

Disordered eating practices in gastrointestinal disorders

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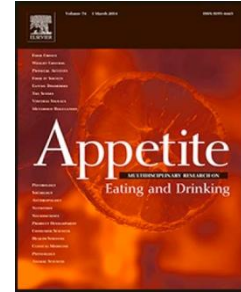
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Disordered Eating Practices in Gastrointestinal Disorders

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Key words: Eating disorder; disordered eating; gastrointestinal; coeliac disease;
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Disordered Eating Practices in Gastrointestinal Disorders**Highlights**

- Evidence of disordered eating patterns in GI disease was reviewed.
- The prevalence of disordered eating in gastrointestinal disease was 5.3-44.4%
- This was associated with distress, symptom severity and dietary management.
- A conceptual model of disordered eating in GI disease was developed.

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22 Abstract

23 **Purpose:** To systematically review evidence concerning disordered eating practices

24 in dietary-controlled gastrointestinal conditions. Three key questions were

25 examined: a) are disordered eating practices a feature of GI disorders?; b) what

26 abnormal eating practices are present in those with GI disorders?; and c) what

27 factors are associated with the presence of disordered eating in those with GI

28 disorders? By exploring these questions, we aim to develop a conceptual model of

29 disordered eating development in GI disease.

30 **Methods:** Five key databases, Web of Science with Conference Proceedings (1900-

31 2014) and MEDLINE (1950-2014), Pubmed, PsychINFO (1967-2014) and Google

32 Scholar, were searched for papers relating to disordered eating practices in those

33 with GI disorders. All papers were quality assessed before being included in the

34 review.

35 **Results:** Nine papers were included in the review. The majority of papers reported

36 that the prevalence of disordered eating behaviours is greater in populations with GI

37 disorders than in populations of healthy controls. Disordered eating patterns in

38 dietary-controlled GI disorders may be associated with both anxiety and GI

39 symptoms. Evidence concerning the correlates of disordered eating was limited.

40 **Conclusions:** The presence of disordered eating behaviours is greater in populations

41 with GI disorders than in populations of healthy controls, but the direction of the

42 relationship is not clear. Implications for further research are discussed.

43

44

Introduction

45 Disruptions to the gastrointestinal (GI) tract result in GI disorders including coeliac
46 disease (CD), irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD).

47 The symptoms associated with these disorders include nausea, bloating,

48 constipation, diarrhoea, changes in weight and abdominal pain. CD, IBS and IBD can

49 all be managed via a life-long modification of the daily diet to avoid GI symptoms

50 (Gibson & Shepherd, 2010). Dietary plans and foods that trigger symptoms vary

51 across GI conditions. In those with CD, it is necessary to follow a strict, life-long

52 gluten-free diet, whereas individuals with IBD and IBS have a less structured dietary

53 regimen that involves trial and error to identify trigger foods (NICE, 2009; Yamaoto,

54 Nakahigashi & Saniabadi, 2009).

55 Dietary-controlled GI disorders may place individuals at risk for the development of

56 disordered eating (DE) patterns. DE describes abnormal eating behaviours that may

57 include skipping meals, binge eating, restricting certain food types or fasting (Grilo,

58 2006). These eating patterns are deviations from the cultural standard of 3 meals a

59 day, which is often found in Western cultures (Fjellstrom, 2004). In this article, we

60 use the term "Disordered Eating (DE)" to indicate any deviation from these cultural

61 norms, including food restriction, skipping meals and over-eating. These deviations

62 from cultural norms may be related to later development of an eating disorder but

63 they do not necessarily indicate that an eating disorder is present. Dietary restraint,

64 GI symptoms, food awareness and the non-specific burden of chronic illness may act

65 as triggers for the development of DE patterns in those with CD, IBS and IBD.

66 Before diagnosis, individuals with dietary-controlled GI disorders will often
67 experience uncomfortable, embarrassing and distressing symptoms when
68 consuming offending food items (Bohn, Storsrud, Tornblom, Bengtsson & Simren,
69 2013; NICE, 2009). These symptoms may become associated with certain types of
70 food or with food in general, creating the potential for a conditioned food aversion
71 to develop (Garcia, Kimeldorf & Koelling, 1955). This may be similar to the
72 development of food aversions in chemotherapy patients (Berteretche et al., 2004).
73 A fear of being contaminated by unknown food sources has repeatedly been
74 reported in the literature across the dietary-controlled GI disorders (Sverker,
75 Hensing & Hallert, 2005; Teufel et al., 2007). This may feed into the development of
76 DE patterns when individuals become too afraid to consume a variety of foods and
77 subsequently begin to restrict their intake.

78 All dietary-controlled GI disorders require some form of prescribed dietary
79 restriction as part of their management. Food restriction, whether it is done as part
80 of a medical regimen or to promote health, is associated with altered eating patterns
81 (Johnson, Pratt, & Wardle, 2012; Herman & Polivy, 1980). The prescribed dietary
82 restraint in IBS, IBD and CD may place these individuals at risk for abnormal eating
83 patterns (Keller, 2008). Prescribed dietary regimens may result in the development
84 of harmful thoughts and attitudes towards food and body weight, which may in turn,
85 lead to inappropriate eating practices (Nicholas et al., 2007). This phenomenon has
86 been demonstrated in those with dietary-controlled chronic health conditions
87 including Diabetes and Cystic Fibrosis (Quick, Byrd-Bredbenner & Neumark-Sztainer,
88 2013).

89 Visible signs of illness in GI disorders are accompanied by embarrassing symptoms
90 (Creed et al., 2006). Subsequently, individuals with GI disorders may become the
91 target of bullying, which may contribute to a lower self-confidence and create a
92 heightened awareness of one's body (Quick, McWilliams & Byrd-Bredbenner, 2014).
93 In combination with essential dietary modifications and subsequent food awareness,
94 these factors may result in those with dietary-controlled GI conditions being at
95 greater risk for DE patterns (De Rosa, Troncone, Vacca & Ciacci, 2004).

96 Numerous case studies have described the co-occurrence of GI disorders and DE
97 behaviours. The incidence of Bulimia Nervosa (BN), pica, obesity, Anorexia Nervosa
98 (AN) and Eating Disorder Not Otherwise Specified (ENDOS) have been reported
99 (Bayle & Bouvard, 2003; Leffler, Dennis, Edwards-George & Kelly, 2007; Mallert &
100 Murch, 1990; Nied, Gillespie & Riedel, 2011, Oso & Fraser, 2005). In addition, the
101 deliberate consumption of trigger foods to avoid weight gain has been indicated
102 (Leffler et al., 2007). However, to our knowledge there has been no systematic
103 review of the prevalence and aetiology of these difficulties in representative
104 samples. The present work aimed to answer three questions: a) are DE practices a
105 feature of GI disorders?; b) what abnormal eating practices are present in those with
106 GI disorders?; and c) what factors are associated with the presence of DE in those
107 with GI disorders?

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111 **Methods**

112 *Search Strategy*

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

118 *Eligibility Criteria*

119 To be included in the review, the articles had to meet stringent criteria. Only studies
120 published during or after 1990 were included as this was a period of change for the
121 diagnosis of GI conditions (ESPGHAN, 1990). In addition, articles had to be written in
122 the English language and include participants between 10-80 years with a physician
123 validated diagnosis of CD, IBS or IBD. Those articles that had not been peer reviewed
124 were excluded, as well as case studies and case series. For a summary of the
125 selection process refer to Figure 1.

126 *Participants:* Studies included youths and working-age adults (10-80 years) with a
127 physician provided diagnosis of CD, IBS or IBD. Those reports focusing on other GI
128 food-related allergies were excluded. Any articles looking at the presence of GI
129 disorders in populations already diagnosed with an eating disorder were excluded.
130 The relationship between eating disorder onset and subsequent GI symptoms has
131 been well documented (Abraham & Kellow, 2013; Peat et al., 2013; Perkins et al.,

132 2005); this review concerns the presence of DE in those with diagnosed GI
133 conditions.

134 *Outcome Measures:* The articles included in the review were related to the eating
135 patterns of those with IBS, IBD or CD. Studies were required to measure food intake
136 or eating patterns as well as any presence of DE behaviours.

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

140

141

142 *Quality Assessment*

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

153 consensus. All of the papers included in the review had high quality scores (M=0.89;
154 see Table 1).

155 *Extraction of Data*

156 *Participant Characteristics*

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

158 *Intervention/Study*

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

162

163 *Comparator/Control Group*

164 The presence and characteristics of the control groups were noted.

165 *Outcome Measure*

166 We extracted the percentage prevalence of DE behaviours evident in the samples, as
167 well as the types of DE behaviours (bingeing, restriction, vomiting) and any factors
168 that were associated with or predicted DE behaviours.

169 *Study Design*

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

172

Results

173 This section will contain a brief overview of the selected studies. After applying the
174 critical appraisal criteria, 9 articles were available for review. These articles used
175 mixed methods. The data from these articles are presented in Table 1.

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178 *Participant Characteristics*

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

183 *Comparator/Control Groups*

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

187 *Outcome Measure*

188 DE patterns were assessed in all of the papers; however, only four of the studies provided
189 information concerning the prevalence of DE across the samples (Addolorato et al., 1997;
190 Arigo et al., 2012; Guthrie et al., 1990; Karwautz et al., 2008). Seven papers provided
191 information concerning the correlates of DE.

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

198

199 *Study Design*

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

202 **Synthesis of Results**

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

206 *Studies Concerning the Prevalence of Disordered Eating in GI Disorders*

207 Four of the articles reported the prevalence of patterns suggestive of DE, although this was
208 assessed using differing methods (Addolorato et al., 1997; Arigo et al., 2012; Guthrie et al.,
209 1990; Karwautz et al., 2008). Across these four papers there were a total of 691 participants
210 with GI disease. Of these, 23.43% displayed eating patterns that were suggestive of DE.
211 Across these papers, DE patterns ranged between 5.3-44.4% in those with GI disease.

212 Prevalence rates for the Eating Disorders Examination (Fairburn & Beglin, 1994) ranged
213 between 22-29.3% (Arigo et al., 2012; Karwautz et al., 2008). These scores are in excess of
214 the scores reported for the general population (10%; Solmi, Hatch, Hotopf, Treasure &
215 Micali, 2014). However, those papers using the EAT (Garner, Olmstead, Bohr & Garfinkel,
216 1982) reported lower prevalence rates (Guthrie et al., 1990; Sullivan et al., 1997). Only one
217 of the papers reported lower prevalence of DE in participants with GI disease than healthy
218 controls (Sullivan et al., 1997). Unfortunately, Sullivan et al. (1997) reported only the means

219 for the EAT scores and did not report what percentage scored above the cut-off criteria.

220 However, they acknowledge that a subgroup of their participants with IBS may have

221 engaged in DE practices.

222 Studies that assessed eating patterns via a food diary reported that for participants with GI

223 disease had lower intake than healthy controls (Addolorato et al., 1997; Fletcher et al.,

224 2008). Addolorato et al. (1997) found that individuals with IBD had a daily calorie intake that

225 was significantly lower than that of controls. Furthermore, 37.2% of those with Crohn's

226 disease and 44.4% of those with ulcerative colitis showed evidence of malnutrition,

227 indicating that these individuals are not meeting their daily dietary needs. Although DE was

228 not assessed, a lack of food intake was observed in this group, the cause of which remains

229 unclear.

230 When combined, the evidence indicates that the presence of DE may be greater in those

231 with GI disease than the reported norms for healthy controls. The conflicting results may be

232 accounted for by the differing use of screening tools as well as factors such as the duration

233 of diagnosis and the type of medical support received.

234 *Studies Concerning the Types of Disordered Eating*

235 Eight of the articles made some reference to the type of DE that was presented by

236 participants (n=2988). This largely depended on the method used to assess DE. However,

237 the majority of articles described DE as a whole, rather than breaking it into subtypes.

238 Food restriction was commonly referred to throughout the articles (Addolorato et al., 1997;

239 Fletcher et al., 2008; Okami et al., 2011). Individuals with GI disease ate more irregular

240 meals and skipped meals more frequently than control participants (Okami et al., 2011).

241 Although consumption of less food was observed in those with GI disorders, it is not clear
242 why this was the case and if intentional food restriction was the cause. Fletcher et al. (2008)
243 found that individuals reported using food restriction as a way to cope with their GI
244 symptoms, often avoiding food when engaging in social activities. Participants said that they
245 would not eat during the day but would eat normally when in the home during the evening,
246 resulting in an abnormal pattern of food intake. In contrast, Tang et al.'s (1997) findings are
247 suggestive of a purging eating pathology. Tang et al. (1997) found that those IBS patients
248 who reported greater vomiting symptoms were more likely to endorse the beliefs of the
249 Bulimia subscale of the EDI. These individuals had thoughts of vomiting as a means of
250 weight reduction but did not necessarily engage in these behaviours. Tang et al. (1997)
251 suggest that those IBS patients with severe vomiting and high scores on the Bulimia subscale
252 (EDI) may have a characteristic in common with people with eating disorders, i.e. the desire
253 to lose weight.

254 Kauwautz et al.'s (2008) findings may shed light on the types of DE present in those with GI
255 disease. When looking at the weight loss mechanisms used by these participants, Kauwautz
256 et al. (2008) found that 58.1% used dieting behaviours, 12.9% used excessive exercising,
257 19.4% used vomiting and 3.2% used laxatives. This suggests that a range of DE behaviours
258 across the clinical spectrum were present, with a majority choosing to restrict their food
259 intake.

260 *Studies Concerning the Correlates and Co-morbidities of Disordered Eating*

261 Seven of the articles made reference to factors associated with higher DE scores. Of
262 particular interest is the reoccurrence of psychological distress, symptom severity and
263 dietary management alongside higher DE scores.

264 Out of the nine articles reviewed, six reported a relationship between DE and psychological
265 distress (Arigo et al., 2012; Addolorato et al., 1997; Fletcher et al., 2008; Guthrie et al., 1990;
266 Okami et al., 2011; Sainsbury et al., 2013). Eating disorder risk was associated with a
267 reduced quality of life, maladaptive coping mechanisms, depression and perceived stress
268 (Addolorato et al., 2011; Sainsbury et al., 2013). Furthermore, greater anxiety and
269 depressive symptomatology was found in those presenting with eating disturbances
270 (Addolorato et al., 1997; Guthrie et al., 1990; Okami et al., 2011). Addolorato et al. (1997)
271 explain that the reason for undernourishment in this patient group is not clear but suggest
272 that it might result from a fear of GI symptoms when consuming food. Fletcher et al.'s
273 (2008) findings suggest that this may be due to anxiety in unfamiliar settings, as participants
274 would restrict their intake in unfamiliar settings due to fears of cross-contamination. Anxiety
275 and depression both seem to be key factors in the development DE in those with GI
276 disorders.

277 Symptom severity was referred to across the papers (Arigo et al., 2012; Sainsbury et al.,
278 2013; Tang et al., 1997). It is not clear at what point symptom severity is most important,
279 with some reports suggesting that symptom severity prior to diagnosis may lead to the
280 development of DE patterns (Sainsbury, et al., 2013), and others suggesting it is the
281 frequency of symptoms during the course of the disease (Tang et al., 1997). More bulimic-
282 type thoughts were reported in those who experienced more extreme vomiting symptoms,
283 however, this does not necessarily translate into behaviour; these individuals acknowledged
284 the use of vomiting as a weight loss strategy but did not necessarily engage in this behaviour
285 (Tang et al., 1997). Arigo et al. (2012) reported that symptom severity was not associated

286 with DE practices. The role that symptom severity plays is not clear and it may only play a
287 role in the development of DE in a subset of those with GI disease.

288 Adherence to dietary regimens shows evidence of being related to DE practices, particularly
289 in those with CD. Arigo et al. (2012) found that management of the prescribed diet was
290 associated with a decreased range of psychological stresses, but was also linked to greater
291 DE concerns and behaviours. This indicates that those who monitor their food intake more
292 closely, to follow their prescribed dietary regime, may be at risk of DE. Karwatz et al.
293 (2008) reported that those with eating pathology also had significantly higher gluten
294 antibody markers, suggesting poorer dietary self-management. Those with eating pathology
295 also had a higher BMI and 85.7% reported the pathology as appearing after the onset of
296 their CD.

297 **Discussion**

298 This paper points towards some important factors that need to be considered in the
299 management of patients with GI disorders. There is an indication that individuals with GI
300 disorders may be more at risk of developing DE practices than the general population.

301 One aim of the review was to examine the prevalence of DE in those with GI disease. DE
302 patterns are present in a subset of those with GI disorders and the prevalence exceeds the
303 rates found in the general population. The prevalence rates identified in this review (5.3-
304 44.4%) are similar to those found in other dietary controlled chronic health conditions
305 (Markowitz et al., 2010; Shearer & Bryon, 2004). Quick, McWilliams & Byrd-Bredbenner
306 (2012) found that those with dietary-controlled health conditions were twice as likely to
307 have been diagnosed with an eating disorder compared to controls. The constant need to

308 monitor food intake may place these individuals, and those with GI disorders, at risk for DE
309 behaviours (Schlundt, Rowe, Pichert & Plant, 1999; Grilo, 2006). However, it is not clear
310 whether the GI disorder is contributing any additional risk factors towards the development
311 of DE, above and beyond that of other dietary controlled chronic health conditions.

312 The types of DE that were present in those with GI disease were also examined. The
313 majority of papers presented in the review indicated that a restrictive eating pathology was
314 most common. Although there was evidence for bulimic patterns of behaviour as well as
315 excessive exercising, food restriction was more frequently reported. It is not clear why
316 these behaviours are more common and if this finding will be replicated in larger samples.
317 However, it may be that those with GI disorders are more likely to fit the psychological
318 profile of someone with a restrictive eating disturbance. However, the majority of
319 investigations simply examined eating disorder risk. This assesses the presence of food
320 restriction, bingeing and purging behaviours. Therefore, it is difficult to get a clear picture of
321 what types of DE are most prevalent in those with GI disorders. In addition, an extensive
322 range of eating patterns such as emotional eating, over eating and nocturnal eating patterns
323 have not been examined. This should be addressed in future research because the ranges of
324 DE practices are associated with distinct psychological profiles (Cassin & von Ranson, 2005).
325 Moreover, the majority investigations did not assess the presence of subclinical eating
326 pathology. Future studies should also consider the role of subclinical eating symptoms in GI
327 disorders; due to their risk of malnutrition, any deviation from traditional eating patterns
328 may have a significant impact in this subset of the population.

329 Another aim of this review was to examine the correlates of DE in GI disease. Psychological
330 distress, symptom severity and dietary adherence were found to be associated with the

331 presence of DE patterns. Anxiety, depression and impaired quality of life were reported in
332 those with DE patterns across the majority of papers. This is not surprising because
333 psychological distress is frequently associated with altered eating patterns in those both
334 with (Colton, Olmsted, Daneman & Rodin, 2013) and without chronic disease (Patrick, Stahl
335 & Sundaram, 2011; Santos, Richards & Bleckley, 2007). However, the specific role that
336 psychological distress plays is not clear. Distress may be both a cause and a consequence of
337 DE behaviours. However, Arigo et al., (2012) suggested that anxiety might be playing a
338 unique role in those with GI disease. According to Arigo and colleagues the fear and anxiety
339 surrounding GI symptoms may lead to DE practices of a restrictive nature. Individuals with
340 GI disease may be so anxious and fearful of the GI symptoms that have been associated with
341 food consumption in their past, that their fear and anxiety results in an aversion to
342 unfamiliar food types and subsequent food restriction.

343 GI symptom severity may also play an important role in the development of DE
344 patterns. The role that GI symptoms play in the development of DE appears rather complex.
345 Some authors report that greater symptoms prior to diagnosis increases DE risk (Sainsbury
346 et al., 2013), whereas others report that greater symptoms throughout their diagnosis led to
347 greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et
348 al., 2008) and good dietary management (Arigo et al., 2012) have been associated with DE
349 patterns. It is possible that there are at least two pathways that lead to increased risk of DE
350 in patients with GI symptoms. On the one hand, individuals who do not follow their dietary
351 regimen experience GI symptoms throughout their diagnosis. These individuals may not be
352 concerned about their diet, and choose to consume their trigger foods for a variety of
353 reasons. This group could be using their trigger foods to promote weight loss. These

354 findings are in line with case studies of individuals with CD, IBS and IBD where deliberate
355 consumption of trigger foods has been reported in order to aid weight loss (Leffler et al.,
356 2007; Mallert & Murch, 1990). In those with good dietary management, their GI symptoms
357 may be playing a unique role in the development of DE patterns. Hypothetically, the
358 presence of GI symptoms may create a food aversion in these individuals, causing
359 alterations to their eating patterns (Garcia et al., 1955). These individuals may be extremely
360 anxious and concerned with the preparation and potential cross-contamination of their food
361 products. Concerns around cross-contamination and anxiety around unfamiliar foods is
362 frequently found across the GI disorders (Schneider & Fletcher, 2008; Sverker, Hensing, &
363 Hallet 2005). Although high concern around unknown food items may be advantageous in
364 some situations, this may also feed into the development of DE patterns. A hypothetical
365 framework based on these two pathways has been developed.

366 *A Hypothetical Framework*

367 Based upon the literature presented in the review, a conceptual model of DE patterns in GI
368 disorders has been developed (Figure 2). The model depicts the theoretical relationship
369 between a collection of GI disorders and DE patterns; however, it is likely that each GI
370 disorder will have a more specific relationship with eating behaviour but to develop specific
371 models more focussed research is required.

372 The model begins at diagnosis. When diagnosed with a chronic health condition, depending
373 on the individual's circumstances, some will adapt well and accept the condition but for
374 others denial may play a role (Alvani, Parvin, Seyed & Alvani, 2012). Coping with any form of
375 chronic illness creates both physical and psychological challenges (Turkel & Pao, 2007). This

376 can contribute to psychological distress, coping problems and a lack of compliance to a
377 medical regime (Seiffge-Krenke & Skaletz, 2006; Suris, Michaud & Viner, 2004).

378 Pathway one describes the potential development of DE patterns for those who have
379 adapted well to their condition. These individuals may have greater GI symptoms at
380 diagnosis; the implementation of their treatment and prescribed dietary regimens is
381 effective in resolving these GI symptoms. Sainsbury et al. (2013) found greater symptoms at
382 diagnosis to be important in the development of DE patterns. These individuals may be
383 anxious about experiencing these GI symptoms again. They may overestimate the negative
384 consequences of their condition and develop the belief that all foods have cross-
385 contamination potential. As a result, these individuals follow their dietary regimens
386 extremely well, like those described by Arigo et al. (2012). Due to their strict dietary self-
387 management, uncertainty surrounding the content of food may be intolerable for this
388 group. High concerns and anxiety around the preparation and cross-contamination of food
389 dominate their thoughts and behaviours. This may result in the consumption of a limited
390 range of foods or eating only in well-known environments. This is similar to the experiences
391 Fletcher et al. (2008) described in those with IBS and IBD. These individuals may restrict
392 their food intake during the day, in order to cope with their anxiety around cross-
393 contamination and food preparation issues and subsequently there is the potential for an
394 excessive amount of food to be consumed in the evening when in the home. These
395 individuals display the food restriction that was found throughout the papers and this is
396 associated with their anxiety surrounding GI symptoms that was described by Arigo et al.
397 (2012) and reported under nutrition in these groups.

398 Individuals in pathway two do not adapt well to their diagnosis and experience distress.
399 When starting their treatment and prescribed dietary regimens, these individuals may react
400 with fear when their weight is restored to a healthy level after diagnosis. This group may
401 believe that their dietary regimen is causing them to gain weight, which leads to
402 dysfunctional illness beliefs and behaviours regarding their dietary regimen. Poor dietary
403 management may follow and the consumption of trigger foods may be motivated by the
404 belief that this can aid with weight loss. These beliefs may lead to a lack of adherence to the
405 prescribed dietary regimen, continued GI symptoms and psychological distress (Lohiniemi,
406 Maki, Kaukinen, Laippala & Collin, 2000; Roth & Ohlsson, 2013). This explains the poor
407 dietary management and increased symptom severity throughout diagnosis, described by
408 Arigo et al. (2012), Tang et al. (1997), Fletcher et al. (2008) and Kawautz et al. (2008).
409 Individuals in pathway two may be at risk for a clinically significant eating disorder.

410

411

412

413 *Strengths and Limitations of Review*

414 The prevalence of GI disorders is increasing rapidly and this is expected to increase as
415 diagnostic measures improve (Lohi et al., 2007; Molodecky et al., 2012; West, Fleming, Tata,
416 Card & Crooks, 2014). We believe our review brings together an important area of research
417 for the first time. We outline gaps in the current literature and pose a number of important
418 research questions that will need answering in the future. This review also highlights several
419 limitations that need to be addressed in order to develop research into DE practices in GI

420 disease. The development of a model of DE in GI disease is of use clinically and provides a
421 guide for future research. However, there is a need to explore the underlying causes of DE
422 patterns in GI disease and explore the functions that these eating patterns may have for this
423 group. In addition, the studies described in the review failed to report long-term outcomes.
424 It is essential for future research to prioritise the long-term effects of DE in GI disease.

425 Only nine articles were included in this review, which highlights the need for research in this
426 population. Despite this limitation, these nine articles had strong quality scores and eight of
427 these articles suggested that DE was occurring in participants with GI disease, suggesting
428 that the findings are reliable. Due to improved diagnostic measures and better access to services,
429 GI diagnosis is increasing rapidly (Lohi et al., 2007; Molodecky et al., 2012; West, Fleming, Tata, Card
430 & Crooks, 2014; WGO, 2009). As more of the population is diagnosed with
431 GI disease, there becomes a need to explore and highlight the psychosocial and physical
432 consequences of GI disease. This includes DE patterns. An increased awareness of this phenomenon
433 should improve awareness amongst healthcare professionals and ultimately can lead to early
434 detection or prevention of the problem in those with GI disease.

435
436 Unfortunately, the results could not be combined in a meta-analysis due to the differing
437 methodologies, outcomes and populations. The development of the hypothetical model of
438 DE in GI disease provides a framework to guide future research. There is a need for studies
439 to document the levels of adherence and anxiety around food in those with GI disease. In
440 addition, the function that these eating patterns may have, should be addressed from the
441 patient perspective.

442 *Pathologizing Behaviours that Work?*

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

450 *Conclusions*

451 The evidence indicates that those with dietary-controlled GI disorders may be at increased
452 risk for DE practices. This is likely to interact with the presence of GI symptoms and
453 psychological distress. The limited research in this area is concerning as it impacts both the
454 physical and psychological well-being of this group. There is a need to fully examine the
455 prevalence of this phenomenon in the GI population, as well as the interaction between the
456 two disorders. These findings may help with plans to manage such cases effectively in order
457 to improve physical health and well-being.

458

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680 Appendices

681 Appendix A: Search Criteria

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702 *Figure 1. Overview of search strategy*

703 *Figure 2. Hypothetical framework between GI disorders and disordered eating*

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Accepted Manuscript

706 Table 1 A summary of studies included in the review

Author	GI Disorder	Experimental Group	Control Group	Exclusion Criteria	Study Type	Eating Behaviour Measure	Other Measures	Disordered Eating Prevalence	Disordered Eating Type	Correlations of Disordered Eating	Quality Score (0-1)
Arigo, Anskis & Smyth (2012) USA	Coeliac Disease	177 females over 18 years (M=39.2 years)	NA	Under 18 years, 23 removed due to insufficient data	Correlational design	Eating Disorders Examination (Fairburn & Beglin, 1994)	Dietary Compliance Scale (Casellas, Vivancos & Maladela da, 2009), Celiac Disease Symptom Questionnaire (Hauser et al., 2007), Short-Form Health Survey (Ware & Sherborne, 1992), Perceived Stress Scale (Cohen, Kamarck & Mermelstein, 1983), Centre for Disease Studies Depression Scale (Radloff, 1977)	22%	Restraint, Eating Concern, Shape Concern and Weight Concern	Illness symptoms, gluten-free diet compliance, depression	0.91
Karwautz, Wagner, Berger, Sinreich, Grylli & Huber (2008) Austria	Coeliac Disease	283 adolescents (10-20 years; M=14.8 years).	Adolescents with Type I Diabetes	NA	Two stage design: between participants and qualitative interviews	Eating Disorder Inventory (Rathner & Waldherr, 1997), Eating Disorder Examination Questionnaire (Hilbert et al., 2007), Eating Disorder Examination (Hilbert, Tuschen-	IgA Anti-Endomysial and IgA Transglutaminase antibodies	29.3%	4.8% lifetime history of eating disorder, 3.9% current eating disorder, 10.2% lifetime history of subclinical eating disorder, 10.7% current subclinical eating disorder.	Poor compliance with gluten-free diet	1

Caffier & Ohms, 2004)											
Addolorato, Capristo, Stefani & Gabbarini (1997) Italy	IBD	79 patients with IBD (M=35 years)	36 healthy controls (M=36 years)	Those receiving steroid therapy or having had previous surgery	Between subjects design	BMI, 7 day food diary	Physical Morbidity Index (Andrews, Barczack & Allan, 1987), STAI (Grillion, Ameli, Footh & Davis, 1993), Zung Self-Rating Depression scale (Zung, Richards & Short, 1965),	37.2% of Crohn's Disease, 44.4% of Ulcerative Colitis	Malnutrition	Anxiety, depression	1
Guthrie, Creed & Whorwell (1990) United Kingdom	IBS/IBD	152 female outpatients with IBS (M=39 years).	34 with IBD and 37 with peptic ulcer	NA	Between subjects design	Eating Attitudes Test (EAT; Garner, Olmstead, Bohr & Garfinkel, 1982)	Psychiatric Assessment Schedule (Dean, Surtees & Sashidharan, 1983)	5.3%	Preoccupation with desire to be thinner, food controlling life, engaging in dieting behaviour and too much time and consideration to food.	NA	0.77
Fletcher, Jamieson, Schneider & Harry (2008) Canada	IBS/IBD	8 females (18-23 years), 5 with IBS and 3 IBD	NA	NA	Qualitative interviews	14-day food diary	Background questionnaire, semi-structured interview	NA	NA	Lack of compliance with medical regimen	0.85
Sullivan, Blewett, Jenkins & Allison (1997) United Kingdom	IBS	48 patients with IBS, 31 with IBD	28 healthy controls	NA	Between subjects design	Eating Attitudes Test (Garner, 1982)	NA	NA	Dieting, bulimia, food preoccupation, oral control	NA	0.62
Tang, Toner, Stuckless, Dion, Kaplan & Ali (1997) Canada	IBS	43 female and 17 male IBS patients (M=36.8 years).	Predetermined normative sample: 271 healthy controls (M=20.3 years)	NA	Between subjects design	Eating Disorder Inventory (Garner & Olmstead, 1984)	Daily GI symptom diary (Neff & Blanchard, 1987)	NA	Bulimic thoughts	Female, vomiting symptoms	0.91

Okami et al., (2011) Japan	IBS	626 students, IBS symptoms aged 18-29 years	NA 1140 healthy controls without IBS symptoms	Over 30 years, previous diagnosis of IBD	Between subjects design	Non-Validated questionnaire	Rome II (Thompson et al., 1999), HADS (Hatta et al., 1998), METS (Ministry of Health, Japan, 2006)	NA	Irregular meals and meal skipping	Anxiety, depression	0.91	707
Sainsbury, Mullan & Sharpe (2013) Australia	Coeliac Disease	390 members of a coeliac society (M=44.2 years)	NA	NA	Correlational design	Eating Disorder Inventory (Garner, 2004)	WHO QoL measure (Murphy et al., 2000), Depression Anxiety Scale (Lovibond & Lovibond, 1995), the Coping Inventory for Stressful Situations, Coeliac Dietary Adherence Scale (Leffler et al., 2009), Perceived Behavioural Control Scale (Sainsbury & Mullan, 2011)	NA	Restraint, Eating Concern, Shape Concern and Weight Concern	QoL, severe GI symptoms at diagnosis, depression, anxiety, stress, emotion-focused coping	1	