indicates that 75-90% of individuals with CD remain undiagnosed (Kaukinen et al., 2010). Individuals with CD report dissatisfaction with the length of time to obtain a diagnosis (Gray & Papanicolas, 2012); the average time from presentation of symptoms to CD diagnosis is thirteen years (Gray & Papanicolas, 2012). This can result from a lack of CD screening across vulnerable populations, positive blood results with a negative biopsy,

negative blood results with a positive biopsy or the exclusion of gluten prior to or during the diagnostic process (Dharmesh et al., 2015).

Failures in screening individuals for CD result from the unspecific nature of CD symptoms, which makes misdiagnosis common. Chrowdhury and Osmani (2016) found that 9% of individuals with an irritable bowel syndrome diagnosis were positive for CD antibodies. Although serological blood tests are highly sensitive, having an accuracy between 95-100% (Hourigan, 2006), false negatives occur, particularly if the individual has already removed gluten from their diet (Dharmesh et al., 2015). If CD is suspected despite a negative serological test, a biopsy may be used. However, misdiagnosis can occur if the biopsy is taken from an incorrect location or if the sample size of biopsies is insufficient (Freeman, 2008).

Once CD diagnosis has been confirmed, there is referral to a gastroenterology clinic for monitoring of any complications associated with CD and to provide support for commencement of the gluten-free diet (GFD). Individuals should be followed-up twice in their first year after diagnosis to assess symptoms, dietary management, body mass index and serological features (Bai et al., 2013; Husby et al., 2012). Subsequently, annual follow-up of individuals with CD is recommended (British Society of Gastroenterology, 2014; NICE, 2015).

1.2. Living with Coeliac Disease

There is no cure for CD; the condition is controlled by maintaining a strict GFD that reverses villous atrophy, reducing physical symptoms and complications and increasing psychological well-being (Bao & Bhagat, 2012; Burger et al., 2016). Untreated CD leads to a number of health complications including infertility, osteoporosis, weight loss, lymphatic

cancers and increased mortality largely resulting from cardiovascular disease (Garcia-Manzanares & Lucendo, 2011; Rubio-Tapia et al., 2009; Tio, Cox & Eslick, 2012). To prevent these complications, it is essential to encourage strict management of the GFD.

Living with CD and managing the GFD has physical, psychological and social consequences (e.g. Ford, Howard & Oyebode, 2012; Rose & Howard, 2014; Sainsbury, Mullan & Sharpe, 2013). As well as living with and managing a chronic health condition, the need to read food labels, the increased cost of gluten-free foods and the difficulties associated with eating gluten-free outside the home all influence the lives of people with CD.

1.2.1. The Gluten-Free Diet

Managing CD is demanding and involves the removal of all foods containing gluten from the diet. Gluten is found in wheat, barley and rye. Some people are also sensitive to oats, because they contain the protein avenin, which is similar to gluten (Londono et al., 2013). However, a recent review indicates that most people with CD can tolerate oats that have not been contaminated by gluten (La Vieille et al., 2016). This GFD is restrictive and requires foods such as bread, cake, pasta, pastry, some condiments and many processed foods to be removed from the diet; this results in a limited diet that is quite different from the typical Western diet that is usually high in cereal-based staple foods; individuals with CD can feel dissatisfied with the restrictive nature of the diet (Bakshi et al., 2012). However, in the UK, staple gluten-free foods are available on prescription (although this is currently under review) and gluten-free food is becoming more readily available in supermarkets and online. People with CD have the option to consume naturally gluten-free foods such as fresh meat and fish, fruit, vegetables, rice and nuts (Martin & Mercer, 2013).

Gluten-free food can become contaminated by gluten when prepared in the same environment as gluten-containing foods; management of the GFD requires the monitoring of cross-contamination (Schuppan, Dennis & Kelly, 2005). Even trace amounts of gluten consumption can affect individuals with CD. A contamination level of less than 20 parts per million is the cut-off for labelling a food as gluten-free (Collin, Thorell, Kaukinen & Maki, 2004). Cross-contamination is particularly common in social settings including work events, eating out at restaurants and when eating food prepared by others (Lee, Anderson & Ryu, 2014; Schuppan et al., 2005). At home, when preparing food, individuals with CD have more control over the food preparation process ensuring cross-contamination is prevented. However, when eating outside the home, poor knowledge about the GFD, unsuitable food preparation and a lack of autonomy and communication from individuals with CD about the GFD can contribute to cross-contamination and accidental gluten exposure (Zarkadas et al., 2012).

Cross-contamination of gluten-free food products can also occur when they are produced on the same factory production line as gluten containing products, when the same utensils are used for serving gluten-containing foods and gluten-free foods, and when sharing kitchen appliances, such as a toasters (Schuppan et al., 2005). As a result, individuals with CD often use their own cooking utensils and have their own food products and kitchen equipment (Zarkadas et al., 2012).

The majority of individuals respond beneficially to the GFD and feel better as the small intestine recovers and the villi regrow; however, this process can take up to five years (Newnham, Shepherd, Strauss & Hosking, 2016; Wahab, Meijer & Mulder, 2002). Although uncommon, approximately 5% of individuals with CD will not respond to the GFD and have a rare type of CD, called Refractory CD (Rubio-Tapia & Murray, 2010). These individuals

continue to experience gastrointestinal symptoms despite good management of the GFD. For those with refractory CD, nutritional support and steroid treatment may be provided in tertiary care centres; immunosuppression has been effective in encouraging clinical remission (Mooney, Evans, Singh & Sanders, 2012).

1.2.2. Management of the Gluten-Free Diet

GFD adherence ranges between 42-91%, indicating that a large proportion of adults struggle with dietary self-management (Hall, Rubin & Charnock, 2009). In a large cross-sectional study of adults with CD, 40% reported intentionally consuming gluten over a 6-month period and 54% reported accidental gluten consumption (Hall, Rubin & Charnock, 2013). Cognitive factors including limited knowledge, attitudes and illness representations (Silvester et al., 2016; Villafuerte-Galvez, 2015); social factors including public awareness, dining out and social events (White, Bannerman & Gillett, 2016); and emotional factors including depression, anxiety and stress (Wagner et al., 2016) have been associated with poor GFD management (Hall, Rubin & Charnock, 2009). Medical support, being a member of a support group, and obtaining regular dietetic follow-up, older age and needing to gain or lose weight are all associated with improved GFD management (Dowd et al., 2014; Kurppa et al., 2012; Rajpoot et al., 2015).

1.2.3. Label Reading

An essential part of managing the GFD and identifying appropriate food requires the checking and reading of food labels. Label checking is an essential part of managing the GFD because product ingredients can change, meaning an individual with CD must keep updated with recipe adjustments. Individuals also need to be familiar with foods that contain hidden sources of gluten including some sausages, marinades and soups; this requires an

understanding of the sources of gluten-containing ingredients (e.g. malt extract which is derived from barley).

Despite the challenges of identifying gluten-containing foods, labelling has improved considerably in recent years. In 2012, the European Commission introduced labelling standards for gluten-free foods. In order to be classified as "gluten-free", the food product must have less than 20 parts per million of gluten. In addition, the European Commission introduced new food allergen labelling requirements in 2016 that requires food products to emphasise the inclusion of gluten on food ingredient lists (usually via a **bold** type-face). This legislation also requires that restaurants and food outlets provide food ingredient lists for their dishes, including the labelling of gluten-free foods.

No studies have yet evaluated the impact of this change in legislation on the lives of people with CD. However, prior to these changes, Zarkadas et al. (2013) found that 79% of individuals with CD reported concern about the accuracy of food labels. Furthermore, individuals with CD report that the continuous reading of food labels is a time-consuming activity that can be associated with both anger and distress, particularly in those who are newly diagnosed (Rose & Howard, 2014).

1.2.4. Cost and Availability of Gluten-Free Foods

The availability of gluten-free food has increased in recent years but individuals with CD still report difficulties in finding gluten-free products, particularly in smaller stores (Singh & Whelan, 2011). Despite improvements in gluten-free food availability, in 2014, only 41% of restaurants surveyed in the UK sold gluten-free food items (Aziz et al., 2014). The GFD is also considered to be more expensive and viewed as a considerable burden to undertake (Whitaker et al., 2009). Gluten-free food products tend to be 2-6 times more expensive than

their gluten-containing counterparts (Coeliac UK, 2009; Lee, Ng, Zivin & Green, 2007; Singh & Whelan, 2011) and this increased cost and the variable availability of gluten-free foods adds an extra barrier to GFD adherence (Hall et al., 2009; Roma et al., 2010). Individuals with CD who are on a lower income and have poor transport options may feel this burden most significantly (Burden et al., 2015; Lambert & Ficken, 2016). These factors may contribute to impaired quality of life and poor self-management of the GFD (Singh & Whelan, 2011).

Individuals in the UK are entitled to gluten-free foods on prescription, provided by the National Health Service. These items are prescribed by a General Practitioner and include essential food items such as bread, rolls and flour. The number of items prescribed is capped based on the individual's age, gender and co-morbid conditions (Coeliac UK, 2015). The prescription of gluten-free foods increases availability and reduces the cost of gluten-free foods but in recent years, the availability of gluten-free prescriptions has reduced. Not all Clinical Commissioning Groups prescribe gluten-free items to people with CD, which results in differential access throughout the country (known as a postcode lottery effect). Evaluations of General Practitioner records indicate that 70% of Clinical Commissioning Groups are under prescribing gluten-free food products relative to the national guidelines (Coeliac UK, 2015; Martin & Mercer, 2014). The psychosocial and physical impact of reduced access to gluten-free prescriptions remains to be seen.

1.2.5. Eating Outside the Home

The need to maintain a strict GFD at all times, can affect social interactions, which often revolve around food (Olsson, Hornell, Ivarsson & Sydner, 2008). Eating out with CD becomes complicated due to the need to find restaurants that source gluten-free products and are

aware of the risks of cross-contamination. Individuals with CD can find it hard to identify suitable gluten-free items to consume outside the home and these concerns are not unjustified since reports indicate that UK chefs' knowledge about CD and the GFD is lower than that of the general population (Karajeh, Hurlstone, Paten & Sanders, 2005). However, recent reports indicate that awareness and understanding are increasing within the food industry, which may alleviate the challenges for individuals with CD when eating outside the home (Aziz et al., 2014).

Difficulties in eating outside the home can be associated with distress and social isolation (Silvester et al., 2016). When consuming food outside the home, individuals with CD report shame, fear and difficulties in maintaining their dietary management, all of which are associated with reduced quality of life (Hauser et al., 2006; Jacobson, Hallert, Midberg & Friedrichsen, 2012; Zarkadas et al., 2013). Individuals also find it challenging asking for gluten-free food products in public for fear of "being a bother" (Black & Orfilia, 2011; Sverker et al., 2005). Furthermore, poor management of the GFD often occurs in social settings, where eating gluten-free is perceived as a social inconvenience (Olsson, Hornell, Ivarsson & Sydner, 2008).

1.3. Psychosocial Well-Being

Living with CD can have a significant psychological and social impact (Silvester et al., 2016). Eating and food consumption involves more than meeting our nutritional requirements; it is also important in meeting our social and emotional needs (Lee et al., 2012). Although the GFD is essential in reversing gut damage and CD-related symptoms, the need to display dietary vigilance, and monitor food intake and preparation may have a critical impact on psychosocial well-being and quality of life (Barratt, Leeds & Sanders, 2011;

Casellas et al., 2015). Additionally, the diagnosis of a chronic health condition and the burden of living with this diagnosis can places individuals at risk for nonspecific psychological distress (Keles et al., 2007).

1.3.1. Depression and Anxiety

Large population studies indicate an increased risk for psychological distress after CD diagnosis (Ludvigsson et al., 2007; Smith & Gerdes, 2012). Depression persists after CD diagnosis and for some individuals this depression worsens when following the GFD (Addolorato et al., 2001; Zingone et al., 2014). Anxiety is also widely reported by individuals with CD; however, a meta-analysis concluded that anxiety in CD was comparable to the level of anxiety found in healthy controls (Smith & Gerdes, 2012). Levels of anxiety appear to increase prior to diagnosis but receiving the CD diagnosis is associated with feelings of relief (Ciacci et al., 2002). Addolorato et al. (2001) described an increase in anxiety at CD diagnosis but this decreased after the first year of maintaining the GFD, suggesting that the development of anxiety in CD is different to healthy controls.

Before CD diagnosis, the feelings of uncertainty and CD-related symptoms may explain the pre-diagnosis increase in psychological distress (Kurppa et al., 2011). Anxiety may start to decrease when the individual has adapted to the GFD and learns how to manage their CD (Zingone et al., 2014). However, depression may persist as a result of the restrictions placed on one's diet and the burden of living with a chronic health condition (Lee et al., 2012). Additionally, distress can affect CD outcomes and contribute to poor dietary self-management, psychological comorbidity, social isolation and a negative evaluation of the GFD (Sainsbury, Mullan & Sharpe, 2014; Zingone et al., 2014). These psychological co-

morbidities, as well as the general stressors of living with a chronic health condition, may further contribute to psychological distress in CD (Zingone et al., 2014).

1.3.2. Quality of Life

The majority of research exploring quality of life has used generic tools designed for chronic health conditions; however, more recently, specific CD quality of life tools have been designed (Dorn et al., 2010; Hauser et al., 2007; van Doorn et al., 2008). Quality of life is reduced in those with undiagnosed CD compared to healthy controls, particularly in those experiencing gastrointestinal symptoms (Gray & Papanicolas, 2010). However, the effect of the GFD on quality of life is unclear and the findings are mixed.

Several papers using cross-sectional surveys have reported a positive effect of the GFD on quality of life; quality of life scores in CD are similar to the general population (Mustalahti et al., 2002; Lee et al., 2015; Paavola et al., 2012). In symptomatic individuals, time-course assessments indicate that most of the improvement in quality of life is seen three months after starting the GFD and this improvement is maintained at twelve months (Nachman et al., 2009). Furthermore, Rosen et al. (2011) found that after starting the GFD, the majority of individuals saw themselves as more healthy and reported improvements in quality of life. However, 5% of these participants viewed their diagnosis as a stigma that limited their daily lives, particularly in social domains.

Although these studies largely indicate an improvement in quality of life after CD diagnosis, others suggest that the quality of life in CD is much lower than that of the general population, and this is particularly common in females with CD (Altobelli et al., 2013; Hopman et al., 2009). Although quality of life may improve after starting the GFD, these improvements are not necessarily maintained. When Nachman et al. (2009) followed their

participants up at four years, quality of life had reduced, particularly in those who reported poor management of the GFD. Furthermore, for those who had fewer symptoms at diagnosis, quality of life had also deteriorated at four years. The authors suggested this might result from the perceived burden of maintaining the restrictive GFD. Individuals with CD often report avoiding social events and limiting food intake in social settings in order to prevent CD-related symptoms and to prevent being a burden on the social group (Biahetti, Naspi & Catassi, 2013; Skjerning, Mahony, Husby & DunnGalvin, 2014). Furthermore, qualitative findings have consistently reported the burden of the GFD and the negative impact this has on quality of life and psychological well-being (Roos et al., 2013; Rose & Howard, 2014; Skjerning, Mahony, Husby & DunnGalvin, 2014). Rose and Howard (2014) reported that after CD diagnosis, individuals experienced grief around the loss of gluten from their diet, which led to an identity change and a loss of social confidence and activities. The restrictive nature of the GFD negatively impacts quality of life for some individuals with CD. This need to focus on food may contribute to the development of depression, anxiety and the development of disordered eating patterns, in addition to reduced quality of life (Arigo, Anskis & Smyth, 2012). The next section will discuss disordered eating patterns and CD in more detail.

1.4. Eating Disorders and Disordered Eating

A healthy eating pattern describes a balanced diet that contains enough nutrition to meet the body's needs (Freeland-Graves & Nitzke, 2013). Healthy eating attitudes are closely linked to eating patterns and describe a positive attitude around food, where foods are not labelled as "good" or "bad" and are strongly related to both physical and psychological health, and should be both flexible and enjoyable (Freeland-Graves & Nitzke,

2013). Although healthy eating patterns can fluctuate based on factors including food availability and proximity, this should not fluctuate to the point of nutrient deficiency or excess weight change. Thoughts around planning and preparing food may be present but should not dominate thoughts and dictate behaviours above and beyond that of other daily activities (Freeland-Graves & Nitzke, 2013).

1.4.1. Eating Disorders

Eating disorders can be understood on a spectrum ranging from disordered eating to clinically significant eating disorders. At one end of the spectrum, eating disorders describe a psychiatric illness that is marked by disordered eating and disordered beliefs surrounding food (APA, 2013).

Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder, Pica, Rumination Disorder and Avoidant/Restrictive Food Intake Disorder are recognised in the current edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5; APA, 2013). Anorexia Nervosa describes those who pursue marked weight loss, dietary restraint and a disturbance in the perception of one's weight and/or body shape. Bulimia Nervosa describes those who engage in binge eating behaviours followed by compensatory behaviours (e.g. vomiting, excessive exercise, laxative misuse); there is also a disturbance in the perception of one's weight and/or body shape. Binge Eating Disorder is characterised by recurrent episodes of overeating associated with feelings of guilt or depression. The diagnostic criteria for these eating disorders can be found in Table One. These eating disorders have a combined prevalence of approximately 13.1% in the general population (Stice, Marti & Rohde, 2013). Pica, Rumination Disorder and Avoidant/Restrictive Food Intake Disorder will not be

discussed in this thesis, as there is no evidence that they are associated with the diagnosis of chronic health conditions.

Table 1

DSM-5 Diagnostic Criteria for Anorexia Nervosa, Bulimia Nervosa and Binge Eating Disorder

	Diagnostic Criteria (DSM-V, 2013)
Anorexia Nervosa	• Restriction of energy intake leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health • Intense fear of gaining weight or becoming fat even though underweight • Disturbance in experience of weight or shape, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
Bulimia Nervosa	• Eating large amounts of food in a discrete time frame • A sense of lack of control over eating during episodes • Recurrent inappropriate compensatory behaviours to prevent weight gain (purging) • Self-evaluation is unduly influenced by body shape and weight
Binge Eating Disorder	• Eating large amounts of food in a discrete time frame • A sense of lack of control over eating during episodes • Associated with three or more of the following: *Eating more rapidly than normal; eating until uncomfortably full; eating large amounts of food while not physically hungry; eating alone due to embarrassment; feeling disgusted, depressed, or guilty afterward*

1.4.2. Disordered Eating

Disordered eating is characterised by dieting, purging, binge eating, fasting and the use of excessive physical activity in order to control weight and/or body shape (Grilo, 2006; Rosen, 2003). These eating patterns are deviations from healthy eating and may develop into clinically significant eating disorders (Cattarin & Thompson, 1994). Although damaging to physical and psychological health, these eating patterns may meet some of the criteria

for eating disorders but they do not meet the full diagnostic criteria for an eating disorder diagnosis (see Table One).

1.4.3. Disordered Eating in Chronic Health Conditions

Individuals with dietary-controlled chronic health conditions, such as CD, may be at increased risk for disordered eating patterns due to the need to restrict certain food groups and monitor the content of food (Quick et al., 2013). The identification of disordered eating is important in those with chronic health conditions, including CD because it may affect prognosis and treatment. Behaviours such as vigilance around food and dietary monitoring are essential in the care of dietary-controlled chronic health conditions but these behaviours may become dysfunctional when they are used to achieve weight loss, interfere with daily living, impair psychosocial well-being or become a health risk (APA, 2013). As the research on disordered eating in CD is limited, the next section will explore the relationship between disordered eating and diabetes, cystic fibrosis, irritable bowel syndrome and inflammatory bowel disease, all of which have dietary components to their treatment. Exploring disordered eating in the context of other dietary-controlled chronic health conditions can also provide insight into the causes of disordered eating and whether this results from the non-specific burden of a chronic health condition or factors specific to CD.

1.4.4. Diabetes

Disordered eating has been studied extensively in type one and type two diabetes, a condition characterised by blood glucose dysregulation (NICE, 2015). Type one diabetes results from an autoimmune reaction in the cells of the pancreas, meaning the body cannot produce insulin; it is commonly diagnosed in infancy to the late 30s (NICE, 2015). Type two diabetes is more common and occurs because the body's cells develop a resistance to

insulin; it is usually diagnosed in those over 40; however, childhood diagnosis is becoming more common (D'Adamo & Caprio, 2011). In order to regulate blood glucose levels, individuals with type one diabetes will inject insulin for the rest of their lives, and monitor their blood glucose levels and carbohydrate intake in order to calculate the insulin dosage. Individuals with type two diabetes can control their condition through insulin injections, tablets, other medication or dietary-management alone.

Some studies indicate that an eating disorder diagnosis is more common in individuals with type one diabetes than healthy controls (Pinhas-Hamiel & Levy-Shraga, 2013; Young et al., 2013); however, others suggest that the risk is no higher than that of the general population (Baechile et al., 2014; Nielsen, 2002). A systematic-review suggested that the prevalence of disordered eating was 39.3% in those with type one diabetes, compared to 32.5% found in healthy controls which represented a medium effect size (Young et al., 2013). The most common types of disordered eating found in type one diabetes are bingeing and purging. In addition, insulin misuse has been reported as a tool to encourage weight loss, which can be defined as an inappropriate compensatory behaviour in the DSM-5 (Favazza, 2010; Merwin et al., 2014; Wisting et al., 2013). The prevalence of insulin omission in individuals with type one diabetes ranges between 2 and 40% (Colton et al., 2004; Stancin et al., 1989).

Eating disorder diagnosis is also prevalent in type two diabetes, with Binge Eating Disorder being more common in those with type two diabetes compared to healthy controls (Affenito & Adams, 2001; Nicolau et al., 2015). The prevalence rates for Binge Eating Disorder in type two diabetes ranges between 2-25% (Crow, Kendall, Praus & Thuras, 2001; Mannucci et al., 2002). Individuals with both type two diabetes and Binge Eating Disorder tend to be younger and have greater depression scores compared to diabetics without

Binge Eating Disorder (Nicolau et al., 2015). Insulin omission has not been associated with disordered eating in individuals with type two diabetes.

It is not clear why disordered eating presents in diabetes; however, this may result from factors common to all chronic health conditions, such as psychological distress, age at diagnosis and stigma (Colton, Rodin, Olmstead & Daneman, 1999) or factors related to the management of diabetes specifically (e.g. fear of injections, food preoccupation; Ismail et al., 2000; Young-Hyman & Davis, 2010). For individuals with type two diabetes, it is not clear whether the presence of Binge Eating Disorder contributes to the development of diabetes or whether this occurs post-diagnosis.

1.4.5. Cystic Fibrosis

Cystic fibrosis is a genetic disorder characterised by the production of abnormally thick mucus (Hayes, Sheehan, Ulchaker & Rebar, 1994). Individuals with cystic fibrosis are at high risk of malnutrition and long-term treatment includes increased energy intake with 35-40% of calories coming from fat (Mahan & Escott-Stump, 2004). The evidence for disordered eating in those with cystic fibrosis is mixed; some research indicates an increased prevalence compared to the general population (Abbott et al, 2007; Shearer & Bryon, 2004) whereas others report no difference (Bryon, Shearer & Davies, 2008; Raymond et al., 2000). Underweight females with cystic fibrosis may maintain their low weight status by not meeting their increased calorific needs (Warlters, 2001). Disordered eating behaviours in people with cystic fibrosis includes the spitting out of chewed foods, bingeing and purging behaviours and misuse of medication. Quick and Byred-Bredbenner (2014) reported that 25% of their cystic fibrosis sample engaged in self-induced vomiting and medicine misuse,

and one third reported binge eating behaviours; however, this study was limited by a small sample size with only 9 individuals taking part.

1.4.6. Inflammatory Bowel Disease and Irritable Bowel Syndrome

Inflammatory bowel disease and irritable bowel syndrome describe a collection of gastrointestinal conditions, including crohn's disease and ulcerative colitis, which result in uncomfortable and unpleasant gastrointestinal symptoms such as pain, altered bowel habit, bloating, nausea and acid reflux. Irritable bowel syndrome is a functional gastrointestinal disorder, meaning the gastrointestinal tract appears healthy, whereas inflammatory bowel disease is a non-functional gastrointestinal disorder where damage, such as ulcers of inflammation, can be seen in the bowel. Both conditions require the avoidance of certain foods that trigger gastrointestinal symptoms. Individuals with inflammatory bowel disease often take steroids to help manage the condition.

Young people with inflammatory bowel disease can struggle with their prescribed dietary regimens and weight gain resulting from steroid use, which may lead to disordered eating attitudes and behaviours (Eubanks et al., 2002; McDermott et al., 2015; Nicholas et al., 2007; Saha et al., 2015). Due to uncontrollable gastrointestinal symptoms such as bloating and bodily discomfort, greater body shame is more common in individuals with inflammatory bowel disease; this can be associated with negative psychosocial outcomes (Hakanson, Sahlberg-Blom, Nyhlin & Ternestedt, 2009). These factors may contribute to disordered eating attitudes and behaviours (Bayle & Bouvard, 2003; Muller et al., 2010). Disordered eating in inflammatory bowel disease and irritable bowel syndrome is an under researched area and there are no formal prevalence studies; however, comorbid disordered

eating practices and body shame have been reported (Bayle & Bouvard, 2003; Gilbert & Miles, 2002).

1.4.7. Coeliac Disease

The literature examining disordered eating in those with CD is largely dominated by case studies. A systematic review of the empirical evidence will be discussed in Chapter Two. However, evidence drawn from case studies, although limited by sample size, is essential in understanding the nuances of the relationship between CD and disordered eating attitudes and behaviours. Disordered eating in CD appears to be closely related to management of the GFD. Leffler et al. (2007) described the cases of two women with CD age 35 and 40 years, who reported intentional gluten consumption as a strategy to promote weight loss by triggering gastrointestinal symptoms. The potential to promote intentional weight loss through poor gluten-free dietary-management may be observed in those whose weight was lower than expected as a result of untreated CD, or in those who had gained weight following the commencement of the GFD. Weight gain can occur after commencing the GFD, as the intestine starts to recover and nutrients are absorbed into the bloodstream (Kabbani et al., 2012). Both individuals in the case study experienced weight gain when starting the GFD and responded to this by consuming gluten to keep their weight down. In contrast, Ricca et al., (2000) described a 23-year-old woman who followed her GFD extremely well but feared experiencing uncomfortable, coeliac-related symptoms. She avoided eating in public to reduce her exposure to gluten, and reduced the size of her meals to prevent weight gain. Ricca et al., (2000) suggested that the restrictive nature of the GFD might have acted as a trigger for the development of her eating disorder. However, this case of dietary restriction may also have resulted from concerns and anxiety around gluten-

consumption and weight gain, as opposed to the restrictive nature of the GFD. Ricca et al., (2000) also reported evidence for a binge/purge pattern of eating. They described a woman aged 23 with CD whose eating behaviour was characterised by an apparent loss of control over eating, during which she would eat a large amount of gluten-free food (bingeing), followed by episodes of purging.

For individuals with dietary-controlled chronic health conditions, an awareness of food and food intake may act as a risk factor for the development of disordered eating patterns (Quick, Byrd-Bredbenner & Neumark-Sztainer, 2013). In addition, the psychological burden that occurs alongside the diagnosis and management of a chronic health condition may indirectly place these individuals at increased risk for disordered eating behaviours. Colton, Rodin, Olmstead and Daneman (1999) propose that the nonspecific burden associated with chronic disease may lower the threshold for disordered eating in those who are susceptible.

The literature regarding disordered eating and CD indicates that disordered eating may serve different functions across individuals and is related to a variety of factors. These may include the general stressors of living with a chronic health condition or factors specific to living with CD (such as post-diagnosis weight change). To enable a clear and testable understanding of disordered eating in the context of CD, theoretical frameworks are needed to guide future research into the factors that may help us understand this phenomenon. The following section examines a number of theories that can be used to help us understand disordered eating within the context of CD.

1.5. Theoretical Context

A thorough search of the literature (Web of Science with Conference Proceedings, 1900-2016; MEDLINE, 1950-2016; Pubmed; PsychINFO, 1967-2016; and Google Scholar) revealed

no theoretical model to explain disordered eating attitudes and behaviours in CD. At the theoretical level, disordered eating in CD needs to be better understood in order to develop appropriate prevention and intervention strategies, and the construct of disordered eating in relation to CD requires further exploration. With an understanding of the interplay between chronic physical and psychological health being a research priority, heath psychology models are used to understand the interface between long-term conditions, their management and psychological well-being. For those with CD, dietary-management, physical and psychological health are essential in adjusting to and managing the condition, making health psychology a strong framework for exploring disordered eating patterns within CD.

Theories combining social, physical and psychological health are recommended for understanding the self management of gastrointestinal conditions (Pojoga & Stanculete, 2014); of these, the Health Belief Model (Rosenstock, Strecher & Becker, 1988) and the Theory of Planned Behaviour (Azjen, 1985) have been applied to dietary-controlled gastrointestinal conditions, such as CD; however, these models do not directly support the understanding of disordered eating attitudes and behaviours. Stice's dual pathway model of disordered eating (Stice, 2002) has been used to explain disordered eating in chronic health conditions; Peterson, Fischer and Young-Hyman (2015) have modified this model, using social, physical and psychological factors to explain disordered eating patterns in individuals with type one diabetes. These theories will be discussed in the following section.

1.5.1. The Health Belief Model

The Health Belief Model (Rosenstock et al., 1988) proposes that behaviour change is based on an analysis of the barriers to and benefits of behaviour change. This model has

been used to explain the management of chronic health conditions (DiMatteo, Haskard & Williams, 2007) and eating disorders (Akey, Rintamaki & Kane, 2013). There are four beliefs that are used to inform the cost-benefit analysis: *Perceived Susceptibility* (what is the likelihood that my behaviour will cause poor health outcomes), *Perceived Severity* (how severe are the consequences of my behaviour), *Perceived Benefit* (changing my behaviour will be good for my health) and *Perceived Barriers* (the GFD is expensive/ eating gluten-free food will cause weight gain). The idea of self-efficacy was later added to the model; this concept explains how competent one feels in engaging in a particular behaviour despite certain barriers (Strecher & Rosenstock, 1997); in CD this may include management of the GFD. Those with a high sense of self-efficacy are more likely to engage in behaviour change, crucial in the management of CD.

The Health Belief Model is flexible, meaning it can be adapted to explain a variety of behaviours. An individual with CD and co-morbid disordered eating must believe they are susceptible to the negative consequences of their disordered eating behaviour before they will engage in more adaptive eating patterns (*perceived susceptibility*; e.g. my eating patterns are harming my CD-management and health); the individual must recognise the seriousness of their disordered eating upon their CD (*perceived severity*; e.g. my eating patterns will result in hospital treatment); the individual must believe in the alternative option to reduce their disordered eating (*perceived benefits*; e.g. if I eat better, I will feel better); and the barriers to adaptive eating must be reduced (*perceived barriers*; e.g. I find it hard to eat healthily because gluten-free food is hard to find).

The Health Belief Model has been reviewed, across a variety of health behaviours (e.g. smoking cessation, breast cancer screening), in four meta-analyses (Carpenter, 2010; Harrison, Mullen & Green, 1992; Janz & Becker, 1984; Zimmerman & Vernberg, 1994). The

most recent (Carpenter, 2010) suggests that *perceived benefits* and *perceived barriers* were the most effective variables in predicting behaviour, however, weak relationships were found between *perceived severity* and *behaviour*. Based on these findings, Carpenter concluded that the current version of the Health Belief Model is no longer applicable to behaviour change in chronic health conditions. The constructs in the Health Belief Model are not valuable as individual constructs but exploring interactions amongst these constructs may explain further variability in health behaviours. In support of these conclusions, Umeh and Jones (2010) explored the interactions amongst the variables of the Health Belief Model in breast cancer screenings, finding that women who did not conduct breast cancer screenings perceived more barriers to this behaviour but only when they perceived the consequences of breast cancer as more severe. Interactions between *perceived benefits* of screening and *perceived susceptibility* to breast cancer were also noted. The findings of Carpenter (2010) and Umeh and Jones (2010) indicate that in its current form, the Health Belief Model is not appropriate for predicting health behaviours and interactions amongst the variables are critical to understanding health behaviours.

For those with CD, factors including the presence of gastrointestinal symptoms and the burden of reading of food labels may play a role in influencing disordered eating attitudes and beliefs in CD (Arigo, Anskis & Smyth, 2012; Zarkadas et al., 2013). The Health Belief Model fails to account for these more habitual health-related behaviours that may become independent of conscious decision-making processes in CD (Janz & Becker, 1984). Additionally, the model assumes that all negative health behaviours stem from psychological processes, when in CD negative health behaviours, such as dietary restriction, may occur because of alternative factors such as the increased cost of gluten-free foods or poor explanation of the GFD from health care professionals (Nelson, Mandozat & McGough,

2007; Ukkola et al., 2012; Whitaker et al., 2009). In addition, this model fails to explain the positive effects of negative behaviours (Stroeb, 2000); this may be particularly important for disordered eating in CD, as dietary restriction may have a positive effect by reducing the risk of gluten consumption.

1.5.2. The Theory of Planned Behaviour

The Theory of Planned Behaviour (Azjen, 1985) has been used to understand behaviours in CD (e.g. Hall et al., 2013; Kothe et al., 2015; Sainsbury & Mullan, 2011; Sainsbury, Mullan & Sharpe, 2013c; Sainsbury, Mullan & Sharpe, 2014), and recognises that attitudes and beliefs do not account for all behaviours. According to the theory, intention to change behaviour is influenced by *Attitudes* (an individual's positive or negative evaluation of self-performance of the behaviour), *Subjective Norms* (an individual's perception about the particular behaviour which is influenced by the judgement of significant others) and *Behavioural Control* (an individual's perceived ease or difficulty in performing the behaviour). This model also includes the concept of self-efficacy; an individual needs to believe they can engage in a specified behaviour before they can adopt that behaviour.

The Theory of Planned Behaviour has been used to explain GFD adherence in CD (Hall et al., 2013; Kothe et al., 2015; Sainsbury & Mullan, 2011; Sainsbury, Mullan & Sharpe, 2013c; Sainsbury, Mullan & Sharpe, 2014). Individuals with CD often report difficulties in managing their GFD, despite having good intentions towards this behaviour (Barratt et al., 2011). However, the application of the theory to CD has had mixed results. Sainsbury and Mullan (2011) found no evidence for the role of intentions in predicting GFD management; however, when including measures of knowledge and symptom severity, intention and *Perceive Behavioural Control* was associated with GFD management (Hall et al., 2013;

Sainsbury et al., 2013c). One interpretation is that the intention-behaviour relationship is poor at predicting GFD management (Sainsbury et al., 2013c). Participants struggling with GFD management had greater psychological distress, which may influence GFD management over and above the factors accounted for by Theory of Planned Behaviour. Alternatively, Kothe et al. (2015) suggests that this may reflect the role of habit in moderating the intention-behaviour relationship; habit is likely to be an important factor in CD as areas of GFD management may have become habitual such as reading food labels and asking questions about cross-contamination.

A systematic review into health behaviours that influence individuals in a positive or negative way (e.g. smoking, weight loss; McEachan, Conner, Taylor & Lawton, 2011) found that the Theory of Planned Behaviour only accounted for 19.3% of variability in health behaviours. The review also found that the theory was less predictive of behaviour when research designs used self-report measures; other methods may find more support for the Theory of Planned Behaviour. Furthermore, the Theory of Planned Behaviour does not account for behaviour that may result from emotional states or negative affect, two factors which are particularly important in relation to disordered eating patterns and the management of CD (Godart et al., 2007; Sainsbury, Mullan & Sharpe, 2014). In addition, the theory cannot account for factors such as varying the salience of behavioural cues, which cause behaviour change without influencing intentions (Marteau, Ogden, Roland, Suhrcke & Kelly, 2011). For example, the Theory of Planned Behaviour cannot account for the association between availability of gluten free foods, improved GFD management and well-being (White, Bannerman & Gillett, 2016).

1.5.3. Dual Pathway Models of Disordered Eating

Stice's (2002) Dual Pathway Model of disordered eating in Western cultures (Stice, 2002; see Figure Two) suggests that disordered eating develops through two pathways: 1) *Dietary Restriction* and 2) *Negative Affect*. The *Dietary Restriction* pathway describes how body dissatisfaction, resulting from the internalisation of society's unattainable thin ideal, results in dietary restriction in an attempt to control weight. Prolonged dietary restraint is thought to trigger overeating as it increases hunger levels and appetitive response to food, in attempt to restore energy levels (Polivy & Herman, 1985). The *Negative Affect* pathway describes how body dissatisfaction leads to difficulties in emotional regulation; disordered eating behaviours are used as a distractor from this emotional arousal.

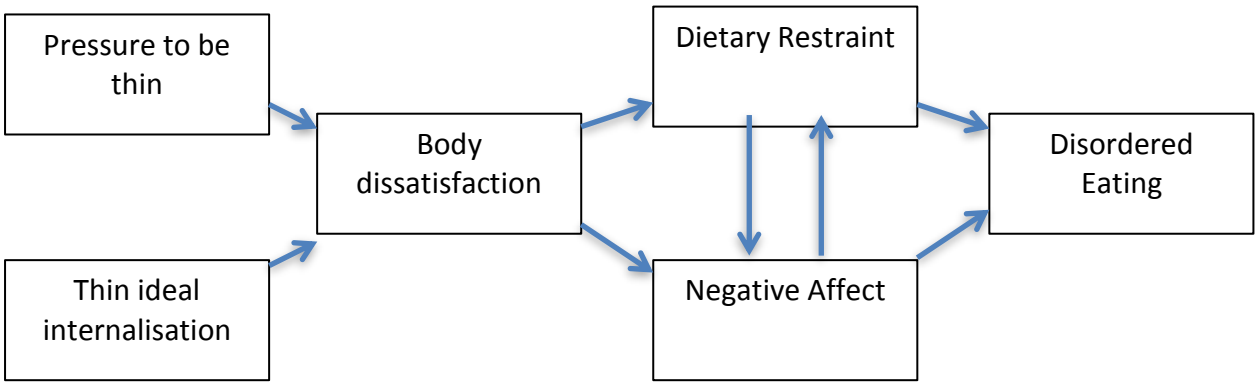

Figure 2: The Dual Pathway Model of Eating Disorders (Stice, 2002)

The Dual Pathway Model of disordered eating has received considerable empirical support (Dakanalis et al., 2014; Urvelyte & Perminas, 2015). In clinical and non-clinical samples, the perceived societal pressure to be thin predicts subsequent body dissatisfaction

and the onset of eating disorders in both developed and developing countries (Allen, Byrne & McLean, 2012; Kroon Van Diest & Perez, 2013; Unikel, Aguilar & Gomez-Peresmitre, 2005). Additionally, emotional regulation difficulties are more common in those with eating disorders and increase with the severity of disordered eating behaviours (Hayes & Napolitano, 2012; Skinner et al., 2012). However, the model fails to describe why some individuals are more susceptible to society's pressure to be thin and there is mixed support for the relationship between dietary restraint and binge eating (Andres & Saldana, 2014); not all binge eating occurs after dietary restraint (Johnson, Pratt & Wardle, 2012). In addition, the model describes relationships between negative affect and disordered eating but not disordered eating and negative affect. This is unusual given the amount of research that indicates a bi-directional relationship between these factors (Pan et al., 2012; Presnell et al., 2009; Skinner et al., 2012).

Although limited in its application to disordered eating in chronic health conditions, the Dual Pathway Model has been modified to account for biological, psychological and social factors that occur in type one diabetes (Peterson, Fischer & Young-Hyman, 2015). This modification has included the addition of biological variables into the model, producing a bio-psychosocial framework (see below); the modified model suggests that factors specific to the diabetes diagnosis may increase the risk of disordered eating. For individuals with CD, a model needs to take into account biological factors such as gastrointestinal symptoms and weight changes that may influence disordered eating attitudes and behaviours.

1.5.4. The Modified Dual Pathway Model

Disordered eating patterns are prevalent in type one diabetes and are associated with poor diabetes-related outcomes and impaired quality of life (Hagger et al., 2016; Nicolucci

et al., 2013). The Modified Dual Pathway Model suggests four factors, in addition to the sociocultural factors described in the original Dual Pathway Model, that contribute to the development of disordered eating patterns in individuals with diabetes (Peterson, Fischer & Young-Hyman, 2015). These factors include diabetes-related hunger dysregulation, weight gain, diabetes-related distress and the need to follow a strict dietary regimen.

Insulin administration is part of the diabetes treatment programme, which is associated with weight gain (Russell-Jones & Khan, 2007). The modified model suggests that this insulin-related weight gain may create an additional vulnerability to body dissatisfaction and subsequent dietary restraint. The second addition proposes that the required dietary regimen may trigger dietary restraint. In addition, the failure to manage this dietary regimen may encourage disordered eating, similar to the breaking of dietary regimens in the general population (Polivy & Herman, 1985).

The Modified Dual Pathway model (Peterson, Fischer & Young-Hyman, 2015) suggests that fluctuations in blood glucose levels result in diabetic-related hunger dysregulation that creates changes in appetite regulation and increases calorie intake (Engstrom et al., 1999; Sabourin & Pursley, 2013; Young-Hyman & Davis, 2010). However, the interactions between diabetic-related hunger dysregulation, insulin and eating patterns have not been empirically supported. The last modification to the model is the addition of diabetes-related distress. Individuals with chronic health conditions, including diabetes, have increased rates of psychological distress (Johnson et al., 2013). This distress can be compounded by the diagnosis of diabetes or diabetes-related weight gain, which leads to disordered eating patterns (Young et al., 2013).

The model for diabetes is in need of assimilation and validation by academic research, as it is not known how much variance in behaviour this model accounts for. Although diabetes-

related weight gain has received considerable support in the development of disordered eating (Pinhas-Hamiel, Hamiel & Levy-Shraga, 2015), Larger's (2005) review indicates that diabetes-related weight change is minimal, with an increase of 2.5kg over 7.5 years. This small weight change may be accounted for by age-related changes in weight, which were not controlled for in this study (Williams & Wood, 2006). Furthermore, depressive symptoms and emotional dysregulation are independently associated with disordered eating (Lavender et al., 2015), so it is unknown how these psychological risk factors act in the context of diabetes. However, this integrated model of disordered eating in diabetes allows the identification of disordered eating in a chronic health group, whilst accounting for factors specific to diabetes, in addition to risk factors found in the general population. The identification of disease specific features that can increase the risk of disordered eating, has the potential to inform clinical interventions, which may significantly improve physical and psychological health outcomes. Furthermore, the Modified Dual Pathway model has the potential to explain disordered eating patterns in a range of chronic health conditions making this a useful tool to aid in our understanding of disordered eating in CD.

1.5.5. Applying the Modified Dual Pathway Model to Coeliac Disease

Disordered eating in those with CD has received little attention. This is unusual given the importance placed on the GFD in the management of CD. The risk factors present in CD, such as managing a strict dietary regimen and disease-related distress, may increase the risk of disordered eating in CD, above and beyond the risk factors present in those without a chronic health condition. Although many of the additional risk factors for disordered eating in CD are similar to those found in diabetes, there are some additional factors that need to be considered.

1.5.6. Weight Gain Secondary to Treatment and Body Dissatisfaction

Individuals diagnosed with the classical presentation of CD, characterised by the presence of gastrointestinal symptoms, tend to be underweight prior to their diagnosis (Olen, Montgomery, Marcus, Ekbom & Ludvigsson, 2009). However, more recently, studies indicate that up to 52% of individuals present as overweight or obese at diagnosis (Kabbani et al., 2012; Olen et al., 2009). Weight increase is likely after commencing the GFD due to changes in diet composition and the increase in nutrient absorption that results from the recovery of the small intestine (Dickey & Kearney, 2006; Kupper, 2005).

The limited research describing weight changes after starting the GFD is contradictory (Dickey & Kearney, 2006; Murray, Watson, Clearman & Mitros, 2004). Dickey and Kearney (2006) described an increase in BMI across 371 adults, with 81% of individuals experiencing weight increase; this occurred across all BMI categories, with 82% of initially overweight participants gaining further weight. However, in a sample of 215, Murray et al. (2004) reported equal numbers of adults gaining and losing weight after commencing the GFD (34% gained and 38% lost weight). In 369 adults with CD, Cheng et al. (2009) found favourable weight changes; individuals, who were underweight or overweight at diagnosis, reached an acceptable BMI after commencing the GFD. Although many participants gained weight on the GFD, this did not result in a change in BMI category.

However, these data failed to take covariates such as GFD management into account. To address this, Kabbani et al. (2012) assessed changes in BMI in 1018 adults with CD, retrospectively assessing Body Mass Index (BMI) at diagnosis from medical records. After initiation of the GFD, the mean BMI of the cohort significantly increased with 21.5%

experiencing clinically significant weight gain (> 7% significant weight gain); this effect was largely seen in those reporting good dietary management whereas those with poor dietary-management had a tendency to lose weight. One interpretation of these findings is that poor management of the GFD may contribute to weight loss whereas strict GFD management may contribute to weight gain; this study highlights the need to take GFD management into account when determining weight change after CD diagnosis. Overall, when controlling for covariates, it appears that weight gain may occur after CD diagnosis and initiation of the GFD. However, other covariates including the perception of this weight change and the emotional impact have not been assessed. These factors may further contribute to weight change after diagnosis and be important in determining how one reacts to these changes in weight.

An increase in weight after initiating the GFD may increase vulnerability for body dissatisfaction and trigger disordered eating attitudes and behaviours. As discussed earlier Leffler et al. (2007) reported the cases of two adult females with CD, who experienced weight gain when starting the GFD and responded by engaging in dietary restriction. Although these females' experienced weight gain after their diagnosis, they did not experience clinically significant weight gain and were still within healthy BMI ranges. The authors argued that the perception of weight gain contributed to the development of their disordered eating behaviours. Factors including the general burden of being diagnosed with a chronic health condition may also explain the post-diagnosis development of disordered eating. Research on the relationship between weight gain and the development of disordered eating has not been conducted in CD but factors related to the diagnosis of CD and living with a chronic health condition need to be considered. These include the role of

distress, the individual perception of weight changes, the restrictive natures of the GFD and the symptoms associated with CD.

1.5.7. The Gluten-Free Diet and Imposed Dietary Restriction

As described in the Dual Pathway Model of disordered eating (Stice, 2002), dietary restraint can lead to the development of disordered eating behaviours such as binge eating, or may encourage excessive food restraint through positive reinforcement (Herman & Polivy, 1984; Stice, 2002). These patterns of behaviour may result in a binge-restrict cycle of eating (Herman & Polivy, 1984).

Despite the increase in the development of gluten-free food products in recent years, in comparison to gluten-containing food, gluten-free food availability is still limited and individuals with CD may feel limited in what they can consume (Estevez, Ayala Vespa & Araya, 2016; White, Bannerman & Gillett, 2016). The need to restrict food intake when gluten-free options are not available may encourage either overeating when gluten-free food becomes available, or conversely, excessive dietary restriction.

At present, the eating patterns of individuals with CD are under-researched; however, increased food consumption may explain the weight changes that have been described in CD (Dicky et al., 2006); as discussed earlier, evidence for a cycle of binge and restrictive eating has been suggested by case reports (Ricca et al., 2000). These eating patterns may develop from the perceived limited availability of gluten-free foods.

1.5.8. A Fear of Gastrointestinal Symptoms

In the majority of individuals, the GFD will encourage intestinal recovery and over time gastrointestinal symptoms will resolve, however, some individuals will continue to

experience gastrointestinal symptoms despite following the GFD (Daum, Cellier & Mulder, 2005). Gastrointestinal symptoms may become associated with certain types of food and could develop into a conditioned food aversion (Garcia, Kimeldorf & Koelling, 1955), which may influence disordered eating attitudes and behaviours (Berstein & Borson, 1986). Food aversions are important for our survival but these can become maladaptive when food aversion occurs in the aetiology of eating disorders (Bernstein & Borson, 1986). Gastrointestinal symptoms have been associated with food aversion in a variety of chronic health conditions including cancer (Coa et al., 2015), autism (Nadon, Feldman, Dunn & Gisel, 2011) and gastroparesis (a condition characterised by delayed gastric emptying; NIDDK Gastroparesis Clinical Research Consortium, 2011). However, the role of gastrointestinal symptoms in CD and the development of disordered eating have received little attention.

The 23 year old woman described by Ricca et al. (2000) managed her GFD extremely well but feared re-experiencing coeliac-related symptoms. She avoided eating in public and reduced the size of her gluten-free meals to prevent weight gain and to cope with her fear of gastrointestinal symptoms. Although this case study is essential in guiding our understanding of gastrointestinal symptoms, disordered eating and CD, there is a need for this to be replicated in larger samples to explore and explain associations between CD, gastrointestinal symptoms and disordered eating. Furthermore, the avoidance of gluten to prevent gastrointestinal symptoms is a sign of essential good dietary-management in CD, so there may be risk of over-pathologising behaviours that are adaptive in CD unless it can demonstrate that this food avoidance results in negative effects on physical and psychological well-being.

1.5.9. Intentional Gluten consumption

Poor management of medical regimens has been identified as a mechanism to induce intentional weight loss in certain groups. For example, the omission of insulin has been described in diabetes (Rodin et al., 2002), the consumption of trigger foods in food allergies such as egg, peanut and lactose intolerance, (Kosky, McCluskey & Lacey, 1993) and poor dietary management in cystic fibrosis (Gilchrist & Lenny, 2008).

For some individuals with CD, the potential to promote intentional weight loss through poor gluten-free dietary-management may be available to those who experience weight loss as a symptom of their untreated CD. Case reports of individuals' intentionally consuming gluten in order to promote weight loss have been described (Leffler et al., 2007; Yucel et al., 2006).

1.5.10. Psychological Distress associated with Chronic Health Conditions

In the general population, psychological distress can contribute to the development of disordered eating attitudes and behaviours (Stice, 2002). Chronic health conditions, including CD, have a high prevalence of comorbid psychological distress, which may contribute to changes in appetite or increase the risk of disordered eating in these groups (Dury, 2015; Turner & Kelly, 2000). This nonspecific burden of chronic health conditions may account for the development of disordered eating attitudes and behaviours in CD. Colton, Rodin, Olmstead and Daneman (1999) suggest that the diagnosis of a chronic health condition may lower the threshold for the onset of disordered eating in those who are susceptible to eating difficulties.

Research consistently reports a high prevalence of psychological distress in CD (Smith & Gerdes, 2011). CD-related distress may further contribute to the development of disordered eating attitudes and behaviours, in addition to the non-specific burden of chronic health

conditions. Psychological distress associated with the diagnosis of CD and the difficulties living with the condition may further exacerbate psychological distress in CD (Rocha, Gandolfi & Santos, 2016). This CD-related distress may further add to the distress associated with managing a chronic health condition, to increase the risk of disordered eating attitudes and behaviours in CD.

1.6. Gaps in the Research and Future Directions

Research addressing CD and disordered eating is limited. Although the research on disordered eating in CD is expanding the focus has been on case studies. Knowledge about the extent of this issue is lacking, and there is a lack of understanding regarding the factors that contribute to disordered eating in CD. Furthermore, the types of disordered eating attitudes and behaviours present in CD are unclear. Greater understanding is needed in order to target interventions for those in need. This thesis will attempt to assess what is already known about disordered eating in CD, in order to develop a new theoretical model of disordered eating in CD using the sociocultural, disease specific and disease general factors that were highlighted in previous models. The second aim of this thesis was to test a number of hypotheses proposed by this model through a series of mixed-research methodologies. The insights from this thesis will be useful for devising new clinical strategies for preventing, assessing and treating disordered eating in CD.

CHAPTER TWO: A MODEL OF DISORDERED EATING IN GASTROINTESTINAL DISEASE

2.0. Chapter Rationale

Chapter One highlighted the potential relationships between disordered eating and a diagnosis of CD. A range of case studies have documented co-morbid disordered eating and CD; however, the construct of disordered eating in CD is not well conceptualised. An underlying theory and/or model is essential for understanding disordered eating in CD as it frames both the methodologies used and the assumptions made.

This chapter will introduce a model to explain the potential relationships between disordered eating and CD and provide a contemporary review of the empirical literature. Due to the limited research exploring disordered eating patterns in CD, this chapter draws upon relevant research from disordered eating practices in dietary-controlled gastrointestinal (GI) conditions. This allowed the development of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.*

Three key questions were examined: a) is disordered eating a feature of GI disorders?; b) what abnormal eating practices are present in those with GI disorders?; and c) what factors are associated with the presence of disordered eating in those with GI disorders? The synthesis of this evidence contributed to the development of a theoretical model of disordered eating development in GI disease, which later chapters will then apply to CD. This chapter provides the basis for the rest of the thesis, as subsequent chapters will test the assumptions held by the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.*

2.1. Introduction

Disruptions to the gastrointestinal (GI) tract result in GI disorders including CD, irritable bowel syndrome and inflammatory bowel disease. The symptoms associated with these disorders include nausea, bloating, constipation, diarrhoea, changes in weight and abdominal pain. CD, irritable bowel syndrome and inflammatory bowel disease can all be managed via a life-long modification of the daily diet to avoid GI symptoms (Gibson & Shepherd, 2010). Dietary plans and foods that trigger symptoms vary across GI conditions. In those with CD, it is necessary to follow a strict, life-long GFD, whereas individuals with irritable bowel syndrome and inflammatory bowel disease have a less structured dietary regimen that involves trial and error to identify trigger foods (NICE, 2015; Yamaoto, Nakahigashi & Saniabadi, 2009).

Dietary-controlled GI disorders may place individuals at risk for the development of disordered eating attitudes and behaviours. Dietary restriction, GI symptoms, food awareness and the non-specific burden of chronic illness may act as triggers for the development of disordered eating attitudes and behaviours in those with CD, irritable bowel syndrome and inflammatory bowel disease.

Numerous case studies have described the co-occurrence of GI disorders and disordered eating attitudes and behaviours (Bayle & Bouvard, 2003; Leffler, Dennis, Edwards-George & Kelly, 2007; Mallett & Murch, 1990; Nied, Gillespie & Riedel, 2011; Oso & Fraser, 2005). However, to our knowledge there has been no systematic review of the prevalence and aetiology of these difficulties in representative samples. The present chapter aimed to answer three questions: a) are disordered eating attitudes and behaviours a feature of GI disorders?; b) what disordered eating attitudes and behaviours are present in those with GI

disorders?; and c) what factors are associated with the presence of disordered eating attitudes and behaviours in those with GI disorders?

2.2. Methods

2.2.1. Search Strategy

Articles were obtained from the two databases that form Web of Knowledge: Web of Science with Conference Proceedings (1900-2014) and MEDLINE (1950-2014), as well as Pubmed, PsychINFO (1967-2014) and Google Scholar. The search criteria were formed of two categories: (i) GI disorder and (ii) terms relating to disordered eating attitudes and behaviours (see Appendix A for search terms used). Retrieved articles were scrutinised for relevant citations.

2.2.2. Eligibility Criteria

To be included in the review, the articles had to meet stringent criteria. Only studies published during or after 1990 were included as this was a period of change for the diagnosis of GI conditions (ESPGHAN, 1990). In addition, articles had to be written in the English language and include participants between 10-80 years with a physician-validated diagnosis of CD, irritable bowel syndrome and inflammatory bowel disease. Those articles that had not been peer reviewed were excluded, as well as case studies and case series. For a summary of the selection process refer to Figure One.

Participants: Studies included youths and working-age adults (10-80 years) with a physician provided diagnosis of CD, irritable bowel syndrome and inflammatory bowel disease. Those reports focusing on other GI food-related allergies were excluded. Any articles looking at the presence of GI disorders in populations already diagnosed with an

eating disorder were excluded. The relationship between eating disorder onset and subsequent GI symptoms has been well documented (Abraham & Kellow, 2013; Peat et al., 2013; Perkins et al., 2005); this review concerns the presence of disordered eating attitudes and behaviours in those with diagnosed GI conditions.

Outcome Measures: The articles included in the review were related to the eating patterns of those with irritable bowel syndrome and inflammatory bowel disease or CD. Studies were required to measure food intake or eating patterns as well as any presence of disordered eating attitudes and behaviours.

Study Design: Studies of both a qualitative and quantitative nature were included in the review. However, those that had not undergone the peer review process were excluded. Case studies and case series were excluded from the review.

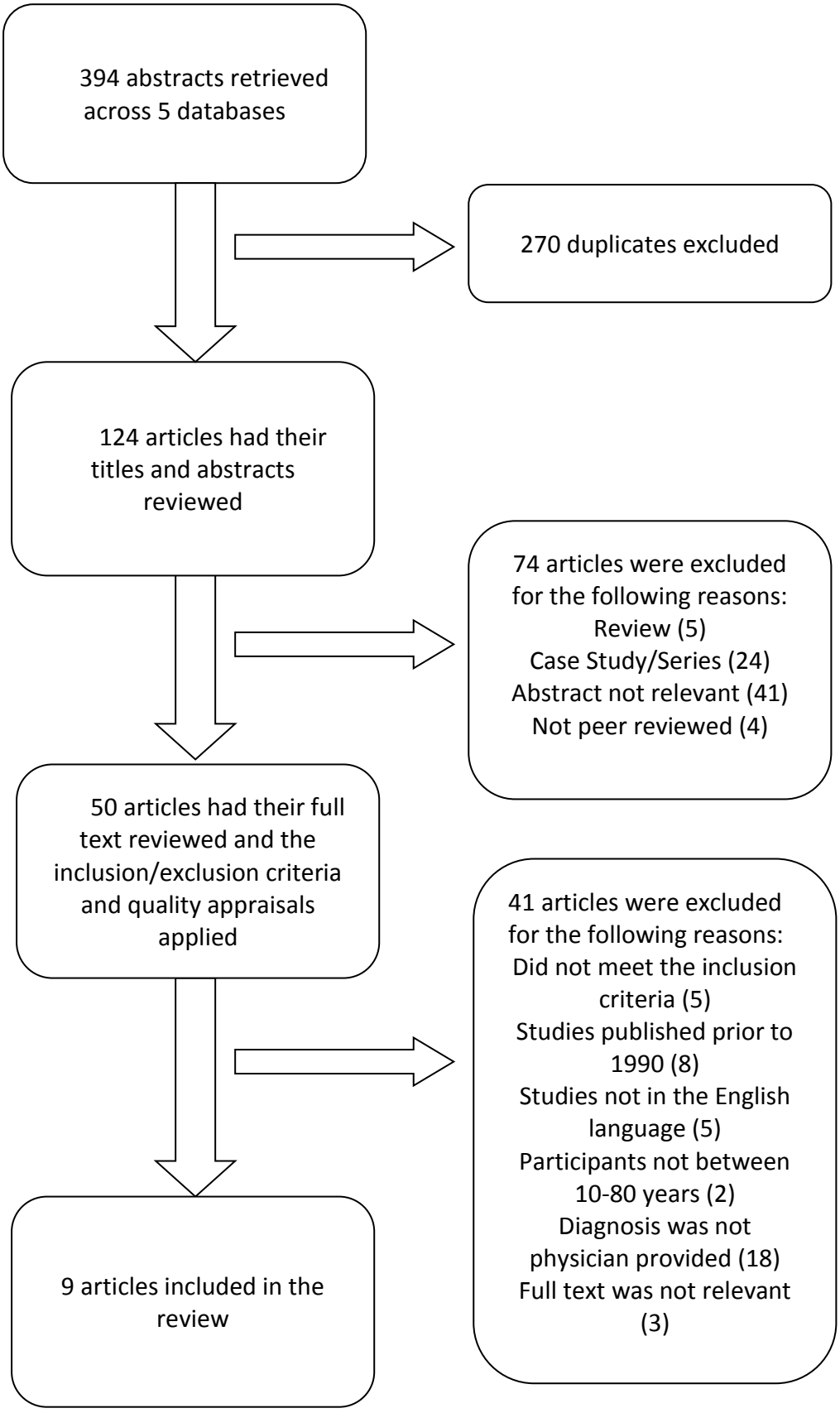

Figure 1. Overview of search strategy

2.2.3. Quality Assessment

Each article underwent an assessment of quality using an established tool (Kmet, Lee & Cook, 2004). Studies were independently rated by two researchers on a 3-point scale, according to established criteria, with a score of 2 (yes) indicating strong evidence for the criteria and a score of 0 (no) indicating a lack of evidence. If some evidence for the criterion was present, a score of 1 was allocated (partial). The criteria were not always applicable (NA) and these criteria were removed from the calculations. A total score was calculated ((number of yes's x 2) + partials) and this was divided by a total possible sum (28-(number of NA's x 2)). This provided a total quality score ranging between 0 and 1. Scores closer to 1 were suggestive of better quality. Difference in ratings between the reviewers was minor and resolved through consensus. The mean quality score across the papers ranged between 0.62 and 1 (see Table One).

2.2.4. Extraction of Data

2.2.5. Participant Characteristics

Sample size, GI diagnosis, age and exclusion criteria were extracted.

2.2.6. Intervention/Study

The research topics that were examined (e.g. Prevalence of disordered eating in GI disease) and the experimental procedure were extracted. Information concerning the method of eating behaviour or dietary assessment was also recorded.

2.2.7. Comparator/Control Group

The presence and characteristics of the control groups were noted.

2.2.8. Outcome Measure

We extracted the percentage prevalence of disordered eating attitudes and behaviours evident in the samples, as well as the types of disordered eating attitudes and behaviours (bingeing, dietary restraint, vomiting) and any factors that were associated with or predicted disordered eating attitudes and behaviours.

2.2.9. Study Design

The study design was noted, whether it was within-subjects or between-subjects and whether it was a qualitative or quantitative investigation.

2.3. Results

This section will contain a brief overview of the selected studies. After applying the critical appraisal criteria, 9 articles were available for review. These articles used mixed methods. The data from these articles are presented in Table One.

Table 1

Summary of studies includes in the literature review; irritable bowel syndrome (IBS), inflammatory bowel disease (IBD).

Author	GI Disorder	Experimental Group	Control Group	Exclusion Criteria	Study Type	Eating Behaviour Measure	Other Measures	Disordered Eating Prevalence	Disordered Eating Type	Correlates of Disordered Eating	Quality Score (0-1)
Arigo, Anskis & Smyth (2012) USA	CD	177 females over 18 years (M=39.2 years)	NA	Under 18 years, 23 removed due to insufficient data	Correlational design	Eating Disorders Examination (Fairburn & Beglin, 1994)	Dietary Compliance Scale (Casellas, Vivancos & Maladelada, 2009), Celiac Disease Symptom Questionnaire (Hauser et al., 2007), Short-Form Health Survey (Ware & Sherborne, 1992), Perceived Stress Scale (Cohen, Kamarck & Mermelstein, 1983), Centre for Disease Studies Depression Scale (Radloff, 1977)	22%	Restraint, Eating Concern, Shape Concern and Weight Concern	Illness symptoms, gluten-free diet compliance, depression	0.91

Karwautz, Wagner, Berger, SInreich, Grylli & Huber (2008) Austria	CD	283 adolescents (10-20 years; M=14.8 years).	Adolescents with Type I Diabetes	NA	Two stage design: between participants and qualitative interviews	Eating Disorder Inventory (Rathner & Waldherr, 1997), Eating Disorder Examination Questionnaire (Hilbert et al., 2007), Eating Disorder Examination (Hilbert, Tuschen-Caffier & Ohms, 2004)	IgA Anti-Endomysial and IgA Tranglutimase antibodies	29.3%	4.8% lifetime history of eating disorder, 3.9% current eating disorder, 10.2% lifetime history of subclinical eating disorder, 10.7% current subclinical eating disorder.	Poor compliance with gluten-free diet	1
Addolorato, Capristo, Stefanini & Gabarrini (1997) Italy	IBD	79 patients with IBD (M=35 years)	36 healthy controls (M=36 years)	Those receiving steroid therapy or having had previous surgery	Between subjects design	BMI, 7 day food diary	Physical Morbidity Index (Andrews, Barczack & Allan, 1987), STAI (Grillion, Ameli, Footh & Davis, 1993), Zung Self-Rating Depression scale (Zung, Richards & Short, 1965),	37.2% of Crohn's Disease, 44.4% of Ulcerative Colitis	Malnutrition	Anxiety, depression	1

Guthrie, Creed & Whorwell (1990) United Kingdom	IBS/ IBD	152 female outpatients with IBS (M=39 years).	34 with IBD and 37 with peptic ulcer	NA	Between subjects design	Eating Attitudes Test (EAT; Garner, Olmstead, Bohr & Garfinkel, 1982)	Psychiatric Assessment Schedule (Dean, Surtees & Sashidharan, 1983)	5.3%	Preoccupation with desire to be thinner, food controlling life, engaging in dieting behaviour and too much time and consideration to food.	NA	0.77
Fletcher, Jamieson, Schneider & Harry (2008) Canada	IBS/ IBD	8 females (18-23 years), 5 with IBS and 3 IBD	NA	NA	Qualitative interviews	14-day food diary	Background questionnaire, semi-structured interview	NA	NA	Lack of compliance with medical regimen	0.85
Sullivan, Blewett, Jenkins & Allison (1997) United Kingdom	IBS	48 patients with IBS, 31 with IBD	28 healthy controls	NA	Between subjects design	Eating Attitudes Test (Garner, 1982)		NA	Dieting, bulimia, food preoccupation, oral control	NA	0.62

Tang, Toner, Stuckless, Dion, Kaplan & Ali (1997) Canada	IBS	43 female and 17 male IBS patients (M=36.8 years).	Predetermined normative sample: 271 healthy controls (M=20.3 years)	NA	Between subjects design	Eating Disorder Inventory (Garner & Olmstead, 1984)	Daily GI symptom diary (Neff & Blanchard, 1987)	NA	Bulimic thoughts	Female, vomiting symptoms	0.91
Okami et al., (2011) Japan	IBS	626 students, IBS symptoms aged 18-29 years	NA 1140 healthy controls without IBS symptoms	Over 30 years, previous diagnosis of IBD	Between subjects design	Non-Validated questionnaire	Rome II (Thompson et al., 1999), HADS (Hatta et al., 1998), METS (Ministry of Health, Japan, 2006)	NA	Irregular meals and meal skipping	Anxiety, depression	0.91

Sainsbury, Mullan & Sharpe (2013b) Australia	CD	390 members of a coeliac society (M=44.2 years)	NA	NA	Correlational design	Eating Disorder Inventory (Garner, 2004)	WHO QoL measure (Murphy et al., 2000), Depression Anxiety Scale (Lovibond & Lovibond, 1995), the Coping Inventory for Stressful Situations, Coeliac Dietary Adherence Scale (Leffler et al., 2009), Perceived Behavioural Control Scale (Sainsbury & Mullan, 2011)	NA	Restraint, Eating Concern, Shape Concern and Weight Concern	QoL, severe GI symptoms at diagnosis, depression, anxiety, stress, emotion-focussed coping	1

2.3.1. Participant Characteristics

Only three of the investigations excluded male participants (Arigo et al., 2012; Fletcher et al., 2008; Guthrie et al., 1990). The remaining six papers had a majority of female participants. The average age of participants across the studies was 29.9 years (10-80 years). Surprisingly, there was a lack of information concerning body mass index across the papers.

2.3.2. Comparator/Control Groups

Five of the studies used control groups (Addolorato et al., 2012; Guthrie et al., 1990; Okami et al., 2011; Sullivan et al., 1997; Tang et al. 1997). Information about the participant characteristics was lacking in two of the papers (Okami et al., 2011; Sullivan et al., 1997).

2.3.3. Outcome Measure

Disordered eating attitudes and behaviours were assessed in all of the papers; however, only four of the studies provided information concerning the prevalence of disordered eating attitudes and behaviours across the samples (Addolorato et al., 1997; Arigo et al., 2012; Guthrie et al., 1990; Karwautz et al., 2008). Seven papers provided information concerning the correlates of DE.

A range of variables were measured but there was no common assessment of eating patterns. Two of the articles used the Eating Disorder Examination Questionnaire (EDE-Q; Hilbert et al., 2007), two used the Eating Attitudes Test (EAT; Garner, Olmstead, Bohr & Garfinkel, 1982) and three used the Eating Disorder Inventory (EDI; Garner, 2004). Other measures of disordered eating and body image were related to general psychosocial well-being questionnaires.

2.3.4. Study Design

The majority of studies used a cross-sectional design, except Fletcher et al. (2008) who used a qualitative design and Karwautz et al. (2008) who used a two-stage qualitative design.

2.4. Synthesis of Results

We discuss the studies in three categories according to the aims of the review: those looking at the prevalence of eating pathology in GI disease, those reporting the types of disordered eating displayed and those that examined the correlates of disordered eating attitudes and behaviours in those with GI disease.

2.4.1. Studies Concerning the Prevalence of Disordered Eating in GI Disorders

Four of the articles reported the prevalence of patterns suggestive of disordered eating, although this was assessed using differing methods (Addolorato et al., 1997; Arigo et al., 2012; Guthrie et al., 1990; Karwautz et al., 2008). Across these four papers there were a total of 691 participants with GI disease. Of these, 23.43% displayed eating patterns that were suggestive of disordered eating attitudes and behaviours. Across these papers, disordered eating attitudes and behaviours ranged between 5.3-44.4% in those with GI disease.

Prevalence rates for the Eating Disorders Examination (Fairburn & Beglin, 1994) ranged between 22-29.3% (Arigo et al., 2012; Karwautz at al., 2008). These scores are in excess of the scores reported for the general population (10%; Solmi, Hatch, Hotopf, Treasure & Micali, 2014). However, those papers using the EAT (Garner, Olmstead, Bohr & Garfinkel, 1982) reported lower prevalence rates (Guthrie et al., 1990; Sullivan et al., 1997). Only one of the papers reported lower prevalence of disordered eating in participants with GI disease

than healthy controls (Sullivan et al., 1997). Unfortunately, Sullivan et al. (1997) reported only the means for the EAT scores and did not report what percentage scored above the cut-off criteria. However, they acknowledged that a subgroup of their participants with irritable bowel syndrome may have engaged in disordered eating practices.

Studies that assessed eating patterns via a food diary reported that participants with GI disease had lower intake than healthy controls (Addolorato et al., 1997; Fletcher et al., 2008). Addolorato et al. (1997) found that individuals with inflammatory bowel disease had a daily calorie intake that was significantly lower than that of controls. Furthermore, 37.2% of those with crohn's disease and 44.4% of those with ulcerative colitis showed evidence of malnutrition, indicating that these individuals are not meeting their daily dietary needs. Although disordered eating attitudes and behaviours were not assessed, a lack of food intake was observed in this group, the cause of which remains unclear.

When combined, the evidence indicates that the presence of disordered eating attitudes and behaviours may be greater in those with GI disease than the reported norms for healthy controls. The conflicting results may be accounted for by the differing use of screening tools as well as factors such as the duration of diagnosis and the type of medical support received.

2.4.2. Studies Concerning the Types of Disordered Eating

Eight of the articles made some reference to the type of disordered eating that was presented by participants (n=2988). This largely depended on the method used to assess disordered eating attitudes and behaviours. However, the majority of articles described disordered eating attitudes and behaviours as a whole, rather than breaking it into subtypes.

Dietary restriction was commonly referred to throughout the articles (Addolorato et al., 1997; Fletcher et al., 2008; Okami et al., 2011). Individuals with GI disease ate more irregular meals and skipped meals more frequently than control participants (Okami et al., 2011). Although consumption of less food was observed in those with GI disorders, it is not clear why this was the case and if intentional dietary restriction was the cause. Fletcher et al. (2008) found that individuals reported using dietary restriction as a way to cope with their GI symptoms, often avoiding food when engaging in social activities. Participants said that they would not eat during the day but would eat normally when in the home during the evening, resulting in an abnormal pattern of food intake. In contrast, Tang et al.'s (1997) findings are suggestive of a purging eating pathology. Tang et al. (1997) found that those irritable bowel syndrome patients who reported greater vomiting symptoms were more likely to endorse the beliefs of the *Bulimia* subscale of the EDI. These individuals had thoughts of vomiting as a means of weight reduction but did not necessarily engage in these behaviours. Tang et al. (1997) suggest that those irritable bowel syndrome patients with severe vomiting and high scores on the *Bulimia* subscale (EDI) may have a characteristic in common with people with eating disorders, i.e. the desire to lose weight.

Kauwautz et al.'s (2008) findings may shed light on the types of disordered eating attitudes and behaviours present in those with GI disease. When looking at the weight loss mechanisms used by these participants, Kauwautz et al. (2008) found that 58.1% used dieting behaviours, 12.9% used excessive exercising, 19.4% used vomiting and 3.2% used laxatives. This suggests that a range of disordered eating behaviours across the clinical spectrum were present, with a majority choosing to restrict their food intake.

2.4.3. Studies Concerning the Correlates and Co-morbidities of Disordered Eating

Seven of the articles made reference to factors associated with higher disordered eating scores. Of particular interest is the reoccurrence of psychological distress, symptom severity and dietary management alongside higher disordered eating attitudes and behaviours scores.

Out of the nine articles reviewed, six reported a relationship between disordered eating attitudes and behaviours and psychological distress (Arigo et al., 2012; Addolorato et al., 1997; Fletcher et al., 2008; Guthrie et al., 1990; Okami et al., 2011; Sainsbury et al., 2013b). Eating disorder risk was associated with a reduced quality of life, maladaptive coping mechanisms, depression and perceived stress (Addolorato et al., 2012; Sainsbury et al., 2013b). Furthermore, greater anxiety and depressive symptomatology was found in those presenting with eating disturbances (Addolorato et al., 1997; Guthrie et al., 1990; Okami et al., 2011). Addolorato et al. (1997) explain that the reason for undernourishment in this patient group is not clear but suggest that it might result from a fear of GI symptoms when consuming food. Fletcher et al.'s (2008) findings suggest that this may be due to anxiety in unfamiliar settings, as participants would restrict their intake in unfamiliar settings due to fears of cross-contamination. Anxiety and depression both seem to be key factors in the development of disordered eating in those with GI disorders.

Symptom severity was referred to across the papers (Arigo et al., 2012; Sainsbury et al., 2013b; Tang et al., 1997). It is not clear at what point symptom severity is most important, with some reports suggesting that symptom severity prior to diagnosis may lead to the development of DE patterns (Sainsbury et al., 2013b), and others suggesting it is the frequency of symptoms during the course of the disease (Tang et al., 1997). More bulimic-type thoughts were reported in those who experienced more extreme vomiting symptoms,

however, this does not necessarily translate into behaviour; these individuals acknowledged the use of vomiting as a weight loss strategy but did not necessarily engage in this behaviour (Tang et al., 1997). Arigo et al. (2012) reported that symptom severity was not associated with disordered eating attitudes and behaviours. The role that symptom severity plays is not clear and it may only play a role in the development of disordered eating in a subset of those with GI disease.

Adherence to dietary regimens shows evidence of being related to disordered eating attitudes and behaviours, particularly in those with CD. Arigo et al. (2012) found that management of the prescribed diet was associated with a decreased range of psychological stresses, but was also linked to greater disordered eating attitudes and behaviours. This indicates that those who monitor their food intake more closely, to follow their prescribed dietary regime, may be at risk of disordered eating attitudes and behaviours. Karwuatz et al. (2008) reported that those with eating pathology also had significantly higher gluten antibody markers, suggesting poorer dietary self-management. Those with eating pathology also had a higher BMI and 85.7% reported the pathology as appearing after the onset of their CD.

2.5. Discussion

This review points towards some important factors that need to be considered in the management of patients with GI disorders. There is an indication that individuals with GI disorders may be more at risk of developing disordered eating attitudes and behaviours than the general population.

One aim of the review was to examine the prevalence of disordered eating attitudes and behaviours in those with GI disease. Disordered eating attitudes and behaviours are present

in a subset of those with GI disorders and the prevalence exceeds the rates found in the general population. The prevalence rates identified in this review (5.3-44.4%) are similar to those found in other dietary controlled chronic health conditions (Markowitz et al., 2010; Shearer & Bryon, 2004). Quick, McWilliams & Byrd-Bredbenner (2012) found that those with dietary-controlled health conditions were twice as likely to have been diagnosed with an eating disorder compared to controls. The constant need to monitor food intake may place these individuals, and those with GI disorders, at risk for disordered eating attitudes and behaviours (Grilo, 2006; Schlundt, Rowe, Pichert & Plant, 1999). However, it is not clear whether the GI disorder is contributing any additional risk factors towards the development of disordered eating attitudes and behaviours, above and beyond that of other dietary controlled chronic health conditions.

The types of disordered eating that were present in those with GI disease were also examined. The majority of papers presented in the review indicated that a restrictive eating pathology was most common. Although there was evidence for bulimic patterns of behaviour as well as excessive exercising, dietary restriction was more frequently reported. It is not clear why these behaviours are more common and if this finding will be replicated in larger samples. However, it may be that those with GI disorders are more likely to fit the psychological profile of someone with a restrictive eating disturbance. However, the majority of investigations simply examined eating disorder risk. This assesses the presence of dietary restriction, bingeing and purging behaviours. Therefore, it is difficult to get a clear picture of what types of disordered eating attitudes and behaviours are most prevalent in those with GI disorders. In addition, an extensive range of eating patterns such as emotional eating, over eating and nocturnal eating patterns have not been examined. This should be addressed in future research because the ranges of disordered eating attitudes and

behaviours are associated with distinct psychological profiles (Cassin & von Ranson, 2005). Moreover, the majority of investigations did not assess the presence of subclinical eating pathology. Future studies should also consider the role of subclinical eating symptoms in GI disorders; due to their risk of malnutrition, any deviation from traditional eating patterns may have a significant impact in this subset of the population.

Another aim of this review was to examine the correlates of disordered eating attitudes and behaviours in GI disease. Psychological distress, symptom severity and dietary adherence were found to be associated with the presence of DE patterns. Anxiety, depression and impaired quality of life were reported in those with disordered eating attitudes and behaviours across the majority of papers. This is not surprising because psychological distress is frequently associated with altered eating patterns in those both with (Colton, Olmsted, Daneman & Rodin, 2013) and without chronic disease (Patrick, Stahl & Sundaram, 2011; Santos, Richards & Bleckley, 2007). However, the specific role that psychological distress plays is not clear. Distress may be both a cause and a consequence of DE behaviours. However, Arigo et al. (2012) suggested that anxiety might be playing a unique role in those with GI disease. According to Arigo and colleagues the fear and anxiety surrounding GI symptoms may lead to disordered eating attitudes and behaviours of a restrictive nature. Individuals with GI disease may be so anxious and fearful of the GI symptoms that have been associated with food consumption in their past, that their fear and anxiety results in an aversion to unfamiliar food types and subsequent dietary restriction.

GI symptom severity may also play an important role in the development of disordered eating attitudes and behaviours. The role that GI symptoms play in the development of disordered eating attitudes and behaviours appears rather complex. Some authors report

that greater symptoms prior to diagnosis increases disordered eating risk (Sainsbury et al., 2013b), whereas others report that greater symptoms throughout their diagnosis led to greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et al., 2008) and good dietary management (Arigo et al., 2012) have been associated with disordered eating attitudes and behaviours. It is possible that there are at least two pathways that lead to increased risk of disordered eating attitudes and behaviours in patients with GI symptoms. On the one hand, individuals who do not follow their dietary regimen experience GI symptoms throughout their diagnosis. These individuals may not be concerned about their diet, and choose to consume their trigger foods for a variety of reasons. This group could be using their trigger foods to promote weight loss. These findings are in line with case studies of individuals with CD, irritable bowel syndrome and inflammatory bowel disease where deliberate consumption of trigger foods has been reported in order to aid weight loss (Leffler et al., 2007; Mallett & Murch, 1990). In those with good dietary management, their GI symptoms may be playing a unique role in the development of disordered eating patterns. Hypothetically, the presence of GI symptoms may create a food aversion in these individuals, causing alterations to their eating patterns (Garcia et al., 1955). These individuals may be extremely anxious and concerned with the preparation and potential cross-contamination of their food products. Concerns around cross-contamination and anxiety around unfamiliar foods is frequently found across the GI disorders (Schneider & Fletcher, 2008; Sverker, Hensing, & Hallet 2005). Although high concern around unknown food items may be advantageous in some situations, this may also feed into the development of disordered eating patterns. A hypothetical framework based on these two pathways has been developed.

2.5.1. A Hypothetical Framework

Based upon the literature presented in the review, a theoretical model of disordered eating attitudes and behaviours patterns in GI disease has been developed (Figure Two). The model depicts the theoretical relationship between a collection of GI disorders and disordered eating attitudes and behaviours; however, it is likely that each GI disorder will have a more specific relationship with eating behaviour but to develop specific models more focussed research is required. In this context, disordered eating is defined as an eating pattern that does not fluctuate beyond the point of nutrient deficient or excess weight change. Although thoughts around planning and preparing food may be present, they should not contaminate thoughts or dictate behaviours above and beyond that of other daily activates (Freeland-Graces & Nitzke, 2013). In addition, the diagnosis of an eating disorder also falls under the criteria for disordered eating.

The model begins at diagnosis, as diagnosis is associated with an imposed change in eating patterns in order to manage the GFD, an increase in psychological distress and weight changes. Receiving a diagnosis and adapting to the new condition brings about risk factors that may contribute to the development of disordered eating attitudes and behaviours. When diagnosed with a chronic health condition, depending on the individual's circumstances, some will adapt well and accept the condition but for others denial may play a role (Alvani, Parvin, Seyed & Alvani, 2012). Coping with any form of chronic illness creates both physical and psychological challenges (Turkel & Pao, 2007). This can contribute to psychological distress, coping problems and a lack of compliance to a medical regime (Seiffge-Krenke & Skaletz, 2006; Suris, Michaud & Viner, 2004).

Pathway one describes the potential development of disordered eating attitudes and behaviours for those who have adapted well to their condition. These individuals may have

greater GI symptoms at diagnosis; the implementation of their treatment and prescribed dietary regimens is effective in resolving these GI symptoms. Sainsbury et al. (2013b) found greater symptoms at diagnosis to be important in the development of disordered eating attitudes and behaviours. These individuals may be anxious about experiencing these GI symptoms again. They may overestimate the negative consequences of their condition and develop the belief that all foods have cross-contamination potential. As a result, these individuals follow their dietary regimens extremely well, like those described by Arigo et al. (2012). Due to their strict dietary self-management, uncertainty surrounding the content of food may be intolerable for this group. High concerns and anxiety around the preparation and cross-contamination of food dominate their thoughts and behaviours; this may result in the consumption of a limited range of foods or eating only in well-known environments. This is similar to the experiences Fletcher et al. (2008) described in those with irritable bowel syndrome and inflammatory bowel disease. These individuals may restrict their food intake during the day, in order to cope with their anxiety around cross-contamination and food preparation issues and subsequently there is the potential for an excessive amount of food to be consumed in the evening when in the home. These individuals display the dietary restriction that was found throughout the papers and this is associated with their anxiety surrounding GI symptoms that was described by Arigo et al. (2012) and reported under nutrition in these groups.

Individuals in pathway two do not adapt well to their diagnosis and experience distress. When starting their treatment and prescribed dietary regimens, these individuals may react with fear when their weight is restored to a healthy level after diagnosis. This group may believe that their dietary regimen is causing them to gain weight, which leads to dysfunctional illness beliefs and behaviours regarding their dietary regimen. Poor dietary

management may follow and the consumption of trigger foods may be motivated by the belief that this can aid with weight loss. These beliefs may lead to a lack of adherence to the prescribed dietary regimen, continued GI symptoms and psychological distress (Lohiniemi, Maki, Kaukinen, Laippala & Collin, 2000; Roth & Ohlsson, 2013). This explains the poor dietary management and increased symptom severity throughout diagnosis, described by Arigo et al. (2012), Tang et al. (1997), Fletcher et al. (2008) and Kawautz et al. (2008). Individuals in pathway two may be at risk for a clinically significant eating disorder. However, poor management of the GFD may be related to factors independent of disordered eating, including a lack of knowledge around the GFD and poor gluten-free food availability. Poor dietary management can be considered a type of disordered eating when gluten ingestion is combined with the belief that this will lead to weight loss. In addition, individuals with CD who skip meals or are cautious around food may indicate disordered eating attitudes or behaviours; alternatively, these individuals may be using these strategies to manage their GFD. These eating attitudes and behaviours, and the motivation behind these behaviours, are poorly understood in CD and further research is needed

Figure 2. Hypothetical framework between GI disorders and disordered eating

2.5.2. Strengths and Limitations of Review

The prevalence of GI disorders is increasing rapidly and this is expected to increase as diagnostic measures improve (Lohi et al., 2007; Molodecky et al., 2012; West, Fleming, Tata, Card & Crooks, 2014). We believe our review brings together an important area of research for the first time. We outline gaps in the current literature and pose a number of important research questions that will need answering in the future. This review also highlights several limitations that need to be addressed in order to develop research into disordered eating attitudes and behaviours in GI disease. The development of a model of disordered eating in GI disease is of use clinically and provides a guide for future research. However, there is a need to explore the underlying causes of disordered eating attitudes and behaviours in GI disease and explore the functions that these eating patterns may have for this group. In addition, the studies described in the review failed to report long-term outcomes. It is essential for future research to prioritise the long-term effects of disordered eating attitudes and behaviours in GI disease.

Only nine articles were included in this review, which highlights the need for research in this population. Despite this limitation, these nine articles had strong quality scores and eight of these articles suggested that disordered eating attitudes and behaviours were occurring in participants with GI disease, suggesting that the findings are reliable. Due to improved diagnostic measures and better access to services, GI diagnosis is increasing rapidly (Lohi et al., 2007; Molodecky et al., 2012; West, Fleming, Tata, Card & Crooks, 2014; WGO, 2009). As more of the population is diagnosed with GI disease, there becomes a need to explore and highlight the

psychosocial and physical consequences of GI disease. This includes disordered eating attitudes and behaviours. An increased awareness of this phenomenon should improve awareness amongst healthcare professionals and ultimately can lead to early detection or prevention of the problem in those with GI disease.

Unfortunately, the results could not be combined in a meta-analysis due to the differing methodologies, outcomes and populations. The development of the hypothetical model of disordered eating attitudes and behaviours in GI disease provides a framework to guide future research. There is a need for studies to document the levels of adherence and anxiety around food in those with GI disease. In addition, the function that these eating patterns may have, should be addressed from the patient perspective.

2.5.3. Pathologising Behaviours that Work?

It is important to note that the majority of individuals with GI disease will not go on to develop disordered eating attitudes and behaviours. Nevertheless this review indicates that some individuals with GI disease will eat in a manner that deviates from the cultural norms of three meals a day (Fjellstrom, 2004). Some behaviour that could be considered disordered may actually result from features of the food environment, which make it difficult to stick to a prescribed diet such as gluten-free foods being unavailable. Further research is needed to explore the specific eating patterns associated with GI disease and how these patterns relate to external constraints on the diet.

2.6. Conclusion

The review indicates that those with dietary-controlled GI disorders, including CD, may be at increased risk for disordered eating attitudes and behaviours. This is likely to interact with the presence of GI symptoms and psychological distress. The limited research in this area is concerning as it impacts both the physical and psychological well-being of this group. There is a need to fully examine the prevalence of this phenomenon in the GI population, as well as the interaction between the two disorders.

The development of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* provides new assumptions, which have not been thoroughly tested in individuals with CD. Because of this, this model has the potential to direct new avenues of research and treatment. However, the core assumptions of this model need to be applied and tested in individuals with CD. This includes testing whether:

1. Disordered eating attitudes and behaviours are related to the CD diagnosis (gastrointestinal symptoms, GFD management) and do not just result from the stressors of chronic health conditions (Chapter Three, Chapter Four, Chapter Six).
2. Exploring the role of food concerns in CD (Chapter Four, Chapter Five, Chapter Six, Chapter Seven).

CHAPTER THREE: THE PREVALENCE AND PREDICTORS OF DISORDERED EATING IN WOMEN WITH CD

3.0. Chapter Rationale

Chapter Two highlighted the limitations in research exploring disordered eating attitudes and behaviours in gastrointestinal disease. The majority of studies have failed to establish whether disordered eating in CD results from the CD diagnosis itself or the general stressors associated with a chronic health condition. The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* explains how factors specific to a GI diagnosis may be associated with the development of disordered eating. These include the presence of gastrointestinal symptoms, poor management of dietary regimens and increased concerns around food.

This chapter will use the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* to explore the associates of disordered eating in CD. These factors are important for identifying and supporting those with CD and disordered eating. In addition, this chapter will build on the limitations of previous research that were discussed in Chapter Two by assessing the prevalence of disordered eating in females with CD, compared with other dietary-controlled conditions.

In order to differentiate between the general stressors of chronic health conditions and factors specific to a CD diagnosis, individuals with CD were compared to other chronic health conditions and the correlates of disordered eating (psychological distress, gastrointestinal symptoms, dietary management) was compared across chronic health conditions.

3.1. Introduction

The management of a dietary-controlled health condition, such as CD, creates pressures that may harm one's relationship with food and have been associated with an increased prevalence of disordered eating attitudes and behaviours (Quick, Byrd-Bredbenner & Neumark-Sztainer, 2013). Disordered eating describes a spectrum of eating behaviours, which can range from clinical eating disorders to skipping meals, binge eating, restricting certain food types or fasting (Grilo, 2006).

The risk of developing disordered eating behaviours increases with psychological distress, which frequently occurs in a range of chronic health conditions (Quick, Byrd-Bredbenner & Neumark-Sztainer, 2013). Furthermore there is an increased risk of developing disordered eating in individuals diagnosed with a chronic health condition during puberty, when their body shape is already changing (Smith, Latchford, Hall & Dickson, 2008). These factors are common across all chronic health conditions. For individuals with CD, the need to monitor the gluten content of food, combined with fears about the effectiveness of their GFD and concerns about the prevention of gastrointestinal symptoms, may additionally contribute to increased risk of disordered eating (Arigo, Anskis & Smyth, 2012; Karwautz et al., 2008).

To date, there have been few studies of the prevalence of disordered eating in CD. The results of two cross-sectional surveys suggest that between 22% and 29% of individuals with CD score above the clinical cut-offs on measures assessing Anorexia and Bulimia Nervosa (Arigo, Anskis & Smyth, 2012; Karwautz et al., 2008). Poor dietary management, psychological distress and physical symptoms related to CD were frequent in those with disordered eating attitudes and behaviours (Arigo, Anskis & Smyth, 2012; Karwautz et al., 2008; Wagner et al., 2015), however, the

absence of a control group means that it is impossible to determine if the disordered eating is related to the coeliac diagnosis or if it results from the nonspecific burden of a chronic health condition. These factors are essential to understand the mechanisms behind disordered eating in CD.

Case studies offer an understanding of the complex relationship between disordered eating and CD (Leffler et al., 2007; Ricca et al., 2000; Yucel, Ozbey, Demir, Polat & Yager, 2006). Yucel et al. (2006) suggested that the long-term dietary restriction, necessary in CD, might contribute to disordered eating attitudes and behaviours whereas Leffler et al. (2007) suggested that problems with maintaining the GFD may be associated with disordered eating attitudes and behaviours. However, to fully understand the extent of this problem and to understand the mechanisms behind disordered eating in CD, larger sample sizes are required.

Prior to diagnosis, some individuals with CD experience severe gastrointestinal symptoms, which may contribute to the development of disordered eating attitudes and behaviours (Arigo, Anskis & Smyth, 2012; Chapter Two). Although most individuals will experience clinical remission on the GFD, some will continue to experience gastrointestinal symptoms, which may result from refractory CD where the individual is not responsive to the GFD (Daum, Cellier & Mulder, 2005). Alternatively, Midhagen and Hallert (2003) suggested that the nutritional composition of the GFD might be responsible for persistent gastrointestinal symptoms, whereas Nachman et al (2010) suggested this results from poor dietary management. Untreated gastrointestinal symptoms may trigger an aversion to food, which can influence disordered eating attitudes and behaviours (Berstein & Borson, 1986). Gastrointestinal symptoms have been associated with food aversion in a

variety of chronic health conditions including cancer (Coa et al., 2015), autism (Nadon, Feldman, Dunn & Gisel, 2011) and gastroparesis (a condition characterised by delayed gastric emptying; IDDK Gastroparesis Clinical Research Consortium). However, the role of gastrointestinal symptoms in CD and the development of disordered eating has received little attention.

Gastrointestinal symptoms and dietary management are closely associated via a bidirectional relationship, where good dietary management is associated with fewer and/or less severe gastrointestinal symptoms, and poor dietary management is associated with increased/more severe gastrointestinal symptoms (Murray, Eason, Clearman & Mitros, 2004). In addition, the severity of symptoms can influence management of the GFD. One interpretation of the associations between gastrointestinal symptoms and disordered eating attitudes and behaviours may be explained by the deliberate consumption of gluten in those diagnosed with CD; Leffler et al. (2007) described cases in which individuals would consume gluten in order to encourage gastrointestinal symptoms to promote weight loss. However, this phenomenon has only been described in case studies and it is not clear how these findings will generalise to larger samples. Misuse of dietary regimens has been reported in diabetes (Young-Hyman & Davis, 2010) and there is potential for this to occur in CD. However, not all individuals who display poor dietary self-management will do so because of disordered eating attitudes and behaviours.

The Theoretical Model of Disordered Eating in Gastrointestinal Disease (Chapter Two) suggests that disordered eating differs depending on beliefs about the disease and dietary management. The first pathway describes individuals who experience extreme anxiety around unfamiliar foods and/or overestimate the negative

consequences associated with their condition. These individuals may fear food prepared outside of their control, and cope with this by eating a limited variety of foods. The second pathway describes individuals who experience weight gain after commencing their prescribed dietary regimen and may use techniques to reverse this weight gain. Not all individuals with CD will experience weight gain after commencing the GFD; however, good dietary management has been associated with a post-diagnosis increase in weight (Kabbani et al., 2012). Prior to coeliac diagnosis, individuals may present as underweight, meaning that increased weight is an indicator of recovery of the intestine, however, for some individuals this weight change may be negatively interpreted and trigger disordered eating. These individuals may recognise the association between weight gain and the GFD and aim to reduce their weight gain through poor dietary management (Leffler et al., 2007). This model has the potential to help us to interpret and understand the relationships between disordered eating and CD by testing specific hypotheses.

This study is the first to apply *The Theoretical Model of Disordered Eating in Gastrointestinal Disease* to CD. Given the limitations of prior studies, this study assessed the prevalence, predictors and types of disordered eating in CD compared to other conditions with dietary controlled components. Individuals with CD, who follow a strict GFD, were compared to those with inflammatory bowel disease and type two diabetes (both of which have dietary components to their management) and healthy controls. Dietary management in inflammatory bowel disease and type two diabetes is unlike that for CD as it is less strict and regimented when compared to the GFD and other medical interventions may be required, which is generally not the case in CD. Individuals with inflammatory bowel disease experience

gastrointestinal symptoms associated with the ingestion of certain restricted foods, which can differ between patients, but will avoid these trigger foods during a flare-up and may use medical or surgical approaches to manage flare-ups (NICE, 2015); those with type two diabetes do not have gastrointestinal symptoms as a feature of their diagnosis and do not avoid particular food types, but will follow a balanced diet with an emphasis on consuming high fibre and low-glycaemic index foods. This may be combined with blood glucose monitoring and insulin injections (NICE, 2015). These control groups allowed us to explore the role of nonspecific factors common to *all* dietary-controlled conditions (years with condition, psychological distress), factors common to gastrointestinal disease (gastrointestinal symptoms) and factors *unique* to the CD diagnosis (GFD management). The most common types of disordered eating patterns related to Binge Eating, Anorexia Nervosa and Bulimia Nervosa, were assessed (NHS, 2015).

The following were anticipated: 1) individuals with dietary-controlled conditions (CD, inflammatory bowel disease and type two diabetes) would score greater on disordered eating measures than healthy controls; 2) psychological distress, a nonspecific factor, would be associated with disordered eating across all groups; 3) in those with gastrointestinal disorders (inflammatory bowel disease and CD), factors unique to these conditions (gastrointestinal symptoms) would explain additional variance in disordered eating scores; 4) additional variance in disordered eating would be explained by dietary-management in CD and 5) based on *The Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two), two types of disordered eating were expected to be present in CD.. One group of disordered eaters was expected to show good dietary self-management and few gastrointestinal

symptoms, associated with increased anxiety around new foods. The second group was expected to have poor dietary management and experience increased gastrointestinal symptoms, associated with gluten ingestion.

3.2. Methods

The cross-sectional survey was conducted between June and December 2014. Individuals living in the United Kingdom, aged between 18-69 years and who reported a diagnosis of CD, type two diabetes or inflammatory bowel disease, were eligible to participate. Healthy controls with no reported health conditions or food allergies were also recruited. Participants were excluded if 1) they reported having a dietary-controlled condition other than CD, type two diabetes or inflammatory bowel disease (e.g. cystic fibrosis, type one diabetes) and 2) if they had any other food allergies. Individuals with type two diabetes were required to be following a prescribed dietary regimen as a part of their treatment programme and individuals with CD were required to self-report a biopsy confirmed diagnosis.

Participants with long-term conditions were recruited through adverts on online support forums (e.g. Facebook) and through Coeliac UK, Diabetes UK and Core, the main charities supporting people with CD, type two diabetes and inflammatory bowel disease in the UK. Healthy controls were university psychology students recruited from the School of Psychology Research Participation Scheme at the University of Birmingham. Interested individuals were directed to an online survey to complete the following questionnaires. Men were recruited but only 14 took part, so data not analysed.

3.2.1. Measures

3.2.2. Demographic and General Health Information

For participants with type two diabetes, inflammatory bowel disease and CD, information was gathered on demographics, information relating to diagnosis (method of diagnosis, date of diagnosis, dietary management) and health status (allergies, medication). For individuals with CD, diagnostic method was assessed on a 3 item scale including 1) biopsy provided diagnosis; 2) blood test; 3) I diagnosed myself based on dietary changes, and dietary self-management was rated on a 5-point Likert scale, in response to the question "*In general, how strictly do you maintain a gluten free diet?*" *ranging from '1) All of the time'; 2) 'Most of the time'; 3) 'Some of the time'; 4) 'Now and then'; 5) 'Not at all'* (Ford, Howard & Oyebode, 2012). For those with inflammatory bowel disease and type two diabetes dietary self-management was also rated on a 5-point Likert scale but the item was phrased "*In general, how strictly do you maintain your prescribed dietary-regimen*?"

The presence of gastrointestinal symptoms was assessed using the Illness Perception Questionnaire Revised (IPQ-R; Moss-Morris et al., 2002). Participants are asked to rate whether they have experienced a range of symptoms since their diagnosis (yes/no). For the purpose of this analysis, a total symptom score was calculated by adding up the total of gastrointestinal symptoms (nausea, weight loss, upset stomach, abdominal pain, bloating, excessive wind, constipation, indigestion) experienced in the last four weeks, providing a score between 0 and 8, with 8 indicating a greater number of gastrointestinal symptoms. Non-gastrointestinal symptoms were removed from the analysis, as they were not central to the aims of

this chapter (sore throat, breathlessness, fatigue, stiff joints, sore eyes, wheeziness, headache, sleep difficulties, dizziness, loss of strength, mouth ulcers, hair loss).

3.2.3. Psychological Distress

The Depression, Anxiety, Stress Scale 21 (DASS-21; Lovibond & Lovibond, 1995) assesses levels of depression, anxiety and stress. The items consist of statements referring to the past week, rated on a 4-point scale. To enable comparison with the longer version of the DASS each subscale is multiplied by 2 creating a total score between 0-63 with higher scores indicating greater distress. The DASS-21 has strong psychometric properties (Brown et al., 1997).

3.2.4. Food Anxiety

The Food Neophobia Scale (FNS; Pliner & Hobden, 1992) is a ten-item scale that measures willingness to try new foods. Scores above 35 are considered high, with lower scores indicating greater willingness to try unfamiliar foods (Pliner & Hobden, 1992). The FNS is a 10-item questionnaire rated on a 7-point Likert-scale from disagree strongly to agree strongly, with total scores ranging from 10 to 70. The scale has been validated numerous times and is the standard measure of food neophobia, with good reliability and validity (Miselman, King & Gillette, 2010). At present no appropriate measures of food anxiety have been developed. The FNS was chosen as the best available tool to measure anxiety around new foods.

3.2.5. Disordered Eating

Two questionnaires were used to target the differing attitudes and behaviours surrounding disordered eating, to account for any overlap in disordered eating categories (Eddy et al., 2008; Swanson et al., 2011).

The Eating Attitudes Test (EAT-26; Garner & Garfinkel, 1979) is used to assess eating disorder risk by measuring the attitudes and behaviours suggestive of Anorexia and Bulimia Nervosa. It has been used to identify eating disturbances in non-clinical samples. It is used as a screening tool for eating disorders, but is not a diagnostic tool. The 26 items are scored on a 3-point scale, with a score of 20 or above requiring further evaluation. The tool has strong psychometric properties (Garner et al., 1982) and has been used in populations with dietary-controlled conditions (Guthrie, Creed & Whorwell, 1990). Confirmatory factor analysis found poor support for Garner et al.'s (1982) three-factor model (RCFI=.889, RMSEA=.075), strongest support was found for a one factor model (RCFI=.922, RMSEA=.066). Therefore, total EAT-26 scores were used throughout the analysis and subscales were not explored.

The Binge Eating Scale (BES; Gormally et al., 1982) assesses the behavioural aspects of binge eating and the thoughts and feelings associated with these behaviours. The BES is a screening tool to help identify individuals who may be at risk for binge eating behaviours. Scores on the BES range from 0-46, with scores above 17 indicating moderate binge eating and scores greater than 27 indicating severe binge eating. The BES has been validated in both obese and non-obese population and used in those with gastrointestinal disorders (Duarte, Pinto-Gouveia & Ferreira, 2015; Passananti et al., 2013; Timmerman, 1999).

3.2.6. Ethical Approval

Ethical approval was granted by the Psychology Research Ethics Committee, University of Birmingham.

3.2.7. Statistical Analysis

Data were analysed using the Statistics for the Social Sciences (SPSS) version 22.0. 69 CD participants were excluded across the groups due to the absence of a biopsy-proven diagnosis, 8 additional participants were excluded for not meeting the inclusion criteria. Overall, 77 individuals were removed from the CD group, 27 from type two diabetes and 9 from inflammatory bowel disease and 4 from health controls, providing 503 participants for analysis.

To assess the predictors of disordered eating, regression analyses were conducted to examine the relationships between disease specific factors, disease non-specific factors and disordered eating scores and to compare these amongst the different diagnostic categories. Correlations were run between BES and EAT-26 scores and all other variables to select covariates for the regression models. The covariates and nonspecific predictors were added into stage one of the hierarchical regression, followed by disease specific predictors (dietary management, gastrointestinal symptoms). All variables were centred before being entered into the regression models. Bonferroni corrections were used to control for multiple comparisons and reduce the chance of type one errors (Armstrong, 2014).

The fit of the model across the groups was assessed using three stages: 1) does the predictor set work better for CD than other groups; 2) are the models substitutable and 3) are the regression weights across the groups different. 1)

Fishers Z test was used to compare the R^2 values from each of the groups regression models. A significant p-value (<.05) would indicate a difference in model fit across the groups. 2) Differences in model structure across the diagnostic groups were explored using a cross validation technique (Palmer & O'Connell, 2009). The regression model from each group was applied to every other group (e.g. the CD regression model was applied to all other diagnostic groups) to create both a "direct" and a "crossed" model. The resulting crossed R^2 and direct R^2 were compared using Hotelling's t-test, a significant p-value (<.05) indicates a difference in model structure across the groups, which requires further investigation. 3) To examine the individual predictors within the models, regression weights across the groups were compared.

To investigate the types of eating behaviours, a two-step cluster analysis was performed on the CD sample. Three theoretical groups were hypothesised to come out of the analysis (two disordered and a healthy type), so we specified three groups to emerge from the analysis. Years with diagnosis, psychological distress, disordered eating scores, food neophobia scores, dietary-management and gastrointestinal symptoms were entered into the analysis. Variables with a predictor importance less than 0.2 were subsequently removed from the analysis. The average silhouette measure of cohesion and separation (ranging from -1 to +1) was used to determine the goodness of model fit. A silhouette measure <0.2 is considered poor, between 0.2 and 0.5 is considered a fair solution and >0.5 is considered a good solution (Mooi & Sarstedt, 2011).

3.3. Results

Overall, 72.8% of participants identified as White British, 18.6% as White Other, 2% as Asian, 1% as Black and 2.8% as Mixed Background. Table One displays the mean age, Body Mass Index (BMI) and years since diagnosis across the groups. The type two diabetes group were older and had a higher BMI when compared to other diagnostic groups. There were no other differences between the groups. The BMI, ethnicity and years with diagnosis for each condition were similar to previous samples; however, across all groups our samples were younger than previous reports (Hauser et al., 2010; Koro, Bowlin, Bourgeois & Fedder, 2004; Wada et al., 2015).

68.5% of participants with CD reported that they followed their GFD “all the time”. Of the remaining 31.5%, 9.4% were completely non-adherent and 22.1% were partially adherent to the GFD

Table 1

Demographic Information (Age, Body Mass Index, Years with Condition) Displayed as Means and Standard Deviations. Ethnicity Displayed as Number and Percentage.

	CD (n=157)	**Inflammatory Bowel Disease (n=116)**	**Type Two Diabetes (n=88)**	**Healthy Controls (n=142)**	**Group Differences**
Age (years)	38 (13.4)	36 (12.0)	47 (12.8)	33 (13.7)	T2D > CD, IBD, HC
Body Mass Index	22.9 (3.8)	23.1 (4.9)	29.1 (3.6)	22.4 (4.8)	T2D > CD, IBD, HC
Years since Diagnosis	9 (10.3)	8 (7.6)	9 (7.3)	-	CD= IBD= T2D
Ethnicity (White)	150 (95.5)	108 (93.1)	84 (95.5)	133 (93.0)	CD= IBD= T2D= HC
Ethnicity (Non-White)	7 (4.5)	8 (6.9)	4 (4.5)	10 (7.0)	CD= IBD= T2D= HC

CD: Coeliac Disease; T2D: Type Two Diabetes; IBD: Inflammatory Bowel Disease; HC: Healthy Controls. Standard deviations are displayed in brackets (for ethnicity, percentage is displayed in brackets).

3.3.1. Prevalence of Disordered Eating in CD compared to Controls

Table Two displays the proportion of participants scoring above the clinical cut-off for the EAT-26 and the BES and the mean total scores for each group. The Kruskal Wallis tests found significant differences in mean EAT-26 scores across the diagnostic groups ($H(3)=31.8$, $p<.001$). EAT-26 scores were higher in those with CD than healthy controls ($U=5312.5$, $p=.001$) and those with CD scored higher than those with type two diabetes ($U=2532$, $p=.001$). There was a significant difference in BES scores across the diagnostic groups ($H(3)=82.4$, $p<.001$). Those with CD had higher BES scores than healthy controls ($U=3947$, $p<.001$) but scored lower than those with type two diabetes ($U=2268$, $p=.001$).

Table 2

Mean Scores and Percentage scoring above the clinical cut-offs for measures of disordered eating

Measure	**CD (n=157)**	**Type Two Diabetes (n=88)**	**Inflammatory Bowel Disease (n=116)**	**Healthy Controls (n=142)**	**Group Differences**
Eating Attitudes Test (>20)	11.1 (15.7%)	7.4 (8.8%)	12.8 (20%)	7.7 (3.8%)	CD > T2D, HC; IBD > T2D, HC
Binge Eating Scale (>17)	11.2 (19.4%)	13.6 (25%)	9.9 (22.2%)	3.9 (2.3%)	CD, T2D, IBD > HC

CD: Coeliac Disease; T2D: Type Two Diabetes; IBD: Inflammatory Bowel Disease; HC: Healthy Controls.

The number in brackets represents the percentage of participants scoring above the pre-determined clinical cut-offs for the BES and EAT-26.

EAT-26 and BES scores were compared across all groups ($p<.05$; see group differences column).

3.3.2. Predictors of Disordered Eating

Associations (p<.008) were found for scores on the EAT-26 and BES, and measures of psychological distress, as well as age, BMI, symptoms and GFD management. These factors were added as covariates. Based on the significant relationships with disordered eating and between the subscales, total DASS-21 scores were entered into step one of the regression model. Years with condition, BMI and age were also added. This model accounted for 23.1% of the variance in EAT-26 scores (F=(4, 90)=8.4, p<.001; see Table Three) with distress having a significant positive regression weight.

The disease specific variables were entered in step two (dietary-management and gastrointestinal symptoms). For the CD group, when predicting EAT-26 score, this model accounted for 54.3% of the variance in EAT-26 scores (F=(6, 90)=20.4, p<.001; see Table Three) with dietary-management and gastrointestinal symptoms having significant positive regression weights. Based on the examination of ß weights, dietary-management had the major contribution.

The overall model predicted total EAT-26 score equally well for all of the diagnostic groups. Comparison of the fit of the model across those with type two diabetes (z=2.9,p=.004) and inflammatory bowel disease (z=6.1,p<.001) revealed that there was no significant difference between the respective R^2 values for the EAT-26 score.

When examining the model structure across the groups, structural differences were found. When looking at CD and inflammatory bowel disease, the combined direct R^2 = .60 and crossed R^2 = .40 were significantly different (z=2.9,p=.004). There

are structural differences between the best regression model for predicting EAT-26 score in those with CD and inflammatory bowel disease. When looking at CD and type two diabetes together, the combined direct R^2 = .60 and crossed R^2 = -.43 were significantly different (z=6.1,p<.001), indicating that there are structural differences between the best regression model for predicting EAT-26 score in those with CD and type two diabetes.

Further analysis revealed that dietary self-management (z=3.6, p<.001) and DASS-21 scores (z=-2.8, p=.006) had significantly different regression weights in the CD and inflammatory bowel disease groups, with dietary-management having more influence on EAT-26 scores in those with CD and DASS-21 scores in those with inflammatory bowel disease. Dietary self-management (z=4.6 p<.001) had a significantly different regression weight in the CD and type two diabetes groups, with poor dietary self-management being associated with EAT-26 scores in those with CD. The regression weights for gastrointestinal symptoms were close to significance across CD and type two diabetes (z=1.9, p= .057). The regression models for the comparison groups are provided in Appendix B for comparison but are not central to the aims of this chapter.

Table 3

Disease specific and Non-Specific Factors in Predicting EAT-26 Scores in CD

Predictors	**B**	***B***	R^2	**F**	R^2 **Change**
Model 1) Non-specific Factors					
Age	*-.02*	*-.03*			
Body Mass Index	*-.24*	*-.12*			
Years with Condition	*.01*	*.08*			
DASS-21	*.21*	*.04**	*.26*	*8.36**	*.26**
Model 2) Disease Specific Factors					
Age	*.02*	*.03*			
Body Mass Index	*-.11*	*-.06*			
Years with Condition	*.05*	*.06*			
DASS-21	*.09*	*.22*			
Gastrointestinal Symptoms	*.65*	*.50**			
Dietary-management	*2.52*	*.24**	*.57*	*20.42**	*.31**

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

For the CD group, when predicting BES score, collectively this model (disease non-specific factors) accounted for 41.8% of the variance in BES scores (F=(4,86)=17.5, p<.001; see Table 4) with distress having a significant positive regression weight. The addition of disease-specific factors only explained no additional variance.

The overall model fit for all of the diagnostic groups fit equally well. Comparison of the fit of the disease-nonspecific model across those with type two diabetes (z=-1.3,p=.180) and inflammatory bowel disease (z=0.6,p=.521) revealed no significant

difference between the respective R^2 values for BES scores between inflammatory bowel disease, type two diabetes and CD. These predictors do equally well across the groups. Examination of ß weights found a positive association between depression and BES scores across all of the groups.

Table 4

Disease specific and Non-Specific Factors in Predicting BES Scores in CD

Predictors	**B**	***B***	R^2	**F**	**R^2 Change**
Model 1) Non-specific Factors					
Age	*-.13*	*-.14*			
Body Mass Index	*.71*	*.23*			
Years with Condition	*-.07*	*-.06*			
DASS-21	*.33*	*.51**	*.44*	*17.53**	*.44**
Model 2) Disease Specific Factors					
Age	*-.13*	*-.15*			
Body Mass Index	*.69*	*.22*			
Years with Condition	*-.09*	*-.07*			
DASS-21	*.35*	*.55**			
Gastrointestinal Symptoms	*-.14*	*-.07*			
Dietary-management	*-.34*	*-.02*	*.67*	*11.61**	*.00*

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

3.4.3. Typologies of Eating Attitudes and Behaviour in CD

Three groups emerged from the cluster analysis producing a “fair” model with a silhouette measure of cohesion and separation of 0.5 (Mooi & Sarstedt, 2011). The

first group was the largest (N=60) containing those with low psychological distress, few gastrointestinal symptoms, good dietary-management and low scores on all disordered eating measures. These were determined to be the “low risk” group. The second group contained 25 participants. This group was named the “critical” group. These individuals’ scored high on EAT-26, and reported poor dietary self-management, many gastrointestinal symptoms and moderate stress scores. The “high distress” group included 11 individuals with high BES scores; this group scored highest on all measures of psychological distress but show good dietary-management. The Kruskal Wallis tests found significant differences in all variables across the three groups (see Table Five). Further post-hoc Mann-Whitney tests revealed that when the critical group and the high distress group were compared to the low risk group, significant differences were found across all of the variables ($p<.05$).

Table 5

Cluster Analysis in Individuals with CD

Variable	**Low Risk (60)**	**Critical (25)**	**High Distress (11)**
Depression (0-21)	1.72	5.4	12
BES Total (0-46)	6.58	11.44	39
Stress (0-17)	3.57	8.72	14.45
GFD Management (Always-Never)	Always	Most of the time	Always
EAT-26 Total (10-40)	8.3	18.96	10.36
Gastrointestinal Symptoms (0-8)	7.13	11.72	13.82

GFD, gluten-free diet; BES, Binge Eating Scale; EAT-26, Eating Attitudes Test-26

Surprisingly, years with diagnosis had a predictor importance less than 0.2 and was subsequently removed from this cluster analysis. We calculated the age of diagnosis and divided this into adult diagnosis, childhood diagnosis and less than 4 years. However, the sample sizes were too small to conduct further analysis.

3.4. Discussion

The primary goal of this study was to explore the prevalence, predictors and types of disordered eating in CD, inflammatory bowel disease, type two diabetes and healthy controls, and examine whether factors unique to the diagnosis of CD contributed to reports of disordered eating above the impact of having a dietary-controlled health condition.

This study used two screening tools for disordered eating, measuring a combination of disordered eating attitudes and self-reported behaviours. Our findings were consistent with previous research; the prevalence of disordered eating as assessed by the EAT-26 was greater in CD compared to healthy controls, with 15.7% scoring above the clinical cut-off. This is lower than previous reports of 22-29% but significantly higher than healthy controls (Arigo, Anskis & Smyth, 2012; Karwautz et al., 2008).

Uniquely, this chapter compared the prevalence of disordered eating across dietary-controlled health conditions. Of those with inflammatory bowel disease, 20% scored above the cut-off on the EAT-26, with no significant differences in prevalence scores between inflammatory bowel disease and CD. Individuals with dietary-controlled gastrointestinal conditions may be placed at a unique risk for the development of Anorexic-type attitudes and behaviours. The nature of these associations is unclear but the presence of gastrointestinal symptoms may be important in the development of disordered eating in those with gastrointestinal disease (Tang et al., 1997). It is not clear how gastrointestinal symptoms are associated with disordered eating but potential mechanisms may include accidental or intentional gluten ingestion, which is consistent with *The Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). Case reports indicate that for some individuals with gastrointestinal disease, their prescribed dietary-regimen may interact with disordered eating; the consumption of foods that trigger gastrointestinal symptoms may be used to promote weight loss (Leffler et al., 2007; Yucel et al., 2006). Furthermore, larger studies in CD have found associations between disordered eating scores and dietary transgressions (Wagner et al., 2015). A

similar phenomenon has been described in type one diabetes, where individuals may withhold insulin to promote weight loss (Jones, Lawson, Daneman, Olmsted & Rodin, 2000). Future research should focus on the role of gastrointestinal symptoms, dietary-management and disordered eating in CD.

This chapter has identified specific factors that are associated with disordered eating in CD. In CD, disease specific factors explained additional variance in EAT-26 scores (29.7%) when compared to disease-nonspecific factors, and dietary management was only important for the CD group. In line with previous research, poor dietary self-management explained additional variance in EAT-26 scores for those with CD (Arigo, Anskis & Smyth 2012; Karwautz et al., 2008; Wagner et al., 2015). In addition, distress was associated with EAT-26 scores in CD; however, distress scores were no longer significant when accounting for gastrointestinal symptoms and dietary management in CD. Furthermore, the cluster analysis produced a "critical" group who scored high on the EAT-26 but reported poorer dietary self-management. This suggests that a small group of individuals with CD may have a difficult relationship with food. Some individuals may engage in poor dietary self-management in order to promote villous atrophy and subsequent weight loss (Leffler et al., 2007). This offers one interpretation of these results; however, the self-reported measures of dietary self-management and the motivations behind poor management are unclear.

When compared with healthy controls, all dietary-controlled diagnostic groups had increased scores on the BES. Binge eating is commonly reported in those with type two diabetes, so it is unsurprising that those with type two diabetes scored highest on these measures (Crow, Kendall, Praus & Thuras, 2001). Binge eating has

not previously been reported in those with CD. In the United Kingdom, it has been reported that up to 81% of individuals gain weight after commencing the GFD (Dickey & Kearney, 2006). This weight gain has been attributed to factors including the poor nutritional quality of some gluten-free foods, resulting in an increased energy intake, and intestinal recovery (Garcia-Manzanares & Lucendo, 2011; Kabbani et al., 2012); however for a subset of individuals, our results suggest that binge eating may also play a role in weight gain. Future research should focus on the relationship between binge eating and weight changes in CD.

Factors common to all conditions (years with condition, psychological distress) were more strongly associated with BES scores across all diagnostic groups. Binge eating in CD may be influenced by distress associated with the presence of a long-term condition. Greater psychological distress has frequently been associated with binge eating behaviours (Dide & Fitzgibbon, 2005). Furthermore, the cluster analysis highlighted a "High Distress" group who were characterised by increased BES scores and psychological distress. Alternatively, following a restricted dietary regimen, like the GFD, may increase the risk of binge eating behaviours through disinhibition (Herman & Polivy, 1985).

3.4.1. Limitations and Future Research

The cross-sectional nature of this study limits any conclusions about the sequence of events between disordered eating and CD diagnosis. Longitudinal studies are essential in determining the timeframe between disordered eating onset and CD diagnosis. Furthermore, online recruitment may create a bias in sampling which may over/under-inflate problems with eating behaviours and dietary self-

management. In addition, these samples were younger than those previously reported across all conditions. This may be due to the nature of online sampling, which is likely to attract a younger population (Remillard et al., 2014). Despite these limitations, this study provides an important extension in exploring disordered eating in those with CD and online methods allowed recruitment of a large sample.

Due to the nature of online data collection, CD diagnosis, dietary management, disordered eating scores and psychological distress were all based on self-report. These findings need replication in a biopsy-confirmed sample of individuals with CD and should focus on more objective measures of dietary-management such as anti-tissue transglutaminase assays, questionnaires designed to assess gluten-free dietary management (Leffler et al., 2009) and multi-modal approaches, including self-report and dietician assessment. However, the comparison across different chronic health conditions, recruited in the same manner, is a strength of this study and provides an extension of existing research in CD and disordered eating.

No evidence was found for the role of anxiety in the development of disordered eating behaviours. Surprisingly the FNS was not a good predictor of disordered eating. We had anticipated that FNS scores might tap into fears about cross-contamination and trying new foods. However, the FNS may lack sensitivity to assess this mechanism in those with CD. The development of a scale measuring food anxiety in CD may allow further investigation of the role of anxiety around food in disordered eating in CD.

3.4.2. Clinical Implications

The observation that individuals with dietary-controlled chronic health conditions have increased scores in disordered eating tools when compared to healthy controls suggesting that the use of screening tools for disordered eating may be valuable in these individuals. More specifically, the observation that gastrointestinal symptoms and poor dietary management were associated with EAT-26 scores in CD, indicates that individuals experiencing difficulties in managing their GFD and reporting gastrointestinal symptoms may benefit from have their eating attitudes and behaviours explored. In addition, for those who do score above clinical cut-offs, it is important to consider how their chronic health condition may interact with disordered eating attitudes and behaviours.

3.5. Conclusions

This chapter indicates factors both common to all dietary-controlled health conditions (psychological distress), gastrointestinal symptoms and factors unique to the CD diagnosis (GFD management) require further assessment in relation to CD and disordered eating.

A small group of people with CD display poor dietary management and this is associated with disordered eating attitudes and beliefs, lending some support to *The Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). The majority of individuals with CD display a typical eating pattern, but for some, disordered eating behaviours are a feature of their CD. This chapter has isolated some factors that are specific to CD that may place individuals at increased risk for disordered eating attitudes and behaviours. Future research should focus on

understanding this sub-group of individuals with CD and look at ways to identify them and provide support.

CHAPTER FOUR: DISORDERED EATING PATTERNS IN CD, A FRAMEWORK ANALYSIS

4.0. Chapter Rationale

Chapter Three suggested that factors specific to a CD diagnosis (gastrointestinal symptoms, GFD management), as well as the non-specific stressors associated with chronic health conditions, were associated with disordered eating attitudes and behaviours in CD. This supports some of the ideas proposed in the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). However, the role of anxiety around food, which is central to the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*, was not found to play an important role and the motivations behind these disordered eating attitudes and behaviours were not explored.

The aim of this chapter was to explore the experiences of typical and disordered eating in CD to gain a greater understanding of these processes and explore specific pathways within this model of disordered eating, particularly in relation to food anxiety.

A qualitative methodology is employed in this chapter to understand the reasons behind disordered eating attitudes and behaviours in CD in order to evaluate the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.*

4.1. Introduction

Management of the GFD requires vigilance around cross-contamination of food products, because small amounts of gluten can cause symptoms in some individuals (Sverker, Hensing & Hallert, 2005). In the majority of people with CD, successful management of the GFD reverses damage to the gut and reduces symptoms. However, the GFD can be challenging to follow and can create concerns around eating outside the home and cross-contamination of food products (Sverker, Hensing & Hallert, 2005). Although the GFD is physically beneficial for the individual, its restrictive nature may impact quality of life and result in maladaptive behaviours, including disordered eating patterns (Leffler et al., 2007; Lohiniemi, Mustalahti & Collin, 1998; Chapter Two).

The majority of individuals with CD score in the healthy range on self-report measures of disordered eating (Karwautz et al., 2008; Chapter Three). However, for some, CD may act as a risk factor for the development of disordered eating via a number of mechanisms. Factors essential in managing the GFD, including food preoccupation and awareness, may harm relationships with food (De Rosa et al., 2004). Additionally, factors relating to the diagnostic experience, including gastrointestinal symptoms and changes in weight, may affect body image and eating patterns (Capristo et al., 2000). Alternatively, the non-specific burden of chronic illness may account for the presence of disordered eating in this population. The results of Chapter Three suggested that factors both unique to the CD diagnosis (gastrointestinal symptoms, dietary management) and nonspecific factors (psychological distress) are important factors in disordered eating and CD.

The present study was theoretically informed by the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). Central to this model are two pathways; the first pathway describes individuals who experience anxiety around food and cope with this by consuming a limited variety of gluten-free foods. The second pathway describes those who struggle with weight changes experienced after diagnosis (usually weight gain) and engage in poor dietary self-management to promote gastrointestinal symptoms and associated weight loss. In an evaluation of this model, dietary-management and gastrointestinal symptoms were associated with disordered eating scores, lending some support to pathway two (Chapter Three). However, the relationships between gastrointestinal symptoms, dietary-management and disordered eating were not clear. Furthermore, no evidence was found to support pathway one, the role of anxiety in disordered eating. This was attributed to a lack of appropriate tools to measure concerns around food in individuals with CD. Understanding these factors and their role in the development of disordered eating is essential if appropriate supportive strategies are to be adopted by healthcare professionals.

The present study aimed to gain a holistic view of the experiences of typical and disordered eating in CD. This was done by exploring the pathways of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* by using a structured framework to analyse interviews with people with CD. According to the model, the type of disordered eating pattern that develops will depend on beliefs about CD and the GFD, as well as the psychological response to weight changes after CD diagnosis. By using this model to create the framework for the interviews, we were able to assess how well this model was supported by qualitative data.

4.2. Method

Participants (18-69 years) with a self-reported biopsy-confirmed diagnosis of CD, for at least 2 years, without additional food allergies or health conditions, were eligible to participate. Purposive sampling was used to recruit both typical and disordered eaters from a previous database. Participants were categorised as disordered eaters (DE) or typical eaters (non-DE) based on a score above 20 on the EAT-26 or a score above 17 on the BES (Garner et al., 1982; Gormally et al., 1982). The EAT-26 is a screening tool that measures symptoms and concerns characteristic of eating disorders and the BES screens for the presence of binge eating behaviour. These are not diagnostic tools but screen for the presence of disordered eating behaviours.

Sample size was based on data saturation, by repeatedly comparing data across participants, which occurred when no new information was obtained from the interviews (Higginbotham, Albrecht & Connor, 2001). Twenty-five participants were invited to take part in the interviews but three withdrew their data and one was removed from analysis, as the inclusion criteria were not met. Participants were informed that the interview would explore eating patterns in CD. Demographic (gender, age, years since diagnosis, Body Mass Index (BMI)) and health information (EAT-26 and BES scores) were taken from the existing database.

A semi-structured interview schedule allowed us to frame questions to fit the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). The interview explored key themes concerning the diagnosis of CD, the daily management of the GFD and how CD has affected participants' relationship with food and body image.

4.2.1. Procedure

Participants provided written consent before their interviews. The lead researcher (RS) conducted and audiotaped the interviews. Each interview lasted between 30 and 45 minutes and was conducted in the participant's home. If any current or past disordered eating was reported, participants were asked to discuss this in more detail, and reflect on any links with their CD diagnosis. The interviewer encouraged participants to elaborate on relevant themes.

4.2.2. Data Analysis

Ritchie and Spencer's (1994) Framework methodology was used as it allows the use of a theoretically-driven framework to structure and explore the data. Framework analysis was beneficial for this study because it can include *a priori* themes drawn directly from the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two), as well as emergent concepts.

Interviews were transcribed verbatim by the lead researcher (RS), and read repeatedly in order to identify key themes. These themes were developed into a framework for coding the entire dataset. Additional categories were created for data that did not fit into the framework. To enhance reliability, the coding process and emerging themes were discussed among the authors until consensus was achieved. Trustworthiness of the data was enhanced using a decision trail to ensure transparency (Koch, 1994).

4.2.3. Ethical Approval

Ethical approval was granted by the Research Ethics Committee, University of Birmingham.

4.3. Results

Five males and 16 females took part in the interviews (mean age = 39 years; mean time since diagnosis = 5 years). Of these, 10 participants scored above EAT-26 or BES cut-offs resulting in them being classified as “disordered eaters”. Participant information can be found in Table One. Illustrative quotes presented are annotated with pseudonyms and participants’ disordered eating status (DE or non-DE). ‘Disordered eaters’ and ‘typical eaters’ displayed significantly different BES ($t(19)=-7.1$, $p=<.001$) and EAT-26 ($t(19)=-.6$, $p<.001$) scores. There were no significant differences between participants for age, BMI or years since diagnosis.

Table 1

Participant Characteristics and Disordered Eating Scores

	Pseudonym	Age (years)	Years since Diagnosis	EAT-26 Score	BES Score
'Typical Eaters'	Katy	19	3	6	8
	John	53	2	3	3
	Mel	26	2	0	3
	Louise	29	10	8	1
	Sue	49	5	0	11
	Colette	59	19	5	2
	Richard	49	4	4	5
	Anna	28	3	7	1
	Katherine	32	3	9	5
	George	36	7	0	2
	Andrea	29	6	3	3
	Mean	*37.2*	*5.8*	*4.1*	*4*
'Disordered Eaters'	Caroline	48	3	12	23**
	Amy	48	3	26*	18**
	Paula	41	3	26*	8
	Georgia	48	2	26*	30**
	Dan	40	6	21*	25**
	Julie	22	4	30*	13
	Martha	35	4	27*	14
	Steve	38	6	19	22**
	Holly	29	2	26*	21**
	Lisa	54	8	27*	19**
	Mean	*40.3*	*4.1*	*24*	*19.3*

Note. * >20 on EAT-26; ** >17 on BES

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two) describes three stages in the development of disordered eating: adaptation to diagnosis, illness beliefs and dietary management. These stages provided the analytic framework for the hierarchical themes. Each hierarchical theme was coded in depth to identify subordinate themes. Each of these subordinate themes was explored, resulting in 8 sub-themes (see Table Two). All themes were reported across participants but experiences and opinions differed across individuals.

Table 2.

Final Thematic Structure and Example Quotes from Disordered and Typical Eaters

Superordinate Theme	Sub-Theme	Example Quotes from Disordered Eaters	Example Quotes from Typical Eaters
Adaptation	*The New Self*	Caroline: I've lost a lot of confidence in the way I look. Julie: My stomach is a lot bigger now, it's hard to accept that that's healthier. Dan: My weight kept going up and down, I found that very difficult.	Sue: I have more strength and energy, so I perceive myself as better. John: My weight hasn't changed much at all. Richard: I've lost some weight, I think that's one of the benefits of being a Coeliac.
	Mourning of Gluten	Julie: Viennese whirls. I miss them, they were my favourite and I get sad thinking about them. Dan: I ate a lot of gluten, to say goodbye to the foods I wouldn't be able to eat anymore. Caroline: My diet is so restrictive, it's impossible not to miss old foods.	Richard: I don't really miss any foods because they made me so ill. Louise: It felt like a mourning for what you couldn't have, I was angry but made peace with it in the end. Sue: I feel so much better now, I don't think I could miss gluten.
Illness Beliefs	*The Dangers of Cross Contamination*	Amy: I clean the surfaces before cooking and reduce the contamination risk. Georgia: I don't let cross-contamination control me, I just do a quick check before eating. Julie: It doesn't overly concern me, I might get ill but a small bit of gluten won't kill me.	Sue: I'm worried about the crumbs, if my husband's bread is in my kitchen, I won't eat. Louise: Sometimes it's safer not to eat because cross-contamination is everywhere. Mel: I have a gluten radar on at all times, if that radar is activated, it's best not to eat.
	My GFD Makes me Fat	Georgia: Gluten-free foods are full of calories, they make me feel fat. Caroline: Gluten-free food is full of rubbish, it definitely contributed to my weight gain.	Katy: Gluten-free cakes are unhealthy but I limit them like anyone else would limit cakes. Richard: I knew that I would gain weight as my body healed.

Dietary Management	*Risk-Taking*	Paula: Sometimes I'll take a very small risk. Georgia: I think I should probably be more careful than I am. Caroline: It's hard outside the home, I may take some risks then.	Mel: Gluten is poison, I would never cheat. Richard: I'm very ill when I make mistakes, I can't let it happen. Louise: I haven't had gluten. I just don't allow it.
Eating Knowledge and Practices	*Eating for Pleasure*	Georgia: Food is my enemy at the moment. Paula: Food makes me upset. It makes me scared. It makes me jealous. Amy: Eating isn't enjoyable anymore, it causes a lot of stress, particularly outside the home	John: Food is just a tool for my body now. Sue: I've gone off food, it causes me a lot of anxiety. Richard: Eating is a lot more difficult than it used to be, it can be done but it involves a lot more planning and isn't as relaxed.
	Food Preoccupation	Caroline: I'm a lot more aware of the calories in food now and more careful about what I eat. Julie: The gluten-free foods are full of fat and calories, I just avoid them. Georgia: Food is always on my mind, I think I'm a little bit obsessive about food.	Katy: You're always thinking about food. You're always cooking food. Mel: It does make you a bit conscious about how you are with foods. Richard: Food is always on my mind but it motivates me to cook and I now want to make a gluten free cake shop.
	New Eating Patterns	Julie: I overcompensate with cakes and cookies. Caroline: I eat a limited range of foods but it works for me. Dan: I always search for the new gluten-free treats. They're hard to find, so I feel like I deserve them when I can have them.	Colette: I will eat anything, as long as it's gluten-free. Richard: I cook a lot more now and I'm more interested in cooking, which makes sourcing food a lot easier. Sue: I don't eat out as much now, but in my home it's just the same as it used to be.

4.3.1 Thoughts, Feelings and Behaviours Underlying Disordered Eating

4.3.2 Adaptation to Diagnosis

The New Self

The diagnostic process was related to physical changes in body image, which were important in the adoption of disordered eating patterns. Disordered eaters described distress around weight changes after diagnosis. This was linked with a desire to lose weight by restricting food intake.

I liked being thin. I was over 30 and I wasn't putting on weight. I've definitely got a big belly now, I've put on weight and it's really bothering me. I really have lost a lot of confidence in terms of the way I look. So I go on more diets now, to try and get back to how I was. I'd like to be back to my pre-diagnosis weight. **(Georgia, DE)**

These weight changes were challenging for disordered eaters and Dan felt that more support could have been provided from healthcare professionals.

Associating thinness with unhealthiness is strange. Putting on weight but being healthy, it goes against the things you read about. I think the dietician could have explained that once your stomach goes back to normal there will be a process where you start to gain weight. I don't remember her explaining that. That may have helped me feel better. **(Dan, DE)**

Some individuals did not experience post-diagnosis weight changes and others felt happier with their weight once they were following the GFD. Typical eaters felt better after diagnosis because of their increased energy, which was associated with an improved body image.

The thing I've really noticed is that when I'm feeling ok, I've got so much more strength and energy. And that makes me perceive my body better. **(Amy, non-DE)**

Mourning Gluten

Participants described the challenging process of mourning gluten-containing foods after diagnosis, which was accompanied by distress. Twelve participants described a ritualistic consumption of gluten "for the last time". These feelings of loss were still present in disordered eaters and were associated with a desire to consume gluten-containing foods.

There's a certain food that I'd normally eat, I remember I cried when I ate that for the last time. I ate loads of it, to try and say goodbye. That was really upsetting. I still miss the food, it's really hard. I just want to eat it again. I get upset seeing friends eat it. **(Paula, DE)**

For typical eaters, this mourning process was brief and no longer occupied their thoughts.

There is a sort of grieving process for maybe a few months. But now it's just a part of life. There's no reason to miss food that made me ill. **(Colette, non-DE)**

After the adjustment process and acceptance of their diagnosis, participants began to develop beliefs about their CD and the GFD.

4.3.3. Illness Beliefs

The Dangers of Cross Contamination

Cross-contamination was frequently referred to during the interviews. However, disordered eaters were less concerned about cross-contamination than were typical eaters, and believed that accidental gluten ingestion would not impact their long-term health.

I'm rarely ill from cross-contamination, so I take risks and deal with the consequences. A tiny amount of gluten every so often won't have adverse effects on your long-term health; it just might make you feel sick. **(Julie, DE)**

Typical eaters had greater concerns around cross-contamination and went to greater lengths to avoid cross-contamination than did the disordered eaters. Louise coped with these concerns by limiting her food intake when outside of her home environment.

Sitting in the staff room with everyone else eating food, that's scary. Um, I know they're not going to touch me or make me eat it or anything but I won't eat anything. There's just too much risk. I only eat my own foods in my own home… if I'm out shopping all day, I won't eat but I'll eat my own safe food when I get home. **(Louise, non-DE)**

For three individuals, these cross-contamination concerns extended into their own home: the kitchen was viewed as an unsafe environment and resulted in a restricted food intake.

The kitchen isn't safe. It's gluten-free, but it's more that food in general isn't safe. I get worried around food. I have a few safe things that I do eat but food has become the enemy now. It's just safer not to eat. **(Mel, non-DE)**

For two individuals, these beliefs around cross-contamination extended to non-food items.

I won't let my husband put up wallpaper because I'm worried about the gluten in the wallpaper paste. Those hidden gluten sources make it hard to eat safely. **(Sue, non-DE)**

Response to Weight Changes due to GFD

Participants were asked about the causes of any weight changes experienced after commencing the GFD. Seventeen participants experienced weight gain after starting the GFD whereas the remainder experienced no change or weight loss. Disordered eaters attributed weight changes to the GFD and the poor nutritional quality of gluten-free foods; they responded by restricting their food intake.

And the gluten-free foods, if it's not super fatty, it's super sugary. Eating gluten-free food made me really fat. It's hard to stay slim on a gluten-free diet. I've had to go on diets to lose the weight but it's hard. **(Paula, DE)**

For typical eaters, weight changes were attributed to the recovery of the intestine and improved health.

My weight has been quite stable, I put on a bit at first but I was really underweight. I read all the books and they said that when your body recovers your weight should be normal. And that's what happened. **(Mel, non-DE)**

4.3.4. Dietary Management

Risk Taking

The majority of participants managed their GFD well. However, four disordered eaters reported consuming small amounts of gluten.

There was this really good sauce and I did take a really small piece of crusty bread. Because crusty bread is the thing I miss the most. And I very gingerly sort of scooped up all the sauces and ate it. It would be a small piece that hopefully I'm going to sort of eat without my stomach noticing. **(Dan, DE)**

Not all individuals with disordered eating reported deliberate gluten ingestion and this was not recognised as a technique to promote weight loss.

For typical eaters, their concerns around cross-contamination and the fear of re-experiencing unpleasant symptoms meant that risk taking was not tolerated.

I don't take risks. I can't take risks. Gluten poisons me, why would you risk being poisoned? **(Sue, non-DE)**

4.3.5. Patterns of Disordered Eating

4.3.6. Eating Knowledge and Practices

All participants felt that their eating patterns and the way they thought about food had changed since their diagnosis. Their thoughts and feelings about their CD affected both their attitudes towards food and the way they consumed food. Three sub-themes emerged related to these changes in eating patterns and beliefs: *food preoccupation, eating for pleasure and new eating patterns.*

Food Preoccupation

All participants reported that their diagnosis of CD had made them more aware of the foods they were consuming and more aware of the nutritional content of food. This awareness arose from the need to manage the GFD and the preparation and planning that this involved. Participants were always thinking about food, what meal they were having next and where this food was coming from. For disordered eaters, this food preoccupation dominated their thoughts.

You've got to think about the range of colours you're eating, the nutrients and about the quantity, you're thinking about a whole range of stuff. I'm a bit obsessive

about food. It does change your relationship with food. You're always thinking about food. **(Paula, DE)**

This awareness of food often led to an increased awareness of the calorific content of food. Seven individuals became dissatisfied with the amount of calories they were consuming and became dissatisfied with their body image.

Since becoming coeliac I'm also a lot more calorie conscious as well. And the gluten free foods. They're full of calories and fat, and that has made me, well, fat. Now I'm much more conscious, about everything I eat. **(Georgia, DE)**

Typical eaters described an awareness of food, but they were able to integrate these thoughts around food into their life.

I'm a lot more aware of food now, it's on my mind a lot but that doesn't bother me. I might see a Chinese recipe but I'd just wonder how I could make it gluten-free. It's just a part of life. **(Richard, non-DE)**

Eating for Pleasure

After CD diagnosis, emotional relationships with food had changed. Meal times were described as challenging and eating was no longer enjoyable. For disordered eaters, a loss of pleasure around eating was common and was strongly interlinked with emotions: food became a source of distress.

Initially I was anxious. Finding out all these foods you couldn't have and thinking why the hell does that have gluten in it, was upsetting. Food is now my enemy, food kills me, food attacks me. I know that sounds really melodramatic but that's how it feels. **(Dan, DE)**

A lack of enjoyment in the eating process resulted in typical eaters simply viewing food as fuel for the body.

I've gone off food really. Food is the baddie in my life at the moment. I just eat what I have to; I've lost the enjoyment of sitting down and going out for a meal.

(Amy, non-DE)

In comparison, the majority of typical eaters enjoyed eating outside the home, whilst managing their GFD.

It's harder to eat out but you can't let that dictate your life. I still enjoy going out with friends for a meal, I just have to be careful. **(Richard, non-DE)**

New Eating Patterns

Some participants reported an improvement in their diet since diagnosis; however, others reported eating patterns that appeared disordered in nature.

For eight disordered eaters, overconsumption of food was reported and this was linked with emotional distress. The restrictive nature of the GFD made participants long for certain foods. When these foods were available, they would be bought in bulk and consumed in a short space of time, indicating a binge-type eating pattern. However, the consumption of this food was not associated with guilt.

When you're unable to eat certain foods, you then overcompensate with other things like wine, chocolate, biscuits. It's depressing not getting these foods, so when you do, you just enjoy it. And eat loads of it. I don't feel guilty, when I eat it, I feel happy again. The cakes aren't going to be there tomorrow, so eat it while you can.

(Paula, DE)

Some disordered eaters felt that because of the restrictive nature of their GFD they deserved to indulge in certain foods. Some participants hoarded gluten-free foods and ate them at a fast rate.

When the gluten-free Kit Kat bars first came out, I hoarded those because they were delicious. If it's good, I'll be hoarding. Sometimes I eat them all myself. I think that's probably my way of dealing with it. And I eat faster than I used to, I just eat it quickly before someone's like – no you can't eat that. **(Julie, DE)**

Other disordered eaters felt a need to limit their food intake due to concerns around weight increase since their CD diagnosis.

It's like being on several diets at once. I can't eat gluten, I eat naturally gluten-free because of all the calories in gluten-free breads and pasta, and I'm on a Slimming World diet because of all the weight I put on after my diagnosis. I just want to lose the weight. **(Martha, DE)**

Typical eaters used strategies to improve food availability. This included cooking large quantities of food and storing them to consume during the week.

I kind of, I think I make up for the fact that I can't eat gluten by baking a lot of gluten free cookies and meals. I portion them and freeze them for later in the week. **(Katy, non-DE)**

Five typical eaters developed a fear of trying new foods or trying foods in new environments. This stemmed from concerns around cross-contamination and the belief that it was dangerous to eat foods outside the home. Some typical eaters reported going for long periods of time without eating outside the home. These participants no longer enjoyed eating in general and felt more at ease when they were not around food, which resulted in restricted food intake.

If I'm out shopping all day, I prefer not to eat. It's just not safe to eat. Eating has become scary because of my coeliac. I only eat if I'm desperate. Food is too dangerous now, when I'm not eating I feel safe. **(George, non-DE)**

Others felt that their eating patterns were not affected by their CD diagnosis. They were still able to maintain a nutritionally balanced diet. These participants were able to consume a range of foods both inside and outside the home, despite sticking to their GFD.

As long as I know it is gluten free, I'll try anything. I'm not a fussy eater at all. I've always been that way. The only restriction to that is whether it's gluten free or not.

(Katy, non-DE)

4.4. Discussion

This chapter investigated the experiences of disordered eating in CD, in order to test the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). Disordered eaters reported eating patterns suggestive of a binge/restrict cycle, which was associated with psychological distress, poor dietary-management and a preoccupation with food.

4.4.1. Disordered Eaters

Disordered eaters, as assessed by the EAT-26 and the BES, developed eating beliefs that stemmed from concerns around weight changes associated with commencing the GFD. These weight changes caused distress and participants found it challenging to adapt to their new body image. They described a desire to reach their pre-diagnostic weight and responded by restricting their dietary intake. Weight increase is a known trigger for disordered eating behaviours that may be viewed positively by those who are underweight at diagnosis but may be unwelcome in those who begin at a normal or higher weight (Andres & Saldana, 2014). These findings are in line with Leffler et al. (2007) who described three cases where

concerns around weight increased after starting the GFD, which led to disordered eating behaviours.

Distress and mourning the loss of gluten-containing foods were associated with disordered eating status. All participants experienced a mourning period, but for disordered eaters, there was an extended period of distress surrounding the loss of gluten-containing foods, that lasted for years after diagnosis. Participants coped with these feelings by overcompensating with high energy-dense, gluten-free foods such as cakes and biscuits. Consumption of high-energy dense foods has frequently been reported in those with CD (Mariani et al., 1998), but our results indicate that this may occur to help manage distress. Participants reported no guilt around the consumption of these foods because they felt they "deserved" to eat them. This resulted in the hoarding of foods and fast food consumption. This could be an indication of binge-eating type behaviour in a sub-group of participants, all of whom were classified as disordered eaters according to the BES (APA, 2013).

Disordered eaters reported that overconsumption occurred in combination with restrictive eating: weight loss was promoted by restricting food intake but this resulted in a preoccupation with food and psychological distress, which resulted in binge eating. These findings are in line with Herman and Polivy's (1984) Boundary Model, which suggests that those who restrict their intake are more responsive to external stimuli and at risk for both under and overconsumption of food. Similar patterns of eating have been described in people with type two diabetics who also follow a prescribed dietary regimen (Herpertz et al., 2001). These findings highlight the complex interplay of emotions and food, which may alter eating attitudes and behaviours in CD. An increased intake of high-density gluten free foods may be used

to cope with feelings of distress that arise from the restrictive nature of the GFD. Mazzeo and Bulik (2009) suggested that disordered eating arises after a stressful event as a way to manage emotions and acts as a coping mechanism.

Intentional gluten consumption to promote weight loss was not reported. When asked about gluten-consumption in an anonymised web-meditated survey, poor dietary management was associated with disordered eating (Chapter Three). In addition, case studies have documented the interaction between intentional gluten consumption and a desire to promote weight loss through villous atrophy (Karwautz et al., 2008; Leffler et al., 2007). However, only four participants, categorised as 'disordered eaters', described occasional gluten ingestion or risk-taking behaviours. Participants may not have been willing to talk about intentional gluten consumption as a way of losing weight with the interviewer due to perceived lack of anonymity.

4.4.2. Typical Eaters

Typical eaters differed from disordered eaters in thoughts, feelings and behaviours. Despite experiencing weight changes after diagnosis, typical eaters felt healthy and energetic with increased confidence. This is in line with findings suggesting that quality of life increases after initiation of the GFD (Casellas et al., 2015; Sainsbury & Mullan, 2011). Typical eaters also experienced a mourning period after diagnosis but these feelings of loss were no longer present at the time of interview. Typical eaters associated gluten-containing foods with the symptoms they had experienced prior to commencing the GFD and had no desire to consume these items again.

Caution around cross-contamination is essential for those with CD but may contribute to limited food consumption, both inside and outside the home. Some typical eaters reported going for long periods of time without consuming food because they believed that limiting food consumption was keeping them safe, particularly when outside the home. Furthermore, two typical eaters described a concern around non-food products, these beliefs affected their ability to eat outside the home. Neither the EAT-26 nor the BES captured the consequences of these cross-contamination beliefs on eating patterns. However, this form of dietary self-management may result in eating behaviours that could be considered 'disordered' (i.e. restricting and bingeing behaviours) as they deviate from the norm (Polloni et al., 2013).

Importantly, not all participants displayed high levels of concern around food. Eight individuals were happy to try new foods that they believed were gluten-free. These individuals described a healthy eating style and adaptive beliefs about food, with the caveat that their diet was gluten-free.

4.4.3. The Theoretical Model of Disordered Eating

These findings provide support for the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). The first pathway of the model suggests that an unwanted increase in weight after diagnosis results in the belief that the GFD is responsible for this weight gain, which results in poor dietary self-management to lose weight. Although our data suggests that distress around weight change is associated with disordered eating attitudes and behaviours, there was no evidence for the role of intentional gluten ingestion to promote weight loss. In addition, the

mourning and distress around the loss of gluten-containing foods was associated with a desire to consume gluten. These findings are closely in line with the CD grief process described by Rose and Howard, whereby the benefits of following a GFD were not always viewed as beneficial, resulting in problems with dietary management (Rose & Howard, 2014). Future revisions of the theoretical model should consider the role of distress and feelings of loss in relation to gluten-containing foods.

The second pathway describes those who adapt well to their CD diagnosis and have good dietary self-management but overly extreme concerns around cross-contamination may develop. Our findings suggest that some participants developed an extreme vigilance around food, which was associated with limited food intake and concerns around food preparation and consumption. However, these individuals did not score above clinical cut-offs on measures of disordered eating. Vigilance around cross-contamination is essential for GFD management but it is unclear from the current data whether these extreme concerns around cross-contamination are maladaptive. Future revisions of the theoretical model need to consider the types of concerns around food in those with CD to identify factors that may promote maladaptive concerns.

Strengths and Limitations

All participants were diagnosed at 16 years of age or older; however, age of diagnosis may have an impact on interactions with food, and this is often associated with the development of disordered eating in chronic health conditions (Davidson, 2014). Childhood diagnosis may differ from adolescent and adult diagnosis in the risk for disordered eating patterns, as diagnosis under four years has been associated

with better dietary-management and better psychological well-being whereas those diagnosed in adolescence show more problems with social interactions and more physical health problems (Hogberg, Grodzinsky & Stenhammar, 2003; Wagner et al., 2008). In addition, the EAT-26 and the BES allow screening of disordered eating but cannot be used as diagnostic tools. Future research could focus on looking at those who display clinically significant disordered eating patterns, assessed through clinical interview and the use of diagnostic tools.

4.5. Conclusions

This qualitative study was guided by the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* and allowed us to gain in-depth understanding into the application of this model to CD (Chapter Two) and has provided insight into the factors that may contribute to the development of disordered eating patterns in CD. The results suggest that experiences of disordered eating differ across individuals with CD but relate closely to the CD diagnosis and management of the GFD. Greater understanding is still needed, especially in regards to atypical eating patterns, which are not detected by current measures of disordered eating. The BES and EAT-26 appear to be effective in identifying individuals who display binge/restrict-eating patterns. However, these tools were not able to select individuals who limited their food intake due to concerns around food and cross-contamination. Directions for future research should focus on tools to assess concerns around food and cross-contamination in CD.

CHAPTER FIVE: DEVELOPMENT AND VALIDATION OF THE COELIAC DISEASE FOOD ATTITUDES AND BEHAVIOURS SCALE

5.0. Chapter Rationale

The qualitative results from Chapter Four suggest that the EAT-26 and the BES are useful for identifying a binge/restrict style of eating in CD. However, despite scoring below the EAT-26 and BES cut-offs, some participants from Chapter Four described concerns around food and the potential for the cross-contamination of food products. As a result, these individuals would only eat food that they had prepared, in familiar environments. This led to food being described as a fearful stimulus and difficulties, or even a refusal, in eating outside the home. Current disordered eating questionnaires were unable to identify individuals who described concerns around food and patterns of food avoidance.

The aim of this chapter was to explore these disordered eating attitudes and behaviours in the context of CD by developing and validating a scale to assess thoughts and behaviours around food and cross-contamination in CD. This chapter describes the development of a tool that is designed to target the attitudes and behaviours described in Chapter Four, as well as the concerns around food that are highlighted in the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two).

5.1. Introduction

Case studies of comorbid CD and disordered eating, although limited by small sample sizes, are concordant with studies suggesting 22-29% of individuals with CD score above clinical cut-offs for disordered eating attitudes and behaviours (Arigo et al., 2012; Karwautz et al., 2008; Leffler et al., 2007). Chapter Two used the Binge Eating Scale (BES; Gormally, Black, Daston & Rardin, 1982) and the Eating Attitudes Test (EAT-26; Garner et al., 1982) to identify disordered eating attitudes and behaviours in CD. These tools appear to be effective at identifying disordered eating attitudes and behaviours in CD that are motivated by the desire to alter weight and reflect a binge/restrict style of eating (Chapter Four). However, some individuals who score below the clinical cut-offs on these measures describe eating attitudes and behaviours that appear disordered in nature (Chapter Four). Fears around cross-contamination and food safety were discussed in combination with a reduced willingness to eat outside the home and a fear around attending social events involving food. Therefore, existing measures of disordered eating may lead to inaccurate estimations of disordered eating in CD, as dietary restriction alongside concerns around food and cross-contamination are not captured by current disordered eating measures (Chapter Four).

The management of CD requires vigilance around food intake and knowledge of food preparation. Although control around food is essential for those with CD following a GFD, these beliefs about food may result in disordered eating attitudes and behaviours (Chapter Four). To better understand how these beliefs develop from adaptive coping mechanisms to disordered attitudes and behaviours, they must be measureable in CD.

Accordingly, we developed a CD food attitudes and behaviours scale (CD-FAB) that will identify disordered eating attitudes and behaviours resulting from beliefs around cross-contamination and food safety. Items in the CD-FAB are based on the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*, which was used to guide item development (Chapter Two). This chapter will report the development of the CD-FAB, including the psychometric properties comprising subscale structure, reliability and validity.

5.2. Methods

A mixed methods approach using three studies was used to develop the CD-FAB (see Figure One).

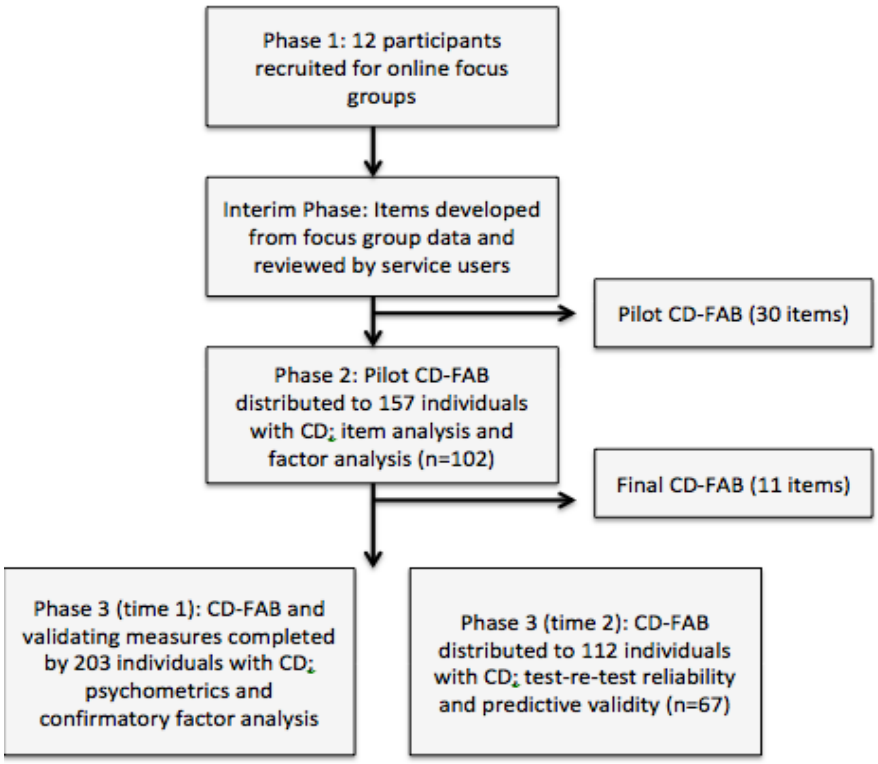

Figure 1. Flow chart of the CD-FAB questionnaire development and validation process.

5.2.1. Ethical Approval

Ethical approval was granted by the Psychology Research Ethics Committee, University of Birmingham.

5.3. Study 1: Item Generation

5.3.1. Methods of Item Generation

CD-FAB items were generated using one online focus group moderated by the lead researcher (RS) using an online platform (LiveMinds). Individuals with a self-reported biopsy-confirmed diagnosis of CD were recruited from online forums. Interested individuals emailed the lead researcher to complete a screening questionnaire and to confirm focus group attendance. All participants gave verbal consent over the phone and then gave online consent before being directed to the focus group. This provided the opportunity for questions about the study to be answered. Participants were aged between 18 and 69 years. Participants experiencing other dietary controlled health conditions (e.g. cystic fibrosis, diabetes mellitus) and food allergies were excluded.

Eight open-ended questions relating to feelings and concerns about food and cross-contamination were designed to answer the key questions of the study: 1) the construct of food attitudes in CD and 2) the everyday interactions with food in CD. These were used as a guide during the 90-minute focus-group session. These questions were developed based on the five stages of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease:* diagnosis of CD, adaptation to diagnosis, illness beliefs, dietary management and eating patterns (Chapter Two).

5.3.2. Data Analysis

Key themes related to food attitudes, concerns and eating behaviours were identified using thematic analysis (Braun & Clarke, 2006). The lead researcher (RS), who is experienced in qualitative analysis, and under the supervision of experienced academics, read through the transcripts noting down initial thoughts and ideas; she then re-read transcripts several times to allow data immersion. After immersion, the notes were consulted and the coding phase began. The codes identified characteristics that were related to food attitudes, concerns and eating behaviours, which were grouped by similarity to create themes. Emerging themes were used to develop items for the CD-FAB. Items were created using interviewee statements from the focus group to target each of the identified themes. Each item was transformed into a 7-point Likert scale (strongly agree to strongly disagree). Questions were phrased so that higher scores indicated greater food concerns; however, four items were reverse scored to minimise response bias. Individuals from the focus group and members from the Birmingham Coeliac UK committee were asked to rate the 33 pilot items of the CD-FAB based on clarity, adequacy and relevance to the focus group discussion. These questions were rated on a 5-point scale (1, Strongly Agree – 5, Strongly Disagree), and those items that consistently scored low were considered for removal.

5.3.3. Results of Item Generation

Twelve individuals took part in the online focus groups (10 females), (mean age =29.1 years, SD=5.7; mean time since diagnosis = 6.2 years, SD=2.3). Of the twelve participants, three had been diagnosed with CD for 10 years or more. The remainder

had been diagnosed for between 1 and 7 years. The majority of participants (66%) were in their late teens or 20's. The remainder ranged in age from 30 to 51 years.

5.3.4. Thematic Analysis

Four themes underlying dimensions of food concerns in CD were identified: *handling of food, trust, risk-taking* and *food safety.* Theme one, *handling of food*, refers to feelings around gluten-containing products, including preparing gluten-containing food for others, having gluten in the home and touching gluten-containing foods. Some individuals would prepare gluten-containing foods for others and had no concern being around gluten, as long as they did not have to consume gluten. Others described a fear around food that was attributed to their need to be vigilant about food content and feelings of anxiety would increase when they were around gluten. These individuals would not allow gluten in the home and for some, feelings of anxiety increased when they were in close proximity to gluten. "*I also get concerned in supermarkets when the gluten-free bread is next to the normal bread. I know they're all wrapped up but they're so close to each other. It just scares me. (Ashley)"*

The second theme, *trust* described the need for control during food preparation especially where others were involved in this process, which affected willingness to consume food. Concerns stemmed from the belief that others may not be vigilant around cross-contamination or may lack understanding of dietary requirements. To reduce concerns around eating food prepared by others, trust in the individual preparing food was needed. "*I don't let him (my boyfriend) prepare my food, I do that all myself. Only I cook for me! I like to be in control of my food, I can't trust*

others to do it (Charlie)". Clem described the impact of trust on the ability to eat outside the home "*Any time people talk about going out for a meal I always have to double check. If they (the restaurant) sound unsure I won't go. I need to be able to trust them (Clem)"*

The third dimension *risk-taking*, reflects the ability to consume new foods in new environments. 12 participants indicated that an element of risk was necessary in order to live a normal life. However, for some individuals, a lack of risk-taking led to isolation from events involving food because of concerns around cross-contamination. "*I have to take risks or I'd never eat anything. I used to be really paranoid that everything had gluten in it but I have to take small risks if I want to have a normal life! (Jamie)"*

Food safety describes the eating strategies that were employed by some participants to manage food concerns. Although the majority of participants were willing to try new gluten-free foods, or employed strategies to ensure that new foods were gluten-free, some viewed food as the enemy. These individuals experienced anxiety around food and felt safer when they were not eating. These individuals reported consuming a limited range of foods, or using long periods of dietary restriction in order to promote their safety and prevent gastrointestinal symptoms. "*I think I cope with my fear of getting glutened by not eating. That makes me feel safe." (Ashley).* These attitudes were related to participant's ability to recall their symptoms and adverse food experiences prior to diagnosis. "*I don't go to restaurants. They remind me of being ill. I don't want to feel like I did before going gluten-free again, so I don't eat much (Alex)"*

5.3.5. Item Development

These four themes were used to generate 33 items for the CD-FAB. Individuals from the focus group were asked to rate the 33 pilot items of the CD-FAB based on clarity, adequacy and relevance to the focus group discussion. In addition, three service users with CD, recruited via email from the Birmingham Coeliac UK committee, who did not take part in the focus groups, were asked to comment on the clarity, adequacy and relevance of the questions to individuals with CD. These questions were rated on a 5-point scale (1, Strongly Agree – 5, Strongly Disagree), and those items that consistently scored low were considered for removal. Based on the feedback, 13 items were re-worded and 3 items removed to create the pilot CD-FAB. Thirty items remained in the pilot CD-FAB to be used in Study Two.

5.4. Study 2: Item Analysis and Initial Exploratory Factor Analysis

5.4.1. Methods of Item Analysis and Initial Exploratory Factor Analysis

Study two identified items for the final scale. Data collection occurred between November-December 2015. The pilot CD-FAB was distributed to a new sample recruited from our research participant database (n=157). This database consists of adults (18-69 years) with self-reported biopsy confirmed CD, recruited from online forums and coeliac food fairs, who had previously volunteered to take part in our research. These individuals have consented to being contacted about taking part in future research. Interested individuals were directed to an online site to complete the questionnaire. The questionnaires included demographic and health information and the pilot CD-FAB.

5.4.2. Data Analysis

Items were reduced to only those that contributed to the questionnaires explanatory power (see Table One for removal criteria; Stevens, 1992). One item that was determined theoretically relevant due to its prevalence in responses during the focus groups, "*I am afraid to touch gluten-containing foods*", was retained despite the removal criteria.

Principle components analysis with orthogonal rotation was used to identify loading patterns within the CD-FAB. The scree plot and factors Eigen values >1 identified the most appropriate factor solution. Factor loadings >0.4 identified the clustering of items onto each factor (Kline, 1994).

Table 1

Criteria Used to Remove Items from the CD-FAB.

Spread of responses across options	High endorsement of a single item suggests poor discriminatory power. Items were considered for removal if >80% or <20% were an agree-type statement or a disagree-type statement	16 removed
Internal consistency	Items with a corrected item-domain total correlation <0.3 or in a domain with a poor Cronbach's alpha <0.7 were considered for removal	1 removed
Timing of administration of questionnaire	Needs to be applicable to people from the point of CD diagnosis onwards, so all individuals with CD can complete the scale	2 reworded
Clarity and relevance of items	Difficult to understand items were reworded or considered for removal	13 reworded
Items deemed theoretically important	These items were retained despite meeting the above criteria because they were deemed theoretically important	1 retained

5.4.3. Results of Item Analysis and Initial Exploratory Factor Analysis

One hundred and two individuals (96 females) completed the pilot stage (mean age = 38.6 years; SD = 16.7; 9.6 years with CD diagnosis; SD = 18.2). Twelve participants were excluded because they reported self-diagnosis and not a biopsy-diagnosis of CD.

5.4.4. Internal Reliability

The CD-FAB was reduced from 30 to 13 items based on the criteria described above. The Cronbach's Alpha for the total score was .88 indicating a good level of internal consistency. The Cronbach's alpha for each subscale was >.7 (see Table Two).

5.4.5. Exploratory Factor Analysis

A three-factor solution was extracted that explained 65.3% of the variance.

Factor one, "*Food Attitudes*" contained items describing concerns around interacting with food and cross contamination. Factor two, "*Fear Response*" contained items that described behaviours designed to control food preparation and a fear of trying new foods. Factor three, "*Adaptive Response*" described behaviours that allowed individuals to manage their food attitudes without compromising their lifestyle.

Table 2

Factor Loadings and Cronbach Alpha Coefficients for CD-FAB Factors

	Fear Response:	**Food Attitudes:**	**Adaptive Response:**
Cronbach's Alpha for Scale	**.788**	**.786**	**.790**
I am afraid to touch gluten-containing foods	.844		
I get concerned being near others when they are eating gluten	.806		
** I will happily prepare gluten for others*	.734		
I am afraid to eat outside my home	.516		
I find it hard to eat gluten-free foods that look like the gluten-containing-foods that have made me ill in the past		.808	
I have a lack of variety in my diet		.799	
I get worried when eating with strangers		.679	
My concerns about cross-contamination prevent me from going to socal events involving food		.548	
I will only eat food that I have prepared myself		.413	
** If I ask questions, I can normally find gluten-free food to eat*			.819
** I enjoy going out for meals as much as I did before my diagnosis*			.734
** Being contaminated by gluten in the past, hasn't stopped me from enjoying restaurants*			.692
** I am comfortable eating gluten-free food from other people's kitchens*			.586

* Represents items that are reverse scored. Numerical values represent factor loadings, a value>.4 is identified the clustering of the items onto each factor.

5.5. Study 3: Confirmatory Factor Analysis and Psychometrics

Study Three assessed the feasibility, reliability and psychometric properties of the CD-FAB, and validated the underlying factor structure.

5.5.1. Methods of Confirmatory Factor Analysis and Psychometrics

Recruitment posters in the food outlets and across campus at the University of Birmingham, UK, directed interested individuals to an online survey. Data collection occurred between January-March 2016. Individuals were asked not to complete the questionnaire if they had completed Study One. All participants completed the online questionnaire (time 1), and were invited to complete the CD-FAB and items assessing predictive validity for the second time, 4 weeks later (time 2). Two-hundred individuals with CD were targeted as this is a sufficient sample size for confirmatory factor analysis (Guilford, 1954). The inclusion/exclusion criteria were as described in Study One.

5.5.2. Data Analysis

Floor and ceiling effects were examined to assess feasibility of the total CD-FAB score and the subscales; these were considered when more than 15% of respondents achieved the lowest or highest possible score.

Confirmatory factor analysis (CFA) was used to confirm the model found in Study Two, based on the goodness fit and assessed using several indices: the comparative fit index (CFI; >.95 indicates acceptable fit), Tucker-Lewis Index (TLI; >.95 indicates acceptable fit) and root mean square errors of approximation (RMSEA; <.08 indicates acceptable fit; Hu & Bentler, 1999). Modification indices were examined and

modifications were made to improve model fit. Higher-order confirmatory factor analysis, with food attitudes and beliefs as the higher order factor, was conducted to explore whether the 3 CD-FAB factors could be combined to create a total score. Total CD-FAB scores were calculated by summing the responses on each item (responses for each item range between 1 and 7). Total CD-FAB scores ranged between 13 and 91, with higher scores indicating greater CD-related food attitudes and behaviours.

A minimum of 50 participants per time period is required to assess test-re-test reliability (Atkinson and Nevi, 2000; Hopkins, 2000). Correlation coefficients (r) were used to assess test-re-test reliability of the total CD-FAB scores, an r >.7 is indicative of strong reliability (Terwee et al., 2007).

5.5.3. Measures for Validation

Food Neophobia Scale (FNS; Pliner & Hobden, 1992*):* The FNS was used to assess anxieties around food in Chapter Three, so was used to assess convergent validity. The FNS measures willingness to try new food, with lower scores indicating a greater willingness to try new foods. The scale consists of 10 items and is the standard measure of food neophobia (Pliner & Hobden, 1992). Correlations were sought to determine the degree to which the CD-FAB reflected a fear of trying new foods. We anticipated a moderately positive relationship between CD-FAB and FNS scores, as individuals with high CD-FAB scores may also be fearful of trying new foods.

Depression, Anxiety, Stress scale 21 (DASS-21; Lovibond & Lovibond, 1995*):* The *Anxiety* subscale from the DASS-21 was used to assess convergent validity. This subscale measures behavioural feelings of anxiety over the last 4 weeks with higher

scores indicating greater anxiety. The DASS-21 has strong psychometric properties; with higher scores indicating greater anxiety (Brown et al., 1997). To demonstrate convergent validity, scores on the CD-FAB should correlate with scores on the *Anxiety* subscale.

CD Quality of Life scale (*CD-QoL*; Dorn et al., 2010): The *Treatment* subscale was used to assess discriminative validity. This subscale assesses satisfaction with one's treatment (the GFD). There is no reason for CD-FAB total scores to be associated with treatment beliefs, so no relationship between these scores was anticipated.

Behavioural Item: Known groups discriminant validity was assessed using the behavioural item, *"Do you consider yourself to be anxious around food?"* This item was rated yes/no. A further behavioural item "*How many times have you eaten outside the home over the last month?"* was assessed at time 2, to assess predictive validity. To show predictive validity, individuals who score high on the CD-FAB at time 1 will eat outside the home less than those scoring low on the CD-FAB at time 2.

Gluten-Free Management: Dietary management was rated on a 5-point Likert scale, in response to the question "*In general, how strictly do you maintain a GFD?*" *ranging from '1) All of the time'; 2) 'Most of the time'; 3) 'Some of the time'; 4) 'Now and then'; 5) 'Not at all'* (Ford, Howard & Oyebode, 2012).

5.5.4. Results of Confirmatory Factor Analysis and Psychometrics

Participants at time 1: 203 (35 males, 2 "other") participants took part in the validation stage with a mean age of 30.9 years (SD = 11.4) and 6.2 years with CD diagnosis (SD = 8.4). Nineteen participants were excluded as they reported a self-diagnosis and not a biopsy proven diagnosis of CD. This sample was older than the

participants recruited for study two (t(1, 285)=-4.2, p<.001). No difference was found in years since diagnosis across the two samples (t (1, 286)=-1.8, p=.072). 56.1% of participants with CD reported that they followed their GFD "all the time". Of the remainder, 1% were completely non-adherent and 42.9% were partially adherent to the GFD.

Participants at time 2: 112 of those recruited at time 1 consented to be contacted at time 2, of these 67 completed the second questionnaire. This sample consisted of 13 males and 54 females with a mean age of 32.8 years (SD=16.5) and these individuals had been diagnosed for a mean of 7.5 years (SD=11.4).

When comparing participant contact details to those participants recruited in Study One, there was only a 3% overlap in participants across the samples. We can be confident that the participants in the current sample were different to those recruited in the original sample. These individuals were removed from the analysis. Missing data for each of the items ranged from 1.4–4.1% and was missing at random. Participants with missing data were removed from the analysis.

5.5.5. Reliability and Feasibility

Floor and ceiling effects ranged between 0.5-1% across all CD-FAB subscales (Table Three). This indicates strong feasibility across the CD-FAB. As demonstrated in Study Two, the CD-FAB subscales and total score showed good internal consistency with Cronbach's Alpha above 0.7 in all cases.

Table 3

Floor and Ceiling Effects for Total CD-FAB Scores and Subscales with Cronbach Alpha Coefficients

Subscales	**Mean**	**SD**	**Cronbach's Alpha**	**Floor (%)**	**Ceiling (%)**
Food Attitudes	16.70	4.45	.805	0.5	0.5
Fear Response	19.59	7.53	.701	1	0.5
Adaptive Response	15.66	5.77	.833	1	1
CD-FAB Total	20.57	15.70	.887	0.5	0.5

5.5.6. Confirmatory Factor Analysis

All items loaded onto their respective factors (standardised loadings ranged between 0.4-1.1 and are all statistically significant ($p<.01$); see Figure Two). Figure Two shows the structural equation model containing the standardised path estimates between the items and factors for the final model. Items 4 (*I have a lack of variety in my diet*) and 9 (*I will happily prepare gluten for others*) were removed from the model due to low factor loadings and improved model fit after removal. Despite item 6 (*I find it hard to eat gluten-free foods that look like the gluten-containing-foods that have made me ill in the past*) having a low factor loading (0.4), this item was retained, as its removal did not improve model fit. The path coefficients represent the direct structural relationship between each factor and its indicators. The correlation between the three factors of the CD-FAB ranged between -.78 and 1.06, suggesting a high level of affinity between the three factors, even though they tap into distinct underlying constructs. An examination of the modification indices

indicated that fit could be improved by co-varying the errors on items 7 and 8, 1 and 3, and 5 and 7. The resulting model fit was good (TLI=.97; CFI=.98; RMSEA=.06). Total CD-FAB scores and subscales were based on this CFA, meaning items 4 and 5 were removed from analyses. Subsequently, the resulting CD-FAB contained 11 items with total scores ranging from 11 to 77. These calculations were used in subsequent analyses.

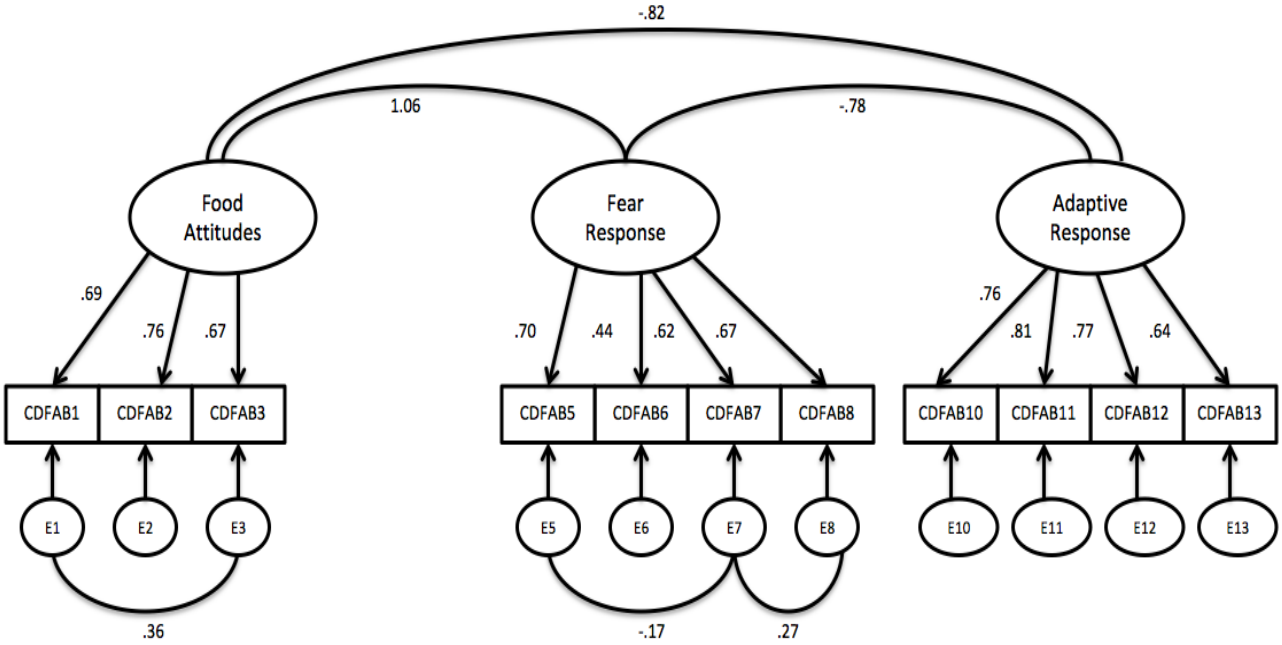

Figure 2: Confirmatory Factor Analysis with standardised Item loadings onto each Factor for the three factors of the CD-FAB. The numbers shown on the diagram from bottom to top are: 1) covariance of the errors, 2) error terms (E), 3) path coefficients of indicators and 4) correlations between the three factors.

5.5.7. Higher Order Factor Analysis

The three factor model (*Food Attitudes, Adaptive Response and Fear Response*) with food attitudes and beliefs (total CD-FAB score) as the second order factor found a good model fit (TLI=.97; CFI=.98; RMSEA=.05).

5.5.8. Content Validity

Convergent Validity: As hypothesised, the total CD-FAB positively correlated with the FNS (r=.25, p<.001) and the *Anxiety* subscale of the DASS-21 (r=.16, p=.025). This indicates that the CD-FAB is similar to measures of anxiety and fear around trying new foods. The relationship between the total CD-FAB score and the *Anxiety* subscale of the DASS-21 was significant but weak.

Discriminant Validity: As hypothesised, beliefs about the effectiveness of the GFD were not related to total CD-FAB scores (r=-.01, p=.880), indicating good discriminant validity.

Known Groups Validity: Overall, 37.2% of participants considered themselves to feel anxious around food. In CD-FAB subscales and total score, those that reported being anxious around food had significantly higher CD-FAB scores than those who were not anxious around food (see Table Four). However, those who reported being more anxious around food had significantly lower scores on the *Adaptive Response* subscale.

Table 4

Means and standard deviations for CD-FAB total and subscales (with reverse scoring for Adaptive Response subscale) for CD patients with and without food anxiety, in response to the item "*Do you consider yourself to be anxious around food?*"

Subscales	*"Do you consider yourself to be anxious around food?"*		P-Value
	Yes; Mean (SD)	*No; Mean (SD)*	
Food Attitudes	16.6 (3.5)	10.1 (4.4)	<.001
Fear Response	18.9 (4.3)	13.0 (4.9)	<.001
Adaptive Response	12.5 (4.9)	17.4 (5.5)	<.001
CD-FAB Total	48.0 (6.4)	40.6 (7.0)	<.001

5.5.9. Predictive Validity

Total CD-FAB scores taken at time 1 were associated with responses to the item "*How many times have you eaten outside the home over the last month?"* taken at time 2 (r=-.36,p=.048), and individual CD-FAB subscale scores showed correlations with this item (p<.05). The *Adaptive Response* subscale showed significant correlations in the opposite direction. High scores on the *Adaptive Response* subscale, indicating healthy food attitudes and behaviours, were associated with a higher number of times eaten outside the home over the last month, taken at time 2 (r=.36, p=.047).

5.5.10. Test-Re-Test Reliability

Correlation coefficients between the total CD-FAB scores at time 1 and time 2 were strong (r=.92, p<.001). Similar results were found for all three CD-FAB subscales. The CD-FAB and its subscales show good test-re-test reliability.

5.6. General Discussion

Recent research has highlighted the potential for disordered eating to develop in individuals with CD (Arigo et al., 2012; Karwautz et al., 2008; Chapter Three). Qualitative studies suggest that existing measures of disordered eating do not identify all atypical eating patterns reported in CD (Chapter Four). These questionnaires do not consider factors related to CD that are essential in managing the GFD that may become maladaptive, including a hypervigilance around cross-contamination of food with gluten. Although vigilance around food is essential in CD, a hypervigilance around food may contribute to the development of disordered eating attitudes and behaviours. Beliefs around cross-contamination and food safety have been implicated in the development of disordered eating attitudes and behaviours in CD (Chapter Four) but there are no specific measurement tools available. Here we developed and validated a self-report food attitudes and behaviours measure for adults with CD (the CD-FAB).

The CD-FAB set out to measure the four themes identified in the focus group, which explored underlying food attitudes, concerns and eating behaviour themes (i.e. *handling of food, trust, risk-taking and food safety*). However, only three factors emerged, but items targeting each of the themes were distributed randomly across the three factors. These factors explained 65.3% of the variance: *Food Attitudes,*

describing the beliefs one has around food and cross-contamination; *Fear Response,* which describes avoidance behaviours and changes in the diet to cope with these food attitudes; and *Adaptive Response,* describing techniques to obtain nutritional information to allow food consumption in a range of environments.

Food cross-contamination and eating outside the home are important concerns for people with CD (Sverker et al., 2005), and these are described by the items in the *Food Attitudes* subscale. The *Adaptive Response* and *Fear Response* subscales reflect two differing strategies to cope with food attitudes. These distinctions mirror the "depressive-anxious" (those who respond to CD with fear, sadness and anger) or "passive-adaptive" (those who have become accustomed to their CD) responses described by Ciacci et al., (2002).

The CD-FAB shows strong psychometric properties with high Cronbach's alphas for all subscales (>.7) and good predictive validity. High inter-factor correlations and similar relationships with the validation measures indicate that the three factors are not independent but the scree plot and Eigenvalues indicated a three-factor solution that was supported by a CFA. The CD-FAB had excellent test-re test reliability over 4 weeks. This may indicate that CD food attitudes and behaviours are a stable trait, supporting previous literature highlighting this issue (Sverker et al., 2005). Additionally, the CD-FAB has good discriminant validity; it is able to identify subtypes of individuals with CD and shows no correlation with the CD-QoL *Treatment* subscale. The direction and magnitude of the correlations between the CD-FAB and the FNS and *Anxiety* subscale of the DASS-21 indicate good convergent validity and moderate correlations with the FNS and *Anxiety* subscales indicate that the CD-FAB is measuring a construct similar to, but unique from food neophobia and anxiety.

A strength of this study lies in the use of focus groups and participant feedback to create the CD-FAB. This information, alongside *a priori themes*, including the framework developed in Chapter Two, was used to make these items relevant to participant experiences. Furthermore, constructs identified by respondents as relevant to their experience, related to social settings, gastrointestinal symptoms and eating behaviours are measured for the first time by the CD-FAB. Pertinent examples of this are that eating at social events is less enjoyable after a CD diagnosis as the GFD can lead to feelings of embarrassment, isolation and a fear of gastrointestinal symptoms and for some, this fear of symptoms and anxiety around food may lead to disordered eating attitudes and behaviours (Chapter Four).

Despite the strengths of the current study, future research needs to examine long-term changes in CD-FAB scores over time, particularly during the first year of diagnosis when the GFD is being initiated, to explore adaptation to CD and the GFD, and the stability of self-management behaviours. In addition, although we included a broad range of individuals with self-reported biopsy proven CD, it is not clear how these results will generalise to those with an objective biopsy confirmed diagnosis. Furthermore, participants in the developmental stages of the CD-FAB were predominantly female. This is representative of the CD population, as diagnosis is two times more common in females than in males (West et al., 2014); however further validation of the CD-FAB is needed in males with CD (Ciacci et al., 2009). Finally, expert review was not used to assess content validity, but the involvement of service users and individuals with CD in generating the items and providing feedback on the overall CD-FAB provides evidence for content validity. Future research should

consult with healthcare professionals working alongside individuals with CD, to fully assess the content validity of the CD-FAB.

These limitations do not detract from the clinical utility of the CD-FAB for assessing those with disordered eating attitudes and behaviours related to a CD diagnosis. The instrument may be used as an outcome measure in clinical research, to evaluate the effectiveness of interventions on eating patterns, and enables eating behaviours to be evaluated in clinical practice to identify people requiring additional support. This will allow a broader understanding of the impact of a CD diagnosis on eating attitudes and behaviours. Given the potential health implications of these attitudes and behaviours, future research should focus on the psychological and physical consequences of high CD-FAB scores. Furthermore, there is a need to establish the responsiveness of the questionnaire to detect the minimal important change and establish clinical cut-off points by calculating receiver operating characteristics curves in larger samples before recommending its use in clinical settings. Further guidance regarding the interpretation of CD-FAB scores (e.g. referral to dietician, clinical psychologist or specialist eating disorders service) can be given following the identification of population norms and health implications.

In summary, the CD-FAB is a brief, self-report questionnaire that shows good reliability and validity in measuring disordered eating attitudes and behaviours in CD. The measure may be a useful tool for clinical practice to help understand eating attitudes and behaviours in adults with CD.

CHAPTER SIX: FACTORS ASSOCIATED WITH SCORES ON THE CD FOOD ATTITUDES AND BEHAVIOURS SCALE

6.0. Chapter Rationale

Chapter Five described the development and validation of the CD Food Attitudes and Behaviours scale (CD-FAB), a tool designed to understand disordered eating in the context of CD that is not detected by current tools of disordered eating. The CD-FAB showed strong reliability and validity; however, at present, it is not clear how scores on the CD-FAB are related to physical and psychosocial wellbeing.

The aim of this chapter was to explore the associations between CD-FAB scores and physical and psychosocial outcomes to gain a greater understanding of the influence of disordered eating attitudes and behaviours in CD. This chapter used cross-sectional survey techniques to explore the correlates of CD-FAB scores. In addition, this chapter aimed to replicate the results of Chapter Three in a second sample of individuals with CD.

6.1. Introduction

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* developed in Chapter Two, proposes two pathways that explain the development of disordered eating in gastrointestinal disease. The first pathway describes individuals who struggle with weight changes experienced after diagnosis. In terms of CD, individuals may associate weight change (commonly weight gain) with the GFD, and aim to reduce their weight gain through poor dietary self-management (Leffler et al., 2007). The second pathway describes those who experience extreme anxiety around unfamiliar foods and overestimate the negative consequences associated with their condition. These individuals may fear food prepared outside of their control, and cope with this by restricting food intake (Chapter Four).

The EAT-26 and the BES are effective in identifying disordered eating attitudes and behaviours in CD that are associated with psychological distress, dietary management and gastrointestinal symptoms (Arigo et al., 2012; Karwautz et al., 2008; Chapter Three). However, qualitative studies suggest that these tools do not identify all disordered eating attitudes and behaviours found in CD (Chapter Four). Some individuals with CD describe fears when eating outside the home, difficulties trusting others to prepare their food and difficulties not having control over the food preparation process, and cope with this by limiting their food intake (Chapter Four). Concern and vigilance around food may result in a unique type of disordered eating specific to those with CD and are similar to the beliefs and behaviours described in the second pathway of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*. There is a need to understand these types of disordered

eating in CD and explore whether this may result in CD-specific disordered eating attitudes and beliefs.

The *CD Food Attitudes and Behaviours* scale (CD-FAB; Chapter Five) was developed to screen for these disordered eating attitudes and behaviours related to food concerns and concern around cross-contamination. Those who score high on the CD-FAB may display a hypervigilance around food and limit food intake, not for weight loss but because they believe that food is dangerous to their health and may encourage gastrointestinal symptoms (Chapter Four). Vigilance around food and limiting food intake when there is a high risk of gluten cross-contamination is essential for individuals with CD (Collin et al., 2004) but hypervigilance can become maladaptive and may negatively impact physical and psychosocial well-being (Pilowsky, 1978).

Negative physical effects may occur in individuals who score high on the CD-FAB, as restricted diets tend to involve the exclusion of important foods groups, such as bread and pasta, which may result in a calorie deficit or a poor nutritional profile (Misra et al., 2006). In addition, the planning and isolation that may result from this hyper-vigilance around food may be associated with a limited social life, increased anxiety, depression and impaired quality of life (See et al., 2015; Chapter Four). Control around food may lead some individuals to limit food intake in certain settings rather than eating food that has been prepared by others (Chapter Four). As well as examining the physical and psychosocial correlates of the CD-FAB, we hoped to extend the discriminative validity of the CD-FAB. For this tool to be clinically useful, it needs to differ from current disordered eating tools. High scores on the CD-FAB may describe behaviours, such as food preoccupation and dietary restriction, so

some overlap with other measures of disordered eating such as the EAT-26 may be expected. However, the CD-FAB also identifies concerns around food and cross-contamination that are specific to CD; although some overlap in disordered eating tools may be expected, the CD-FAB should identify a pattern of behaviour that is distinct from established tools.

In the only evaluation of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Three) a combination of CD-specific factors (dietary management and gastrointestinal symptoms) and non-specific factors (psychological distress) explained a significant proportion of disordered eating scores in CD. A subsequent cluster analysis produced three typologies of disordered eating in CD, a "low risk" group, a "critical" group and a "high distress" group. The "critical group" contained individuals who had difficulty managing their GFD, greater current gastrointestinal symptoms and symptoms of Anorexia and Bulimia Nervosa, as assessed by the Eating Attitudes Test (EAT-26; Garner, Olmsted, Bohr & Garfinkel, 1992). The "high distress" group contained individuals who scored high on binge eating measures, as assessed by the Binge Eating scale (BES; Gormally, Black, Daston & Rardin, 1982), and psychological distress. The data from Chapter Three provides some support for the notion of poor dietary management described in pathway one of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.* Although a significant step forward in the CD and disordered eating literature, Chapter Three used online recruitment strategies from online support forums, potentially resulting in sample bias. In addition, these findings drop in clinical utility unless they can be observed in a second, independent study.

The present study examines the physical and psychosocial correlates of CD-FAB scores. Furthermore, we hoped to further examine the discriminative validity of the CD-FAB by comparing it to the EAT-26 and the BES. Given the limitations of previous research, an additional aim was to find out whether the findings of Chapter Three would replicate in a new sample of individuals with CD recruited without the use of the Internet. We anticipated that high CD-FAB scores would be associated with impaired physical and psychosocial well-being and the CD-FAB will show modest correlations with the EAT-26 but no association with the BES.

6.2. Method

The methods used were similar to those described in Chapter Three. Briefly, this cross-sectional survey was conducted between January and February 2016. Females living in the United Kingdom aged between 18 and 69 years and who self-reported a physician-confirmed diagnosis of CD were eligible to participate. Participants were excluded if 1) they reported having a dietary-controlled condition in addition to CD (e.g. cystic fibrosis, type one diabetes); 2) they did not have a self-reported biopsy confirmed diagnosis for their condition; and 3) if they had any other food allergies.

Chapter Three recruited individuals from online support forums whereas in the present study, participants were recruited from the University of Birmingham campus. This method was selected to avoid the potential biases of the previous study, related to online recruitment. Adverts were distributed across the University of Birmingham campus food outlets. Participants were asked to contact other individuals with CD who may be interested in taking part in the research, by

distributing the participant information sheet. Interested individuals were directed to an online survey, hosted by Qualtrics, to complete the questionnaires.

6.2.1. Measures

All of the measures and procedures used in this study are described in Chapter Three. Participants completed the Depression Anxiety Stress 21 scale (DASS-21; Lovibond & Lovibond, 1995), the Food Neophobia Scale (FNS; Pliner & Hobden, 1992), the EAT-26 (Garner, Olmsted, Bohr & Garfinkel, 1992) and the BES (Gormally, Black, Daston & Rardin, 1982). Gastrointestinal symptoms were assessed using the *Identity* subscale of the Illness Perception Questionnaire Revised (IPQ-R; Moss-Morris et al., 2002), dietary-management was assessed on a 5-point Likert scale, in response to the question "*In general, how strictly do you maintain a gluten free diet?*" *ranging from 1) 'All of the time'; 2) 'Most of the time'; 3) 'Some of the time'; 4) 'Now and then'; 5) 'Not at all'* (Ford, Howard & Oyebode, 2012). The only exceptions were the addition of the newly developed CD-FAB (Chapter Five) and the CD Quality of Life scale (Dorn et al., 2010), which are detailed below.

The CD Food Attitudes and Behaviour scale(CD-FAB; Chapter Five): The CD-FAB is a CD-specific measure that assesses beliefs and concerns around food and food environments. It consists of 11 items, scored on a 7-point Likert scale. The CD-FAB has good reliability and validity (Chapter Five) and is formed of three subscales: *Food Attitudes, Fear Response* and *Adaptive Response.*

The CD Quality of Life Scale (CD-QoL; Dorn et al., 2010): The CD-QoL assesses quality of life across four clinically relevant subscales (*CD-related limitations*, *dysphoria*, *health concerns* and *inadequate treatment*). The scale consists of 20

items, with higher scores indicating greater quality of life (maximum score is 100). There is no defined clinical cut-off for this scale.

6.2.2. Ethical Approval

Ethical approval was granted by the Psychology Research Ethics Committee, University of Birmingham.

6.2.3. Statistical Analysis

Data were analysed using the Statistics Package for the Social Sciences (SPSS) version 22.0. Cronbach's alphas were calculated for all scales and all measures exceeded the acceptable minimum of 0.7 (DeVelis, 2003). Missing data for each of the items ranged from 2.4–3.1% and was missing at random. Participants with missing data were removed from the analysis.

A tertiary split was applied to the CD-FAB responses received in this dataset, to divide individuals into high, medium and low scorers based on the 33rd and 66th percentiles. By using a tertiary split, we were able to explore differences in outcomes between high and low CD-FAB scores whilst avoiding noise within the data from participants that scored close to the high/low cut-off. Analysis of variance was used to compare physical and psychosocial outcomes across the three groups and t-tests were used to compare the means across the low and high scorers on the CD-FAB.

To replicate the results of Chapter Three, independent t-tests were used to compare the studies in terms of participant characteristics. The regression analyses used in Chapter Three were repeated. To test the stability of the cluster analysis, Blashfield and Macintyre's (1980) method was used. This procedure performs the cluster analysis using the same rules and parameters from the original cluster

analysis (Chapter Three) and applies these rules to the new data set. The results from the original sample are used to classify the data in the current sample. The cluster centres from the second sample are then compared to the original sample and the Cohen's kappa coefficient is calculated to measure the agreement between the clusters.

6.3. Results

6.3.1. Participant Characteristics

The entire sample consisted of 166 women, with a self-reported biopsy diagnosis of CD (see Table One). The mean age was 30.1 years (SD=11.0; range =18-68), and participants had a mean of 6.1 years with a CD diagnosis (SD=8.2; range =2-61). Participants in the current sample were younger ($p<.001$) and had CD for fewer years ($p=.03$) when compared to the original study sample (Chapter Three).

Inspection of participant contact details revealed that 7% of participants who participated in Study 1 also participated in the present study. These individuals were excluded from the analysis (n=10).

6.3.2. Phase One: Impact of CD-FAB Scores

Demographic, psychosocial and physical outcomes were compared across CD-FAB scores using an ANOVA (see Table One).

High scorers had greater psychological distress, fewer years with CD and a more impaired quality of life across a variety of domains ($p < 0.003$). The low scorers scored in the "normal" ranges for DASS-21, whereas the medium to high scorers scored within the "mild" and "moderate" ranges (Lovibond & Lovibond, 1995).

Compared to the low scorers, the medium scorers had fewer years with diagnosis, increased psychological distress, a greater fear of trying new foods and impaired quality of life. No significant differences were found between the medium and high scorers, and no differences were found across the physical outcomes (gastrointestinal symptoms, dietary management). Notably, although the relationship between EAT-26 scores and CD-FAB scores was not significant, the trend was approaching significance with higher CD-FAB scorers having higher EAT-26 scores ($F=2.8$, $p=.006$) suggesting that a similar but unique construct in being assessed by the CD-FAB, whereas BES scores did not differ according to CD-FAB scores ($F=.75$, $p=.48$).

Table 1

Demographic, psychosocial and Physical Outcomes Using the Tertiary Split on the CD-FAB. Data are presented as means.

	Low Scorers	**Medium Scorers**	**High Scorers**	**F Statistic**
Demographic Outcomes				
Age (years)	32.3	29.0	29.0	1.75
BMI	22.6	22.7	21.9	.29
Years with CD	8.5	5.0a	5.0[b]	3.69*
Psychosocial Outcomes				
Depression	8.6	13.5[a]	13.5[b]	3.81*
Anxiety	6.6	10.1[a]	11.6[b]	4.34*
Stress	11.6	16.9[a]	15.8 [b]	4.13*
Total DASS-21	26.7	40.5[a]	40.9[b]	5.3*
EAT-26	9.1	13.9	13.9	2.80
BES	10.8	11.9	12.8	.75
Food Neophobia	27.4	31.3[a]	33.9[b]	10.7*
Total Quality of Life	71.1	57.9[a]	53.6 [b]	16.03*
Limitations	31.4	24.4[a]	21.9[b]	17.32*
Health	17.1	13.6[a]	12.4 [b]	10.92*
Treatment	5.5	5.5	5.5	.032
Dysphoria	17.1	14.6[a]	14.2 [b]	7.12*
Physical Outcomes				
GI Symptoms	1.6	1.8	1.8	.27
Dietary Management	1.6	1.7	1.6	.65

*p=.003 for ANOVA across all three groups; [a]p=.003 for T-Test across low and medium scorers; [b]p=.003 for T-Test across low and high CD-FAB scorers.

6.3.3. Phase Two: The Prevalence and Characteristics Associated with Disordered Eating in Coeliac Disease; Replication of Chapter Three

6.3.4. Prevalence of Disordered Eating in Coeliac Disease

Table Two reports the proportion of participants scoring above the clinical cut-off for the EAT-26 and the BES and the mean total scores for the current study and the original study reported in Chapter Three for comparison. No difference in disordered eating scores across the two samples was found ($p>.05$), however, the distributions of EAT-26 and BES scores were positively skewed whereas the original study described a normal distribution. The decision was made to continue with the parametric analyses conducted in the original study, as the distribution of the residual errors was normal.

Table 2

Mean Scores and Percentage scoring above the clinical cut-offs for measures of disordered eating for the initial and current samples

Measure	Current Sample	Initial Sample	T-Test
Eating Attitudes Test (>20)	12.3 (17.7%)	11.1 (15.7%)	-.91
Binge Eating Scale (>17)	11.8 (21.6%)	11.2 (19.4%)	-.47

The number in brackets represents the percentage of participants scoring above the pre-determined clinical cut-offs for the BES and EAT-26.

6.3.5. Factors Associated with EAT-26 and BES scores

Table Three shows the results for the hierarchical regressions for both EAT-26 and BES scores from the current study. When predicting EAT-26 score, collectively

this model accounted for 43.5% of the variance in EAT-26 scores ($F=(8,155)=14.1$, $p<.001$; see Table Three) with dietary-management having a significant regression weight. Based on the examination of ß weights, gastrointestinal symptoms and dietary management had the major contribution to the model.

When predicting BES score, collectively the disease non-specific factors model accounted for 45.5% of the variance in BES scores ($F=(6,147)=19.6$, $p<.001$; see Table Three) with age, depression and stress having significant regression weights. The addition of disease-specific factors only explained an additional 3% of the variance, with no significant change in R Square.

Table 3

Disease specific and Non-Specific Factors in Predicting EAT-26 and BES Scores in CD

	Eating Attitudes Test				Binge Eating Scale			
Predictors	**B**	***B***	***R^2***	**F**	**B**	***B***	***R^2***	**F**
Non-specific Factors								
Age	*-.04*	*-.04*			*-.23*	*-.29**		
Body Mass Index	*.04*	*.02*			*.30*	*.19**		
Years with Condition	*.01*	*.01*			*.09*	*.09*		
Depression	*.37*	*.16*			*.53*	*.35**		
Anxiety	*.74*	*.24**			*-.18*	*-.11*		
Stress	*.51*	*.22*	*.34*	*12.68**	*.50*	*.32**	*.46*	*19.61**
Disease Specific Factors								
Age	*-.03*	*-.03*			*-.22*	*-.28**		
Body Mass Index	*.01*	*.02*			*.29*	*.18**		
Years with Condition	*.02*	*.02*			*.10*	*.11*		
Depression	*.39*	*.17*			*.54*	*.36**		
Anxiety	*.56*	*.21*			*-.24*	*-.14*		
Stress	*.50*	*.21*			*.49*	*.31**		
Gastrointestinal Symptoms	*1.55*	*1.8**			*.44*	*.09*		
Dietary-management	*3.39*	*.23**	*.44*	*14.13**	*1.41*	*.14*	*.49*	*16.35**

* Significance at $p < 0.008$. The significance of the F value refers to the F associated with each step.

6.3.6. Cluster Analysis

Conducting a replication analysis using Blashfied and Macintyres (1980) method produced clusters that were similar to the original cluster analysis reported in Chapter Three. Cohen's kappa coefficient was .42 ($p<.001$), suggesting moderate agreement between the current and the original cluster analysis.

6.4. Discussion

Individuals with CD frequently report concerns around food and cross-contamination of food products which may lead to atypical food attitudes and behaviours (Chapter Two; Chapter Four). Recently, the CD-FAB has been created to assess these food attitudes and behaviours in CD (Chapter Five). The primary goal of this chapter was to explore the correlates of CD related food attitudes and beliefs, as assessed by the CD-FAB. Our second aim was to replicate the results of Chapter Three in a new sample of individuals with CD.

This study used the CD-QoL, DASS-21 and measures of gastrointestinal symptoms and GFD management to explore the physical and psychosocial correlates of CD-FAB scores. Quality of life was affected across a variety of domains in individuals with high CD-FAB scores. Individuals above the 66th percentile of CD-FAB scores felt more socially limited by their CD, felt more concerned about their CD and were more concerned about the health consequences of CD compared to those below the 33rd percentile. The increased concern around food may result from concerns around one's health and a fear of not managing the GFD. In addition, Hauser et al. (2010) found that anxiety was lower in individuals who lived alone, suggesting that certain social environments, such as eating with other people, may be perceived as a burden

for those with CD, which may provide one explanation for the perceived social limitations in the CD-FAB group with the highest scores.

In addition, high CD-FAB scorers were more distressed, with individuals scoring in the “mild” and “moderate” severity categories on the DASS-21 (Lovibond & Lovibond, 1995) and had fewer years with CD. Those scoring low on the CD-FAB were in the “normal” ranges for the DASS-21. These findings are consistent with research suggesting that anxiety is greater in the first few years following a CD diagnosis when an individual is adjusting to the GFD (Addolorato et al., 2001). High scores on the CD-FAB indicate impaired psychosocial well-being but the interactions between these factors and years with a CD diagnosis are not clear.

Self-reported gastrointestinal symptoms and GFD management were used to assess the physical impact of high CD-FAB scores. The number of gastrointestinal symptoms did not vary based on CD-FAB score and neither did self-reported GFD management. This is unsurprising, as according to the *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* individuals with high CD-FAB scores should show strict management of the GFD, resulting in fewer gastrointestinal symptoms (Chapter Four).

This research has added to the discriminative validity of the CD-FAB and suggests this tool does differ from established measures of disordered eating (the EAT-26 and BES). There was a weak but positive association between the CD-FAB and EAT-26, suggesting that the CD-FAB is measuring a construct similar to, but unique from disordered eating as assessed by the EAT-26. The EAT-26 contains items that assess a preoccupation around food and dietary restriction, so the small overlap between the CD-FAB and the EAT-26 makes theoretical sense. The CD-FAB was not associated

with BES scores, which contains items regarding over eating and a lack of control around food intake, highlighting the discriminant validity between these two questionnaires. Furthermore, when the CD-FAB was used as an outcome variable, the predictors differed from those that were important in predicting BES and EAT-26 scores. Increased food neophobia was an important correlate of greater CD-FAB scores. This implies that those who score high on the CD-FAB are fearful around new foods; this reflects some of the beliefs presented in the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). These results are also in line with the qualitative study presented in Chapter Four, which suggests that individuals who are fearful of cross contamination will restrict their food intake in certain environments to manage these fears. Further research should focus on how these concerns around food affect actual eating behaviours.

In line with previous research, we found that 11-12% of individuals with CD scored above the clinical cut-offs for disordered eating according to the EAT-26 and BES (Chapter Three; Arigo, Anskis & Smyth 2012; Karwautz et al., 2008). We previously observed that factors related to the CD diagnosis (gastrointestinal symptoms and dietary management) were associated with the development of anorexic and bulimic attitudes and behaviours, whereas the nonspecific burden of a chronic health condition was associated with the development of binge eating behaviours in CD (Chapter Three). In line with these findings, we found that nonspecific factors were associated with the presence of binge eating behaviours, and gastrointestinal symptoms and dietary management were associated with anorexic and bulimic attitudes and behaviours. In addition, we successfully

replicated the cluster analysis reported in Chapter Three, in a new sample of individuals with CD.

6.4.1. Limitations and Future Research

The limitations reported in the original study (Chapter Three) are relevant to this study. Because of the cross-sectional nature of this design, we cannot determine the direction of causality between disordered eating patterns and other variables assessed. As in the original study, we relied on a self-reported biopsy confirmed diagnosis of CD. Future research should replicate this findings in a physician-reported biopsy-confirmed sample of individuals with CD and should focus on using more objective measures of dietary-management such as anti-tissue transglutaminase assays, questionnaires designed to assess gluten-free dietary management (Leffler et al., 2009) and multi-modal approaches, including self-report and dietician assessment. The use of CD serology can enhance this type of research by assessing whether dietary management is improving or worsening over time, in combination with self-reported dietary management, psychosocial outcomes and disordered eating measures (Ho, 2012). However, what is striking and consistent across these two studies is the differing predictors for both EAT-26 scores and BES scores.

High scores on the CD-FAB were associated with self-reported impaired quality of life and increased psychosocial impairment, indicating that individuals with increased CD-FAB scores are struggling with their CD. Although the physical impact of CD-FAB scores was assessed through dietary management and gastrointestinal symptoms, the physical outcome variables were limited. Impaired physical

consequences may result from the restrictive diets employed by those with high CD-FAB scores; for example, these individuals may exclude certain types of food, preferring to only eat those foods that are deemed as safe (Chapter Four). This type of eating pattern may be associated with a poor nutritional profile, potentially resulting in anaemia and osteoporosis (Misra et al., 2006). Future research should examine these correlates in relation to the CD-FAB.

6.4.2. Clinical Implications

The observation that individuals with high CD-FAB scores have an impaired self-reported quality of life and psychosocial well-being suggests that the use of the CD-FAB is valuable in this population. More specifically, the observation that years with diagnosis, food neophobia and reported social limitations were associated with CD-FAB scores indicates that individuals reporting limited dietary choices and those who are newly diagnosed may benefit from having dietician support, including assessment using the CD-FAB, to explore food attitudes and behaviours in relation to their CD diagnosis.

The replication of the cluster analysis demonstrates consistent subtypes of eating pathology in individuals with CD. A focus on subgroups of people may allow a more efficient way of targeting healthcare and health resources; assessment and intervention around eating attitudes and behaviours within the context of CD may be essential in supporting psychosocial health. The replication of the cluster analysis indicates that there are three groups of individuals with CD: the low risk group who manage their CD well and have adaptive eating patterns, the high distress group who have problems with psychological distress and binge eating and the critical group

who score high on measures of anorexic and bulimic attitudes, and describe more difficulties in managing their GFD. Additionally, the CD-FAB appears to identify a group of individuals who experience concern around food. Although the traditional eating disorder measures such as the EAT-26 and the BES are useful in identifying disordered eating in CD, the CD-FAB may be a useful tool to understand eating concerns in the context of CD. Clinically, the CD-FAB may be used by dieticians to aid in the understanding of the beliefs around food and how these are related to eating behaviours in CD.

6.5. Conclusions

Research into the relationship between CD management and food attitudes and behaviours is becoming increasingly important. The results of the present study indicate that negative food attitudes and behaviours specifically related to CD are associated with impaired quality of life and psychosocial well-being. Future research should focus on understanding this sub-group of individuals with CD and look at ways to identify them and provide support.

CHAPTER SEVEN: BEHAVIOURAL CORRELATES OF THE COELIAC DISEASE FOOD ATTITUDES AND BEHAVIOURS QUESTIONNAIRE

7.0. Chapter Rationale

Chapter Six assessed the psychosocial and physical correlates of the CD-FAB. The results from Chapter Six and the qualitative results from Chapter Four provide support for the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*. However, both of these studies were based on self-report measures of disordered eating. At present, it is not clear whether CD-FAB scores are related to actual food intake.

The aim of this chapter, therefore, was to explore the associations between CD-FAB scores, food intake and the processing of food-related information to explore the behavioural validity of the CD-FAB. Secondly, we hoped to replicate our previous findings by exploring the associations between the CD-FAB and psychosocial outcomes, using self-report questionnaires in a biopsy-confirmed sample of individuals with CD. This chapter utilised a laboratory-based experiment in order to evaluate the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.*

7.1. Introduction

To avoid the consumption of gluten, individuals with CD need to be vigilant around the food they consume (Remes-Troche et al., 2006). However, according to the *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* for some individuals with CD, hypervigilance can create concerns around food that are associated with disordered eating attitudes and behaviours (Chapter Four, Chapter Six). The *Coeliac Disease Food Attitudes and Behaviours* scale (CD-FAB; Chapter Five) has been designed to screen for these food concerns that contribute to disordered eating attitudes and behaviours.

Individuals with CD, who report increased vigilance and concern around food, discuss these concerns in combination with dietary restriction (Chapter Four). These concerns and dietary-restriction are reported in unfamiliar settings or when an unknown individual is preparing food, in an attempt to maintain strict adherence to the GFD and prevent gastrointestinal symptoms (Chapter Four). High scores on the CD-FAB have been associated with a fear of trying new foods and an impaired quality of life, particularly in social domains (Chapter Six). The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* assumed that vigilance around food might also contribute to disordered eating attitudes and behaviours in CD. Biased information processing of food may reinforce concerns about food content and preparation, resulting in concerns which contribute to disordered eating attitudes and behaviours.

The measurement of eating behaviours and vigilance to food in CD has come from self-report measures. These self-report measures assume that individuals can accurately assess their own eating behaviours and tell us little about actual food

consumption and the behavioural validity of the CD-FAB. Additionally, the CD-FAB has only been validated in those with a self-reported diagnosis of CD (Chapter Six); there is need for replication in a sample with a biopsy-confirmed diagnosis of CD to extend the validity of the CD-FAB in this population. One way to address the limitations surrounding self-report measures is to measure food intake and food vigilance behaviourally. To examine the behavioural correlates of the CD-FAB we used a taste test to assess food intake and the dot-probe task to assess attentional bias towards food images.

Taste-test paradigms have been widely used to examine food consumption in the laboratory (Healtherton, Herman & Polivy, 1991; Polivy & Herman 1991). Vartanian at el. (2013) described a modified version of the taste test paradigm, whereby participants are provided with three types of cookie and are asked to rate each of the cookies, taking a sip of water between each tasting. Participants were left alone, with the cookies, for ten minutes. Participants were told to help themselves to more cookies whilst waiting for the experimenter to return, as any left over cookies will be thrown away, to encourage consumption of the cookies. Although the task is presented as a taste test, the aim is to measure food consumption. This task has been used to assess the influence of social models and portion sizes on food intake but has not been used in individuals with CD (Robinson et al., 2016; Vartanian et al., 2013). Using this procedure in CD allows us to test food consumption and dietary restriction in an unfamiliar environment with unknown individuals (the researchers) presenting the food, creating further anxiety.

To examine relationships between the CD-FAB and vigilance to food, the dot-probe task was used (MacLeod, Mathews & Tata, 1986). Behaviourally, this vigilance

towards food is defined by the response to images of the threat stimulus, in the dot-probe task. In a typical dot-probe task, participants are shown a pair of stimuli (one threatening, one neutral); these stimuli appear to the left and right of a fixation cross. The stimuli will disappear and a dot will either replace the threatening stimulus (congruent trials) or the neutral stimulus (incongruent trials). Attentional bias is calculated by subtracting the consistent trial reaction times from the inconsistent trial reaction times. Anxious individuals will respond faster to congruent trials when compared to incongruent trials; this is interpreted as a bias towards threatening information (MacLeod, Mathews & Tata, 1986). Visual attention biases have frequently been used to measure threat towards stimuli in phobias and eating disorders, but this task has not been used in CD (Cisler & Koster, 2010).

As anxiety, in general, is the primary emotion related to food attitudes and concerns we anticipated that greater CD-FAB scores would be associated with a greater attentional bias towards threatening stimuli (i.e. gluten-containing food images). Based on the *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* we further anticipated that greater CD-FAB scores would be associated with less food consumption.

Aims

In this chapter, we report the first behavioural, laboratory-based study in CD. Given the limitations of previous cross-sectional studies (Chapters Four, Six), this study explored the relationships between CD-FAB scores, attentional bias towards gluten-free and gluten-containing foods and food consumption in those with biopsy confirmed CD.

7.2. Methods

Adults with CD living in the United Kingdom, ages between 18 and 69 years, with a biopsy-confirmed diagnosis of CD (confirmed via doctor's letter) were eligible to take part in the study. Forty individuals were recruited using posters placed in the University of Birmingham food outlets and through snowballing methods, including social media. The study was advertised as "Individual Differences and Dietary Preferences in Coeliac Disease: A Taste Test" and the recruitment methods asked individuals to not eat for three hours prior to the study, to ensure a similar level of hunger across participants. The taste test was used to ensure participants were not aware that the aim was to assess food intake.

All individuals were required to bring confirmation of their biopsy-confirmed CD diagnosis, in the form of a doctor's letter. All participants had normal or corrected-to-normal vision. Participants were excluded if 1) they reported having a dietary-controlled condition other than CD (e.g. cystic fibrosis, type one diabetes); 2) they did not have evidence for a biopsy confirmed diagnosis for their condition (doctor's letter or gluten-free prescription); and 3) if they had any other food allergies. No participants met the exclusion criteria.

7.2.1. Measures

The questionnaire measures used in this study are fully described in Chapter Six. Participants completed the Depression-Anxiety-Stress-21 scale (DASS-21; Lovibond & Lovibond, 1995), the Illness Perception Questionnaire-Revised (IPQ-R; Moss-Morris et al., 2002), the Food Neophobia Scale (FNS; Pliner & Hobden, 1992), the Eating Attitudes Test-26 (EAT-26; Garner, Olmsted, Bohr & Garfinkel, 1992), the Binge

Eating Scale (BES; Gormally, Black, Daston & Rardin, 1982), the Coeliac Disease Food Attitudes and Behaviours scale (CD-FAB; Chapter Five), the Coeliac Disease Quality of Life scale (Dorn et al., 2010). In addition, a series of visual analogue scales (VAS) were completed throughout the procedure (see below). Two open-ended questions, asking participants what they had eaten and drunk that day and when (based on a similar measure used by Thomas et al., 2014), were used to check individuals had not eaten for three hours prior to attending the laboratory.

Visual Analogue Scales: A series of five VAS were completed at three points: prior to the experimental procedure, prior to the food taste test and on completion of the food taste test. These assessed current nausea, hunger, fullness, happiness and sadness (e.g. "*Please rate how nauseous you feel at this moment*") and allowed us to control for these factors that influence food intake throughout the procedure. Participants were asked to mark the VAS on a 10cm line, describing how they felt with "Not at all" and "Extremely" as the anchors.

Dot-Probe Task: The dot-probe task was presented on a computer using E-Prime 2.0. One-hundred and twenty images, 30 gluten-free foods, 30 gluten-containing foods and 60 control stimuli matched for shape and colour were used; each image was presented twice. Gluten-containing and gluten-free images were matched for calorie, sugar and fat content. In order to make foods easily identifiable, all gluten-free images were of foods that are naturally gluten-free (e.g. fruit, vegetables, nuts) and gluten-containing images consisted of typical gluten-containing foods (e.g. bread, cake, pasta; see Figure One).

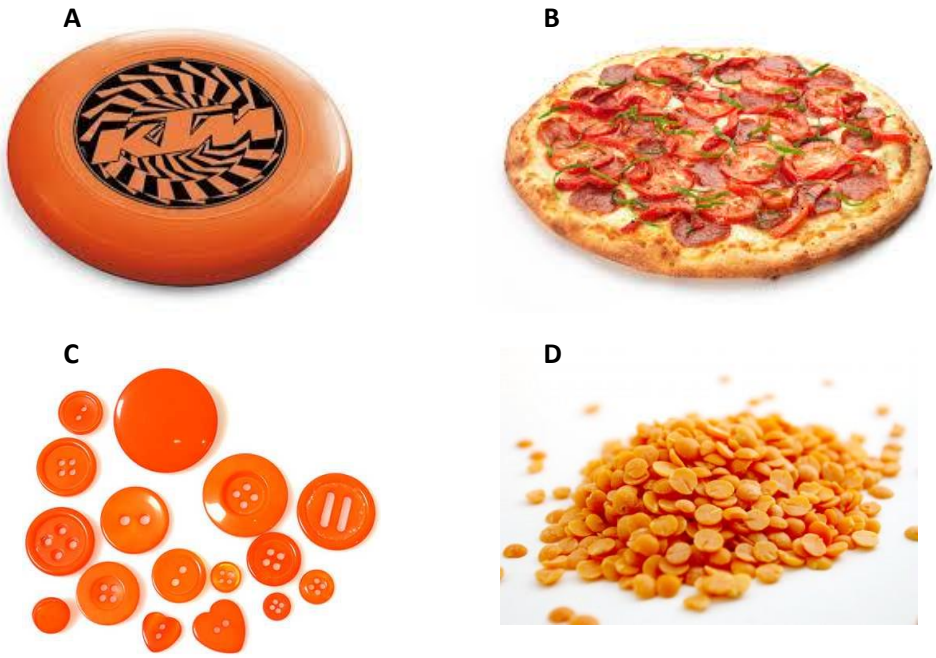

Figure 1: Examples of food and control stimuli in the dot-probe task. A = control image for image B and gluten-containing image; C = control image for image D and gluten-free image

Each trial began with the presentation of a fixation cross for 500 milliseconds (ms). Following this, a food stimulus (gluten-containing or gluten-free) and a control stimulus would appear either side (left/right) of the fixation cross for 500ms. A dot-probe then replaced one of the stimuli. Participants were required to detect the location of the dot-probe as quickly and accurately as possible. The next trial would begin once participants had made their response. Participants completed a series of 10 practice trials, followed by 1 block of experimental trials. In total, the dot-probe task consisted of 180 experimental trials. All trials were randomly ordered and the position of the probe was counterbalanced for side of presentation (left and right) and for image type (food image and control image) to control for order effects.

Ten healthy controls and 10 individuals with CD piloted the dot-probe task by rating their familiarity with the food images and whether the food images were

gluten-free or gluten containing. The control participants also completed the dot-probe task to ensure there was no difference between gluten-free and gluten-containing attentional biases.

Food Taste Task: Participants were served four foods to taste and rate on familiarity, palatability and previous consumption (e.g. "*Have you seen this food item before?*" rated on a yes/no scale), to control for factors that may influence food intake in CD (Four). Likability, sweetness, saltiness and bitterness (e.g. "*How salty would you rate this food item?*") were rated on a 10cm VAS with "Extremely" and "Not at all" as the anchors. These items were used to maintain the cover story of the taste test and were not used in the analysis.

The food taste task contained four gluten-free food types: Mrs Crimbles cheese bites (60g, 263.4 kcals), Pombears (30g, 190 kcals), Schar custard creams (125g, 625 kcals) and Dairy Milk chocolate buttons (80g, 420 kcals). This combination of foods provided a selection of sweet and salty gluten-free foods in snack-sized pieces, making it hard to determine appropriate portion size (Herman & Polivy, 2005). This encouraged greater food consumption and ensured that there were a variety of food products for palatability. To ensure equal knowledge across participants and to reduce the risk of cross-contamination of gluten, the foods were presented in their packaging so participants could confirm the foods were gluten-free.

The arrangement of the food was the same for all participants and individuals were free to eat as much food as they desired from each bowl. The foods were presented in four, separate glass bowls and were labelled plate one, two, three and four. A jug of water and a glass was provided.

Body Composition: A BC-418 Tanita Body Composition Analyser was used to measure weight, body mass index (BMI) and body fat percentage. The lead researcher (RS) measured height.

7.2.2. Procedure

Sessions took place over lunchtime, between 12.00 and 14.00 pm. Participants were greeted and shown to a testing cubicle. After gaining written informed consent, confirming eligibility for the study and completing demographic measures, participants were asked to show the researcher their confirmation of CD diagnosis. Participants then completed a VAS to assess baseline mood and hunger.

The researcher returned and described the dot-probe task. Individuals were sat approximately 50cm in front of the computer monitor and were required to place their left index finger on the "Z" key and their right index finger on the "M" key. Participants were instructed to select the "Z" key if the probe appeared on the left of the fixation cross and the "M" key if the probe appeared on the right of the fixation cross. Ten practice trials were completed, after which, the experimenter left the room. A second VAS was completed after completing the dot-probe task.

On completion of the VAS, individuals were taken into a separate testing cubicle where the food taste test was conducted. Participants sat at a table with four glass bowls containing the packaged gluten-free foods (the weight of the food and packaging combined was recorded by the experimenter prior to the food taste task). Participants were presented with each food item one at a time, and were asked to read the ingredients and confirm the foods were gluten-free. The researcher

removed each food from its packaging and placed the food into its bowl (one food type per bowl).

Participants tasted and rated the four foods, using the VAS scales, taking a sip of water between each food type. They were informed that all of the food would be thrown away at the end of the experiment, so after conducting the taste test they were informed they could eat as much food as they desired. Participants were left in the room for 10 minutes to complete the food taste test whilst the researcher left the room. The researcher then weighed and recorded the packaging of each food type. After 10 minutes, the researcher returned and removed the food bowls and the experimenter weighed the remaining food. The participant then completed the final VAS and questionnaire pack.

Upon completion of the questionnaire pack, participants had their height, weight and body fat percentage calculated. Participants were asked what they believed the aims of the study to be and whether they were aware that their food intake was being measured. Finally, participants were provided with any of the food that they had not consumed in the food taste test, if desired, debriefed and thanked for their time.

7.2.3. Ethical Approval

Ethical approval was granted by the Psychology Research Ethics Committee, University of Birmingham.

7.2.4. Statistical Analysis

Data were analysed using the Statistics Package for the Social Sciences (SPSS) version 22.0. Due to the small sample size, effect sizes are reported throughout to

aid in the interpretation of results. Effects sizes were calculated for the various statistical tests using established effect size calculations as follows: 1) eta-squared (η^2) for Analysis of Variance (small effect= 0.01; medium effect= 0.05; large effect= 0.13; Cohen, 1988), and 2) Cohen's d (*d*) for t-tests (small effect= 0.2; medium effect= 0.5; large effect= 0.8; Cohen, 1988). Increase in r^2 was examined for regression models. (Cohen, 1988)

Outliers: Dot-probe results were screened for accuracy and only the correct trials were analysed (15 trial data points removed). Data points +/- 3 standard deviations from the each participants' mean were removed (55 data points removed). 0.9% of data points were removed from the analysis.

Box-plots of the reaction time data and calories consumed were examined for outliers. One outlier was identified, this data point was not removed as removing this outlier had no effect on the results.

A tertiary split was applied to CD-FAB scores responses received in this dataset, to divide individuals into high, medium and low scorers based on the 33rd and 66th percentiles. By using a tertiary split, we were able to explore differences in outcomes between high and low CD-FAB score whilst avoiding noise within the data from participants that scored close to the high/low cut-off.

Demographics: Differences in psychosocial outcomes and baseline demographics were explored by conducting one-way ANOVAs. Two-way ANOVAs were used to explore associations between CD-FAB scores, hunger, mood, fullness and nausea throughout the procedure.

Food Taste Test: Food intake was calculated as follows (start food weight – leftover food = total food consumed (grams)). The calorie content of consumed food

was calculated using the nutritional information on the packaging. The data were also analysed as grams consumed; there was no difference in the pattern of results so these data are not reported. Multiple regressions were used to explore the association between CD-FAB score and food intake whilst accounting for theoretical covariates and factors associated with food intake. Food familiarity, BMI, disordered eating scores and gender were used as covariates in the regression models. The four ratings of familiarity for each of the food types were combined into a composite food familiarity score ranging from 1 being unfamiliar with all of the food types, to 8 being familiar with all food types.

Dot-Probe Task: Attentional bias to gluten-free and gluten-containing food images was calculated separately. Attentional bias is calculated by subtracting the consistent trial reaction times from the inconsistent trial reaction times. Positive scores are taken as evidence for an attentional bias towards the stimulus and negative scores as a bias away from the stimulus (MacLeod et al., 1986). Paired t-tests were used to compare differences in attentional bias for gluten-free and gluten-containing images. The relationship between CD-FAB scores and attentional bias was explored through one-way ANOVA. Differences between all individuals with CD, regardless of CD-FAB score, were compared to the data from the pilot healthy controls to explore differences in attentional bias to gluten-free and gluten containing stimuli.

7.3. Results

7.3.1. Pilot Study

10 healthy controls (9 females, 1 male) piloted the dot-probe task (18-69 years). Healthy controls showed a mean bias of -5.1 to gluten-containing foods and -1.2 to gluten-free foods. Paired samples t-tests found no difference and a small effect size for attentional bias between food types (t= -.29,p= .78, *d*=-0.10). Mean attentional bias scores were compared to zero, the theoretical non-bias point. No significant differences from zero, and only small effect sizes, were found for gluten-free (t= -.10, p= .92, *d*=0.05) and gluten-containing images (t= -.43, p= .68, *d*=0.19). All foods were named and identified by control participants.

7.4. Main Results

7.4.1. Sample Characteristics

The sample consisted of 12 males and 29 females, with a mean age of 40.5 years (SD= 18.2; range= 18-69), a mean BMI of 24.6 (SD=5.2; range=17.6-44.7) and 8.8 years with CD diagnosis (SD=11.1; range=2-55). The mean score on the CD-FAB was 34.9 (SD=12.2; range=13-58).

One-way ANOVAs found that high, medium and low CD-FAB scorers differed in terms of anxiety, overall distress, EAT-26 scores and quality of life (overall, and *Limitations* and *Health* sub-scales; see Table One). Post-hoc t-tests indicated that compared to the low scorers, high CD-FAB scorers had significantly higher anxiety, overall distress, EAT-26 and quality of life scores ($p<.05$); compared to the low CD-FAB scorers, medium scorers had more impaired quality of life on the *Limitations*

subscale (see Table One). No differences were found in demographics between CD-FAB groups.

Table 1

Sample Characteristics Using the tertiary Split on the CD-FAB. Data are presented as Means.

	High CD-FAB Scorers	Medium CD-FAB Scorers	Low CD-FAB Scorers	F Statistic	Effect Size
Demographics					
Age	42.9	41.4	37.2	.33	0.02[e]
BMI	23.4	26.6	23.7	1.65	0.08[e]
Years with CD	8.6	8.6	9.2	.01	0.00 [e]
Body Fat %	24.6	29.2	25.1	.85	0.04[e]
Depression Anxiety Stress Scale 21					
Depression	8.3	5.9	9.1	1.29	0.07[ee]
Anxiety	9.3	5.0	4.3[b]	1.99*	0.09[ee]
Stress	12.6	11.4	10.3	.30	0.02[e]
Total DASS	31.1	22.3	19.2[b]	1.41*	0.07[ee]
CD Quality of Life					
Quality of Life	35.7	48.5	54.0[bb]	9.54**	0.36[eee]
CDQoL Limitations	14.9	22.1[a]	26.2[bb]	15.22**	0.45[eee]
CDQoL Dysphroia	9.8	5.8	6.6	1.25	0.06[ee]
CDQoL Health	10.1	13.8	14.4[bb]	4.64*	0.21[eee]
CDQoL Treatment	6.2	5.9	6.8	1.02	0.05[e]
Disordered Eating and GFD Adherence (% scoring above clinical cut-off)					
GFD Adherence	13.0	12.9	11.6	.85	0.04[e]
Total EAT	8.9 (0%)	6.4 (0%)	3.4[b] (0%)	3.78*	0.17[eee]
Total BES	9.5 (26.7%)	10.8 (25%)	6.2 (7.1%)	1.64	0.08[ee]

*p=.05; **p<.001 for ANOVA across all three groups; [a]p=.05; [aa]p<.001 for T-Test across low and medium scorers; [b]p=.05; [bb]p<.001 for T-Test across low and high CD-FAB scorers. Effect size using eta-squared: [e]small effect; [ee]medium effect; [eee]large effect.

Baseline characteristics that could affect food intake were assessed throughout the procedure. Two-way ANOVAs revealed a main effect with large effect sizes, whereby participants felt less hungry ($f (1, 39) = 12.83$, $p< .001$, $\eta^2 = .19$) and more full over time ($f (1, 39) = 10.58$, $p< .001$, $\eta^2 = .16$; likely to be due to food consumption), this effect remained across high, medium and low CD-FAB scorers at each measurement time point (prior to dot-probe task, prior to taste test and post taste test). In addition there were two main effects, with medium effect sizes, whereby increasing CD-FAB score was linked with increases in both reported nausea ($f (1, 39) = 6.78$, $p= .002$, $\eta^2 = .12$) and fullness ($f (1, 39) = 4.49$, $p= .013$, $\eta^2 = .07$). No significant interactions were found.

Table 2

VAS Scales Across the Experimental Procedure (Before the experiment (1), Prior to Taste Task (2) and After Taste Task (3)). Data are presented as means and standard deviations. Higher scores indicate greater happiness, sadness, nausea, fullness and hunger.

	High CD-FAB			**Medium CD-FAB**			**Low CD-FAB**		
	1	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**
Happy	66.7 (14.8)	76.0 (12.4)	77.3 (10.5)	72.3 (20.7)	69.1 (16.3)	70.6(11.9)	72.7 (19.8)	80.5 (10.1)	80.3 (10.3)
Nauseous (A)	10.4 (17.0)	8.9 (12.2)	13.2 (16.4)	3.5 (3.6)	4.7 (4.1)	6.3 (6.9)	3.0 (2.6)	3.4 (6.3)	3.3 (3.2)
Sad	12.4 (15.4)	7.8 (8.5)	9.4 (14.4)	7.2 (11.9)	10.7 (11.6)	8.1 (9.9)	5.8 (12.0)	3.6 (3.5)	4.1 (4.4)
Hungry (B)	53.7 (24.6)	51.1 (31.4)	25.0 (25.5)	40.8 (24.1)	51.6 (28.1)	23.0 (22.0)	50.8 (21.5)	63.5 (16.8)	39.5 (20.1)
Fullness (A, B)	31.4 (25.9)	40.2 (28.3)	61.4 (28.0)	31.9 (26.1)	23.7 (18.9)	49.2 (20.3)	24.6 (21.7)	23.2 (18.2)	39.1 (20.8)

A= main effect of CD-FAB group, B = main effect of time, C = interaction

7.4.2. Food Taste Test

Associations were found for overall calorie intake and three variables: BES, psychological distress and food familiarity ($p<.05$). Based on these significant relationships, these variables were entered into step one of the regression model. BMI, gender and EAT-26 scores were also included in step one, as these factors are commonly associated with food consumption (Pollard, Kirk & Cade, 2002). Total CD-FAB scores were entered in step two to explore the relationship between CD-FAB scores and calorie intake.

The first model accounted for 34.9% of the variance in calories consumed ($f(1,39)=4.48$, $p=.002$; see Table Three), with food familiarity and BES scores having significant positive regression weights. The addition of CD-FAB scores did not explain any additional variance in calories consumed.

Table 3

Factors Associated with Overall Calories Consumed

Predictors	B	*B*	R^2	F	R^2 Change
Model 1) Covariates					
Gender	*-102.26*	*-.21*			
Body Mass Index	*-5.73*	*-.13*			
Composite Food Familiarity	*132.61*	*-.40**			
BES Total	*11.63*	*.36**			
EAT-26	*-1.78*	*-.04*			
DASS-21	*3.56*	*.81*	*.45*	*4.48***	*.45***
Model 2) CD-FAB					
Gender	*-101.20*	*-.21*			
Body Mass Index	*-5.60*	*-.13*			
Composite Food Familiarity	*132.42*	*.40**			
BES Total	*11.42*	*.35**			
EAT-26	*-2.35*	*-061*			
DASS-21	*3.51*	*.30*			
CD-FAB Total	*.79*	*.04*	*.45*	*3.75***	*.00*

* = significance at p<.05; **p<.005. The significance of the F value refers to the F associated with each step.

7.4.5. Dot-Probe Task

Across CD participants, the overall mean attentional bias score for the gluten-free foods was -1.0 (SD=60.3) compared to a mean of 5.5 (SD=34.0) for the gluten containing foods. A 2 (gluten-free images and gluten-containing images) x 3 (high, medium, low CD-FAB scorers) ANOVA was conducted to explore relationships between CD-FAB scores and attentional bias to gluten-free and gluten-containing food images.

No significant main effects were found; there were small effects for food stimulus (f (1, 37) = .33, p= .57, η^2= 0.01) and a medium effect size for CD-FAB score

(f (1, 37) = 1.80, p= .18, η^2= 0.13). No significant interactions were found and only small effect sizes (f (2, 37) = 1.13, p= .34, η^2= 0.06). The same pattern of results was found for the CD-FAB subscales.

Post-hoc tests were used to further explore this data. The combined CD data was compared with the pilot data from healthy controls using T-tests. No differences were found between healthy controls and individuals with CD on measures of attentional bias for gluten-free images, and only small effect sizes (t (1,48)=1.08, p=.29, *d*= 0.16) but individuals with CD had a greater attentional bias towards gluten-containing images compared to healthy controls, demonstrating a medium effect size (t (1,48)=2.03, p=.048, *d*= -.58). No other significant differences were found.

7.4.6. Manipulation Check

Participants were asked what they thought the aims of the study were and whether they were aware that the amount of food they consumed was being measured. 2.4% (n=1) of participants correctly guessed the aims of the study. 34.1% (n=17) of the sample reported that they were aware their food intake was being measured.

7.5. Discussion

The aim of this chapter was to explore the relationship between CD-FAB scores, food intake and attentional bias towards gluten-free and gluten-containing foods in individuals with a biopsy-confirmed CD diagnosis. CD-FAB scores were explored alongside measures of attentional bias towards gluten-free and gluten containing food images, and the amount of food consumed in a laboratory environment. Results revealed that CD-FAB scores were not associated with calorie intake and

attentional bias did not significantly differ according to CD-FAB scores but strong effect sizes were apparent for the dot-probe task; we were also able to replicate the psychological correlates of the CD-FAB in a biopsy-confirmed sample of individuals with CD.

7.5.1. Food Taste Test

Alterations in the amount of food consumed are a key feature of disordered eating patterns (APA, 2013). In relation to CD, some individuals find it challenging and may be unwilling to eat food outside the home in order to prevent gluten cross-contamination and subsequent gastrointestinal symptoms (Chapter Four; Olsson, Hornell, Ivarsson & Sydner, 2008; Simpson et al., 2011). Contrary to our hypothesis, CD-FAB scores were not associated with calories consumed.

There are two potential interpretations of this finding; greater concerns around food and cross-contamination may have no relationship with food intake in individuals with CD. However, this explanation seems unlikely, as the qualitative findings in Chapter Four suggested that dietary-restriction occurred in novel environments for those who had increased concerns around food; in addition, Chapter Six reported that increased CD-FAB scores were associated with an unwillingness to try new foods. Alternatively, these findings may stem from methodological limitations within this study. Although the laboratory environment is novel and we had anticipated that increased CD-FAB scores would be associated with reduced food intake, participants may have viewed this environment as a “safe” location to consume food. Participants were presented with pre-packaged gluten-free foods, and were able to confirm this by checking the ingredients list. It is

possible that even those with concerns around food feel safe enough to consume food in a controlled, laboratory environment. Furthermore, individuals tended to be familiar with the foods presented and food familiarity was independently associated with calorie intake, highlighting the lack of novelty within the experimental set-up.

The association between BES scores, food familiarity scores and increased food consumption is not surprising. As the BES measure contains items targeting food consumption, this positive association may be expected. BES scores have been associated with increased calorie consumption in healthy controls and this finding appears to be consistent in CD (Laessle & Schulz, 2009; Peterson et al., 2012). Additionally, our results show that food familiarity was associated with total calories consumed. This is in line with the qualitative findings from Chapter Four, which found that individuals with CD preferred to eat at food places they had been previously and preferred to consume foods they had experienced before. Sticking to eating and buying familiar food products is a strategy adopted by many individuals with CD, our findings highlight that this also translates into the amount of calories consumed (Food Standards Agency, 2009).

7.5.2. Dot-Probe Task

Within the disordered eating and anxiety/phobia literature, there is considerable evidence to suggest that individuals with concerns around food will orient their attention to the stimulus of fear (food images; Cisler & Koster, 2010; Faunce, 2002). Our data suggest that individuals with CD had a greater attentional bias towards gluten-containing images when compared to healthy controls, irrespective of CD-FAB scores. This highlights potential mechanisms by which individuals with CD may differ

from healthy controls. Individuals with CD appear to be more sensitive to gluten-containing food cues than healthy controls and this increased attentional bias towards food in CD may result from the need to be vigilant around gluten. Although no other literature reports this finding in CD, this is in line with qualitative reports that indicate increased food preoccupation in CD (Leffler et al., 2007; Yucel et al., 2006).

No significant main effects or interactions were found for food stimulus or CD-FAB scores (gluten-free and gluten-containing images); this indicates that food stimuli are processed similarly across individuals with CD. It is premature to conclude that CD-FAB scores are not associated with attentional bias for food-related stimuli. Given the small sample size, limited statistical power may explain the lack of significance in the attentional bias comparisons. Alternatively, picture-based images may not be enough to influence attentional bias in those with high CD-FAB scores; paradigms that use actual food items instead of food images, may better reflect attentional bias in CD. Furthermore, this study would have benefited from an additional manipulation check to see whether the food images were associated with an increase in self-reported concern in participants.

7.5.3. Questionnaires

In line with our previous findings (Chapter Six), increased CD-FAB scores were associated with impaired quality of life and psychological distress in individuals with a biopsy-confirmed diagnosis of CD. The replication of these findings in a biopsy-confirmed sample of individuals with CD further adds to the evidence that high CD-FAB scores are associated with negative psychosocial outcomes and requires

assessment and intervention in the CD population. Additionally, we were able to replicate the finding that EAT-26 scores were associated with CD-FAB scores but there was a non-significant association between BES and CD-FAB scores. This adds to the discriminative validity of the CD-FAB, as discussed in Chapter Six.

7.5.4. Limitations and Directions for Future Research

The limitations of this study need to be noted. The distribution of CD-FAB scores across this sample was within the lower end of CD-FAB scores, with a maximum score of 58 out of a possible 77. The advertising of this study as a "taste test" may have prevented individuals with more extreme food concerns from volunteering to take part. Furthermore, the use of a tertiary split to divide individuals into high and low CD-FAB groups divides the group based on an arbitrary number. Without further exploration of the CD-FAB properties in large samples, we cannot establish clinically meaningful cut-offs. Future research needs to establish the cut-off for clinical impairment within the CD-FAB in order to examine the needs of those most at risk.

By using a highly controlled environment for the experimental procedure; whereby individuals could see the food items in packages and read these packages, this may have created a setting that even those with food concerns may have viewed as safe. Further procedures should focus on modifying the food environment, by presenting participants with unpackaged gluten-free foods, to explore whether different levels of control within the environment affect food intake in CD.

The replication of the association between CD-FAB scores and psychosocial outcomes in a biopsy-confirmed sample of CD further extends the findings from Chapter Six. Despite this, the tertiary split was applied to CD-FAB responses received

within this dataset. As a result, the split of CD-FAB scores may differ across this dataset and Chapter Six, meaning the findings from both chapters are not directly comparable. Despite the limitations, this study demonstrates the application of laboratory based behavioural methods in CD. These methods provide a way of assessing the attentional biases within CD and measuring food consumption. In addition,

The results from this study also further inform the development of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*. Future revisions of this model need to consider whether eating behaviours are affected by food concerns and whether there is a perceived level of safety that will allow even individuals with high CD-FAB scores to consume food in novel settings.

CHAPTER EIGHT: GENERAL DISCUSSION

8.0. Chapter Rationale

The overall aim of this thesis was to examine the relationship between disordered eating attitudes and behaviours and CD. This included the following aims: 1) to develop a theoretical model to explain the development of disordered eating in CD; and 2) to empirically test some of the core assumptions proposed by this model. I will discuss how the thesis has answered these questions by assessing the contribution from each study and integrating the findings to evaluate and further develop the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.*

I will then discuss the clinical implications of this work and how this may be used in clinical practice to inform the treatment and support of individuals with CD. This will be followed by a discussion of the strengths and limitations of this work and directions for future research.

8.1. Overview of Findings

This thesis sought to develop and evaluate a theory explaining disordered eating attitudes and behaviours in CD. The results presented in this thesis have explored the prevalence, experiences and factors associated with disordered eating in CD, in order to evaluate the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two).

8.1.1. Development of the Theoretical Model of Disordered Eating in Gastrointestinal Disease

A systematic review of the literature relating to disordered eating in dietary-controlled gastrointestinal conditions assessed the current state of the literature and was used to develop the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* (Chapter Two). This model was formed of distinct stages that were assessed in later chapters, through mixed methodologies.

The literature review identified that the majority of research on disordered eating in gastrointestinal conditions was based on case studies; only nine papers were eligible to be included in the review. Overall, the prevalence rates of disordered eating across dietary-controlled gastrointestinal disorders were 5.3-44.4% and these consisted of dietary restriction and bulimic pathologies. No assessments of binge-eating behaviours were made in any of the studies reported in the review (Chapter Two). Psychological distress, symptom severity and dietary management were found to be associated with disordered eating patterns; however, the direction of these associations were varied across papers.

Based upon the findings of this literature review, the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* was developed. Briefly, the model suggests that there are two pathways that lead to disordered eating attitudes and behaviours in dietary-controlled gastrointestinal disease. The first pathway describes how distress around post-diagnosis weight change contributes to poor dietary-management and disordered eating attitudes and behaviours. The second pathway describes increased hypervigilance and concern around food prepared in novel or unfamiliar environments. These beliefs stem from the concern that all food has the potential to trigger gastrointestinal symptoms. In order to adapt to these beliefs, individuals respond by only eating in familiar settings, only eating familiar foods, or only eating foods that they have prepared themselves, resulting in a limited food intake and an impaired quality of life, particularly in social domains. The next section will discuss the findings of this thesis in the context of this model and suggest further modifications to this model.

8.1.2. Prevalence of Disordered Eating in CD

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* assumes that individuals with CD will be at risk for the development of disordered eating attitudes and behaviours. The combined prevalence rates in the general population for Anorexia Nervosa, Bulimia Nervosa and Binge Eating Disorder is 13.1% (Stice, Marti & Rohde, 2013) and disordered eating is estimated at 10% (Solmi et al., 2014).

Throughout this thesis, clinical cut offs on the Eating Attitudes Test 26 (EAT-26; Garner & Garfinkel, 1979) and the Binge Eating scale (BES; Gormally, Black, Daston & Rardin, 1982) have been used to estimate the prevalence of disordered eating

attitudes and behaviours in CD. Within this thesis, 15.7-17.7% of participants scored above the cut-off on the EAT-26 and 19.4-21.6% on the BES (Chapters Three and Six), which is higher than the prevalence found in healthy controls. Furthermore, Chapter Three reported findings demonstrating that more individuals with CD scored above the clinical cut-off on the EAT-26 compared to healthy controls and those with type two diabetes. However, there was no difference in BES scores across all dietary-controlled conditions reported compared to healthy controls. These findings support the assumption that there is an increased risk of disordered eating attitudes and behaviours in CD compared to healthy controls, as assessed by the EAT-26 and the BES. Individuals with CD may also be at greater risk for bulimic and restriction behaviours, as assessed by the EAT-26, than those with other dietary-controlled conditions (type two diabetes and inflammatory bowel disease), whereas the presence of binge eating behaviours was similar across dietary-controlled chronic health conditions, but greater than healthy controls (Chapter Three).

8.2. An Evaluation of Pathway One: Weight Change and Disordered Eating in Coeliac Disease

Pathway one of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* is based on three main assumptions: 1) individuals will be upset by weight change that occurs after CD diagnosis and commencement of the GFD; 2) individuals will attribute this weight change to the GFD and 3) individuals will engage in the consumption of gluten to encourage the weight loss that can result from the gastrointestinal symptoms associated with CD. The consumption of gluten to promote weight loss can be conceptualised as a form of disordered eating, as misuse

of medication is defined as an inappropriate compensatory behaviour in the DSM-5 (Simon et al., 2011). The evidence from this thesis will be used to evaluate each of the main assumptions in this pathway.

8.2.1. Distress Surrounding Coeliac Disease Related Weight Change

After CD diagnosis and initiation of the GFD, weight change (usually gain) commonly occurs (Kabbani et al., 2012). According to the model, this weight change can lead to distress and the development of disordered eating attitudes and behaviours (Chapter Two). Not all individuals will experience weight change after CD diagnosis; however, when weight change does occur this can be interpreted in a negative manner and strategies to reverse this weight change are made (Dowd et al., 2013; Madden, Riordan & Knowles, 2016).

The qualitative findings of Chapter Four found that individuals who scored high on measures of disordered eating, as assessed by the EAT-26 and the BES, described distress around post-diagnosis weight gain and a desire to regain their pre-diagnosis weight. Furthermore, difficulties in adapting to post-diagnosis body shape were described in combination with psychological distress, and dietary restriction was used to achieve weight loss in these individuals. These findings are in line with Leffler et al. (2007) who described three case studies where concerns around weight increased after starting the GFD and led to dietary restriction. In addition, Madden, Riordan and Knowles (2016) found that individuals were concerned with the rate of weight increase after starting the GFD and this led to concerns around weight management.

8.2.2. Post-Diagnosis Weight Change will be attributed to the Gluten-Free Diet

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* proposes that those who experience distress around post-diagnosis weight change develop the belief that these weight changes are caused by the initiation of the GFD (Chapter Two). These beliefs are not unfounded: prior to CD diagnosis individuals may present as underweight and 21.5% experience clinically significant weight gain after starting the GFD (Kabbani et al., 2012). In addition, gluten-free foods have a greater nutritional imbalance compared to gluten-containing alternatives, including an increased calorie, sugar and fat content which may contribute to weight change in CD (Miranda et al., 2014).

Madden, Riordan and Knowles (2016) found some support for this assumption; individuals with CD were concerned about unwanted weight gain and attributed this to the fat, sugar and salt content of manufactured gluten-free foods, leading to a desire to remove these foods from the diet. Additionally, the qualitative findings reported in Chapter Four found that individuals scoring high on measures of disordered eating had an increased awareness of the nutritional composition of gluten-free foods. Participants were distressed about the poor quality of gluten-free food, and this was perceived to be the cause of post-diagnosis weight gain. Furthermore, Rocha, Gandolfi and Snatos (2016) found that individuals who believed the GFD was nutritionally poor, reflected on the benefit of removing manufactured gluten-free foods from their diet, as they believed this would increase health and reduce their risk of obesity.

Weight change in CD appears to be attributed to the nutritional content of gluten-free foods and this can lead to a desire to alter weight and/or diet. These

findings are similar to the process of insulin-related weight gain described in the Modified Dual Pathway Model (Peterson, Fischer & Young-Hyman, 2015). This model proposes that post-diagnosis weight gain in diabetes, associated with insulin therapy, creates a vulnerability to body dissatisfaction and dietary restraint. The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* places this weight change in the context of CD; beliefs around the quality of the GFD can contribute to disordered eating attitudes and beliefs.

8.2.3. Poor Dietary-Management will be used to Promote Weight Loss

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* suggests that these beliefs around gluten-free foods will result in the intentional consumption of gluten to encourage gastrointestinal symptoms and subsequent weight loss as reported in case studies (Leffler et al., 2007; Yucel et al., 2006; Young et al., 2013).

Within this thesis, no evidence was found for the intentional consumption of gluten to promote weight loss. No reports of intentional gluten consumption were described in the interviews with CD participants (Chapter Four); however, those scoring high on measures of disordered eating described less strict management of their GFD, particularly when eating outside the home. Furthermore, when asked about gluten consumption in an anonymous web-meditated survey, poor dietary management was associated with disordered eating scores (Chapter Three and Six).

Chapters Three and Six found increased disordered eating scores, as assessed by the EAT-26, to be associated with self-reported gastrointestinal symptoms and self-reported poor dietary management. In addition, these factors produced a separate group within the cluster analyses. One interpretation of the associations between

GFD management and gastrointestinal symptoms is that this results from intentional gluten consumption. However, another explanation is that this may reflect a bi-directional relationship between gluten consumption and gastrointestinal symptoms, whereby poor dietary management leads to increased gastrointestinal symptoms and vice versa. In support of this interpretation, cross sectional studies have also found an association between disordered eating patterns and poor GFD management in CD (Arigo et al., 2012; Karwautz et al., 2008). However, it is not clear what the reasons underlying deliberate gluten consumption are, i.e. whether it is intentional, and whether deliberate gluten consumption relates to weight change.

8.2.4. Disordered Eating Patterns Related to Pathway One

The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* suggests that a combination of the previous three factors can lead to the development of disordered eating attitudes and behaviours in CD. The EAT-26 was effective in identifying disordered eating attitudes and behaviours that were associated with difficulties in GFD management, gastrointestinal symptoms and distress around post-diagnosis weight change.

Those who reported difficulties following the GFD and increased gastrointestinal symptoms were identified by the EAT-26 (Chapters Three, Four and Six). This tool appears to be effective in identifying disordered eating in those with CD who engage in dietary-restriction, and/or deliberate gluten ingestion, with the intention of losing weight. As this behaviour is of particular importance to clinicians, further research should focus on the role of deliberate gluten consumption and disordered eating in CD, to establish the prevalence of and motivations behind this behaviour.

8.3. An Evaluation of Pathway Two: Food Concerns and Disordered Eating in Coeliac Disease

The second pathway of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* consists of three main assumptions: 1) individuals experience severe gastrointestinal symptoms prior to diagnosis and a resolution of these symptoms when following the GFD; 2) the belief that all foods have cross-contamination potential and an increased concern around food and 3) a fear of consuming food prepared in new environments or by unfamiliar people. These factors can lead to eating attitudes and behaviours that can be conceptualised as disordered, as these eating patterns are inflexible and impair psychosocial well-being (Freeland-Graves & Nitzke, 2013).

8.3.1. Severe Gastrointestinal Symptoms Prior to Diagnosis and Reversal of Symptoms After Initiation of the Gluten-Free Diet

According to the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*, individuals at risk for disordered eating attitudes and behaviours will experience increased gastrointestinal symptoms prior to CD diagnosis. However, once commencing the GFD, these symptoms will resolve and individuals will believe in the effectiveness of their treatment. Severe symptoms surrounding diagnosis and beliefs about the effectiveness of the GFD have previously been associated with strict management of the GFD (Sainsbury & Mullan, 2011), however, no research has explored the relationship between these factors and the development of disordered eating attitudes and behaviours.

Symptoms at CD diagnosis and the resolution of these symptoms were not directly assessed in this thesis. However, the qualitative findings from Chapter Four indicated that greater concerns around food and cross-contamination were described by those who talked about the effectiveness of their GFD in terms of symptom resolution. The majority of these individuals also reflected on their pre-diagnosis symptoms as uncomfortable and distressing. Although this lends some support to the model's assumptions, this assumption was not fully assessed and further studies are needed to assess the symptoms prior to diagnosis, the effect these have on GFD management and on disordered eating attitudes and behaviours.

8.3.2. The Belief that all Foods have Cross-Contamination potential

According to the theoretical model, those who feel that their GFD is effective in reversing their CD-related symptoms may develop the belief that all foods have cross-contamination potential and an increased concern around food (Chapter Two). This occurs because the CD-related aversion symptoms that were triggered by gluten-containing foods may generalise across all unfamiliar food types, creating a general concern around food. Hypervigilance and preoccupation with food and sources of cross-contamination can also contribute to the development of disordered eating attitudes and behaviours. These concerns have consistently been reported in the CD literature and have been associated with reduced food consumption at social events (Rocha, Gandolfi & Santos, 2016; Silvester et al., 2016; Zarkadas et al., 2013).

During qualitative interviews (Chapter Four), concerns around food and cross-contamination were discussed in combination with restrictive eating behaviours and

strict management of the GFD. Although vigilance around food was described as necessary by the majority of participants, eating attitudes and behaviours were only affected in those with hypervigilance around food. Hypervigilance led to individuals being fearful of gluten in non-ingested-items, including wallpaper and food packaging, and these thoughts were allowed to affect their daily lives. Surprisingly, individuals describing these concerns did not score above the clinical cut-offs on the EAT-26 or the BES, and so these instruments would not have identified their disordered behaviours, and they did not discuss a desire to alter their body-shape and/or weight (Chapter Four). Other assessment tools were needed to understand and identify the concerns described by this pathway, leading to the work reported in Chapter Five.

Chapter Five reported the development of the *CD Food Attitudes and Behaviours Scale* (CD-FAB) that was designed to explore these food concerns in the context of CD. This scale contained items that demonstrated a fear of food and the impact that this could have on individuals (e.g. *I won't eat food unless I have complete control over the preparation*), as well as adaptive strategies to cope with concerns around food (e.g. *I will happily prepare gluten foods for others, as long as it doesn't come into contact with the food I'm preparing for myself*). A high score on the CD-FAB indicates greater concerns around food, and was associated with an increase in anxiety and an impaired quality of life, particularly in social domains (Chapters Five, Six and Seven), supporting the assumptions of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*.

The development of the CD-FAB allowed us to test the assumption that there would be food preoccupation in individuals with concerns around food and cross-

contamination. Chapter Seven explored associations between the CD-FAB and attentional bias to gluten-free and gluten-containing food stimuli. Contrary to the model's assumptions, CD-FAB scores were not significantly associated with attentional bias towards food images (gluten-free or gluten-containing), however individuals with CD had a greater attentional bias towards gluten-containing foods when compared to healthy controls. Furthermore, using the qualitative methods in Chapter Four found that individuals scoring high on measures of disordered eating consistently report greater focus around food and food content after their CD diagnosis. This thesis presents mixed evidence for the role of food preoccupation in the development of disordered eating attitudes and behaviours in CD. Given the limitations that were reported in Chapter Seven and the qualitative findings, it is too premature to conclude that food preoccupation does not contribute to disordered eating attitudes and behaviours, particularly given the importance of this relationship in other dietary-controlled chronic health conditions (Quick, Byrd-Bredbenner & Neumark-Sztainer, 2013). Vigilance around food is important for the management of CD, however, the food concerns and preoccupation present in those scoring high on the CD-FAB may contribute to the development of disordered eating attitudes.

8.3.3. A Fear of eating novel foods or food in novel environments

The theoretical model proposed that beliefs and hypervigilance, previously discussed, around food and cross-contamination contribute to a fear of eating novel foods or eating in novel environments. These eating behaviours can be considered

disordered as they dominate daily thoughts and impair one's ability to eat flexibly (Freeland-Graves & Nitzke, 2013).

Vigilance around food, particularly when eating outside the home, is essential for all individuals with CD. Previous, qualitative reports have indicated that novel foods and food situations cause increased concern for individuals with CD. Individuals report constant anxiety mainly concerning instances of cross-contamination when eating outside the home and this is associated with the refusal of social invitations involving food (Black & Orfilia, 2011; Rose & Howard, 2013; Sverker et al., 2009). However, this vigilance becomes maladaptive when strategies to promote safety outside the home are not adopted and a fear of food develops. By carrying gluten-free foods, reading restaurant menus and asking questions to restaurant staff, individuals with CD should be able to eat novel gluten-free items in novel environments (Rordian & Frognel, 2014).

The development of the CD-FAB allowed us to test the assumption that there would be a fear of consuming food prepared in new environments or food prepared by unfamiliar people. Increased CD-FAB scores were associated with eating outside the home less often and a fear of trying new foods, as assessed by the Food Neophobia scale, which is in line with the concerns around food reported in qualitative accounts (Chapter Six). These findings are consistent with the model's assumptions.

Chapter Seven explored the relationships between the CD-FAB and its association with food intake in a laboratory setting. According to the *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* individuals scoring high on the CD-FAB should consume less food in the laboratory, as this is an unfamiliar

environment. Contrary to the model's assumptions, food intake (in terms of weight of food consumed in the lab) CD-FAB scores were not related. These findings may result from methodological limitations and the procedures failure to induce food anxiety; for example, the food in the laboratory was not unfamiliar to individuals with CD and was presented in a clinical environment. Further, the food was pre-packaged and unwrapped in the laboratory at the University of Birmingham; participants may have believed that the University would not contaminate them with gluten. These factors may have encouraged food consumption by individuals despite increased food concerns. Furthermore, the majority of individuals reported being familiar with the food items presented in the laboratory set-up. This food familiarity was independently associated with the amount of food consumed, providing some support to the assumption that unfamiliar foods would not be consumed in individuals with high CD-FAB scores.

The proposal that hypervigilance around food is associated with the development of eating behaviours that can be considered disordered is not reflected in any of the theoretical models outlined in Chapter One. This is a unique component in the Theoretical Model of Disordered Eating in Gastrointestinal Disease and future research needs to explore its occurrence in other gastrointestinal conditions and the willingness to change this behaviour.

8.3.4. Eating Attitudes and Behaviours Resulting from Pathway Two

The CD-FAB is a novel tool that is effective in identifying those who report concerns around food and food preparation. Limiting food intake outside the home and in unfamiliar environments may be considered adaptive for those with CD;

however, the association of this with clinical levels of distress and impaired quality of life indicates that the eating attitudes and behaviours described by the CD-FAB can be conceptualised as maladaptive (Chapter Six). Further research is needed to explore the impact of these eating attitudes and beliefs on physical, behavioural and psychological outcomes.

8.3.5. The Potential for a Third Pathway?

The results described so far have largely provided support for the assumptions of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease.* However, the findings of this thesis also suggest further modifications that need to be taken into account in future revisions of this model.

Chapter Three explored the disease specific and nonspecific factors associated with disordered eating scores in CD. All of the dietary-controlled conditions (CD, inflammatory bowel disease, type two diabetes) scored equally high on the BES, and these scores were associated with psychological distress. These findings are consistent with Stice's Dual Pathway Model (2002) which proposes that psychological distress is important in the development of disordered eating attitudes and behaviours (Dury, 2015; Stice, 2002). Additionally, the Modified Dual Pathway Model (Peterson, Fischer & Young-Hyman, 2015) proposes that disease-related distress further contributes to the risk of disordered eating in those with dietary-controlled chronic health conditions. One interpretation of these findings is that binge eating behaviours result from the non-specific burden of living with a chronic health condition.

Strength to this argument was added by the results of Chapter Six, which found a positive association between psychological distress and BES scores in CD. Furthermore, cluster analyses indicated that individuals who scored high on measures of binge eating and psychological distress were a distinct group from those who had increased gastrointestinal symptoms, poor dietary management, and high scores of measures of Anorexic and Bulimic symptoms, as described by the first pathway of the model (Chapter Three and Six).

The qualitative findings from Chapter Four indicate that individuals with high disordered eating scores would long for gluten-containing foods after their CD diagnosis. These feelings of loss and distress around food were associated with increased food consumption to compensate for the restrictive GFD. For some individuals this was associated with the need to hoard foods. In addition, individuals who reported feeling restricted by the need to follow a restricted dietary-regimen would consume increased amounts of food when it was available to them and use this food consumption to elevate their mood. Binge-eating behaviours have consistently been reported alongside low mood in the general population, and food consumption is used to elevate mood in these individuals (Emery, King, Fischer & Davis, 2013).

These finding indicate that in addition to the two pathways described by the *Theoretical Model of Disordered Eating in Gastrointestinal Disease*, some individuals may find it hard to cope with the general stressors of a chronic health condition and the restrictive nature of the GFD, and cope with this by engaging in binge-type eating patterns. The pathways of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease,* do not account for these factors; the addition of a third

pathway that describes how the general stressors of a chronic health condition, and the need to maintain a strict dietary regimen, can lead to binge eating behaviours in CD as assessed by the BES.

This binge-eating pathway is similar to the relationships between negative affect, dietary restraint and disordered eating described in Stice's (2002) Dual Pathway Model. Furthermore, Herman and Polivy's (1984) Boundary Model can be used to understand the relationship between dietary restraint and binge eating. The Boundary Model suggests that those who restrict their intake are more responsive to food-related external stimuli and at risk for both under and overconsumption of food. Similar patterns of eating have been described in people with Type Two Diabetics who also follow a prescribed dietary regimen (Herpertz et al., 2001).

8.4. The Revised Theoretical Model of Disordered Eating in Coeliac Disease

Based on the findings from studies contained within this thesis, we were able to modify the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* to describe the development of disordered eating attitudes and behaviours in CD and recommend appropriate tools to explore these eating attitudes and beliefs further. Stice's Dual Pathway Model (Stice, 2001), which has been extensively researched in adolescents without long-term conditions, captures the broader aspects of disordered eating which are still relevant to the CD population. This model was not developed to be an all-encompassing model of disordered eating but rather a framework for the factors that occur alongside the diagnosis and management of CD, that are not accounted for in Stice's model. As a result, the Theoretical Model of Disordered Eating in Gastrointestinal Disease can be seen as a model accounting for

the disease-specific factors that contribute to disordered eating attitudes and behaviours that occur within the broader context of the sociocultural factors accounted for by Stice's model

Figure One describes the revised model as applied to CD. The model is formed of three pathways, two disease-specific pathways that were described in the original model and one nonspecific pathway resulting from the burden of living with a chronic health condition. A brief description of each pathway can be found below.

Pathway One: This pathway described individuals with increased concerns around food and cross-contamination. These individuals will have experienced distressing gastrointestinal symptoms prior to CD diagnosis but respond well to the GFD, experiencing symptom resolution. As a result, these individuals develop increased concerns around food prepared in unfamiliar environments or by unfamiliar individuals. Therefore, food consumption is limited in novel settings, but eating patterns are typical when food preparation is controlled. This dysfunctional focus on GFD management may lead to disordered eating motivated by the fear of gastrointestinal symptoms or the need to engage in dietary-restriction in environments that are viewed as unsafe but increased food consumption in environments that are perceived as safe. These eating attitudes and behaviours can be screened for using the CD-FAB.

Pathway Two: This pathway describes those who exhibit disordered eating attitudes and behaviours more in line with traditional eating disorders that are motivated by the desire to change weight or body shape. These individuals will experience distress in response to weight changes that occur after the CD diagnosis and initiation of the GFD. As a result, the belief that the GFD is responsible for these

weight changes results in the deliberate consumption of gluten in order to promote weight loss through the physiological effect of gluten. These eating attitudes and behaviours can be screened for using the EAT-26.

Pathway Three: This pathway describes disordered eating attitudes and behaviours that result from the nonspecific burden of living with and managing a chronic health condition. Those who experience distress surrounding their CD diagnosis and the adoption of the GFD, may feel that the GFD is too restrictive and report distress around the loss of gluten-containing foods and as a result may consume large quantities of food as a coping strategy to elevate their mood. These eating attitudes and behaviours can be screened for using the BES.

In its present form, the model implies that disordered eating in CD is unidirectional in nature. The arrows between the constructs imply no association among the factors that may contribute to disordered eating attitudes and behaviours in CD. In addition, it is not clear whether individuals can move across disordered eating pathways or whether the pathways feed into one another. The qualitative results from Chapter Four indicate that individuals may engage in both a binge and restrictive eating pathology, suggesting that a multidimensional model where the pathways interact requires further exploration. Additionally, the exploration of operational feedback mechanisms that may reinforce disordered eating attitudes and behaviours is essential in this population.

The clustering of items within the model needs further exploration. Although the model describes a poor or good adaptation to diagnosis, the items reflected in the clusters (*distress in response to weight gain; greater symptoms at diagnosis, resolution of these symptoms*) relates more specifically to the physical and

psychosocial response to GFD treatment, and it is these responses to treatment that were reflected in the findings (Chapter Four). Furthermore, the model refers to *Dysfunctional Illness Beliefs*, which includes illness beliefs as well as emotional states (e.g. *high anxiety*). This lack of differentiation between emotional states and cognitions makes it challenging to specify relationships between disordered eating attitudes and behaviours in CD. Future revisions on the model need to explore the role of anxiety and illness beliefs in the development of disordered eating attitudes and behaviours and reflect on the terminology used within this model, to allow further understanding of the relationships between these emotions and cognitions.

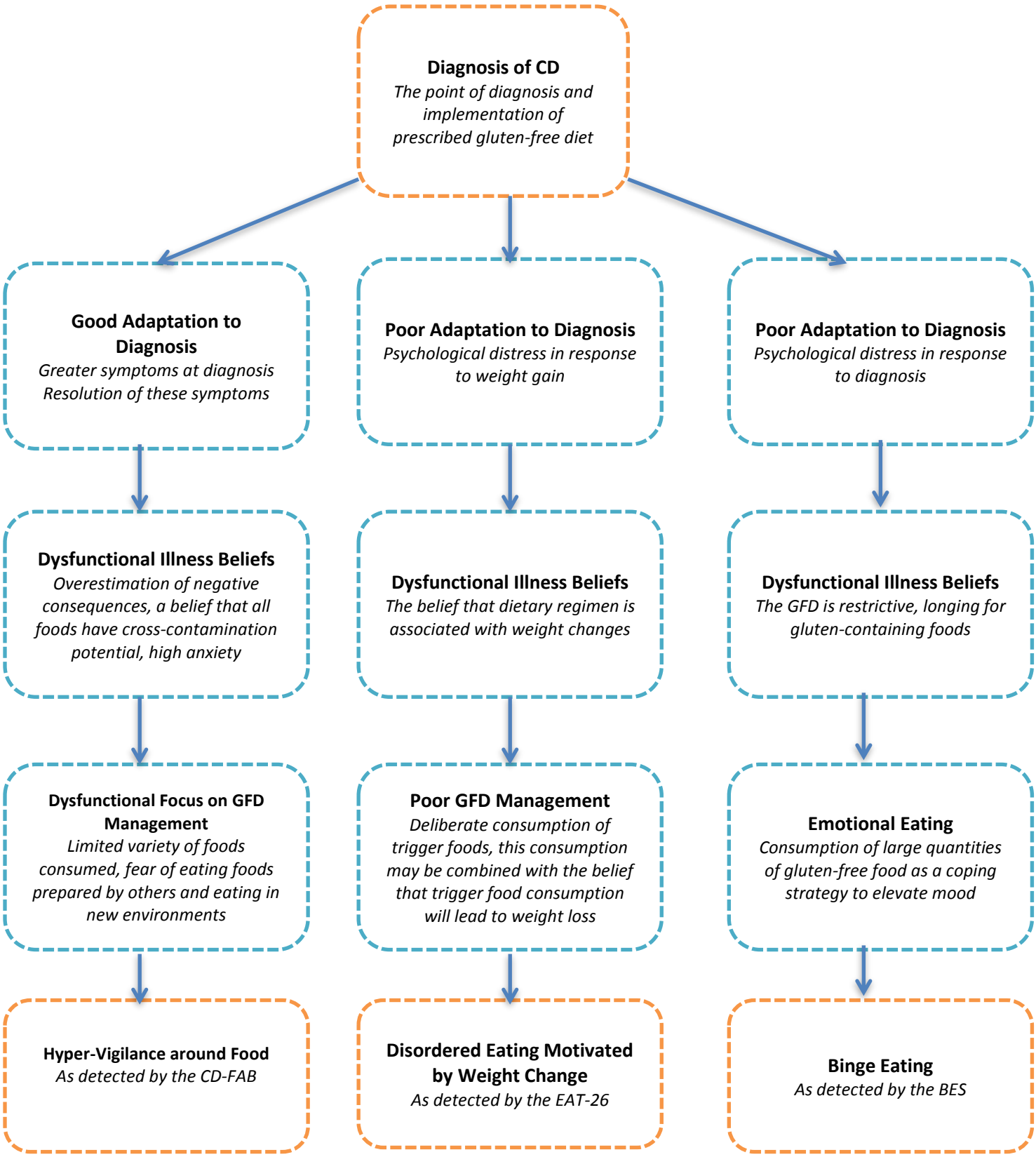

Figure One: The revised model of disordered eating as applied to CD. Orange boxes indicate the type of disordered eating and method of identifying behaviour.

8.5. Clinical Implications

At present, there are no studies assessing the effectiveness of interventions for disordered eating attitudes and behaviours in CD. The development and evaluation of the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* provides a framework to explore this concept and can be utilised in the development of appropriate tools, to allow prevention and intervention strategies to follow. The *Theoretical Model of Disordered Eating in Gastrointestinal Disease* can help us understand disordered eating in CD and provides insight into service development.

In combination with previous literature, the evidence throughout this thesis indicates that disordered eating attitudes and behaviours are present in CD (Chapters Two-Four, Six) and that these attitudes and behaviours are related specifically to the CD diagnosis (Chapter Three, Six). The presence of disordered eating attitudes and behaviours in CD affects both physical (gastrointestinal symptoms, GFD management) and psychosocial outcomes (quality of life, depression, anxiety, stress). As a result, these attitudes and behaviours need to be addressed in clinical practice as part of a full assessment into the management of CD and the GFD. The need to give higher priority to the psychological needs of those with CD has been consistently documented in the literature by numerous studies that show the interaction between psychological health and physical outcomes (e.g. Barratt, Leeds & Sanders, 2011; Casellas et al., 2015; Rose & Howard, 2014). Despite this evidence, psychological support is not available in the majority of gastrointestinal clinics in the UK and research assessing the impact of psychological interventions in CD is minimal (Addolorato et al., 2004; Sainsbury, Mullan & Sharpe, 2013a). Based on the current evidence, specific intervention guidelines cannot be

made. However, within the chronic health literature a patient-centred approach that emphasised patient self-management in combination with professional support is the underlying principle for interventions in chronic health conditions (Mirzaei et al., 2013). Patient-centred care has decreased symptom burden, reduced hospital admission rates and increased quality of life in a variety of chronic health conditions (Kane et al., 2015; McMillan et al., 2013; Rathert, Wyrwich & Boren, 2013). Due to the heterogeneity of disordered eating in coeliac disease, we recommend that future research focus on a personalised patient-centred approach in the assessment and care of disordered eating in CD.

8.5.1. NICE Guidelines

The current NICE guidelines for CD recommend that an individual be referred to a gastroenterology clinic when a diagnosis of CD has been confirmed (NICE, 2015). Upon diagnosis, the NICE guidelines recommend that the importance of the GFD is explained as well as information about food labelling, how to manage social situations, how to avoid cross contamination and the role of national and local CD support groups. However, the depth of information covered will largely depend on the expertise of the dietician (Madden, Riordan & Knowles, 2016). It is recommended that individuals be offered an annual review where weight and height are assessed, alongside symptoms, dietary-management and the need for specialist dietetic and nutritional advice. Further support is recommended if concerns are identified during the annual review. Despite these guidelines, follow-up at gastroenterology clinics is poor, dietetic support would need to be increased three-fold in order to provide adequate support for CD across the UK, and specialist

dietician knowledge varies across the country (Madden, Riordan & Knowles, 2016; Nelson, Mendoza & McGough, 2007).

The results of Chapter Four suggest discontent around the dietary support provided at diagnosis. Individuals expressed a desire for more information regarding potential weight change after commencing the GFD ("*I think it would be helpful if the dietician had explained the weight change was to be expected. It's unusual associating unhealthiness with thinness, it would have helped having that explained*"). This is in line with previous research, which highlights the value of dietician-led services and the desire for more dietetic support in individuals with CD (Bebb, Lawson, Knight & Long, 2006; Kurien, Trott & Sanders, 2016; Madden, Riordan & Knowles, 2016). Furthermore, disordered eating attitudes and behaviours in CD were associated with distress surrounding weight change at diagnosis (Chapters Two and Four). The current NICE guidelines do not recommend that individuals newly diagnosed with CD are consulted about the benefits of a nutritionally balanced GFD and how the initiation of the GFD may influence weight change and body shape, despite individuals with CD explaining the benefits of this type of support (Madden, Riordan & Knowles, 2016). This is surprising given the evidence for poor nutritional status in CD, which has been attributed to poor diet quality and management of the GFD (Abenavoli et al., 2015; Oso & Fraser, 2005; Theethira, Dennis & Leffler, 2014; Zuccotti et al., 2012). Therefore, it is recommended that research informing clinical guidelines should focus on the role of educating all newly diagnosed individuals with CD about the nutritional content of gluten-free foods and possible weight changes after starting the GFD, as well as how to manage these weight changes.

8.5.2. Support for Individuals with CD and Disordered Eating Attitudes and Behaviours

As discussed previously, upon CD diagnosis, dietetic support should focus on supporting healthy eating in the context of CD, promoting firstly, healthy eating but including education about the nutritional content of gluten-free foods and the potential for weight change after starting the GFD. For individuals who attend annual clinical appointments, these should be used to assess concerns that might indicate disordered eating attitudes and behaviours. The findings of this thesis indicate that healthcare professionals working with CD need educating about the symptoms of disordered eating attitudes and behaviours. Furthermore, for individuals who express concerns around food or weight, referrals should be made to a specialist dietary service where more targeted support can be provided. It is at this point of contact where the presence of more traditional eating disorders can be assessed, using the EAT-26 or the BES. The CD-FAB will be a useful tool for those who experience concerns around food and express difficulties eating outside the home. The CD-FAB, and its subscales, will allow the dietary team to assess the adaption of the individual to their GFD, it also enables assessment of food concerns, the severity of these concerns and whether these are resulting in maladaptive or adaptive behaviour. This information can be used to develop further research and patient-centred intervention plans in order to improve physical and psychosocial health outcomes in individuals with CD.

The presence of disordered eating attitudes and behaviours in CD and the complex interplay of psychosocial factors, suggests the need for psychological services in gastroenterology practices. Although dieticians and gastroenterologists

are ideally placed to reduce the risk of disordered eating through CD-related education, information on the consequences of disordered eating, and the importance of the GFD and healthy eating, psychological assessment may be beneficial for identifying individuals with disordered eating attitudes and behaviours who may be a greater risk of distress. Early referral to an eating disorder specialist should occur once a diagnosis has been confirmed to allow appropriate formulations and treatment plans to be developed.

8.5.3. Strengths, Limitations and Future Research

Throughout the thesis, a number of limitations have been noted. In this section, a number of noteworthy limitations will be noted. These limitations centre on recruitment bias, the limitations of self-report methods, the measures of disordered eating used and the limitations of cross-sectional studies.

Throughout this thesis there are limitations in terms of recruitment. Chapters Three and Four used online strategies to recruit individuals with CD. Online strategies tend to target young individuals who are considered Internet "savvy" and may direct attention away from older age groups (Remillard et al., 2014). In addition, recruitment took place via the charity Coeliac UK. Individuals recruited from Coeliac UK are predominantly female, white and well educated (Ford et al., 2012); these factors are reflected throughout our participant characteristics. To address these limitations, Chapters Five, Six and Seven used offline recruitment strategies across the University of Birmingham campus and snowball techniques. Although this addresses the limitations of online recruitment techniques, this may result in a younger and more educated sample (Sear, 1986). Despite these limitations in

sampling, the correlates of disordered eating attitudes and behaviours were consistent through all studies. Future research may focus on extending these findings to a more representative sample of individuals with CD. Given the increasing age of diagnosis within CD it is important to prioritise and see whether these findings extend to this age group (Rashtak & Murray, 2011).

The majority of chapters within this thesis have used self-report methods to assess CD diagnosis, psychological and physical well-being. These methodologies are largely limited by participant's introspective ability, whether they can take on an accurate view of their psychological well-being, and response bias is a particularly important limitation when assessing dietary management (Shim, Oh & King, 2014). Furthermore, Chapters Three-Six recruited individuals based on a self-reported diagnosis of CD. Although individuals had the option to select their method of diagnosis, we cannot confirm that all participants were biopsy confirmed. However, the results from Chapter Seven required evidence for a biopsy confirmed diagnosis for CD and the questionnaire results in this chapter replicated the results of Chapters Three and Six.

Improvements could have been made throughout the thesis by using more objective measures of dietary management such as the CD Adherence scale (Leffler et al., 2009) or dietician assessment of dietary management; however, self-report measures were a useful first step to identify the direction of associations. In addition, physical examinations may be used to assess gastrointestinal symptoms and the physical impact of disordered eating attitudes and behaviours, and assessments of antibody levels would further inform the impact on CD-related outcomes. As a result, future research should prioritise a multidisciplinary

perspective in order to assess the psychological and physical factors related to disordered eating in CD.

This thesis has used the EAT-26 and BES to evaluate the presence of disordered eating in CD. Although these tools are useful for assessing disordered eating symptoms, they cannot provide a clinical diagnosis of an eating disorder. Diagnostic clinical interviews should be used to establish the presence of clinical levels of disordered eating. Furthermore, although used in CD (Arigo et al., 2012; Karwautz et al., 2008), these tools have not been validated in a CD population. Chapter Three conducted confirmatory factor analyses on these questionnaires and identified an appropriate factor solution for the CD population, however, this needs to be replicated in larger samples before we can fully assess what these scores mean for those with CD.

The development of the CD-FAB, a CD specific tool for disordered eating attitudes and behaviours, is a considerable strength of this thesis. This tool was shown to have good psychometric properties and high scores are associated with clinical impairment in psychological distress and quality of life. However, further development is needed to assess the physical consequences of high CD-FAB scores and to establish a clinical cut off for CD-FAB scores. In addition, further development in large samples is needed to understand meaningful changes on the CD-FAB.

The majority of analyses conducted in this thesis are cross-sectional in nature, meaning the directional nature of the variables related to disordered eating attitudes and behaviours in CD could not be assessed. Although the *Theoretical Model of Disordered Eating in Gastrointestinal Disease* assumes that disordered eating attitudes and behaviours develop after CD diagnosis, due to the cross-

sectional nature of data collection, causality cannot be assumed. Gastrointestinal symptoms are frequently associated with eating disorder symptomology, however, when CD is not evident these symptoms resolve once weight has been restored (Kaltsa et al., 2015). The direction of this relationship can be understood further through the use of longitudinal studies in individuals before the point of diagnosis and throughout their years living with CD. This will not only allow a better understanding of the prevalence and factors associated with disordered eating and CD, but would shed light on the directional relationship between disordered eating and CD. However, despite these limitations, the use of mixed methodologies, using both qualitative and quantitative techniques, to explore disordered eating in the context of CD is a considerable strength of this thesis.

8.6. Conclusions

The aim of this thesis was to further our understanding of disordered eating in CD by developing and evaluating a model that emerged from a systemic review. Overall, this thesis indicates that disordered eating is prevalent in CD and is associated with factors directly related to CD management as well as more general factors such as psychological distress. In addition, the types of disordered eating attitudes and behaviours vary from Binge Eating, anorexic and bulimic thoughts to a CD-specific hyper-vigilance around food. All disordered eating types were associated with negative psychosocial outcomes. However, despite the high prevalence of disordered eating attitudes and behaviours, the majority of individuals with CD will consume a healthy GFD.

This thesis highlights how complex the management of CD can be, and the complex interactions between biological, psychological and social factors. This has implications for both researchers and clinicians working with CD. Due to the importance of physical (gastrointestinal symptoms and weight change) and psychological (anxiety, depression, stress) factors in the development of disordered eating attitudes and behaviours, this thesis highlights the need for a person-centred, biopsychosocial approach in the management of CD.

References

Abbott, J., Morton, A. M., Musson, H., Conway, S. P., Etherington, C., Gee, L., … Webb, A. K. (2007). Nutritional status, perceived body image and eating behaviours in adults with cystic fibrosis. *Clinical Nutrition (Edinburgh, Scotland)*, *26*(1), 91–99. https://doi.org/10.1016/j.clnu.2006.08.002

Abraham, S., & Kellow, J. E. (2013). Do the digestive tract symptoms in eating disorder patients represent functional gastrointestinal disorders? *BMC Gastroenterology*, *13*, 38. https://doi.org/10.1186/1471-230X-13-38

Ackard, D. M., Vik, N., Neumark-Sztainer, D., Schmitz, K. H., Hannan, P., & Jacobs, D. R. (2008). Disordered eating and body dissatisfaction in adolescents with type 1 diabetes and a population-based comparison sample: comparative prevalence and clinical implications. *Pediatric Diabetes*, *9*(4 Pt 1), 312–319. https://doi.org/10.1111/j.1399-5448.2008.00392.x

Addolorato, G., Capristo, E., Ghittoni, G., Valeri, C., Mascianà, R., Ancona, C., & Gasbarrini, G. (2001). Anxiety but not depression decreases in coeliac patients after one-year gluten-free diet: a longitudinal study. *Scandinavian Journal of Gastroenterology*, *36*(5), 502–506.

Addolorato, G., Capristo, E., Stefanini, G. F., & Gasbarrini, G. (1997). Inflammatory bowel disease: a study of the association between anxiety and depression, physical morbidity, and nutritional status. *Scandinavian Journal of Gastroenterology*, *32*(10), 1013–1021.

Addolorato, G., De Lorenzi, G., Abenavoli, L., Leggio, L., Capristo, E., & Gasbarrini, G. (2004). Psychological support counselling improves gluten-free diet compliance in coeliac patients with affective disorders. *Alimentary*

Pharmacology & Therapeutics, *20*(7), 777–782. https://doi.org/10.1111/j.1365-2036.2004.02193.x

Affenito, S. G., & Adams, C. H. (2001). Are eating disorders more prevalent in females with type 1 diabetes mellitus when the impact of insulin omission is considered? *Nutrition Reviews*, *59*(6), 179–182.

Ajzen, I. (1985). From Intentions to Actions: A Theory of Planned Behavior. In P. D. J. Kuhl & D. J. Beckmann (Eds.), *Action Control* (pp. 11–39). Springer Berlin Heidelberg. Retrieved from http://link.springer.com/chapter/10.1007/978-3-642-69746-3_2

Akey, J. E., Rintamaki, L. S., & Kane, T. L. (2013). Health Belief Model deterrents of social support seeking among people coping with eating disorders. *Journal of Affective Disorders*, *145*(2), 246–252. https://doi.org/10.1016/j.jad.2012.04.045

Allen, K. L., Byrne, S. M., & McLean, N. J. (2012). The dual-pathway and cognitive-behavioural models of binge eating: prospective evaluation and comparison. *European Child & Adolescent Psychiatry*, *21*(1), 51–62. https://doi.org/10.1007/s00787-011-0231-z

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.).

Andrés, A., & Saldaña, C. (2014). Body dissatisfaction and dietary restraint influence binge eating behavior. *Nutrition Research (New York, N.Y.)*, *34*(11), 944–950. https://doi.org/10.1016/j.nutres.2014.09.003

Arigo, D., Anskis, A. M., & Smyth, J. M. (2012). Psychiatric comorbidities in women with celiac disease. *Chronic Illness*, *8*(1), 45–55.

https://doi.org/10.1177/1742395311417639

Armstrong, R. A. (2014). When to use the Bonferroni correction. *Ophthalmic & Physiological Optics: The Journal of the British College of Ophthalmic Opticians (Optometrists)*, *34*(5), 502–508. https://doi.org/10.1111/opo.12131

Aziz, I., Karajeh, M. A., Zilkha, J., Tubman, E., Fowles, C., & Sanders, D. S. (2014). Change in awareness of gluten-related disorders among chefs and the general public in the UK: a 10-year follow-up study. *European Journal of Gastroenterology & Hepatology*, *26*(11), 1228–1233. https://doi.org/10.1097/MEG.0000000000000166

Bai, J. C., Fried, M., Corazza, G. R., Schuppan, D., Farthing, M., Catassi, C., … World Gastroenterology Organization. (2013). World Gastroenterology Organisation global guidelines on celiac disease. *Journal of Clinical Gastroenterology*, *47*(2), 121–126. https://doi.org/10.1097/MCG.0b013e31827a6f83

Bakshi, A., Stephen, S., Borum, M. L., & Doman, D. B. (2012). Emerging Therapeutic Options for Celiac Disease. *Gastroenterology & Hepatology*, *8*(9), 582–588.

Bao, F., & Bhagat, G. (2012). Histopathology of Celiac Disease. *Gastrointestinal Endoscopy Clinics of North America*, *22*(4), 679–694. https://doi.org/10.1016/j.giec.2012.07.001

Bao, F., Green, P. H. ., & Bhagat, G. (2012). An Update on Celiac Disease Histopathology and the Road Ahead. *Archives of Pathology & Laboratory Medicine*, *136*(7), 735–745. https://doi.org/10.5858/arpa.2011-0572-RA

Barratt, S. M., Leeds, J. S., & Sanders, D. S. (2011). Quality of life in Coeliac Disease is determined by perceived degree of difficulty adhering to a gluten-free diet, not the level of dietary adherence ultimately achieved. *Journal of*

Gastrointestinal and Liver Diseases: JGLD, *20*(3), 241–245.

Baum, A. (1997). *Cambridge Handbook of Psychology, Health and Medicine*. Cambridge University Press.

Baylé, F. J., & Bouvard, M. P. (2003). Anorexia nervosa and Crohn's disease dual diagnosis: a case study. *European Psychiatry: The Journal of the Association of European Psychiatrists*, *18*(8), 421–422.

Bebb, J. R., Lawson, A., Knight, T., & Long, R. G. (2006). Long-term follow-up of coeliac disease--what do coeliac patients want? *Alimentary Pharmacology & Therapeutics*, *23*(6), 827–831. https://doi.org/10.1111/j.1365-2036.2006.02824.x

Beck, A. T., Emery, G., & Greenberg, R. L. (2005). *Anxiety Disorders and Phobias: A Cognitive Perspective*. Basic Books.

Belsley, D. A., Kuh, E., & Welsch, R. E. (1980). Detecting and Assessing Collinearity. In *Regression Diagnostics* (pp. 85–191). John Wiley & Sons, Inc. Retrieved from http://onlinelibrary.wiley.com/doi/10.1002/0471725153.ch3/summary

Bernstein, I. L., & Borson, S. (1986). Learned food aversion: a component of anorexia syndromes. *Psychological Review*, *93*(4), 462–472.

Biagetti, C., Naspi, G., & Catassi, C. (2013). Health-Related Quality of Life in Children with Celiac Disease: A Study Based on the Critical Incident Technique. *Nutrients*, *5*(11), 4476–4485. https://doi.org/10.3390/nu5114476

Biggs, M. M., Basco, M. R., Patterson, G., & Raskin, P. (1994). Insulin withholding for weight control in women with diabetes. *Diabetes Care*, *17*(10), 1186–1189.

Bryon, M., Shearer, J., & Davies, H. (2008). Eating Disorders and Disturbance in Children and Adolescents With Cystic Fibrosis. *Children's Health Care*, *37*(1),

67–77. https://doi.org/10.1080/02739610701766909

Burden, M., Mooney, P. D., Blanshard, R. J., White, W. L., Cambray-Deakin, D. R., & Sanders, D. S. (2015). Cost and availability of gluten-free food in the UK: in store and online. *Postgraduate Medical Journal*, *91*(1081), 622–626. https://doi.org/10.1136/postgradmedj-2015-133395

Burger, J. P. W., de Brouwer, B., IntHout, J., Wahab, P. J., Tummers, M., & Drenth, J. P. H. (n.d.). Systematic review with meta-analysis: Dietary adherence influences normalization of health-related quality of life in coeliac disease. *Clinical Nutrition*. https://doi.org/10.1016/j.clnu.2016.04.021

Capristo, E., Addolorato, G., Mingrone, G., De Gaetano, A., Greco, A. V., Tataranni, P. A., & Gasbarrini, G. (2000). Changes in body composition, substrate oxidation, and resting metabolic rate in adult celiac disease patients after a 1-y gluten-free diet treatment. *The American Journal of Clinical Nutrition*, *72*(1), 76–81.

Carpenter, C. J. (2010). A meta-analysis of the effectiveness of health belief model variables in predicting behavior. *Health Communication*, *25*(8), 661–669. https://doi.org/10.1080/10410236.2010.521906

Carvajal, A., Centeno, C., Watson, R., & Bruera, E. (2011). A comprehensive study of psychometric properties of the Edmonton Symptom Assessment System (ESAS) in Spanish advanced cancer patients. *European Journal of Cancer*, *47*(12), 1863–1872. https://doi.org/10.1016/j.ejca.2011.03.027

Casellas, F., Rodrigo, L., Lucendo, A. J., Fernández-Bañares, F., Molina-Infante, J., Vivas, S., … López-Vivancos, J. (2015). Benefit on health-related quality of life of adherence to gluten-free diet in adult patients with celiac disease. *Revista*

Española De Enfermedades Digestivas: Organo Oficial De La Sociedad Española De Patología Digestiva, *107*(4), 196–201.

Cassin, S. E., & von Ranson, K. M. (2005). Personality and eating disorders: a decade in review. *Clinical Psychology Review*, *25*(7), 895–916. https://doi.org/10.1016/j.cpr.2005.04.012

Cattarin, J. A., & Thompson, J. K. (1994). A Three-Year Longitudinal Study of Body Image, Eating Disturbance, and General Psychological Functioning in Adolescent Females. *Eating Disorders*, *2*(2), 114–125. https://doi.org/10.1080/10640269408249107

Mahan, K. (2011). *Krause's Food & the Nutrition Care Process,* (13 edition). St. Louis, Mo: Saunders.

Cheng, J., Brar, P. S., Lee, A. R., & Green, P. H. R. (2010). Body mass index in celiac disease: beneficial effect of a gluten-free diet. *Journal of Clinical Gastroenterology*, *44*(4), 267–271. https://doi.org/10.1097/MCG.0b013e3181b7ed58

Chowdhury, M. K., Chakraborty, R., Gope, S., Rahman, M. A., Miah, A. R., Raihan, A. S., … Ferdousi, K. R. (2016). Celiac Disease in Patients Fulfilling the Rome III Criteria for Irritable Bowel Syndrome Attending Gastroenterology Department of A Tertiary Care Hospital in Bangladesh. *Mymensingh Medical Journal : MMJ*, *25*(1), 102–108.

Ciacci, C., Cirillo, M., Sollazzo, R., Savino, G., Sabbatini, F., & Mazzacca, G. (1995). Gender and Clinical Presentation in Adult Celiac Disease. *Scandinavian Journal of Gastroenterology*, *30*(11), 1077–1081. https://doi.org/10.3109/00365529509101610

Ciacci, C., Iavarone, A., Siniscalchi, M., Romano, R., & De Rosa, A. (2002). Psychological dimensions of celiac disease: toward an integrated approach. *Digestive Diseases and Sciences*, *47*(9), 2082–2087.

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of Attentional Biases towards Threat in the Anxiety Disorders: An Integrative Review. *Clinical Psychology Review*, *30*(2), 203. https://doi.org/10.1016/j.cpr.2009.11.003

Coa, K. I., Epstein, J. B., Ettinger, D., Jatoi, A., McManus, K., Platek, M. E., … Moskowitz, B. (2015). The Impact of Cancer Treatment on the Diets and Food Preferences of Patients Receiving Outpatient Treatment. *Nutrition and Cancer*, *67*(2), 339–353. https://doi.org/10.1080/01635581.2015.990577

Collin, P., Thorell, L., Kaukinen, K., & Mäki, M. (2004). The safe threshold for gluten contamination in gluten-free products. Can trace amounts be accepted in the treatment of coeliac disease? *Alimentary Pharmacology & Therapeutics*, *19*(12), 1277–1283. https://doi.org/10.1111/j.1365-2036.2004.01961.x

Colton, P. A., Olmsted, M. P., Daneman, D., & Rodin, G. M. (2013). Depression, disturbed eating behavior, and metabolic control in teenage girls with type 1 diabetes. *Pediatric Diabetes*, *14*(5), 372–376. https://doi.org/10.1111/pedi.12016

Colton, P. A., Rodin, G. M., Olmsted, M. P., & Daneman, D. (1999). Eating disturbances in young women with type I diabetes mellitus: Mechanisms and consequences. *Psychiatric Annals*, *29*(4), 213–218.

Colton, P., Olmsted, M., Daneman, D., Rydall, A., & Rodin, G. (2004). Disturbed Eating Behavior and Eating Disorders in Preteen and Early Teenage Girls With Type 1 Diabetes. *Diabetes Care*, *27*(7), 1654–1659.

https://doi.org/10.2337/diacare.27.7.1654

Crow, S., Kendall, D., Praus, B., & Thuras, P. (2001). Binge eating and other psychopathology in patients with type II diabetes mellitus. *The International Journal of Eating Disorders*, *30*(2), 222–226.

Dandeneau, S. D., Baldwin, M. W., Baccus, J. R., Sakellaropoulo, M., & Pruessner, J. C. (2007). Cutting stress off at the pass: reducing vigilance and responsiveness to social threat by manipulating attention. *Journal of Personality and Social Psychology*, *93*(4), 651–666. https://doi.org/10.1037/0022-3514.93.4.651

Daum, S., Cellier, C., & Mulder, C. J. J. (2005). Refractory coeliac disease. *Best Practice & Research. Clinical Gastroenterology*, *19*(3), 413–424. https://doi.org/10.1016/j.bpg.2005.02.001

Davidson, J. (2014). Diabulimia: how eating disorders can affect adolescents with diabetes. *Nursing Standard (Royal College of Nursing (Great Britain): 1987)*, *29*(2), 44–49. https://doi.org/10.7748/ns.29.2.44.e7877

de Rosa, A., Troncone, A., Vacca, M., & Ciacci, C. (2004). Characteristics and quality of illness behavior in celiac disease. *Psychosomatics*, *45*(4), 336–342. https://doi.org/10.1176/appi.psy.45.4.336

Denham, J. M., & Hill, I. D. (2013). Celiac Disease and Autoimmunity: Review and Controversies. *Current Allergy and Asthma Reports*, *13*(4), 347–353. https://doi.org/10.1007/s11882-013-0352-1

DeVellis, R. F. (2003). *Scale Development: Theory and Applications*. Sage.

Dickey, W., & Kearney, N. (2006). Overweight in celiac disease: prevalence, clinical characteristics, and effect of a gluten-free diet. *The American Journal of Gastroenterology*, *101*(10), 2356–2359. https://doi.org/10.1111/j.1572-

0241.2006.00750.x

Didie, E. R., & Fitzgibbon, M. (2005). Binge eating and psychological distress: is the degree of obesity a factor? *Eating Behaviors*, *6*(1), 35–41. https://doi.org/10.1016/j.eatbeh.2004.08.007

Dieli-Crimi, R., Cénit, M. C., & Núñez, C. (2015). The genetics of celiac disease: A comprehensive review of clinical implications. *Journal of Autoimmunity*, *64*, 26–41. https://doi.org/10.1016/j.jaut.2015.07.003

Dietary Intake and Nutritional Deficiencies in Patients with Diabetic or Idiopathic Gastroparesis. (2011). *Gastroenterology*, *141*(2), 486–498.e7. https://doi.org/10.1053/j.gastro.2011.04.045

DiMatteo, M. R., Haskard, K. B., & Williams, S. L. (2007). Health beliefs, disease severity, and patient adherence: a meta-analysis. *Medical Care*, *45*(6), 521–528. https://doi.org/10.1097/MLR.0b013e318032937e

Dorn, S. D., Hernandez, L., Minaya, M. T., Morris, C. B., Hu, Y., Leserman, J., … Drossman, D. A. (2010). The development and validation of a new coeliac disease quality of life survey (CD-QOL). *Alimentary Pharmacology & Therapeutics*, *31*(6), 666–675. https://doi.org/10.1111/j.1365-2036.2009.04220.x

Dowd, A. J., Tamminen, K. A., Jung, M. E., Case, S., McEwan, D., & Beauchamp, M. R. (2014). Motives for adherence to a gluten-free diet: a qualitative investigation involving adults with coeliac disease. *Journal of Human Nutrition and Dietetics*, *27*(6), 542–549. https://doi.org/10.1111/jhn.12203

Duarte, C., Pinto-Gouveia, J., & Ferreira, C. (2015). Expanding binge eating assessment: Validity and screening value of the Binge Eating Scale in women

from the general population. *Eating Behaviors*, *18*, 41–47. https://doi.org/10.1016/j.eatbeh.2015.03.007

Dury, R. (2015). Mental and physical long-term conditions in the UK: spanning the boundaries. *British Journal of Community Nursing*, *20*(4), 190; 192-193. https://doi.org/10.12968/bjcn.2015.20.4.190

Eddy, K. T., Dorer, D. J., Franko, D. L., Tahilani, K., Thompson-Brenner, H., & Herzog, D. B. (2008). Diagnostic crossover in anorexia nervosa and bulimia nervosa: implications for DSM-V. *The American Journal of Psychiatry*, *165*(2), 245–250. https://doi.org/10.1176/appi.ajp.2007.07060951

Edwards George, J. B., Leffler, D. A., Dennis, M. D., Franko, D. L., Blom-Hoffman, J., & Kelly, C. P. (2009). Psychological Correlates of Gluten-free Diet Adherence in Adults With Celiac Disease: *Journal of Clinical Gastroenterology*, *43*(4), 301–306. https://doi.org/10.1097/MCG.0b013e31816a8c9b

Elfström, P., Montgomery, S. M., Kämpe, O., Ekbom, A., & Ludvigsson, J. F. (2008). Risk of thyroid disease in individuals with celiac disease. *The Journal of Clinical Endocrinology and Metabolism*, *93*(10), 3915–3921. https://doi.org/10.1210/jc.2008-0798

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science*, *196*(4286), 129–136. https://doi.org/10.1126/science.847460

Engström, I., Kroon, M., Arvidsson, C.-G., Segnestam, K., Snellman, K., & Åman, J. (1999). Eating disorders in adolescent girls with insulin-dependent diabetes mellitus: a population-based case-control study. *Acta Pædiatrica*, *88*(2), 175–180. https://doi.org/10.1111/j.1651-2227.1999.tb01078.x

Estévez, V., Ayala, J., Vespa, C., & Araya, M. (2016). The gluten-free basic food

basket: a problem of availability, cost and nutritional composition. *European Journal of Clinical Nutrition*. https://doi.org/10.1038/ejcn.2016.139

Eubanks, V., Koppersmith, N., Wooldridge, N., Clancy, J. P., Lyrene, R., Arani, R. B., … Makris, C. M. (2002). Effects of megestrol acetate on weight gain, body composition, and pulmonary function in patients with cystic fibrosis. *The Journal of Pediatrics*, *140*(4), 439–444. https://doi.org/10.1067/mpd.2002.121936

Fasano, A., & Catassi, C. (2012). Clinical practice. Celiac disease. *The New England Journal of Medicine*, *367*(25), 2419–2426. https://doi.org/10.1056/NEJMcp1113994

Faunce, G. J. (2002). Eating disorders and attentional bias: a review. *Eating Disorders*, *10*(2), 125–139. https://doi.org/10.1080/10640260290081696

Fjellström, K. (2008). Mealtime and meal patterns from a cultural perspective. *Food & Nutrition Research*, *48*(4), 161–164. https://doi.org/10.3402/fnr.v48i4.1630

Fletcher, P. C., Jamieson, A. E., Schneider, M. A., & Harry, R. J. (2008). "I know this is bad for me, but...": a qualitative investigation of women with irritable bowel syndrome and inflammatory bowel disease: part II. *Clinical Nurse Specialist CNS*, *22*(4), 184–191. https://doi.org/10.1097/01.NUR.0000311707.32566.c8

Ford, S., Howard, R., & Oyebode, J. (2012). Psychosocial aspects of coeliac disease: a cross-sectional survey of a UK population. *British Journal of Health Psychology*, *17*(4), 743–757. https://doi.org/10.1111/j.2044-8287.2012.02069.x

Freeland-Graves, J. H., & Nitzke, S. (2013). Position of the Academy of Nutrition and Dietetics: Total Diet Approach to Healthy Eating. *Journal of the Academy of*

Nutrition and Dietetics, *113*(2), 307–317. https://doi.org/10.1016/j.jand.2012.12.013

Freeman, H. J. (2008). Pearls and pitfalls in the diagnosis of adult celiac disease. *Canadian Journal of Gastroenterology = Journal Canadien De Gastroenterologie*, *22*(3), 273–280.

Fuchs, V., Kurppa, K., Huhtala, H., Collin, P., Mäki, M., & Kaukinen, K. (2014). Factors associated with long diagnostic delay in celiac disease. *Scandinavian Journal of Gastroenterology*, *49*(11), 1304–1310. https://doi.org/10.3109/00365521.2014.923502

Garcia, J., Kimeldorf, D. J., & Koelling, R. A. (1955). Conditioned aversion to saccharin resulting from exposure to gamma radiation. *Science (New York, N.Y.)*, *122*(3160), 157–158.

García-Manzanares, A., & Lucendo, A. J. (2011). Nutritional and dietary aspects of celiac disease. *Nutrition in Clinical Practice: Official Publication of the American Society for Parenteral and Enteral Nutrition*, *26*(2), 163–173. https://doi.org/10.1177/0884533611399773

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: an index of the symptoms of anorexia nervosa. *Psychological Medicine*, *9*(2), 273–279. https://doi.org/10.1017/S0033291700030762

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine*, *12*(4), 871–878.

Gibson, P. R., & Shepherd, S. J. (2010). Evidence-based dietary management of functional gastrointestinal symptoms: The FODMAP approach. *Journal of*

Gastroenterology and Hepatology, *25*(2), 252–258. https://doi.org/10.1111/j.1440-1746.2009.06149.x

Gilbert, P., & Miles, J. (Eds.). (2002). *Body Shame: Conceptualisation, Research and Treatment*. Hove, East Sussex ; New York: Routledge.

Gilchrist, F. J., & Lenney, W. (2008). Distorted body image and anorexia complicating cystic fibrosis in an adolescent. *Journal of Cystic Fibrosis*, *7*(5), 437–439. https://doi.org/10.1016/j.jcf.2008.04.003

Godart, N. T., Perdereau, F., Rein, Z., Berthoz, S., Wallier, J., Jeammet, P., & Flament, M. F. (2007). Comorbidity studies of eating disorders and mood disorders. Critical review of the literature. *Journal of Affective Disorders*, *97*(1–3), 37–49. https://doi.org/10.1016/j.jad.2006.06.023

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*(1), 47–55.

Gray, A. M., & Papanicolas, I. N. (2010). Impact of symptoms on quality of life before and after diagnosis of coeliac disease: results from a UK population survey. *BMC Health Services Research*, *10*, 105. https://doi.org/10.1186/1472-6963-10-105

Green, P. H. R., & Jabri, B. (2003). Coeliac disease. *Lancet (London, England)*, *362*(9381), 383–391. https://doi.org/10.1016/S0140-6736(03)14027-5

Green P. H. R., Stavropoulos, S. N., Panagi, S. G., Goldstein, S. L., Mcmahon, D. J., Absan, H., & Neugut, A. I. (2001). Characteristics of adult celiac disease in the USA: results of a national survey. *The American Journal of Gastroenterology*, *96*(1), 126–131. https://doi.org/10.1111/j.1572-0241.2001.03462.x

Grilo, C. (2006). *Eating and weight disorders*. New York: Psychology Press.

Guthrie, E. A., Creed, F. H., & Whorwell, P. J. (1990). Eating disorders in patients with the irritable bowel syndrome: A comparison with inflammatory bowel disease and peptic ulceration. *European Journal of Gastroenterology and Hepatology, 6,* 471-473.

Hagger, V., Hendrieckx, C., Sturt, J., Skinner, T. C., & Speight, J. (2016). Diabetes Distress Among Adolescents with Type 1 Diabetes: a Systematic Review. *Current Diabetes Reports*, *16*(1), 9. https://doi.org/10.1007/s11892-015-0694-2

Hakanson, C., Sahlberg-Blom, E., Nyhln, H., & Ternestedt, B. (2009). Struggling with an unfamiliar and unreliable body: the experience of irritable bowel syndrome. *Journal of Nursing and Healthcare of Chronic Illness, 1*(1), 29-38.

Hall, N. J., Rubin, G., & Charnock, A. (2009). Systematic review: adherence to a gluten-free diet in adult patients with coeliac disease. *Alimentary Pharmacology & Therapeutics*, *30*(4), 315–330. https://doi.org/10.1111/j.1365-2036.2009.04053.x

Hall, N. J., Rubin, G. P., & Charnock, A. (2013). Intentional and inadvertent non-adherence in adult coeliac disease. A cross-sectional survey. *Appetite*, *68*, 56–62. https://doi.org/10.1016/j.appet.2013.04.016

Hallert, C., Grant, C., Grehn, S., Grännö, C., Hultén, S., Midhagen, G., … Valdimarsson, T. (2002). Evidence of poor vitamin status in coeliac patients on a gluten-free diet for 10 years. *Alimentary Pharmacology & Therapeutics*, *16*(7), 1333–1339.

Harrison, J. A., Mullen, P. D., & Green, L. W. (1992). A meta-analysis of studies of the Health Belief Model with adults. *Health Education Research*, *7*(1), 107–116.

https://doi.org/10.1093/her/7.1.107

Häuser, W., Janke, K.-H., Klump, B., Gregor, M., & Hinz, A. (2010). Anxiety and depression in adult patients with celiac disease on a gluten-free diet. *World Journal of Gastroenterology : WJG*, *16*(22), 2780–2787. https://doi.org/10.3748/wjg.v16.i22.2780

Hayes, D. R., Sheehan, J. P., Ulchaker, M. M., & Rebar, J. M. (1994). Management dilemmas in the individual with cystic fibrosis and diabetes. *Journal of the American Dietetic Association*, *94*(1), 78–80.

Hayes, S., & Napolitano, M. A. (2012). Examination of weight control practices in a non-clinical sample of college women. *Eating and Weight Disorders: EWD*, *17*(3), e157-163.

Heatherton, T. F., Herman, C. P., & Polivy, J. (1991). Effects of physical threat and ego threat on eating behavior. *Journal of Personality and Social Psychology*, *60*(1), 138–143.

Herman, C. P., & Polivy, J. (2005). Normative influences on food intake. *Physiology & Behavior*, *86*(5), 762–772. https://doi.org/10.1016/j.physbeh.2005.08.064

Herpertz, S., Albus, C., Kielmann, R., Hagemann-Patt, H., Lichtblau, K., Köhle, K., … Senf, W. (2001). Comorbidity of diabetes mellitus and eating disorders: a follow-up study. *Journal of Psychosomatic Research*, *51*(5), 673–678.

Higginbotham, N., Albrecht, G., & Connor, L. (2001). *Health Social Science: A Transdisciplinary and Complexity Perspective*. Oxford University Press.

Hilbert, A., Tuschen-Caffier, B., Karwautz, A., Niederhofer, H., & Munsch, S. (2007). Eating Disorder Examination-Questionnaire. *Diagnostica*, *53*(3), 144–154. https://doi.org/10.1026/0012-1924.53.3.144

Högberg, L., Grodzinsky, E., & Stenhammar, L. (2003). Better dietary compliance in patients with coeliac disease diagnosed in early childhood. *Scandinavian Journal of Gastroenterology*, *38*(7), 751–754.

Hopper, A. D., Hadjivassiliou, M., Butt, S., & Sanders, D. S. (2007). Adult coeliac disease. *BMJ : British Medical Journal*, *335*(7619), 558–562. https://doi.org/10.1136/bmj.39316.442338.AD

Hourigan, C. S. (2006). The molecular basis of coeliac disease. *Clinical and Experimental Medicine*, *6*(2), 53–59. https://doi.org/10.1007/s10238-006-0095-6

Husby, S., Koletzko, S., Korponay-Szabó, I. R., Mearin, M. L., Phillips, A., Shamir, R., … Zimmer, K. P. (2012). European Society for Pediatric Gastroenterology, Hepatology, and Nutrition Guidelines for the Diagnosis of Coeliac Disease: *Journal of Pediatric Gastroenterology and Nutrition*, *54*(1), 136–160. https://doi.org/10.1097/MPG.0b013e31821a23d0

Hauser, W., Gold, J., Stallmach, A., Caspary, W. F., & Stein, J. (2007). Development and Validation of the Celiac Disease Questionnaire (CDQ), a Disease-specific Health-related Quality of Life Measure for Adult Patients With Celiac Disease: *Journal of Clinical Gastroenterology*, *41*(2), 157–166. https://doi.org/10.1097/01.mcg.0000225516.05666.4e

Hauser, W., Gold, J., Stein, J., Caspary, W. F., & Stallmach, A. (2006). Health-related quality of life in adult coeliac disease in Germany: results of a national survey: *European Journal of Gastroenterology & Hepatology*, *18*(7), 747–754. https://doi.org/10.1097/01.meg.0000221855.19201.e8

Ismail, I. S., Nazaimoon, W. M., Mohamad, W. B., Letchuman, R., Singaraveloo, M.,

Pendek, R., … Khalid, B. A. (2000). Sociodemographic determinants of glycaemic control in young diabetic patients in peninsular Malaysia. *Diabetes Research and Clinical Practice*, *47*(1), 57–69.

Jacobsson, L. R., Hallert, C., Milberg, A., & Friedrichsen, M. (2012). Coeliac disease--women's experiences in everyday life. *Journal of Clinical Nursing*, *21*(23–24), 3442–3450. https://doi.org/10.1111/j.1365-2702.2012.04279.x

Janz, N. K., & Becker, M. H. (1984). The Health Belief Model: a decade later. *Health Education Quarterly*, *11*(1), 1–47.

Johnson, B., Eiser, C., Young, V., Brierley, S., & Heller, S. (2013). Prevalence of depression among young people with Type 1 diabetes: a systematic review. *Diabetic Medicine*, *30*(2), 199–208. https://doi.org/10.1111/j.1464-5491.2012.03721.x

Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*, *36*(5), 665–674. https://doi.org/10.1038/ijo.2011.156

Johnston, S. D., Rodgers, C., & Watson, R. G. (2004). Quality of life in screen-detected and typical coeliac disease and the effect of excluding dietary gluten. *European Journal of Gastroenterology & Hepatology*, *16*(12), 1281–1286.

Jones, C. J., Smith, H. E., Frew, A. J., Toit, G. D., Mukhopadhyay, S., & Llewellyn, C. D. (2014). Explaining adherence to self-care behaviours amongst adolescents with food allergy: A comparison of the health belief model and the common sense self-regulation model. *British Journal of Health Psychology*, *19*(1), 65–82. https://doi.org/10.1111/bjhp.12033

Jones, J. M., Lawson, M. L., Daneman, D., Olmsted, M. P., & Rodin, G. (2000). Eating

disorders in adolescent females with and without type 1 diabetes: cross sectional study. *BMJ (Clinical Research Ed.)*, *320*(7249), 1563–1566.

Kabbani, T. A., Goldberg, A., Kelly, C. P., Pallav, K., Tariq, S., Peer, A., … Leffler, D. A. (2012). Body mass index and the risk of obesity in coeliac disease treated with the gluten-free diet. *Alimentary Pharmacology & Therapeutics*, *35*(6), 723–729. https://doi.org/10.1111/j.1365-2036.2012.05001.x

Kabir, A., Deb, S. R., Akhter, R., Bhuyian, M. R., & Hasan, P. (2016). Diagnostic delay in clinical practice: A case report of Coeliac disease. *Bangabandhu Sheikh Mujib Medical University Journal*, *7*(1), 68–70. https://doi.org/10.3329/bsmmuj.v7i1.29152

Kaltsa, M, Garoufi, A., Tsitsika, A., Tsirogianni, A., Papasteriades, C., & Kossiva, L. (2015). Patients with eating disorders showed no signs of coeliac disease before and after nutritional intervention. Acta Paediatrica, 104, e319–323. doi:10.1111/apa.12985

Karajeh, M. A., Hurlstone, D. P., Patel, T. M., & Sanders, D. S. (2005). Chefs' knowledge of coeliac disease (compared to the public): a questionnaire survey from the United Kingdom. *Clinical Nutrition (Edinburgh, Scotland)*, *24*(2), 206–210. https://doi.org/10.1016/j.clnu.2004.08.006

Karwautz, A., Wagner, G., Berger, G., Sinnreich, U., Grylli, V., & Huber, W.-D. (2008). Eating pathology in adolescents with celiac disease. *Psychosomatics*, *49*(5), 399–406. https://doi.org/10.1176/appi.psy.49.5.399

Kaswala, D. H., Veeraraghavan, G., Kelly, C. P., & Leffler, D. A. (2015). Celiac Disease: Diagnostic Standards and Dilemmas. *Diseases*, *3*(2), 86–101. https://doi.org/10.3390/diseases3020086

Kaukinen, K., Lindfors, K., Collin, P., Koskinen, O., & Mäki, M. (2010). Coeliac disease--a diagnostic and therapeutic challenge. *Clinical Chemistry and Laboratory Medicine*, *48*(9), 1205–1216. https://doi.org/10.1515/CCLM.2010.241

Khan, N. Z., Ferdous, S., Islam, R., Sultana, A., Durkin, M., & McConachie, H. (2009). Behaviour Problems in Young Children in Rural Bangladesh. *Journal of Tropical Pediatrics*, *55*(3), 177–182. https://doi.org/10.1093/tropej/fmn108

Kmet, L. M., Lee, R. C., & Cook, L. S. (2004). Standard quality assessment criteria for evaluating primary research papers from a variety of fields. *AHFMR, HTA Initiative, 97,* 1-21.

Koch, T. (1994). Establishing rigour in qualitative research: the decision trail. *Journal of Advanced Nursing*, *19*(5), 976–986.

Koro, C. E., Bowlin, S. J., Bourgeois, N., & Fedder, D. O. (2004). Glycemic control from 1988 to 2000 among U.S. adults diagnosed with type 2 diabetes: a preliminary report. *Diabetes Care*, *27*(1), 17–20.

Kosky, N., McCluskey, S., & Lacey, J. H. (1993). Bulimia nervosa and food allergy: A case report. *International Journal of Eating Disorders*, *14*(1), 117–119. https://doi.org/10.1002/1098-108X(199307)14:1<117::AID-EAT2260140116>3.0.CO;2-H

Kothe, E. J., Sainsbury, K., Smith, L., & Mullan, B. A. (2015). Explaining the intention-behaviour gap in gluten-free diet adherence: The moderating roles of habit and perceived behavioural control. *Journal of Health Psychology*, *20*(5), 580–591. https://doi.org/10.1177/1359105315576606

Kroon Van Diest, A. M., & Perez, M. (2013). Exploring the integration of thin-ideal internalization and self-objectification in the prevention of eating disorders.

Body Image, *10*(1), 16–25. https://doi.org/10.1016/j.bodyim.2012.10.004

Kupper, C. (2005). Dietary guidelines and implementation for celiac disease. *Gastroenterology*, *128*(4 Suppl 1), S121-127.

Kurien, M., Trott, N., & Sanders, D. S. (2016). Long-term care for patients with coeliac disease in the UK: a review of the literature and future directions. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*. https://doi.org/10.1111/jhn.12379

Kurppa, K., Collin, P., Mäki, M., & Kaukinen, K. (2011). Celiac disease and health-related quality of life. *Expert Review of Gastroenterology & Hepatology*, *5*(1), 83–90. https://doi.org/10.1586/egh.10.81

Kurppa, K., Lauronen, O., Collin, P., Ukkola, A., Laurila, K., Huhtala, H., … Kaukinen, K. (2012). Factors Associated with Dietary Adherence in Celiac Disease: A Nationwide Study. *Digestion*, *86*(4), 309–314. https://doi.org/10.1159/000341416

La Vieille, S., bastien, Pulido, O. M., Abbott, M., Koerner, T. B., & Godefroy, S. (2016). Celiac Disease and Gluten-Free Oats: A Canadian Position Based on a Literature Review. *Canadian Journal of Gastroenterology and Hepatology*, *2016*, e1870305. https://doi.org/10.1155/2016/1870305

Lambert, K., & Ficken, C. (2016). Cost and affordability of a nutritionally balanced gluten-free diet: Is following a gluten-free diet affordable? *Nutrition & Dietetics*, *73*(1), 36–42. https://doi.org/10.1111/1747-0080.12171

Larger, E. (2005). Weight gain and insulin treatment. *Diabetes & Metabolism*, *31*(4 Pt 2), 4S51-54S56.

Lavender, J. M., Wonderlich, S. A., Engel, S. G., Gordon, K. H., Kaye, W. H., & Mitchell,

J. E. (2015). Dimensions of emotion dysregulation in anorexia nervosa and bulimia nervosa: A conceptual review of the empirical literature. *Clinical Psychology Review*, *40*, 111–122. https://doi.org/10.1016/j.cpr.2015.05.010

Lee, A. R., Ng, D. L., Diamond, B., Ciaccio, E. J., & Green, P. H. R. (2012). Living with coeliac disease: survey results from the U.S.A. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *25*(3), 233–238. https://doi.org/10.1111/j.1365-277X.2012.01236.x

Lee, A. R., Ng, D. L., Zivin, J., & Green, P. H. R. (2007). Economic burden of a gluten-free diet. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *20*(5), 423–430. https://doi.org/10.1111/j.1365-277X.2007.00763.x

Lee, H. J., Anderson, Z., & Ryu, D. (2014). Gluten Contamination in Foods Labeled as "Gluten Free" in the United States. *Journal of Food Protection*, *77*(10), 1830–1833. https://doi.org/10.4315/0362-028X.JFP-14-149

Lee, S. K., & Green, P. H. R. (2005). Endoscopy in celiac disease. *Current Opinion in Gastroenterology*, *21*(5), 589–594.

Leffler, D. A., Dennis, M., Edwards George, J. B., Jamma, S., Magge, S., Cook, E. F., … Kelly, C. P. (2009). A simple validated gluten-free diet adherence survey for adults with celiac disease. *Clinical Gastroenterology and Hepatology: The Official Clinical Practice Journal of the American Gastroenterological Association*, *7*(5), 530–536, 536–2. https://doi.org/10.1016/j.cgh.2008.12.032

Leffler, D. A., Dennis, M., Edwards George, J. B., & Kelly, C. P. (2007). The interaction between eating disorders and celiac disease: an exploration of 10 cases. *European Journal of Gastroenterology & Hepatology*, *19*(3), 251–255.

https://doi.org/10.1097/MEG.0b013e328012db37

Lohi, S., Mustalahti, K., Kaukinen, K., Laurila, K., Collin, P., Rissanen, H., … Mäki, M. (2007). Increasing prevalence of coeliac disease over time. *Alimentary Pharmacology & Therapeutics*, *26*(9), 1217–1225. https://doi.org/10.1111/j.1365-2036.2007.03502.x

Lohiniemi, S., Mäki, M., Kaukinen, K., Laippala, P., & Collin, P. (2000). Gastrointestinal symptoms rating scale in coeliac disease patients on wheat starch-based gluten-free diets. *Scandinavian Journal of Gastroenterology*, *35*(9), 947–949.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy*, *33*(3), 335–343.

Ludvigsson, J. F., Bai, J. C., Biagi, F., Card, T. R., Ciacci, C., Ciclitira, P. J., … Sanders, D. S. (2014). Diagnosis and management of adult coeliac disease: guidelines from the British Society of Gastroenterology. *Gut*, *63*(8), 1210–1228. https://doi.org/10.1136/gutjnl-2013-306578

Ludvigsson, J. F., Leffler, D. A., Bai, J. C., Biagi, F., Fasano, A., Green, P. H. R., … Ciacci, C. (2013). The Oslo definitions for coeliac disease and related terms. *Gut*, *62*(1), 43–52. https://doi.org/10.1136/gutjnl-2011-301346

Ludvigsson, J. F., Ludvigsson, J., Ekbom, A., & Montgomery, S. M. (2006). Celiac disease and risk of subsequent type 1 diabetes: a general population cohort study of children and adolescents. *Diabetes Care*, *29*(11), 2483–2488. https://doi.org/10.2337/dc06-0794

Ludvigsson, J. F., Reutfors, J., Ösby, U., Ekbom, A., & Montgomery, S. M. (2007).

Coeliac disease and risk of mood disorders — A general population-based cohort study. *Journal of Affective Disorders*, *99*(1–3), 117–126. https://doi.org/10.1016/j.jad.2006.08.032

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*(1), 15–20.

Madden, A. M., Riordan, A. M., & Knowles, L. (2016). Outcomes in coeliac disease: a qualitative exploration of patients' views on what they want to achieve when seeing a dietitian. *Journal of Human Nutrition and Dietetics*, *29*(5), 607–616. https://doi.org/10.1111/jhn.12378

Mallett, P., & Murch, S. (1990). Anorexia nervosa complicating inflammatory bowel disease. *Archives of Disease in Childhood*, *65*(3), 298–300.

Mariani, P., Viti, M. G., Montuori, M., La Vecchia, A., Cipolletta, E., Calvani, L., & Bonamico, M. (1998). The gluten-free diet: a nutritional risk factor for adolescents with celiac disease? *Journal of Pediatric Gastroenterology and Nutrition*, *27*(5), 519–523.

Markowitz, J. T., Butler, D. A., Volkening, L. K., Antisdel, J. E., Anderson, B. J., & Laffel, L. M. B. (2010). Brief screening tool for disordered eating in diabetes: internal consistency and external validity in a contemporary sample of pediatric patients with type 1 diabetes. *Diabetes Care*, *33*(3), 495–500. https://doi.org/10.2337/dc09-1890

Marteau, T. M., Ogilvie, D., Roland, M., Suhrcke, M., & Kelly, M. P. (2011). Judging nudging: can nudging improve population health? *BMJ (Clinical Research Ed.)*, *342*, d228.

Martin, U., & Mercer, S. W. (2014). A comparison of general practitioners prescribing

of gluten-free foods for the treatment of coeliac disease with national prescribing guidelines. *Journal of Human Nutrition and Dietetics*, *27*(1), 96–104. https://doi.org/10.1111/jhn.12116

Mazzeo, S. E., & Bulik, C. M. (2009). Environmental and genetic risk factors for eating disorders: What the clinician needs to know. *Child and Adolescent Psychiatric Clinics of North America*, *18*(1), 67–82. https://doi.org/10.1016/j.chc.2008.07.003

McEachan, R. R. C., Conner, M., Taylor, N. J., & Lawton, R. J. (2011). Prospective prediction of health-related behaviours with the Theory of Planned Behaviour: a meta-analysis. *Health Psychology Review*, *5*(2), 97–144. https://doi.org/10.1080/17437199.2010.521684

McIntyre, R. M., & Blashfield, R. K. (1980). A Nearest-Centroid Technique For Evaluating The Minimum-Variance Clustering Procedure. *Multivariate Behavioral Research*, *15*(2), 225–238. https://doi.org/10.1207/s15327906mbr1502_7

Meadows, G., & Treasure, J. (1989). Bulimia nervosa and Crohn's disease: two case reports. *Acta Psychiatrica Scandinavica*, *79*(4), 413–414.

Meiselman, H. L., King, S. C., & Gillette, M. (2010). The demographics of neophobia in a large commercial US sample. *Food Quality and Preference*, *21*(7), 893–897. https://doi.org/10.1016/j.foodqual.2010.05.009

Meltzer, L. J., Johnson, S. B., Prine, J. M., Banks, R. A., Desrosiers, P. M., & Silverstein, J. H. (2001). Disordered Eating, Body Mass, and Glycemic Control in Adolescents With Type 1 Diabetes. *Diabetes Care*, *24*(4), 678–682. https://doi.org/10.2337/diacare.24.4.678

Merwin, R. M., Moskovich, A. A., Dmitrieva, N. O., Pieper, C. F., Honeycutt, L. K., Zucker, N. L., … Buhi, L. (2014). Disinhibited eating and weight-related insulin mismanagement among individuals with type 1 diabetes. *Appetite*, *81*, 123–130. https://doi.org/10.1016/j.appet.2014.05.028

Midhagen, G., & Hallert, C. (2003). High rate of gastrointestinal symptoms in celiac patients living on a gluten-free diet: controlled study. *The American Journal of Gastroenterology*, *98*(9), 2023–2026. https://doi.org/10.1111/j.1572-0241.2003.07632.x

Mills, J. R., & Murray, J. A. (2016). Contemporary celiac disease diagnosis: is a biopsy avoidable? *Current Opinion in Gastroenterology*, *32*(2), 80–85. https://doi.org/10.1097/MOG.0000000000000245

Miranda, J., Lasa, A., Bustamante, M. A., Churruca, I., & Simon, E. (2014). Nutritional Differences Between a Gluten-free Diet and a Diet Containing Equivalent Products with Gluten. *Plant Foods for Human Nutrition*, *69*(2), 182–187. https://doi.org/10.1007/s11130-014-0410-4

Mirzaei, M., Aspin, C., Essue, B., Jeon, Y.-H., Dugdale, P., Usherwood, T., & Leeder, S. (2013). A patient-centred approach to health service delivery: improving health outcomes for people with chronic illness. *BMC Health Services Research*, *13*, 251. https://doi.org/10.1186/1472-6963-13-251

Misra, M., Miller, K. K., Tsai, P., Gallagher, K., Lin, A., Lee, N., … Klibanski, A. (2006). Elevated peptide YY levels in adolescent girls with anorexia nervosa. *The Journal of Clinical Endocrinology and Metabolism*, *91*(3), 1027–1033. https://doi.org/10.1210/jc.2005-1878

Molodecky, N. A., Soon, I. S., Rabi, D. M., Ghali, W. A., Ferris, M., Chernoff, G., …

Kaplan, G. G. (2012). Increasing incidence and prevalence of the inflammatory bowel diseases with time, based on systematic review. *Gastroenterology*, *142*(1), 46–54.e42; quiz e30. https://doi.org/10.1053/j.gastro.2011.10.001

Mooi, E., & Sarstedt, M. (2011). *A Concise Guide to Market Research*. Berlin, Heidelberg: Springer Berlin Heidelberg. Retrieved from http://link.springer.com/10.1007/978-3-642-12541-6

Mooney, P. D., Evans, K. E., Singh, S., & Sanders, D. S. (2012). Treatment failure in coeliac disease: a practical guide to investigation and treatment of non-responsive and refractory coeliac disease. *Journal of Gastrointestinal and Liver Diseases: JGLD*, *21*(2), 197–203.

Moss-Morris, R., Weinman, J., Petrie, K., Horne, R., Cameron, L., & Buick, D. (2002). The Revised Illness Perception Questionnaire (IPQ-R). *Psychology & Health*, *17*(1), 1–16. https://doi.org/10.1080/08870440290001494

Muller, K., Prosser, R., Bampton, P., Mountifield, R., & Andrews, J. M. Female gender and surgery impair relationships, body image and sexuality in inflammatory bowel disease: patient perceptions. *Inflammatory Bowel Disease, 16* (4), 657-663.

Murray, J. A., Watson, T., Clearman, B., & Mitros, F. (2004). Effect of a gluten-free diet on gastrointestinal symptoms in celiac disease. *The American Journal of Clinical Nutrition*, *79*(4), 669–673.

Mustalahti, K., Lohiniemi, S., Collin, P., Vuolteenaho, N., Laippala, P., & Mäki, M. (2002). Gluten-free diet and quality of life in patients with screen-detected celiac disease. *Effective Clinical Practice: ECP*, *5*(3), 105–113.

Nachman, F., del Campo, M. P., González, A., Corzo, L., Vázquez, H., Sfoggia, C., … Bai, J. C. (2010). Long-term deterioration of quality of life in adult patients with celiac disease is associated with treatment noncompliance. *Digestive and Liver Disease: Official Journal of the Italian Society of Gastroenterology and the Italian Association for the Study of the Liver*, *42*(10), 685–691. https://doi.org/10.1016/j.dld.2010.03.004

Nachman, F., Mauriño, E., Vázquez, H., Sfoggia, C., Gonzalez, A., Gonzalez, V., … Bai, J. C. (2009). Quality of life in celiac disease patients: prospective analysis on the importance of clinical severity at diagnosis and the impact of treatment. *Digestive and Liver Disease: Official Journal of the Italian Society of Gastroenterology and the Italian Association for the Study of the Liver*, *41*(1), 15–25. https://doi.org/10.1016/j.dld.2008.05.011

Nadon, G., Feldman, D. E., Dunn, W., & Gisel, E. (2011). Mealtime problems in children with autism spectrum disorder and their typically developing siblings: a comparison study. *Autism: The International Journal of Research and Practice*, *15*(1), 98–113. https://doi.org/10.1177/1362361309348943

Nelson, M., Mendoza, N., & McGough, N. (2007). A survey of provision of dietetic services for coeliac disease in the UK. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *20*(5), 403–411. https://doi.org/10.1111/j.1365-277X.2007.00813.x

Neumark-Sztainer, D., Patterson, J., Mellin, A., Ackard, D. M., Utter, J., Story, M., & Sockalosky, J. (2002). Weight control practices and disordered eating behaviors among adolescent females and males with type 1 diabetes: associations with sociodemographics, weight concerns, familial factors, and

metabolic outcomes. *Diabetes Care*, *25*(8), 1289–1296.

Newnham, E. D., Shepherd, S. J., Strauss, B. J., Hosking, P., & Gibson, P. R. (2016). Adherence to the gluten-free diet can achieve the therapeutic goals in almost all patients with coeliac disease: A 5-year longitudinal study from diagnosis. *Journal of Gastroenterology and Hepatology*, *31*(2), 342–349. https://doi.org/10.1111/jgh.13060

NICE. (2015). Coeliac disease: Recognition and assessment of disease. Retrieved September 21 2016, from http://www.nice.org.uk/nicemedia/pdf/cg061niceguideline.pdf

NICE. (2016). Inflammatory bowel disease. Retrieved September 21 2016, from https://www.nice.org.uk/guidance/conditions-and-diseases/digestive-tract-conditions/inflammatory-bowel-disease

NICE. (2016). Type 1 diabetes in adults: diagnosis and management. Retrieved September 21 2016, from https://www.nice.org.uk/guidance/ng17?unlid=107165640920164921152

NICE. (2016). Type 2 diabetes in adults: management. Retrieved September 21 2016, from https://www.nice.org.uk/guidance/ng28?unlid=177237790201681645256

Nicholas, D. B., Otley, A., Smith, C., Avolio, J., Munk, M., & Griffiths, A. M. (2007). Challenges and strategies of children and adolescents with inflammatory bowel disease: a qualitative examination. *Health and Quality of Life Outcomes*, *5*, 28. https://doi.org/10.1186/1477-7525-5-28

Nicolau, J., Simó, R., Sanchís, P., Ayala, L., Fortuny, R., Zubillaga, I., & Masmiquel, L.

(2015). Eating disorders are frequent among type 2 diabetic patients and are associated with worse metabolic and psychological outcomes: results from a cross-sectional study in primary and secondary care settings. *Acta Diabetologica*, *52*(6), 1037–1044. https://doi.org/10.1007/s00592-015-0742-z

Nicolucci, A., Kovacs Burns, K., Holt, R. I. G., Comaschi, M., Hermanns, N., Ishii, H., … the DAWN2 Study Group. (2013). Diabetes Attitudes, Wishes and Needs second study (DAWN2™): Cross-national benchmarking of diabetes-related psychosocial outcomes for people with diabetes. *Diabetic Medicine*, *30*(7), 767–777. https://doi.org/10.1111/dme.12245

Nield, L. S., Gillespie, S., & Riedel, B. D. (2011). A 16-year-old boy with anemia, pica. *Pediatric Annals*, *40*(8), 391–392. https://doi.org/10.3928/00904481-20110708-03

Nielsen, S. (2002). Eating disorders in females with type 1 diabetes: an update of a meta-analysis. *European Eating Disorders Review*, *10*(4), 241–254. https://doi.org/10.1002/erv.474

Okami, Y., Kato, T., Nin, G., Harada, K., Aoi, W., Wada, S., … Fukudo, S. (2011). Lifestyle and psychological factors related to irritable bowel syndrome in nursing and medical school students. *Journal of Gastroenterology*, *46*(12), 1403–1410. https://doi.org/10.1007/s00535-011-0454-2

Olén, O., Montgomery, S. M., Marcus, C., Ekbom, A., & Ludvigsson, J. F. (2009). Coeliac disease and body mass index: a study of two Swedish general population-based registers. *Scandinavian Journal of Gastroenterology*, *44*(10), 1198–1206. https://doi.org/10.1080/00365520903132013

Olsson, C., Hörnell, A., Ivarsson, A., & Sydner, Y. M. (2008). The everyday life of adolescent coeliacs: issues of importance for compliance with the gluten-free diet. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *21*(4), 359–367.

Oso, O., & Fraser, N. C. (2006). A boy with coeliac disease and obesity. *Acta Pædiatrica*, *95*(5), 618–619. https://doi.org/10.1111/j.1651-2227.2006.tb02292.x

Paarlahti, P., Kurppa, K., Ukkola, A., Collin, P., Huhtala, H., Mäki, M., & Kaukinen, K. (2013). Predictors of persistent symptoms and reduced quality of life in treated coeliac disease patients: a large cross-sectional study. *BMC Gastroenterology*, *13*, 75. https://doi.org/10.1186/1471-230X-13-75

Palmer, P. B., & O'Connell, D. G. (2009). Regression Analysis for Prediction: Understanding the Process. *Cardiopulmonary Physical Therapy Journal*, *20*(3), 23–26.

Passananti, V., Siniscalchi, M., Zingone, F., Bucci, C., Tortora, R., Iovino, P., & Ciacci, C. (2013). Prevalence of Eating Disorders in Adults with Celiac Disease. *Gastroenterology Research and Practice*, *2013*, e491657. https://doi.org/10.1155/2013/491657

Patrick, J. H., Stahl, S. T., & Sundaram, M. (2011). Disordered eating and psychological distress among adults. *International Journal of Aging & Human Development*, *73*(3), 209–226.

Peat, C. M., Huang, L., Thornton, L. M., Von Holle, A. F., Trace, S. E., Lichtenstein, P., … Bulik, C. M. (2013). Binge Eating, Body Mass Index, and Gastrointestinal Symptoms. *Journal of Psychosomatic Research*, *75*(5).

https://doi.org/10.1016/j.jpsychores.2013.08.009

Perkins, S. J., Keville, S., Schmidt, U., & Chalder, T. (2005). Eating disorders and irritable bowel syndrome: is there a link? *Journal of Psychosomatic Research*, *59*(2), 57–64. https://doi.org/10.1016/j.jpsychores.2004.04.375

Peterson, C. M., Fischer, S., & Young-Hyman, D. (2015). Topical review: a comprehensive risk model for disordered eating in youth with type 1 diabetes. *Journal of Pediatric Psychology*, *40*(4), 385–390. https://doi.org/10.1093/jpepsy/jsu106

Peveler, R. C., Fairburn, C. G., Boller, I., & Dunger, D. (1992). Eating disorders in adolescents with IDDM. A controlled study. *Diabetes Care*, *15*(10), 1356–1360.

Pilowsky, I. (1978). A general classification of abnormal illness behaviours. *Psychology and Psychotherapy Theory, Research and Practice, 51* (2), 131-137.

Pinhas-Hamiel, O., Hamiel, U., & Levy-Shraga, Y. (2015). Eating disorders in adolescents with type 1 diabetes: Challenges in diagnosis and treatment. *World Journal of Diabetes*, *6*(3), 517–526. https://doi.org/10.4239/wjd.v6.i3.517

Pinhas-Hamiel, O., & Levy-Shraga, Y. (2012). Eating Disorders in Adolescents with Type 2 and Type 1 Diabetes. *Current Diabetes Reports*, *13*(2), 289–297. https://doi.org/10.1007/s11892-012-0355-7

Pliner, P., & Hobden, K. (1992). Development of a scale to measure the trait of food neophobia in humans. *Appetite*, *19*(2), 105–120.

Pojoga, C., & Stănculete, M. F. (2014). BIOPSYCHOSOCIAL APPROACH OF

GASTROINTESTINAL DISORDERS. *Clujul Medical*, *87*(2), 95–97. https://doi.org/10.15386/cjmed-289

Polivy, J., & Herman, C. P. (1985). Dieting and binging. A causal analysis. *The American Psychologist*, *40*(2), 193–201.

Polivy, J., & Herman, C. P. (1991). Good and bad dieters: Self-perception and reaction to a dietary challenge. *International Journal of Eating Disorders*, *10*(1), 91–99. https://doi.org/10.1002/1098-108X(199101)10:1<91::AID-EAT2260100110>3.0.CO;2-6

Polivy, J., & Herman, C. P. (2002). Causes of Eating Disorders. *Annual Review of Psychology*, *53*(1), 187–213. https://doi.org/10.1146/annurev.psych.53.100901.135103

Pollard, J., Kirk, S. F. L., & Cade, J. E. (2002). Factors affecting food choice in relation to fruit and vegetable intake: a review. *Nutrition Research Reviews, 15 (2),* 373-387. https://doi.org/10.1079/NRR200244

Polloni, L., Toniolo, A., Lazzarotto, F., Baldi, I., Foltran, F., Gregori, D., & Muraro, A. (2013). Nutritional behavior and attitudes in food allergic children and their mothers. *Clinical and Translational Allergy*, *3*(1), 41. https://doi.org/10.1186/2045-7022-3-41

Polonsky, W. H., Anderson, B. J., Lohrer, P. A., Aponte, J. E., Jacobson, A. M., & Cole, C. F. (1994). Insulin omission in women with IDDM. *Diabetes Care*, *17*(10), 1178–1185.

Quick, V., & Byrd-Bredbenner, C. (2014). Disordered eating behaviors and attitudes of females with cystic fibrosis (628.7). *The FASEB Journal*, *28*(1 Supplement), 628.7.

Quick, V. M., Byrd-Bredbenner, C., & Neumark-Sztainer, D. (2013). Chronic Illness and Disordered Eating: A Discussion of the Literature. *Advances in Nutrition: An International Review Journal*, *4*(3), 277–286. https://doi.org/10.3945/an.112.003608

Rajpoot, P., Sharma, A., Harikrishnan, S., Baruah, B. J., Ahuja, V., & Makharia, G. K. (2015). Adherence to gluten-free diet and barriers to adherence in patients with celiac disease. *Indian Journal of Gastroenterology*, *34*(5), 380–386. https://doi.org/10.1007/s12664-015-0607-y

Rashtak, S., & Murray, J. A. (2009). Celiac Disease in the Elderly. *Gastroenterology Clinics of North America*, *38*(3), 433–446. https://doi.org/10.1016/j.gtc.2009.06.005

Raymond, N. C., Chang, P.-N., Crow, S. J., Mitchell, J. E., Dieperink, B. S., Beck, M. M., … Warwick, W. J. (2000). Eating disorders in patients with cystic fibrosis. *Journal of Adolescence*, *23*(3), 359–363. https://doi.org/10.1006/jado.2000.0321

Remillard, M. L., Mazor, K. M., Cutrona, S. L., Gurwitz, J. H., & Tjia, J. (2014). Systematic Review of the Use of Online Questionnaires of Older Adults. *Journal of the American Geriatrics Society*, *62*(4), 696–705. https://doi.org/10.1111/jgs.12747

Ricca, V., Mannucci, E., Calabrò, A., Bernardo, M. D., Cabras, P. L., & Rotella, C. M. (2000). Anorexia nervosa and celiac disease: two case reports. *The International Journal of Eating Disorders*, *27*(1), 119–122.

Robinson, E., Oldham, M., Cuckson, I., Brunstrom, J. M., Rogers, P. J., & Hardman, C. A. (2016). Visual exposure to large and small portion sizes and perceptions of

portion size normality: Three experimental studies. *Appetite*, *98*, 28–34. https://doi.org/10.1016/j.appet.2015.12.010

Rocha, S., Gandolfi, L., Santos, J. E. dos, Rocha, S., Gandolfi, L., & Santos, J. E. dos. (2016). The psychosocial impacts caused by diagnosis and treatment of Coeliac Disease. *Revista Da Escola de Enfermagem Da USP*, *50*(1), 65–70. https://doi.org/10.1590/S0080-623420160000100009

Rodin, G., Olmsted, M. P., Rydall, A. C., Maharaj, S. I., Colton, P. A., Jones, J. M., … Daneman, D. (2002). Eating disorders in young women with type 1 diabetes mellitus. *Journal of Psychosomatic Research*, *53*(4), 943–949. https://doi.org/10.1016/S0022-3999(02)00305-7

Roma, E., Roubani, A., Kolia, E., Panayiotou, J., Zellos, A., & Syriopoulou, V. P. (2010). Dietary compliance and life style of children with coeliac disease. *Journal of Human Nutrition and Dietetics*, *23*(2), 176–182. https://doi.org/10.1111/j.1365-277X.2009.01036.x

Roos, S., Hellström, I., Hallert, C., & Wilhelmsson, S. (2013). Everyday Life for Women With Celiac Disease: *Gastroenterology Nursing*, *36*(4), 266–273. https://doi.org/10.1097/SGA.0b013e31829ed98d

Rose, C., & Howard, R. (2014). Living with coeliac disease: a grounded theory study. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *27*(1), 30–40. https://doi.org/10.1111/jhn.12062

Rosen, D. S. (2003). Eating disorders in children and young adolescents: Etiology, classification, clinical features, and treatment. *Adolescent Medicine*, *14*(1), 49.

Rosmark, B., Berne, C., Holmgren, S., Lago, C., Renholm, G., & Sohlberg, S. (1986).

Eating disorders in patients with insulin-dependent diabetes mellitus. *Journal of Clinical Psychiatry*, *47*(11), 547–550.

Roth, B., & Ohlsson, B. (2013). Gastrointestinal symptoms and psychological well-being in patients with microscopic colitis. *Scandinavian Journal of Gastroenterology*, *48*(1), 27–34. https://doi.org/10.3109/00365521.2012.741614

Rubio-Tapia, A., Kyle, R. A., Kaplan, E. L., Johnson, D. R., Page, W., Erdtmann, F., … Murray, J. A. (2009). Increased prevalence and mortality in undiagnosed celiac disease. *Gastroenterology*, *137*(1), 88–93. https://doi.org/10.1053/j.gastro.2009.03.059

Rubio-Tapia, A., & Murray, J. A. (2010). Classification and management of refractory coeliac disease. *Gut*, *59*(4), 547–557. https://doi.org/10.1136/gut.2009.195131

Russell-Jones, D., & Khan, R. (2007). Insulin-associated weight gain in diabetes--causes, effects and coping strategies. *Diabetes, Obesity & Metabolism*, *9*(6), 799–812. https://doi.org/10.1111/j.1463-1326.2006.00686.x

Sabourin, B. C., & Pursley, S. (2013). Psychosocial Issues in Diabetes Self-Management: Strategies for Healthcare Providers. *Canadian Journal of Diabetes*, *37*(1), 36–40. https://doi.org/10.1016/j.jcjd.2013.01.002

Sainsbury, K., & Mullan, B. (2011). Measuring beliefs about gluten free diet adherence in adult coeliac disease using the theory of planned behaviour. *Appetite*, *56*(2), 476–483. https://doi.org/10.1016/j.appet.2011.01.026

Sainsbury, K., Mullan, B., & Sharpe, L. (2013a). A randomized controlled trial of an online intervention to improve gluten-free diet adherence in celiac disease.

The American Journal of Gastroenterology, *108*(5), 811–817. https://doi.org/10.1038/ajg.2013.47

Sainsbury, K., Mullan, B., & Sharpe, L. (2013b). Gluten free diet adherence in coeliac disease. The role of psychological symptoms in bridging the intention–behaviour gap. *Appetite*, *61*, 52–58. https://doi.org/10.1016/j.appet.2012.11.001

Sainsbury, K., Mullan, B., & Sharpe, L. (2013c). Reduced quality of life in coeliac disease is more strongly associated with depression than gastrointestinal symptoms. *Journal of Psychosomatic Research*, *75*(2), 135–141. https://doi.org/10.1016/j.jpsychores.2013.05.011

Sainsbury, K., Mullan, B., & Sharpe, L. (2014). Depression in Coeliac Disease and the Impact on ADHERENCE: a Systematic Review of the Literature. *European Health Psychologist*, *16*(S), 910.

Santos, M., Richards, C. S., & Bleckley, M. K. (2007). Comorbidity between depression and disordered eating in adolescents. *Eating Behaviors*, *8*(4), 440–449. https://doi.org/10.1016/j.eatbeh.2007.03.005

Schlundt, D. G., Rowe, S., Pichert, J. W., & Plant, D. D. (1999). What are the eating cognitions of children whose chronic diseases do and do not require attention to diet? *Patient Education and Counseling*, *36*(3), 279–286.

Schneider, M. A., & Fletcher, P. C. (2008). “I feel as if my IBS is keeping me hostage!” Exploring the negative impact of irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD) upon university-aged women. *International Journal of Nursing Practice*, *14*(2), 135–148. https://doi.org/10.1111/j.1440-172X.2008.00677.x

Schuppan, D., Dennis, M. D., & Kelly, C. P. (2005). Celiac disease: epidemiology, pathogenesis, diagnosis, and nutritional management. *Nutrition in Clinical Care: An Official Publication of Tufts University*, *8*(2), 54–69.

See, J. A., Kaukinen, K., Makharia, G. K., Gibson, P. R., & Murray, J. A. (2015). Practical insights into gluten-free diets. *Nature Reviews Gastroenterology & Hepatology*, *12*(10), 580–591. https://doi.org/10.1038/nrgastro.2015.156

Seiffge-Krenke, I., & Skaletz, C. (2006). [Chronically ill and psychologically impaired? Results of a new literature analysis]. *Praxis Der Kinderpsychologie Und Kinderpsychiatrie*, *55*(1), 73–90.

Serra, J., Azpiroz, F., & Malagelada, J. R. (1998). Intestinal gas dynamics and tolerance in humans. *Gastroenterology*, *115*(3), 542–550.

Sharma, T. R., Kline, D. B., Shreeve, D. F., & Hartman, D. W. (2011). Psychiatric comorbidities in patients with celiac disease: Is there any concrete biological association? *Asian Journal of Psychiatry*, *4*(2), 150–151. https://doi.org/10.1016/j.ajp.2011.03.001

Shaw, A., & Favazza, A. (2010). Deliberate Insulin Underdosing and Omission Should Be Included in DSM-V Criteria for Bulimia Nervosa. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *22*(3), 352.e13-352.e13. https://doi.org/10.1176/jnp.2010.22.3.352.e13

Shearer, J. E., & Bryon, M. (2004). The nature and prevalence of eating disorders and eating disturbance in adolescents with cystic fibrosis. *Journal of the Royal Society of Medicine*, *97*(Suppl 44), 36–42.

Shim, J. S., Oh, K., & Kim, H. C. (2014). Dietary assessment methods in epidemiologic studies. *Epidemiology and Health*, *36*.

https://doi.org/10.4178/epih/e2014009

Silvester, J. A., Weiten, D., Graff, L. A., Walker, J. R., & Duerksen, D. R. (2016). Is it gluten-free? Relationship between self-reported gluten-free diet adherence and knowledge of gluten content of foods. *Nutrition*, *32*(7–8), 777–783. https://doi.org/10.1016/j.nut.2016.01.021

Simione, L., Calabrese, L., Marucci, F. S., Belardinelli, M. O., Raffone, A., & Maratos, F. A. (2014). Emotion Based Attentional Priority for Storage in Visual Short-Term Memory. *PLoS ONE*, *9*(5). https://doi.org/10.1371/journal.pone.0095261

Simon, S. L., Duncan, C. L., Horky, S. C., Nick, T. G., Castro, M. M., & Riekert, K. A. (2011). Body satisfaction, nutritional adherence, and quality of life in youth with cystic fibrosis. *Pediatric Pulmonology*, *46*(11), 1085–1092. https://doi.org/10.1002/ppul.21477

Simpson, S., Lebwohl, B., DiGiacomo, D., Minaya, M. T., Sanders, D. S., & Green, P. H. (2011). Awareness of Celiac Disease and Gluten Sensitivity in the United States. *Gastroenterology*, *140*(5), S-443. https://doi.org/10.1016/S0016-5085(11)61816-7

Singh, J., & Whelan, K. (2011). Limited availability and higher cost of gluten-free foods. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *24*(5), 479–486. https://doi.org/10.1111/j.1365-277X.2011.01160.x

Skjerning, H., Mahony, R. O., Husby, S., & DunnGalvin, A. (2014). Health-related quality of life in children and adolescents with celiac disease: patient-driven data from focus group interviews. *Quality of Life Research*, *23*(6), 1883–1894.

https://doi.org/10.1007/s11136-014-0623-x

Smith, D. F., & Gerdes, L. U. (2012). Meta-analysis on anxiety and depression in adult celiac disease. *Acta Psychiatrica Scandinavica*, *125*(3), 189–193. https://doi.org/10.1111/j.1600-0447.2011.01795.x

Snyder, M. R., & Murray, J. A. (2016). Celiac disease: advances in diagnosis. *Expert Review of Clinical Immunology*, *12*(4), 449–463. https://doi.org/10.1586/1744666X.2016.1130625

Sollid, L. M. (2002). Coeliac disease: dissecting a complex inflammatory disorder. *Nature Reviews Immunology*, *2*(9), 647–655. https://doi.org/10.1038/nri885

Solmi, F., Hatch, S. L., Hotopf, M., Treasure, J., & Micali, N. (2014). Prevalence and correlates of disordered eating in a general population sample: the South East London Community Health (SELCoH) study. *Social Psychiatry and Psychiatric Epidemiology*, *49*(8), 1335–1346. https://doi.org/10.1007/s00127-014-0822-3

Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychological Bulletin*, *128*(5), 825–848.

Stice, E., Nathan, C., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology*, *122*(2), 445–457. https://doi.org/10.1037/a0030679

Sullivan, G., Blewett, A. E., Jenkins, P. L., & Allison, M. C. (1997). Eating attitudes and the irritable bowel syndrome. *General Hospital Psychiatry*, *19*(1), 62–64.

Suris, J. C., Michaud, P. A., & Viner, R. (2004). The adolescent with a chronic condition. Part I: developmental issues. *Archives of Disease in Childhood*,

89(10), 938–942. https://doi.org/10.1136/adc.2003.045369

Sverker, A., Hensing, G., & Hallert, C. (2005). “Controlled by food”- lived experiences of coeliac disease. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *18*(3), 171–180. https://doi.org/10.1111/j.1365-277X.2005.00591.x

Swanson S. A., Crow S. J., Le Grange D., Swendsen J., & Merikangas K. R. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry*, *68*(7), 714–723. https://doi.org/10.1001/archgenpsychiatry.2011.22

Tang, T. N., Toner, B. B., Stuckless, N., Dion, K. L., Kaplan, A. S., & Ali, A. (1998). Features of eating disorders in patients with irritable bowel syndrome. *Journal of Psychosomatic Research*, *45*(2), 171–178.

Terwee, C. B., Bot, S. D. M., Boer, M. R. de, Windt, D. A. W. M. van der, Knol, D. L., Dekker, J., … Vet, H. C. W. de. (2007). Quality criteria were proposed for measurement properties of health status questionnaires. *Journal of Clinical Epidemiology*, *60*(1), 34–42. https://doi.org/10.1016/j.jclinepi.2006.03.012

Thomas, J. M., Dourish, C. T., Tomlinson, J. W., Hassan-Smith, Z., & Higgs, S. (2014). Effects of the 5-HT2C receptor agonist meta-chlorophenylpiperazine on appetite, food intake and emotional processing in healthy volunteers. *Psychopharmacology*, *231*(12), 2449–2459. https://doi.org/10.1007/s00213-013-3409-x

Timmerman, G. M. (1999). Binge Eating Scale: Further Assessment of Validity and Reliability1. *Journal of Applied Biobehavioral Research*, *4*(1), 1–12.

https://doi.org/10.1111/j.1751-9861.1999.tb00051.x

Tio, M., Cox, M. R., & Eslick, G. D. (2012). Meta-analysis: coeliac disease and the risk of all-cause mortality, any malignancy and lymphoid malignancy. *Alimentary Pharmacology & Therapeutics*, *35*(5), 540–551. https://doi.org/10.1111/j.1365-2036.2011.04972.x

Turkel, S., & Pao, M. (2007). Late consequences of chronic pediatric illness. *The Psychiatric Clinics of North America*, *30*(4), 819–835. https://doi.org/10.1016/j.psc.2007.07.009

Turner, J., & Kelly, B. (2000). Emotional dimensions of chronic disease. *Western Journal of Medicine*, *172*(2), 124–128.

Ukkola, A., Mäki, M., Kurppa, K., Collin, P., Huhtala, H., Kekkonen, L., & Kaukinen, K. (2011). Diet Improves Perception of Health and Well-being in Symptomatic, but Not Asymptomatic, Patients With Celiac Disease. *Clinical Gastroenterology and Hepatology*, *9*(2), 118–123.e1. https://doi.org/10.1016/j.cgh.2010.10.011

Ukkola, A., Mäki, M., Kurppa, K., Collin, P., Huhtala, H., Kekkonen, L., & Kaukinen, K. (2012). Patients' experiences and perceptions of living with coeliac disease - implications for optimizing care. *Journal of Gastrointestinal and Liver Diseases: JGLD*, *21*(1), 17–22.

Unikel, C., Aguilar, J., & Gómez-Peresmitré, G. (2005). Predictors of eating behaviors in a sample of Mexican women. *Eating and Weight Disorders: EWD*, *10*(1), 33–39.

Urvelyte, E., & Perminas, A. (2015). The Dual Pathway Model of Bulimia: Replication and Extension with Anorexia. *Procedia - Social and Behavioral Sciences*, *205*,

178–183. https://doi.org/10.1016/j.sbspro.2015.09.054

van Doorn, R. K., Winkler, L. M., Zwinderman, K. H., Mearin, M. L., & Koopman, H. M. (2008). CDDUX: A Disease-specific Health-related Quality-of-life Questionnaire for Children With Celiac Disease: *Journal of Pediatric Gastroenterology and Nutrition*, *47*(2), 147–152. https://doi.org/10.1097/MPG.0b013e31815ef87d

Vartanian, L. R., Sokol, N., Herman, C. P., & Polivy, J. (2013). Social Models Provide a Norm of Appropriate Food Intake for Young Women. *PLoS ONE*, *8*(11). https://doi.org/10.1371/journal.pone.0079268

Villafuerte-Galvez, J., Vanga, R. R., Dennis, M., Hansen, J., Leffler, D. A., Kelly, C. P., & Mukherjee, R. (2015). Factors governing long-term adherence to a gluten-free diet in adult patients with coeliac disease. *Alimentary Pharmacology & Therapeutics*, *42*(6), 753–760. https://doi.org/10.1111/apt.13319

Wada, Y., Hisamatsu, T., Naganuma, M., Matsuoka, K., Okamoto, S., Inoue, N., … Kanai, T. (2015). Risk factors for decreased bone mineral density in inflammatory bowel disease: A cross-sectional study. *Clinical Nutrition (Edinburgh, Scotland)*, *34*(6), 1202–1209. https://doi.org/10.1016/j.clnu.2015.01.003

Wagner, G., Berger, G., Sinnreich, U., Grylli, V., Schober, E., Huber, W.-D., & Karwautz, A. (2008). Quality of life in adolescents with treated coeliac disease: influence of compliance and age at diagnosis. *Journal of Pediatric Gastroenterology and Nutrition*, *47*(5), 555–561. https://doi.org/10.1097/MPG.0b013e31817fcb56

Wagner, G., Zeiler, M., Berger, G., Huber, W.-D., Favaro, A., Santonastaso, P., &

Karwautz, A. (2015). Eating Disorders in Adolescents with Celiac Disease: Influence of Personality Characteristics and Coping. *European Eating Disorders Review: The Journal of the Eating Disorders Association*, *23*(5), 361–370. https://doi.org/10.1002/erv.2376

Wahab, P. J., Meijer, J. W. R., & Mulder, C. J. J. (2002). Histologic follow-up of people with celiac disease on a gluten-free diet: slow and incomplete recovery. *American Journal of Clinical Pathology*, *118*(3), 459–463. https://doi.org/10.1309/EVXT-851X-WHLC-RLX9

Walters, S. (2001). Sex differences in weight perception and nutritional behaviour in adults with cystic fibrosis. *Journal of Human Nutrition and Dietetics*, *14*(2), 83–91. https://doi.org/10.1046/j.1365-277X.2001.00274.x

West, J., Fleming, K. M., Tata, L. J., Card, T. R., & Crooks, C. J. (2014). Incidence and prevalence of celiac disease and dermatitis herpetiformis in the UK over two decades: population-based study. *The American Journal of Gastroenterology*, *109*(5), 757–768. https://doi.org/10.1038/ajg.2014.55

Whitaker, J. K. H., West, J., Holmes, G. K. T., & Logan, R. F. A. (2009). Patient perceptions of the burden of coeliac disease and its treatment in the UK. *Alimentary Pharmacology & Therapeutics*, *29*(10), 1131–1136. https://doi.org/10.1111/j.1365-2036.2009.03983.x

White, L. E., Bannerman, E., & Gillett, P. M. (2016). Coeliac disease and the gluten-free diet: a review of the burdens; factors associated with adherence and impact on health-related quality of life, with specific focus on adolescence. *Journal of Human Nutrition and Dietetics*, *29*(5), 593–606. https://doi.org/10.1111/jhn.12375

Wisting, L., Frøisland, D. H., Skrivarhaug, T., Dahl-Jørgensen, K., & Rø, Ø. (2013). Disturbed Eating Behavior and Omission of Insulin in Adolescents Receiving Intensified Insulin Treatment. *Diabetes Care*, *36*(11), 3382–3387. https://doi.org/10.2337/dc13-0431

Wolfgang, S. (2011). *Social Psychology And Health*. McGraw-Hill Education (UK).

Yamamoto, T., Nakahigashi, M., & Saniabadi, A. R. (2009). Review article: diet and inflammatory bowel disease--epidemiology and treatment. *Alimentary Pharmacology & Therapeutics*, *30*(2), 99–112. https://doi.org/10.1111/j.1365-2036.2009.04035.x

Young, V., Eiser, C., Johnson, B., Brierley, S., Epton, T., Elliott, J., & Heller, S. (2013). Eating problems in adolescents with Type 1 diabetes: a systematic review with meta-analysis. *Diabetic Medicine*, *30*(2), 189–198. https://doi.org/10.1111/j.1464-5491.2012.03771.x

Young-Hyman, D. L., & Davis, C. L. (2010). Disordered eating behavior in individuals with diabetes: importance of context, evaluation, and classification. *Diabetes Care*, *33*(3), 683–689. https://doi.org/10.2337/dc08-1077

Yucel, B., Ozbey, N., Demir, K., Polat, A., & Yager, J. (2006). Eating disorders and celiac disease: A case report. *International Journal of Eating Disorders*, *39*(6), 530–532. https://doi.org/10.1002/eat.20294

Zarkadas, M., Cranney, A., Case, S., Molloy, M., Switzer, C., Graham, I. D., … Burrows, V. (2006). The impact of a gluten-free diet on adults with coeliac disease: results of a national survey. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *19*(1), 41–49. https://doi.org/10.1111/j.1365-277X.2006.00659.x

Zarkadas, M., Dubois, S., MacIsaac, K., Cantin, I., Rashid, M., Roberts, K. C., … Pulido, O. M. (2013). Living with coeliac disease and a gluten-free diet: a Canadian perspective. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association*, *26*(1), 10–23. https://doi.org/10.1111/j.1365-277X.2012.01288.x

Zimmerman, R. S., & Vernberg, D. (1994). Models of preventative health behavior: Comparison, critique, and meta-analysis. *Advances in Medical Sociology, 4*, 45-67.

Zingone, F., Swift, G. L., Card, T. R., Sanders, D. S., Ludvigsson, J. F., & Bai, J. C. (2014). Psychological morbidity of celiac disease: A review of the literature. *United European Gastroenterology Journal*, 2050640614560786. https://doi.org/10.1177/2050640614560786

Appendix A: Chapter Two Search Criteria

Search Term One: CD; C*oeliac disease; Gluten intolerance; IBS; Irritable bowel syndrome; IBD; Inflammatory bowel disease; Crohn's disease; Ulcerative colitis

Search Term Two: Eating disorder; Anorexi*; Bulimi*; Binge; EDNOS; Obes*; Eating distress; Dysfunctional eating; Disturbed eating; Eating habits; Nocturnal eating; Night eating; Pica; Eating attitudes

Appendix B: Chapter Three Supplementary Tables

Supplementary Table One

Disease specific and Non-Specific Factors in Predicting EAT-26 Scores in Inflammatory Bowel Disease

Predictors	B	*B*	R^2	F	R^2 Change
Model 1) Non-specific Factors					
Age	*.01*	*.01*			
Body Mass Index	*-.09*	*-.05*			
Years with Condition	*-.09*	*-.07*			
DASS-21	*.16*	*.50*	*.25*	*6.48**	*.25**
Model 2) Disease Specific Factors					
Age	*-.03*	*-.04*			
Body Mass Index	*.02*	*.01*			
Years with Condition	*-.08*	*-.07*			
DASS-21	*.12*	*.39**			
Gastrointestinal Symptoms	*.51*	*.34**			
Dietary-management	*.61*	*.05*	*.59*	*6.57**	*.09*

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

Supplementary Table Two

Disease specific and Non-Specific Factors in Predicting EAT-26 Scores Type Two Diabetes

Predictors	**B**	***B***	***R^2***	**F**	**R^2 Change**
Model 1) Non-specific Factors					
Age	*.25*	*.50*			
Body Mass Index	*.40*	*.23*			
Years with Condition	*.12*	*.15*			
DASS-21	*.11*	*.53*	*.21*	*3.76*	*.22*
Model 2) Disease Specific Factors					
Age	*.16*	*.32*			
Body Mass Index	*.54*	*.31*			
Years with Condition	*.14*	*.19*			
DASS-21	*.11*	*.53*			
Gastrointestinal Symptoms	*-.46*	*-.08*			
Dietary-management	*-1.79*	*-.21*	*.24*	*2.84*	*.03*

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

Supplementary Table Three

Disease specific and Non-Specific Factors in Predicting BES Scores Type Two Diabetes

Predictors	**B**	***B***	***R^2***	**F**	**R^2 Change**
Model 1) Non-specific Factors					
Age	*-.37*	*-.58**			
Body Mass Index	*.64*	*.29**			
Years with Condition	*-.12*	*-.13*			
DASS-21	*.09*	*.35**	*.74*	*38.15**	*.74**
Model 2) Disease Specific Factors					
Age	*-.34*	*-.53**			
Body Mass Index	*.61*	*.28**			
Years with Condition	*-.12*	*-.13*			
DASS-21	*.09*	*.33*			
Gastrointestinal Symptoms	*1.01*	*.14*			
Dietary-management	*.39*	*.04*	*.75*	*6.75**	*.02*

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

Supplementary Table Four

Disease specific and Non-Specific Factors in Predicting BES Scores in Inflammatory Bowel Disease

Predictors	**B**	***B***	R^2	**F**	R^2 **Change**
Model 1) Non-specific Factors					
Age	*-.06*	*-.09*			
Body Mass Index	*.10*	*.07*			
Years with Condition	*.01*	*.01*			
DASS-21	*.10*	*.38**	*.40*	*3.74*	*.156*
Model 2) Disease Specific Factors					
Age	*-.05*	*-.08*			
Body Mass Index	*.07*	*.05*			
Years with Condition	*.02*	*.01*			
DASS-21	*.10*	*.40**			
Gastrointestinal Symptoms	*-.08*	*-.06*			
Dietary-management	*.63*	*.06*	*.41*	*2.58**	*.01*

* = significance at p<.008. The significance of the F value refers to the F associated with each step.

Appendix C - The CD-FAB

The Coeliac Disease Food Attitudes and Beliefs Scale (CD-FAB)

Instructions: This is a questionnaire is designed to explore food attitudes and beliefs in coeliac disease. Some questions may not apply to you; this is because we are trying to assess a range of beliefs about coeliac disease and managing the gluten-free diet. Please fill out the form below as accurately, honestly and completely as possible. There are no right or wrong answers. All of your responses are confidential.

Please tick the box that best describes your response to the question.

	Strongly Agree (7)	**Agree (6)**	**Somewhat Agree (5)**	**Neither Agree nor Disagree (4)**	**Somewhat Disagree (3)**	**Disagree (2)**	**Strongly Disagree (1)**
Because of My Coeliac Disease…							
I get concerned being near others when they are eating gluten							
I am afraid to eat outside my home							
I am afraid to touch gluten-containing foods							
I get worried when eating with strangers							
I find it hard to eat gluten-free foods that look like the gluten-containing-foods that have made me ill in the past							
I will only eat food that I have prepared myself							
My concerns about cross-contamination prevent me from going to socal events involving food							

Despite having Coeliac Disease…							
I enjoy going out for meals as much as I did before my diagnosis *							
I am comfortable eating gluten-free food from other people's kitchens *							
Being contaminated by gluten in the past, hasn't stopped me from enjoying restaurants *							
If I ask questions, I can normally find gluten-free food to eat *							

Reverse 8, 9, 10, 11 and add to make total score.

Appendix D – Questionnaires

DASS- 21

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.

Over the past week…	Did not apply to me at all	Applied to me to some degree, or some of the time	Applied to me to a considerable degree, or a good part of the time	Applied to me very much, or most of the time
I found it hard to wind down	0	1	2	3
I was aware of dryness in my mouth	0	1	2	3
I couldn't seem to experience any positive feeling at all	0	1	2	3
I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
I found it difficult to work up the initiative to do things	0	1	2	3
I tended to over-react to situations	0	1	2	3
I experienced trembling (e.g., in the hands)	0	1	2	3
I felt that I was using a lot of nervous energy	0	1	2	3
I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
I felt that I had nothing to look forward to	0	1	2	3

I found myself getting agitated	0	1	2	3
I found it difficult to relax	0	1	2	3
I felt down-hearted and blue	0	1	2	3
I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
I felt I was close to panic	0	1	2	3
I was unable to become enthusiastic about anything	0	1	2	3
I felt I wasn't worth much as a person	0	1	2	3
I felt that I was rather touchy	0	1	2	3
I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)	0	1	2	3
I felt scared without any good reason	0	1	2	3
I felt that life was meaningless	0	1	2	3

Eating Attitudes Test-26

Instructions: This tool looks at your patterns of eating. Please fill out the form below as accurately, honestly and completely as possible. There are no right or wrong answers.

Please tick the box that applies most to you

	Always	Usually	Often	Sometimes	Rarely	Never
I am terrified about being overweight						
I avoid eating when I am hungry						
I find myself preoccupied with food						
I have gone on eating binges where I feel that I may not be able to stop						
I cut my food into small pieces						
I am aware of the calorie content of the foods that I eat						
I particularly avoid foods with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)						
I feel that others would prefer it if I ate more						
I vomit after I have eaten						
I feel extremely guilty after eating						
	Always	Usually	Often	Sometimes	Rarely	Never
I am occupied with a desire to be thinner						
I think about burning up calories when I exercise						
Other people think I am too thin						

I am preoccupied with the thought of having fat on my body						
I take longer than others to eat my meals						
I avoid foods with sugar in them						
I eat diet foods						
I feel that food controls my life						
I display self-control around food						
I feel that others pressure me to eat						
I give too much time and thought to food						
I feel uncomfortable after eating sweets						
I engage in dieting behaviour						
I like my stomach to be empty						
I have the impulse to vomit after meals						
I enjoy trying new rich foods						

Binge Eating Scale

Below are groups of numbered statements. Read all of the statements in each group and mark on this sheet the one that best describes the way you feel about the problems you have controlling your eating behaviour.

Question Number	Statement	Please tick the statement that best describes the way you feel
1	I don't feel self-conscious about my weight or body size when I'm with others.	
	I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.	
	I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.	
	I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.	
2	I don't have any difficulty eating slowly in the proper manner.	
	Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.	
	At times, I tend to eat quickly and then, I feel uncomfortably full afterwards.	
	I have the habit of bolting down my food, without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.	
3	I feel capable to control my eating urges when I want to.	
	I feel like I have failed to control my eating more than the average person.	
	I feel utterly helpless when it comes to feeling in control of my eating urges.	
	Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.	
4	I don't have the habit of eating when I'm bored.	
	I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.	
	I have a regular habit of eating when I'm bored, but occasionally, I can use some other activity to get my mind off eating.	
	I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.	

5	I'm usually physically hungry when I eat something.	
	Occasionally, I eat something on impulse even though I really am not hungry.	
	I have the regular habit of eating foods that I might not really enjoy, to satisfy a hungry feeling even though physically, I don't need the food.	
	Even though I'm not physically hungry, 1 get a hungry feeling in my mouth that only seems to be satisfied when I eat a food, like a sandwich, that fills my mouth. Sometimes, when I eat the food to satisfy my mouth hunger, I then spit the food out so I won't gain weight.	
6	I don't feel any guilt or self-hate after I overeat.	
	After I overeat, occasionally I feel guilt or self-hate.	
	Almost all the time I experience strong guilt or self-hate after I overeat.	
7	I don't lose total control of my eating when dieting even after periods when I overeat.	
	Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.	
	Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I overeat on a diet. When that happens I eat even more.	
	I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either a "feast" or "famine."	
8	I rarely eat so much food that I feel uncomfortably stuffed afterwards.	
	Usually about once a month, I eat such a quantity of food, I end up feeling very stuffed.	
	I have regular periods during the month when I eat large amounts of food, either at mealtime or at snacks.	
	I eat so much food that I regularly feel quite uncomfortable after eating and sometimes a bit nauseous.	
9	My level of calorie intake does not go up very high or go down very low on a regular basis	
	Sometimes after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten	
	I have a regular habit of overeating during the night. It seems that my routine is not to be hungry in the morning but I overeat in the evening	
	In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I overeat. It seems I live a life of either "feast or famine"	

10	I usually am able to stop eating when I want to. I know when "enough is enough."	
	Every so often, I experience a compulsion to eat which I can't seem to control.	
	Frequently, I experience strong urges to eat which I seem unable to control, but at other times I can control my eating urges.	
	I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.	
11	I don't have any problem stopping eating when I feel full.	
	I usually can stop eating when I feel full but occasionally overeat leaving me feeling uncomfortably stuffed.	
	I have a problem stopping eating once I start and usually I feel uncomfortable stuffed after I eat a meal.	
	Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling.	
12	I seem to eat just as much when I'm with others (family, social gatherings) as when I'm by myself.	
	Sometimes, when I'm with other persons, I don't eat as much as I want to eat because I'm self-conscious about my eating.	
	Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.	
	I feel so ashamed about overeating that I pick times to overeat when I know no one will see me. I feel like a "closet eater."	
13	I eat three meals a day with only an occasional between meal snacks.	
	I eat 3 meals a day, but I also normally snack between meals.	
	When I am snacking heavily, I get in the habit of skipping regular meals.	
	There are regular periods when I seem to be continually eating, with no planned meals.	
14	I don't think much about trying to control unwanted eating urges.	
	At least some of the time, I feel my thoughts are pre-occupied with trying to control my eating urges.	
	I feel that frequently I spend much time thinking about how much I ate or about trying not to eat anymore.	
	It seems to me that most of my waking hours are pre-occupied by thoughts about eating or not eating. I feel like I'm constantly struggling not to eat.	

15	I don’t think about food a great deal.	
	I have strong cravings for food but they last only for brief periods of time.	
	I have days when I can’t seem to think about anything else but food.	
	Most of my days seem to be pre-occupied with thoughts about food. I feel like I live to eat.	
16	I usually know whether or not I’m physically hungry. I take the right portion of food to satisfy me.	
	Occasionally, I feel uncertain about knowing whether or not I’m physically hungry. At these times it’s hard to know how much food I should take to satisfy me.	
	Even though I might know how many calories I should eat, I don’t have any idea what is a “normal” amount of food for me.	

Coeliac Disease Quality of Life

Please think about your life over the past month (30 days), and look at the statements below. Each statement has five possible responses. For each statement, please tick one box that best describes your feelings

	Not at all	Slightly	Moderately	Quite a bit	A great deal
I feel limited by this disease					
I feel worried that I will suffer from this disease					
I feel concerned that this disease will cause other health problems					
I feel worried about my increased risk of cancer from this disease					
I feel socially stigmatised for having this disease					
I feel like I'm limited in eating meals with co-workers					
I feel like I am not able to have special foods like birthday cake and pizza					
I feel that the diet is sufficient treatment for my disease					
I feel that there are not enough choices for treatment					
I feel depressed because of my disease					
I feel frightened by having this disease					
I feel like I don't know enough about the disease					
I feel overwhelmed about having this disease					
I have trouble socialising because of my disease					
I find it difficult to take long trips because of my disease					
I feel like I cannot live a normal life because of my disease					
I feel afraid to eat out because my food may be contaminated					
I feel worried about the increased risk of one of my family members having coeliac disease					
I feel like I think about food all the time					
I feel concerned that my long-term health will be affected					

Gluten-Free Diet Adherence Questionnaire

Please circle the response that most adequately reflects how you feel

Have you been bothered by low energy level during the past 4 weeks?	None of the time	A little of the time	Some of the time	Most of the time	All of the time
Have you been bothered by headaches during the past 4 weeks?	None of the time	A little of the time	Some of the time	Most of the time	All of the time
I am able to follow a GFD when dining outside my home	Strongly agree	Somewhat agree	Neither agree nor disagree	Somewhat disagree	Strongly disagree
Before I do something I carefully consider the consequences	Strongly agree	Somewhat agree	Neither agree nor disagree	Somewhat disagree	Strongly disagree
I do not consider myself a failure	Strongly agree	Somewhat agree	Neither agree nor disagree	Somewhat disagree	Strongly disagree
How important to your health are accidental gluten exposures?	Very important	Somewhat important	Neutral/unsure	A little important	Not at all important
Over the past 4 weeks, how many times have you eaten foods containing gluten on purpose?	0 (never)	1–2	3–5	6–10	>10

Food Neophobia Scale

Please select the statement which most applies to you:

	Disagree Strongly	Disagree Moderately	Disagree Slightly	Neither Disagree nor Agree	Agree Slightly	Agree Moderately	Agree Strongly
I am very particular about the foods I will eat							
I don't trust new foods							
If I don't know what is in a food, I won't try it							
I will eat almost anything							
I am afraid to eat things I have never had before							
I am constantly sampling new and different foods							
I like to try new ethic restaurants							
At dinner parties, I would try a new food							
I like food from different countries							
Ethnic foods look weird to eat							

Illness Perception Questionnaire Revised – Symptom Scale

Listed below are a number of symptoms that you may or may not have experienced this symptom over the last four weeks. Please indicate by circling Yes or No, whether you have experienced any of these symptoms and if you have.

Symptom	I have experienced this symptom over the last 4 weeks	
Abdominal Pain	Yes	No
Sore Throat	Yes	No
Nausea	Yes	No
Weight Loss	Yes	No
Fatigue	Yes	No
Stiff Joints	Yes	No
Sore Eyes	Yes	No
Headaches	Yes	No
Upset Stomach/Diarrhoea	Yes	No
Sleep Difficulties	Yes	No
Dizziness	Yes	No
Loss of Strength	Yes	No
Bloating	Yes	No
Excessive Wind	Yes	No
Breathlessness	Yes	No
Constipation	Yes	No
Heartburn/Indigestion	Yes	No
Mouth Ulcers	Yes	No
Wheeziness	Yes	No
Hair Loss	Yes	No

Appendix E – Focus Group Guide

Introduction

Hello, welcome to the focus group. Firstly, I'd like to thank you all for coming today and I look forward to hearing all of your contributions. My name is Rosie and I am a Phd student from the University of Birmingham. I am currently involved in some research looking into the anxiety around food in people with coeliac disease.

You were invited to this group because you all live with coeliac disease and therefore, know the most about coeliac disease! Please feel free to share your points of view with the group, even if it differs from what others have said. There are no right or wrong answers in this group and I am interested in what all of you have to say. Any difference in opinion will help to fuel a discussion, so please don't be afraid to state any alternative views.

Please help yourself to tea and coffee, and [directions to bathroom]. If you would all read through your information sheet and sign the consent form if you are happy to take part in the discussion and don't mind being recorded during the discussion.

[Health and safety rules depending on location]

Ground Rules

Before we begin, let me share some ground rules. Please speak up clearly, no matter what your opinion. Only one person should talk at a time and everyone should listen to the person who is speaking and respect their opinions.. I will be recording the session because I don't want to miss any of your comments, but, please note that all contributions will be anonymised and neither your identity nor the identity of your service will be associated with transcribed material.Please turn off your mobile phones but if you do need to leave the room, please do so quietly

The duration of the focus group discussion will be approximately 1 hour. Does anybody have any questions?

Firstly, let's find out some more about each person. Can you tell us your name and background?

Questions

- How do you manage your coeliac disease and what symptoms do you experience?
    - Nausea, tummy ache, headache
    - Gluten-free diet
    - What effect would accidental gluten-consumption have on you?
- How does it differ managing your diet inside your home compared to outside your home?
    - Is it more difficult managing the diet outside the home?
    - How does awareness about cross-contamination affect your dietary management?
    - Do you avoid eating outside the home because there is no guarantee that the food is gluten free?

    - Does the availability of gluten free foods make it difficult to manage your diet in some settings?
    - Does your coeliac disease make it harder to socialise around food?
    - Do the challenges involved in managing your diet mean that you have to eat less food in some settings?
- What kind of eating environment makes you feel safe and what makes you feel more concerned?
    - Do you trust gluten free foods?
    - Do you find it more difficult to eat outside the house with your friends?
    - Do you get concerned when eating new or unfamiliar foods?
    - Is it important for you to clean up your kitchenware after contact with gluten-containing foods?
    - Is it important for you to have a sense of control over your food preparation?
    - Does reading food labels make you feel safer around unfamiliar foods?
- When you're out and about, shopping for the day, do you ever have concerns about food availability?
    - How do you cope with the food availability?
    - Are you adventurous with food and restaurant choices?
    - Do you limit the amount you eat when you are out all day?
    - Do you feel that restaurants are able to accommodate your dietary needs?
    - Do you trust it when people say their food is gluten free?
- Do you feel safe preparing gluten-containing foods for others?
    - What if the gluten-containing food comes into contact with the food you have prepared for yourself?
    - Do you get anxious when others are eating gluten around you?
    - Do you prefer to keep a completely gluten free home?
- Do you find it hard to eat foods that resemble those you have previously had a severe reaction to?
    - Has this changed throughout your diagnosis?
    - Has your coeliac disease make you scared of food?
    - Does coeliac disease affect the way you feel about food?

Summary

What do you all feel were the main points that came from this session? Did anything surprise you? Is there anything you would like to discuss further?

Closing Statements

Thank you for taking part today, I'm hoping the answers you have given will help direct future research into coeliac disease. If you do want to withdraw your data

please let me know as soon as possible, either after the session or by email. Does anyone have any questions?

Appendix E – Ethical Approval

Chapter Three

Re: "Disturbed Eating Practices in Coeliac Disease"
Application for amendment ERN_14-0015B

Thank you for the above application for amendment, which was reviewed by the Science, Technology, Engineering and Mathematics Ethical Review Committee.

On behalf of the Committee, I can confirm that this amendment now has full ethical approval.

Chapter Four

Re: "Food Preferences and Individual Differences in Coeliac Disease"
Application for Ethical Review ERN_15-0370

Thank you for your application for ethical review for the above project, which was reviewed by the Science, Technology, Engineering and Mathematics Ethical Review Committee.

On behalf of the Committee, I can confirm the conditions of approval for the study have been met and this study now has full ethical approval.

Chapters Five and Six

Re: "Development of a Food Anxiety Questionnaire in Coeliac Disease"
Application for Amendment ERN_15-0370A

Thank you for your application for amendment to the above study. This has now been reviewed by the Science, Technology, Engineering and Mathematics Ethical Review Committee.

On behalf of the Committee, I can confirm that this amendment now has full ethical approval.

Chapter Seven

Re: "Food Attitudes, Attentional Bias and Eating Behaviours in Coeliac Disease: A Laboratory Study"
Application for Ethical Review ERN_16-0410

Thank you for your application for ethical review for the above project, which was reviewed by the Science, Technology, Engineering and Mathematics Ethical Review Committee.

On behalf of the Committee, I confirm that this study now has full ethical approval.