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THE ASSOCIATION OF OBESITY WITH IRRITABLE BOWEL SYNDROME IN ADULTS: A SYSTEMATIC REVIEW

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ABSTRACT

The relationship between obesity and Irritable Bowel Syndrome (IBS) remains a subject of ongoing debate, as epidemiological evidence has yielded inconsistent findings. Although both conditions are highly prevalent and significantly impair quality of life, a universal association between them has not been established. This systematic review aims to synthesize the available evidence and clarify the nature of this relationship by examining specific clinical phenotypes, diagnostic criteria, and underlying biological mechanisms. Following the PRISMA 2020 guidelines, a comprehensive search was performed across multiple databases. Eligible studies included adult populations, used standardized measures of obesity such as Body Mass Index (BMI) or waist circumference, and diagnosed IBS using the Rome criteria or physician assessment. Thirteen studies—including systematic reviews, meta-analyses, observational studies, and clinical trials—met the inclusion criteria and were incorporated into a narrative synthesis. The findings indicate no consistent overall association between BMI-defined obesity and IBS prevalence. However, significant associations were observed in specific subgroups. A Mendelian randomization study demonstrated strong genetic evidence supporting a causal link between visceral adiposity and IBS risk. Additional studies identified significant relationships for the diarrhea-predominant subtype (IBS-D) and for IBS diagnosed using Rome IV criteria. Interventional studies further showed that reductions in abdominal fat were associated with clinically meaningful improvements in IBS symptoms and quality of life. Overall, the evidence suggests that the most relevant association is not with general obesity but specifically with metabolically active visceral adiposity. These findings support incorporating targeted weight-management strategies into the clinical approach for patients with coexisting obesity and IBS.

KEYWORD:

Obesity, Irritable Bowel Syndrome, Visceral Adiposity, Systematic Review, Gut Microbiota

ABSTRAK

Hubungan antara obesitas dan Irritable Bowel Syndrome (IBS) masih menjadi perdebatan karena bukti epidemiologis menunjukkan hasil yang tidak konsisten. Meskipun kedua kondisi tersebut memiliki prevalensi tinggi dan berdampak signifikan terhadap kualitas hidup, belum terdapat kesepakatan mengenai adanya hubungan langsung yang bersifat universal. Penelitian ini bertujuan untuk mensintesis dan memperjelas bukti ilmiah terkini mengenai hubungan tersebut dengan menelaah perbedaan fenotipe klinis, penggunaan kriteria diagnosis, serta mekanisme biologis yang mungkin menghubungkan obesitas dan IBS. Kajian dilakukan melalui tinjauan sistematis berdasarkan pedoman PRISMA 2020, dengan pencarian literatur pada berbagai basis data. Kriteria inklusi mencakup penelitian pada populasi dewasa, penggunaan ukuran obesitas yang terstandarisasi seperti Body Mass Index (BMI) atau lingkar pinggang, serta diagnosis IBS menggunakan kriteria Rome atau penilaian klinis. Sebanyak tiga belas studi, yang terdiri dari tinjauan sistematis, meta-analisis, studi observasional, dan uji klinis, memenuhi syarat dan dianalisis secara naratif. Hasil menunjukkan tidak terdapat hubungan konsisten antara obesitas berbasis BMI dan prevalensi IBS. Namun, hubungan bermakna ditemukan pada subkelompok tertentu. Satu studi Mendelian randomization memberikan bukti genetik kuat mengenai hubungan kausal antara adipositas visceral dan peningkatan risiko IBS. Temuan signifikan juga muncul pada subtipe IBS diare (IBS-D) dan diagnosis berdasarkan kriteria Rome IV. Intervensi penurunan lemak abdominal terbukti memperbaiki gejala IBS dan kualitas hidup. Secara keseluruhan, bukti menunjukkan bahwa hubungan yang paling relevan bukan pada obesitas umum, melainkan adipositas visceral yang bersifat metabolik aktif.

KATA KUNCI

Obesitas, Irritable Bowel Syndrome, Adipositas Visceral, Sistematik Review, Mikrobiota Usus

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INTRODUCTION

Irritable Bowel Syndrome (IBS) is a common functional gastrointestinal disorder characterized by chronic and recurrent abdominal pain, bloating, and alterations in bowel habits, including diarrhea or constipation. The condition significantly reduces the quality of life for millions of adults and places a substantial burden on healthcare systems worldwide (Mahadeva, 2023). Diagnosis relies primarily on symptom-based criteria, particularly the Rome guidelines, which have been refined over time to improve clinical accuracy (Yau et al., 2024). At the same time, obesity has emerged as a major global health issue, defined by excessive body fat accumulation and commonly assessed using a Body Mass Index (BMI) of 30 kg/m² or higher. Obesity is associated with chronic low-grade inflammation influenced by genetic, metabolic, and environmental factors (Hadi et al., 2016; Shen et al., 2019).

In recent years, scientific interest in the potential comorbidity between obesity and IBS has increased. Both conditions are highly prevalent, and several observational studies suggest that their coexistence may not be coincidental. This has led to hypotheses proposing a bidirectional association in which obesity may trigger or exacerbate IBS symptoms, while IBS-related mechanisms—including altered motility, visceral sensitivity, and microbiome disruption may also contribute to weight changes (Rawat et al., 2024; Simanenkov et al., 2019). Shared pathophysiological pathways such as inflammation, gut microbiota alteration, and metabolic dysregulation have been proposed as potential mechanistic links between the two disorders (Oliveira et al., 2022; Pogodina et al., 2021).

Despite the growing body of evidence, findings regarding the association between obesity and IBS remain inconsistent. Several systematic reviews and meta-analyses report a positive association, indicating an increased IBS risk among individuals with obesity (Açik et al., 2025), whereas others have found no significant overall relationship (Yau et al., 2024; Neo et al., 2020). These discrepancies complicate clinical interpretation and may reflect heterogeneity in diagnostic criteria, populations studied, and the multifactorial nature of both conditions. Notably, some associations appear only within specific subgroups, including individuals with diarrhea-predominant IBS (IBS-D) or those diagnosed using the more stringent Rome IV criteria (Neo et al., 2020; Yau et al., 2024). Recent interventional findings also suggest that reductions in abdominal or visceral fat may improve IBS symptoms and quality of life, indicating that weight management may serve as a therapeutic option for some patients (Elrashidy et al., 2024; Aasbrenn et al., 2018). Proceedings and recent publications from the past five years additionally highlight the need to clarify the specific role of central adiposity in IBS pathogenesis, suggesting that visceral fat may be more predictive of IBS symptoms than general obesity (Chen et al., 2024; Abboud et al., 2025). Given the conflicting epidemiological findings, subgroup-specific distinctions, and evolving mechanistic insights, a comprehensive synthesis of current evidence is warranted. This systematic review aims to evaluate the strength and nature of the association between obesity and IBS in adults by integrating findings across diverse study designs and populations. The study contributes to existing knowledge by offering an updated synthesis that incorporates recent scientific publications and proceedings, identifies key sources of heterogeneity, and highlights mechanistic pathways most consistently supported in the literature. This review also introduces a novel perspective by emphasizing the potential causal role of visceral adiposity—rather than general BMI-defined obesity in shaping IBS risk, thereby informing future research directions and clinical practice (Chen et al., 2024). This systematic review aims to synthesize the available evidence and clarify the nature of this relationship by examining specific clinical phenotypes, diagnostic criteria, and underlying biological mechanisms. Following the PRISMA 2020 guidelines, a comprehensive search was performed across multiple databases.

METHOD

This systematic review was conducted in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. The study employed a systematic review design aimed at evaluating the association between obesity and Irritable Bowel Syndrome (IBS) among adults. The **population** targeted in this review consisted of adults aged 18 years or older who participated in empirical studies assessing obesity and IBS. The **samples or subjects** in the context of this review were peer-reviewed articles that met the inclusion criteria and passed all screening stages.

Articles were selected through a comprehensive, multi-stage screening procedure. Studies were eligible for inclusion if they involved adult participants, utilized standardized metrics for assessing obesity—such as a Body Mass Index (BMI) of ≥30 kg/m² or measurements of central adiposity—and diagnosed IBS using the Rome criteria or a physician-confirmed diagnosis. To ensure relevance, studies were required to examine the obesity–IBS association as either a primary or secondary outcome. Eligible research designs included observational studies (cross-sectional, cohort, case-control), systematic reviews, and meta-analyses. Case reports, case series, animal studies, and laboratory-based experiments were excluded.

During data extraction, all available study designs were documented; if the design was unclear, this was noted explicitly. Extraction procedures emphasized capturing exact numerical values and percentages, acknowledging missing information, and documenting value ranges when provided. All measurement instruments and statistical procedures used within the included studies were comprehensively listed. Key findings were recorded with priority given to statistically significant outcomes, while ensuring that all reported numerical results were included for accuracy.

The extracted data elements comprised: (1) study design; (2) participant characteristics, including sample size, sex distribution, and age; (3) IBS diagnostic criteria; (4) definitions and measurement tools for obesity; (5) primary statistical outcomes such as effect sizes, odds ratios, confidence intervals, p-values, and subgroup analyses; and (6) methods of data collection and analysis, including survey tools, clinical assessments, and statistical models such as meta-analysis or meta-regression where applicable.

Search Strategy

The literature search applied the PICO framework (Population, Intervention, Comparison, Outcome) to determine relevant keywords and indexing terms. The Boolean and MeSH keyword string used across the databases was:

Table 1. Keywords

Element	Keyword 1	Keyword 2	Keyword 3	Keyword 4
Population (P)	Adults Population	Participants aged 18 years or older	Adults	General adult populations
Intervention (I)	Obesity	High BMI	High Body Mass Index	Overweight
Comparison (C)	Non-Obese	Normal BMI	Healthy weight	Non-overweight
Outcome (O)	Irritable Bowel Syndrome (IBS)	IBS Subtypes	Functional Gastrointestinal Disorders	IBS Symptoms

The Boolean MeSH keywords inputted on databases for this research are: ("Adults Population" OR "Participants aged 18 years or older" OR "Adults" OR "General adult populations") AND ("Obesity" OR "High BMI" OR "High Body Mass Index" OR "Overweight") AND ("Non-Obese" OR "Normal BMI" OR "Healthy weight" OR "Non-overweight") AND ("Irritable Bowel Syndrome (IBS)" OR "IBS Subtypes" OR "Functional Gastrointestinal Disorders" OR "IBS Symptoms").

Data retrieval

Following the initial data retrieval, all records were compiled, and duplicate entries were removed using reference management software to ensure each study was assessed only once. The screening process was then conducted independently by two reviewers, Alif Ramadhan and Reza Muchlas Fauziansyah, to determine eligibility. They first screened the titles and abstracts of the retrieved articles against the predefined inclusion criteria. Any disagreements or uncertainties between the two reviewers were resolved through discussion to reach a consensus. If a consensus could not be achieved, a third reviewer, Sagita Nindra Pratama, was consulted to make the final decision regarding inclusion or exclusion. Studies that fulfilled all criteria after this rigorous screening process were selected for full-text analysis, during which data such as titles, authors, publication dates, study locations, methodologies, and key parameters were thoroughly examined.

Quality Assessment and Data Synthesis

For the quality assessment, the methodological rigor of each included study was independently evaluated by two reviewers, Alif Ramadhan and Reza Muchlas Fauziansyah, using the appropriate Joanna Briggs Institute (JBI) Critical Appraisal Tools. These tools consist of a series of questions designed to assess a study's risk of bias across key domains such as participant selection, confounding factors, and outcome measurement. Any discrepancies in the quality ratings between the two reviewers were resolved through discussion to reach a consensus. If an agreement could not be reached, a third reviewer, Sagita Nindra Pratama, was consulted to make the final determination. Based on this appraisal, each study's overall methodological quality was categorized, which informed the strength and interpretation of its findings. Following the quality assessment, the data was synthesized narratively, summarizing and integrating the key findings and characteristics from all included articles to provide a comprehensive overview of the evidence.

Table 2. Article Search Strategy

Database	Keywords	Hits
Pubmed	*("Adults Population" OR "Participants aged 18 years or older" OR "Adults" OR "General adult populations") AND ("Obesity" OR "High BMI" OR "High Body Mass Index" OR "Overweight") AND ("Non-Obese" OR "Normal BMI" OR "Healthy weight" OR "Non-overweight") AND ("Irritable Bowel Syndrome (IBS)" OR "IBS Subtypes" OR "Functional Gastrointestinal Disorders" OR "IBS Symptoms")*	2
Cochrane	*("Adults Population" OR "Participants aged 18 years or older" OR "Adults" OR "General adult populations") AND ("Obesity" OR "High BMI" OR "High Body Mass Index" OR "Overweight") AND ("Non-Obese" OR "Normal BMI" OR "Healthy weight" OR "Non-overweight") AND ("Irritable Bowel Syndrome (IBS)" OR "IBS Subtypes" OR "Functional Gastrointestinal Disorders" OR "IBS Symptoms")*	1
Semantic Scholar	*("Adults Population" OR "Participants aged 18 years or older" OR "Adults" OR "General adult populations") AND ("Obesity" OR "High BMI" OR "High Body Mass*	250

	Index" OR "Overweight") AND ("Non-Obese" OR "Normal BMI" OR "Healthy weight" OR "Non-overweight") AND ("Irritable Bowel Syndrome (IBS)" OR "IBS Subtypes" OR "Functional Gastrointestinal Disorders" OR "IBS Symptoms")	
Google Scholar	*("Adults Population" OR "Participants aged 18 years or older" OR "Adults" OR "General adult populations") AND ("Obesity" OR "High BMI" OR "High Body Mass Index" OR "Overweight") AND ("Non-Obese" OR "Normal BMI" OR "Healthy weight" OR "Non-overweight") AND ("Irritable Bowel Syndrome (IBS)" OR "IBS Subtypes" OR "Functional Gastrointestinal Disorders" OR "IBS Symptoms")*	1,410

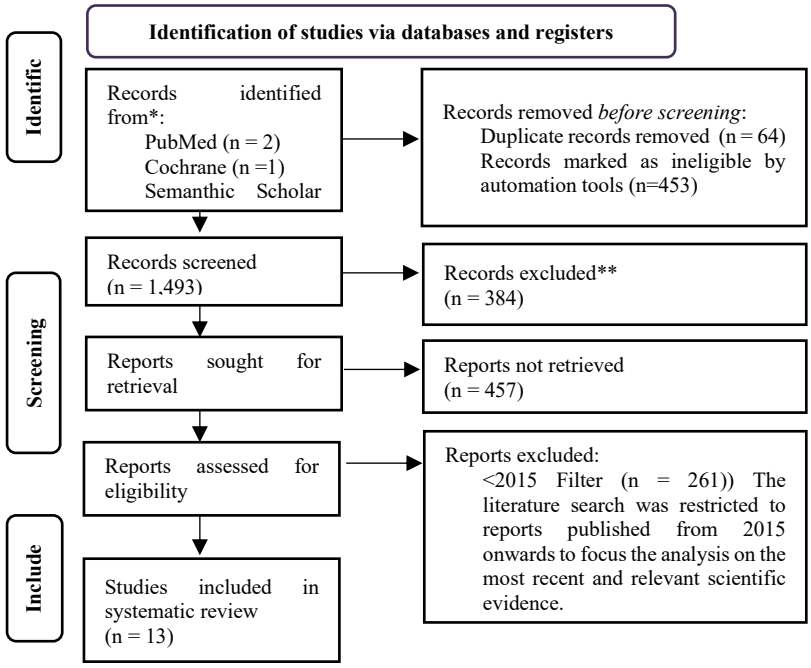

Figure 1. Article search flowchart

RESULTS AND DISCUSSION

Result

Characteristics of Included Studies

Table 3. Characteristics of Included Studies

Study	Study Design	Sample Size	Population Characteristics	Primary Findings
Abboud et al., 2025	Cross-sectional study	221	Lebanese adults, mean age 43.4, 62.9% female	Metabolic Syndrome (MetS) and its components (waist circumference, blood pressure, fasting blood sugar) positively associated with IBS symptoms, especially diarrhea
Açık et al., 2025	Systematic review	Not reported in abstract	Obese adults (BMI 30), Turkey	10.5% IBS prevalence in obese patients; smoking also a significant risk factor
Simanenkov et al., 2019	Systematic review	Not reported in abstract	Adults, IBS and obesity	Reviews genetic, hormonal, microbiota, and psychological

Study	Study Design	Sample Size	Population Characteristics	Primary Findings
				mechanisms for IBS-obesity comorbidity
Rawat et al., 2024	Narrative review	Not reported in abstract	Adults, FGIDs, IBS, obesity	Reviews bidirectional and mechanistic links between obesity and IBS; highlights shared pathophysiology
Oliveira et al., 2022	Systematic review	14 studies (825 women)	Overweight/obese women, 18-65 years	Nutritional interventions (low fermentable oligosaccharides, disaccharides, monosaccharides, and polyols [low-FODMAP], pre/probiotics) improve IBS symptoms; no direct statistical link between obesity and IBS
Погодина et al.,	Systematic review	Not reported in abstract	Adults, Functional Bowel Disorders (FBD)	Stronger evidence for obesity-diarrhea link than for obesity-IBS; insufficient data for obesity-IBS association
Pogodina et al., 2021	Systematic review	Not reported in abstract	Adults, FBD	Obesity more clearly linked to diarrhea than IBS; insufficient data for obesity-IBS association
Elrashidy et al., 2024	Randomized controlled trial (RCT)	60	Adults 20-45 years, BMI 30-39.9, IBS and abdominal obesity	Abdominal fat reduction (focused ultrasound, exercise, diet) improved IBS symptoms and quality of life more than exercise/diet alone
Aasbrenn et al., 2018	Prospective cohort study	88	Morbidly obese adults, mean age 44, 81% female, BMI >40 or >35 with complications	Weight loss led to significant improvement in IBS symptoms, especially in those with IBS at baseline

Study	Study Design	Sample Size	Population Characteristics	Primary Findings
Chen et al., 2024	Mendelian randomization, Genome-Wide Association Study (GWAS); meta-analysis	Genome-Wide Association Study (GWAS): 40,548 cases/293,220 controls (UK Biobank); 12,852/139,981 (Bellygenes); 8,116/276,683 (FinnGen)	Adults, genetic data, IBS by Rome III/ICD codes	Genetically predicted visceral adiposity, BMI, and lean body mass causally associated with increased IBS risk
Mahadeva, 2023	Systematic review, cross-sectional	Not reported in abstract	Adults, Functional Gastrointestinal Disorders (FGIDs), various populations	Obesity is a risk factor for IBS in some studies, but associations are inconsistent; mechanisms may involve visceral hypersensitivity and inflammation
Yau et al., 2024	Systematic review, meta-analysis	27 studies (sample size not reported)	Adults, Body Mass Index (BMI) categories, Irritable Bowel Syndrome (IBS) by Rome criteria	No overall association between overweight/obese BMI and IBS; significant association for obese BMI and IBS with Rome IV criteria
Neo et al., 2020	Systematic review, meta-analysis	34 studies (sample size not reported)	Adults, BMI/Waist Circumference (WC), IBS subtypes	No significant overall association; significant positive association for obesity and diarrhea-predominant IBS (IBS-D)

The research landscape on this topic is built on various study designs, dominated by nine systematic reviews or meta-analyses, including one Mendelian randomization study. This high-level evidence was supplemented by a randomized controlled trial, a prospective cohort study, a cross-sectional study, and a narrative review. The primary findings from these studies are notably mixed. Three major systematic reviews and meta-analyses concluded there was no overall association between obesity and IBS. In contrast, two studies did report a positive association. A more nuanced view was presented in five studies, which found associations only within specific subgroups, such as patients with diarrhea-predominant IBS, those meeting the Rome IV criteria, or individuals with diarrhea symptoms. Clinically, three studies showed that interventions like weight loss improved IBS symptoms. However, the evidence base was further complicated by three studies that described the data as inconsistent or insufficient, three that only reviewed potential biological mechanisms without testing the association directly, and one study that simply reported IBS prevalence in an obese population, noting smoking as a risk factor.

The studies predominantly focused on general adult populations (nine studies). However, three studies specifically recruited obese or overweight adults, and one was conducted exclusively with women. The geographic context was mostly undefined, with the exceptions of one study conducted in Lebanon and another in Turkey.

Prevalence Patterns and Risk Relationships

Table 4. Effects of Obesity on IBS Prevalence and Risk

Study	Obesity Measures	IBS Prevalence	Effect Size	Statistical Significance
Yau et al., 2024	BMI (study-defined)	No mention found	Overweight: Odds Ratio (OR) 1.02 (95% Confidence Interval [CI] 0.89–1.17); Obese: OR 1.11 (0.91–1.37); Rome IV: OR 1.59 (1.13–2.23)	No significant association overall; significant for Rome IV criteria ($p<0.01$)
Neo et al., 2020	BMI, Waist Circumference	No mention found	BMI: Standardized Mean Difference (SMD) +0.227 (-0.093 to 0.547), OR 1.312 (0.974-1.767); IBS-D: SMD +1.940 (0.679-3.200)	No significant association overall; significant for diarrhea-predominant IBS ($p=0.003$)
Elrashidy et al., 2024	BMI 30-39.9	No mention found	No mention found	Significant improvement in IBS symptoms with intervention (no effect size reported)
Aasbrenn et al., 2018	BMI >40 or >35 with complications	No mention found	BMI reduced from 42.0 to 38.7 ($p<0.001$); IBS-SSS reduced from 116 to 81 ($p<0.001$); IBS-SSS improvement: 88 (95% CI 55–121) in IBS group	Significant improvement in IBS symptoms with weight loss
Chen et al., 2024	Genetically predicted BMI, visceral adiposity	No mention found	Visceral adiposity: OR 1.15 (1.06-1.24, $p=7.96\times10^{-6}$); BMI: OR 1.08 (1.03-1.15)	Statistically significant
Mahadeva, 2023	BMI >30	No mention found	No mention found	Inconsistent associations; some studies show risk, others do not
Oliveira et al., 2022	BMI, Waist Circumference, visceral adiposity	No mention found	No mention found	Insufficient data for obesity-IBS association
Pogodina et al., 2021	No mention found	No mention found	No mention found	Insufficient data for obesity-IBS association
Abboud et al., 2025	Waist circumference (MetS)	No mention found	Waist circumference: Beta coefficient = 5.38 ($p=0.010$) for Visual Analogue Scale (VAS)-Diarrhea	Statistically significant for MetS components and IBS symptoms
Açik et al., 2025	BMI 30	10.5% in obese patients	No mention found	Smoking $p=0.003$; no effect size for obesity-IBS
Simanenkov et al., 2019	No mention found	No mention found	No mention found	No mention found
Rawat et al., 2024	No mention found	No mention found	No mention found	No mention found

In the reviewed studies, Body Mass Index (BMI) was the most common method for measuring obesity, used in eight studies. Other metrics included waist circumference, which was utilized in three studies, visceral adiposity in two, and genetically predicted obesity measures in one. Four studies did not specify the particular obesity measure employed. The reporting of effect sizes was inconsistent, with eight studies not providing this information. Among those that did, three studies reported odds ratios, while one study each reported a standardized mean difference, a beta coefficient, and an IBS symptom severity score.

The statistical significance of the findings was varied. A statistically significant association between obesity and IBS was found in two studies, which included a Mendelian randomization meta-analysis. Two other studies reported significant associations, but only for specific subgroups, such as patients with diarrhea-predominant IBS, or for particular symptoms. Additionally, two studies showed a significant improvement in IBS symptoms following an intervention but did not find a direct association between obesity and IBS at baseline. The evidence was further complicated by one study reporting inconsistent associations, two studies having insufficient data to assess the link, and four studies not mentioning statistical significance at all.

Mechanistic Pathways Linking Obesity and IBS

Table 5. Proposed Biological Mechanisms Linking Obesity and IBS

Study	Mechanism Type	Evidence Quality	Clinical Relevance	Key Findings
Yau et al., 2024	No mention found	High (meta-analysis)	Moderate	Suggests excess body weight may not be a primary IBS driver; calls for mechanistic studies
Neo et al., 2020	No mention found	High (meta-analysis)	Moderate	Supports shared pathophysiology for diarrhea-predominant IBS and obesity
Elrashidy et al., 2024	No mention found	Moderate (randomized controlled trial)	High	Abdominal fat reduction improves IBS symptoms
Aasbrenn et al., 2018	No mention found	Moderate (cohort study)	High	Weight loss associated with improved bowel symptoms and well-being
Chen et al., 2024	Genetic, inflammation, microbiota	High (Mendelian randomization, meta-analysis)	High	Genetic evidence suggests a possible causal association; mechanisms may involve motility, microbiota, inflammation
Mahadeva, 2023	Visceral hypersensitivity, inflammation, microbiota	Moderate (review)	Moderate	Obesity may increase visceral hypersensitivity, alter microbiota, promote inflammation
Oliveira et al., 2022	Diet, inflammation, microbiota	Moderate (review)	High	Diet-induced inflammation and microbiota changes implicated in IBS symptoms
Погодина et al.,” ”	Diet, bile acids, microbiota, inflammation	Moderate (review)	Moderate	Obesity-diarrhea link via diet, bile acids, microbiota, inflammation
Pogodina et al., 2021	Diet, bile acids, microbiota, inflammation	Moderate (review)	Moderate	Similar to above; insufficient data for IBS

Abboud et al., 2025	Metabolic, inflammation	Moderate (cross-sectional study)	Moderate	Metabolic syndrome components (waist circumference, blood pressure, fasting blood sugar) linked to IBS symptoms
Açik et al., 2025	No mention found	Moderate (review)	Moderate	Notes dietary triggers and low-FODMAP efficacy
Simanenkov et al., 2019	Genetic, hormonal, microbiota, psychological	Moderate (review)	Moderate	Reviews multiple mechanisms for IBS-obesity comorbidity
Rawat et al., 2024	Inflammation, microbiota, permeability, hypersensitivity	Moderate (review)	Moderate	Highlights shared pathophysiology and bidirectional links

Based on the research, inflammation and alterations in gut microbiota are the most frequently proposed mechanisms linking obesity and Irritable Bowel Syndrome (IBS), with each being mentioned in seven studies. Other potential mechanisms include diet, genetics, bile acids, metabolic factors, visceral hypersensitivity, hormonal influences, psychological factors, and intestinal permeability, though these were discussed less frequently. The quality of the evidence is varied, comprising three high-quality studies (meta-analyses or Mendelian randomization), a number of moderate-quality studies including a randomized controlled trial, a cohort study, a cross-sectional study, and seven reviews. Five studies did not provide detailed information on mechanisms.

Clinically, the findings suggest several management considerations. Weight loss interventions have been shown to significantly improve IBS symptoms and quality of life in obese individuals. Similarly, dietary approaches, such as the low-FODMAP diet and the use of pre/probiotics, have demonstrated effectiveness in reducing symptoms, particularly in overweight or obese women, with professional guidance enhancing adherence and outcomes. Furthermore, the association between metabolic syndrome components and IBS symptoms suggests that screening for this condition may be beneficial in IBS patients. Several studies underscore the necessity of personalized management strategies that account for the heterogeneity of IBS subtypes and obesity phenotypes.

In summary, while some studies, particularly those focusing on specific subgroups or measures of central adiposity, report associations between obesity and IBS, the overall evidence is mixed, with some high-quality research finding no general association. Although mechanistic studies point towards shared pathways involving inflammation, gut microbiota, and metabolic factors, direct causal links have not been definitively established in most research.

Discussion

This systematic review aimed to synthesize the evidence on the association between obesity and Irritable Bowel Syndrome (IBS) in adults. The findings reveal a complex and nuanced relationship, moving beyond a simple question of direct correlation to a more sophisticated understanding shaped by specific clinical phenotypes, diagnostic criteria, and underlying biological mechanisms. While several high-quality meta-analyses concluded there is no significant overall association between an elevated Body Mass Index (BMI) and IBS, our synthesis of the literature demonstrates that this conclusion masks highly significant relationships within specific subgroups (Yau, et al., 2024).

The most compelling evidence for an association emerges when the heterogeneity of both obesity and IBS is considered. The link is not with obesity as a monolithic condition defined solely by BMI, but rather with more specific and metabolically significant measures of adiposity. The landmark Mendelian randomization study by Chen et al. (2024) provided robust genetic evidence for a causal association between visceral adiposity and an increased risk of IBS. This finding is critical, as it suggests that the location and metabolic activity of fat, rather than total body mass alone, are the key drivers of the relationship (Chen, et al., 2024).

This emphasis on visceral fat is strongly supported by clinical and observational data. The cross-sectional study by Abboud et al. (2025) found that central obesity, measured by waist circumference as a component of Metabolic Syndrome, was significantly associated with IBS symptoms, particularly diarrhea. This highlights that metabolic dysfunction, intrinsically linked to visceral adiposity, is a critical factor in the gut-brain axis

disturbances characteristic of IBS. The findings suggest a shared pathophysiology rooted in metabolic health rather than just weight (Abboud, et al., 2025).

Further reinforcing this point is the interventional study by Elrashidy et al. (2024), which demonstrated that targeted reduction of abdominal fat led to a significant improvement in IBS symptoms and quality of life. This provides direct evidence that addressing central adiposity can be a potent therapeutic strategy for IBS management. The positive outcomes from this trial lend practical, clinical weight to the mechanistic theories linking visceral fat to gastrointestinal dysfunction (Elrashidy, et al., 2024).

The specificity of the association also extends to the subtype of IBS. A meta-analysis by Neo et al. (2020) identified a significant positive association specifically between obesity and the diarrhea-predominant subtype of IBS (IBS-D). This finding suggests that the pathophysiological mechanisms linking obesity and IBS may be most relevant to pathways that accelerate colonic transit and alter fluid secretion, which are characteristic of IBS-D (Neo, et al., 2020).

The diagnostic criteria used for IBS also appear to be a significant moderator of the association. The meta-analysis by Yau et al. (2024), while finding no overall link, reported a statistically significant association between obesity and IBS when the diagnosis was made using the more stringent and recent Rome IV criteria. This may indicate that the Rome IV criteria identify a more specific IBS phenotype that is genuinely linked to obesity-related pathophysiology, whereas older, broader criteria may have diluted this association by including patients with different underlying issues (Yau, et al., 2024).

The convergence of evidence from genetic, clinical, and interventional studies points toward shared biological pathways as the foundation of the obesity-IBS link. Among the most frequently cited mechanisms are chronic low-grade inflammation and alterations to the gut microbiota. Obesity is well-established as a pro-inflammatory state, characterized by the increased production of inflammatory cytokines from adipose tissue, which can have systemic effects, including on the gastrointestinal system (Rawat, et al., 2024).

A key aspect of this shared pathophysiology is the role of systemic inflammation in modulating gut function. Adipose tissue, particularly visceral fat, is not an inert storage depot but an active endocrine organ that secretes a range of pro-inflammatory mediators. These inflammatory markers can circulate throughout the body and directly impact the enteric nervous system and intestinal barrier, contributing to two of the core features of IBS: visceral hypersensitivity and increased intestinal permeability (Mahadeva, 2023).

The inflammatory state in obesity may directly sensitize the nerve endings in the gut, lowering the threshold for pain and discomfort in response to normal stimuli like gas or intestinal contractions. This increased visceral hypersensitivity is a hallmark of IBS and explains why many patients experience pain disproportionate to the degree of intestinal activity. The inflammatory milieu fostered by obesity could therefore be a primary driver of the abdominal pain that defines the IBS experience (Rawat, et al., 2024).

Based on a review of current scientific literature, the general pro-inflammatory state linking obesity and IBS involves several specific, measurable markers. Key among these are the cytokines Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6), which are secreted by adipose tissue and are often found at significantly higher levels in patients with both conditions. The elevation in IL-6 also drives an increase in hepatic C-reactive protein (CRP), a well-known marker of systemic inflammation that positively correlates with BMI and IBS symptom severity. Furthermore, an imbalance in adipokines is critical; studies report higher levels of the pro-inflammatory hormone leptin and lower levels of the anti-inflammatory hormone adiponectin in overweight and obese IBS patients, which is associated with more severe abdominal pain and poorer quality of life. This systemic inflammation is often complemented by local gut inflammation, characterized by an increased number and activation of mast cells in the intestinal mucosa, which release mediators like histamine and contribute directly to visceral hypersensitivity and pain. (Hadi et al, 2016; Livzan et al, 2023; Shen et al, 2019).

Furthermore, chronic inflammation is known to compromise the integrity of the intestinal epithelial barrier. This can lead to increased permeability, or a "leaky gut," allowing luminal contents like bacteria and toxins to pass into the bloodstream, triggering further immune activation and perpetuating a cycle of inflammation. This mechanism is implicated in both the local gut symptoms and the systemic malaise often reported by patients with IBS, providing a direct biological link to the inflammatory state of obesity (Chen, et al., 2024).

The gut microbiota represents another critical mechanistic link. Both obesity and IBS are independently associated with significant alterations in the composition and function of the gut microbiome, a condition known as dysbiosis. The obese gut microbiome is often characterized by a reduced diversity of bacterial species and a shift in the balance of major phyla, which can impact energy extraction from the diet, gut motility, and immune regulation (Simanenkov, et al., 2019).

These microbial alterations can directly contribute to IBS symptomatology. For instance, certain bacterial populations can increase gas production through fermentation, leading to bloating and discomfort. Other changes can affect the production of short-chain fatty acids, which are crucial for maintaining gut health and regulating

motility. This shared pathway of dysbiosis offers a compelling explanation for the comorbidity and suggests that interventions targeting the microbiome could benefit both conditions (Oliveira, et al., 2022).

Diet plays a central role in modulating both inflammation and the gut microbiota, acting as a key environmental factor in the obesity-IBS relationship. Diets high in fat and processed foods, which are often associated with obesity, can promote a pro-inflammatory gut environment and contribute to dysbiosis. Conversely, dietary interventions have shown significant efficacy in managing IBS symptoms, particularly in overweight or obese individuals (Pogodina, et al., 2021)

The effectiveness of the low fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (low-FODMAP) diet in reducing IBS symptoms is well-documented and particularly relevant in this context. By reducing the intake of poorly absorbed carbohydrates, the diet limits the substrate available for bacterial fermentation, thereby reducing gas production, bloating, and pain. This provides a practical therapeutic tool that directly addresses the diet-microbiota-symptom axis in patients with both obesity and IBS (Oliveira, et al., 2022).

The clinical implications of these findings are significant. The strong evidence that weight loss and, more specifically, the reduction of abdominal fat can alleviate IBS symptoms provides a clear therapeutic target. The prospective cohort study by Aasbrenn et al. (2018) showed that a conservative weight loss program in morbidly obese individuals led to a significant improvement in IBS symptoms, underscoring the value of weight management as a primary or adjunctive therapy for IBS in this population (Aasbrenn, et al., 2018).

This review reinforces the need for a personalized and holistic approach to managing patients presenting with both obesity and IBS. Clinicians should look beyond BMI and consider measures of central adiposity and metabolic health, as these appear to be more strongly linked to IBS pathophysiology. Screening for components of Metabolic Syndrome may be warranted in IBS patients, as this could identify individuals who would most benefit from aggressive lifestyle and metabolic interventions (Abboud, et al., 2025).

The strength of this review lies in its synthesis of diverse and high-quality evidence, including multiple meta-analyses, a Mendelian randomization study, and clinical trials. By examining the evidence through the lens of specific phenotypes and mechanisms, it provides a clearer picture than what is offered by studies looking only at the overall association. This approach helps to resolve some of the inconsistencies in the literature.

Future research should focus on further elucidating the causal mechanisms, particularly the specific inflammatory and microbial pathways that link visceral fat to IBS-D and Rome IV-defined IBS. Longitudinal studies that track changes in inflammatory markers, gut microbiota composition, and IBS symptoms in response to targeted weight loss and dietary interventions are needed to confirm the directionality of these relationships.

In summary, while a general association between BMI-defined obesity and IBS is not consistently supported, this review reveals significant and clinically relevant links when focusing on specific phenotypes. There is strong evidence for a causal relationship between visceral adiposity and IBS risk, with a particular connection to the diarrhea-predominant subtype. This association appears to be driven by shared pathophysiological mechanisms, most notably chronic low-grade inflammation and gut dysbiosis. These findings strongly support the integration of weight management, particularly the reduction of central adiposity, into the therapeutic strategy for patients with co-occurring IBS and obesity.

CONCLUSION

In conclusion, the association between obesity and Irritable Bowel Syndrome (IBS) is a complex and specific relationship, rather than a universal link. While broad analyses using BMI often show no connection, compelling evidence from genetic and clinical studies reveals that visceral adiposity—the metabolically active fat stored around internal organs is the primary driver. This link is strongest for the diarrhea-predominant subtype of IBS (IBS-D) and is more evident when using the stringent Rome IV diagnostic criteria. The biological foundation for this connection lies in shared pathways where visceral fat promotes chronic low-grade inflammation and gut microbiome dysbiosis, which in turn increase gut sensitivity and permeability, core features of IBS.

These findings have direct clinical implications, providing a strong rationale for integrating weight management, specifically targeting central obesity, into the therapeutic strategy for affected IBS patients. Interventional studies have confirmed that reducing abdominal fat can significantly improve symptoms and quality of life. Clinicians are therefore encouraged to adopt a holistic approach that assesses metabolic health and central adiposity, rather than relying solely on BMI. Alongside weight management, dietary interventions like the low-FODMAP diet are validated tools, paving the way for more personalized treatments as research continues to unravel the precise mechanisms at play.

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