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Disorders of gut–brain interaction common among outpatients with eating disorders including avoidant/restrictive food intake disorder

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Abstract

Objective: Little research exists on Rome IV disorders of gut–brain interaction (DGBI; formerly called functional gastrointestinal disorders) in outpatients with eating disorders (EDs). These data are particularly lacking for avoidant/restrictive food intake disorder (ARFID), which shares core features with DGBI. We aimed to identify the frequency and nature of DGBI symptoms among outpatients with EDs.

Method: Consecutively referred pediatric and adult patients diagnosed with an ED (n = 168, 71% female, ages 8–76 years) in our tertiary care ED program between March 2017 and July 2019 completed a modified Rome IV Questionnaire for DGBI and psychopathology measure battery.

Results: The majority (n = 122, 72%) of participants reported at least one bothersome gastrointestinal symptom. Sixty-six (39%) met criteria for a DBGI, most frequently functional dyspepsia—post-prandial distress syndrome subtype (31%). DGBI were surprisingly less frequent among patients with ARFID (30%) versus EDs that are associated with shape or weight concerns (45%; $X^2[1] = 3.61$, $p = .058$, Cramer's $V = .147$). Among those with ARFID, DGBI presence

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Jennifer J. Thomas and Kyle Staller were co-senior authors.

AUTHOR CONTRIBUTIONS

KS conceptualized the study and KS, JJT, and KTE oversaw the study. JJT, KTE, LB, KRB, and MJD all contributed to data collection. HBM conducted statistical analyses, wrote the manuscript, edited the manuscript, and approved the final draft submitted. KS and JJT provided substantial editing of the manuscript and supervised the project. All authors edited the manuscript and approved the final draft submitted.

CONFLICTS OF INTEREST

KS has received research support from Takeda and Gelesis, has served as a speaker for Shire, and has served as a consultant to Arena, Boston Pharmaceuticals, and Shire. BK has received research support from AstraZeneca, Takeda, Gelesis, Medtronic, Genzyme and has served as a consultant to Shire, Takeda, and Ironwood. KS and BK have no personal conflicts to declare. HBM, LB, and MJD have no personal or financial conflicts to declare. JJT, KTE, and KRB receive royalties from Cambridge University Press for the sale of their books on ARFID.

was associated with the fear of aversive consequences prototype and multiple comorbid prototype presence.

Discussion: We demonstrated notable overlap between DGBI and EDs, particularly post-prandial distress symptoms. Further research is needed to examine if gastrointestinal symptoms predict or are a result of greater ED pathology, including ARFID prototypes.

Keywords

avoidant/restrictive food intake disorder; disorder of gut–brain interaction; dyspepsia; feeding and eating disorders; functional constipation; functional dyspepsia—post-prandial distress; functional gastrointestinal disorder; irritable bowel syndrome

1 | INTRODUCTION

Patients with eating disorders (EDs) often report gastrointestinal (GI) symptoms, particularly functional symptoms characteristic of disorders of gut–brain interaction (DGBI; formerly called functional GI disorders; Drossman et al., 2016). Furthermore, GI symptoms may in fact contribute to ED symptom maintenance (Chami, Andersen, Crowell, Schuster, & Whitehead, 1995; Thomas & Eddy, 2019). However, the frequency and nature of DGBI is unclear among outpatients with the full-spectrum of EDs, including avoidant/restrictive food intake disorder (ARFID) and shape/weight-motivated EDs (i.e., anorexia nervosa, bulimia nervosa, binge-eating disorder, other specified feeding or eating disorder).

DGBI are GI conditions without underlying structural abnormality (i.e., no ulcers, cancer, inflammation). Rome IV holds the current widely accepted symptom-based classification scheme for DGBI across the GI tract (Drossman et al., 2016). Two of the most common DGBI include functional dyspepsia (Aziz et al., 2018) and irritable bowel syndrome (IBS; Palsson, Whitehead, Törnblom, Sperber, and Simren, 2020). Functional dyspepsia is characterized by chronic, post-prandial discomfort or upper abdominal pain. IBS is characterized by chronic abdominal pain associated with a change in frequency or form of stool.

Understanding the presence of DGBI among patients with EDs is important to identify potential treatment targets. Although some research suggests that GI symptoms improve with shape/weight-motivated ED treatment (Chami et al., 1995), DGBI symptoms may persist beyond ED treatment and could—in turn—put affected patients at risk for relapse. Previous data suggest that symptoms of DGBI persisted beyond ED treatment for up to 77% of patients (Boyd, Abraham, & Kellow, 2010), and patients who continue to experience DGBI symptoms may then later present for gastroenterology consultation. Thus, understanding the frequency and nature of DGBI in the ED population could inform case conceptualization and potential adjunctive treatment targets to integrate into ED treatment.

ED pathology is common in DGBI samples: between 13 and 23% for shape/weight-motivated ED symptoms (Murray et al., 2020; Murray, Jehangir, Silvernale, Kuo, & Parkman, 2020; Zia, Riddle, DeCou, McCann, & Heitkemper, under review) and between 24 and 43% for ARFID (Murray, Bailey, et al., 2020; Murray, Jehangir, et al., 2020; Zia et

al., under review). However, research on the frequency of DGBI among ED groups has been limited to inpatient samples (e.g., Boyd, Abraham, and Kellow, 2005), one small outpatient sample (e.g., Santonicola et al., 2012), and samples solely focused on IBS (e.g., DeJong, Perkins, Grover, and Schmidt, 2011). To date, there is limited data on the frequency of DGBI symptoms across the full spectrum of both EDs and DGBI symptom classification (Rome IV; Drossman et al., 2016).

We examined the frequency and nature of DGBI symptoms in pediatric and adult outpatients with the full-spectrum of EDs presenting for treatment evaluation. Given that previous research has shown a relatively higher frequency of ARFID compared to other shape/weight-motivated EDs among individuals with DGBI, we hypothesized that DGBI would be significantly more frequent among patients with ARFID compared to patients with shape/weight EDs. We also explored associations between DGBI presence and clinical characteristics including non-ED psychopathology.

2 | METHODS

2.1 | Participants and procedure

Participants included 186 consecutively referred patients (ages 8–76 years; sex = 73.1% female, 25.8% male, 1.1% other) seeking ED evaluation at a tertiary care ED program between March 2017 and July 2019. Of these patients, 168 were diagnosed with an ED and included in this study. Average age (SD) was 24.5 ± 13.0 years. The Massachusetts General Hospital Institutional Review Board approved the study. Patients completed self-report surveys, and self-reported demographics and height/weight. Evaluating psychology and psychiatry providers conferred clinical ED diagnoses (including ARFID prototypes—sensory sensitivity, fear of aversive consequences, lack of interest/low appetite).

2.2 | GI symptoms

Patients completed a modified *Rome IV Questionnaire for Functional GI Disorders* that included 12 items that mapped on to DGBI criteria for functional dyspepsia (post-prandial distress syndrome and epigastric pain syndrome), IBS (constipation-predominant, diarrhea-predominant, mixed, unspecified), functional constipation, functional diarrhea, functional abdominal bloating/distension, and belching disorders. By Rome IV, DGBI diagnoses are made based off of self-report questionnaire. DGBI presence is confirmed with the exclusion of structural abnormalities; although we were not able to confirm absence of structural abnormalities, most patients evaluated at our center had already been evaluated by a medical professional prior to ED consult.

2.3 | ED symptoms

The *Eating Disorder Examination Questionnaire (EDE-Q)* includes 28 items modeled after the Eating Disorder Examination with a Global score and four subscales (Restraint, Eating Concern, Weight Concern, Shape Concern). Higher scores indicate greater symptom severity (Fairburn & Beglin, 2008). Cronbach alphas was in the current sample were as follows: Global score = .969, Restraint subscale = .889, Eating Concern subscale = .813, Shape Concern subscale = .949, Weight Concern subscale = .896.

The *Food Neophobia Scale* includes 10 items on a 7-point Likert scale, with higher scores representing greater reluctance to try new foods (Pliner & Hobden, 1992). Cronbach alpha was .951 in the current sample.

The *Clinical Impairment Assessment (CIA)* includes 16 items on a 4-point Likert scale, with higher scores representing greater eating-related psychosocial impairment (Bohn et al., 2008). Cronbach alpha was .951 in the current sample.

2.4 | Other psychopathology

The *Center for Epidemiological Studies Depression Questionnaire* includes 20 items on a 4-point Likert scale, with higher scores representing greater depression symptoms over the preceding week (Eaton, Smith, Ybarra, Muntaner, & Tien, 2004). Cronbach alpha was .936 in the current sample.

The *State–Trait Anxiety Inventory—Trait Anxiety* scale includes 20 items on a 4-point Likert scale, with higher scores representing greater trait anxiety symptoms (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Cronbach alpha was .945 in the current sample.

2.5 | Statistical analysis

We summarized means and standard deviations for continuous variables and calculated proportions for categorical variables. We calculated the frequencies of bothersome GI symptoms and DGBI. To explore the data univariately, we compared those with DGBI versus those without on age, sex (male/female), BMI, ED diagnosis frequency, and each psychopathology measure. We used Chi-square tests for categorical variables. We used Kruskal–Wallis H tests for continuous variables, as log-transformation failed to improve significant skew present in all variables. We calculated Hedge's g for continuous and Cramer's V for categorical comparisons as measures of effect size. To identify psychological factors associated with likelihood of meeting DGBI criteria, we performed logistic regression with covariates selected *a priori* based on clinical relevance and reduced in number based on univariate screen. Covariates included in the model included biological sex (male/female) and age. We also reported frequencies of ARFID clinical characteristics between DGBI and no-DGBI groups.

3 | RESULTS

We first examined the frequency and nature of GI symptoms and DGBI specifically. One-hundred twenty-two patients (72%) reported at least one bothersome GI symptom (Table 1). Criteria for at least one DBGI were present in 66 patients (39%), with functional dyspepsia—post-prandial distress syndrome subtype being the most frequent (31%), followed by IBS (10%) and functional constipation (7%; Table 1). Those with DGBI were significantly older (mean difference = 5.4 years, medium effect size—Hedge's $g = .422$) and more likely to be female (81% versus 65%; small effect size—Cramer's $V = .173$) versus those without, but had similar mean BMI (Table 2). In addition, the shape/weight-motivated ED group was significantly older (mean difference = 10.9 years; large effect size—Hedge's $g = .929$) and were more likely to be female (16% versus 46%; medium effect size—Cramer's $V = .331$) compared to the ARFID group.

Contrary to our hypothesis, although there were differences in proportion of DGBI by ED type, these differences were not significant—DGBI were less frequent among patients with ARFID (30%) compared to patients with shape/weight EDs (45%; $X^2[1] = 3.61$, $p = .058$, small effect size—Cramer's $V = .147$), excluding rumination ($n = 1$) and pica ($n = 1$). Among patients with ARFID, the DGBI group had a higher frequency of the fear of aversive consequences prototype (50% vs. 11%) and the lack of interest/low appetite prototype (60% vs. 44%), but lower frequency of the sensory sensitivity prototype (55% vs. 85%). See Table 2 for frequencies of single prototypes and multiple comorbid prototypes, and ARFID medical/psychosocial impairment criteria. Notably, the sensory sensitivity prototype alone was infrequent in the DGBI group ($n = 2$; 10%), but the most frequent presentation in the no-DGBI group ($n = 23$; 50%). For ARFID criteria frequencies, weight loss/failure to gain/grow and psychosocial interference were the most frequent criteria met in both DGBI and no-DGBI groups.

We then examined the association between DGBI presence and self-report measures of ED-related and general psychopathology. Although those with versus without DGBI had significantly greater severity (with medium to large effect sizes) of shape/weight ED symptoms, eating-related quality of life difficulties, depression, and trait anxiety on univariate screen, these differences did not remain on multivariate analysis when controlling for biological sex (male/female) and age (ORs = 0.89–1.05, $p = .056$–$.880$, 95% CIs = 0.64–1.24). The model provided a good fit ($X^2[6] = 24.7$, $p < .001$; Hosmer-Lemeshow $X^2[8] = 11.3$, $p = .183$]. Food neophobia did not differ by DGBI status, so was not included in the multivariate model.

4 | DISCUSSION

Among outpatients presenting for ED treatment evaluation, we found that bothersome GI symptoms were common (72%) and DGBI (particularly for functional dyspepsia—post-prandial distress syndrome subtype) were also relatively frequent (39%). DGBI presence was associated with older age and female sex, but not BMI or psychopathology severity. Contrary to our hypothesis, there was not a higher rate of DGBI among patients with ARFID compared to shape/weight EDs. However, those with DGBI more frequently had ARFID fear of aversive consequences prototype and presence of multiple comorbid prototypes than those without DGBI, possibly indicating that fear and anxiety around GI symptoms is a mechanism present in DGBI and ED comorbidity.

The types of DGBI present among our sample could suggest specific treatment targets. DGBI are maintained by biopsychosocial processes, including visceral hypersensitivity (i.e., heightened sensitivity to normal GI tract sensations), motility disturbances (i.e., abnormal movement through the digestive tract), and psychological factors including negative thinking patterns and behavioral avoidance (Ljótsson et al., 2013). In our sample, functional dyspepsia was the most common DGBI, occurring at a rate roughly 3× that of the general population, suggesting a concentration of this pathophysiology in this ED sample (Aziz et al., 2018). Among this enriched group, post-prandial distress syndrome was the most common DGBI, mirroring findings that patients with EDs have dysregulated satiety

signaling (e.g., van Dyck et al., 2020), and aligning with treatment recommendations to use regular eating intervention to normalize signaling (e.g., Thomas & Eddy, 2019).

Depending on the DGBI, additional treatment targets in the context of EDs may be useful. For example, patients with DGBI characterized by lower GI symptoms (e.g., IBS) may experience abdominal symptoms (e.g., bloating, distension) that they interpret to indicate weight gain, perpetuating ED behavior (e.g., fasting, laxative use) that in-turn actually keeps their DGBI symptoms going (e.g., by reinforcing visceral hypersensitivity and hypervigilance around abdominal symptoms). Anxiety around and difficulty tolerating GI symptoms is frequently targeted in behavioral exposure for ARFID (Thomas, Wons, & Eddy, 2018), but is often not a *direct* target for other shape/weight-motivated EDs. Presence of DGBI symptoms could indicate behavioral exposure targets related to DGBI symptoms (Ljótsson et al., 2013). In addition, common treatments for DGBIs include neuromodulators (i.e., tricyclic antidepressants) to target visceral hypersensitivity, antibiotics and probiotics to alter the gut microbiome, and motility agents to speed up or slow down altered gut motility; such pharmacologic approaches could be used to supplement behavioral treatment in targeting brain–gut dysregulation in EDs.

The high frequency of bothersome GI symptoms aligns with previous reports showing frequent GI complaints in those with EDs, but our study expands on previous work by demonstrating a high prevalence of chronic, formally defined Rome IV DGBI. However, there are several limitations that should be considered. First, it is possible that the frequency of DGBI among our tertiary outpatient population is either higher or lower than among the wider population of individuals with EDs. For example, the frequency of DGBI may be lower than the true frequency of DGBI symptoms—the new Rome IV criteria for IBS require the presence of abdominal *pain* (Drossman et al., 2016), but some patients with IBS symptoms may only experience discomfort (Palsson et al., 2020). Second, the shape/weight ED group was both older and more frequently female than the ARFID group, which could have affected the proportion of DGBI between groups (e.g., DGBI may be present among more adults with EDs than children/adolescents). Third, our modified Rome IV questionnaire did not include all DGBI, including chronic nausea and vomiting disorders (e.g., cyclic vomiting syndrome). Although these DGBI are less common than the DGBI evaluated in the current study, they warrant further study. Finally, we did not capture previous medical evaluations that would allow us to definitely rule out gastrointestinal structural or organic abnormalities.

This cross-sectional study adds to the growing literature on the overlap between DGBI and EDs, including ARFID. Further research is needed to understand if screening for DGBI and targeting brain–gut dysregulation could improve treatment outcomes for some patients.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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TABLE 1

Frequency of bothersome GI symptoms and disorders of gut–brain interaction in patients with eating disorders (*N* = 168)

GI symptom	***N* (%)**
Post-prandial fullness[a]	79 (47%)
Early satiety	66 (39%)
Constipation	66 (39%)
Bloating ≥1 day/week[a]	62 (37%)
Upper abdominal pain[a]	46 (27%)
Nausea ≥1 day/week[a]	35 (21%)
< 3 Bowel movements/week	30 (18%)
Severe abdominal pain ≥1 day/week	30 (18%)
Loose stool ≥25% Bowel movements	28 (17%)
Upper abdominal burning[a]	19 (11%)
Belching ≥3 days/week[a]	14 (8%)
Disorders of gut–brain interaction (DGBI)[b]	***N* (%)**
Functional dyspepsia [c]	53 (32%)
Post-prandial distress syndrome	52 (31%)
Epigastric pain syndrome	8 (5%)
Irritable bowel syndrome [d]	16 (10%)
Constipation predominant	0 (0%)
Diarrhea predominant	3 (2%)
Mixed	8 (5%)
Unspecified	5 (3%)
Functional constipation	11 (7%)
Functional diarrhea	8 (5%)
Functional abdominal bloating/distension	4 (2%)

[a] Each symptom was qualified with “bothersome”.

[b] *n* = 7 met criteria for two DGBI, *n* = 1 met criteria for three DGBI.

[c] Functional dyspepsia is subtyped into postprandial distress syndrome (post-prandial bothersome fullness *or* early satiety at least 2–3 days/week for 3 months) and epigastric pain syndrome (bothersome pain or burning in the upper abdomen at least 1 day/week for 3 months). Seven participants met criteria for both post-prandial distress syndrome and epigastric pain syndrome.

[d] Irritable bowel syndrome (IBS) is characterized by recurrent abdominal pain (at least 1 day/week for 3 months) related to a change in bowel frequency or consistency and subtyped into four presentations by predominant bowel consistency—constipation, diarrhea, mixed (alternating constipation/diarrhea), and unspecified.

TABLE 2

Characteristics of patients presenting for eating disorder evaluation by DGBI status

Characteristics among all presenting patients (*N* = 168)					
	DGBI (*n* = 67)	**No DGBI (*n* = 101)**	***H or Chi*** [a]	***p*-value**	**Hedge's *g* or Cramer's *V***
Age, *M* (*SD*)	27.8 (12.9)	22.4 (12.6)	11.52	.001	.422
Sex, *N* (%) [b]			4.97	.026	.173
Female	54 (81%)	66 (65%)			
Male	12 (18%)	34 (34%)			
Other	1 (2%)	1 (1%)			
BMI-kg/m^2, *M* (*SD*)	23.1 (6.9)	22.4 (7.4)	0.86	.353	.097
Race/Ethnicity [c]					
American Indian or Alaska Native	0 (0%)	2 (2%)			
Asian	4 (6%)	6 (6%)			
Black or African American	2 (3%)	2 (2%)			
Native Hawaiian or Other Pacific Islander	0 (0%)	0 (0%)			
White	64 (96%)	95 (94%)			
Hispanic or Latino	3 (5%)	8 (8%)			
ED diagnosis, *N* (%) [d,e]					
Anorexia nervosa	11 (16%)	15 (15%)	0.08	.783	.021
Bulimia nervosa	11 (16%)	9 (9%)	2.16	.141	.114
Binge-eating disorder	11 (16%)	10 (10%)	1.56	.211	.096
Avoidant/restrictive food intake disorder	20 (30%)	45 (45%)	3.61	.058	.148
Other Specified Feeding or Eating Disorder[d]	12 (18%)	18 (18%)	0.00	.988	.001
Unspecified Feeding or Eating Disorder	1 (2%)	2 (2%)			
Rumination Disorder	1 (1%)	0 (0%)			
Pica	0 (0%)	1 (1%)			
EDE-Q Global (range 0–6), ***M* (*SD*)**	2.8 (1.8)	1.9 (1.8)	8.17	.004	.498
Restraint subscale	2.2 (1.9)	1.4 (1.8)	7.65	.006	.433
Eating Concern subscale	2.4 (1.7)	1.8 (1.7)	5.06	.024	.351
Shape Concern subscale	3.3 (2.0)	2.4 (2.2)	8.38	.004	.422

Characteristics among all presenting patients (*N* = 168)					
	DGBI (*n* = 67)	**No DGBI (*n* = 101)**	***H or Chi*** [a]	***p*-value**	**Hedge's *g* or Cramer's *V***
Weight Concern subscale	3.0 (2.1)	2.2 (1.9)	7.22	.007	.402
ED behavior frequency, *N* (%)					
Self-induced vomiting	10 (15%)	15 (15%)	0.00	.989	.001
Laxatives/diuretics	8 (12%)	6 (6%)	1.90	.168	.016
Binge eating	30 (45%)	45 (45%)	0.00	.977	.002
Compulsive exercise	18 (27%)	37 (37%)	1.75	.186	.102
Food Neophobia Scale (range = 10–70) ***M* (*SD*)**	43.0 (18.1)	46.1 (18.3)	0.92	.339	−.169
Clinical Impairment Assessment (range = 0–48) ***M* (SD)**	26.5 (13.2)	19.2 (12.7)	12.11	.001	.563
Center for Epidemiological Studies Depression Questionnaire (range = 0–60) ***M* (SD)**	25.7 (13.5)	16.1 (11.9)	18.97	<.001	.761
State-Trait Anxiety Inventory (range = 20–80) ***M* (SD)**	54.7 (12.4)	47.9 (12.3)	10.85	.001	.549
Characteristics among patients diagnosed with ARFID (*n* = 65)					
		DGBI (*n* = 20)		**No DGBI (*n* = 45)** [a]	
ARFID prototypes, *N* (%)					
Sensory sensitivity alone		2 (10%)		23 (50%)	
Fear of aversive consequences alone		4 (20%)		0 (0%)	
Lack of interest/low appetite alone		3 (15%)		3 (7%)	
Sensory sensitivity + fear of aversive consequences		2 (10%)		2 (5%)	
Sensory sensitivity + lack of interest/low appetite		5 (25%)		14 (31%)	
Fear of aversive consequences + lack of interest/low appetite		2 (10%)		3 (7%)	
Sensory sensitivity + fear of aversive consequences + lack of interest/low appetite		2 (10%)		0 (0.0%)	
ARFID criteria met, *N* (%)					
Weight loss or failure to gain/grow		12 (60%)		15 (33%)	
Nutritional deficiency		3 (15%)		8 (17%)	
Dependence on enteral feeding/oral supplements		2 (10%)		11 (24%)	
Psychosocial interference		12 (60%)		36 (78%)	

Abbreviations: ARFID, avoidant/restrictive food intake disorder; BMI, body mass index; DGBI, disorders of gut–brain interaction; M, mean.

[a] Comparisons conducted for exploratory purposes only. Independent sample Kruskal–Wallis *H* tests (continuous) and Chi-square tests (categorical). Hedge's *g* calculated for continuous and Cramer's *V* calculated for categorical comparisons as measures of effect size.

[b] Comparison on male versus female sex.

[c] Percentages do not add up to 100% because some participants reported more than one racial category.

[d] Statistical comparisons conducted with exclusion of participants with pica ($n = 1$) and rumination ($n = 1$).

[e] DGBI group included atypical anorexia nervosa ($n = 8$) and binge-eating disorder of limited frequency and/or duration ($n = 3$). No DGBI group included atypical anorexia nervosa ($n = 8$), bulimia nervosa of limited frequency and/or duration ($n = 3$), binge eating disorder of limited frequency and/or duration ($n = 3$), purging disorder ($n = 2$), other ($n = 4$).