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The Prevalence and Predictors of Disordered Eating in Women with Coeliac Disease

Purpose: The need for dietary management in coeliac disease may lead to the development of disordered eating. This study examined the prevalence of disordered eating and factors predicting disordered eating in women with coeliac disease, compared with other dietary-controlled conditions.

Methods: A cross-sectional, online survey assessing psychological well-being, disordered eating behaviours (Eating Attitudes Test 26 (EAT-26); Binge Eating Scale (BES)) was distributed using online forums, to those with coeliac disease (N=157), inflammatory bowel disease (N=116), type two diabetes (N=88) and healthy controls (N=142). Hierarchical regressions were conducted to explore and compare the predictors of EAT-26 and BES scores across all groups. Within the coeliac disease group, a cluster analysis was conducted to examine types of disordered eating.

Results: Higher EAT-26 scores were found in those with coeliac disease and inflammatory bowel disease compared with healthy controls and type two diabetes; participants with a chronic health condition had higher BES than healthy control participants. The factors associated with EAT-26 scores differed across the dietary-controlled health conditions, with dietary management being important for those with coeliac disease. Psychological distress was associated with binge-eating behaviour across all groups. Cluster analyses found two types of disordered eating in coeliac disease; a binge eating type and a restrictive type.

Conclusions: Disordered eating attitudes and behaviours are more prevalent in participants with chronic health conditions relative to healthy controls. The presence of binge eating behaviours in coeliac disease may be related to non-coeliac disease specific factors such as

the distress associated with dietary-controlled illness. EAT-26 scores in coeliac disease are associated with disease specific factors, unique to following the gluten-free diet. These factors are important for identifying and supporting those with coeliac disease and disordered eating.

Introduction

Coeliac disease is an autoimmune condition characterised by damage to the small intestine following the ingestion of the protein gluten (NICE, 2015). The condition is managed by a life-long gluten-free diet, requiring the exclusion of wheat, rye, barley and sometimes oats (GFD; Di Sabatino & Corazza, 2009; NICE, 2015). The GFD is the only treatment for coeliac disease; it is effective in reversing intestinal damage and is necessary to avoid complications such as osteoporosis and gastrointestinal cancers (Valdimarsson, Toss, Ross, Lofman & Strom, 1994) However, management of a dietary-controlled health condition, such as coeliac disease, 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 coeliac disease, 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 coeliac disease. The results of two cross-sectional surveys suggest that between 22% and 29% of individuals with coeliac disease 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 coeliac disease 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 coeliac disease.

Case studies offer an understanding of the complex relationship between disordered eating and coeliac disease (Leffler et al., 2007; Ricca et al., 2000; Yucel, Ozbey, Demir, Polat & Yager, 2006). Yucel et al., (2006) suggested that the long-term dietary restraint, necessary in coeliac disease, 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 coeliac disease, larger sample sizes are required.

Prior to diagnosis, some individuals with coeliac disease experience severe gastrointestinal symptoms, which may contribute to the development of disordered eating attitudes and behaviours (Arigo, Anskis & Smyth, 2012; Satherley, Howard & Higgs, 2014). Although most individuals will experience clinical remission on the GFD, some will continue to experience gastrointestinal symptoms, which may result from refractory coeliac disease 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; NIDDK Gastroparesis Clinical Research Consortium). However, the role of gastrointestinal symptoms in coeliac disease 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, 2003). The associations between gastrointestinal symptoms and disordered eating attitudes and behaviours may be explained by the deliberate consumption of gluten in those diagnosed with coeliac disease; 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 coeliac disease.

Satherley, Howard and Higgs' (2014) developed a two-path, theoretical model of disordered eating in gastrointestinal disease, suggesting 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 coeliac disease 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). The model proposed by Satherley, Howard and Higgs (2014) has the potential to help us to interpret and understand the relationships between disordered eating and coeliac disease by testing specific hypotheses.

This study is the first to apply Satherley, Howard and Higgs' (2014) model of disordered eating in gastrointestinal disease to coeliac disease. Given the limitations of prior studies, this study assessed the prevalence, predictors and types of disordered eating in coeliac disease compared to other dietary-controlled conditions. Individuals with coeliac disease, 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 coeliac disease 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 coeliac disease. 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, 2009). 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 coeliac disease diagnosis (GFD management). The most common types of disordered eating patterns related to Binge Eating, Anorexia Nervosa and Bulimia Nervosa, were assessed (NHS, 2015).

We anticipated the following: 1) individuals with dietary-controlled conditions (coeliac disease, 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 coeliac disease), 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 coeliac disease and 5) based on the theoretical model of disordered eating (Satherley, Howard & Higgs, 2014), we expected two types of disordered eating to be present in coeliac disease. 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.

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 self-reported a biopsy-confirmed diagnosis of coeliac disease, 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 coeliac disease, type two diabetes or inflammatory bowel disease (e.g. cystic fibrosis, type I 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 coeliac disease were required to self-report a biopsy confirmed diagnosis.

Participants were recruited through adverts on online support forums (e.g. Facebook) and through Coeliac UK, the main charity supporting people with coeliac disease in the UK. Interested individuals were directed to an online survey to complete the following questionnaires. Men were recruited but only 14 took part, so this data was not analysed.

Measures

Demographic and General Health Information

For participants with type two diabetes, inflammatory bowel disease and coeliac disease, 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 coeliac disease, 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 prescribe 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 symptom since their diagnosis (yes/no). A total

gastrointestinal symptom 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.

The IPQ-R also measures an individual's perceptions of illness, the cause of their illness and their personal views of the illness. Only those with coeliac disease only completed this questionnaire but the results are not reported here, as they are not directly relevant to the aims of this study.

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. Scores on each subscale range from 0 to 42 with higher scores indicating greater distress. The DASS-21 has strong psychometric properties (Brown et al., 1997).

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 scale has been validated numerous times and is the standard measure of food neophobia, with good reliability and validaty (Miselman, King & Gilette, 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.

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 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 bingeing and scores greater than 27 indicating severe binging. 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).

Ethical Approval

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

Statistical Analysis

Data was analysed using the Statistics for the Social Sciences (SPSS) version 22.0. 69 coeliac disease participants were excluded across the groups due to the absence of a biopsy-proven diagnosis. Overall, 77 individuals were removed from the coeliac disease 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 centered 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 coeliac disease 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 coeliac disease 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 coeliac disease sample. Three theoretical groups were hypothesised to come out of the analysis (two disordered and a healthy type) so 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).

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 1 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 coeliac disease 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.

	Coeliac Disease (n=157)	**Inflammatory Bowel Disease (n=116)**	**Type Two Diabetes (n=88)**	**Healthy Controls (n=142)**	**Group Differences**
Age (years)	38 (13.4)	36 (11.98)	47 (12.83)	33 (13.72)	T2D > CD, IBD, HC
Body Mass Index	22.91 (3.83)	23.05 (4.91)	29.13 (3.63)	22.39 (4.75)	T2D > CD, IBD, HC
Years since Diagnosis	9 (10.25)	8 (7.62)	9 (7.29)	-	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).

Prevalence of Disordered Eating in Coeliac Disease 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 ($H(3)=31.84$, $p<.001$). EAT-26 scores were higher in those with coeliac disease than healthy controls ($U=5312.5$, $p=.001$) and those with coeliac disease scored higher than those with type two diabetes ($U=2532$, $p=.001$). There was a significant difference in BES scores across the diagnostic groups ($H(3)=82.41$, $p<.001$). Those with coeliac disease 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	**Coeliac Disease (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 Binge Eating Scale and Eating Attitudes Test-26. EAT-26 and BES scores were compared across all groups ($p<.05$; see group differences column).

Predictors of Disordered Eating

Strong 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)=8.36, p<.001; see Table 3) with distress having a significant positive regression weight.

The disease specific variables were entered in step two (dietary-management and gastrointestinal symptoms). For the coeliac disease group, when predicting EAT-26 score, this model accounted for 54.3% of the variance in EAT-26 scores (F=(6, 90)=20.42, p<.001; see Table 3) with dietary-management and gastrointestinal symptoms having significant positive regression weights. Based on the examination of ß weights, dietary-management has 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=2.87,p=.004) and inflammatory bowel disease (z=6.12,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 coeliac disease and inflammatory bowel disease, the combined direct R^2 = .60 and crossed R^2 = .40 were significantly different (z=2.87,p=.004). There are structural differences between the best regression model for predicting EAT-26 score in those with coeliac disease and inflammatory bowel disease. When looking at coeliac disease and type

two diabetes together, the combined direct R^2 = .60 and crossed R^2 = -.43 were significantly different (z=6.12,p<.001), indicating that there are structural differences between the best regression model for predicting EAT-26 score in those with coeliac disease and type two diabetes.

Further analysis revealed that dietary self-management (z=3.62, p<.001) and DASS-21 scores (z=-2.80, p=.006) had significantly different regression weights in the coeliac disease and inflammatory bowel disease groups, with dietary-management having more influence on EAT-26 scores in those with coeliac disease and DASS-21 scores in those with inflammatory bowel disease. Dietary self-management (z=4.60 p<.001) had a significantly different regression weight in the coeliac disease and type two diabetes groups, with poor dietary self-management being associated with EAT-26 scores in those with coeliac disease. The regression weights for gastrointestinal symptoms were close to significance across coeliac disease and type two diabetes (z=1.90, p=.057). The regression models for the comparison groups are provided in the supplementary materials for comparison but are not central to the aims of the research.

Table 3

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

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 coeliac disease 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)=17.53, 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 all of the diagnostic groups equally well. Comparison of the fit of the disease-nonspecific model across those with type two diabetes (z=-1.33,p=.180) and inflammatory bowel disease (z=0.64,p=.521) revealed no significant difference between the respective R^2 values for BES scores between inflammatory bowel disease, type two diabetes and coeliac disease. 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 Coeliac Disease

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.

Typologies of Eating Attitudes and Behaviour in Coeliac Disease

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 5). 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 Coeliac Disease

Variable	**Low Risk (60)**	**Critical (25)**	**High Distress (11)**
Depression (0-14)	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 (0-15)	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.

Discussion

The primary goal of this study was to explore the prevalence, predictors and types of disordered eating in coeliac disease, inflammatory bowel disease, type two diabetes and healthy controls, and examine whether factors unique to the diagnosis of coeliac disease 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 coeliac disease 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, our research 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 coeliac disease. Individuals with dietary-controlled gastrointestinal conditions may be placed at a unique risk for the development of Anorexic-type attitudes and behaviours. We do not know the nature of these associations, however, 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 model of disordered eating in gastrointestinal disease (Satherley, Howard & Higgs, 2014). 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 coeliac disease 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 coeliac disease.

Our research has identified specific factors that are associated with disordered eating in coeliac disease. In coeliac disease, 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 coeliac disease group. In line with previous research, poor dietary self-management explained addition variance in EAT-26 scores for those with coeliac disease (Arigo, Anskis & Smyth 2012; Karwautz et al., 2008; Wagner et al., 2015). In addition, distress was associated with EAT-26 scores in coeliac disease, however, distress scores were no longer significant when accounting for gastrointestinal symptoms and dietary management in coeliac disease. 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 coeliac disease 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 our 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 coeliac disease. 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 coeliac disease.

Factors common to all conditions (years with condition, psychological distress) were more strongly associated with BES scores across all diagnostic groups. Binge eating in coeliac disease 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).

Limitations and Future Research

The cross-sectional nature of this study limits any conclusions about the sequence of events between disordered eating and coeliac disease diagnosis. Longitudinal studies are essential in determining the timeframe between disordered eating onset and coeliac disease diagnosis. Furthermore, we recognise that online recruitment may create a bias in sampling

which may over/under-inflate problems with eating behaviors and dietary self-management. In addition, our 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 coeliac disease and online methods allowed recruitment of a large sample.

Due to the nature of online data collection, coeliac disease 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 coeliac disease 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 coeliac disease 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 coeliac disease. The development of a scale measuring food anxiety in coeliac disease may allow further investigation of the role of anxiety around food in disordered eating in coeliac disease.

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 dietary management were associated with EAT-26 scores in coeliac disease, indicates that individuals experiencing difficulties in managing their gluten-free diet and reporting gastrointestinal symptoms may benefit from have their eating attitudes and behaviors 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.

Conclusions

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

A small group of people with coeliac disease display poor dietary management and this is associated with disordered eating attitudes and beliefs, lending some support to models of disordered eating in gastrointestinal disorders (Satherley, Howard & Higgs, 2014). The majority of individuals with coeliac disease display a typical eating pattern, but for some, disordered eating behaviours are a feature of their coeliac disease. We have isolated some factors that are specific to coeliac disease 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 coeliac disease and look at ways to identify them and provide support.

References

1. Arigo, D., Anskis, A. M., & Smyth, J. M. (2002). Psychiatric comorbidities in women with celiac disease. *Chronic Illness, 8,* 45-55.
2. Armstrong, R. A. (2014). When to use the bonferroni correction. *Ophthalmic and Physiological Optics, 34,* 502-508.
3. Bernstein, I. L., & Borson, S. Learned food aversion: A component of anorexia syndromes. (1986). Learned food aversion: a component of anorexia syndromes. *Psychological Review, 93,* 462-472.
4. Brown, T. A., Chorpita, B. F., Korotitsch, W., & Barlow, D. H. (1997). Psychometric properties of the Depression Anxiety Stress Scales (DASS) in clinical samples. *Behaviour Research and Therapy, 35*, 79-89.
5. 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,* 339-353.
6. Crow, S., Kendall, D., Praus, B., & Thuras, P. (2001). Binge eating and other psychopathology in patients with type II diabetes mellitus. *International Journal of Eating Disorders, 30*, 222-226.
7. Daum, S., Cellier, C., & Mulder, C. J. (2005). Refractory coeliac disease. Best Practice and Research. *Clinical Gastroenterology, 19*, 413–424.
8. DeVellis, R. F. (2003). *Scale development: Theory and applications* (2nd ed.), California: Sage.
9. Di Sabatino, A., & Corazza, G. R. (2009). Coeliac disease. *Lancet*, *373*, 1480-1493.

10. Dickey, W., & Kearney, N. (2006). Overweight in celiac disease: prevalence, clinical characteristics, and effect of a gluten-free diet. *American Journal of Gastroenterology, 101*, 2356-2359.
11. Dide, E. R., & Fitzgibbon, M. (2005). Binge eating and psychological distress: is the degree of obesity a factor? *Eating Behaviour, 6,* 35-41.
12. 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 Behaviours, 18*, 41-47.
13. 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*, 245-250.
14. 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,* 743-757.
15. Garcia-Manzanares, A., & Lucendo, A. J. (2011). Nutritional and dietary aspects of celiac disease. *Nutrition in Clinical Practice, 26,* 163-173.
16. Garner, D. M., & Garfinkel, P. E. (1979). The eating attitudes test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9,* 273-279.
17. Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine, 12*, 871-878.
18. Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7,* 47–55.
19. Grilo, C. (2006). *Eating and Weight Disorders.* New York: Psychology Press.

20. Guthrie, E. A., Creed, F. H., & Whorwell, P. J. (1990). Eating disorders in patients with irritable bowel syndrome: a comparison with Inflammatory Bowel Disease and peptic ulceration. *European Journal of Gastroenterology and Hepatology, 2*, 471-473.

21. Hauser, W., Janke, K., Lump, B., Gregor, M., & Hinz, A. (2012). Anxiety and depression in adult patients with celiac disease on a gluten-free diet. *World Journal of Gastroenterology, 16,* 2780-2787.

22. 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. *British Medical Journal, 320,* 1563.

23. 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 and Therapeutics, 35*, 723-729.

24. Karwautz, A., Wagner, G., Berger, G., Sinnreich, U., Grylli, V., & Huber, W. D. (2008). Eating pathology in adolescents with celiac disease. *Psychosomatics, 49*, 399-406.

25. 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*, 17-20.

26. 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, 7,* 530-536.

27. 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,* 251-255.

28. Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales.* (2nd. Ed.) Sydney: Psychology Foundation. ISBN 7334-1423-0.

29. Meiselman, H. L., King, S. C., & Gilette, M. (2010). The demographics of neophobia in a large commercial US sample. *Food Quality and Preference, 21*, 893-897.

30. 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,* 2023-2026.

31. Mooi, E., & Sarstedt, M. (2011). A concise guide to market research: the process, data and methods using IBM SPSS statistics. *International Journal of Market Research, 53*, 563–564.

32. Moss-Morris, R., Weinman, J., Petrie, K. J., Horne, R., Cameron, L. D., & Buick, D. (2002). The revised illness perception questionnaire (IPQ-R). *Psychology and Health, 17,* 1-16.

33. Murray, J. A., Watson, T., Clearman, B., & Mitros, F. (2003). Effect of a gluten-free diet on gastrointestinal symptoms in celiac disease. *The American Journal of Clinical Nutrition, 79,* 669-673.

34. Nachman, F., Planzer del Campo, M., Gonzalez, A., Corzo, L., Vazquez, 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, 42,* 685-691.

35. Nadon, G., Feldman, D. E., Dunn, W., & Gisel, E. (2011). Association of sensory processing and eating problems in children with autism spectrum disorders. *Autism Research and Treatment,* DOI: 10.1155/2011/541926

598 36. NHS (2015). Eating disorders. Retrieved June 20 2015, from
599 http://www.nhs.uk/conditions/Eating-disorders/Pages/Introduction.aspx.
600 37. NICE. (2009). The management of type two diabetes. Retrieved March 21 2016,
601 from https://www.nice.org.uk/guidance/cg87
602 38. NICE. (2015). Coeliac disease: Recognition and assessment of disease. Retrieved
603 October 21 2013, from
604 http://www.nice.org.uk/nicemedia/pdf/cg061niceguideline.pdf
605 39. NICE. (2015). Inflammatory bowel disease. Retrieved October 21 2013, from
606 https://www.nice.org.uk/guidance/qs81
607 40. Palmer, P. B., & O'Connell, D. G. (2009). Regression analysis for prediction:
608 understanding the process. *Cardiopulmonary Physical Therapy Journal, 20,* 23-26.
609 41. Passananti, V., Siniscalchi, M., Zingone, F., Bucci, C., Tortora, R., Iovino, P., & Ciacci,
610 C. (2013). Prevalence of eating disorders in adults with celiac disease.
611 *Gastroenterology Research and Practice,* doi: 10.1155/2013/491657
612 42. Pliner, P., & Hobden, K. (1992). Development of a scale to measure the trait of
613 food neophobia in humans. *Appetite, 19,* 105-120.
614 43. Polivy, J., & Herman, P. C. (1985). Dieting and binging: A causal analysis. *American*
615 *Psychologist, 40,* 193-201.
616 44. Quick, V. M., Byrd-Bredbenner, C., & Neumark-Sztainer, D. (2013). Chronic illness
617 and disordered eating: A discussion of the literature. *Advanced Nutrition, 4*, 277-
618 286.
619 45. Remillard, M. L., Mazor, K. M., Cutrona, S. L., Gurwitz, J. H., Tija, J. (2014).
620 Systematic review of the use of online questionnaires among the geriatric
621 population. *Journal of the American Geriatrics Society, 62,*696-705.

46. Ricca, V., Mannucci, E., Calabro, A., Bernardo, M. D., Cabras, P. L., & Rotella, C. M. (2000). Anorexia nervosa and celiac disease: two case reports. *International Journal of Eating Disorders, 27*, 119-122.

47. Satherley, R., Howard, R., & Higgs, S. (2014). Disordered eating in gastrointestinal disorders. *Appetite, 84*, 240-250.

48. Smith, F. M., Latchford, G. J., Hall, R. M., & Dickson, R. A. (2008). Do chronic medical conditions increase the risk of eating disorder? A cross-sectional investigation of eating pathology in adolescent females with scoliosis and diabetes. *Journal of Adolescent Health, 42,* 58-63.

49. Swanson, S. A., Crown, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlated of eating disordered in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry, 7*, 714-723.

50. Tang, T. N., Toner, B. B., Stuckless, N., Dion, K. L., Kaplan, A. S., & Ali, A. (1997). Features of eating disorders in patients with irritable bowel syndrome. *Journal of Psychosomatic Research, 4,* 171-178.

51. The NIKDDK Gastroparesis Clinical Research Consortium. (2011). Dietary intake and nutritional deficiencies in patients with diabetic or idiopathic gastroparesis. *Gastroenterology, 141,* 486-498.

52. Timmerman G. M. (1999). Binge eating scale: further assessment of validity and reliability. *Journal of Applied Biobehavioural Research, 4,* 1-12.

53. Valdimarsson, T., Toss, G., Ross, I., Lofman, 0., & Strom, M. (1994). Bone mineral density in coeliac disease. *Scandinavian Journal of Gastroenterology, 29,* 457-461.

645 54. Wada, Y., Hismatsu, T., Naganuma, M., Matsuoka, K., Okamoto, S., Inoue, N., …
646 Kanai, T. (2015). Risk factors for decreased bone mineral density in inflammatory
647 bowel disease: a cross-sectional study. *Clinical Nutrition, 34,* 1202-1209.
648 55. Wagner, G., Zeiler, M., Berger, G., Huber, W., Favaro, A., Santonastaso, P., &
649 Karwautz, A. (2015). Eating disordered in adolescents with celiac disease: influence
650 of personality characteristics and coping. *European Eating Disorders Review, 23,*
651 361-370.
652 56. Young-Hyman, D. L., & Davis, C. L. (2010). Disordered eating behaviour in
653 individuals with diabetes. *Diabetes Care, 33,* 686-689.
654 57. Yucel, B., Ozbey, N., Demir, K., Plat, A., & Yager, J. (2006). Eating disorders and
655 celiac disease: a case report. *International Journal of Eating Disorders, 39,* 530-532.