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REVIEW

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Prevalence of metabolic syndrome in patients with inflammatory bowel disease: a meta-analysis on a global scale

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Abstract

Background Metabolic syndrome (MetS) is a cluster of metabolic abnormalities that increase the risk of cardiovascular diseases (CVD). Patients with inflammatory bowel disease (IBD) may be at higher risk of developing MetS due to chronic inflammation, altered adipokine profiles, and the effects of corticosteroid treatment. However, the prevalence of MetS in IBD patients remains inconsistent across studies. This meta-analysis aims to estimate the prevalence of MetS in IBD patients and compare its occurrence between Crohn's disease (CD) and ulcerative colitis (UC).

Methods A systematic search was conducted across PubMed, Scopus, Embase, and Web of Science from their inception up to January 19, 2025. Eligible observational studies reporting MetS prevalence in IBD patients were included. Meta-analysis was performed using a random-effects model, with heterogeneity assessed via the I^2 statistic. Comprehensive Meta-Analysis (CMA) software, version 4.0 was used for analysis.

Results The pooled prevalence of MetS in IBD patients was 21.8% (95% CI: 14.3–31.6%). The prevalence was higher in UC patients (32.7%, 95% CI: 16.0–55.5%) compared to CD patients (14.1%, 95% CI: 8.6–22.3%). Patients with UC had significantly higher odds of MetS than those with CD (OR = 1.38, 95% CI: 1.03–1.85, $P = 0.02$). Additionally, IBD patients with MetS were significantly older than those without (MD: 9.89, 95% CI: 5.12–14.67, $P < 0.01$).

Conclusion In summary, this meta-analysis reveals a notable prevalence of MetS among patients with IBD, particularly in those with UC, where the prevalence is higher than in CD. The analysis also shows that IBD patients with MetS tend to be older, suggesting age as a contributing factor. These findings underscore the need for routine metabolic screening in IBD care, especially in UC and elderly patients.

Keywords Inflammatory bowel disease, Metabolic syndrome, Crohn's disease, Ulcerative colitis, Meta-analysis

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Introduction

Metabolic syndrome (MetS) refers to a cluster of interconnected metabolic abnormalities—such as central obesity, insulin resistance, high blood pressure, elevated triglyceride levels, and low levels of high-density lipoprotein (HDL) cholesterol—that together heighten the risk of developing cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), stroke, and increased overall mortality [1–7]. The global prevalence of metabolic syndrome (MetS) has been steadily increasing in recent decades, reflecting the widespread adoption of sedentary lifestyles, poor dietary habits, and the escalating rates of obesity. This has positioned MetS as a major public health concern worldwide [7–11].

In parallel, the incidence and prevalence of inflammatory bowel disease (IBD)—which encompasses Crohn's disease (CD) and ulcerative colitis (UC)—have also increased globally, especially in newly industrialized countries [12–15]. IBD is a chronic, immune-mediated condition characterized by relapsing inflammation of the gastrointestinal tract, leading to a wide range of gastrointestinal and systemic complications [15–17]. Beyond the primary intestinal manifestations, IBD patients frequently experience extraintestinal symptoms, including metabolic disturbances, hepatic involvement, and increased cardiovascular risk [18–21].

Recent research has suggested that individuals with IBD may be at an elevated risk for developing MetS [22, 23]. The pathophysiological mechanisms linking IBD to MetS are multifactorial and complex. Chronic low-grade systemic inflammation—a hallmark of IBD—plays a central role in disrupting insulin signaling and lipid metabolism, which may predispose patients to insulin resistance, dyslipidemia, and visceral adiposity [24–27]. Furthermore, the dysregulation of adipokines such as leptin and adiponectin, which are altered in both IBD and MetS, may mediate inflammation-driven metabolic dysfunction [7, 28–31]. Another contributing factor is long-term corticosteroid use, a common therapeutic strategy in IBD management, which is known to adversely affect glucose metabolism, promote weight gain, and increase the risk of hypertension and lipid abnormalities [32–35].

Despite the growing interest in this topic, estimates of the prevalence of MetS in IBD patients remain inconsistent across studies, with results varying depending on the population studied, diagnostic criteria for MetS, and the methods used to assess metabolic abnormalities [36–40]. Given these discrepancies and the clinical importance of identifying metabolic risk in IBD patients, a comprehensive synthesis of the existing literature is warranted. Therefore, the aim of this study is to conduct a systematic review and meta-analysis to estimate the overall prevalence of MetS in patients with IBD. Additionally, we compare prevalence rates between Crohn's disease and ulcerative colitis. By consolidating evidence from multiple studies, this analysis aims to provide clinicians with a clearer understanding of the metabolic burden in IBD populations and inform strategies for early screening and intervention.

Methods

This meta-analysis was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines and the Cochrane Handbook for Systematic Reviews [41, 42] (Table S1). The study protocol was registered on PROSPERO with the registration number CRD420250655049.

Search strategy

A comprehensive search was conducted across multiple electronic databases including PubMed, Scopus, Embase, and Web of Science, with no restrictions on language or publication date, from inception up to January 19,2025. The search terms included multiple keywords including "metabolic syndrome," "IBD," "inflammatory bowel disease," " Crohn's disease," and " Ulcerative colitis " combined with Boolean operators to ensure a broad search. The detailed search strategy for each database is provided in Table S2.

Study selection and eligibility criteria

Studies were included if they met the following criteria: [1] patients diagnosed with IBD, including CD and UC; [2] studies that reported the prevalence of MetS in IBD patients; [3] studies providing quantitative data on MetS components; [4] observational studies, including cohort, case-control, or cross-sectional designs. We excluded studies that did not report relevant data, conference abstracts, reviews, editorials, or studies on non-human subjects. Two independent reviewers (P.S, and S.M.H) (screened the titles and abstracts of identified articles, followed by full-text assessments to confirm eligibility. Discrepancies were resolved through discussion or consultation with a third reviewer (E.AS).

Quality assessment

The quality of included studies was independently assessed by two reviewers (P.S, and S.M.H), using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for observational studies [43–45]. Disagreements between reviewers were resolved through discussion or involvement of a third reviewer (E.AS) to reach a consensus.

Data extraction

Data were extracted from eligible studies using a standardized form. Extracted data included: author information, year of publication, country, sample size, age, gender

distribution, disease type (Crohn's disease or ulcerative colitis), and MetS criteria (e.g., NCEP ATP III, IDF).

Statistical analyses

Statistical analyses were performed using CMA software, version 4.0 [46]. Pooled estimates of the prevalence of metabolic syndrome in IBD patients were calculated using a random-effects model to account for between-study variability. The results were reported with 95% confidence intervals (CIs). Heterogeneity between studies was assessed using the I^2 statistic and Cochran's Q test, with I^2 values greater than 50% and a p-value of < 0.1 indicating significant heterogeneity. Meta-regression analyses were performed to explore potential sources of heterogeneity, including. Sensitivity analyses were conducted to assess the robustness of the results by systematically excluding each study. Publication bias was evaluated using funnel plots, and Egger's and Begg's tests were conducted to assess asymmetry. A p-value of < 0.1 was considered indicative of significant publication bias.

Results

Study selection

A total of 4,687 records were initially identified from the databases. After removing 2,175 duplicate records, 2,512 records remained for screening. Following the screening process, 2,484 records were excluded based on irrelevant content. Out of the remaining records, 28 reports were sought for retrieval, and all 28 reports were successfully retrieved. These reports were assessed for eligibility, with 8 reports excluded due to insufficient data, 5 reports excluded for not reporting Mets group data, and 3 reports excluded because they reported IBD prevalence in Mets. Ultimately, 12 new studies were included in the review (Fig. 1).

Study characteristics

This meta-analysis includes studies that evaluated the prevalence of MetS in patients with IBD, including both UC and CD (Table 1). A total of twelve studies were analyzed, comprising cross-sectional and retrospective cohort designs [22, 38–40, 47–54]. These studies were conducted across various countries, including Japan [40], Turkey [22], the United States [47, 51, 53, 54], Serbia [39, 50], South Korea [48], Italy [49, 52], and Portugal [38], covering time frames ranging from a few months to over a decade.

Sample sizes varied significantly, ranging from 78 [52] to a large nationwide database of over 1 million IBD cases (369,466 UC and 772,144 CD) [47]. The mean age of participants differed across studies, with some reporting separate values for UC and CD, while others provided a single mean age for all IBD patients. Gender distribution also varied, with most studies reporting a near-equal male-to-female ratio.

Several studies reported additional clinical characteristics, including disease duration, body mass index (BMI), and smoking status. The prevalence of MS among IBD patients demonstrated substantial variability, ranging from small cross-sectional cohorts with fewer than 20 cases [40] to large administrative datasets identifying over 100,000 cases [47]. These differences highlight variations in study design, sample size, and clinical settings. All included studies were considered high quality, based on their evaluation using the JBI Critical Appraisal Checklists. A detailed quality assessment for each study is provided in Table S3.

Results of meta-analysis

Total

The prevalence of Mets in patients with IBD included was 21.8% (95% CI: 14.3–31.6%) (Fig. 2A). The results were accompanied by significant heterogeneity (I^2 = 97.24%, $P < 0.01$). The sensitivity analysis demonstrated the stability of the results after removing individual studies, showing that the overall estimate remains consistent despite exclusions (Fig. 2B). The prediction interval analysis suggested that the true effect size in 95% of comparable populations falls between 4.00% and 68.00% (Fig. 2C).

Prevalence of Mets in UC and CD

The prevalence of Mets in patients with UC was 32.7% (95% CI: 16.0–55.5%) (Fig. 3.A). The results were accompanied by significant heterogeneity (I^2 = 97.54%, $P < 0.01$). The sensitivity analysis demonstrated the stability of the results after removing individual studies, showing that the overall estimate remains consistent despite exclusions (Fig. 3.B). The prediction interval analysis suggested that the true effect size in 95% of comparable populations falls between 2.0% and 94.0% (Fig. 3.C).

The prevalence of Mets in patients with CD was 14.1% (95% CI: 8.6–22.3%) (Fig. 4.A). The analysis revealed significant heterogeneity (I^2 = 92.39%, $P < 0.01$). The sensitivity analysis showed that the overall estimate remained consistent even after removing individual studies (Fig. 4.B). The prediction interval analysis indicated that the true effect size in 95% of comparable populations ranges from 2.0 to 52.0% (Fig. 4.C).

Patients with UC had significantly higher Mets prevalence compared to those with CD (OR = 1.38, 95% CI: 1.03 to 1.85, P = 0.02) (Fig. 5.A). The sensitivity analysis showed no significant difference between UC and CD regarding Mets after the removal of M. Carr, 2017; Sztembis, 2018; and Yorulmaz, 2011 (Fig. 5.B). Publication bias was not significant based on both Egger's test ($P = 0.26$) and Begg's test ($P = 0.13$). However, the trim and fill analysis identified two studies on the left side of

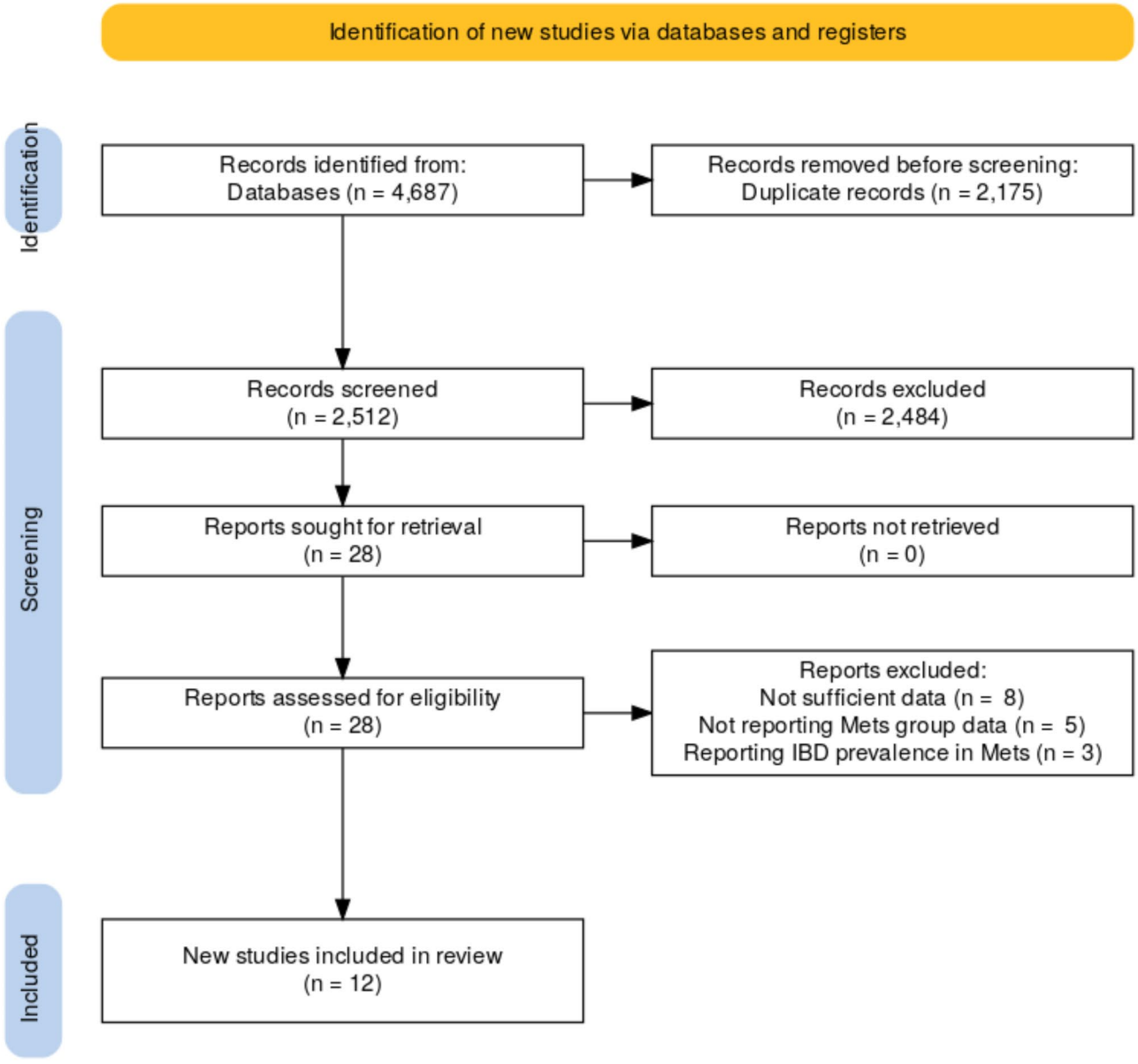

Fig. 1 Study selection process

the funnel plot, with an OR of 1.26 (95% CI: 0.93 to 1.71) (Fig. 5.C). The prediction interval analysis suggested that the true effect size in 95% of comparable populations falls between 0.70 and 2.74 (Fig. 5.D).

Comparison of the age of IBD patient with and without Mets

Patients with Mets were significantly older than those without Mets, with a mean difference (MD) of 9.89 years (95% CI: 5.12 to 14.67, $P < 0.01$) (Fig. 6). The I^2 statistic was 0.00%, with a P-value of 0.7. Due to the limited number of studies, evaluation of publication bias, prediction intervals, and sensitivity analysis was not possible.

Discussion

This meta-analysis underscores a critical issue in the management of IBD: the substantial prevalence of MetS in this patient population. Our findings reveal that over one-fifth of individuals with IBD meet the criteria for MetS, reinforcing the notion that chronic inflammation, metabolic dysregulation, and treatment-related effects create a unique metabolic phenotype distinct from that seen in the general population. Notably, UC patients exhibit a significantly higher prevalence of MetS (32.7%) compared to those with CD (14.1%), suggesting that disease-specific mechanisms, including inflammatory burden, gut microbiota alterations, and pharmacologic exposure, play a decisive role in metabolic risk.

Table 1 Characteristics of included studies

First author	Year of publication	Time set	Study Design	Mean Age IBD (UC/CD)	Population IBD (UC/CD)	Gender Distribution IBD (M/F)	The country	Disease duration IBD (UC/CD)	BMI (UC/CD)	MetS in IBD frequency (UC/CD)	Smoking IBD (UC/CD)	Quilty	Prevalence report
Nagahori et al. **(40)**	2010	December 2008 to May 2009	Cross-sectional	(43.6 ± 13.5/31.5 ± 8.1)	102 (74/28)	31/71	Japan	(6.9 ± 7.1/8.7 ± 9.0)	(23 ± 3.93/22.7 ± 5.14)	19 (17/2)	15 (9/6)	High	Point prevalence
Yorulmaz et al. (55)	2011	June 2009 to 2011	Cross-sectional	(43.93 ± 13.59/36.74 ± 13.88)	177(115/62)	100/77	Turkey	(50.18 ± 55.59/47.71 ± 70.16)	(26.27 ± 4.89/23.67 ± 5.18)	45 (34/11)	51 (22/29)	High	Point prevalence
Njeim et al. (47)	2024	2016 to 2020	Retrospective cohort	54.51 (52.56/56.35)	N. A	369,466/772,144	USA	N. A	N. A	147,598 (68455/79143)	N. A	High	Period prevalence
Fitzmorris et al. (54)	2015	2000 to 2012	Retrospective cohort	N. A	868 (0/868)	N. A	USA	N. A	N. A	37 (0/37)	N. A	High	Period prevalence
Dragasevic et al. (39)	2020	N. A	Cross-sectional	(43.5 ± 14.5/35 ± 13.25)	104 (54/50)	57/47	Serbia	N. A	N. A	34 (16/18)	N. A	High	Point prevalence
M. Carr et al. (53)	2017	January 1997 to December 2011	Retrospective cohort	52.4 ± 14.5	84 (24/60)	37/47	USA	N. A	N. A	19 (7/12)	N. A	High	Period prevalence
Jovanovic et al. (50)	2019	N. A	Cross-sectional	N. A	89(89/0)	52/37	Serbia	N. A	N. A	89 (72/0)	42(42/0)	High	Point prevalence
Kang et al. (48)	2020	2004 to 2017	Retrospective cohort	39.3 ± 15.47	443 (169/274)	N. A	South korea	(87.4 ± 64.3/91.3 ± 48.6)	N. A	47	N. A	High	Period prevalence

Table 1 (continued)

First author	Year of publication	Time set	Study Design	Mean Age IBD (UC/CD)	Population IBD (UC/CD)	Gender Distribution IBD (M/F)	The country	Disease duration IBD (UC/CD)	BMI (UC/CD)	MetS in IBD frequency (UC/CD)	Smoking IBD (UC/CD)	Quilty	Prevalence report
Magrì et al. (49)	2019	December 2016 to January 2018	Cross-sectional	49.73 ± 13.26	178 (95/83)	97/81	Italy	134.16 ± 99.24	21.0 ± 3.24	34	N. A	High	Point prevalence
Arieiraa et al. (38)	2018	January to March 2017	Cross-sectional	40.6 ± 12.8	161 (60/101)	75/86	Portugal	91.5 ± 77.4	25.1 ± 3.9	21	35	High	Point prevalence
Sartini et al. (52)	2018	March 2012 and March 2016	Cross-sectional	51.19 ± 11.82	78 (36/42)	49/29	Italy	99.9 ± 91.1	N. A	18	N. A	High	Point prevalence
Sztembis et al. (51)	2018	2016 to 2017	Cross-sectional	N. A	116 (48/68)	N. A	USA	N. A	N. A	31 (17/14)	N. A	High	Point prevalence

IBD (Inflammatory Bowel Disease), UC (ulcerative colitis), CD (Crohns disease), MetS (Metabolic Syndrome), N.A (Not Available)

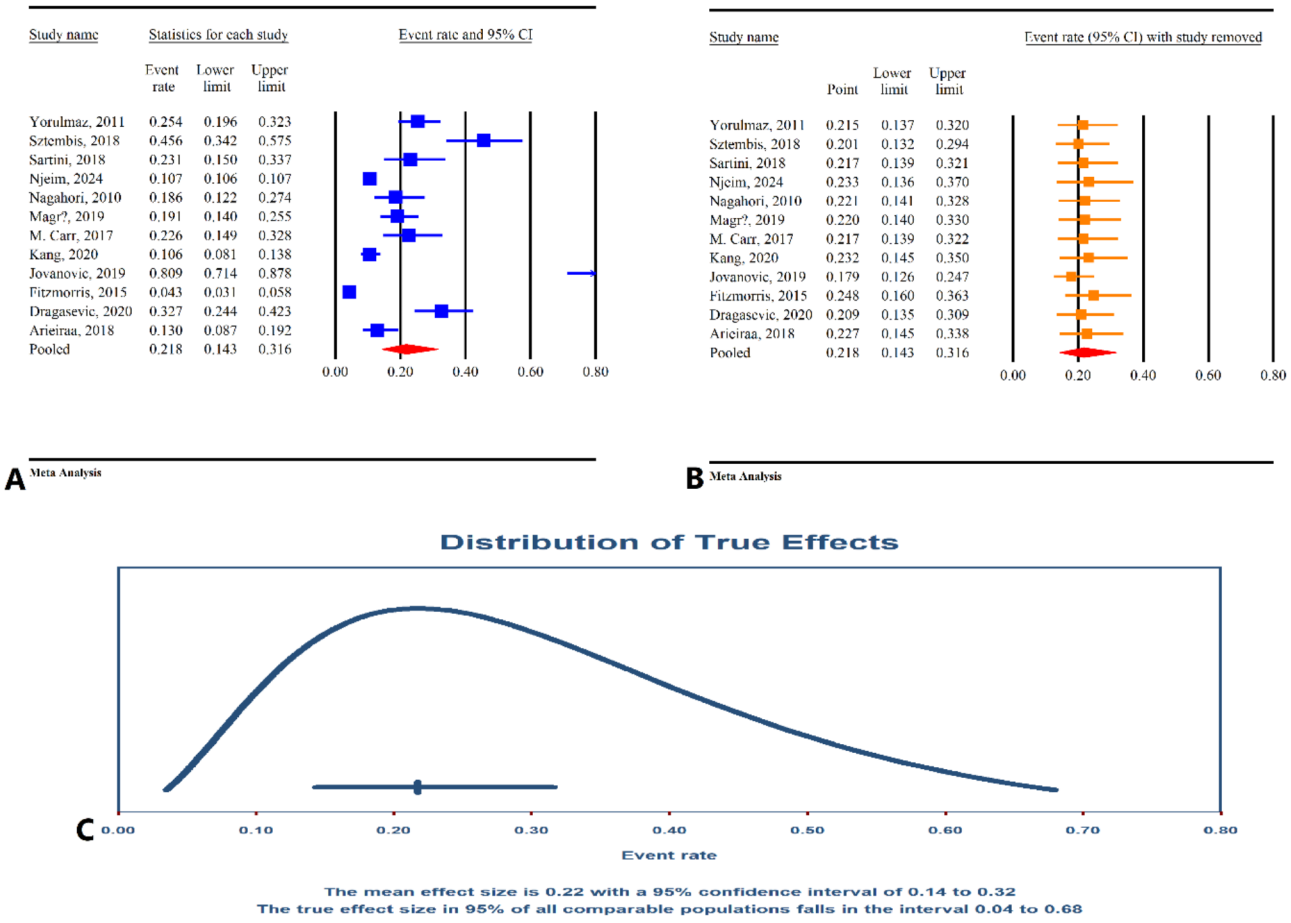

Fig. 2 Prevalence of Mets in Mets in patients with IBD. A: Forest plot B: sensitivity analysis C: Prediction interval analysis

Several studies have consistently demonstrated a robust association between MetS and IBD, indicating that patients with IBD are at an elevated risk for metabolic abnormalities. Epidemiological research underscores that IBD patients exhibit a higher propensity for developing these metabolic disturbances compared to the general population, thereby increasing their overall cardiovascular risk.

For instance, Hyun and Cheon explore the interplay between these conditions, highlighting shared inflammatory and metabolic dysfunctions. Although the overall prevalence of MetS components may be lower in IBD patients compared to non-IBD individuals, the chronic inflammatory state in IBD significantly amplifies cardiovascular risks. The authors advocate for early detection and a multidisciplinary management strategy to address the metabolic complications associated with IBD, ultimately improving patient outcomes [7, 20]. In a complementary study, Njeim et al. observed that, a higher metabolic score—reflecting the cumulative presence of MetS components—correlated strongly with increased risks of CVD in both UC and CD patients. These findings underscore the urgent need for comprehensive metabolic and cardiovascular risk management in IBD [47].

Both MetS and IBD are becoming increasingly prevalent worldwide [55, 56]. The pathophysiology of MetS in IBD is fundamentally driven by chronic systemic inflammation, which extends beyond the gut to disrupt metabolic homeostasis [47, 57]. Unlike classic obesity-associated MetS, in which excess adiposity triggers metabolic dysfunction, IBD-associated MetS appears to be primarily inflammation-driven, leading to distinct metabolic consequences. Elevated levels of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP), hallmark features of IBD-related inflammation [58–60], have been shown to impair insulin receptor signaling, promote hepatic gluconeogenesis, and reduce glucose uptake in peripheral tissues, resulting in insulin resistance and hyperglycemia Furthermore, chronic inflammation contributes to endothelial dysfunction, arterial stiffness, and dyslipidemia, all of which are well-established precursors to CVD [61–64] (Fig. 7).

Beyond systemic inflammation, adipose tissue dysfunction further exacerbates metabolic dysregulation in IBD. Adipose tissue, an active endocrine organ, plays a pivotal

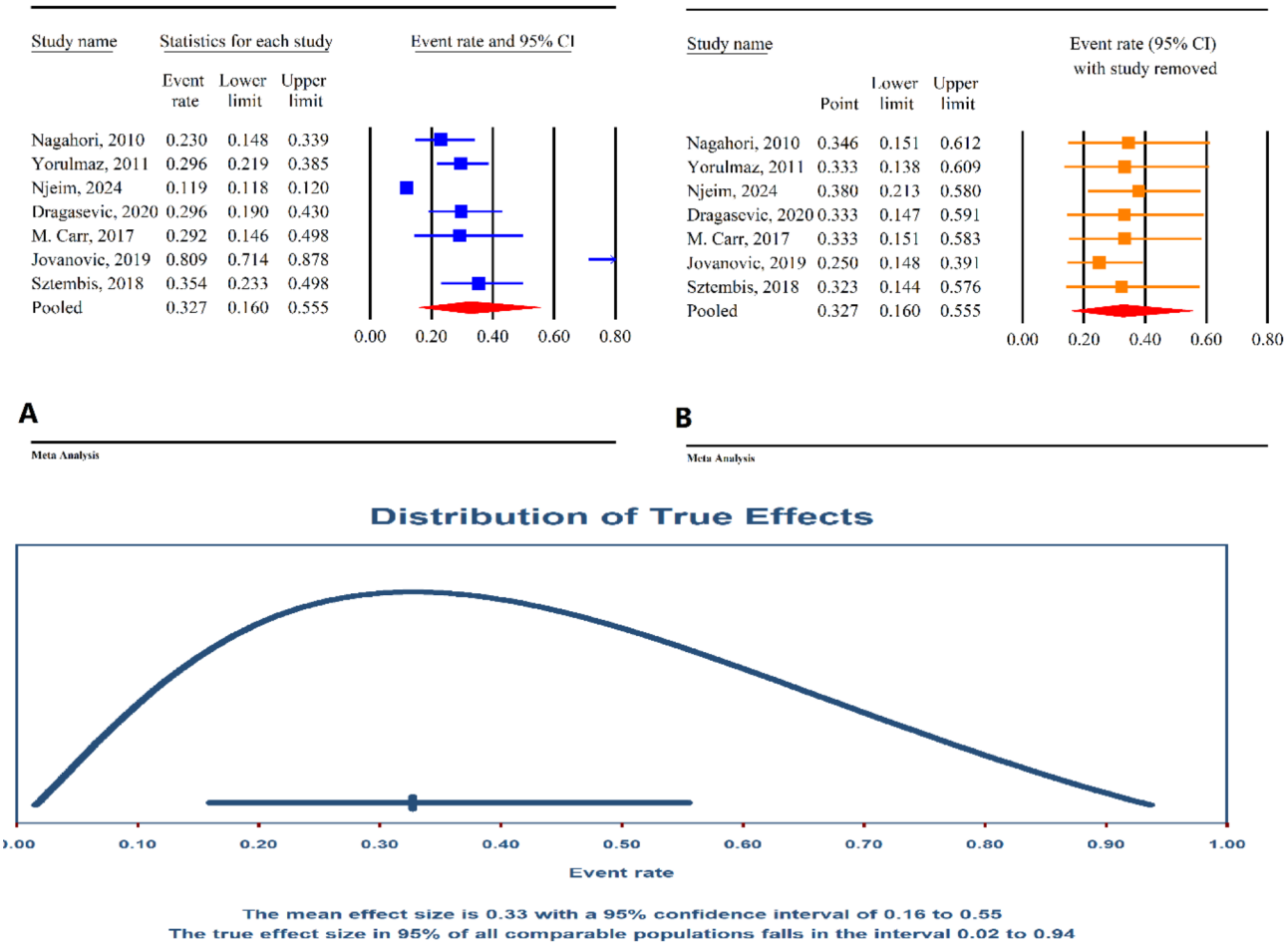

Fig. 3 Prevalence of Mets in Mets in patients with UC. A: Forest plot B: sensitivity analysis C: Prediction interval analysis

role in regulating glucose and lipid metabolism through adipokine secretion. In IBD, this regulatory function is altered, with elevated leptin and reduced adiponectin levels—a pattern strongly linked to worsening insulin resistance, visceral adiposity, and lipid abnormalities [65, 66]. Leptin, a pro-inflammatory adipokine, amplifies TNF-α production, macrophage activation, and central obesity, while adiponectin, a protective anti-inflammatory adipokine, is paradoxically reduced, further impairing metabolic homeostasis. These disruptions collectively contribute to an inflammatory-metabolic state that predisposes IBD patients to MetS, even in the absence of significant weight gain [67–69].

Another critical factor is gut microbiota dysbiosis, a well-documented feature of both UC and CD [70, 71]. The gut microbiome plays an essential role in immune regulation, energy metabolism, and bile acid processing. In IBD, a reduction in short-chain fatty acid (SCFA)-producing bacteria, particularly butyrate, weakens insulin sensitivity and promotes lipid dysregulation [72, 73]. Concurrently, an increase in pro-inflammatory bacterial strains leads to intestinal permeability, systemic endotoxemia, and chronic immune activation, all of which contribute to metabolic dysfunction. Moreover, altered bile acid metabolism in IBD disrupts lipid absorption and hepatic glucose regulation, further compounding MetS risk [14, 74–77].

Pharmacologic therapies, particularly corticosteroids, also exert significant metabolic consequences. While essential for controlling inflammation during acute IBD flares, long-term corticosteroid exposure is strongly linked to increased visceral adiposity, dyslipidemia, insulin resistance, and hypertension [78]. Corticosteroids promote hepatic gluconeogenesis and impair peripheral glucose uptake, predisposing patients to steroid-induced diabetes and metabolic dysfunction [79]. In contrast, TNF-α inhibitors have shown potential benefits by improving insulin sensitivity through inflammation reduction [80, 81], although their overall metabolic impact remains incompletely characterized. These findings underscore the need for careful consideration of long-term corticosteroid use in IBD management, particularly in patients with preexisting metabolic risk factors [82–85].

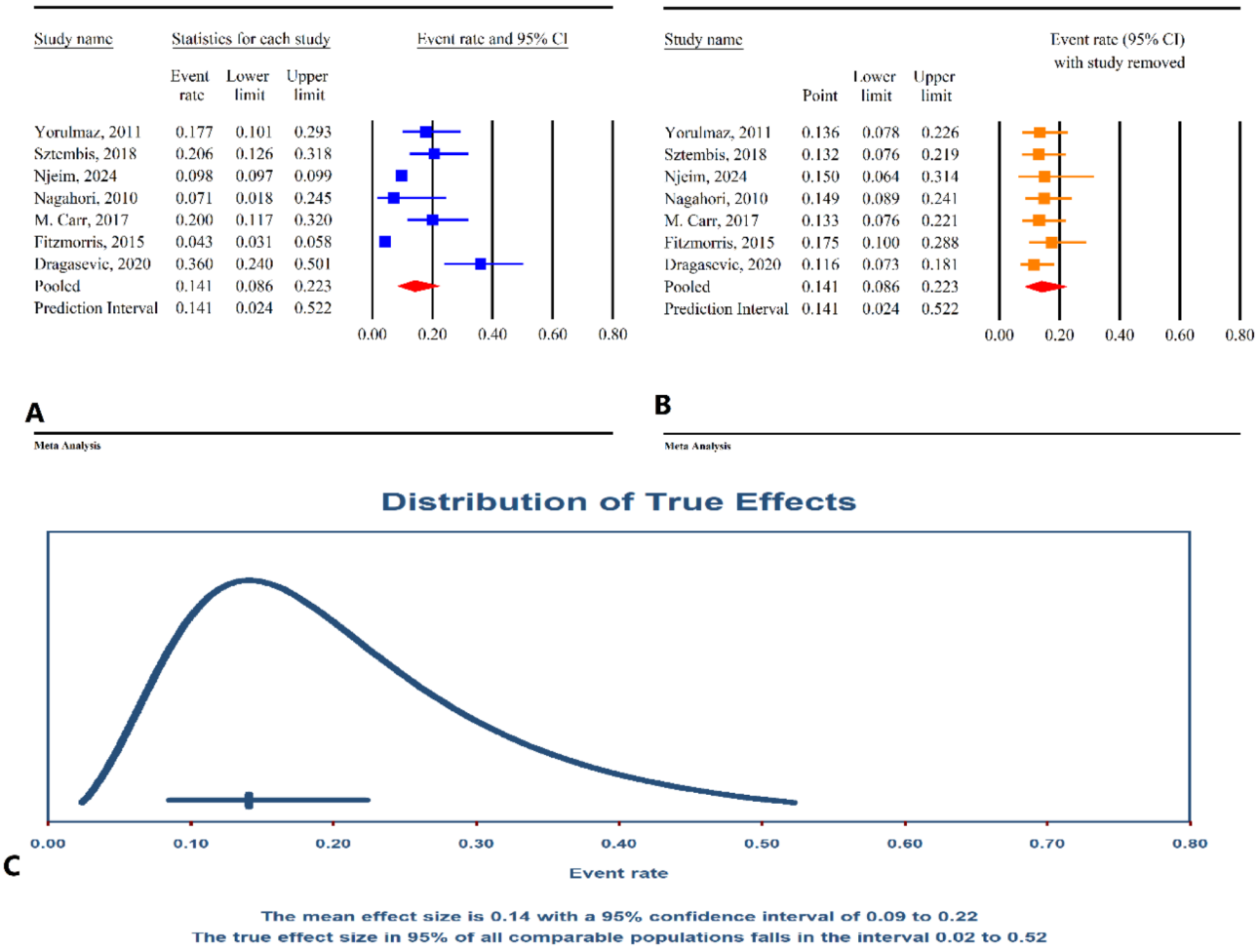

Fig. 4 Prevalence of Mets in Mets in patients with CD. A: Forest plot B: sensitivity analysis C: Prediction interval analysis

The significantly higher prevalence of MetS in UC compared to CD suggests that distinct disease characteristics shape metabolic risk. UC is characterized by continuous colonic inflammation, which directly influences gut microbiota composition, immune responses, and metabolic regulation [86]. Conversely, CD primarily affects the small intestine, leading to nutrient malabsorption, altered bile acid metabolism, and energy deficiency, which may exert different metabolic consequences compared to UC [87, 88] (Fig. 7).

Another key differentiator is systemic inflammatory burden, which is markedly higher in UC, as evidenced by persistently elevated CRP, TNF-α, and IL-6 levels. These inflammatory mediators directly impair insulin signaling, promote endothelial dysfunction, and drive lipid abnormalities, exacerbating MetS risk [89–91]. According to Njeim et al., the inflammatory pattern in UC is markedly different from that in CD. In UC, continuous inflammation confined to the mucosal layer leads to elevated levels of inflammatory markers, indicating that inflammation is more pronounced in UC. This sustained, mucosal-restricted inflammation may explain the higher systemic inflammatory burden and metabolic disturbances observed in UC patients compared to those with CD, which is characterized by transmural and discontinuous lesions [47] (Fig. 7).

Pharmacologic factors also play a crucial role in shaping this disparity. Corticosteroid therapy is more frequently used in UC, contributing to greater metabolic burden compared to CD, where biologic therapies such as TNF-α inhibitors are more commonly employed. Given their potential benefits in improving insulin sensitivity and reducing inflammation, biologics may confer a metabolic advantage in CD relative to UC [7, 92, 93]. Surgical interventions further differentiate metabolic outcomes. Colectomy with ileal pouch-anal anastomosis (IPAA), a common procedure in UC, has been linked to altered microbiota composition, nutrient absorption, and bile acid metabolism, all of which may influence metabolic pathways [94]. In contrast, small bowel resections in CD often result in malabsorption-driven metabolic shifts rather than classical MetS phenotypes [95] (Fig. 7).

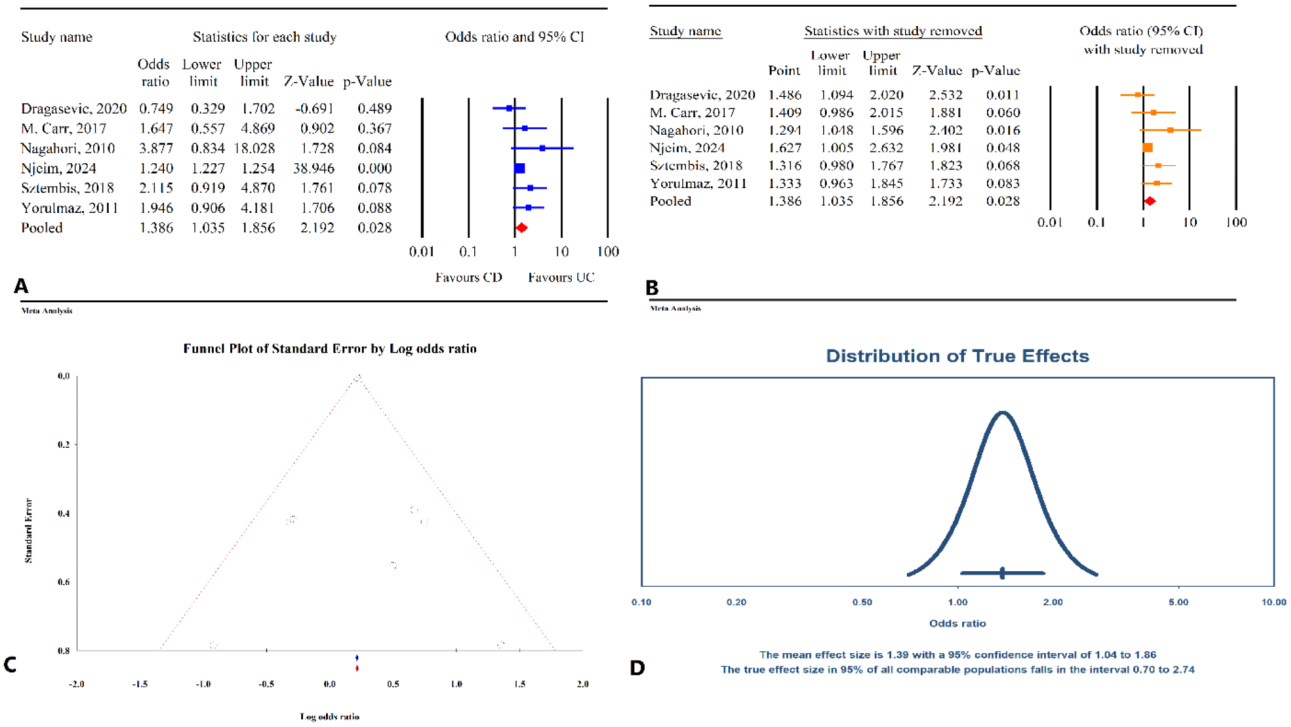

Fig. 5 Comparison of the prevalence of Mets in Mets in patients with CD and UC. A: Forest plot B: sensitivity analysis C: Trim and fill analysis D: Prediction interval analysis

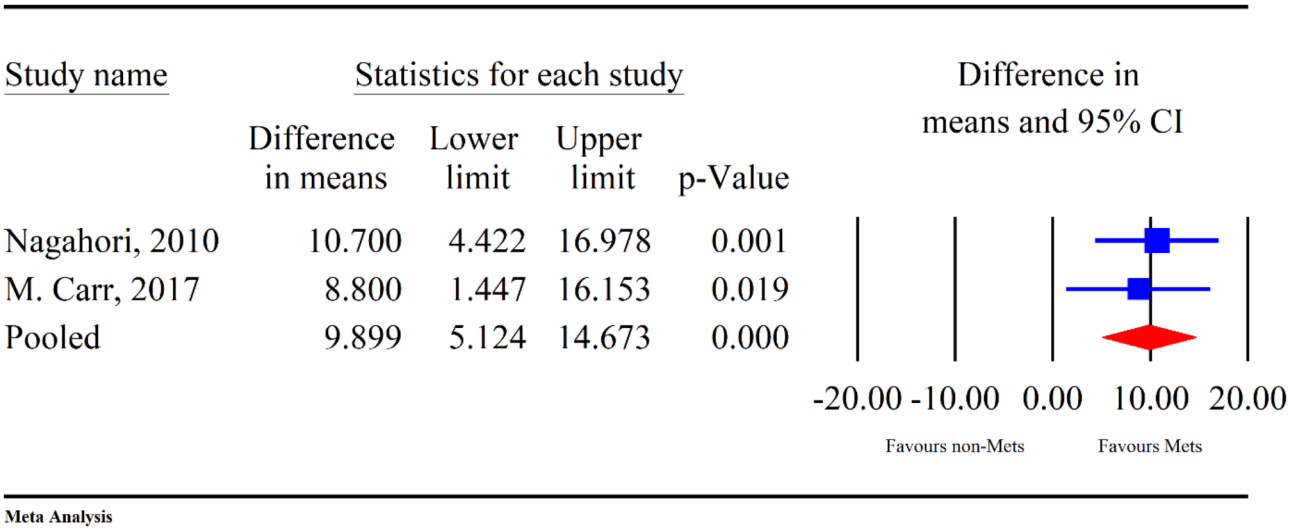

Fig. 6 Comparison of the age of IBD patient with and without Mets

Our findings also revealed that age plays a crucial role in MetS development among IBD patients, with affected individuals being nearly 10 years older, on average, than those without MetS. This aligns with well-established metabolic principles, as aging is inherently linked to insulin resistance, adiposity redistribution, and increased systemic inflammation—all of which predispose individuals to MetS [96–98].

In the context of IBD, the metabolic impact of aging may be further compounded by prolonged exposure to disease-related inflammation and long-term medication use. Older IBD patients are more likely to have undergone multiple rounds of corticosteroid therapy, experienced cumulative inflammatory insults, and exhibited declining physical activity levels due to disease-related fatigue or mobility limitations. These factors may accelerate metabolic deterioration, placing older individuals

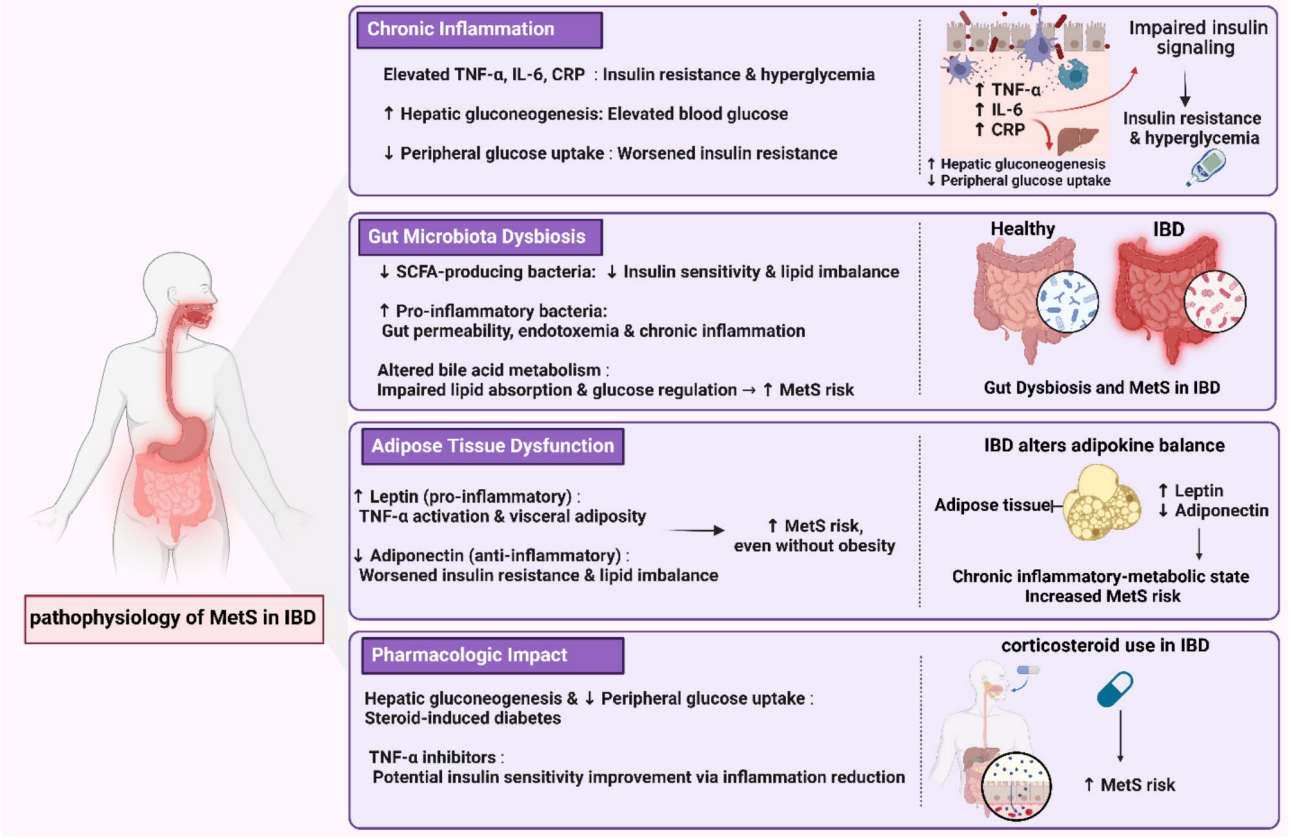

Fig. 7 Potential mechanism of action

at an even greater risk for cardiovascular and metabolic complications [99–102].

The implications of this age-related MetS risk are substantial. Given that IBD itself is increasingly recognized as a systemic disease with extraintestinal manifestations, clinicians should incorporate metabolic assessments as part of routine long-term disease monitoring, particularly for older patients who may already be vulnerable to cardiovascular complications [103].

From a clinical perspective, our findings reinforce the necessity of integrating metabolic screening into standard IBD management protocols. Traditionally, IBD treatment has been focused on controlling gastrointestinal inflammation and preventing disease progression [104], but our results highlight an urgent need to expand this focus to include metabolic health. For UC patients—who appear to be at greater risk of MetS —this means closer lipid and glucose monitoring, early lifestyle interventions, and careful consideration of long-term corticosteroid use. In older IBD patients, tailored metabolic risk assessments should be incorporated into routine follow-ups, ensuring that cardiovascular and metabolic risks are addressed alongside gut health.A multidisciplinary approach that involves gastroenterologists, endocrinologists, dietitians, and primary care providers is likely the most effective way to mitigate these risks. Lifestyle interventions, including personalized nutrition plans, structured physical activity programs, and weight management strategies, should be considered part of comprehensive IBD care.

Limitations and future directions

This meta-analysis has several limitations that should be considered when interpreting the findings. First, significant heterogeneity was observed among the included studies. This can be attributed to substantial variability in study design, patient characteristics, geographic regions, and diagnostic criteria used to define MetS. Although we performed sensitivity analyses and meta-regression, the sources of heterogeneity could not be fully accounted for.

Second, most studies included in this meta-analysis were cross-sectional, limiting the ability to establish causal relationships between IBD and MetS. Longitudinal cohort studies are needed to determine whether MetS develops as a consequence of chronic inflammation, medication effects, or other metabolic alterations in IBD patients.

Third, confounding factors such as dietary habits, physical activity, medication use (especially corticosteroids and biologics), and genetic predisposition were not consistently reported, making it difficult to assess

their impact on MetS development. Future studies should control for these variables to provide more robust conclusions.

Fourth, the meta-analysis is based solely on published observational studies, which introduces the potential for publication bias. Studies that reported no significant association between MetS and IBD may be underrepresented in the literature. While Egger's test, Begg's test, and trim-and-fill analysis were performed and showed no significant bias, the possibility of subtle underreporting cannot be excluded.

Lastly, while our analysis found that IBD patients with MetS were significantly older than those without, the underlying mechanisms linking age and MetS in this population remain a key knowledge gap. Factors such as chronic inflammation, long-term corticosteroid use, and age-related metabolic alterations are likely contributors, but were not systematically addressed in the included studies.

To address these limitations, future research should focus on standardizing MetS diagnostic criteria across studies to improve comparability. Large-scale, multicenter longitudinal studies are needed to explore causal links between IBD and MetS, as most of the current studies are cross-sectional and do not establish causality. Additionally, future studies should better control for confounding factors such as diet, physical activity, medication use (especially corticosteroids and biologics), and genetic predisposition, which were inconsistently reported in the current analysis. Investigating the role of inflammation control and medication effects on MetS development in IBD patients would also provide more insight into the underlying mechanisms. Furthermore, it is essential to incorporate patient lifestyle factors, including diet and exercise, as well as comorbidities, to better understand modifiable risks. Finally, exploring potential therapeutic interventions, such as anti-inflammatory treatments and metabolic-targeted therapies, could help reduce MetS risk in IBD populations. By addressing these gaps, future research can enhance our understanding of the relationship between IBD and MetS and contribute to improving clinical management and patient outcomes.

Conclusion

This meta-analysis highlights a significant prevalence of MetS in patients with IBD, particularly in those with UC. The pooled prevalence of MetS in IBD patients was 21.8%, with UC patients demonstrating a higher prevalence compared to those with CD. Additionally, IBD patients with MetS were significantly older than those without, suggesting a potential age-related metabolic risk.

These findings underscore the importance of routine metabolic screening in IBD patients, particularly for those with UC and older individuals. Given the role of chronic inflammation, gut microbiota dysbiosis, corticosteroid use, and altered adipokine profiles in MetS development, multidisciplinary management strategies integrating gastroenterologists, endocrinologists, and nutritionists may improve patient outcomes. Early identification and intervention, including lifestyle modifications, targeted pharmacotherapy, and personalized treatment plans, could help mitigate the long-term cardiovascular and metabolic risks associated with MetS in IBD patients. Further research is warranted to explore the underlying mechanisms linking IBD and MetS, the impact of different therapeutic interventions, and the role of inflammation control in reducing metabolic risk.

Abbreviations

BMI	Body Mass Index
CD	Crohn's Disease
CIs	Confidence Intervals
CMA	Comprehensive Meta–Analysis
CVD	Cardiovascular Disease
CRP	C–reactive Protein
IBD	Inflammatory Bowel Disease
IDF	International Diabetes Federation
IL	6–Interleukin–6
IPAA	Ileal Pouch–Anal Anastomosis
MetS	Metabolic Syndrome
MD	Mean Difference
NCEP ATP III	National Cholesterol Education Program Adult Treatment Panel III
N.A	Not Available
OR	Odds Ratio
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta–Analyses
SCFA	Short–Chain Fatty Acids
T2DM	Type 2 Diabetes Mellitus
TNF	α–Tumor Necrosis Factor Alpha
UC	Ulcerative Colitis

Supplementary Information

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Supplementary Material 1

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Author contributions

The research concept was originally conceived by E.AS, A.G, K.V.J, and N.R.K. The study design, including the methodological framework needed to achieve the outcomes, was managed by S.M.H, A.G H.B.P and P.S. Supervision, coordination, and manuscript preparation were overseen by E.AS, A.P and A.G. Data collection and processing—encompassing experiments, patient management, data analysis, and interpretation—were undertaken by K.V.J, P.S, S.M.H, and A.E. All authors made substantial contributions to drafting the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

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