

# The prevalence and predictors of disordered eating in women with coeliac disease

Satherley, Rose-Marie; Higgs, Suzanne; Howard, Ruth

DOI:

[10.1016/j.appet.2016.07.038](https://doi.org/10.1016/j.appet.2016.07.038)

License:

Creative Commons: Attribution-NonCommercial-NoDerivs (CC BY-NC-ND)

*Document Version*

Peer reviewed version

*Citation for published version (Harvard):*

Satherley, R-M, Higgs, S & Howard, R 2016, 'The prevalence and predictors of disordered eating in women with coeliac disease', *Appetite*, vol. 107, pp. 260-267. <https://doi.org/10.1016/j.appet.2016.07.038>

[Link to publication on Research at Birmingham portal](#)

## General rights

Unless a licence is specified above, all rights (including copyright and moral rights) in this document are retained by the authors and/or the copyright holders. The express permission of the copyright holder must be obtained for any use of this material other than for purposes permitted by law.

- Users may freely distribute the URL that is used to identify this publication.
- Users may download and/or print one copy of the publication from the University of Birmingham research portal for the purpose of private study or non-commercial research.
- User may use extracts from the document in line with the concept of 'fair dealing' under the Copyright, Designs and Patents Act 1988 (?)
- Users may not further distribute the material nor use it for the purposes of commercial gain.

Where a licence is displayed above, please note the terms and conditions of the licence govern your use of this document.

When citing, please reference the published version.

## Take down policy

While the University of Birmingham exercises care and attention in making items available there are rare occasions when an item has been uploaded in error or has been deemed to be commercially or otherwise sensitive.

If you believe that this is the case for this document, please contact [UBIRA@lists.bham.ac.uk](mailto:UBIRA@lists.bham.ac.uk) providing details and we will remove access to the work immediately and investigate.

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

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

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

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

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

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

## 27 **Introduction**

28 Coeliac disease is an autoimmune condition characterised by damage to the small intestine  
29 following the ingestion of the protein gluten (NICE, 2015). The condition is managed by a  
30 life-long gluten-free diet, requiring the exclusion of wheat, rye, barley and sometimes oats  
31 (GFD; Di Sabatino & Corazza, 2009; NICE, 2015). The GFD is the only treatment for coeliac  
32 disease; it is effective in reversing intestinal damage and is necessary to avoid complications  
33 such as osteoporosis and gastrointestinal cancers (Valdimarsson, Toss, Ross, Lofman &  
34 Strom, 1994). However, management of a dietary-controlled health condition, such as  
35 coeliac disease, creates pressures that may harm one's relationship with food and have  
36 been associated with an increased prevalence of disordered eating attitudes and behaviours  
37 (Quick, Byrd-Bredbenner & Neumark-Sztainer, 2013). Disordered eating describes a  
38 spectrum of eating behaviours, which can range from clinical eating disorders to skipping  
39 meals, binge eating, restricting certain food types or fasting (Grilo, 2006).

40 The risk of developing disordered eating behaviours increases with psychological distress,  
41 which frequently occurs in a range of chronic health conditions (Quick, Byrd-Bredbenner &  
42 Neumark-Sztainer, 2013). Furthermore there is an increased risk of developing disordered  
43 eating in individuals diagnosed with a chronic health condition during puberty, when their  
44 body shape is already changing (Smith, Latchford, Hall & Dickson, 2008). These factors are  
45 common across all chronic health conditions. For individuals with coeliac disease, the need

46 to monitor the gluten content of food, combined with fears about the effectiveness of their  
47 GFD and concerns about the prevention of gastrointestinal symptoms, may additionally  
48 contribute to increased risk of disordered eating (Arigo, Anskis & Smyth, 2012; Karwautz et  
49 al., 2008).

50 To date, there have been few studies of the prevalence of disordered eating in coeliac  
51 disease. The results of two cross-sectional surveys suggest that between 22% and 29% of  
52 individuals with coeliac disease score above the clinical cut-offs on measures assessing  
53 Anorexia and Bulimia Nervosa (Arigo, Anskis & Smyth, 2012; Karwautz et al., 2008). Poor  
54 dietary management, psychological distress and physical symptoms related to coeliac  
55 disease were frequent in those with disordered eating attitudes and behaviours (Arigo,  
56 Anskis & Smyth, 2012; Karwautz et al., 2008; Wagner et al., 2015), however, the absence of  
57 a control group means that it is impossible to determine if the disordered eating is related  
58 to the coeliac diagnosis or if it results from the nonspecific burden of a chronic health  
59 condition. These factors are essential to understand the mechanisms behind disordered  
60 eating in coeliac disease.

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

69 Prior to diagnosis, some individuals with coeliac disease experience severe gastrointestinal  
70 symptoms, which may contribute to the development of disordered eating attitudes and  
71 behaviours (Arigo, Anskis & Smyth, 2012; Satherley, Howard & Higgs, 2014). Although most  
72 individuals will experience clinical remission on the GFD, some will continue to experience  
73 gastrointestinal symptoms, which may result from refractory coeliac disease where the  
74 individual is not responsive to the GFD (Daum, Cellier & Mulder, 2005). Alternatively,  
75 Midhagen and Hallert (2003) suggested that the nutritional composition of the GFD might  
76 be responsible for persistent gastrointestinal symptoms, whereas Nachman et al, (2010)  
77 suggested this results from poor dietary management. Untreated gastrointestinal symptoms  
78 may trigger an aversion to food, which can influence disordered eating attitudes and  
79 behaviours (Berstein & Borson, 1986). Gastrointestinal symptoms have been associated with  
80 food aversion in a variety of chronic health conditions including cancer (Coa et al., 2015),  
81 autism (Nadon, Feldman, Dunn & Gisel, 2011) and gastroparesis (a condition characterised  
82 by delayed gastric emptying; NIDDK Gastroparesis Clinical Research Consortium). However,  
83 the role of gastrointestinal symptoms in coeliac disease and the development of disordered  
84 eating has received little attention.

85 Gastrointestinal symptoms and dietary management are closely associated via a  
86 bidirectional relationship, where good dietary management is associated with fewer and/or  
87 less severe gastrointestinal symptoms, and poor dietary management is associated with  
88 increased/more severe gastrointestinal symptoms (Murray, Eason, Clearman & Mitros,  
89 2003). The associations between gastrointestinal symptoms and disordered eating attitudes  
90 and behaviours may be explained by the deliberate consumption of gluten in those  
91 diagnosed with coeliac disease; Leffler et al., (2007) described cases in which individuals

92 would consume gluten in order to encourage gastrointestinal symptoms to promote weight  
93 loss. However, this phenomenon has only been described in case studies and it is not clear  
94 how these findings will generalise to larger samples. Misuse of dietary regimens has been  
95 reported in diabetes (Young-Hyman & Davis, 2010) and there is potential for this to occur in  
96 coeliac disease.

97 Satherley, Howard and Higgs' (2014) developed a two-path, theoretical model of disordered  
98 eating in gastrointestinal disease, suggesting disordered eating differs depending on beliefs  
99 about the disease and dietary management. The first pathway describes individuals who  
100 experience extreme anxiety around unfamiliar foods and/or overestimate the negative  
101 consequences associated with their condition. These individuals may fear food prepared  
102 outside of their control, and cope with this by eating a limited variety of foods. The second  
103 pathway describes individuals who experience weight gain after commencing their  
104 prescribed dietary regimen and may use techniques to reverse this weight gain. Not all  
105 individuals with coeliac disease will experience weight gain after commencing the GFD;  
106 however, good dietary management has been associated with a post-diagnosis increase in  
107 weight (Kabbani et al., 2012). Prior to coeliac diagnosis, individuals may present as  
108 underweight, meaning that increased weight is an indicator of recovery of the intestine,  
109 however, for some individuals this weight change may be negatively interpreted and trigger  
110 disordered eating. These individuals may recognise the association between weight gain and  
111 the GFD and aim to reduce their weight gain through poor dietary management (Leffler et  
112 al., 2007). The model proposed by Satherley, Howard and Higgs (2014) has the potential to  
113 help us to interpret and understand the relationships between disordered eating and coeliac  
114 disease by testing specific hypotheses.

115 This study is the first to apply Satherley, Howard and Higgs' (2014) model of disordered  
116 eating in gastrointestinal disease to coeliac disease. Given the limitations of prior studies,  
117 this study assessed the prevalence, predictors and types of disordered eating in coeliac  
118 disease compared to other dietary-controlled conditions. Individuals with coeliac disease,  
119 who follow a strict GFD, were compared to those with inflammatory bowel disease and type  
120 two diabetes (both of which have dietary components to their management) and healthy  
121 controls. Dietary management in inflammatory bowel disease and type two diabetes is  
122 unlike that for coeliac disease as it is less strict and regimented when compared to the GFD  
123 and other medical interventions may be required, which is generally not the case in coeliac  
124 disease. Individuals with inflammatory bowel disease experience gastrointestinal symptoms  
125 associated with the ingestion of certain restricted foods, which can differ between patients,  
126 but will avoid these trigger foods during a flare-up and may use medical or surgical  
127 approaches to manage flare-ups (NICE, 2015); those with type two diabetes do not have  
128 gastrointestinal symptoms as a feature of their diagnosis and do not avoid particular food  
129 types, but will follow a balanced diet with an emphasis on consuming high fibre and low-  
130 glycaemic index foods. This may be combined with blood glucose monitoring and insulin  
131 injections (NICE, 2009). These control groups allowed us to explore the role of nonspecific  
132 factors common to *all* dietary-controlled conditions (years with condition, psychological  
133 distress), factors common to gastrointestinal disease (gastrointestinal symptoms) and  
134 factors *unique* to the coeliac disease diagnosis (GFD management). The most common types  
135 of disordered eating patterns related to Binge Eating, Anorexia Nervosa and Bulimia  
136 Nervosa, were assessed (NHS, 2015).

137 We anticipated the following: 1) individuals with dietary-controlled conditions (coeliac  
138 disease, inflammatory bowel disease and type two diabetes) would score greater on  
139 disordered eating measures than healthy controls; 2) psychological distress, a nonspecific  
140 factor, would be associated with disordered eating across all groups; 3) in those with  
141 gastrointestinal disorders (inflammatory bowel disease and coeliac disease), factors unique  
142 to these conditions (gastrointestinal symptoms) would explain additional variance in  
143 disordered eating scores; 4) additional variance in disordered eating would be explained by  
144 dietary-management in coeliac disease and 5) based on the theoretical model of disordered  
145 eating (Satherley, Howard & Higgs, 2014), we expected two types of disordered eating to be  
146 present in coeliac disease. One group of disordered eaters was expected to show good  
147 dietary self-management and few gastrointestinal symptoms, associated with increased  
148 anxiety around new foods. The second group was expected to have poor dietary  
149 management and experience increased gastrointestinal symptoms, associated with gluten  
150 ingestion.

## 151 **Methods**

152 The cross-sectional survey was conducted between June and December 2014. Individuals  
153 living in the United Kingdom, aged between 18-69 years and who self-reported a biopsy-  
154 confirmed diagnosis of coeliac disease, type two diabetes or inflammatory bowel disease,  
155 were eligible to participate. Healthy controls with no reported health conditions or food  
156 allergies were also recruited. Participants were excluded if 1) they reported having a dietary-  
157 controlled condition other than coeliac disease, type two diabetes or inflammatory bowel  
158 disease (e.g. cystic fibrosis, type I diabetes) and 2) if they had any other food allergies.  
159 Individuals with type two diabetes were required to be following a prescribed dietary



160 regimen as a part of their treatment programme and individuals with coeliac disease were  
161 required to self-report a biopsy confirmed diagnosis.

162 Participants were recruited through adverts on online support forums (e.g. Facebook) and  
163 through Coeliac UK, the main charity supporting people with coeliac disease in the UK.

164 Interested individuals were directed to an online survey to complete the following  
165 questionnaires. Men were recruited but only 14 took part, so this data was not analysed.

## 166 *Measures*

### 167 *Demographic and General Health Information*

168 For participants with type two diabetes, inflammatory bowel disease and coeliac disease,  
169 information was gathered on demographics, information relating to diagnosis (method of  
170 diagnosis, date of diagnosis, dietary management) and health status (allergies, medication).

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

179 The presence of gastrointestinal symptoms was assessed using the Illness Perception  
180 Questionnaire Revised (IPQ-R; Moss-Morris et al., 2002). Participants are asked to rate  
181 whether they have experienced a symptom since their diagnosis (yes/no). A total

182 gastrointestinal symptom was calculated by adding up the total of gastrointestinal  
183 symptoms (nausea, weight loss, upset stomach, abdominal pain, bloating, excessive wind,  
184 constipation, indigestion) experienced in the last four weeks, providing a score between 0  
185 and 8, with 8 indicating a greater number of gastrointestinal symptoms.

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

#### 190 *Psychological Distress*

191 The Depression, Anxiety, Stress Scale 21 (DASS-21; Lovibond & Lovibond, 1995) assesses  
192 levels of depression, anxiety and stress. The items consist of statements referring to the  
193 past week, rated on a 4-point scale. Scores on each subscale range from 0 to 42 with higher  
194 scores indicating greater distress. The DASS-21 has strong psychometric properties (Brown  
195 et al., 1997).

#### 196 *Food Anxiety*

197 The Food Neophobia Scale (FNS; Pliner & Hobden, 1992) is a ten-item scale that measures  
198 willingness to try new foods. Scores above 35 are considered high, with lower scores  
199 indicating greater willingness to try unfamiliar foods (Pliner & Hobden, 1992). The scale has  
200 been validated numerous times and is the standard measure of food neophobia, with good  
201 reliability and validity (Miselman, King & Gilette, 2010). At present no appropriate  
202 measures of food anxiety have been developed. The FNS was chosen as the best available  
203 tool to measure anxiety around new foods.

204 *Disordered Eating*

205 Two questionnaires were used to target the differing attitudes and behaviours surrounding  
206 disordered eating, to account for any overlap in disordered eating categories (Eddy et al.,  
207 2008; Swanson et al., 2011).

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

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

225

226 *Ethical Approval*

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

229 *Statistical Analysis*

230 Data was analysed using the Statistics for the Social Sciences (SPSS) version 22.0. 69 coeliac  
231 disease participants were excluded across the groups due to the absence of a biopsy-proven  
232 diagnosis. Overall, 77 individuals were removed from the coeliac disease group, 27 from  
233 type two diabetes and 9 from inflammatory bowel disease and 4 from health controls,  
234 providing 503 participants for analysis.

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

244 The fit of the model across the groups was assessed using three stages: 1) does the  
245 predictor set work better for coeliac disease than other groups; 2) are the models  
246 substitutable and 3) are the regression weights across the groups different. 1) Fishers Z test  
247 was used to compare the  $R^2$  values from each of the groups regression models. A significant

248 p-value ( $<.05$ ) would indicate a difference in model fit across the groups. 2) Differences in  
249 model structure across the diagnostic groups were explored using a cross validation  
250 technique (Palmer & O'Connell, 2009). The regression model from each group was applied  
251 to every other group (e.g. the coeliac disease regression model was applied to all other  
252 diagnostic groups) to create both a "direct" and a "crossed" model. The resulting crossed  $R^2$   
253 and direct  $R^2$  were compared using Hotelling's t-test, a significant p-value ( $<.05$ ) indicates a  
254 difference in model structure across the groups, which requires further investigation. 3) To  
255 examine the individual predictors within the models, regression weights across the groups  
256 were compared.

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

267

## Results

268 Overall, 72.8% of participants identified as White British, 18.6% as White Other, 2% as Asian,  
269 1% as Black and 2.8% as Mixed Background. Table 1 displays the mean age, Body Mass Index  
270 (BMI) and years since diagnosis across the groups. The type two diabetes group were older

271 and had a higher BMI when compared to other diagnostic groups. There were no other  
 272 differences between the groups. The BMI, ethnicity and years with diagnosis for each  
 273 condition were similar to previous samples; however, across all groups our samples were  
 274 younger than previous reports (Hauser et al., 2010; Koro, Bowlin, Bourgeois & Fedder, 2004;  
 275 Wada et al., 2015).

276 68.5% of participants with coeliac disease reported that they followed their GFD “all the  
 277 time”. Of the remaining 31.5%, 9.4% were completely non-adherent and 22.1% were  
 278 partially adherent to the GFD

279 Table 1

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

	<b>Coeliac Disease (n=157)</b>	<b>Inflammatory Bowel Disease (n=116)</b>	<b>Type Two Diabetes (n=88)</b>	<b>Healthy Controls (n=142)</b>	<b>Group Differences</b>
<b>Age (years)</b>	38 (13.4)	36 (11.98)	47 (12.83)	33 (13.72)	T2D > CD, IBD, HC
<b>Body Mass Index</b>	22.91 (3.83)	23.05 (4.91)	29.13 (3.63)	22.39 (4.75)	T2D > CD, IBD, HC
<b>Years since Diagnosis</b>	9 (10.25)	8 (7.62)	9 (7.29)	-	CD= IBD= T2D
<b>Ethnicity (White)</b>	150 (95.5)	108 (93.1)	84 (95.5)	133 (93.0)	CD= IBD= T2D= HC
<b>Ethnicity (Non- White)</b>	7 (4.5)	8 (6.9)	4 (4.5)	10 (7.0)	CD= IBD= T2D= HC

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

285 *Prevalence of Disordered Eating in Coeliac Disease compared to Controls*

286 Table two displays the proportion of participants scoring above the clinical cut-off for the  
287 EAT-26 and the BES and the mean total scores for each group. The Kruskal Wallis tests found  
288 significant differences in mean EAT-26 scores across the diagnostic groups ( $H(3)=31.84$ ,  
289  $p<.001$ ). EAT-26 scores were higher in those with coeliac disease than healthy controls  
290 ( $U=5312.5$ ,  $p=.001$ ) and those with coeliac disease scored higher than those with type two  
291 diabetes ( $U=2532$ ,  $p=.001$ ). There was a significant difference in BES scores across the  
292 diagnostic groups ( $H(3)=82.41$ ,  $p<.001$ ). Those with coeliac disease had higher BES scores  
293 than healthy controls ( $U=3947$ ,  $p<.001$ ) but scored lower than those with type two diabetes  
294 ( $U=2268$ ,  $p=.001$ ).

295 Table 2

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

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

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

298 The number in brackets represents the percentage of participants scoring above the pre-determined clinical cut-offs for the Binge Eating Scale

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



300 *Predictors of Disordered Eating*

301 Strong associations ( $p < .008$ ) were found for scores on the EAT-26 and BES, and measures of  
302 psychological distress, as well as age, BMI, symptoms and GFD management. These factors  
303 were added as covariates. Based on the significant relationships with disordered eating and  
304 between the subscales, total DASS-21 scores were entered into step one of the regression  
305 model. Years with condition, BMI and age were also added. This model accounted for 23.1%  
306 of the variance in EAT-26 scores ( $F(4, 90) = 8.36, p < .001$ ; see Table 3) with distress having a  
307 significant positive regression weight.

308 The disease specific variables were entered in step two (dietary-management and  
309 gastrointestinal symptoms). For the coeliac disease group, when predicting EAT-26 score,  
310 this model accounted for 54.3% of the variance in EAT-26 scores ( $F(6, 90) = 20.42, p < .001$ ;  
311 see Table 3) with dietary-management and gastrointestinal symptoms having significant  
312 positive regression weights. Based on the examination of  $\beta$  weights, dietary-management  
313 has the major contribution.

314 The overall model predicted total EAT-26 score equally well for all of the diagnostic groups.  
315 Comparison of the fit of the model across those with type two diabetes ( $z = 2.87, p = .004$ ) and  
316 inflammatory bowel disease ( $z = 6.12, p < .001$ ) revealed that there was no significant  
317 difference between the respective  $R^2$  values for the EAT-26 score.

318 When examining the model structure across the groups, structural differences were found.  
319 When looking at coeliac disease and inflammatory bowel disease, the combined direct  $R^2 =$   
320  $.60$  and crossed  $R^2 = .40$  were significantly different ( $z = 2.87, p = .004$ ). There are structural  
321 differences between the best regression model for predicting EAT-26 score in those with  
322 coeliac disease and inflammatory bowel disease. When looking at coeliac disease and type

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

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

338

339

340

341

342

343

344

345 Table 3

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

Predictors	B	B	R <sup>2</sup>	F	R <sup>2</sup> Change
<b>Model 1) Non-specific Factors</b>					
Age	-.02	-.03			
Body Mass Index	-.24	-.12			
Years with Condition	.01	.08			
DASS-21	.21	.04*	.26	8.36*	.26*
<b>Model 2) Disease Specific Factors</b>					
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*

347 \* = significance at p&lt;.008. The significance of the F value refers to the F associated with each

348 step.

349 For the coeliac disease group, when predicting BES score, collectively this model (disease

350 non-specific factors) accounted for 41.8% of the variance in BES scores (F=(4,86)=17.53,

351 p&lt;.001; see table 4) with distress having a significant positive regression weight. The

352 addition of disease-specific factors only explained no additional variance.

353 The overall model fit all of the diagnostic groups equally well. Comparison of the fit of the

354 disease-nonspecific model across those with type two diabetes (z=-1.33,p=.180) and

355 inflammatory bowel disease (z=0.64,p=.521) revealed no significant difference between the

356 respective R<sup>2</sup> values for BES scores between inflammatory bowel disease, type two diabetes357 and coeliac disease. These predictors do equally well across the groups. Examination of  $\beta$

358 weights found a positive association between depression and BES scores across all of the  
359 groups.

360 Table 4

361 *Disease specific and Non-Specific Factors in Predicting BES Scores in Coeliac Disease*

Predictors	B	B	R <sup>2</sup>	F	R <sup>2</sup> Change
<b>Model 1) Non-specific Factors</b>					
Age	-.13	-.14			
Body Mass Index	.71	.23			
Years with Condition	-.07	-.06			
DASS-21	.33	.51*	.44	17.53*	.44*
<b>Model 2) Disease Specific Factors</b>					
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

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

364 *Typologies of Eating Attitudes and Behaviour in Coeliac Disease*

365 Three groups emerged from the cluster analysis producing a “fair” model with a silhouette  
366 measure of cohesion and separation of 0.5 (Mooi & Sarstedt, 2011). The first group was the  
367 largest (N=60) containing those with low psychological distress, few gastrointestinal  
368 symptoms, good dietary-management and low scores on all disordered eating measures.  
369 These were determined to be the “low risk” group. The second group contained 25

370 participants. This group was named the “critical” group. These individuals’ scored high on  
 371 EAT-26, and reported poor dietary self-management, many gastrointestinal symptoms and  
 372 moderate stress scores. The “high distress” group included 11 individuals with high BES  
 373 scores; this group scored highest on all measures of psychological distress but show good  
 374 dietary-management. The Kruskal Wallis tests found significant differences in all variables  
 375 across the three groups (see Table 5). Further post-hoc Mann-Whitney tests revealed that  
 376 when the critical group and the high distress group were compared to the low risk group,  
 377 significant differences were found across all of the variables ( $p < .05$ ).

378 Table 5

379 *Cluster Analysis in Individuals with Coeliac Disease*

<i>Variable</i>	<b>Low Risk (60)</b>	<b>Critical (25)</b>	<b>High Distress (11)</b>
<b>Depression (0-14)</b>	1.72	5.4	12
<b>BES Total (0-46)</b>	6.58	11.44	39
<b>Stress (0-17)</b>	3.57	8.72	14.45
<b>GFD Management (Always-Never)</b>	Always	Most of the time	Always
<b>EAT-26 Total (10- 40)</b>	8.3	18.96	10.36
<b>Gastrointestinal Symptoms (0-15)</b>	7.13	11.72	13.82

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

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

385

**Discussion**

386

387

388

389

390

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.

391

392

393

394

395

396

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).

397

398

399

400

401

402

403

404

405

406

407

408

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

409 prescribed dietary-regimen may interact with disordered eating; the consumption of foods  
410 that trigger gastrointestinal symptoms may be used to promote weight loss (Leffler et al.,  
411 2007; Yucel et al., 2006). Furthermore, larger studies in coeliac disease have found  
412 associations between disordered eating scores and dietary transgressions (Wagner et al.,  
413 2015). A similar phenomenon has been described in type one diabetes, where individuals  
414 may withhold insulin to promote weight loss (Jones, Lawson, Daneman, Olmsted & Rodin,  
415 2000). Future research should focus on the role of gastrointestinal symptoms, dietary-  
416 management and disordered eating in coeliac disease.

417 Our research has identified specific factors that are associated with disordered eating in  
418 coeliac disease. In coeliac disease, disease specific factors explained additional variance in  
419 EAT-26 scores (29.7%) when compared to disease-nonspecific factors, and dietary  
420 management was only important for the coeliac disease group. In line with previous  
421 research, poor dietary self-management explained additional variance in EAT-26 scores for  
422 those with coeliac disease (Arigo, Anskis & Smyth 2012; Karwautz et al., 2008; Wagner et  
423 al., 2015). In addition, distress was associated with EAT-26 scores in coeliac disease,  
424 however, distress scores were no longer significant when accounting for gastrointestinal  
425 symptoms and dietary management in coeliac disease. Furthermore, the cluster analysis  
426 produced a “critical” group who scored high on the EAT-26 but reported poorer dietary self-  
427 management. This suggests that a small group of individuals with coeliac disease may have a  
428 difficult relationship with food. Some individuals may engage in poor dietary self-  
429 management in order to promote villous atrophy and subsequent weight loss (Leffler et al.,  
430 2007). This offers one interpretation of our results; however, the self-reported measures of  
431 dietary self-management and the motivations behind poor management are unclear.

432 When compared with healthy controls, all dietary-controlled diagnostic groups had  
433 increased scores on the BES. Binge eating is commonly reported in those with type two  
434 diabetes, so it is unsurprising that those with type two diabetes scored highest on these  
435 measures (Crow, Kendall, Praus & Thuras, 2001). Binge eating has not previously been  
436 reported in those with coeliac disease. In the United Kingdom, it has been reported that up  
437 to 81% of individuals gain weight after commencing the GFD (Dickey & Kearney, 2006). This  
438 weight gain has been attributed to factors including the poor nutritional quality of some  
439 gluten-free foods, resulting in an increased energy intake, and intestinal recovery (Garcia-  
440 Manzanares & Lucendo, 2011; Kabbani et al., 2012); however for a subset of individuals, our  
441 results suggest that binge eating may also play a role in weight gain. Future research should  
442 focus on the relationship between binge eating and weight changes in coeliac disease.

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

#### 451 *Limitations and Future Research*

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



456 which may over/under-inflate problems with eating behaviors and dietary self-  
457 management. In addition, our samples were younger than those previously reported across  
458 all conditions. This may be due to the nature of online sampling, which is likely to attract a  
459 younger population (Remillard et al., 2014). Despite these limitations, this study provides an  
460 important extension in exploring disordered eating in those with coeliac disease and online  
461 methods allowed recruitment of a large sample.

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

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

478 *Clinical Implications*

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

#### 488 *Conclusions*

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

493 A small group of people with coeliac disease display poor dietary management and this is  
494 associated with disordered eating attitudes and beliefs, lending some support to models of  
495 disordered eating in gastrointestinal disorders (Satherley, Howard & Higgs, 2014). The  
496 majority of individuals with coeliac disease display a typical eating pattern, but for some,  
497 disordered eating behaviours are a feature of their coeliac disease. We have isolated some  
498 factors that are specific to coeliac disease that may place individuals at increased risk for  
499 disordered eating attitudes and behaviours. Future research should focus on understanding  
500 this sub-group of individuals with coeliac disease and look at ways to identify them and  
501 provide support.

502

**References**

503

1. Arigo, D., Anskis, A. M., & Smyth, J. M. (2002). Psychiatric comorbidities in women with celiac disease. *Chronic Illness, 8*, 45-55.

504

505

2. Armstrong, R. A. (2014). When to use the bonferroni correction. *Ophthalmic and Physiological Optics, 34*, 502-508.

506

507

3. Bernstein, I. L., & Borson, S. Learned food aversion: A component of anorexia syndromes. (1986). Learned food aversion: a component of anorexia syndromes.

508

509

*Psychological Review, 93*, 462-472.

510

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.

511

512

*Behaviour Research and Therapy, 35*, 79-89.

513

5. Coa, K. I., Epstein, J. B., Ettinger, D., Jatoi, A., McManus, K., Platek, M. E., ...

514

Moskowitz, B. (2015). The impact of cancer treatment on the diets and food

515

preferences of patients receiving outpatient treatment. *Nutrition and Cancer, 67*,

516

339-353.

517

6. Crow, S., Kendall, D., Praus, B., & Thuras, P. (2001). Binge eating and other

518

psychopathology in patients with type II diabetes mellitus. *International Journal of*

519

*Eating Disorders, 30*, 222-226.

520

7. Daum, S., Cellier, C., & Mulder, C. J. (2005). Refractory coeliac disease. Best

521

Practice and Research. *Clinical Gastroenterology, 19*, 413-424.

522

8. DeVellis, R. F. (2003). *Scale development: Theory and applications* (2nd ed.),

523

California: Sage.

524

9. Di Sabatino, A., & Corazza, G. R. (2009). Coeliac disease. *Lancet, 373*, 1480-1493.

- 525 10. Dickey, W., & Kearney, N. (2006). Overweight in celiac disease: prevalence, clinical  
526 characteristics, and effect of a gluten-free diet. *American Journal of*  
527 *Gastroenterology*, *101*, 2356-2359.
- 528 11. Dide, E. R., & Fitzgibbon, M. (2005). Binge eating and psychological distress: is the  
529 degree of obesity a factor? *Eating Behaviour*, *6*, 35-41.
- 530 12. Duarte, C., Pinto-Gouveia, J., & Ferreira, C. (2015). Expanding binge eating  
531 assessment: Validity and screening value of the binge eating scale in women from  
532 the general population. *Eating Behaviours*, *18*, 41-47.
- 533 13. Eddy, K. T., Dorer, D. J., Franko, D. L., Tahilani, K., Thompson-Brenner, H., &  
534 Herzog, D. B. (2008). Diagnostic crossover in anorexia nervosa and bulimia  
535 nervosa: implications for DSM-V. *The American Journal of Psychiatry*, *165*, 245-  
536 250.
- 537 14. Ford, S., Howard, R., & Oyeboode, J. (2012). Psychosocial aspects of coeliac disease:  
538 A cross-sectional survey of a UK population. *British Journal of Health Psychology*,  
539 *17*, 743-757.
- 540 15. Garcia-Manzanares, A., & Lucendo, A. J. (2011). Nutritional and dietary aspects of  
541 celiac disease. *Nutrition in Clinical Practice*, *26*, 163-173.
- 542 16. Garner, D. M., & Garfinkel, P. E. (1979). The eating attitudes test: An index of the  
543 symptoms of anorexia nervosa. *Psychological Medicine*, *9*, 273-279.
- 544 17. Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating  
545 attitudes test: psychometric features and clinical correlates. *Psychological*  
546 *Medicine*, *12*, 871-878.
- 547 18. Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge  
548 eating severity among obese persons. *Addictive Behaviors*, *7*, 47-55.
- 549 19. Grilo, C. (2006). *Eating and Weight Disorders*. New York: Psychology Press.

- 550 20. Guthrie, E. A., Creed, F. H., & Whorwell, P. J. (1990). Eating disorders in patients  
551 with irritable bowel syndrome: a comparison with Inflammatory Bowel Disease  
552 and peptic ulceration. *European Journal of Gastroenterology and Hepatology*, 2,  
553 471-473.
- 554 21. Hauser, W., Janke, K., Lump, B., Gregor, M., & Hinz, A. (2012). Anxiety and  
555 depression in adult patients with celiac disease on a gluten-free diet. *World*  
556 *Journal of Gastroenterology*, 16, 2780-2787.
- 557 22. Jones, J. M., Lawson, M. L., Daneman, D., Olmsted, M. P., & Rodin, G. (2000).  
558 Eating disorders in adolescent females with and without type 1 diabetes: cross  
559 sectional study. *British Medical Journal*, 320, 1563.
- 560 23. Kabbani, T. A., Goldberg, A., Kelly, C.P, Pallav, K., Tariq, S., Peer, A., ... Leffler, D. A.  
561 (2012). Body mass index and the risk of obesity in Coeliac disease treated with the  
562 gluten-free diet. *Alimentary Pharmacology and Therapeutics*, 35, 723-729.
- 563 24. Karwautz, A., Wagner, G., Berger, G., Sinnreich, U., Grylli, V., & Huber, W. D.  
564 (2008). Eating pathology in adolescents with celiac disease. *Psychosomatics*, 49,  
565 399-406.
- 566 25. Koro, C. E., Bowlin, S. J., Bourgeois, N., & Fedder, D. O. (2004). Glycemic control  
567 from 1988 to 2000 among U.S. adults diagnosed with type 2 diabetes: a  
568 preliminary report. *Diabetes Care*, 27, 17-20.
- 569 26. Leffler, D. A., Dennis, M., Edwards George, J. B., Jamma, S., Magge, S., Cook, E. F...  
570 Kelly, C. P. (2009). A simple validated gluten-free diet adherence survey for adults  
571 with celiac disease. *Clinical Gastroenterology and Hepatology*, 7, 530-536.
- 572 27. Leffler, D. A., Dennis, M., Edwards-George, J. B., & Kelly, C. P. (2007). The  
573 interaction between eating disorders and celiac disease: an exploration of 10  
574 cases. *European Journal of Gastroenterology & Hepatology*, 19, 251-255.

- 575 28. Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress*  
576 *Scales*. (2<sup>nd</sup>. Ed.) Sydney: Psychology Foundation. ISBN 7334-1423-0.
- 577 29. Meiselman, H. L., King, S. C., & Gilette, M. (2010). The demographics of neophobia  
578 in a large commercial US sample. *Food Quality and Preference, 21*, 893-897.
- 579 30. Midhagen, G., & Hallert, C. (2003). High rate of gastrointestinal symptoms in celiac  
580 patients living on a gluten-free diet: controlled study. *The American Journal of*  
581 *Gastroenterology, 98*, 2023-2026.
- 582 31. Mooi, E., & Sarstedt, M. (2011). A concise guide to market research: the process,  
583 data and methods using IBM SPSS statistics. *International Journal of Market*  
584 *Research, 53*, 563–564.
- 585 32. Moss-Morris, R., Weinman, J., Petrie, K. J., Horne, R., Cameron, L. D., & Buick, D.  
586 (2002). The revised illness perception questionnaire (IPQ-R). *Psychology and*  
587 *Health, 17*, 1-16.
- 588 33. Murray, J. A., Watson, T., Clearman, B., & Mitros, F. (2003). Effect of a gluten-free  
589 diet on gastrointestinal symptoms in celiac disease. *The American Journal of*  
590 *Clinical Nutrition, 79*, 669-673.
- 591 34. Nachman, F., Planzer del Campo, M., Gonzalez, A., Corzo, L., Vazquez, H., Sfoggia,  
592 C... Bai, J. C. (2010). Long-term deterioration of quality of life in adult patients with  
593 celiac disease is associated with treatment noncompliance. *Digestive and Liver*  
594 *Disease, 42*, 685-691.
- 595 35. Nadon, G., Feldman, D. E., Dunn, W., & Gisell, E. (2011). Association of sensory  
596 processing and eating problems in children with autism spectrum disorders.  
597 *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.

- 622 46. Ricca, V., Mannucci, E., Calabro, A., Bernardo, M. D., Cabras, P. L., & Rotella, C. M.  
623 (2000). Anorexia nervosa and celiac disease: two case reports. *International*  
624 *Journal of Eating Disorders*, 27, 119-122.
- 625 47. Satherley, R., Howard, R., & Higgs, S. (2014). Disordered eating in gastrointestinal  
626 disorders. *Appetite*, 84, 240-250.
- 627 48. Smith, F. M., Latchford, G. J., Hall, R. M., & Dickson, R. A. (2008). Do chronic  
628 medical conditions increase the risk of eating disorder? A cross-sectional  
629 investigation of eating pathology in adolescent females with scoliosis and diabetes.  
630 *Journal of Adolescent Health*, 42, 58-63.
- 631 49. Swanson, S. A., Crown, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R.  
632 (2011). Prevalence and correlated of eating disorders in adolescents. Results  
633 from the national comorbidity survey replication adolescent supplement. *Archives*  
634 *of General Psychiatry*, 7, 714-723.
- 635 50. Tang, T. N., Toner, B. B., Stuckless, N., Dion, K. L., Kaplan, A. S., & Ali, A. (1997).  
636 Features of eating disorders in patients with irritable bowel syndrome. *Journal of*  
637 *Psychosomatic Research*, 4, 171-178.
- 638 51. The NIKDDK Gastroparesis Clinical Research Consortium. (2011). Dietary intake  
639 and nutritional deficiencies in patients with diabetic or idiopathic gastroparesis.  
640 *Gastroenterology*, 141, 486-498.
- 641 52. Timmerman G. M. (1999). Binge eating scale: further assessment of validity and  
642 reliability. *Journal of Applied Biobehavioural Research*, 4, 1-12.
- 643 53. Valdimarsson, T., Toss, G., Ross, I., Lofman, O., & Strom, M. (1994). Bone mineral  
644 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 disorders 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.