



A Comprehensive Systematic Review of The Relationship Between Obesity and The Prevalence of Dyspepsia

¹ Yuliana Sintia, ² Abilio Noviandi Jung

¹ Gerbang Sehat Mahulu Regional General Hospital, Indonesia /
Graduate of Duta Wacana Christian University, Indonesia

² Mamahak Besar Regional General Hospital, Indonesia, Graduate of
Wijaya Kusuma University, Indonesia

Corresponding Email : yulianasintia26@gmail.com

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ABSTRACT

Introduction: The parallel rise in global obesity rates and the prevalence of functional gastrointestinal disorders, particularly dyspepsia and gastroesophageal reflux disease (GERD), presents a significant public health challenge. Understanding the nature and strength of this association is crucial for effective clinical management and public health strategies (Eslick, 2012; Mahadeva, 2023).

Methods: A comprehensive review was conducted by screening studies from multiple databases. Studies were included if they investigated the obesity-dyspepsia association as a primary objective, used validated measures for both obesity (e.g., BMI, waist circumference) and dyspepsia (e.g., Rome criteria, clinical diagnosis), involved adult populations, and reported quantitative data. Data on study characteristics, obesity measures, dyspepsia definitions, population demographics, association

findings, proposed mechanisms, and limitations were systematically extracted from 59 included sources.

Results: The evidence demonstrates a consistent positive association between obesity—particularly central adiposity—and dyspepsia/GERD. Meta-analyses show overweight individuals (BMI 25-30 kg/m²) have an odds ratio (OR) of 1.43-1.60 for GERD, rising to 1.94-2.15 for those with obesity (BMI >30 kg/m²) (Hampel, Abraham, & El-Serag, 2005; Corley & Kubo, 2006; Cai et al., 2012). Central obesity measures like waist circumference show even stronger correlations (Zhan et al., 2021). However, findings are heterogeneous, with associations attenuated when controlling for diet and physical activity (Levy et al., 2005), and weaker or absent in some ethnic groups, particularly Asian populations (Goh, 2007).

Discussion: The relationship is mediated by multiple interconnected pathophysiological mechanisms, including mechanical effects (increased intra-abdominal pressure, lower esophageal sphincter dysfunction), inflammatory pathways, hormonal alterations, and visceral hypersensitivity (Mathus-Vliegen & Tytgat, 2002; Al Mushref & Srinivasan, 2013; Doerfler et al., 2020). Weight loss of at least 10% significantly improves symptoms and reduces medication need (de Bortoli et al., 2016), while proton pump inhibitor (PPI) efficacy appears unaffected by BMI (Sharma et al., 2013; Peura et al., 2011).

Conclusion: Obesity, especially central adiposity, is a significant modifiable risk factor for dyspepsia and

GERD. Clinical management should prioritize weight loss and waist circumference measurement alongside conventional therapy. Future research should focus on ethnic-specific mechanisms and the role of lifestyle confounders.

Keywords: Obesity, Dyspepsia, Gastroesophageal Reflux Disease (GERD), Body Mass Index (BMI), Central Adiposity, Pathophysiology.

INTRODUCTION

Background

The global epidemics of obesity and functional gastrointestinal disorders represent two major intersecting public health concerns. Dyspepsia, characterized by epigastric pain or discomfort, and gastroesophageal reflux disease (GERD), defined by troublesome heartburn and regurgitation, are among the most prevalent digestive conditions worldwide, significantly impairing quality of life and incurring substantial healthcare costs (Karimian et al., 2021; Eusebi et al., 2017). Concurrently, obesity rates have tripled since 1975, with over 650 million adults now classified as obese (WHO, 2021). The co-occurrence of these conditions is frequently observed in clinical practice, prompting extensive investigation into a potential causal or associative link. The pathophysiological plausibility stems from obesity's mechanical effects on gastroesophageal anatomy and pressure gradients, as well as its systemic metabolic and inflammatory consequences (Falk, 2008; Mahadeva, 2023).

Research Gap

Despite numerous studies, the evidence remains heterogeneous. While many cross-sectional and case-control studies report a strong positive association (Hampel, Abraham, & El-Serag, 2005; Foster et al., 2003), others, particularly those adjusting for lifestyle factors like diet and physical activity, find attenuated or null effects (Levy et al., 2005). Furthermore, significant geographic and ethnic variations exist, with weaker associations reported in Asian compared to Western populations (Goh, 2007; Deng & Wong, 2023). There is also a relative paucity of studies focusing specifically on functional dyspepsia (FD) as distinct from GERD, with most literature conglomerating upper GI symptoms or focusing solely on reflux. The relative importance of general adiposity (BMI) versus central/visceral adiposity also requires clearer delineation for clinical risk stratification.

Novelty

This review synthesizes evidence from a wide array of study designs, including recent Mendelian randomization analyses that strengthen causal inference (Deng & Wong, 2023; Mao et al., 2025). It places particular emphasis on disentangling the effects of obesity itself from confounding lifestyle behaviors, examining ethnic disparities in the association, and evaluating the differential impact of various adiposity measures. Furthermore, it comprehensively reviews not just epidemiological associations but also underlying mechanisms and implications for treatment response, including pharmacological management, weight loss interventions, and surgical outcomes.

Hypothesis

It is hypothesized that obesity, particularly visceral adiposity, is independently and causally associated with an increased prevalence and severity of dyspepsia and GERD, mediated through mechanical, inflammatory, hormonal, and neural pathways. This association is expected to be modified by ethnicity, lifestyle factors, and the specific anthropometric measure used.

Research Objectives

1. To systematically review and quantify the association between obesity (measured by BMI, waist circumference, etc.) and the prevalence of dyspepsia/GERD.
2. To compare the strength of association using different measures of adiposity (general vs. central).
3. To explore proposed biological mechanisms linking obesity to upper gastrointestinal symptoms.
4. To examine the impact of obesity on treatment outcomes for dyspepsia/GERD, including PPI response and surgical results.
5. To identify sources of heterogeneity in the literature, including ethnic variations and confounding by lifestyle.

Research Benefits

The findings of this review will: 1) inform clinicians about obesity as a key modifiable risk factor in patients with dyspepsia, guiding management priorities; 2) provide evidence for public health strategies aimed at preventing upper GI disorders through weight management; 3) highlight subgroups (e.g., those with central obesity, specific ethnicities) for targeted intervention; and 4) identify gaps in the current literature to direct future research efforts.

METHODS

Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

Criteria for Eligibility

This systematic review aims to evaluate The Relationship Between Obesity and The Prevalence of Dyspepsia.

Screening

We screened in sources based on their abstracts that met these criteria:

- **Research Focus:** Does this study investigate the association between obesity and dyspepsia as a primary research objective?
- **Obesity Measurement:** Does this study use clearly defined and validated obesity measures (such as BMI ≥ 30 kg/m², waist circumference, or other validated anthropometric measures)?
- **Dyspepsia Diagnosis:** Does this study use validated dyspepsia diagnosis methods (such as clinical diagnosis, Rome criteria, or standardized questionnaires)?
- **Study Design:** Is this study a cross-sectional, case-control, cohort study, systematic review, or meta-analysis?

- **Population Age:** Does this study involve adult participants (≥ 18 years of age)?
- **Quantitative Data:** Does this study report quantitative measures of association or prevalence data related to the obesity-dyspepsia relationship?
- **Study Type Quality:** Is this study NOT a case report, case series, editorial, or opinion piece?
- **Primary vs Incidental Focus:** Are obesity and/or dyspepsia examined as primary variables of interest (rather than only mentioned incidentally or as secondary findings)?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

Search Strategy

The keywords used for this research based PICO :

Element	P (Population)	I (Intervention/Exposure)	C (Comparison/Context)	O (Outcome)
Keyword 1	Adult	Obesity	Non-obese	Dyspepsia
Keyword 2	Patient	Body Mass Index	Normal BMI	Gastroesophageal reflux disease
Keyword 3	Human	Central obesity	Normal weight	Gastrointestinal symptoms
Keyword 4	Clinical population	Weight	Healthy weight	Prevalence

The Boolean MeSH keywords inputted on databases for this research are: ("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")

Data extraction

- **Obesity Measures:**

Extract all measures of obesity/body composition used in the study including:

- BMI categories and cutoff points used
- Waist circumference measurements and cutoffs
- Waist-to-hip ratio
- Central/abdominal obesity definitions
- Visceral adiposity measures
- Any other anthropometric measurements
- How these were measured (self-reported vs measured)
- Mean values and ranges for each group

- **Dyspepsia Definition:**

Extract how dyspepsia and related GI disorders were defined and diagnosed including:

- Specific diagnostic criteria used (Rome criteria, clinical diagnosis, etc.)
- Type of dyspepsia (functional dyspepsia, organic dyspepsia, GERD-related, etc.)
- Symptom definitions (heartburn, epigastric pain, early satiety, etc.)
- Diagnostic methods (questionnaires, endoscopy, clinical assessment)
- Severity classifications used
- Duration requirements for diagnosis
- Any validated instruments used (name and details)

- **Population Characteristics:**

Extract key demographic and clinical characteristics that may modify the obesity-dyspepsia relationship including:

- Age (mean, range, categories)
- Gender distribution and any gender-specific analyses
- Ethnicity/race and ethnic-specific findings
- Geographic location/country
- Clinical setting (primary care, specialty clinic, population-based)
- Comorbidities that may affect results
- Any subgroup analyses performed

- **Relationship Findings:**

Extract specific quantitative results about the obesity-dyspepsia relationship including:

- Prevalence rates of dyspepsia by obesity categories
- Odds ratios or risk ratios with 95% confidence intervals
- Dose-response relationships across BMI categories
- Statistical significance (p-values)
- Adjusted vs unadjusted analyses
- Variables controlled for in multivariate analyses
- Any null findings or lack of association
- Strength of association descriptors

- **Study Design:**

Extract study methodology details including:

- Study design (cross-sectional, case-control, cohort, etc.)
- Sample size and power calculations
- Recruitment methods and setting
- Inclusion and exclusion criteria
- Response rates and loss to follow-up

- Time period of study conduct
- Funding source and conflicts of interest
- Whether this is primary research or secondary analysis

- **Mechanisms Proposed:**

Extract any proposed biological or pathophysiological mechanisms linking obesity to dyspepsia including:

- Mechanical effects (increased intragastric pressure, gastroesophageal pressure gradient)
- Inflammatory pathways (low-grade inflammation, cytokines)
- Hormonal effects (leptin, ghrelin, other gut hormones)
- Gut microbiome changes
- Gastric motility effects
- Visceral hypersensitivity
- Any other proposed mechanisms
- Supporting evidence provided for mechanisms

- **Key Limitations:**

Extract study limitations and potential sources of bias including:

- Measurement limitations (self-reported data, recall bias)
- Selection bias issues
- Confounding variables not controlled for
- Temporal relationship unclear (cross-sectional designs)
- Generalizability concerns
- Sample size limitations
- Diagnostic accuracy concerns

- Any other methodological limitations noted by authors

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")	188
Semantic Scholar	("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")	1
Springer	("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")	26,066
Google Scholar	("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")	18,800
Wiley Online Library	("Adult" OR "Patient" OR "Human" OR "Clinical population") AND ("Obesity" OR "Body Mass Index" OR "Central obesity" OR "Weight") AND ("Non-obese" OR "Normal BMI" OR "Normal weight" OR "Healthy weight") AND ("Dyspepsia" OR "Gastroesophageal reflux disease" OR "Gastrointestinal" OR "Prevalence")	27,778

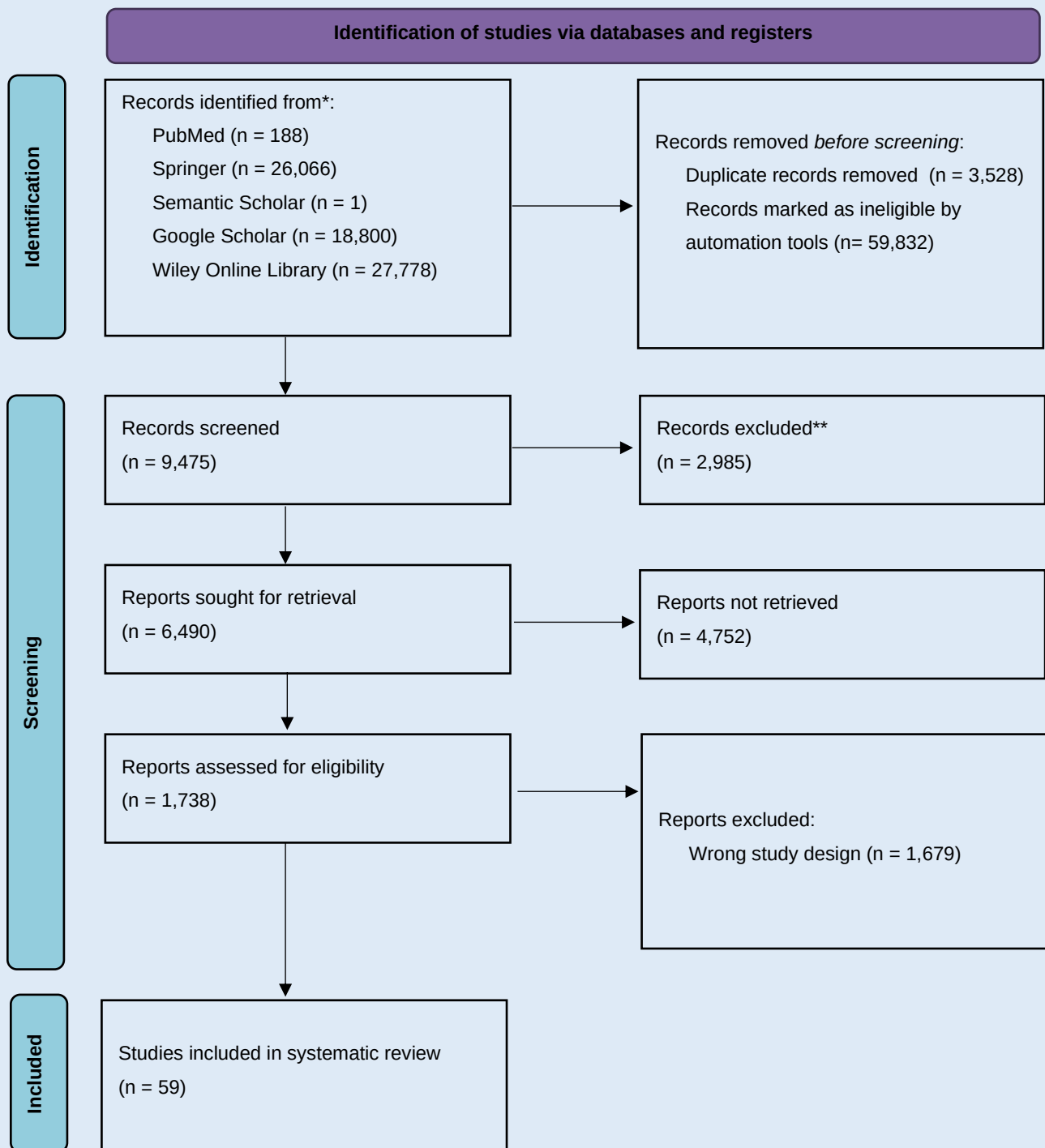


Figure 1. Article search flowchart

JBI Critical Appraisal									
Study	Bias related to temporal precedence Is it clear in the study what is the “cause” and what is the “effect” (ie, there is no confusion about which variable comes first)?	Bias related to selection and allocation Was there a control group?	Bias related to confounding factors Were participants included in any comparisons similar?	Bias related to administration of intervention/exposure Were the participants included in any comparisons receiving similar treatment/care, other than the exposure or intervention of interest?	Were there multiple measurements of the outcome, both pre and post the intervention/exposure?	Were the outcomes of participants included in any comparisons measured in the same way?	Were outcomes measured in a reliable way?	Bias related to participant retention Was follow-up complete and, if not, were differences between groups in terms of their follow-up adequately described and analyzed?	Statistical conclusion validity Was appropriate statistical analysis used?

H. Hampel et al., 2005									
L. Eusebi et al., 2017									
G. Eslick et al., 2012									
S. Mahadeva et al., 2023									
Junyi Zhan et al., 2021									
R. Levy et al., 2005									
F. Mazzoleni et al., 2019									
R. Clements et al., 2003									

A. Foster et al., 2003	✓	✓	✓	✗	✓	✗	✓	✓	✓
Z. Gökteş et al., 2016	✓	✓	✓	✗	✓	✗	✓	✓	✓
T. Taft et al., 2021	✓	✓	✓	✗	✓	✗	✓	✓	✓
K. Goh et al., 2007	✓	✓	✓	✗	✓	✗	✓	✓	✓
G. Falk et al., 2008	✓	✓	✓	✗	✓	✗	✓	✓	✓
Philip O Katz et al., 2013	✓	✓	✓	✗	✓	✗	✓	✓	✓
Prateek Sharma et al., 2007	✓	✓	✓	✗	✓	✗	✓	✓	✓
N. de Bortoli et al., 2016	✓	✓	✓	✗	✓	✗	✓	✓	✓
J. Fletcher et al., 2010	✓	✓	✓	✗	✓	✗	✓	✓	✓
H. Suto et al., 2009	✓	✓	✓	✗	✓	✗	✓	✓	✓

Yunyang Deng et al., 2023									
D. Peura et al., 2011									
B. Doerfler et al., 2020									
D. Corley et al., 2006									
N. Bortoli et al., 2014									
Beata Moczulska et al., 2019									
Ting Li et al., 2022									
Mazen Al Mushref et al., 2013									
Vivien Hanel et al., 2021									

E. M. H. Mathus-Vliegen et al., 2002									
M. Mohammad i et al., 2016									
Priyata Dutta et al., 2024									
Yiqing Mao et al., 2025									
M. Mokhtare et al., 2017									
S. Fu et al., 2021									
Min Seo Kim et al., 2024									
Prateek Sharma et al., 2013									
V. Meineche-									

schmidt et al., 2000									
A. Kurmangulov et al., 2018	✓	✓	✓	✗	✓	✗	✓	✓	✓
Abdelnaser Elzouki et al., 2021	✓	✓	✓	✗	✓	✗	✓	✓	✓
Maria Aparecida A Serra et al., 2025	✓	✓	✓	✗	✓	✗	✓	✓	✓
B. Jacobson et al., 2007	✓	✓	✓	✗	✓	✗	✓	✓	✓
T. C. Крелевец et al., 2016	✓	✓	✓	✗	✓	✗	✓	✓	✓
Ridho Prip et al., 2025	✓	✓	✓	✗	✓	✗	✓	✓	✓
M. Karimian et al., 2021	✓	✓	✓	✗	✓	✗	✓	✓	✓

Song Lin et al., 2019									
N. Cai et al., 2012									
T. Abdelrahm an et al., 2018									
M. Y. Zak et al., 2025									
B. Kendall et al., 2015									
P. Chhabra et al., 2019									
Y. Bashir et al., 2019									
M. Küper et al., 2009									
J. Lagergren et al.,									

2007									
Ruzica- Rosalia Luketina et al., 2015	✓	✓	✓	✗	✓	✗	✓	✓	✓
M. Memon et al., 2025	✓	✓	✓	✗	✓	✗	✓	✓	✓
Faisal Nadeem et al., 2024	✓	✓	✓	✗	✓	✗	✓	✓	✓
S. Bruley des Varannes et al., 2013	✓	✓	✓	✗	✓	✗	✓	✓	✓
O. Şen et al., 2022	✓	✓	✓	✗	✓	✗	✓	✓	✓
Patrycja Wachowska -Kelly et al., 2014	✓	✓	✓	✗	✓	✗	✓	✓	✓
Марина Михайловн а Романова et al.,	✓	✓	✓	✗	✓	✗	✓	✓	✓

2012									
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RESULTS

Characteristics of Included Studies

The 59 sources included in this review comprise a diverse range of study designs examining the relationship between obesity and dyspepsia or related gastrointestinal disorders. The majority of studies focused on gastroesophageal reflux disease (GERD) as the primary outcome, with several also examining functional dyspepsia, erosive esophagitis, and other upper gastrointestinal symptoms.

Study	Primary Outcome	Population
H. Hampel et al., 2005	GERD symptoms, erosive esophagitis	Cross-sectional and case-control studies
L. Eusebi et al., 2017	Gastro-oesophageal reflux symptoms	460,984 subjects from 108 populations
G. Eslick et al., 2012	Gastrointestinal symptoms	Not specified
S. Mahadeva et al., 2023	Functional gastrointestinal disorders	Population-based and primary care
Junyi Zhan et al., 2021	Reflux esophagitis	42 observational studies
R. Levy et al., 2005	GI symptoms including IBS	983 weight-loss program participants

Study	Primary Outcome	Population
F. Mazzoleni et al., 2019	Erosive esophagitis	404 H. pylori positive patients
R. Clements et al., 2003	GI symptoms post-bariatric surgery	43 morbidly obese patients
A. Foster et al., 2003	GI symptom clusters	43 obese vs 36 controls
Z. Göktaş et al., 2016	Functional dyspepsia	168 FD patients, 135 controls
T. Taft et al., 2021	GERD symptoms	66 patients with BMI ≥ 25
K. Goh et al., 2007	GERD and esophagitis	Asian populations
G. Falk et al., 2008	GERD-type symptoms	80,110 Kaiser-Permanente members
Philip O Katz et al., 2013	GERD diagnosis and management	Western and Asian populations
Prateek Sharma et al., 2007	Heartburn resolution in NERD	704 NERD patients
N. de Bortoli et al., 2016	GERD symptoms with weight loss	Overweight/obese GERD patients
J. Fletcher et al., 2010	Response to PPI	105 patients with normal endoscopy
H. Suto et al., 2009	BMI changes after H. pylori eradication	163 H. pylori-positive patients

Study	Primary Outcome	Population
Yunyang Deng et al., 2023	GERD	European populations
D. Peura et al., 2011	Heartburn in NERD and EE	3052 patients
B. Doerfler et al., 2020	GERD symptoms	22 PPI-dependent GERD patients
D. Corley et al., 2006	GERD	18,346 GERD patients
N. Bortoli et al., 2014	Erosive esophagitis	386 female patients
Beata Moczulska et al., 2019	Dyspepsia and glucose disorders	90 dyspeptic patients
Ting Li et al., 2022	GERD, erosive esophagitis, CAD	20 studies
Mazen Al Mushref et al., 2013	GI motility in obesity	Review of literature
Vivien Hanel et al., 2021	IBS and FD in eating disorders	36 publications
E. M. H. Mathus-Vliegen et al., 2002	Gastro-oesophageal reflux	42 obese patients
M. Mohammadi et al., 2016	GERD and metabolic syndrome	23,687 participants
Priyata Dutta et al., 2024	GERD and MASLD	Literature review

Study	Primary Outcome	Population
Yiqing Mao et al., 2025	GERD	484,219 participants from 43 papers
M. Mokhtare et al., 2017	Metabolic syndrome criteria	91 dyspeptic patients
S. Fu et al., 2021	GERD and metabolic syndrome	Not specified
Min Seo Kim et al., 2024	GI diseases	594 observational studies
Prateek Sharma et al., 2013	Heartburn and EE healing	11,731 patients
V. Meineche-schmidt et al., 2000	Omeprazole response in dyspepsia	471 patients
A. Kurmangulov et al., 2018	Obesity and dietary fiber	50 obese patients
Abdelnaser Elzouki et al., 2021	GERD after bariatric surgery	Not specified
Maria Aparecida A Serra et al., 2025	Glycemic parameters in dyspepsia	103 patients
B. Jacobson et al., 2007	PPI efficacy in erosive GERD	560 patients
T. C. Крoлeвeц et al., 2016	GERD in overweight patients	131 patients
Ridho Prip et al., 2025	GERD prevalence and severity	20 studies

Study	Primary Outcome	Population
M. Karimian et al., 2021	Dyspepsia prevalence	54,118 subjects
Song Lin et al., 2019	FD and depression	59,029 individuals
N. Cai et al., 2012	Erosive esophagitis	21 studies
T. Abdelrahman et al., 2018	LARS outcomes	Not specified
M. Y. Zak et al., 2025	Chronic pancreatitis in obesity	182 patients
B. Kendall et al., 2015	Barrett's esophagus	Case-control studies
P. Chhabra et al., 2019	Barrett's esophagus risk factors	1948 patients
Y. Bashir et al., 2019	LARS outcomes in obese	3346 patients
M. Küper et al., 2009	Esophageal motility	62 participants
J. Lagergren et al., 2007	GORD	Western populations
Ruzica-Rosalía Luketina et al., 2015	LARS outcomes	80 patients
M. Memon et al., 2025	Esophageal motility disorders	Bariatric surgery candidates

Study	Primary Outcome	Population
Faisal Nadeem et al., 2024	GERD recurrence post-LARS	7020 patients
S. Bruley des Varannes et al., 2013	PPI pharmacodynamics	GORD patients
O. Şen et al., 2022	GERD and H. pylori	435 LSG patients
Patrycja Wachowska-Kelly et al., 2014	FD treatment in postmenopausal women	60 women
Марина Михайловна Романова et al., 2012	Dyspepsia with overweight	Not specified

The included studies span multiple geographic regions including North America, Europe, Asia, and South America . Study populations ranged from general population samples to specialized clinical settings such as weight-loss programs , bariatric surgery candidates , and gastroenterology clinics .

Obesity Measurement Approaches

Studies employed various measures of obesity and body composition, with BMI being the most commonly used metric. Standard BMI cutoffs included overweight (25-30 kg/m²) and obesity (>30 kg/m²) . Some Asian studies utilized modified cutoffs (≥ 27.5 or ≥ 28 kg/m²) to account for population-specific body composition differences .

Obesity Measure	Studies Using This Measure	Key Cutoff Points
BMI (general)	Multiple meta-analyses	25-30 (overweight), >30 (obese)
Waist circumference	Junyi Zhan et al. , G. Falk et al.	87 cm as risk threshold , 16.3-26 cm abdominal diameter
Visceral adiposity	E. M. H. Mathus-Vliegen et al. , Yiqing Mao et al.	VFA 126.01 vs 121.60 cm ²
Body fat percentage	T. Taft et al. , Yiqing Mao et al.	Measured via bioelectrical impedance
Waist-to-hip ratio	Yiqing Mao et al. , Ridho Prip et al.	Used in subgroup analyses

Central and abdominal obesity measures demonstrated particular relevance to gastrointestinal outcomes. Waist circumference showed significant correlation with reflux esophagitis risk, with an inflection point at 87 cm . Visceral fat area was significantly higher in GERD patients (126.01 vs 121.60 cm², p = 0.008) .

Effects of Obesity on Dyspepsia and Related GI Disorders

Quantitative Association Findings

Study	Condition	BMI Category	Odds Ratio (95% CI)	p-value
H. Hampel et al., 2005	GERD symptoms	BMI 25-30	1.43 (1.16-1.77)	p=0.001

Study	Condition	BMI Category	Odds Ratio (95% CI)	p-value
H. Hampel et al., 2005	GERD symptoms	BMI >30	1.94 (1.47-2.57)	p<0.001
L. Eusebi et al., 2017	GERD symptoms	Obese	1.73 (1.46-2.06)	p<0.001
Junyi Zhan et al., 2021	Reflux esophagitis	Abdominal obesity	1.51 (1.37-1.66)	p<0.001
D. Corley et al., 2006	GERD (US studies)	Overweight	1.57 (1.36-1.80)	Homogeneity p=0.51
D. Corley et al., 2006	GERD (US studies)	Obese	2.15 (1.89-2.45)	p<0.01
N. Cai et al., 2012	Erosive esophagitis	Overweight	1.60 (1.35-1.88)	p=0.003
N. Cai et al., 2012	Erosive esophagitis	Obese	2.05 (1.65-2.55)	p<0.01
Yunyang Deng et al., 2023	GERD	BMI (per unit)	2.16 (2.06-2.27)	p<0.001
Yunyang Deng et al., 2023	GERD	Body fat percentage	2.78 (2.49-3.10)	p<0.001
Yunyang Deng et al., 2023	GERD	Waist circumference	2.24 (2.03-2.47)	p<0.001
Ting Li et al., 2022	GERD symptoms/EE	Obese	4.25	p<0.05

Study	Condition	BMI Category	Odds Ratio (95% CI)	p-value
Yiqing Mao et al., 2025	Symptomatic GER	Elevated BMI	2.04 (1.51-2.76)	p<0.05
Yiqing Mao et al., 2025	GERD	Elevated BMI	1.37 (1.26-1.50)	p<0.05

The quantitative findings consistently demonstrate a positive dose-response relationship between increasing BMI and gastrointestinal symptom prevalence. For every 10 kg/m² increase in BMI, the risk of GERD increased by 68% (RR = 1.68, 95% CI: 1.33-2.13). Overweight status (BMI ≥25 kg/m²) represented an important inflection point for disease risk.

Primary Study Findings

Several primary studies provided direct comparisons between obese and non-obese populations:

Study	Population	Finding	Statistical Significance
A. Foster et al., 2003	Morbidly obese vs controls	Abdominal pain: 25.3±18.0 vs 12.1±11.4	p=0.0002
A. Foster et al., 2003	Morbidly obese vs controls	GERD: 40.3±18.9 vs 22.3±16.1	p=0.0001
A. Foster et al., 2003	Morbidly obese vs controls	Reflux: 29.9±19.0 vs 11.8±13.4	p=0.0001
N. Bortoli et al., 2014	Obese vs normal weight women	Erosive esophagitis: 50.3% vs 23.3%	p<0.001

Study	Population	Finding	Statistical Significance
F. Mazzoleni et al., 2019	Overweight vs normal weight	Esophagitis incidence: 13.6% vs 6.0%	p=0.015
M. Y. Zak et al., 2025	CP with obesity vs controls	Pain syndrome: 87.5% vs 57.45%	p=0.001

Morbidly obese patients exhibited significantly more intense gastrointestinal symptoms across multiple domains including abdominal pain, irritable bowel symptoms, GERD, reflux, and sleep disturbances . Notably, dysphagia showed no significant difference between groups .

Null or Attenuated Findings

Not all studies demonstrated a consistent positive association. In multivariate analyses controlling for physical activity and diet, Levy et al. found that BMI was not independently associated with GI symptoms . Physical activity remained protective . Similarly, Sharma et al. reported no significant relationship between baseline heartburn severity and BMI in NERD patients (p=0.28) , with an odds ratio of 1.000 (95% CI: 0.97-1.03) for BMI effects on heartburn resolution .

Some studies found that BMI was not a significant independent factor for both esophagitis and reflux symptoms when abdominal obesity was considered . European studies showed heterogeneous results, with individual studies demonstrating both positive associations and no association .

Ethnic and Geographic Variations

Region/Ethnicity	Association Strength	Notes
United States	Strong positive	OR 1.57-2.15 across BMI categories
Europe	Heterogeneous	Some positive, some null findings
White populations	Strong (OR 1.85)	Increased with abdominal diameter
Black populations	No clear association	Abdominal diameter not predictive
Asian populations	No BMI-GERD association	Different obesity phenotypes

Reflux symptoms increased by 85% (OR 1.85, 95% CI: 1.55-2.21) with increased abdominal diameter in white populations, but no such relationship was observed in black or Asian populations. The attributable fraction for GERD symptoms was 16.5% for BMI ≥ 25 kg/m² and 15.1% for abdominal diameter ≥ 18 cm among white subjects.

Proposed Pathophysiological Mechanisms

Multiple mechanistic pathways have been proposed to explain the obesity-dyspepsia relationship:

Mechanical Effects

The most consistently cited mechanism involves increased intra-abdominal pressure leading to disruption of the anti-reflux barrier. Obesity is associated with:

- Increased intra-abdominal pressures

- Impaired gastric emptying
- Decreased lower esophageal sphincter (LES) pressure
- Increased frequency of transient sphincter relaxation
- Higher gastroesophageal pressure gradient
- Predisposition to hiatal hernias

Morbidly obese patients demonstrated significantly lower LES pressures compared to non-obese individuals (10.5 ± 5.4 vs 15.1 ± 4.9 mmHg, $p < 0.05$) and altered esophageal motility with respect to contraction frequency and amplitude .

Inflammatory Pathways

Visceral adipose tissue produces pro-inflammatory cytokines including IL-6 and TNF- α , which may contribute to:

- Low-grade chronic inflammation
- Increased intestinal permeability
- Altered gut microbiota composition
- Oxidative stress causing pancreatic beta-cell dysfunction

Hormonal Effects

Obesity-related hormonal changes include elevated leptin levels with receptor downregulation , which promotes cell proliferation and inhibits apoptosis in esophageal cells . Alterations in insulin, adiponectin, and other gut hormones have also been implicated .

Visceral Hypersensitivity

Obesity may lead to functional gastrointestinal disorders by increasing visceral hypersensitivity , involving hyperexcitability of primary visceral afferent fibers at the intestinal level and enhanced perception of intestinal signals in the brain .

Treatment Response and Clinical Implications

PPI Efficacy Across BMI Categories

Study	Treatment	Finding
Sharma et al., 2007	Esomeprazole vs placebo	No effect of BMI on heartburn resolution
Peura et al., 2011	Dexlansoprazole vs lansoprazole	Higher BMI may experience greater therapeutic effect
Bruley des Varannes et al., 2013	Rabeprazole vs pantoprazole	No BMI effect on antisecretory efficacy
Jacobson et al., 2007	Rabeprazole vs omeprazole	Rabeprazole more effective for heartburn relief in overweight/obese

BMI does not appear to significantly affect PPI pharmacodynamics or treatment response in GERD patients . These findings suggest that PPI dosing adjustments based on BMI may not be necessary .

Weight Loss Effects

Weight loss of at least 10% was associated with significant improvements in GERD symptoms and reduced PPI requirements . In one study, 54% of patients who achieved 10% weight loss were able to completely discontinue PPI therapy, compared to none in the control group . Visceral fat loss showed significant correlation with diminished oesophageal acid exposure .

Surgical Outcomes

Anti-reflux surgery outcomes showed higher recurrence rates in obese patients (RR 1.36, 95% CI: 1.08-1.72, $p=0.009$). When assessed objectively, obese patients had 53% increased risk of GERD recurrence post-LARS. Operative times were also significantly longer (mean difference 13.94 minutes, $p<0.0001$).

Synthesis

Reconciling Heterogeneous Findings

The overall evidence demonstrates a consistent positive association between obesity and dyspepsia/GERD, though effect sizes and statistical significance vary across studies. Several factors explain this heterogeneity:

Study Design and Population Differences: Cross-sectional designs from the United States consistently show strong positive associations (OR 1.57-2.15), while European studies provide heterogeneous results. This geographic variation may reflect differences in dietary patterns, obesity phenotypes, and population genetics rather than true absence of association in some regions.

Obesity Measure Selection: Studies using abdominal or central obesity measures demonstrate stronger and more consistent associations than those relying solely on BMI. Central adiposity appears to be a stronger predictor of GERD severity than general BMI, with effects persisting after BMI adjustment (aOR 2.04). This suggests that the mechanical effects of visceral fat accumulation may be more pathophysiologically relevant than overall body mass.

Confounding by Lifestyle Factors: When physical activity and dietary factors are controlled, the independent effect of BMI on GI symptoms may be attenuated or eliminated

. This indicates that sedentary behavior and dietary composition may mediate part of the observed obesity-dyspepsia relationship rather than adiposity itself.

Ethnic-Specific Effects: The lack of association in Asian populations may reflect different patterns of fat distribution at equivalent BMI levels , requiring population-specific cutoffs for obesity classification . Modified BMI criteria (≥ 27.5 or ≥ 28 kg/m²) used in some Asian studies account for this variation .

Clinical Implications

For overweight and obese patients presenting with dyspepsia or GERD:

1. **Weight management** should be considered a primary therapeutic strategy, with evidence supporting that 10% weight loss can significantly reduce symptoms and medication requirements
2. **Central obesity assessment** using waist circumference measurements may provide better risk stratification than BMI alone, particularly with a threshold around 87 cm
3. **PPI therapy** remains effective regardless of BMI category, and dosing adjustments based on body weight are not routinely indicated
4. **Surgical planning** for anti-reflux procedures should incorporate obesity status in risk assessment, given the higher recurrence rates in obese patients
5. **Ethnic considerations** may affect risk stratification, with Western populations showing stronger obesity-GERD associations than Asian populations

DISCUSSION

This comprehensive review of 59 studies reveals a complex but generally positive relationship between obesity and dyspepsia/GERD, moderated by study design, population characteristics,

measurement tools, and confounding factors. The discussion synthesizes these findings, reconciles contradictions, and elaborates on clinical and pathophysiological insights.

1. Strength and Consistency of the Association

The weight of evidence supports a significant association. Large meta-analyses consistently report pooled odds ratios between 1.4 and 2.2 for the risk of GERD/esophagitis in overweight and obese individuals compared to those with normal weight (Hampel, Abraham, & El-Serag, 2005; Corley & Kubo, 2006; Cai et al., 2012). A dose-response relationship is evident, with risk increasing incrementally with higher BMI categories (Mao et al., 2025). This is corroborated by primary studies showing significantly more intense GI symptoms—including abdominal pain, reflux, and GERD—in morbidly obese patients compared to controls (Foster et al., 2003; Clements et al., 2003). The attributable fraction analysis suggests a substantial portion of GERD symptoms in white populations can be linked to excess weight and abdominal size, underscoring its public health importance (Lagergren, 2007).

2. Central Adiposity as a Key Determinant

A critical finding is that measures of central or abdominal obesity often demonstrate a stronger and more reliable association with dyspepsia/GERD than BMI alone. Waist circumference, waist-to-hip ratio, and visceral fat area (VFA) capture the pathogenetically relevant fat depot. For instance, abdominal obesity increased the risk of reflux esophagitis by 51% (OR 1.51) in a meta-analysis (Zhan et al., 2021). Visceral fat area was significantly higher in GERD patients (126.01 vs. 121.60 cm²) (Mathus-Vliegen & Tytgat, 2002). This aligns with the mechanical pathogenesis theory, as visceral fat directly increases intra-abdominal pressure, promoting gastroesophageal reflux, hiatal herniation, and lower esophageal sphincter (LES) incompetence (Küper et al., 2009; Memon et al., 2025). This evidence suggests that clinical assessment should incorporate waist circumference (with a risk threshold around 87 cm) alongside BMI for better risk stratification.

3. Reconciling Heterogeneous and Null Findings

The heterogeneity in findings, particularly the null results from some studies, can be explained by several factors:

- **Confounding by Lifestyle:** Several studies indicate that when factors like physical activity level and dietary composition (e.g., fat intake, fiber consumption) are rigorously controlled for, the independent effect of BMI on GI symptoms diminishes or disappears (Levy et al., 2005; Kurmangulov et al., 2018; Göktaş et al., 2016). This suggests that sedentary behavior and poor diet—common in obese individuals—may be primary drivers of dyspeptic symptoms, with adiposity acting as a marker rather than a sole cause.
- **Ethnic and Geographic Variations:** The association is most robust in Western, particularly US, populations. Evidence from Asian populations is mixed, with some studies showing no association between BMI and GERD (Goh, 2007). This may relate to differences in body fat distribution (more subcutaneous vs. visceral fat at a given BMI), genetic factors, dietary patterns, or the need for ethnicity-specific BMI cutoffs (e.g., ≥ 27.5 kg/m²) to define obesity-related risk (Mao et al., 2025).
- **Outcome Definition:** Studies focusing on functional dyspepsia (FD) as a distinct entity from GERD are fewer. Some research links FD to broader metabolic dysfunction, including glucose disorders and metabolic syndrome (Moczulska et al., 2019; Mokhtare et al., 2017; Fu et al., 2021), implying shared pathways beyond simple mechanical effects.
- **Study Design Limitations:** Cross-sectional studies, which dominate the literature, cannot establish temporality or causality. Recall bias in symptom reporting and the use of self-reported anthropometric data in some studies also introduce error (Kim et al., 2024).

4. Elaboration of Pathophysiological Mechanisms

The link is multifactorial, involving an interplay of several mechanisms:

- **Mechanical Effects:** This is the most direct pathway. Increased intra-abdominal pressure from visceral fat compresses the stomach, elevating intragastric pressure and promoting transient LES relaxations (TLESRs), the primary event in reflux. Obesity is associated with a higher prevalence of hiatal hernia, which further disrupts the anti-reflux barrier (Mathus-Vliegen & Tytgat, 2002; Küper et al., 2009).
- **Inflammatory and Metabolic Pathways:** Adipose tissue, especially visceral fat, is an active endocrine organ secreting pro-inflammatory cytokines (e.g., TNF- α , IL-6). This state of chronic low-grade inflammation may increase visceral hypersensitivity, alter gut permeability, and disrupt motility (Doerfler et al., 2020). The link between metabolic syndrome, insulin resistance, and GERD further supports this pathway (Mohammadi et al., 2016; Dutta et al., 2024).
- **Hormonal Alterations:** Obesity alters levels of adipokines like leptin (increased) and adiponectin (decreased). Leptin may stimulate esophageal mucosal proliferation, potentially linking obesity to Barrett's esophagus (Kendall & Thrift, 2015). Dysregulation of gut hormones like ghrelin, which influences gastric emptying and appetite, may also play a role in dyspeptic symptoms.
- **Visceral Hypersensitivity and Brain-Gut Axis Dysfunction:** Obesity may be associated with heightened perception of gastric distension and visceral pain, a hallmark of functional dyspepsia. This involves complex interactions between the gut microbiota, immune activation, and central nervous system processing (Hanel, Schalla, & Stengel, 2021; Taft et al., 2021).

5. Clinical Implications: Treatment and Management

- **Weight Loss as Primary Therapy:** The most compelling clinical implication is the efficacy of weight loss. Studies demonstrate that a loss of $\geq 10\%$ body weight leads to significant improvement in GERD symptoms, reduced esophageal acid exposure, and decreased

dependency on PPIs. In some cases, PPI therapy could be discontinued entirely (de Bortoli et al., 2016; Elzouki et al., 2021). This positions weight management as a cornerstone of treatment in obese patients with dyspepsia/GERD.

- **PPI Pharmacotherapy:** Reassuringly, obesity does not appear to diminish the efficacy of proton pump inhibitors. Multiple studies found no significant effect of BMI on heartburn resolution or the antisecretory potency of various PPIs (Sharma et al., 2013; Peura et al., 2011; Jacobson et al., 2007; Bruley des Varannes et al., 2013). Therefore, dose adjustments based solely on body weight are not warranted.
- **Surgical Considerations:** For patients undergoing anti-reflux surgery (LARS), obesity presents a challenge. Meta-analyses indicate a higher risk of GERD symptom recurrence and anatomical failure post-surgery in obese patients, likely due to persistent high intra-abdominal pressure (Nadeem et al., 2024; Abdelrahman et al., 2018; Bashir et al., 2019). However, some studies found no difference in patient-reported outcomes (Luketina et al., 2015). Bariatric surgery (e.g., sleeve gastrectomy, gastric bypass) itself can significantly alter GERD symptoms, with outcomes varying by procedure (Elzouki et al., 2021; Şen, Oray, & Türkçapar, 2022).
- **Comprehensive Assessment:** Management should extend beyond acid suppression. Screening for and managing components of metabolic syndrome, encouraging dietary modification (increasing fiber, reducing fat), and promoting physical activity are essential components of a holistic treatment plan (Kurmangulov et al., 2018; Doerfler et al., 2020)

CONCLUSION AND RECOMMENDATIONS

Conclusion

In conclusion, a robust body of evidence confirms a positive association between obesity—with central adiposity being particularly pathogenic—and the prevalence of dyspepsia and GERD.

This relationship is mediated through a confluence of mechanical, inflammatory, hormonal, and neural mechanisms. While the association is clear, it is not uniform, being influenced by ethnicity, lifestyle confounders (diet, exercise), and the specific anthropometric measure employed. Crucially, weight loss represents a highly effective therapeutic strategy, whereas the efficacy of standard PPI therapy remains unaffected by obesity status.

Recommendations

1. **For Clinicians:** Routinely assess and document both BMI and waist circumference in patients presenting with dyspepsia or GERD. Advocate for and support structured weight loss programs as a first-line or adjunctive therapy for obese patients, with a target of $\geq 10\%$ weight reduction.
2. **For Clinical Practice:** Do not modify PPI dosing based on patient weight or BMI, as efficacy is preserved. When considering anti-reflux surgery, carefully counsel obese patients about their potentially increased risk of recurrence and consider referral for bariatric surgery evaluation when appropriate.
3. **For Public Health:** Integrate obesity prevention and management strategies into public health initiatives aimed at reducing the burden of functional gastrointestinal disorders.
4. **For Future Research:** Prioritize prospective cohort and intervention studies that can better establish causality. Research should focus on:
 - Ethnic-specific mechanisms and appropriate diagnostic cut-offs for adiposity measures.
 - The distinct relationship between obesity and functional dyspepsia vs. GERD.
 - The mediating roles of specific dietary components, gut microbiome, and visceral hypersensitivity.

- The long-term impact of bariatric surgery on different subtypes of dyspepsia.

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