DOI: 10.1002/erv.2847

REVIEW

Irritable bowel syndrome and functional dyspepsia in patients with eating disorders - a systematic review

Vivien Hanel¹ | Martha A. Schalla¹ | Andreas Stengel^{1,2}

¹Charité Center for Internal Medicine and Dermatology, Department for Psychosomatic Medicine, Charité-Universitätsmedizin Berlin, Corporate Member of Freie Universität Berlin, Humboldt-Universität zu Berlin, and Berlin Institute of Health, Berlin, Germany

²Department of Psychosomatic Medicine and Psychotherapy, University Hospital Tübingen, Tübingen, Germany

Correspondence

Andreas Stengel, Department of Psychosomatic Medicine and Psychotherapy, University Hospital Tübingen, Osianderstr. 5, Tübingen 72076, Germany. Email: andreas.stengel@med.unituebingen.de

Funding information

German Research Foundation, Grant/ Award Number: STE 1765/3-2; Charité University Funding, Grant/Award Number: UFF 89/441-176, to A.S.

Abstract

Objectives: The prevalence of eating disorders is rising worldwide. The low body weight in anorexia nervosa as well as the increase in body mass index due to binge eating disorder are contributing to a strikingly high morbidity and mortality. In a similar pattern, the prevalence and burden of the disease of functional gastrointestinal disorders such as functional dyspepsia and irritable bowel syndrome is increasing. As gastrointestinal complaints are commonly reported by patients with eating disorders, the question arose whether there is a relationship between eating disorders and functional gastrointestinal disorders.

Methods: To address the need to better understand the interplay between eating disorders and functional gastrointestinal disorders as well as factors that might influence this connection, the data bases Medline, Web of Science and Embase were systematically searched.

Results: After removal of duplicates the search yielded 388 studies which were screened manually. As a result, 36 publications were selected for inclusion in this systematic review.

Conclusion: The occurrence of functional gastrointestinal disorders like irritable bowel syndrome and functional dyspepsia in patients with eating disorders is considerably high and often associated with psychological, hormonal and functional alterations. In the future, further research addressing the underlying mechanisms accounting for this relationship is required.

KEYWORDS

anorexia nervosa, binge eating disorder, bulimia nervosa, dyspepsia, irritable bowel syndrome

Abbreviations: AN, anorexia nervosa; BED, binge eating disorder; BMI, body mass index; BN, bulimia nervosa; CCK, cholecystokinin; DSM, Diagnostic and Statistical Manual of Mental Disorders; ED(s), eating disorder(s); EDNOS, eating disorders not otherwise specified; EPS, epigastric pain syndrome; FD, functional dyspepsia; FGID(s), functional gastrointestinal disorder(s); GI, gastrointestinal; HC, healthy controls; IBS, irritable bowel syndrome; PDS, postprandial distress syndrome; QoL, quality of life.

Vivien Hanel and Martha Anna Schalla contributed equally to this work.

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

© 2021 The Authors. European Eating Disorders Review published by Eating Disorders Association and John Wiley & Sons Ltd.

- Functional gastrointestinal disorders (FGIDs) like irritable bowel syndrome and functional dyspepsia commonly occur in patients with eating disorders (EDs) and often improve with ED treatment
- Especially disordered eating behaviour and psychological disturbances often affect the relationship of FGIDs/FGID symptoms and EDs
- The majority of patients with EDs developed FGIDs/FGID symptoms after the onset of the ED

1 | INTRODUCTION

The prevalence of eating disorders (EDs), namely anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED) and eating disorders not otherwise specified (EDNOS) and Other Specified Feeding and Eating Disorders is rising worldwide from a mean point prevalence of 3.5% in the early 2000s to 7.8% in the period of 2013-2018, leading to a mean lifetime prevalence for EDs of 8.4% for women and 2.2% for men (Galmiche et al., 2019). Due to ED-related changes in body weight impairing physiological organ-related functioning, the rates for morbidity and mortality are strikingly high, and most pronounced in AN (Fichter & Quadflieg, 2016; Smink et al., 2012). Thus, there is a need for a better understanding of eating disorders and the possible underlying mechanism responsible for their development, chronicity and deterioration. Patients with EDs often report gastrointestinal (GI) complaints (Sato & Fukudo, 2015) associated with lower quality of life (QoL) (Enck et al., 2016; Winkler et al., 2014) but also higher healthcare costs, suggesting that GI symptoms could adversely impact the course of the diseases. The burden of functional gastrointestinal disorders (FGIDs), also referred to as disorders of gut-brain interaction since the introduction of the Rome-IV criteria (Tack & Drossman, 2017), is enormous, with worldwide prevalence rates from 20% to more than 40% for at least one FGID (Sperber et al., 2020). Functional dyspepsia (FD) and irritable bowel syndrome (IBS) are among the most commonly occurring FGIDs (Oshima & Miwa, 2015) with distinct diagnostic features as summarized in Table 1.

As early as in the 1990s research observed an increased prevalence of GI symptoms in individuals with EDs (Crowell et al., 1994) as well as symptoms of EDs in those affected by an FGID (Guthrie et al., 1990). Thus, the question arose whether there is a direct relationship between EDs and FGIDs like FD and IBS. To investigate this question, we performed a systematic review of the literature to evaluate how FGID symptoms and FGIDs, focusing on IBS and FD, interface with ED

symptoms. Our specific aims were (1) to ascertain the prevalence of FGIDs/FGID symptoms in patients with EDs and vice versa, (2) to examine the effect of ED treatment on FGIDs/FGID symptoms and (3) to evaluate the development of FGIDs/FGID symptoms in patients with EDs and vice versa and which factors (e.g., psychological, hormonal, behavioural, functional) affect this development.

In accordance with the PICOS scheme, we focused on human studies of any type (study design) investigating patients with EDs (population) with symptoms or a diagnosis of IBS/FD (intervention), where applicable in comparison to patients with other EDs or healthy controls (HC, comparison) regarding prevalence, magnitude and associations of EDs and FGIDs like IBS and FD (outcome).

2 | METHODS

For the systematic data search the PRISMA Guidelines were applied (Figure 1). First, the data bases Pubmed-Medline, Web of Science and EMBASE were searched for English literature released from the earliest day of publication to the day the search was performed (21st of April 2020). The following keywords: "anorexia nervosa", "bulimia nervosa", "binge eating disorder" as well as "eating disorders not otherwise specified" OR "EDNOS" were each coupled with the following terms: "dyspepsia" OR "irritable bowel syndrome" OR "somatoform disorder (s)" OR "medically unexplained symptom(s)" OR "functional distress" OR "bodily distress".

After removal of duplicates, two independent researchers (VH, MAS) screened the titles and abstracts manually for eligibility regarding topic and article type. The inclusion criteria contained English original articles including case reports and human studies that investigated patients with an ED (AN, BN, BED, EDNOS) and co-occurring FGID symptoms/FGIDs (IBS, FD) or vice versa. Hence, review articles, metaanalyses, editorials, animal studies, articles written

TABLE 1	Comparison of diagnostic criteria for eating disorders and functional gastrointestinal c	lisorders	694
	Irritable bowel syndrome	Functional dyspepsia	
Diagnostic Criteria	 Manning criteria: Abdominal distension Looser stools at pain onset More frequent stools at pain onset More frequent stools at pain onset Alleviation of pain with defecation Feeling of incomplete emptying of the bowel Passing of mucus Rome I criteria: ≥3 months (ongoing or recurring) abdominal pain or discomfort Pain/discomfort is alleviated with defecation and/or associated with a change in stool frequency and/or stool consistency Additionally two of the following criteria on at least 25% of days/defaecations: Altered stool frequency, altered stool form, altered defecation (straining or urgency, feeling of incomplete evacuation), passage of mucus, bloating or feeling of abdominal distension Rome II criteria: 	Rome I criteria: - ≥3 months of pain or discomfort in the centre of the upper abdomen - No evidence of organic disease that could explain the symptoms - Subgroups: Ulcer-like dyspepsia, dysmotility-like dyspepsia, unspecified dyspepsia Rome II criteria:	
	 Abdominal pain or discomfort ≥12 weeks (not necessarily consecutive) in the last 12 months Pain/discomfort fulfils two of the following criteria: Alleviation with defecation and/ or onset is associated with a change in stool frequency and/or stool form (appearance) 	 Abdominal pain or discomfort in the centre of the upper abdomen ≥12 weeks (not necessarily consecutive) in the last 12 months No evidence of organic disease that could explain the symptoms No evidence that pain/discomfort fulfils criteria for IBS (i.e., alleviation with defecation, onset associated with change of stool frequency and/or form) Subgroups (according to the predominant symptom): Ulcer-like dyspepsia, dysmotility-like dyspepsia, unspecified dyspepsia 	
	 Rome III criteria: Relapsing abdominal pain or discomfort ≥3 days/month in the last 3 months Pain/discomfort fulfils ≥2 of the following criteria: Alleviation with defecation, onset associated with a change in stool frequency, onset associated with a change in stool form (appearance) Onset of symptoms ≥6 months prior to diagnosis and criteria met for the last 3 months Introduction of IRS-enhymmet IRS.C (constituation) IRS.D (diarrhoca) IRS.M (mixed) 	 Rome III criteria: ≥1 of the following symptoms: Bothersome postprandial fullness, early satiation, epigastric pain, epigastric burning No evidence of a structural disease that could explain the symptoms Onset of symptoms ≥6 months prior to diagnosis and criteria met for the last 3 months Subtypes: Postprandial distress syndrome (PDS), epigastric pain syndrome (EPS) 	
	 IBS-U (unsubtyped) IBS-U (unsubtyped) Rome IV criteria: Relapsing abdominal pain ≥1 day/week in the last 3 months Relapsing abdominal pain ≥1 day/week in the last 3 months Pain fulfils ≥2 of the following criteria: Relation to defecation, association with a change in stool frequency, association with a change in stool form (appearance) Onset of symptoms ≥6 months prior to diagnosis and criteria met for the last 3 months 	 Rome IV criteria: ≥1 of the following symptoms: Bothersome postprandial fullness, bothersome early satiety, bothersome epigastric pain, bothersome epigastric burning No evidence of a structural disease that could explain the symptoms Onset of symptoms ≥6 months prior to diagnosis and criteria met for the last 3 months 	
	 IBS-subtypes now classified according to the predominant subtype Further common symptoms: Abdominal bloating, abdominal distension 	- Subtypes: PDS, EPS	HANEI

	Irritable bowel syndrome		Functional dyspepsia	
Reference	Manning criteria (Manning et al.	, 1978)	Rome I (Drossman, 1995)	
	Rome I & II (Hillilä & Färkkilä, Rome III & IV (Grad & Dumitras	2004) scu, 2020; Lacy & Patel, 2017)	Rome II (Talley et al., 1999) Rome III (Tack & Talley, 2013)	
			Rome IV (Stanghellini et al., 2016)	
	Anorexia nervosa	Bulimia nervosa	Binge-eating disorder	Eating disorders not otherwise specified
Diagnostic criteria	 Less than minimally normal or expected body weight due to restriction of energy intake Intense fear of gaining weight and/or repetitious behavioural patterns impeding reaching a normal body weight Body image distortion and inability to conceive the seriousness of the severe underweight 	 Recurring binge eating episodes associated with lack of control followed by inappropriate compensatory behaviours (self-induced vomiting, laxative abuse, fasting) to counteract weight gain Episodes occur ≥1x/week for at least 3 months Body image has excessive impact on self-evaluation Supportive: Normal/above normal weight 	 Recurring, distressing binge eating episodes eating episodes associated with lack of control not regularly followed by compensatory behaviours followed by compensatory behaviours during episodes: Very rapid food ingestion, eating until feeling uncomfortable, eating without sensation of hunger, eating alone due to embarrassment; feeling of self-disgust, depression or guilt afterwards Episodes occur ≥1x/week for at least 3 months of the DSM-5-criteria 	 DSM-4: EDs not fully meeting the diagnostic criteria for specific EDs (like AN or BN) DSM-5: Division into OSFED and UFED DSM-5: Division into OSFED and UFED OSFED: EDs not completely fulfilling criteria for specified EDs fulfilling criteria for specified EDs Atypical AN Atypical AN Atypical AN BN with less frequency/duration Purging disorder without BE BED with lower frequency/ length Night eating syndrome UFED: Insufficient information for making more specific diagnosis
Reference	Zipfel et al. (2015)	Castillo and Weiselberg (2017)	Brownley et al. (2016); Erzegovesi and Bellodi (2016)	Erzegovesi and Bellodi (2016); Grad and Dumitrascu (2020)
<i>Note:</i> This table Abbreviations: <i>i</i> syndrome; IBS,	provides a summary of the diagno AN, anorexia nervosa; BED, binge ε irritable bowel syndrome; OSFED,	stic criteria for the EDs and FGIDs or eating disorder; BN, bulimia nervosa; other specified feeding and eating dis	onsidered in this systematic review. DSM, Diagnostic and Statistical Manual of Mental Disorder sorders; PDS, postprandial distress syndrome; UFED, unspe	s; ED(s), eating disorder(s); EPS, epigastric pain cified feeding or eating disorders.



FIGURE 1 Prisma flow chart. ED(s), eating disorder(s); FGID(s), functional gastrointestinal disorder(s) [Colour figure can be viewed at wileyonlinelibrary.com]

languages other than English, studies on other EDs and FGIDs than the ones mentioned above and studies reporting on patients with EDs lacking co-occurring FGID symptoms/FGIDs (or vice versa) were excluded. If abstracts and titles were not detailed enough to decide whether the inclusion criteria were met, the full texts were assessed for eligibility. Additional records were identified through examining the reference lists of the included literature and independent searching for publications belonging to conference abstracts discovered through the systematic search. The quality of the studies included was assessed with respect to risk of bias by thoroughly evaluating the study designs, selection of participants, methodological procedures applied as well as presentation of the results.

Since no human studies were conducted by the authors in association with the preparation of this review, no ethical approval was obtained. However, of the studies presented and discussed here, 28 studies indicated that the study was approved by an ethics committee, 10 studies stated that the study was conducted in accordance with the recognized standards (e.g. Declaration of Helsinki) and 22 studies indicated that informed consent was obtained by the participants.

3 | RESULTS

3.1 | Data base search

Titles and abstracts of 388 studies were screened. 36 fulltext articles from the systematic search were assessed for eligibility, of which seven were excluded for the following reasons: no ED diagnosis (n = 5) and no co-occurrence of

Reference	Disorder	Assessment of FGIDs/FGID symptoms	Outcome	Possible bias
Abraham and Kellow (2011)	AN BN EDNOS	 Self-report questionnaires: Rome-If^a QOL-IBS Bowel Symptom Severity Index^b 	 FGIDs occurred in 93% of patients with EDs IBS showed the strongest relation among FGIDs to QOL in EDs Symptom severity of IBS and QoL regarding ED and IBS influenced each other 	Response bias
Abraham et al. (2012)	AN BN EDNOS	 Self-report questionnaires: Rome II^a 2 questions asking for abdominal bloating/distension^b 	 Abdominal bloating occurred more often (78%) than abdominal distension (58%) BMI, pelvic floor symptoms and IBS predicted abdominal bloating Pelvic floor symptoms are a predictor for abdominal distension 	Response bias
Abraham and Kellow (2013)	AN BN EDNOS	Self-report questionnaire: - Rome II ^a	- Common G1-symptoms in patients with EDs were mainly consistent with FGIDs of the Rome-II criteria	Response bias
Benini et al. (2004)	AN, restricting type AN, purging type	 Self-report questionnaires: Bowel symptom Questionnaire^b Visual analogue scale for emptying studies^b 	 Gastric emptying was delayed in patients irrespective of subgroup Gastric emptying improved after long-term rehabilitation only in the restricting subgroup Dyspeptic symptoms improved in AN after treatment 	Response bias
Bluemel et al. (2017)	AN	 Self-report questionnaires: Short-form Leeds dyspepsia Questionnaire^b Gastroparesis Cardinal symptom Index^b 	 GI sensory functioning and gastric emptying were altered in AN and improved after weight rehabilitation Dyspeptic symptoms and psychological comorbidities were common in AN 	Response bias Selection bias Performance bias
Boyd et al. (2005)	AN BN EDNOS	Self-report questionnaire: - Rome II ^a	 ≥1 FGIDs occurred in 98% ≥3 FGIDs appeared in 52% Psychological characteristics were predictors of FGIDs IBS was most prevalent FGID (52%) 	Response bias
Boyd et al. (2010)	V/N	Self-report questionnaire: - Rome II ^a	 FGIDs remained frequent despite of treatment and improvement of EDs FGID symptoms were often fluctuating/changing over time 	Response bias Information bias (Continues)

TABLE 2 Study results

Reference	Disorder	Assessment of FGIDs/FGID symptoms	Outcome	Possible bias
Cremonini et al. (2009)	BED	Self-report questionnaire: - Bowel Disease Questionnaire (modified) ^b	 FGID symptoms appeared frequently in patients with BED Psychological characteristics might affect the rela- tionship between FGIDs and EDs 	Response bias
Crowell et al. (1994)	BED, obese BED, non-obese	 Manning criteria^a Self-report questionnaire: Bowel symptom questionnaire^b 	 Highest prevalence and greatest GI symptom severity was obeserved in obese patients with BED Association of IBS-symptoms, binge eating and obesity 	Recall bias Response bias Selection bias
Cuntz et al. (2013)	AN, restricting type AN, purging type	Self-report questionnaire: - Gastro-Questionnaire ^b	 CCK-levels significantly increased after initial weight regain in AN CCK-levels tend to be lower in purging type of AN CCK-levels on admission predicted improvement of GI symptoms under therapy 	Response bias Selection bias Performance bias
Dejong et al. (2011)	BN EDNOS-BN Unknown	Self-report questionnaires: - Irritable bowel syndrome Question- naire ^{1/2}	 IBS was frequent among patients with EDs but rarely treated Features of EDs did not predict the IBS-status 	Response bias
Diamanti et al. (2003)	AN BN	N/A	 Dyspeptic symptoms occurred similarly in AN and BN Gastric emptying and gastric electric activity were disturbed in BN only 	Selection bias
Guerdjikova et al. (2012)	BN	N/A	- Rectal purging was used to compensate for binge eating in BN and as an attempt to alleviate IBS- symptoms	High risk of bias
Guthrie et al. (1990)	IBS	 Confirmation of IBS diagnosis: Presence of abdominal pain and distension, abnormal bowel habit Inconspicuous laboratory values and instrument-based diagnostics 	- ED patterns occurring in IBS patients were more likely caused by psychiatric comorbidity than con- cerns of aggravation of symptoms induced by food intake	Response bias
Hanachi et al. (2019)	AN	Self-report questionnaire: - Francis score for functional intesti- nal disorders ^b	- Gut dysbiosis in severe malnourished patients with AN was associated with severity of functional intestinal disorders and BMI	Response bias
Heruc et al. (2019)	AN, restricting type	Self-report questionnaires: - GI symptom score ^b questionnaire - Visual analogue scale ^b	- Appetite increased and fullness decreased after short- term refeeding therapy, whereas bloating and anxiety still persisted in patients with restricting AN	Response bias Inception bias

TABLE 2 (Continued)

NEL et al							
Possible bias	selection bias	Response bias Dbserver bias	Response bias	٨/٨	tesponse bias decall bias	Response bias selection bias	Response bias Recall bias Attrition bias (Continues)
Outcome	 GI symptoms were commonly reported in patients with AN and BN Gastric emptying was delayed in patients with AN but not BN compared to controls Severity of GI complaints did not correlate with gastric emptying 	 IBS and other psychiatric and medical disorders were 1 often comorbid in patients with BED Overreporting of adverse events did not markedly attenuate these co-occurrences 	 IBS-symptoms were correlated with somatization and ED-symptoms Somatization predicted IBS-symptom severity, whereas anxiety and depression did not 	 GI complaints were common in BN Patients with BN reported greater fullness, satiety and nausea after intake of water Gastric myoelectrical activity was altered in BN 	 BED was associated with abdominal pain and bloating Physical activity was a protective factor for GI- Symptoms 	 Thought-shape fusion was associated with FD in pa- tients with EDs QoL was predicted by depression, dyspepsia, thought- shape fusion 	 GI symptoms were more frequent and more severe in AN AN Alterations in gut microbiota as well as several GI symptoms persist after weight regain in patients with AN
Assessment of FGIDs/FGID symptoms	 Clinical interview^b Inconspicuous instrument-based diagnostics 	 Questionnaire in structured clinical interview format^a 	 Assessment by a medical doctor to exclude organic GI disorders^a Self-report questionnaire: Irritable bowel syndrome severity scoring system^b 	 Clinical interview on gastrointes- tinal symptoms^b Visual analogue scale^b 	Self-report questionnaires: - Rome II ^b	 Rome III criteria^a Self-report questionnaires: Visual Analogue scale^b 	Self-report questionnaires: - Gastro-Questionnaire ^b
Disorder	AN BN	BED	AN	BN	BED	ED (subtype N/A)	AN, restricting type AN, purging type
Reference	Hutson and Wald (1990)	Javaras et al. (2008)	Kessler et al. (2020)	Koch et al. (1998)	Levy et al. (2005)	Lobera et al. (2011)	Mack et al. (2016)

TABLE 2 (Continued)

699

TABLE 2 (Continued)				
Reference	Disorder	Assessment of FGIDs/FGID symptoms	Outcome	Possible bias
Perez et al. (2013)	AN	 Self-report questionnaires: Questionnaire on paediatric gastro- intestinal symptoms - Rome III version^b 	 Gastric accommodation but not gastric emptying was impaired in AN and normalized under therapy Anxiety, somatization and FGIDs occurred more frequent in AN, whereas only somatization and FGIDs improved during treatment 	Response bias Selection bias
Perkins et al. (2005)	ED (subtype N/A)	 Self-report questionnaire: IBS-questionnaire based on the Manning criteria^{a,b} 	 IBS-symptoms were common in patients with EDs and mostly occurred several years after onset of the ED ED-symptoms were associated with IBS-symptoms 	Response bias Recall bias
Salvioli et al. (2013)	AN BN	 Self-report questionnaires: GI symptom questionnaire^b Wexner constipation scores^b 	 Postprandial fullness and abdominal distension were the most reported GI-symptoms Pooled GI symptoms and individual symptom scores in general improved under therapy Hypochondriasis was significantly associated with pooled GI-symptoms and predicted symptom outcome 	Response bias Attrition bias
Santonicola et al. (2013)	BED	 Self-report questionnaires: Rome III^a Standardized questionnaire (0-6) for intensity-frequency score of dyspeptic symptoms^b 	 PDS was more prevalent in obese patients who binge ate Binge eating in obese patients was associated with higher frequency-intensity scores of nausea and epigastric fullness 	Response bias
Santonicola et al. (2012)	AN BN EDNOS	 Self-report questionnaires: Rome III^a Standardized questionnaire (0-6) for intensity-frequency score of broader dyspeptic symptoms^b 	 PDS occurred frequently and epigastric pain syn- drome rarely in EDs Postprandial fullness and early satiety were the most common dyspeptic symptoms in AN Postprandial fullness, epigastric pressure and nausea were the most marked dyspeptic symptoms in EDNOS and BN 	Response bias
Sherman et al. (1993)	AN BN Unclassified ED	- Self-report standardized question- naire (not specified) ^b	 20.8% patients with EDs were seropositive for <i>H. pylori</i> No relationship between <i>H. pylori</i> status and GI symptoms could be detected 	Response bias Selection bias Attrition bias
Singh et al. (2012)	IBS	 Rome III^a IBS severity scoring system^b 	 Patients with severe IBS symptoms had a higher prevalence of somatic, psychiatric and functional gastrointestinal comorbidities The prevalence of BN and BED was not different in IBS patients compared to controls Psychiatric comorbidities were rarely treated 	Response bias Information/detection bias

Reference	Disorder	Assessment of FGIDs/FGID symptoms	Outcome	Possible bias
Spillebout et al. (2019)	ED (subtype N/A) IBS	Self-report questionnaire: - Rome III ^a	 IBS, EDs and their coexistences were common, especially in female university students Behavioural variables as depression, emotional exhaustion, stress, insomnia and cyberaddiction were determinants of IBS, EDs and their coexistence respectively 	Response bias Recall bias
Tang et al. (1998)	IBS	 Rome I^a Daily gastrointestinal symptom diary^b 	 Eating disorder Inventory variables correlated with IBS symptom severity but not with IBS diagnosis Severe vomiting in IBS correlated with drive for thinness 	Response bias Selection bias
Waldholtz and Andersen (1990)	AN	 Evaluation by a gastroenterologist Admission blood test GI symptom survey^b 	 > 21 GI symptoms were reported in 80% of AN patients GI symptoms improved during therapy remaining more pronounced in AN compared to controls 	Response bias Selection bias
Wang et al. (2014)	AN, restricting type AN, purging type BN EDNOS, restricting type EDNOS, purging type	Self-report questionnaires: - Rome II or Rome III, respectively ^a	 ROME III criteria seemed to be more precise in diagnosing FGIDs In Rome III, eating behaviours were the most significant predictors of FGIDs 83%/34% met the criteria for ≥1/3 FGIDs, respectively PDS was the most common FGID when Rome III criteria were applied 	Response bias
Winstead and Willard (2006)	AN BN EDNOS	- Standardized self-report study questionnaire ^{a.b}	 Patients with EDs were more likely to seek medical advice because of GI symptoms BN patients consulted a specialist due to GI complaints prior to seek treatment for their ED 	Response bias Recall bias Selection bias
Yeh et al. (2018)	IBS	- Rome I-IV ^a	 Psychiatric disorders were commonly occurring in IBS IBS patients showed a 7-fold increased risk to develop AN 	Missing statistical information
Note: This table displays the main Abbreviations: AN, anorexia nervo: specified: EDNOS-RN esting disor	results of all studies that we sa; BED, binge eating disorde ders not otherwise specified	rre included into this systematic review. rr; BMI, body mass index; BN, bulimia nervos bulimic tyne: EPS, enicastric nain syndrom.	t; CCK, cholecystokinin; ED(s), eating disorder(s); EDNOS, ea	ting disorders not otherwise nal disorder(s): GI

Ď gastrointestinal; IBS, irritable bowel syndrome; N/A, not applicable; PDS, post-prandial distress syndrome; QoL, quality of life.

^aQuestionnaire used to confirm FGID diagnosis.

^bQuestionnaire used to measure FGID symptoms.

TABLE 2 (Continued)

TABLE 3 Stud	y characteristics								
Reference	Study design	Type of sample	Disorder	Participants	Gender (n)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
Abraham and Kellow	Cross-sectional	Inpatient treatment (specialized	AN BN	71 29	th th	24 ± 6^{a} 25 ± 6^{a}	15.5 ± 1.5^{a} 22.6 ± 3.4^{a}	Australia ⁱ	n.a.
(2011)		ED unit)	EDNOS	60	f	25 ± 7^{a}	19.6 ± 2.4 ^a		
Abraham	Cross-sectional	Inpatient treatment	AN	84	f	23.6 ± 7.3^{a}	15.6 ± 1.4^{a}	Australia ⁱ	n.a.
et al. (2012)		(specialized ED unit)	BN	33	f	23.7 ± 6.6^{a}	22.5 ± 3.3^{a}		
			EDNOS	67	f	25.8 ± 8.9^{a}	19.4 ± 2.9^{a}		
Abraham and	Cross-sectional	Inpatient treatment	AN	84	N/A	$24.6 \pm 8.4^{a,d}$	$18.3 \pm 3.6^{a,d}$	Australia ⁱ	n.a.
Kellow (2013)		(specialized FD unit)	BN	33					
			EDNOS	68					
Benini et al. (2004)	Case-control	Inpatient treatment and follow-up (after 1	ATN, restricting type	6	f	19.9 ± 0.7^{b}	13.2 ± 0.6^{b}	Italian ⁱ	n.a.
		and 5 months of rehabilitation)	AN, purging type	8 (24 age- & sex-matched HC)	f	25.4 ± 1.1^{b}	$15.5 \pm 0.7^{\mathrm{b}}$		
Bluemel et al. (2017)	Cross-sectional and cohort	Inpatient treatment (specialized ED centre)	AN	24	f	23 (18–41) ^f	$14.4 (11.9 - 16.0)^{f}$	Switzerland ⁱ	n.a.
Boyd	Cross-sectional	Inpatient	AN	45	f	20.8 ± 5.2^{a}	15.6 ± 1.3^{a}	Australia ⁱ	n.a.
et al. (2005)		treatment (specialized ED unit)	BN EDNOS	22 34	f	19.9 ± 4.5^{a} 20.7 ± 7.0^{a}	22.1 ± 2.3^{a} 18.3 ± 3.3^{a}		
Boyd et al. (2010)	Cohort	Inpatient treatment (specialized ED unit) and follow-up (1 year after discharge)	N/A	73	÷	20 ± 5^{a}	17.7 ± 3.1 ^a	Australia ⁱ	n.a.
Cremonini et al. (2009)	Cross-sectional	Population-based study	BED	250/4096	m & f	45.1 ± 0.8^{b}	31.1 ± 0.5^{b}	US, Minnesota (presumably 90% Caucasians)	n.a.

⁷⁰² WILEY-

(Continued)	Study design
TABLE 3	Reference

Reference	Study design	Type of sample	Disorder	Participants	Gender (n)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
Crowell	Cross-sectional	Outpatient	BED, obese	73	f	$45.1 \pm 11.8^{\circ}$	N/A	>90% Caucasian	n.a.
et al. (1994)			BED, non- obese	14	f	44.9 ± 12.8 ^c			
Cuntz et al. (2013)	Cohort	Inpatient treatment	AN, restricting type	10	f (22), m (1)	26.1 ± 7.2 ^c	$14.6 \pm 1.4^{\rm c}$	Germany ⁱ	n.a.
		(specialized clinic)	AN, purging type	13(8 sex- & age- matched controls)		24.7 ± 8.1°	$15.5 \pm 1.4^{\circ}$		
Dejong	Cross-sectional	Outpatient ED	BN	51	f (60), m (4)	N/A	$23.38 \pm 4.65^{a,d}$	UK/London ⁱ	n.a.
et al. (2011)		service	EDNOS-BN	12					
			Unknown	1					
Diamanti et al. (2003)	Cross-sectional	Inpatient (paediatric neuropsychiatric service for ED) before treatment	AN	18 (14) ^e	Ţ	15.0 ± 3.9^{a} (15.5 ± 3.2) ^{a.e}	15.2 ± 1.3^{a} (14.9 ± 1.7) ^{a.e}	Italian ⁱ	n.a.
			BN	10 (6) ^e	ţ	17.8 ± 2.1^{a} (17.3 \pm 3.8) ^{a,e}	20.0 ± 4.1^{a} (20.1 ± 4.5) ^{a,e}		
Guerdjikova et al. (2012)	Case report	Inpatient	BN	1	f	20	17.2 (lowest ever)	White	n.a.
							20.4 (on admission)		
Guthrie et al. (1990)	Cross-sectional	Hospital outpatient clinic	IBS	152	f	39 ± 14.2^{a}	N/A	UK/Manchester ⁱ	n.a.
Hanachi et al. (2019)	Case-control	Inpatient treatment	AN	33	f	32 ± 12^{a}	11.7 ± 1.5^{a}	France ⁱ	n.a.
Heruc et al. (2019)	Interventional study	Inpatient treatment	AN, restricting type	22 (17 age matched HC)	ب	$15.9 \pm 0.4^{\mathrm{b}}$	N/A	Australia ⁱ	n.a.
Hutson and Wald (1990)	Cross-sectional	Inpatient treatment (EDs Centre)	AN	10	f (9), m (1)	28 (18–39) ^f	48 (41–58) ^f (mean weight in kg)	NSi	n.a.

TABLE 3 (Con	itinued)								
Reference	Study design	Type of sample	Disorder	Participants	Gender (<i>n</i>)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
			BN	11 (15 sex-matched controls)	f	27 (20–37) ^f	61 (50–73) ^f (mean weight in kg)		
Javaras et al. (2008)	Case-control	Community-based study	BED	 150 subjects 150 age- & sex-matched controls 135 of interviewed relatives 	f(114), m(36) f(101), m (34)	49.5 ± 12.8^{4} 43.0 ± 14.3^{8}	35.8 ± 6.7^{4} 33.4 ± 8.6^{4}	US/Boston ⁱ	n.a.
Kessler et al. (2020)	Cross-sectional	Outpatient unit	AN	19	f	22.3 ± 6.4 ^a	$15.7 \pm 1.7^{\rm a}$	Norway ⁱ	n.a.
Koch et al. (1998)	Cross-sectional	Inpatient treatment (psychiatry unit)	BN	12 (13 age- matched HC)	ц	25 (16-41) ^f	<i>57.7</i> (mean weight in kg)	US/ Pennsylvania ⁱ	n.a.
Levy et al. (2005)	Cross-sectional	Outpatient treatment for weight loss	BED	56/967	m (294), f (689)	$52.7 \pm 12.4^{a,d}$	$33.2 \pm 5.7^{a,d}$	White (917) Non-white (65)	High school or less (194) Some college or more (789)
Lobera et al. (2011)	Cross-sectional	Outpatient treatment	ED (subtype N/ A)	78	f (70), m (8)	22.9 ± 8.3 ^a	N/A	Spain ⁱ	n.a.
Mack et al. (2016)	Interventional study	Inpatient treatment	AN, restricting type AN, purging type	39 16 (55 age- & gender- matched HC)	¢.	23.8 ± 6.8 (14-39) ^f	15.3 \pm 1.4 (11.6-17.7) f (initial) (initial) 17.7 \pm 1.4 (13.9-21.8) f (after treatment)	Germany ⁱ	n.a.
Perez et al. (2013)	Cohort	Inpatient treatment and follow-up (after 3-4 months)	AN	16 (22 age & sex-matched controls)	ۍ ا	15.5 (mean)	17.3 (mean; initial)18.32 (mean; after treatment)	Caucasian	Parents: Some col- lege or more (14) Annual household income ≥75.000 \$ (11)

TABLE 3 (Con	tinued)								
Reference	Study design	Type of sample	Disorder	Participants	Gender (<i>n</i>)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
Perkins et al. (2005)	Cross-sectional	Volunteers from a register of an ED unit	ED (subtype N/ A)	234	f (229), m (4)	34.1 ± 11.7 ^a	24.6 \pm 5.4 ^a (highest BMI) 14.8 \pm 3.4 ^a (lowest BMI)	White British (204), Asian British (2), non-British white (6), other (2), N/A (20)	n.a.
Salvioli et al. (2013)	Cohort	Inpatient treatment (specialized unit) and follow-up (after 1 and 6 months)	AN BN	39	f (41), m (7)	15 (13–16) ⁸	-3 (-3.4 to -1.9) ^{£.h}	Caucasian	n.a.
Santonicola et al. (2013)	Case-control	Outpatient (clinic for surgical therapy of obesity)	BED	22/100	m (44), f (56)	34.3 ± 1.1 ^{b.d}	$45.9 \pm 0.7^{\rm b,d}$	Caucasian	n.a.
Santonicola et al. (2012)	Cross-sectional	Outpatient (clinic for eating behaviour disorders)	AN BN	20 6	N/A	22.45 ± 0.94^{b} 24.83 ± 2.76^{b}	42.79 ± 1.18^{b} (weight in kg) 0.8 ± 6.13^{b} (weight in kg)	Italy ⁱ	n.a.
			EDNOS	10 (22 age- & sex-matched HC)		24.5 ± 1.82 ^b	54.65 ± 2.51^{b} (weight in kg)		
Sherman et al. (1993)	Cohort	Inpatient	AN BN Unclassified ED	23 18 7	ų	N/A	N/A	Canada ⁱ	n.a.
Singh et al. (2012)	Cross-sectional	Outpatient (department of gastroenterology and human nutrition)	IBS	184	m (134), f (50)	32.9 ± 9.4ª	N/A	India ⁱ	Upper (25), upper middle (58), lower middle (70), upper lower (30), lower (1)
									(Continues)

HANEL ET AL.

10990968, 2021, 5, Downloaded from https://anlinelibrary.wiley.com/doi/10.1002/erv.2847 by Chainté - Universitatesmedizin, Wiley Online Library on [08/122022] See the Terms and Conditions (https://onlinelibrary.wiley.com/ethany) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Reference	Study design	Type of sample	Disorder	Participants	Gender (n)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
Spillebout et al. (2019)	Cross-sectional	University students	ED (subtype N/ A)	122/731	f (97), m (25)	20.4 ± 2.2^{a}	10% uw, 65.8% nw, 24.2% ow	France ⁱ	Financial difficulties:
			IBS	57/731	f (49), m (12)	20.8 ± 3.9^{a}	8.8% uw, 70.1% nw, 21.1% ow		47.5% Financial difficulties: 45.6%
Tang et al. (1998)	Cross-sectional	Inpatient	IBS	60	f (43), m (17)	36.8 (mean)	N/A	Canada ⁱ	n.a.
Waldholtz and Andersen (1990)	Interventional study	Inpatient treatment (ED unit)	AN	16	N/A	Early 20s	N/A	USi	Higher than average
Wang et al. (2014)	Cross sectional	Inpatient treatment (specialized ED unit)	AN, restricting type	27	Ļ	25.1 ± 79 ^a (whole ROME III cohort)	18.5 ± 3.8 ^a (whole Rome III cohort)	Australia	n.a.
			AN, purging type	20		24.7 ± 6.4^{a} (whole Rome	18.2 ± 3.6 ^a (whole Rome		
			BN	6		II cohort)	II cohort)		
			EDNOS, restricting	14					
			type						
			EDNOS, purging type	30					
			AN, restricting type	42					
			AN, purging type	32					
			BN	29					
			EDNOS,	18					
			restricting type						
			EDNOS,	40					
			purging type						

MILEY-

TABLE 3 (Continued)

	κ.								
Reference	Study design	Type of sample	Disorder	Participants	Gender (n)	Age (y)	BMI (kg/m ²)	Ethnicity/ Race (n)	Socioeconomic status
Winstead and Willard (2006)	Case-control	Inpatient treatment (ED unit)	AN BN EDNOS	341811 (47 age- matched controls)	f (57), m (6)	$27.6 \pm 9.8^{a,d}$	N/A	White (57), African American (4), Asian (1), hispanic (1)	n.a.
Yeh et al. (2018)	Cohort	Nationwide, population- based study	IBS	22,356 (67,068 age-, sex- & index year- matched controls)	m (11 980) f (10 376)	55.1 ± 18.4 ^c	N/A	Taiwan ⁱ	Education (years): <12 years (15212), ≥12 years (7144)
<i>Note:</i> This table displ Abbreviations: AN, at BN, eating disorders 1 IBS, irritable bowel s; ^a Data presented as m	ays the main charac norexia nervosa; BEL not otherwise specifi- yndrome; m, male; r ean + SD	:teristics of all studies that), binge eating disorder; BV ed, bulimic type; EPS, epig. n.a., not assessed; nw, norr	were included into 11, body mass index astric pain syndrom mal weight; ow, ow	t this systematic rev ; BN, bulimia nervo he; f, female; FD, fu erweight; PDS, post	/iew. sa; CCK, cholecys nctional dyspepsie t-prandial distress	tokinin; ED(s), eating t; FGID(s), functional syndrome; QoL, qual	disorder(s); EDNOS, e gastrointestinal disorc ity of life; uw, underv	ating disorders not otherv er(s); GI, gastrointestinal /eight.	ise specified; EDNOS- ; HC, healthy controls;

TABLE 3 (Continued)

^bData presented as mean \pm SE(M).

 $^{\rm c}{\rm Study}$ did not specify whether data are presented as SD or ${\rm SE}({\rm M}).$

^dData presented as sum of study population.

^eIn brackets data for patients who consented to electrogastrography.

^fData presented as mean (range).

^gDate presented as median (interquartile range).

^hBMI expressed as Body mass index standard deviations (BMIsds) referred to standardized BMI ascertained by a reference population.

ⁱStudy did not report about the ethnicity/race of participants, the country/city mentioned is the location where the study was conducted.

EDs and FGIDs (n = 2). Additional seven publications were found by screening the reference lists of the included articles for further relevant literature; thus, 36 publications were selected for inclusion in this systematic review.

3.2 | Studies included

Details of the study results and characteristics are listed in Tables 2 and 3, respectively, which will be discussed in this review.

Several studies were found that reported on FGID symptoms such as postprandial fullness, abdominal distension, abdominal pain, gastric distension, early satiety, bloating and nausea in patients with AN, BN and EDNOS before treatment for their ED (Hutson & Wald, 1990; Koch et al., 1998; Salvioli et al., 2013; Waldholtz & Andersen, 1990). In one study from 2013 researchers reported that it is highly probable that GI symptoms frequently reported by inpatients with EDs are representative of FGIDs defined by the Rome-II criteria (Abraham & Kellow, 2013). Unsurprisingly, various studies reported on IBS and symptoms of IBS in AN, BN and EDNOS (Abraham & Kellow, 2011; Boyd et al., 2005; Dejong et al., 2011; Guerdjikova et al., 2012; Perkins et al., 2005; Santonicola et al., 2012; Wang et al., 2014). A few studies focused on FGID symptoms in patients with BED (Cremonini et al., 2009; Crowell et al., 1994; Javaras et al., 2008; Levy et al., 2005; Santonicola et al., 2013). Nevertheless, one study found no association between BN or BED with IBS (Singh et al., 2012).

There were several studies reporting on an improvement of FGID symptoms after treatment for EDs (Benini et al., 2004; Boyd et al., 2010; Cuntz et al., 2013; Mack et al., 2016; Perez et al., 2013; Waldholtz & Andersen, 1990).

Many studies aimed to identify factors contributing to the evolution of dyspeptic symptoms in patients with EDs such as gastric dysmotility, psychiatric disorders, obesity, bacterial infection or medical attention (Benini et al., 2004; Bluemel et al., 2017; Diamanti et al., 2003; Hutson & Wald, 1990; Koch et al., 1998; Lobera et al., 2011; Santonicola et al., 2012; Sherman et al., 1993; Winstead & Willard, 2006). Factors responsible for the development of IBS in patients with EDs were also studied, which included QoL, BMI, hypochondriasis, somatization and anxiety (Abraham & Kellow, 2011; Abraham et al., 2012; Boyd et al., 2005; Kessler et al., 2020; Perkins et al., 2005; Salvioli et al., 2013).

Since only less than 20% of patients with EDs had IBS symptoms before the diagnosis of their ED (Perkins et al., 2005) there are only few studies that investigated

contributing factors inducing the development of EDs in patients with IBS (Guthrie et al., 1990; Spillebout et al., 2019; Tang et al., 1998).

3.3 | Quality assessment

The current review consisted of studies with a prospective, retrospective or cross-sectional design as well as one case report, assuming a high risk of bias generally for the latter. Since the studies were more often observational than interventional, this systematic review is predominantly descriptive. As almost all studies applied (selfreport) questionnaires to assess symptoms, the risk of response bias was the most frequent type of bias. Interview bias might be present in studies conducting interviews of semi- or unstructured character by influencing the response behaviour of individual participants, especially if the interviewer was not blinded. Additionally, recall bias may have occurred, particularly in retrospective studies that evaluated for example the presence of GI symptoms in the past, as patients suffering from EDs might be more aware of them than controls. Selection bias most likely resulted from self-selection, as individuals suffering from specific symptoms might be more likely to return a questionnaire. In addition, it was not always possible to recruit controls belonging to the same population as patients. Performance bias may occur in studies applying instrument-based techniques, for example if the investigator was not blinded. Prospective studies with follow-up periods were affected by attrition bias when patients refused to fill in questionnaires or undergo diagnostic investigation, when ED therapy was discontinued or when participants were lost to follow-up or dropped out of the study.

Several points possibly contributed to a limited comparability across studies and thus limited transferability of results to the general population. First, different instruments were used to assess FGIDs in patients with EDs and vice versa (e.g., Rome criteria, Manning criteria, Diagnostic and Statistical Manual of Mental Disorders [DSM] criteria) which might be a reason for different prevalence rates. Secondly, stages of the disease as well as inclusion and exclusion criteria applied were very heterogeneous. Furthermore, only very few studies consisted of a population-based study sample, whereas the majority of the studies was conducted in samples in higher levels of care groups. This is a major limitation, as results and conclusions drawn for participants with a course of the disease requiring care in specialized units cannot be reliably generalized to patients of the general population or to other levels of care. In addition, therapeutic management, follow-up periods

as well as diagnostic investigations were different, as for example organic diseases as a cause for GI symptoms were not always ruled out, potentially leading to distorted prevalence rates. Furthermore, some studies lacked a control group. Taken together, bias cannot be ruled out for the studies included in this systematic review; thus, these limitations should be kept in mind when interpreting the results discussed here.

4 | DISCUSSION

4.1 | Prevalence of FGIDs and their symptoms in patients with EDs

The included studies show that IBS and FD and their symptoms are common in patients with EDs (Salvioli et al., 2013). Inpatients with AN and BN on admission for ED treatment, of whom 40% sought medical advice from a gastroenterologist in the past and 17% have been diagnosed with IBS, frequently reported postprandial fullness and abdominal distension, followed by abdominal pain, gastric distension and early satiety (Salvioli et al., 2013). In an early prospective study in inpatients with restricting and purging type of AN, all patients reported GI symptoms such as bloating, constipation or diarrhoea, of whom 80% complained of at least one severe GI symptom (Waldholtz & Andersen, 1990). In another clinical study in inpatients with AN and BN especially bloating, but also early satiety and abdominal pain were reported by both groups of patients, whereas controls did not report any GI symptoms (Hutson & Wald, 1990). Focusing only on BN, in another study it was shown that GI symptoms like early satiety, bloating, nausea and upper abdominal pain were often reported by female inpatients with BN (Koch et al., 1998). FGIDs, especially IBS, functional abdominal bloating, functional constipation and functional dyspepsia were prevalent comorbidities in inpatients suffering from EDs attending a specialized Eating Disorder Unit, with 98% reporting at least one and 52% three or more FGIDs applying the Rome-II criteria (Boyd et al., 2005). Within a study sample from a register of an ED unit (n = 234), 64% fulfilled the Manning criteria for IBS, of whom only 25% were ever diagnosed with IBS and those with confirmed IBS were rarely treated (Perkins et al., 2005). In a clinical study in inpatients with AN, BN and EDNOS on admission to a specialized ED unit for treatment, it was shown that FGID-like disorders were common among participants (Abraham & Kellow, 2011).

IBS occurred also frequently in outpatients from a specialized eating disorder service suffering from BN and EDNOS-BN (n = 64), respectively, while only 11%

reported to receive treatment for their FGID symptoms (Dejong et al., 2011). Also criteria for postprandial distress syndrome (PDS) were fulfilled in 90% of patients with AN and EDNOS attending an outpatient department for eating disorders and by 83.3% of patients with BN (Santonicola et al., 2012). Epigastric pain syndrome (EPS) was present in one patient suffering from BN only, whereas in none of the patients a coexistence of EPS and PDS could be detected (Santonicola et al., 2012). A study applying the Rome-II criteria described that IBS, functional abdominal bloating and functional constipation were common among inpatients with EDs on admission for treatment (Abraham & Kellow, 2013). It was observed that GI symptoms encompassing nausea, vomiting, bloating, abdominal pain and difficult defecation emerge more often in individuals with BED compared to normal weight controls and obese patients without BED, whereas obese patients with BED displayed the highest prevalence and severity of these symptoms in general (Crowell et al., 1994). A case report of a young woman with BN and IBS introduced "rectal purging" as a symptom of BN, meaning excessive rectal insertion of the finger to induce defecation (Guerdjikova et al., 2012). This behaviour was performed to compensate for episodes of binge eating but also to relieve constipation, abdominal pain and bloating accompanying IBS (Guerdjikova et al., 2012).

A cross-sectional population-based study observed that upper (bloating, abdominal pain) and lower (diarrhoea, constipation, faecal urgency) FGID symptoms often appear in patients with BED, possibly due to inappropriate compensation of exceedance of the already increased gastric capacity and altered intestinal secretion and motility caused by the large amount of food ingested (Cremonini et al., 2009). Additionally, a case-control study applying the Rome-III criteria reported that the prevalence of FD, functional constipation, functional diarrhoea and functional bloating did not greatly differ between morbidly obese outpatients of a tertiary care centre and HCs, except for IBS (Santonicola et al., 2013). Obese patients with binge eating behaviour reported higher frequency-intensity scores regarding epigastric fullness and nausea compared to the remaining obese patients, whereas interestingly, none of the patients with BED met the criteria for EPS (Santonicola et al., 2013). In contrast, in another study a significant association between obesity, binge eating behaviour and occurrence of IBS symptoms was detected (Crowell et al., 1994). Contrarily, in a study on 983 obese outpatients, of whom 5.8% fulfilled the criteria for probable BED, BED was found to be strongly and positively linked to abdominal pain and bloating but not with the diagnosis of IBS (Levy et al., 2005).

Comparison of these different studies shows that the prevalence for FGIDs is very different among studies

ranging between 42% and 98% in specialized ED units, with IBS and FD being most frequent. As PDS, a subtype of FD, occurred considerably more often than EPS in patients with EDs, it might be speculated that dyspeptic symptoms seem to be especially related to food consumption in patients with EDs. There are different factors that might have contributed to inconsistent prevalence rates: selection of participants (different sex, age, type of ED, duration and severity of the disease, comorbidities), criteria used to diagnose the ED/FGID symptoms (e.g., Rome criteria, Manning criteria) and bias (e.g., reporting bias, selection bias, performance bias). The importance of consistent diagnostic criteria when directly comparing results of different studies is further highlighted by a study by Wang et al. that determined prevalence rates of FGIDs applying the Rome-II and Rome-III criteria, respectively, in female inpatients on admission for ED treatment (Wang et al., 2014). PDS (45%) and IBS (41%) were the most common diagnoses in the Rome-III group, whereas the most prevalent FGIDs in the Rome II cohort were IBS (45%), functional bloating (30%) and functional constipation (26%) (Wang et al., 2014). However, on applying the Rome-III criteria, an increased prevalence (+29-46%) of functional gastroduodenal disorders, depending on the status of cyclic vomiting, was observed, whereas the prevalence of functional bowel disorders decreased by 35% using Rome-III (Wang et al., 2014). The criteria for at least one FGID were met by 83% in the Rome-III group and 94% in the Rome-II sample, whereas the prevalence of at least three FGIDs was similar in both groups (34% and 36%, respectively) (Wang et al., 2014). Moreover, several studies reported that IBS and FD frequently overlap (Choi et al., 2017; von Wulffen et al., 2019), indicating that these diseases are not mutually exclusive (Suzuki & Hibi, 2011).

4.2 | Effect of ED treatment on FGIDs/ FGID symptoms in patients with EDs

A strong association between EDs and FGIDs is further supported by observations of improving FGID symptoms after nutritional and psychiatric treatment of EDs. The total GI symptom score markedly improved in inpatients with AN under nutritional and psychiatric treatment; however, their total symptom scores remained higher compared to controls (Waldholtz & Andersen, 1990). Refeeding tended to ameliorate individual symptoms like abdominal pain but only appetite, bloating, constipation, vomiting and diarrhoea improved significantly with this treatment (Waldholtz & Andersen, 1990). Also, dyspeptic symptoms such as nausea, vomiting and early satiety were alleviated in inpatients with restricting and purging

type of AN, with a longer duration of nutritional rehabilitation and psychotherapy having a more pronounced effect than short-term refeeding (Benini et al., 2004). One vear after inpatient ED treatment, ED symptoms improved in general (BMI, ED behaviours, psychological features) in patients with EDs (Boyd et al., 2010). Although during the follow-up period 34% of patients developed one new regional category of FGIDs that often differed from the category present at the beginning, the investigators also reported that in some patients the FGIDs resolved, resulting in the observation that the overall prevalence of FGIDs decreased from 97% on admission to 77% over time (Boyd et al., 2010). In contrast to the results of other studies, no correlations between BMI changes, ED behaviours, psychological features and changes in FGIDs could be observed (Boyd et al., 2010).

Somatization, anxiety and existence of FGIDs were compared in adolescents with AN to age-matched controls before and after 3-4 months of inpatient treatment (not specified in the manuscript), which improved caloric intake and BMI in patients with AN (Perez et al., 2013). Gastric emptying, assessed using ultrasonography after a liquid meal, did not markedly differ between groups, whereas maximum postprandial antral diameter as a surrogate for gastric accommodation was initially lower in AN and similar to controls after nutritional rehabilitation (Perez et al., 2013). Scores for somatization (e.g., bloating, constipation, abdominal pain) improved under therapy, while those for anxiety, which remained considerably higher in AN than controls, did not (Perez et al., 2013), possibly due to the short follow-up period of 14 weeks. FGID symptoms were reported significantly more often by inpatients with AN than in controls also fulfilling the criteria for at least one FGID (controls were recruited from an adolescent primary care unit) (Perez et al., 2013). IBS was the most common FGID among the study population (Perez et al., 2013). Notwithstanding, after nutritional rehabilitation, 25% of the patients still met the criteria for IBS, indicating that IBS improved over time of treatment in 31% of the patients (Perez et al., 2013).

In a study including inpatients of a specialized clinic with restricting and purging type of AN, cholecystokinin (CCK) concentrations on admission predicted improvement of FGID symptoms (e.g. abdominal pain, nausea, diarrhoea, abdominal fullness) under treatment, as patients with initially higher CCK levels reported lesser FGID symptoms alleviation (Cuntz et al., 2013). However, more recently this potential predictive role of CCK could not be reproduced in inpatients with AN (Heruc et al., 2019), possibly due to the small sample size and different measurement methods. Overall, further investigations examining whether hormonal changes might predict the improvement of GI symptoms in patients with EDs, as suggested for CCK are necessary. Indeed, dysregulations of gastrointestinal peptides like ghrelin and peptide YY, both involved in the regulation of hunger and satiety as well as gastrointestinal functions, have been observed in patients with EDs which potentially perpetuate disturbed eating behaviours and therefore complicate treatment of EDs (Tong & D'Alessio, 2011) but potentially also recovery from FGIDs.

Other studies underlined the role of serotonin, which plays a crucial role in initiating gastrointestinal peristalsis (Mohammad-Zadeh et al., 2008). Regarding EDs, serotonin dysregulation has been linked to binge eating in BN and BED (Hainer et al., 2006), while the decreased serotonin tone in patients with AN arose most likely from malnutrition (Steiger, 2004), leading to the hypothesis that this decreased serotonin activity might induce often comorbid psychiatric disorders like depression in patients with EDs (Haleem, 2017) but potentially also affects gastric emptying, which is often delayed in patients with AN. Additionally, it was speculated that patients with diarrhoea-predominant IBS might present with higher plasma levels of serotonin due to an impaired re-uptake, while patients with constipation-predominant IBS might have lower postprandial plasma levels due to a disturbance of the serotonin release (Holtmann et al., 2017), indicating that an impaired serotonin signalling seems to be involved in the pathogenesis of IBS affecting intestinal motility. Furthermore, an association of serotonin transporter polymorphisms and psychiatric illnesses, frequent in patients with IBS, has been established (Cristina et al., 2019). Taking these results into account could help to explain why central neuromodulators such as antidepressants affecting the serotonin system are also useful to treat FGIDs (Drossman et al., 2018). However, more studies are needed to further illuminate the importance of serotonin in EDs and FGID symptoms. Moreover, the reproducibility and possible relationships of these findings should be assessed in subsequent studies including patients with FGIDs and comorbid EDs to get further insight into the aetiological interplay of gastrointestinal hormones, EDs, psychological alterations and FGID symptoms/FGIDs.

In contrast to the above-mentioned studies, several publications described that patients with FGIDs like IBS and FD remained symptomatic even after recovery of the ED. A study including inpatients with EDs indicated that FGIDs often persist or even emerge after long-term treatment (Boyd et al., 2010). Similarly, in inpatients with AN who also have GI symptoms, most notably several lower GI complaints such as abdominal pain, changes in stool frequency and consistency as well as feeling of incomplete evacuation improved after weight regain, while upper GI symptoms like abdominal fullness, bloating and distension were not markedly

alleviated by ED treatment (Mack et al., 2016). In addition, due to the treatment the richness of microbiota rose in inpatients suffering from AN; however, alterations in the gut microbiota, such as increased clostridium clusters I, XI and XVIII and reduced Roseburia spp. persisted (Mack et al., 2016). In line with these findings, a casecontrol study investigating inpatients with AN reported that microbiome dysbiosis was moderately correlated with functional symptom severity and BMI during enteral nutrition (Hanachi et al., 2019). These findings led to the hypothesis that persisting alterations in microbial composition contribute to the persistence of GI symptoms in patients with EDs. Notwithstanding, these observations should be further confirmed by investigations after weight normalization in these patients, and large long-term studies are required to ascertain if bacterial richness contributes to the improvement of GI symptoms and therefore to identify potential new therapeutic approaches, also for other EDs.

Overall, persistent FGID symptoms after ED therapy led to the hypothesis that the evolution of FGIDs in patients with EDs might be irrespective of how the ED develops especially in the presence of psychological distress and that EDs are not the only cause of the FGIDs. These differences are probably due to, among other things, different ages of the study populations investigated, as adolescents might benefit from an early treatment.

4.3 | Development of FGIDs in patients with EDs

Several studies aimed to identify factors contributing to the evolution of FGID symptoms in patients with EDs. Strikingly, AN, BN and EDNOS were all associated with bloating and distension (Abraham et al., 2012), giving rise to the hypothesis that altered eating behaviour and psychological factors that define EDs in general are more important in the pathogenesis of FGIDs like IBS and FD in EDs than subtype-specific characteristics such as low body weight in AN or vomiting in BN.

4.3.1 | Eating behaviour

In a cross-sectional study in individuals with present or recovered EDs (n = 234), a moderate positive relationship between ED symptoms and number of IBS symptoms was observed, whereas no significant associations regarding duration of EDs and lowest ever BMI could be detected (Perkins et al., 2005). Similarly, it was reported that among typical ED features and behaviours, laxative use correlated significantly with all factors except for

esophageal discomfort, whereas current BMI and binge eating were correlated with the factor vomiting (Abraham & Kellow, 2013). The association of disordered eating behaviours like binge eating and GI complaints in patients with EDs indicates that patients suffering from FGIDs should be screened for these behaviours and that they should be considered in the management of GI symptoms as they potentially impede therapeutic success. However, it requires further investigation whether and how specific eating patterns and behaviours contribute to the development of FGID symptoms. In patients with IBS neither eating behaviours nor characteristics associated with BN could be identified as predictors for the IBS status, indicating that IBS does not seem to be markedly associated with the current ED features (Dejong et al., 2011). Noteworthy, according to the study of Wang and colleagues, disturbed eating behaviours (food restriction, exercise, starvation, binge eating, laxative use, self-induced vomiting) seem to be the strongest predictors for the occurrence of FGIDs in patients with EDs applying the Rome-III criteria, but not Rome-II criteria (Wang et al., 2014), underlying again the importance of the use of specifying diagnosing criteria. Moreover, most of the studies discovered no association of BMI with FGIDs in patients with EDs, leading to the hypothesis that the occurrence of FGIDs like IBS and FD might be more related to altered eating behaviours in patients with EDs than changes in BMI alone.

4.3.2 | Psychological disturbances

There are studies indicating that psychological disturbances like depression, hypochondriasis, anxiety, neuroticism, somatization and thought-shape fusion are often reported in patients with EDs and GI symptoms. In female inpatients on admission for ED treatment, somatization was associated with less likelihood to develop unspecified functional bowel disorders, while no association was observed with anxiety (Wang et al., 2014). In addition, it was reported that for the Rome-II criteria, especially, the psychological factors (somatization, neurocitism, state and trait anxiety, depression, Eating Attitudes Test score) were the main predictors for the occurrence of FGIDs in patients with EDs (Wang et al., 2014). Also in outpatients suffering from AN moderate-strong positive correlations of IBS symptoms with ED symptoms as wells as somatization were observed, while only somatization was identified as the predictor for severe IBS symptoms (Kessler et al., 2020). In this study, IBS symptom severity was not markedly affected by BMI and psychopathological features like commonly occurring depression and anxiety (Kessler et al., 2020). Similarly, it was reported that in patients with

EDs psychological aspects encompassing anxiety and depression could not be identified as predictors for the IBS status, indicating that IBS does not seem to be markedly associated with psychological alterations (Dejong et al., 2011). This contradicts the findings from another study showing that anxiety predicted several FGIDs in patients with EDs (Boyd et al., 2005). Moreover, in this study it was shown that not only binge eating but also somatization and neuroticism were identified as predictors for several FGIDs (Boyd et al., 2005). Interestingly, only neuroticism was associated with the coexistence of three or more FGIDs, potentially being an expression of chronic stress (Boyd et al., 2005). In inpatients with AN and BN on admission, a moderate positive correlation of pooled GI symptoms with hypochondriasis was detected and after follow-up, pooled GI symptoms significantly improved in patients with normal values for hypochondriasis on admission compared to those with higher scores (Salvioli et al., 2013). A significant co-occurrence with disorders such as anxiety disorders, major depressive disorder, body dysmorphic disorder as well as IBS was detected for participants with BED in a community-based study compared to controls (Javaras et al., 2008). Similar but less pronounced, associations were found for subthreshold BED and IBS (Javaras et al., 2008). After adjustment for overreporting adverse events, which was more common in BED, the odds for co-occurring disorders did not markedly decrease (Javaras et al., 2008). In line with these findings a cross-sectional population-based study observed that upper and lower FGID symptoms often appear in patients with BED irrespective of BMI and physical activity level, whereas adjustment for mental health features weakened almost all associations (except fecal urgency and diarrhoea) between FGID symptoms and BED (Cremonini et al., 2009). In inpatients with AN, BN and EDNOS it was shown that the diagnosis of IBS exhibited the strongest correlation with global and subscores of the QoL ED questionnaire (Abraham & Kellow, 2011). Additionally, poor QoL caused by features of EDs was accompanied by greater symptom severity regarding IBS (Abraham & Kellow, 2011). A study enrolling outpatients with EDs (n = 78), patients with psychiatric disorders (n = 77) and students (n = 90), all suffering from dyspepsia reported that although total scores of dyspeptic symptoms did not significantly differ between groups, satiety and bloating were more common in outpatients with EDs (Lobera et al., 2011). Additionally, a lower QoL was reported by patients with EDs and psychiatric disorders compared to students (Lobera et al., 2011). Initially, all groups displayed a moderate positive correlation of thought-shape fusion (believing in weight gain, committing moral misconduct and feeling fat due to thinking of consuming forbidden food) with

symptoms of FD (Lobera et al., 2011). Interestingly, after adjustment for psychopathological variables, only in patients with EDs a moderate positive correlation between thought-shape fusion and FD symptoms was still visible, possibly due to visceral hypersensitivity (Lobera et al., 2011). Furthermore, dyspepsia, thought-shape fusion and depression but not anxiety were identified to predict QoL in patients with EDs (Lobera et al., 2011).

These studies point towards similar aetiologies of EDs and other psychiatric disorders. All these studies give rise to the hypothesis that psychological features play a crucial role in the pathogenesis and development of FGIDs like IBS and FD in patients with EDs and that psychopathological distress experienced by patients with EDs might result in an expression of this discomfort as GI symptoms. As some studies reported that these comorbidities persisted after weight rehabilitation (Perez et al., 2013), the impact of psychological factors (e.g. anxiety) on the evolution of FGID symptoms should be further examined to detect whether therapies specializing on psychological alterations are advantageous for the recovery of FGID symptoms in patients with EDs and on the other hand to assess if and to which extent these alterations contribute to the delayed recovery of FGID symptoms despite ED rehabilitation. If necessary, this should be incorporated better in the treatment of EDs, as it is likely that FGIDs/FGID symptoms arise due to a complex interplay of biological, psychological and social factors (Van Oudenhove et al., 2016). In contrast, some studies did not detect a correlation of psychological alterations and FGIDs/FGID symptoms in patients with EDs. Therefore, longitudinal population-based studies further examining the relationship between psychological characteristics, EDs and GI alterations as well as the impact of therapeutic interventions need to be conducted, also in male subjects to identify possible gender-specific differences.

4.3.3 | Organic dysregulation

Several studies investigated whether in patients with EDs the presence of organic dysregulation is responsible for upper GI symptoms rather than FD. In inpatients with AN and BN neither body weight nor GI symptom severity correlated with gastric emptying, which was assessed after intake of a mixed meal (Hutson & Wald, 1990). Similarly, in inpatients with BN compared to female HCs, cutaneous electrogastrography detected no significant differences in gastric emptying between groups except for an accelerated lag phase in patients with BN (Koch et al., 1998). Nonetheless, disturbed gastric myoelectrical activity such as increased episodes of bradygastria was detected in patients with BN compared to controls, as well as more fullness, satiety and nausea before and after a water load (Koch et al., 1998). In line with this finding, inpatients with BN (n = 10) displayed irregular and disturbed gastric motility leading to delayed gastric emptying, whereas motility of inpatients with AN (n = 18) did not differ from HC (n = 16) as assessed using cutaneous electrogastrography (Diamanti et al., 2003). Interestingly, dyspeptic symptoms encompassing fullness, epigastric pain, postprandial discomfort and nausea occurred equally in AN and BN groups (Diamanti et al., 2003). In another study in inpatients with restricting and purging type of AN, gastric symptoms occurred more frequently in patients than in HC and both subgroups of AN displayed delayed gastric emptying and greater antral diameters in ultrasonography after a solid meal compared to controls (Benini et al., 2004). Interestingly, no associations between dyspeptic symptoms, gastric emptying and psychological distress were detected at any time (Benini et al., 2004).

Compared to obese patients, constitutionally thin participants and HC, postprandial fullness was reported more frequently by outpatients with EDs in a study examining the presence of FD, its subgroups EPS and PDS as wells as other dyspeptic symptoms (e.g. epigastric pressure, belching, nausea, vomiting) (Santonicola et al., 2012). Early satiety occurred most often in AN, whereas nausea and epigastric pressure occurred frequently in EDNOS and BN (Santonicola et al., 2012). Interestingly, dyspeptic symptoms in general were rarely found in obese patients (Santonicola et al., 2012). A study on inpatients with AN demonstrated that while gastric emptying was delayed in AN compared to HCs and obese patients, no significant differences in antral motility and oro-cecal transit time were detected (Bluemel et al., 2017). At any time of measurement, the AN group reported more postprandial fullness and a slower increase of hunger after meal ingestion over time than the other groups, whereas nausea, bloating and abdominal pain did not differ (Bluemel et al., 2017). These alterations were reversed at least in parts after weight restoration in AN: gastric emptying accelerated and postprandial fullness as well as hunger improved (Bluemel et al., 2017). In addition, dyspeptic symptoms, anxiety and depression were more present in patients suffering from AN than in the other groups (Bluemel et al., 2017). Furthermore, no correlation of the Helicobacter pylori status (20.8% positive, 79.2% negative) with dyspeptic symptoms in inpatient adolescents with EDs (n = 48) was detected (Sherman et al., 1993). Lastly, in a case-control study, inpatients with EDs (n = 63) were more likely to see a doctor because of GI complaints than controls, as 41 (65%) subjects of the ED



FIGURE 2 Factors potentially contributing to the interplay between eating disorders and functional gastrointestinal disorders presented and discussed in this systematic review. This figure provides an overview on factors whose potential contribution to the relationship between eating disorders and functional gastrointestinal disorders (particularly irritable bowel syndrome and functional dyspepsia) is described and discussed in this systematic review. GI, gastrointestinal [Colour figure can be viewed at wileyonlinelibrary.com]

group sought gastrointestinal healthcare (Winstead & Willard, 2006). Moreover, patients with EDs tended to receive medication to alleviate GI discomfort more often than the control group (Winstead & Willard, 2006). Among patients with EDs, only those suffering from BN sought medical advice for burdensome GI symptoms significantly earlier before seeking treatment for their ED (Winstead & Willard, 2006).

Overall, although most studies did not detect organic alterations in EDs that could be made responsible for upper GI symptoms, these observations could contribute to the explanation why FD is prevalent in EDs. Due to the observation that gastric emptying seems to be mainly altered in adults with AN that improves with treatment (Bluemel et al., 2017) but lacks correlations with GI symptoms (Benini et al., 2004; Hutson & Wald, 1990), it was hypothesized that disturbed gastric emptying is rather a result than a cause of disordered eating behaviour with subsequent low body weight. As gastric motility was also altered in inpatient adolescents with BN but dyspeptic symptoms occurred equally frequent in AN and BN (Diamanti et al., 2003), alterations in motility are likely not the only factor leading to upper GI symptoms especially in patients with AN. Hence, altered gastric emptying does not seem to be the primary factor

contributing to the development and perpetuation of FGIDs like FD and is possibly not the main influencing factor of GI symptom severity in patients with EDs (Figure 2). Similarly, several studies in patients with gastroparetic symptoms (such as nausea, epigastric pain, early satiety, bloating) reported that the severity of symptoms did not correlate or at best weakly correlated with the severity of the delay in gastric emptying (DiBaise et al., 2016; Pasricha et al., 2011) or other motility disturbances like impaired gastric accommodation (Karamanolis et al., 2007). However, delayed gastric emptying is a phenomenon often blamed for GI complaints in patients with EDs especially during the initial refeeding period (Weterle-Smolińska et al., 2015). Additionally, it is commonly observed in the context of starvation in general which also induces physiological and psychological alterations seen in patients with EDs that could be partly responsible for the development or aggravation of GI complaints in these patients (Keys et al., 1950). However, results regarding normalization of gastric emptying after weight restoration and the use of prokinetics are controversial, so that the impact on the evolution of FGID symptoms remains unclear (Norris et al., 2016) and correlations are often missing, most likely due to small study samples and different methods used.

Therefore, it might be hypothesized that FGID symptoms and their severity in patients with EDs might be more dependent on other factors such as GI sensory disturbances (Farzaei et al., 2016). Indeed, comparing gastric myoelectrical activity and upper GI symptoms in patients with BN to HCs (Koch et al., 1998) indicates that altered gastric myoelectrical activity may contribute to altered visceral perceptions in patients with BN. In some patients with FD and IBS, visceral hypersensitivity (increased perception of gastrointestinal stimuli) is thought to be an important factor in the pathogenesis of FGID symptoms (Enck et al., 2017; Farzaei et al., 2016). A multinational study in patients with IBS and FD assessing visceral sensitivity to distension demonstrated a significant association of GI symptom severity and visceral hypersensitivity, a finding further reinforcing this theory (Simrén et al., 2018). Interestingly, this association persisted after adjustment for psychological comorbidities like anxiety and depression, suggesting that mental illness may not be the main mediator of this relationship (Simrén et al., 2018), which is partly contrary to former studies presuming a greater impact of comorbid mental disorders (Van Oudenhove et al., 2007) and an increased tendency to report symptoms (Dorn et al., 2007). Nevertheless, the significance of FGID-related altered visceral perception should be further investigated in patients with EDs suffering from FGID symptoms, also considering the psychological state of the individual which is altered due to starvation as well and was reported to be associated with GI symptom severity and QoL in IBS (Jerndal et al., 2010) and FD (Van Oudenhove & Aziz, 2013). Moreover, it was observed that psychological alterations like anxiety and depressiveness often persist after weight regain in patients with EDs and FGID symptoms. Therefore, larger long-term studies are required to clarify the role of delayed gastric emptying, sensitivity disturbances as well as the impact of psychological diseases in the context of FGID symptoms in patients with EDs.

4.4 | Development of EDs in patients with FGIDs

Since only less than 20% of patients with EDs had IBS symptoms before the diagnosis of EDs (Perkins et al., 2005), there are only few studies that investigated contributing factors inducing the development of EDs in patients with IBS. One study described that patients with IBS reported at least slightly altered eating behaviours (e.g. disturbed attitude towards eating, thought to become thinner by vomiting) (Tang et al., 1998), pointing

towards the occurrence of at least sub-syndromal disordered eating in patients with IBS. Comparing inpatients with IBS (n = 60) to university students (n = 271), it was observed that women showed higher levels of ineffectiveness and body dissatisfaction than men; however, no differences regarding IBS symptom severity were observed (Tang et al., 1998). Interestingly, only the score for ineffectiveness was higher in female patients with IBS compared to controls, whereas no differences regarding perfectionism and body dissatisfaction could be detected (Tang et al., 1998). A moderate, positive relationship between IBS symptom severity and perfectionism as well as ineffectiveness was detected (Tang et al., 1998). In addition, in a large sample of university students (n = 731) it was demonstrated that an ED occurred in 16.7% of the students, IBS in 7.8% and both together in 2.7% (Spillebout et al., 2019). Furthermore, EDs were more frequent in students suffering from IBS (35%) than in those who did not (11.5%), leading to a twofold increased risk to have an ED in students with IBS (Spillebout et al., 2019). Female gender, financial difficulties, anxiety, depression, stress, emotional exhaustion, insomnia and cyber addiction were positively associated with IBS, EDs or their coexistence (Spillebout et al., 2019). Lastly, a large nationwide, population-based cohort study examining 22,356 patients with IBS and 67,068 non-IBS controls described a 7-fold increased risk for AN in patients with IBS compared to controls (Yeh et al., 2018). In summary, female sex, ineffectiveness and thoughts to become thinner could be the risk factors for the development of EDs that should be studied in more detail in patients with IBS.

Noteworthy, patients with IBS and comorbid psychiatric illness reached higher levels in scores assessing EDrelated symptoms. In an early study, no women with organic GI disorders (n = 27) but 8 of 152 female outpatients with IBS (5%) reached a score beyond 30 in the Eating Attitudes Test, indicating the presence of AN or BN (Guthrie et al., 1990). Within the IBS group, 23% reported a disturbance regarding their attitude towards eating (Guthrie et al., 1990). 48% of the patients with IBS and therefore significantly more than in the control group (26%) depicted signs of mental disorders as depression and anxiety (Guthrie et al., 1990). Interestingly, patients with IBS and comorbid psychiatric illness scored higher in the Eating Attitudes Test than those without (Guthrie et al., 1990). These observations led to the hypothesis that ED patterns in patients with IBS are more probably conditioned by psychiatric disturbances than due to concerns of symptom exacerbation subsequent to food consumption; therefore, psychological factors are major contributors to the development of EDs in IBS. Nevertheless, there is a lack of studies investigating the development of EDs in patients with IBS. Therefore, long-term studies are required to assess predictors and additional underlying factors leading to a development of EDs in patients suffering from IBS.

4.5 | Chicken or egg dilemma with EDs and FGIDs

In several studies, a significant relationship between EDs and IBS symptoms was detected. It could be hypothesized that EDs are a potential risk factor for the development or severity of IBS as the majority of cases (87%) reported an onset of IBS symptoms within 10 years after developing the ED (Perkins et al., 2005). This temporal sequence was also observed in an early study in which 80% of patients with FGIDs such as IBS and FD and a past ED had an onset of GI complaints when the ED was already present, while 10% developed GI symptoms several months before or after the ED episode, respectively (Porcelli et al., 1998).

5 | SUMMARY AND CONCLUSION

Taken together, the results of the studies included in this systematic review highlight the need for close multidisciplinary collaboration. Since patients with EDs may consult a physician earlier for gastrointestinal complaints than for ED symptoms, it might be beneficial to screen patients presenting with unexplained GI symptoms for an underlying ED. This systematic review provides a comprehensive overview of studies examining the frequency and potential pathophysiological alterations leading to the development and persistence of FGIDs, especially IBS and FD in patients with EDs and vice versa. Despite the systematic approach, it cannot be guaranteed that all relevant literature on this topic has been identified and included, which is a limitation of this systematic review. As most of the included studies consisted of small sample sizes, there is a need for large controlled long-term studies with well-defined follow-up periods also taking into account subtypes of EDs (e.g. restricting and purging type of AN) but also other EDs like avoidant restrictive food intake disorder. It should be examined how and to what extent GI symptoms impede therapeutic success in patients with EDs and how they develop over the course of therapy. Lastly, the impact of psychopathological alterations, hormones and GI functions on this relationship should be investigated to optimize the diagnostic and therapeutic procedure, outcome and in the end the QoL in patients with EDs suffering from FGIDs like IBS and FD and vice versa.

ACKNOWLEDGMENTS

This work was supported by funding of the German Research Foundation (STE 1765/3-2) and Charité University Funding (UFF 89/441-176, to A.S.).

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

ORCID

Vivien Hanel Dhttps://orcid.org/0000-0001-6333-4460 Martha A. Schalla Dhttps://orcid.org/0000-0001-9739-0786

REFERENCES

- Abraham, S., & Kellow, J. (2011). Exploring eating disorder quality of life and functional gastrointestinal disorders among eating disorder patients. *Journal of Psychosomatic Research*, 70(4), 372–377. https://doi.org/10.1016/j.jpsychores.2010.11.009
- Abraham, S., & Kellow, J. E. (2013). Do the digestive tract symptoms in eating disorder patients represent functional gastrointestinal disorders? *BMC Gastroenterology*, 13, 38. https://doi. org/10.1186/1471-230x-13-38
- Abraham, S., Luscombe, G. M., & Kellow, J. E. (2012). Pelvic floor dysfunction predicts abdominal bloating and distension in eating disorder patients. *Scandinavian Journal of Gastroenterology*, 47(6), 625–631. https://doi.org/10.3109/00365521. 2012.661762
- Benini, L., Todesco, T., Dalle Grave, R., Deiorio, F., Salandini, L., & Vantini, I. (2004). Gastric emptying in patients with restricting and binge/purging subtypes of anorexia nervosa. *American Journal of Gastroenterology*, 99(8), 1448–1454. https://doi.org/ 10.1111/j.1572-0241.2004.30246.x
- Bluemel, S., Menne, D., Milos, G., Goetze, O., Fried, M., Schwizer, W., Fox, M., & Steingoetter, A. (2017). Relationship of body weight with gastrointestinal motor and sensory function: Studies in anorexia nervosa and obesity. *BMC Gastroenterology*, *17*(1), 4. https://doi.org/10.1186/s12876-016-0560-y
- Boyd, C., Abraham, S., & Kellow, J. (2005). Psychological features are important predictors of functional gastrointestinal disorders in patients with eating disorders. *Scandinavian Journal of Gastroenterology*, 40(8), 929–935. https://doi.org/10.1080/ 00365520510015836
- Boyd, C., Abraham, S., & Kellow, J. (2010). Appearance and disappearance of functional gastrointestinal disorders in patients with eating disorders. *Neurogastroenterology and Motility*, 22(12), 1279–1283. https://doi.org/10.1111/j.1365-2982.2010. 01576.x
- Brownley, K. A., Berkman, N. D., Peat, C. M., Lohr, K. N., Cullen, K. E., Bann, C. M., & Bulik, C. M. (2016). Binge-eating disorder in adults: A systematic review and meta-analysis. *Annals of Internal Medicine*, 165(6), 409–420. https://doi.org/10.7326/ m15-2455
- Castillo, M., & Weiselberg, E. (2017). Bulimia nervosa/Purging disorder. Current Problems in Pediatric and Adolescent Health Care, 47(4), 85–94. https://doi.org/10.1016/j.cppeds.2017.02. 004

- HANEL ET AL.
- Choi, Y. J., Kim, N., Yoon, H., Shin, C. M., Park, Y. S., Kim, J. W., Kim, Y. S., Lee, D. H., & Jung, H. C. (2017). Overlap between irritable bowel syndrome and functional dyspepsia including subtype analyses. *Journal of Gastroenterology and Hepatology*, 32(9), 1553–1561. https://doi.org/10.1111/jgh.13756
- Cremonini, F., Camilleri, M., Clark, M. M., Beebe, T. J., Locke, G. R., Zinsmeister, A. R., & Talley, N. J. (2009). Associations among binge eating behavior patterns and gastrointestinal symptoms: A population-based study. *International Journal of Obesity*, 33(3), 342–353. https://doi.org/10.1038/ijo.2008.272
- Cristina, S., Sinan, S., & Stefano, M. (2019). The relationship between the serotonin metabolism, gut-microbiota and the gutbrain axis. *Current Drug Metabolism*, 20(8), 646–655. https:// doi.org/10.2174/1389200220666190725115503
- Crowell, M. D., Cheskin, L. J., & Musial, F. (1994). Prevalence of gastrointestinal symptoms in obese and normal-weight binge eaters. *American Journal of Gastroenterology*, 89(3), 387–391.
- Cuntz, U., Enck, P., Fruhauf, E., Lehnert, P., Riepl, R. L., Fichter, M. M., & Otto, B. (2013). Cholecystokinin revisited: CCK and the hunger trap in anorexia nervosa. *Plos One*, *8*(1), 7. https:// doi.org/10.1371/journal.pone.0054457
- Dejong, H., Perkins, S., Grover, M., & Schmidt, U. (2011). The prevalence of irritable bowel syndrome in outpatients with bulimia nervosa. *International Journal of Eating Disorders*, 44(7), 661–664. https://doi.org/10.1002/eat.20901
- Diamanti, A., Bracci, F., Gambarara, M., Ciofetta, G. C., Sabbi, T., Ponticelli, A., Montecchi, F., Marinucci, S., Bianco, G., & Castro, M. (2003). Gastric electric activity assessed by electrogastrography and gastric emptying scintigraphy in adolescents with eating disorders. *Journal of Pediatric Gastroenterology and Nutrition*, 37(1), 35–41. https://doi.org/ 10.1097/00005176-200307000-00006
- DiBaise, J. K., Patel, N., Noelting, J., Dueck, A. C., Roarke, M., & Crowell, M. D. (2016). The relationship among gastroparetic symptoms, quality of life, and gastric emptying in patients referred for gastric emptying testing. *Neuro-Gastroenterology* and Motility, 28(2), 234–242. https://doi.org/10.1111/nmo. 12718
- Dorn, S. D., Palsson, O. S., Thiwan, S. I., Kanazawa, M., Clark, W. C., van Tilburg, M. A., Drossman, D. A., Scarlett, Y., Levy, R. L., Ringel, Y., Crowell, M. D., Olden, K. W., & Whitehead, W. E. (2007). Increased colonic pain sensitivity in irritable bowel syndrome is the result of an increased tendency to report pain rather than increased neurosensory sensitivity. *Gut*, 56(9), 1202–1209. https://doi.org/10.1136/gut.2006.117390
- Drossman, D. A. (1995). Diagnosing and treating patients with refractory functional gastrointestinal disorders. *Annals of Internal Medicine*, 123(9), 688–697. https://doi.org/10.7326/0003-4819-123-9-199511010-00008
- Drossman, D. A., Tack, J., Ford, A. C., Szigethy, E., Törnblom, H., & Van Oudenhove, L. (2018). Neuromodulators for functional gastrointestinal disorders (disorders of gut-brain interaction): A rome foundation working team report. *Gastroenterology*, 154(4), 1140–1171. https://doi.org/10.1053/j.gastro.2017.11.279
- Enck, P., Aziz, Q., Barbara, G., Farmer, A. D., Fukudo, S., Mayer, E. A., Spiller, R. C., Quigley, E. M. M., Rajilić-Stojanović, M., Schemann, M., Schwille-Kiuntke, J., Simren, M., Zipfel, S., & Spiller, R. C. (2016). Irritable bowel syndrome. *Nature Reviews Disease Primers*, 2, 16014. https://doi.org/10.1038/nrdp.2016.14

Enck, P., Azpiroz, F., Boeckxstaens, G., Elsenbruch, S., Feinle-Bisset, C., Holtmann, G., Lackner, J. M., Ronkainen, J., Schemann, M., Stengel, A., Tack, J., Zipfel, S., & Talley, N. J. (2017). Functional dyspepsia. *Nature Reviews Disease Primers*, *3*, 17081. https://doi.org/10.1038/nrdp.2017.81

Erzegovesi, S., & Bellodi, L. (2016). Eating disorders. *CNS Spectrums*, *21*(4), 304–309. https://doi.org/10.1017/s109285 2916000304

- Farzaei, M. H., Bahramsoltani, R., Abdollahi, M., & Rahimi, R. (2016). The role of visceral hypersensitivity in irritable bowel syndrome: Pharmacological targets and novel treatments. *Journal of Neurogastroenterology and Motility*, 22(4), 558–574. https://doi.org/10.5056/jnm16001
- Fichter, M. M., & Quadflieg, N. (2016). Mortality in eating disorders results of a large prospective clinical longitudinal study. *International Journal of Eating Disorders*, 49(4), 391–401. https://doi.org/10.1002/eat.22501
- Galmiche, M., Déchelotte, P., Lambert, G., & Tavolacci, M. P. (2019). Prevalence of eating disorders over the 2000–2018 period: A systematic literature review. *American Journal of Clinical Nutrition*, 109(5), 1402–1413. https://doi.org/10.1093/ ajcn/nqy342
- Grad, S., & Dumitrascu, D. L. (2020). Irritable bowel syndrome subtypes: New names for old medical conditions. *Digestive Diseases*, 38(2), 122–127. https://doi.org/10.1159/000505287
- Guerdjikova, A. I., O'Melia, A., Riffe, K., Palumbo, T., & McElroy, S. L. (2012). Bulimia nervosa presenting as rectal purging and rectal prolapse: Case report and literature review. *International Journal of Eating Disorders*, 45(3), 456–459. https://doi.org/10. 1002/eat.20959
- Guthrie, E. A., Creed, F. H., & Whorwell, P. J. (1990). Eating disorders in patients with the irritable bowel syndrome: A comparison with inflammatory bowel disease and peptic ulceration. *European Journal of Gastroenterology and Hepatology*, 2(6), 471–473.
- Hainer, V., Kabrnova, K., Aldhoon, B., Kunesova, M., & Wagenknecht, M. (2006). Serotonin and norepinephrine reuptake inhibition and eating behavior. *Annals of the New York Academy of Sciences*, 1083(1), 252–269. https://doi.org/10. 1196/annals.1367.017
- Haleem, D. J. (2017). Improving therapeutics in anorexia nervosa with tryptophan. *Life Sciences*, 178, 87–93. https://doi.org/10. 1016/j.lfs.2017.04.015
- Hanachi, M., Manichanh, C., Schoenenberger, A., Pascal, V., Levenez, F., Cournede, N., Melchior, J. C., & Melchior, J.-C. (2019). Altered host-gut microbes symbiosis in severely malnourished anorexia nervosa (AN) patients undergoing enteral nutrition: An explicative factor of functional intestinal disorders? *Clinical Nutrition*, 38(5), 2304–2310. https://doi.org/ 10.1016/j.clnu.2018.10.004
- Heruc, G. A., Little, T. J., Kohn, M., Madden, S., Clarke, S., Horowitz, M., & Feinle-Bisset, C. (2019). Appetite perceptions, gastrointestinal symptoms, ghrelin, peptide YY and state anxiety are disturbed in adolescent females with anorexia nervosa and only partially restored with short-term refeeding. *Nutrients*, 11(1), 16. https://doi.org/10.3390/nu11010059
- Hillilä, M. T., & Färkkilä, M. A. (2004). Prevalence of irritable bowel syndrome according to different diagnostic criteria in a nonselected adult population. *Alimentary Pharmacology and*

Therapeutics, *20*(3), 339–345. https://doi.org/10.1111/j.1365-2036.2004.02034.x

- Holtmann, G., Shah, A., & Morrison, M. (2017). Pathophysiology of functional gastrointestinal disorders: A holistic overview. *Digestive Diseases*, 35(Suppl 1), 5–13. https://doi.org/10.1159/ 000485409
- Hutson, W. R., & Wald, A. (1990). Gastric emptying in patients with bulimia nervosa and anorexia nervosa. *American Journal of Gastroenterology*, 85(1), 41–46.
- Javaras, K. N., Pope, H. G., Lalonde, J. K., Roberts, J. L., Nillni, Y. I., Laird, N. M., Bulik, C. M., Crow, S. J., McElroy, S. L., & Walsh, B. T. (2008). Co-occurrence of binge eating disorder with psychiatric and medical disorders. *Journal of Clinical Psychiatry*, 69(2), 266–273. https://doi.org/10.4088/JCP.v69n0213
- Jerndal, P., Ringström, G., Agerforz, P., Karpefors, M., Akkermans, L. M., Bayati, A., & Simrén, M. (2010). Gastrointestinal-specific anxiety: An important factor for severity of GI symptoms and quality of life in IBS. *Neurogastroenterology and Motility*, 22(6), 646–e179. https://doi.org/10.1111/j.1365-2982.2010.01493.x
- Karamanolis, G., Caenepeel, P., Arts, J., & Tack, J. (2007). Determinants of symptom pattern in idiopathic severely delayed gastric emptying: Gastric emptying rate or proximal stomach dysfunction? *Gut*, 56(1), 29–36. https://doi.org/10.1136/gut. 2005.089508
- Kessler, U., Rekkedal, G. A., Ro, O., Berentsen, B., Steinsvik, E. K., Lied, G. A., & Danielsen, Y. (2020). Association between gastrointestinal complaints and psychopathology in patients with anorexia nervosa. *International Journal of Eating Disorders*, 53, 802–806. https://doi.org/10.1002/eat.23243
- Keys, A., Brožek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The biology of human starvation*. (Vol. 2). University of Minnesota Press.
- Koch, K. L., Bingaman, S., Tan, L., & Stern, R. M. (1998). Visceral perceptions and gastric myoelectrical activity in healthy women and in patients with bulimia nervosa. *Neuro*gastroenterology and Motility, 10(1), 3–10. https://doi.org/10. 1046/j.1365-2982.1998.00080.x
- Lacy, B. E., & Patel, N. K. (2017). Rome criteria and a diagnostic approach to irritable bowel syndrome. *Journal of Clinical Medicine*, 6(11), 99. https://doi.org/10.3390/jcm6110099
- Levy, R. L., Linde, J. A., Feld, K. A., Crowell, M. D., & Jeffery, R. W. (2005). The association of gastrointestinal symptoms with weight, diet, and exercise in weight-loss program participants. *Clinical Gastroenterology and Hepatology*, *3*(10), 992–996. https://doi.org/10.1016/s1542-3565(05)00696-8
- Lobera, I. J., Santed, M. A., & Rios, P. B. (2011). Impact of functional dyspepsia on quality of life in eating disorder patients: The role of thought-shape fusion. *Nutricion Hospitalaria*, 26(6), 1363–1371. https://doi.org/10.3305/nh.2011.26.6.5288
- Mack, I., Cuntz, U., Grämer, C., Niedermaier, S., Pohl, C., Schwiertz, A., Zipfel, S., Enck, P., & Penders, J. (2016). Weight gain in anorexia nervosa does not ameliorate the faecal microbiota, branched chain fatty acid profiles, and gastrointestinal complaints. *Scientific Reports*, 6, 26752. https://doi.org/10.1038/ srep26752
- Manning, A. P., Thompson, W. G., Heaton, K. W., & Morris, A. F. (1978). Towards positive diagnosis of the irritable bowel. *British Medical Journal*, 2(6138), 653–654. https://doi.org/10. 1136/bmj.2.6138.653

- Mohammad-Zadeh, L. F., Moses, L., & Gwaltney-Brant, S. M. (2008). Serotonin: A review. Journal of Veterinary Pharmacology and Therapeutics, 31(3), 187–199. https://doi.org/10. 1111/j.1365-2885.2008.00944.x
- Norris, M. L., Harrison, M. E., Isserlin, L., Robinson, A., Feder, S., & Sampson, M. (2016). Gastrointestinal complications associated with anorexia nervosa: A systematic review. *International Journal of Eating Disorders*, 49(3), 216–237. https://doi.org/10. 1002/eat.22462
- Oshima, T., & Miwa, H. (2015). Epidemiology of functional gastrointestinal disorders in Japan and in the world. *Journal of Neurogastroenterology and Motility*, *21*(3), 320–329. https://doi.org/10.5056/jnm14165
- Pasricha, P. J., Colvin, R., Yates, K., Hasler, W. L., Abell, T. L., Unalp-Arida, A., Farrugia, G., Koch, K. L., Parkman, H. P., Snape, W. J., Lee, L., Tonascia, J., & Hamilton, F. (2011). Characteristics of patients with chronic unexplained nausea and vomiting and normal gastric emptying. *Clinical Gastroenterology and Hepatology*, 9(7), 567–576. https://doi.org/10. 1016/j.cgh.2011.03.003
- Perez, M. E., Coley, B., Crandall, W., Di Lorenzo, C., & Bravender, T. (2013). Effect of nutritional rehabilitation on gastric motility and somatization in adolescents with anorexia. *The Journal of Pediatrics*, 163(3), 867–872.e1. https://doi.org/10.1016/j.jpeds. 2013.03.011
- Perkins, S. J., Keville, S., Schmidt, U., & Chalder, T. (2005). Eating disorders and irritable bowel syndrome: Is there a link? *Journal of Psychosomatic Research*, 59(2), 57–64. https://doi.org/10. 1016/j.jpsychores.2004.04.375
- Porcelli, P., Leandro, G., & De Carne, M. (1998). Functional gastrointestinal disorders and eating disorders - relevance of the association in clinical management. *Scandinavian Journal* of *Gastroenterology*, 33(6), 577–582. https://doi.org/10.1080/ 00365529850171819
- Salvioli, B., Pellicciari, A., Iero, L., Di Pietro, E., Moscano, F., Gualandi, S., De Giorgio, R., Ruggeri, E., & Franzoni, E. (2013). Audit of digestive complaints and psychopathological traits in patients with eating disorders: A prospective study. *Digestive and Liver Disease*, 45(8), 639–644. https://doi.org/10. 1016/j.dld.2013.02.022
- Santonicola, A., Angrisani, L., Ciacci, C., & Iovino, P. (2013). Prevalence of functional gastrointestinal disorders according to Rome III criteria in Italian morbidly obese patients. *Scientific World Journal*, 2013, 532503. https://doi.org/10.1155/ 2013/532503
- Santonicola, A., Siniscalchi, M., Capone, P., Gallotta, S., Ciacci, C., & Iovino, P. (2012). Prevalence of functional dyspepsia and its subgroups in patients with eating disorders. *World Journal* of Gastroenterology, 18(32), 4379–4385. https://doi.org/10. 3748/wjg.v18.i32.4379
- Sato, Y., & Fukudo, S. (2015). Gastrointestinal symptoms and disorders in patients with eating disorders. *Clinical Journal of Gastroenterology*, 8(5), 255–263. https://doi.org/10.1007/s12328-015-0611-x
- Sherman, P., Leslie, K., Golderg, E., MacMillan, J., Hunt, R., & Ernst, P. (1993). Helicobacter pylori infection in adolescents with eating disorders and dyspeptic symptoms. *The Journal of Pediatrics*, 122(5 Pt 1), 824–826. https://doi.org/10.1016/s0022-3476(06)80037-5

- Simrén, M., Törnblom, H., Palsson, O. S., van Tilburg, M. A. L., Van Oudenhove, L., Tack, J., & Whitehead, W. E. (2018). Visceral hypersensitivity is associated with GI symptom severity in functional GI disorders: Consistent findings from five different patient cohorts. *Gut*, 67(2), 255–262. https://doi.org/10.1136/ gutjnl-2016-312361
- Singh, P., Agnihotri, A., Pathak, M. K., Shirazi, A., Tiwari, R. P., Sreenivas, V., & Makharia, G. K. (2012). Psychiatric, somatic and other functional gastrointestinal disorders in patients with irritable bowel syndrome at a tertiary care center. *Journal of Neurogastroenterology and Motility*, 18(3), 324–331. https://doi. org/10.5056/jnm.2012.18.3.324
- Smink, F. R., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, 14(4), 406–414. https://doi.org/10. 1007/s11920-012-0282-y
- Sperber, A. D., Bangdiwala, S. I., Drossman, D. A., Ghoshal, U. C., Simren, M., Tack, J., & Palsson, O. S. (2020). Worldwide prevalence and burden of functional gastrointestinal disorders, results of Rome foundation global study. *Gastroenterology*. https://doi.org/10.1053/j.gastro.2020.04.014
- Spillebout, A., Dechelotte, P., Ladner, J., & Tavolacci, M. P. (2019). Mental health among university students with eating disorders and irritable bowel syndrome in France. *Revue D' Épidémiologie Et De Santé Publique*, 67(5), 295–301. https://doi. org/10.1016/j.respe.2019.04.056
- Stanghellini, V., Chan, F. K., Hasler, W. L., Malagelada, J. R., Suzuki, H., Tack, J., & Talley, N. J. (2016). Gastroduodenal disorders. *Gastroenterology*, 150(6), 1380–1392. https://doi.org/ 10.1053/j.gastro.2016.02.011
- Steiger, H. (2004). Eating disorders and the serotonin connection: State, trait and developmental effects. *Journal of Psychiatry & Neuroscience*, 29(1), 20–29.
- Suzuki, H., & Hibi, T. (2011). Overlap syndrome of functional dyspepsia and irritable bowel syndrome - are both diseases mutually exclusive? *Journal of Neurogastroenterology and Motility*, *17*(4), 360–365. https://doi.org/10.5056/jnm.2011.17.4.360
- Tack, J., & Drossman, D. A. (2017). What's new in Rome IV? Neurogastroenterology and Motility, 29(9), e13053. https://doi. org/10.1111/nmo.13053
- Tack, J., & Talley, N. J. (2013). Functional dyspepsia--symptoms, definitions and validity of the Rome III criteria. *Nature Reviews Gastroenterology and Hepatology*, 10(3), 134–141. https://doi. org/10.1038/nrgastro.2013.14
- Talley, N. J., Stanghellini, V., Heading, R. C., Koch, K. L., Malagelada, J. R., & Tytgat, G. N. (1999). Functional gastroduodenal disorders. *Gut*, 45 (Suppl 2), II37-42. https://doi.org/10.1136/ gut.45.2008.ii37
- Tang, T. N., Toner, B. B., Stuckless, N., Dion, K. L., Kaplan, A. S., & Ali, A. (1998). Features of eating disorders in patients with irritable bowel syndrome. *Journal of Psychosomatic Research*, 45(2), 171–178. https://doi.org/10.1016/S0022-3999%2897% 2900300-0
- Tong, J., & D'Alessio, D. (2011). Eating disorders and gastrointestinal peptides. *Current Opinion in Endocrinology Diabetes and Obesity*, 18(1), 42–49. https://doi.org/10.1097/MED.0b013 e328341e12b
- Van Oudenhove, L., & Aziz, Q. (2013). The role of psychosocial factors and psychiatric disorders in functional dyspepsia.

Nature Reviews Gastroenterology and Hepatology, 10(3), 158–167. https://doi.org/10.1038/nrgastro.2013.10

- Van Oudenhove, L., Levy, R. L., Crowell, M. D., Drossman, D. A., Halpert, A. D., Keefer, L., Murphy, T. B., & Naliboff, B. D. (2016). Biopsychosocial aspects of functional gastrointestinal disorders: How central and environmental processes contribute to the development and expression of functional gastrointestinal disorders. *Gastroenterology*, 150(6), 1355–1367. e2. https://doi.org/10.1053/j.gastro.2016.02.027
- Van Oudenhove, L., Vandenberghe, J., Geeraerts, B., Vos, R., Persoons, P., Demyttenaere, K., Tack, J., & Tack, J. (2007). Relationship between anxiety and gastric sensorimotor function in functional dyspepsia. *Psychosomatic Medicine*, 69(5), 455–463. https://doi.org/10.1097/PSY.0b013e3180600a4a
- von Wulffen, M., Talley, N. J., Hammer, J., McMaster, J., Rich, G., Shah, A., Kendall, B. J., Jones, M., & Holtmann, G. (2019). Overlap of irritable bowel syndrome and functional dyspepsia in the clinical setting: Prevalence and risk factors. *Digestive Diseases and Sciences*, 64(2), 480–486. https://doi.org/10.1007/ s10620-018-5343-6
- Waldholtz, B. D., & Andersen, A. E. (1990). Gastrointestinal symptoms in anorexia nervosa. A prospective study. *Gastroenterology*, *98*(6), 1415–1419. https://doi.org/10.1016/0016-5085(90)91070-m
- Wang, X. J., Luscombe, G. M., Boyd, C., Kellow, J., & Abraham, S. (2014). Functional gastrointestinal disorders in eating disorder patients: Altered distribution and predictors using ROME III compared to ROME II criteria. *World Journal of Gastroenterology*, 20(43), 16293–16299. https://doi.org/10.3748/wjg.v20.i43.16293
- Weterle-Smolińska, K. A., Banasiuk, M., Dziekiewicz, M., Ciastoń, M., Jagielska, G., & Banaszkiewicz, A. (2015). Gastrointestinal motility disorders in patients with anorexia nervosa - a review of the literature. *Psychiatria Polska*, 49(4), 721–729. https://doi. org/10.12740/pp/35482
- Winkler, L. A.-D., Christiansen, E., Lichtenstein, M. B., Hansen, N. B., Bilenberg, N., & Støving, R. K. (2014). Quality of life in eating disorders: A meta-analysis. *Psychiatry Research*, 219(1), 1–9. https://doi.org/10.1016/j.psychres.2014.05.002
- Winstead, N. S., & Willard, S. G. (2006). Gastrointestinal complaints in patients with eating disorders. *Journal of Clinical Gastroenterology*, 40(8), 678–682. https://doi.org/10.1097/00004836-200609000-00003
- Yeh, H. W., Chien, W. C., Chung, C. H., Hu, J. M., & Tzeng, N. S. (2018). Risk of psychiatric disorders in irritable bowel syndrome - A nationwide, population-based, cohort study. *International Journal of Clinical Practice*, 72(7), 8. https://doi.org/ 10.1111/ijcp.13212
- Zipfel, S., Giel, K. E., Bulik, C. M., Hay, P., & Schmidt, U. (2015). Anorexia nervosa: Aetiology, assessment, and treatment. *Lancet Psychiatry*, 2(12), 1099–1111. https://doi.org/10.1016/ s2215-0366(15)00356-9

How to cite this article: Hanel, V., Schalla, M. A., & Stengel, A. (2021). Irritable bowel syndrome and functional dyspepsia in patients with eating disorders - a systematic review. *European Eating Disorders Review*, *29*(5), 692–719. <u>https://doi.org/</u>10.1002/erv.2847