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Disordered eating practices in gastrointestinal disorders

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4	Disordered Eating Practices in Gastrointestinal Disorders
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10 11	Key words: Eating disorder; disordered eating; gastrointestinal; coeliac disease; inflammatory bowel disease; irritable bowel syndrome
12	Disordered Eating Practices in Gastrointestinal Disorders
13	Highlights
14	Evidence of disordered eating patterns in GI disease was reviewed.
15	• The prevalence of disordered eating in gastrointestinal disease was 5.3-44.4%
16	 This was associated with distress, symptom severity and dietary
17	management.
18	A conceptual model of disordered eating in GI disease was developed.
19	

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22 Abstract

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

in dietary-controlled gastrointestinal conditions. Three key questions were

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

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

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

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

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

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

review.

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

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

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

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

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

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

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

1	3

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
80	of a medical regimen or to promote health, is associated with altered eating patterns (Johnson, Pratt, & Wardle, 2012; Herman & Polivy, 1980). The prescribed dietary
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81 82 83 84 85	(Johnson, Pratt, & Wardle, 2012; Herman & Polivy, 1980). The prescribed dietary restraint in IBS, IBD and CD may place these individuals at risk for abnormal eating patterns (Keller, 2008). Prescribed dietary regimens may result in the development of harmful thoughts and attitudes towards food and body weight, which may in turn, lead to inappropriate eating practices (Nicholas et al., 2007). This phenomenon has

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?

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.,

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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.
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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

was divided by a total possible sum (28-(number of NA's x 2)). This provided a total

quality score ranging between 0 and 1. Scores closer to 1 were suggestive of better

quality. Difference in ratings between the reviewers was minor and resolved through

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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.

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questionnaires.

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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 Five of the studies used control groups (Addolorato et al., 2012; Guthrie et al., 1990; Okami 184 et al., 2011; Sullivan et al., 1997; Tang et al. 1997). Information about the participant 185 characteristics was lacking in two of the papers (Okami et al., 2011; Sullivan et al., 1997). 186 Outcome Measure 187 DE patterns were assessed in all of the papers; however, only four of the studies provided 188 189 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 information concerning the correlates of DE. 191 192 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

measures of DE and body image were related to general psychosocial well-being

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Study Design

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

Synthesis of Results

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

Studies Concerning the Prevalence of Disordered Eating in GI Disorders

assessed using differing methods (Addolorato et al., 1997; Arigo et al., 2012; Guthrie et al., 1990; Karwautz et al., 2008). Across these four papers there were a total of 691 participants with GI disease. Of these, 23.43% displayed eating patterns that were suggestive of DE. Across these papers, DE patterns ranged between 5.3-44.4% in those with GI disease.

Prevalence rates for the Eating Disorders Examination (Fairburn & Beglin, 1994) ranged between 22-29.3% (Arigo et al., 2012; Karwautz at al., 2008). These scores are in excess of

Four of the articles reported the prevalence of patterns suggestive of DE, although this was

Micali, 2014). However, those papers using the EAT (Garner, Olmstead, Bohr & Garfinkel,

1982) reported lower prevalence rates (Guthrie et al., 1990; Sullivan et al., 1997). Only one

of the papers reported lower prevalence of DE in participants with GI disease than healthy

controls (Sullivan et al., 1997). Unfortunately, Sullivan et al. (1997) reported only the means

the scores reported for the general population (10%; Solmi, Hatch, Hotopf, Treasure &

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.
253254	to lose weight. Kauwautz et al.'s (2008) findings may shed light on the types of DE present in those with GI
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254 255 256 257 258 259	Kauwautz et al.'s (2008) findings may shed light on the types of DE present in those with GI disease. When looking at the weight loss mechanisms used by these participants, Kauwautz et al. (2008) found that 58.1% used dieting behaviours, 12.9% used excessive exercising, 19.4% used vomiting and 3.2% used laxatives. This suggests that a range of DE behaviours across the clinical spectrum were present, with a majority choosing to restrict their food intake. Studies Concerning the Correlates and Co-morbidities of Disordered Eating

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

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

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

297 Discussion

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

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

monitor food intake may place these individuals, and those with GI disorders, at risk for DE
behaviours (Schlundt, Rowe, Pichert & Plant, 1999; Grilo, 2006). However, it is not clear
whether the GI disorder is contributing any additional risk factors towards the development
of DE, above and beyond that of other dietary controlled chronic health conditions.
The types of DE that were present in those with GI disease were also examined. The
majority of papers presented in the review indicated that a restrictive eating pathology was
most common. Although there was evidence for bulimic patterns of behaviour as well as
excessive exercising, food restriction was more frequently reported. It is not clear why
these behaviours are more common and if this finding will be replicated in larger samples.
However, it may be that those with GI disorders are more likely to fit the psychological
profile of someone with a restrictive eating disturbance. However, the majority of
investigations simply examined eating disorder risk. This assesses the presence of food
restriction, bingeing and purging behaviours. Therefore, it is difficult to get a clear picture of
what types of DE are most prevalent in those with GI disorders. In addition, an extensive
range of eating patterns such as emotional eating, over eating and nocturnal eating patterns
have not been examined. This should be addressed in future research because the ranges of
DE practices are associated with distinct psychological profiles (Cassin & von Ranson, 2005).
Moreover, the majority investigations did not assess the presence of subclinical eating
pathology. Future studies should also consider the role of subclinical eating symptoms in GI
disorders; due to their risk of malnutrition, any deviation from traditional eating patterns
may have a significant impact in this subset of the population.
Another aim of this review was to examine the correlates of DE in GI disease. Psychological
distress, symptom severity and dietary adherence were found to be associated with the

presence of DE patterns. Anxiety, depression and impaired quality of life were reported in
those with DE patterns across the majority of papers. This is not surprising because
psychological distress is frequently associated with altered eating patterns in those both
with (Colton, Olmsted, Daneman & Rodin, 2013) and without chronic disease (Patrick, Stahl
& Sundaram, 2011; Santos, Richards & Bleckley, 2007). However, the specific role that
psychological distress plays is not clear. Distress may be both a cause and a consequence of
DE behaviours. However, Arigo et al., (2012) suggested that anxiety might be playing a
unique role in those with GI disease. According to Arigo and colleagues the fear and anxiety
surrounding GI symptoms may lead to DE practices of a restrictive nature. Individuals with
GI disease may be so anxious and fearful of the GI symptoms that have been associated with
food consumption in their past, that their fear and anxiety results in an aversion to
unfamiliar food types and subsequent food restriction.
GI symptom severity was may also play an important role in the development of DE
patterns. The role that GI symptoms play in the development of DE appears rather complex.
Some authors report that greater symptoms prior to diagnosis increases DE risk (Sainsbury
et al., 2013), whereas others report that greater symptoms throughout their diagnosis led to
et al., 2013), whereas others report that greater symptoms throughout their diagnosis led to greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et
greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et
greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et al., 2008) and good dietary management (Arigo et al., 2012) have been associated with DE
greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et al., 2008) and good dietary management (Arigo et al., 2012) have been associated with DE patterns. It is possible that there are at least two pathways that lead to increased risk of DE
greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et al., 2008) and good dietary management (Arigo et al., 2012) have been associated with DE patterns. It is possible that there are at least two pathways that lead to increased risk of DE in patients with GI symptoms. On the one hand, individuals who do not follow their dietary
greater DE risk (Tang et al., 1997). In addition, both poor (Fletcher et al., 2008; Karwautz et al., 2008) and good dietary management (Arigo et al., 2012) have been associated with DE patterns. It is possible that there are at least two pathways that lead to increased risk of DE in patients with GI symptoms. On the one hand, individuals who do not follow their dietary regimen experience GI symptoms throughout their diagnosis. These individuals may not be

findings are in line with case studies of individuals with CD, IBS and IBD where deliberate consumption of trigger foods has been reported in order to aid weight loss (Leffler et al., 2007; Mallert & Murch, 1990). In those with good dietary management, their GI symptoms may be playing a unique role in the development of DE patterns. Hypothetically, the presence of GI symptoms may create a food aversion in these individuals, causing alterations to their eating patterns (Garcia et al., 1955). These individuals may be extremely anxious and concerned with the preparation and potential cross-contamination of their food products. Concerns around cross-contamination and anxiety around unfamiliar foods is frequently found across the GI disorders (Schneider & Fletcher, 2008; Sverker, Hensing, & Hallet 2005). Although high concern around unknown food items may be advantageous in some situations, this may also feed into the development of DE patterns. A hypothetical framework based on these two pathways has been developed. A Hypothetical Framework Based upon the literature presented in the review, a conceptual model of DE patterns in GI disorders has been developed (Figure 2). The model depicts the theoretical relationship between a collection of GI disorders and DE patterns; however, it is likely that each GI disorder will have a more specific relationship with eating behaviour but to develop specific models more focussed research is required. The model begins at diagnosis. When diagnosed with a chronic health condition, depending on the individual's circumstances, some will adapt well and accept the condition but for others denial may play a role (Alvani, Parvin, Seyed & Alvani, 2012). Coping with any form of

chronic illness creates both physical and psychological challenges (Turkel & Pao, 2007). This

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can contribute to psychological distress, coping problems and a lack of compliance to a
medical regime (Seiffge-Krenke & Skaletz, 2006; Suris, Michaud & Viner, 2004).
Pathway one describes the potential development of DE patterns for those who have
adapted well to their condition. These individuals may have greater GI symptoms at
diagnosis; the implementation of their treatment and prescribed dietary regimens is
effective in resolving these GI symptoms. Sainsbury et al. (2013) found greater symptoms at
diagnosis to be important in the development of DE patterns. These individuals may be
anxious about experiencing these GI symptoms again. They may overestimate the negative
consequences of their condition and develop the belief that all foods have cross-
contamination potential. As a result, these individuals follow their dietary regimens
extremely well, like those described by Arigo et al. (2012). Due to their strict dietary self-
management, uncertainty surrounding the content of food may be intolerable for this
group. High concerns and anxiety around the preparation and cross-contamination of food
dominate their thoughts and behaviours. This may result in the consumption of a limited
range of foods or eating only in well-known environments. This is similar to the experiences
Fletcher et al. (2008) described in those with IBS and IBD. These individuals may restrict
their food intake during the day, in order to cope with their anxiety around cross-
contamination and food preparation issues and subsequently there is the potential for an
excessive amount of food to be consumed in the evening when in the home. These
individuals display the food restriction that was found throughout the papers and this is
associated with their anxiety surrounding GI symptoms that was described by Arigo et al.
(2012) and reported under nutrition in these groups.

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

Strengths and Limitations of Review

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

Pathologizing Behaviours that Work?

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Page 21 of 36

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

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

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Page 32 of 36

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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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706 Table 1 A summary of studies included in the review

Author	GI Disor der	Experim ental Group	Control Group	Exclusi on Criteria	Study Type	Eating Behaviou r Measure	Other Measure s	Disord ered Eating Prevale nce	Disordere d Eating Type	Correlat es of Disorde red Eating	Qual ity Scor e (0- 1)
Arigo, Anskis & Smyth (2012) USA	Coeli ac Disea se	177 females over 18 years (M=39.2 years)	NA	Under 18 years, 23 remov ed due to insuffic ient data	Correlati onal design	Eating Disorders Examinat ion (Fairburn & Beglin, 1994)	Dietary Complian ce Scale (Casellas, Vivancos & Maladela da, 2009), Celiac Disease Symptom Question naire (Hauser et al., 2007), Short- Form Health Survey (Ware & Sherborn e, 1992), Perceived Stress Scale (Cohen, Kamarck & Mermelst ein, 1983), Centre for Disease Studies Depressio n Scale (Radloff, 1977)	22%	Restraint, Eating Concern, Shape Concern and Weight Concern	Illness sympto ms, gluten- free diet complia nce, depress ion	0.91
Karwau tz, Wagner Berger, Sinreich , Grylli & Huber (2008) Austria	Coeli ac Disea se	283 adolesce nts (10- 20 years; M=14.8 years).	Adolescen ts with Type I Diabetes	NA	Two stage design: between participa nts and qualitati ve intervie ws	Eating Disorder Inventory (Rathner & Waldherr , 1997), Eating Disorder Examinat ion Question naire (Hilbert et al., 2007), Eating Disorder Examinat ion (Hilbert, Tuschen-	IgA Anti- Endomysi al and IgA Trangluti mase antibodie s	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 complia nce with gluten- free diet	1

						Caffier & Ohms, 2004)					
Addolor ato, Caprist o, Stefani ni & Gabarri ni (1997) Italy	IBD	79 patients with IBD (M=35 years)	36 healthy controls (M=36 years)	Those receiving steroid therap y or having had previous surgery	Between subjects design	BMI, 7 day food diary	Physical Morbidity Index (Andrews , Barczack & Allan, 1987), STAI (Grillion, Andeli, Footh & Davis, 1993), Zung Self- Rating Depressio n scale (Zung, Richards & Short, 1965),	37.2% of Crohn's Disease , 44.4% of Ulcerat ive Colitis	Malnutriti on	Anxiety, depress ion	1
Guthrie, Creed & Whorw ell (1990) United Kingdo m	IBS/IB D	152 female outpatie nts 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)	Psychiatri c Assessme nt Schedule (Dean, Surtees & Sashidhar an, 1983)	5.3%	Preoccup ation with desire to be thinner, food controllin g life, engaging in dieting behaviour and too much time and consideration to food.	NA	0.77
Fletcher , Jamieso n, Schneid er & Harry (2008) Canada	IBS/IB D	8 females (18-23 years), 5 with IBS and 3 IBD	NA	NA	Qualitati ve intervie ws	14-day food diary	Backgrou nd question naire, semi- structure d interview	NA	NA	Lack of complia nce with medical regimen	0.85
Sullivan , Blewett , Jenkins & Allison (1997) United Kingdo m	IBS	48 patients with IBS, 31 with IBD	28 healthy controls	NA	Between subjects design	Eating Attitudes Test(Gar ner, 1982)		NA	Dieting, bulimia, food preoccup ation, oral control	NA	0.62
Tang, Toner, Stuckln ess, Dion, Kaplan & Ali (1997) Canada	IBS	female and 17 male IBS patients (M=36.8 years).	Predeter mined normative sample: 271 healthy controls (M=20.3 years)	NA	Between subjects design	Eating Disorder Inventory (Garner & Olmstead , 1984)	Daily GI symptom diary (Neff & Blanchar d, 1987)	NA	Bulimic thoughts	Female, vomitin g sympto ms	0.91

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Okami et al., (2011) Japan	IBS	626 students, IBS sympto ms aged 18-29 years	NA 1140 healthy controls without IBS symptoms	Over 30 years, previou s diagno sis of IBD	Between subjects design	Non- Validated question naire	Rome II (Thomps on et al., 1999), HADS (Hatta et al., 1998), METS (Ministry of Health, Japan, 2006)	NA	Irregular meals and meal skipping	Anxiety, depress ion	0.91
Sainsbu ry, Mullan & Sharpe (2013) Australi a	Coeli ac Disea se	390 member s of a coeliac society (M=44.2 years)	NA	NA	Correlati onal design	Eating Disorder Inventory (Garner, 2004)	WHO QoL measure (Murphy et al., 2000), Depressio n Anxiety Scale (Lovibond & Lovibond, 1995), the Coping Inventory for Stressful Situations , Coeliac Dietary Adherenc e Scale (Leffler et al., 2009), Perceived Behaviou ral Control Scale (Sainsbur y & Mullan, 2011)	NA	Restraint, Eating Concern, Shape Concern and Weight Concern	QoL, severe GI sympto ms at diagnosi S, depress ion, anxiety, stress, emotio n- focusse d coping	1
					2	800					