

Repurposing glucagon-like peptide-1 receptor agonists in inflammatory bowel disease: clinical and metabolic outcomes from a systematic review and meta-analysis

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Abstract

Background Obesity is increasingly prevalent among individuals with inflammatory bowel disease (IBD). Glucagon-like peptide-1 receptor agonists (GLP-1RAs) induce clinically meaningful weight loss and may exert anti-inflammatory effects in the gut, but their effectiveness in modifying the course of IBD remains uncertain.

Methods We conducted a systematic review and meta-analysis of studies that enrolled adults with IBD who were treated with a GLP-1RA, searching PubMed, Scopus and CENTRAL from inception to 30 June 2025. Primary outcomes were IBD-related surgery, hospitalization, oral and intravenous corticosteroid use, escalation to advanced therapy and intestinal obstruction. We performed random-effects meta-analyses and reported pooled odds ratios (ORs) with 95% confidence intervals (CIs). Risk of bias was assessed with ROBINS-I for observational studies and RoB 2 for randomized trials.

Results Thirteen studies met the inclusion criteria—12 observational and 1 randomized controlled trial (RCT)—comprising 74,513 patients overall; 3 studies contributed data suitable for meta-analysis. Pooled estimates demonstrated a significantly lower odds of IBD-related surgery among GLP-1RA recipients (OR 0.46, 95%CI 0.34-0.61). Estimates for hospitalization, corticosteroid use, therapy escalation and intestinal obstruction were not statistically significant. Across studies, GLP-1RA treatment consistently achieved weight reduction in patients with IBD, aligning with established efficacy in general obesity care.

Conclusions GLP-1RA therapy may reduce the need for surgery in IBD and consistently lower weight. Well-powered RCTs should clarify treatment effect, safety and how best to integrate GLP-1RAs agents into IBD care.

Keywords Inflammatory bowel disease, glucagon-like peptide-1 receptor agonists, obesity, systematic review, meta-analysis

Ann Gastroenterol 2026; 39 (XX): 1-13

Conflict of Interest: None

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Received 20 January 2025; accepted 26 July 2026;
published online 30 September 2026

DOI: <https://doi.org/10.20524/aog.2026.1115>

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Introduction

Inflammatory bowel disease (IBD), which includes ulcerative colitis (UC) and Crohn's disease (CD), is a chronic immune-mediated condition associated with a substantial disease burden. IBD affects nearly 5 million people worldwide, with prevalence rising in Western countries and newly industrialized regions such as Asia, the Middle East and South America [1]. The therapeutic landscape has expanded beyond aminosaliclates, corticosteroids and conventional immunosuppressants. It now includes biologic therapies targeting tumour necrosis factor (TNF), interleukin (IL)-12/23 and integrins, as well as oral small molecules such as

Janus kinase inhibitors and sphingosine-1-phosphate receptor modulators [2,3]. Together, these options have improved clinical and endoscopic outcomes for many patients, and increased the likelihood of achieving steroid-free remission.

However, the response is incomplete and often not durable. Up to a third of patients fail biologic induction, and others lose response or discontinue therapy because of adverse events or intolerance [4]. Flares, hospitalization and surgery remain common. Ongoing challenges also include treatment-related toxicities and the consequences of chronic inflammation [5]. These limitations have prompted a growing interest in novel adjunctive therapies to enhance efficacy and safety in IBD management.

One such area of interest stems from the increasing overlap between IBD and obesity, which now affects up to 40% of patients, with an additional 25–40% classified as overweight [6]. Visceral adipose tissue is an immunometabolic organ that promotes systemic inflammation through mediators such as leptin, TNF- α and IL-6. It may also disrupt the gut microbiota and intestinal barrier, thereby contributing to persistent intestinal inflammation and fibrosis [7]. Observational studies link obesity to more active or complicated disease, a poorer response to therapy, and higher surgical risk, though findings are inconsistent [8]. Excess adiposity may also influence drug pharmacokinetics and pharmacodynamics, potentially attenuating the effectiveness of biologic therapies and small-molecule agents [9,10]. Taken as whole, these concerns have strengthened interest in treatments that can both promote weight loss and favorably modulate inflammatory pathways in patients with IBD.

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), developed for type 2 diabetes and obesity, are of interest as adjunctive therapy in IBD with metabolic comorbidity. By enhancing incretin signaling, delaying gastric emptying, suppressing appetite and influencing energy expenditure, they improve glycemic control and induce meaningful weight loss. Experimental and early clinical evidence suggests additional anti-inflammatory and immunomodulatory effects. These include reduced production of proinflammatory cytokines, support of regulatory immune cell populations, and improved intestinal barrier function through enhanced epithelial integrity and mucosal healing [11]. Proposed mechanisms include a shift toward M2 macrophage polarization and expansion of regulatory T cells. Additional pathways may involve suppression of NF- κ B-mediated inflammatory signaling in T cells and improved epithelial barrier function, including enhanced tight-junction integrity and mucin production [12]. In IBD, GLP-1RAs may therefore influence key pathogenic pathways, such as chronic inflammatory circuits, tight junction dysfunction and gut microbiome dysbiosis. This provides a

biological rationale for their evaluation as adjunctive therapy in IBD patients with coexisting diabetes or obesity.

Clinical data on GLP-1RAs in IBD are still limited and mostly come from retrospective studies in patients treated for diabetes or obesity, but those studies had important issues, such as confounding and non-standardized IBD outcomes. These gaps, together with ongoing concerns about gastrointestinal side effects and possible disease flares, highlight the need for a systematic review and meta-analysis to clarify how effective and safe GLP-1RAs are for patients with IBD.

Materials and methods

Search strategy and selection criteria

This systematic review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Supplementary Table 1) [13]. We conducted a search of PubMed, Scopus, and Cochrane Library (all databases) up to 30 June 2025 using an assistive Large Language Model (LLM) tool (Synthesa AI). Although our dataset was small, we used a LLM tool to assist screening. The goal was to ease reviewer workload by triaging obvious exclusions and to test the tool in a human-in-the-loop process. After duplicate removal, Synthesa AI was used only to support title and abstract triage by identifying records that appeared clearly irrelevant according to the prespecified eligibility criteria. The tool did not make final eligibility decisions and no record was excluded solely on the basis of the tool output. Two reviewers (CM and DP) screened titles and abstracts independently. Only humans advanced records to full text and made all full text decisions, while disagreements were resolved by discussion. We included studies that enrolled adults (≥ 18 years) who had a diagnosis of IBD and reported IBD-specific and/or metabolic outcomes after GLP-1RA exposure. The comparator group consisted of patients with IBD who did not receive GLP-1RAs, including those managed with standard of care, treated with other antidiabetic or anti-obesity agents, or serving as their own controls in pre-post study designs. We excluded case reports, non-human or preclinical studies and gray literature. Baseline use of biologics or small-molecule therapies did not constitute an exclusion. The protocol was registered in PROSPERO (CRD420251102686).

Outcomes

Primary outcomes were: (i) IBD-related hospitalization (unplanned admission attributed to IBD); (ii) IBD-related surgery (intestinal resection, colectomy, stoma or pouch procedures); (iii) systemic corticosteroid use (initiation or escalation of oral or intravenous steroids for IBD); (iv) escalation to advanced therapy (start, switch or major dose escalation of biologic or small-molecule agents); and (v) intestinal obstruction (clinically, radiologically or surgically

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confirmed). Events were captured after GLP-1RA initiation, and when several time points were reported we used the longest follow up.

Secondary outcomes included changes in C-reactive protein (CRP), fecal calprotectin, clinical disease activity indices, endoscopic scores and metabolic parameters (weight change, body mass index, glycated hemoglobin and lipid profile).

Risk of bias assessment

The risk of bias in observational studies was assessed using the ROBINS-I tool across 7 domains (D1–D7): bias due to confounding, selection of participants, classification of interventions, deviations from intended interventions, missing data, measurement of outcomes, and selection of the reported result [14]. For each domain and overall, studies were rated as having low, moderate or serious risk of bias. The randomized controlled trials (RCTs) were evaluated using the RoB 2 tool [15]. Two reviewers (CM, DP) independently performed all assessments, resolving any discrepancies through discussion and consensus.

Data analysis

Meta-analyses were performed in R (version 4.3.1) using the “meta” and “metafor” packages. We applied a random-effects model fitted by restricted/residual maximum likelihood to account for between-study variance. For dichotomous outcomes, we calculated odds ratios (ORs) with 95% confidence intervals (CI). Where possible, we pooled the most fully adjusted estimates. Efficacy was summarized as pooled ORs. Because included studies reported different effect measures, we pooled only studies with extractable event-count data suitable for odds-ratio meta-analysis. Studies reporting adjusted hazard ratios (aHRs) or adjusted incidence rate ratios without compatible event counts were not statistically combined with odds-ratio estimates and were summarized narratively. Between-study heterogeneity was assessed with Cochran's Q and the I^2 index, with $I^2 > 50\%$ considered substantial heterogeneity. Publication bias was assessed using funnel plots and Egger's test, when at least 10 studies were available for a given outcome.

Results

Study identification and characteristics

A total of 566 records were identified through database searching, and 13 studies (12 observational, 1 RCT) that met the inclusion criteria were included for the systematic review (Fig. 1). Only 3 studies provided quantitative data suitable for meta-analysis across prespecified outcomes; 1 study reported only aHRs, with event counts unavailable despite our

attempt to contact the authors [16]. Of the included studies, 9 were from the United States, with the remaining studies conducted in Denmark (n=2), Israel (n=1), and Spain (n=1). The data were derived from a range of sources, including single-center and multicenter clinical cohorts, as well as large nationwide registries. The overall population included 74,513 patients with IBD, most of whom had UC (64.9%) and CD (35.0%). Overall, 9202 patients (12.3%) were exposed to a GLP-1RA. Semaglutide was the most frequently used GLP-1RA (67%), with dulaglutide (13.3%) and liraglutide (7.0%) used less often. Tirzepatide (1.5%) and exenatide (0.2%) contributed minimally. A summary of the included study characteristics is presented in Table 1, and detailed summaries of the IBD and metabolic endpoints are summarized in Supplementary Tables 2 and 3, respectively.

Among the observational studies, most outcomes were at serious risk of bias from confounding, because treatment was not randomly assigned and baseline differences probably persisted. Missing data added further concern over several outcomes. Risks in other domains were generally low to moderate (Fig. 2). The single randomized trial by Herfarth *et al* was judged to have a high overall risk of bias, mainly due to substantial missing outcome data, concerns about outcome measurement, and selective reporting (Fig. 3) [17].

IBD-related outcomes

Corticosteroid initiation

In the evaluation of oral corticosteroid use, a total of 3195 IBD patients treated with GLP-1RAs and 4983 controls were included. GLP-1RA exposure was not associated with a statistically significant difference in oral steroid initiation (OR 0.70, 95%CI 0.29–1.68), although between-study heterogeneity was substantial ($I^2=98.7\%$) (Fig. 4). For intravenous corticosteroid use, the pooled analysis similarly showed no significant association (OR 1.16, 95%CI 0.96–1.40), with low heterogeneity ($I^2=11.9\%$) (Fig. 4). In addition, since Gorelik *et al* reported time-to-event estimates, their data could not be pooled with the odds-ratio meta-analysis [16]. In that study, GLP-1RA exposure was associated with a lower hazard of corticosteroid initiation in the overall IBD population (aHR 0.66, 95%CI 0.48–0.99), while the corresponding subgroup estimates were not statistically significant for CD (aHR 0.66, 95%CI 0.41–1.08) or UC (aHR 0.75, 95%CI 0.42–1.35).

Escalation to advanced therapy

Regarding the initiation of advanced therapy, a total of 2720 IBD patients treated with GLP-1RAs and 4533 controls were included in the analysis. GLP-1RA exposure was not associated with a statistically significant difference in the need for advanced therapy (pooled OR 0.79, 95%CI 0.43–1.44), with substantial between-study heterogeneity ($I^2=85.7\%$) (Fig. 4). Gorelik *et al* found no significant association between GLP-1RA exposure and initiation of advanced therapy in the overall

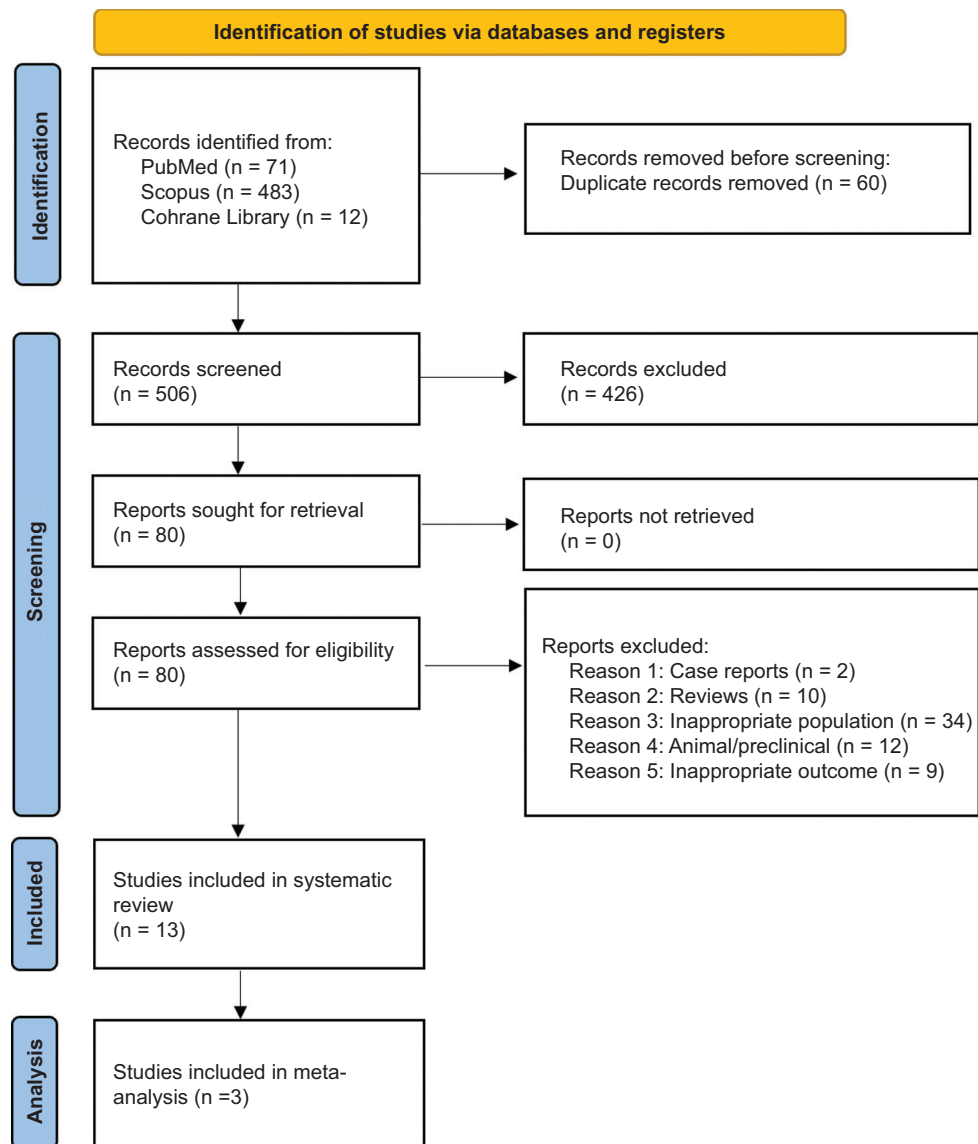


Figure 1 PRISMA flowchart

IBD cohort (aHR 0.82, 95%CI 0.59-1.11), with non-significant findings for both CD (aHR 0.92, 95%CI 0.61-1.39) and UC (aHR 0.70, 95%CI 0.37-1.33) [16].

IBD-related surgery

For surgical interventions, a total of 3196 IBD patients treated with GLP-1RAs and 4,998 controls were included. Notably, GLP-1RA exposure was associated with significantly lower odds of surgery in IBD patients (OR 0.46, 95%CI 0.34-0.61), with moderate between-study heterogeneity ($I^2=42.8\%$) (Fig. 4). In contrast, Gorelik *et al* reported no clear evidence of a protective association, with GLP-1RA exposure not significantly associated with a reduced risk of surgery in the overall IBD cohort (aHR 0.84, 95%CI 0.47-1.50) or in CD

(aHR 0.94, 95%CI 0.47-1.91) and UC (aHR 0.74, 95%CI 0.27-2.07) [16].

Hospitalization

For hospitalization rates, a total of 3200 IBD patients treated with GLP-1RAs and 4,985 controls were included. GLP-1RA exposure was not associated with a statistically significant difference in hospitalization risk in the pooled analysis (OR 0.57, 95%CI 0.23-1.39), with substantial heterogeneity ($I^2=98.3\%$) (Fig. 4). In the study by Gorelik *et al*, GLP-1RA exposure was associated with a lower hazard of hospitalization in the overall IBD population (aHR 0.74, 95%CI 0.61-0.91), with a statistically significant reduction in UC (aHR 0.63, 95%CI 0.45-0.90) but not in CD (aHR 0.82, 95%CI 0.65-1.05) [16].

Table 1 Study and population characteristics

Author (Year) [ref.]	Study origin and years of registration/follow up	Study type	Patients	Intervention (GLP-1 drugs)	Comparator	Outcome	Adverse events	Discontinuation reasons
Anderson <i>et al</i> , 2024 [21]	St. Louis, U.S., Missouri, 2014-2024 (10 years)	Retrospective cohort study	Total-120 UC- 59 (49.2%) CD- 61 (50.8%) T2DM-72 (60.0%) Weight loss-43 (35.8%) MASH-5 (4.2%)	Dulaglutide- 26 (21.7%) Exenatide- 8 (6.7%) Liraglutide- 12 (10%) Semaglutide- 74 (61.7%) Tirzepatide- 0 (0%)	Before and after treatment with GLP-1 RAs within the same patients.	Adverse events Weight change (%) Clinical severity scores IBD-related hospitalizations CRP change	Abdominal pain (1.1%), bloating (1.1%), constipation (2.9%), diarrhea (2.2%), gastroparesis (0.57%), nausea (2.2%), IBD flare (0.57%), pancreatitis (0.57%)	Gastrointestinal side-effects (11.5%) Cost (19.5%) Switching to another GLP-1RA (18.4%) Hypoglycemia (1.1%) Injection site reaction (1.1%) Death (1.1%)
Belinchón <i>et al</i> , 2024 [25]	Hospital General Universitario, Madrid, Spain, 2019-2021	Retrospective case series	Total - 16 UC-7 (43.8%) CD-9 (56.3%) All patients had obesity	Semaglutide (1.0 mg) - 5 (31%) Liraglutide (3.0 mg) -11 (69%)	Before and after treatment with GLP-1 RAs within the same patients.	Weight change (%) Change in IBD activity, fecal calprotectin and CRP IBD therapy escalation	Nausea (12.5%) Diarrhea (12.5%)	Lack of effect-2 patients Diarrhea-1 patient
Clarke <i>et al</i> , 2025 [26]	Brigham and Women's Hospital, Harvard Medical School, Boston, U.S., January 1, 2020 - December 31, 2023 (4 years)	Retrospective cohort study	Total-272 UC-130 (47.8%) CD-137 (50.4%) Indeterminate colitis- 5 (1.8%) All had obesity; 42% had T2DM	Semaglutide (44.4%, n=120) Dulaglutide (26.3%, n=71) Liraglutide (18.9%, n=51) Tirzepatide (9.3%, n=25) Exenatide (1.1%, n=5)	Before and after treatment with GLP-1 RAs within the same patients.	≥5%TWL at 12 months IBD flares (composite outcome: IBD-related ED visits or hospitalizations, surgeries, escalation of IBD therapy, or steroid use)	40% experienced adverse events; 93% of these were GI, nausea/vomiting (22%) constipation (11%) diarrhea (6%) reflux, indigestion, abdominal pain,	24% discontinued GLP-1 RA- 48% due to adverse events 18% for access/cost 3% for poor efficacy 30% for unknown reasons
Desai <i>et al</i> , 2024 (1) [18]	60 healthcare organizations across the U.S., January 1, 2010 - December 31, 2022 (13 years)	Retrospective cohort study	Total- 4136 UC-2020 CD-2016 UC GLP-1RA cohort-1010 CD GLP-1RA cohort-1058, All patients had T2DM	Dulaglutide (UC-546 [48.3%], CD-520 [45.6%]) Liraglutide (UC-212 [18.7%], CD-212 [18.5%]) Semaglutide (UC- 555 [49.1%], CD-512 [44.9%])	Propensity score-matched patients with IBD and T2DM	Hospitalization IBD-related surgery Oral steroid use Intravenous steroid use IBD therapy escalation Ileus CRP, fecal calprotectin change	No significant difference in risk of adverse events	Not reported

(Contd...)

Table 1 (Continued)

Author (Year) [ref.]	Study Origin and Years of Registration/ Follow up	Study Type	Patients	Intervention (GLP-1 drugs)	Comparator	Outcome	Adverse Events	Discontinuation Reasons
Desai et al, 2024 (2) [28]	Multi-institutional, U.S. June 4, 2021 – December 11, 2023 (3 years)	Retrospective cohort study	Total - 300 IBD-150 UC-51 (34%) CD- 99 (66%), All patients with obesity (BMI≥30), (T2DM excluded)	Semaglutide (n=150) Liraglutide (n=75) Tirzepatide (n=95)	Propensity score-matched non-IBD patients with obesity, prescribed semaglutide, Other anti-obesity medications in IBD	TBW change Comparison to other AOMs IBD-specific outcomes: oral/IV steroid use, initiation of advanced therapy, IBD-related surgery, any-cause hospitalization, ED visits	No increased risk of adverse events	Not reported
Gorelik et al, 2024 [16]	Nationwide cohort (epi-IIRN)- Israel, 2005–2021	Retrospective cohort study	Total-3,737, UC-1883 (50.4%) CD-1854 (49.6%) All patients had T2DM	GLP-1 RAs-633 patients	IBD patients with T2DM not exposed to GLP-1 analogs- 3104	Composite outcome, hospitalization, surgery, steroid dependency, treatment escalation, death	No increase in death or other adverse outcomes	Not reported
Herfarth et al, 2024 [17]	University of North Carolina, U.S., March 2022 - July 2023 (16 months)	Randomized, double-blind, placebo-controlled, crossover study	Total-8 UC-7, CD-1 Patients with IBD and colectomy with ileal pouch-anal anastomosis	Liraglutide titrated to 1.8 mg daily	Placebo	30% reduction (BF) Fecal calprotectin	Nausea, diarrhea, headache fatigue, abdominal pain, acid reflux, hypoglycemia UTI, joint pain, hair loss	Not reported
Nielsen et al, 2024 [40]	Denmark, July 1, 2018 – June 30, 2024 (6 years)	Observational cohort study	Total - 61,927 (GLP-1RA- 4430; not exposed- 57,497) UC-41191 CD-20736 UC GLP-1RA - 2930 (66.1%) CD GLP-1RA- 1500 (33.9%), 25.3% of exposed had T2DM, 3.6% of unexposed had T2DM	Semaglutide- 4430	IBD patients not exposed to GLP-1RAs	Ileus/intestinal obstruction	Not specially reported	Not reported

(Contd...)

Table 1 (Continued)

Author (Year) [ref.]	Study Origin and Years of Registration/ Follow up	Study Type	Patients	Intervention (GLP-1 drugs)	Comparator	Outcome	Adverse Events	Discontinuation Reasons
Pham <i>et al</i> , 2024 [27]	Mayo Clinic, US, January 2001–January 2022 (21 years)	Case-control study	Total - 72, IBD-36 UC-15 (41.7%) CD-21 (58.3%) All had obesity	Liraglutide (various, up to 3.0 mg)- 9 Semaglutide (various, up to 2.4 mg or 14 mg PO)- 10	Matched non-IBD controls	TBWL(%) IBD flares	Gastrointestinal symptoms (nausea, vomiting, diarrhea, constipation) - 6 IBD cases (31.6%) - 5 controls (26.3%) on GLP-1RA	Side-effects - 4/36 IBD (11%) - 1/36 controls (2.8%)
St-Pierre <i>et al</i> , 2024 [24]	Chicago, Illinois, US, January 2021–April 2024	Observational cross-sectional cohort study	Total - 36 UC-12 CD-24 IBD-U- 1 All had obesity	Semaglutide- 32 Tirzepatide- 4 7/32 on semaglutide were switched to tirzepatide	Comparison is between before and after treatment with GLP-1 agonists within the same patients.	TBW Changes in metabolic risk factors (lipids, A1c, ALT, CRP), IBD activity (CRP, Calprotectin)	Nausea (11, 31%), constipation (9, 25%), vomiting (3, 8%), abdominal cramping (1)	8/36 (22%) 4 due to lack of insurance coverage 3 due to side-effects for unknown reasons
Villumsen <i>et al</i> , 2021 [41]	Denmark, January 1, 2007 – March 31, 2019 (12 years)	Observational cohort study	Total - 3751 UC-2791 (74.4%) CD-960 (25.6%) UC GLP-IRAs- 761 (77.5%) CD GLP-IRAs- 221 (22.5%) non-users: UC- 2030 (73.3%) CD-739 (26.7%) All had T2DM	GLP-1 RAs and DPP-4i- 982	Other antidiabetic drugs	Composite of the need for oral corticosteroids, TNF- α inhibitor treatment, IBD-related hospitalization, or IBD-related major surgery	Not reported	Not reported
Sehgal <i>et al</i> , 2025 [22]	University of Pennsylvania, US, 2017 – 2023 (6 years)	Retrospective cohort study	Total-244 UC-102 (42%) CD-142 (58%) All had obesity, 155 (64%) had T2DM	Semaglutide- 131 (54%) Dulaglutide- 47 (19%) Liraglutide- 38 (16%) Exenatide- 3 (1%) Tirzepatide- 5 (2%)	Comparison is between before and after treatment	TBW Change in CRP and fecal calprotectin	Nausea/vomiting (8%) Diarrhea (2%) Constipation (2%) Rash (1%) Headache (0.5%) Pancreatitis (0.5%)	Adverse events: 26 (11%) IBD flare: 1 (0.5%) No weight loss: 5 (2%) Polypharmacy: 2 (1%) Unknown: 7 (3%)

A1c, glycated hemoglobin; ALT, alanine aminotransferase; AOMs, anti-obesity medications; BF, bowel frequency; BMI, body mass index; CD, Crohn's disease; CRP, C-reactive protein; DPP-4i, dipeptidyl peptidase-4 inhibitor; ED, emergency department; epi-IBD, Epidemiology Group of the Israeli IBD Research Nucleus; GI, gastrointestinal; GLP-1, glucagon-like peptide-1; GLP-1RA, glucagon-like peptide-1 receptor agonist; IBD, inflammatory bowel disease; IBD-U, inflammatory bowel disease unclassified; IV, intravenous; MASH, metabolic dysfunction-associated steatohepatitis; PO, per os; T2DM, type 2 diabetes mellitus; TBWL, total body weight; TBWL, total body weight loss; TNF- α , tumor necrosis factor- α ; UC, ulcerative colitis; UTI, urinary tract infection

	Risk of bias domains							Overall
	D1	D2	D3	D4	D5	D6	D7	
Anderson <i>et al</i> , 2024	X	+	+	+	X	+	+	X
Belinchón <i>et al</i> , 2024	X	+	+	+	X	+	+	X
Clarke <i>et al</i> , 2025	+	+	+	+	X	+	+	X
Desai <i>et al</i> , 2024(1)	+	+	+	+	+	+	+	+
Dosai <i>et al</i> , 2024(2)	+	+	+	+	+	+	+	+
Gorelik <i>et al</i> , 2024	+	+	+	+	+	+	+	+
Levine <i>et al</i> , 2024	X	+	+	+	X	+	+	X
Nielsen <i>et al</i> , 2024	+	+	+	+	+	+	+	+
Pham <i>et al</i> , 2024	X	+	+	+	-	+	+	X
St-Pierre <i>et al</i> , 2024	X	+	+	+	-	+	+	X
Villumsen <i>et al</i> , 2021	+	+	+	+	+	+	+	+
Sehgal <i>et al</i> , 2025	-	+	+	+	-	+	+	-

Study

Domains:
D1: Bias due to confounding.
D2: Bias due to selection of participants.
D3: Bias in classification of interventions.
D4: Bias due to deviations from intended interventions.
D5: Bias due to missing data.
D6: Bias in measurement of outcomes.
D7: Bias in selection of the reported result.

Judgement
X Serious
- Moderate
+ Low

Figure 2 Risk of bias assessment using ROBINS-I tool

	Risk of bias domains					Overall
	D1	D2	D3	D4	D5	
Study Herfarth <i>et al</i> , 2024	+	+	X	+	X	X

Domains:
D1: Bias arising from the randomization process.
D2: Bias due to deviations from intended intervention.
D3: Bias due to missing outcome data.
D4: Bias in measurement of the outcome.
D5: Bias in selection of the reported result.

Judgement
X High
+ Low

Figure 3 Risk of bias assessment using ROB-2 tool

Intestinal obstruction

When evaluating the risk of intestinal obstruction, only 2 studies contributed data, which included 6230 IBD patients treated with GLP-1RAs and 59,267 controls [18]. GLP-1RA exposure was not associated with a statistically significant difference in intestinal obstruction risk (OR 0.43, 95%CI 0.15-1.25), with substantial heterogeneity ($I^2=93.9\%$) (Fig. 4).

C-reactive protein

CRP was evaluated as a systemic marker of inflammation, to explore whether GLP-1RA exposure was accompanied by measurable changes in inflammatory burden [19,20]. Given the heterogeneity in reporting and follow up across studies, CRP outcomes were synthesized narratively. Anderson *et al* reported a decrease from 12.9 to 6.4 mg/dL ($P=0.005$) and Sehgal *et al* a reduction from 10.1 to 3.0 mg/dL ($P<0.01$) [21,22]. No significant reduction in CRP levels was observed in the studies by Levine *et al* ($P=0.45$), St-Pierre *et al* ($P=0.21$) and Belinchón *et al* ($P=0.66$) [23-25].

Fecal calprotectin

Fecal calprotectin outcomes were also reported in a limited number of studies and showed mixed findings across cohorts.

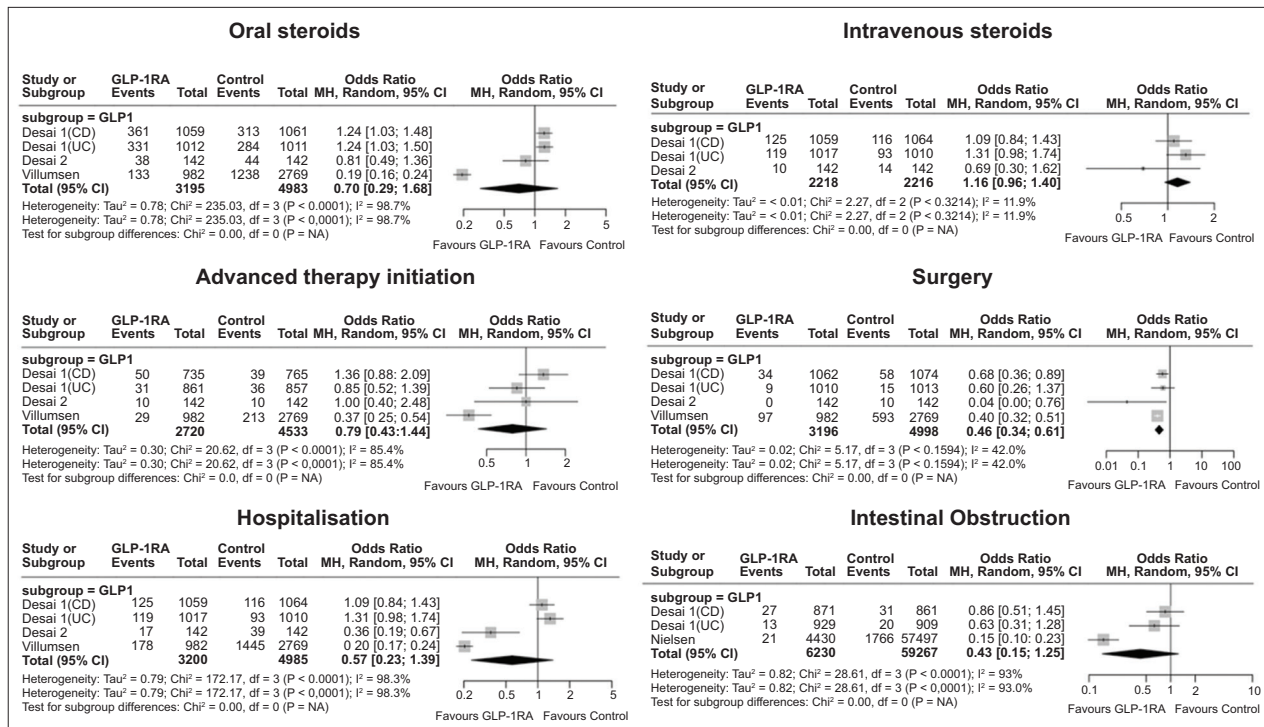


Figure 4 Summary of forest plots
CI, confidence interval; GLP-1RA, glucagon-like peptide-1 receptor agonist; OR, odds ratio

Sehgal *et al* reported a decrease from 824.9 to 234.7 mg/g at 24 weeks ($P=0.13$) [22]. Belinchón *et al* observed a reduction from 40.9 to 32.7 $\mu\text{g/g}$ ($P=0.376$) [25]. Desai *et al* reported lower mean values in GLP-1RA-treated cohorts than controls (CD: 260 vs. 324 $\mu\text{g/g}$, $P=0.29$; UC: 324.7 vs. 369.7 $\mu\text{g/g}$, $P=0.67$) [18]. Herfarth *et al* found no significant differences [17].

IBD exacerbations

An important clinical question is whether GLP-1RA exposure influences day-to-day disease control in IBD, including the ability to maintain clinical remission without precipitating flares, despite the background use of biologics and other therapies. Across the included studies, flare rates were generally stable, with several cohorts reporting reductions after GLP-1RA initiation. Clarke *et al* reported a reduction from 16.6% pre-treatment to 13.1% post-treatment ($P=0.40$) [26]. Levine *et al* observed a decrease from 34.4% to 30.4% ($P=0.36$) [23]. Anderson *et al* and Sehgal *et al* reported exacerbation rates of 0.57% and 0.5%, respectively [21]. Belinchón *et al* observed 2 cases with IBD flare among 15 patients, while Pham *et al* found rates of 19.4% overall (CD: 28.6%, UC: 6.7%) [25, 27].

Clinical severity scores

Clinical activity indices were reported to assess whether GLP-1RA initiation was accompanied by meaningful changes

in symptom-based disease activity. Anderson *et al* reported a decrease in Harvey-Bradshaw Index (HBI) for CD from 3.52 to 3.18 ($P=0.61$) and in Modified Mayo Score for UC from 1.61 to 1.54 ($P=0.67$) [21]. Belinchón *et al* observed no significant change in HBI (3 to 4, $P=0.388$) or Partial Mayo Score (1.29 to 1.00, $P=0.089$) after treatment [25].

Endoscopic scores

Endoscopic indices were reported in only 1 study. Anderson *et al* reported endoscopic scores, observing non-significant reductions in Simplified Endoscopic Score (3.10 to 2.60, $P=0.35$) and Mayo Endoscopy Score (0.67 to 0.00, $P=0.16$) following GLP-1RAs initiation [21].

Metabolic outcomes

Weight change

GLP-1RAs consistently led to clinically meaningful weight loss in IBD patients. Anderson *et al* reported a mean weight loss (WL) of 3.9%, with semaglutide showing the greatest effect (5.3%) [21]. Clarke *et al* found a mean total weight loss (TWL) of 8.72% and a mean body mass index (BMI) reduction of 3.26 kg/m^2 [26]. Semaglutide achieved the highest $\geq 5\%$ TWL rate at 76.0% ($P=0.02$). Belinchón *et al* noted a significant median WL from 92.17 kg to 81.95 kg at 6 months ($P=0.002$) [25]. Desai *et al* observed similar 12-month BMI in semaglutide and control

groups (34.0 vs. 34.1 kg/m², $P=0.87$) [18]. In a separate study, Desai *et al* observed a 16-pound WL in IBD vs. 15 pounds in non-IBD ($P=0.24$); semaglutide outperformed liraglutide ($P=0.04$) and other anti-obesity agents, but was inferior to tirzepatide ($P=0.01$) [28]. Sehgal *et al* reported a mean WL of 5.2% ($P<0.01$), with semaglutide again being the most effective (-6.2%) [22]. St-Pierre *et al* showed a significant median BMI drop from 34.0 to 31.0 ($P<0.001$) and a TWL of 8.15 kg ($P<0.001$) [24]. Pham *et al* found a mean 6-month TWL of 5.4% in IBD versus 8.6% in controls ($P=0.03$) [27]. Levine *et al* reported a median BMI decrease of 1.9 kg/m² in IBD patients ($P<0.01$) [23].

Glycemic control (HbA1c%)

GLP-1RAs modestly improved HbA1c in IBD patients. Belinchón *et al* noted a reduction from 6.2% to 5.9% ($P=0.14$) [25]. Desai *et al* reported post-treatment HbA1c of 6.8% (semaglutide) vs. 7.0% (controls, $P=0.12$) [18]. Levine *et al* and St-Pierre *et al* found mean reductions of 0.3% ($P=0.01$) and 0.2% ($P=0.054$), respectively [23].

Modification in the lipid profile

Only 3 studies reported lipid outcomes in IBD patients treated with GLP-1RAs. Belinchón *et al* observed modest, non-significant changes across total cholesterol (TC) changes ($P=0.10$), low-density lipoprotein cholesterol (LDL-C) ($P=0.58$), high-density lipoprotein cholesterol (HDL-C) ($P=0.20$), and triglycerides ($P=0.58$) [25]. In contrast, Levine *et al* found statistically significant increases in TC, LDL-C and HDL-C during follow up ($P=0.03$, $P<0.01$ and $P<0.01$, respectively), while no significant lipid changes were seen in the non-IBD control group ($P=0.85$, $P=0.20$ and $P=0.69$, respectively) [23]. St-Pierre *et al* reported a significant reduction in HDL-C ($P=0.0427$), whereas changes in TC, LDL-C and triglycerides did not significantly change ($P=0.0634$, $P=0.4618$ and $P=0.1859$, respectively) [24].

Safety and discontinuation reasons

Across the included studies, GLP-1RAs were generally well tolerated in patients with IBD, and adverse events were predominantly gastrointestinal and consistent with the established class profile. The most frequently reported events were nausea or vomiting (19.67%), followed by diarrhea (8.6%), constipation (6.5%) and abdominal pain (2.3%). Serious adverse events were rare, with pancreatitis reported in 0.57% of patients, and hypoglycemia accounted for 1.1% of discontinuations. Treatment discontinuation was most often attributed to adverse events (18.7%) and cost (11.5%), whereas discontinuation due to lack of efficacy was infrequent (2.9%), indicating that tolerability and access issues were more common reasons for stopping therapy than perceived ineffectiveness.

Discussion

This systematic review and meta-analysis provides preliminary evidence that GLP-1RAs may have a role as an add-on treatment in patients with IBD, especially in those with concomitant obesity. Among the most notable findings, GLP-1RA exposure was associated with lower odds of IBD-related surgical interventions in pooled observational data. The analysis did not find significant reductions in corticosteroid dependency, hospitalizations, escalation to advanced therapies, or intestinal obstruction. Still, the general trend across these outcomes leaned towards benefit, suggesting that these areas should be further studied. Measures of IBD activity were mixed and largely non-significant across CRP and fecal calprotectin. Symptom indices showed no meaningful change in the limited reporting studies, and endoscopic outcomes were rarely assessed and non-significant when reported.

The observed association with surgery risk may reflect fewer severe downstream events in some settings. The study by Gorelik *et al* did not show a clear reduction in surgery risk (aHR 0.84, 95%CI 0.47-1.50) [16]. The discordance between event-count and time-to-event estimates may reflect differences in follow-up duration, time at risk and censoring, baseline disease severity, outcome definitions, and analytical adjustment strategies. Residual confounding may also contribute. These issues, together with heterogeneity, probably contributed to the directionally favorable, yet non-robust findings for other endpoints.

Several factors may explain the inconsistent signals of disease activity, despite the biological plausibility of an effect. GLP-1RAs were usually prescribed for metabolic indications rather than for active IBD: many patients probably had stable disease, and were on maintenance therapy, leaving limited room for additional improvement. Biomarker sampling was sparse and variable. CRP is non-specific and calprotectin fluctuates, while symptoms may diverge from mucosal inflammation. The small RCT was not designed to detect slow-moving outcomes such as mucosal healing. Overall, the current evidence supports a metabolic benefit and acceptable tolerability, while consistent improvement in objective IBD inflammation remains unproven.

Across the included studies, GLP-1RAs were consistently associated with clinically meaningful weight loss. This finding aligns with evidence from populations with obesity and appears consistent with the direction of effect observed in IBD cohorts in the present synthesis [29]. Given the growing overlap between metabolic disease and IBD, this effect may be clinically useful. The association between obesity and IBD outcomes remains uncertain, some studies suggest higher relapse rates and a poorer response to biologics, whereas others show no clear effect [8]. GLP-1RAs may therefore offer a practical option for weight management in patients with IBD, but any impact on IBD activity or treatment response should be interpreted cautiously and confirmed in prospective studies.

The safety profile was broadly consistent with the known class profile of GLP-1RAs. Adverse events were predominantly gastrointestinal, and discontinuation occurred in a meaningful

minority in some cohorts, while large datasets did not show a clear excess of serious outcomes, albeit with inconsistent reporting. This pattern aligns with prior meta-analyses in obesity and diabetes populations, where gastrointestinal symptoms are the dominant adverse events and represent a common reason for stopping therapy [30,31]. However, broader safety reviews suggest that less common gastrointestinal and hepatobiliary side-effects still require careful monitoring. This is especially important, because observational IBD studies may not reliably detect rare adverse events [32].

Consistent with prior syntheses, our pooled analysis suggests that GLP-1RA exposure may be associated with a lower likelihood of IBD-related surgery, while other major clinical endpoints remain uncertain despite generally favorable directions. Bayoumy *et al* similarly observed an association with reduced surgery risk, using both time-to-event and event-frequency approaches, and noted that any apparent reduction in hospitalizations was most evident in patients with BMI ≥ 30 kg/m², with heterogeneity across other outcomes [33]. Yang *et al* focused on IBD with metabolic comorbidity, and also reported reduced surgery risk, whereas broader “complications” composites showed only non-significant trends with very high heterogeneity [34]. Maracle *et al* reached broadly similar conclusions, but did not pool effect estimates because of variation in study designs and effect measures. This highlights the limited methodological consistency in the field [35]. In parallel, Shirmard *et al* approached the question from a metabolic perspective and reported consistent reductions in weight and BMI in IBD cohorts, while effects on glycemic and lipid outcomes were limited or inconsistent, and overall certainty was rated low to very low [36]. This pattern is consistent with our findings.

Differences across reviews probably reflect variation in evidence selection and outcome definitions. Eligibility criteria range from metabolically enriched cohorts to broader IBD populations, which may influence baseline risk and event rates [34]. Outcomes are also defined inconsistently, particularly for composite endpoints and hospitalization thresholds. In addition, effect measures vary across studies, which limits direct comparability. Against this background, our review extends prior work by integrating a broader set of IBD-specific clinical endpoints together with metabolic outcomes in a single synthesis. We pooled only prespecified outcomes with available event counts. We reported time-to-event results that could not be pooled separately, which helped show where findings agreed and where they differed across analyses.

This review used transparent, reproducible methods consistent with the PRISMA guidelines, including a comprehensive multi-database search. The work covers a wide range of outcomes in a large real-world cohort (74,513 patients with IBD, including 9202 exposed to GLP-1RAs). It assessed key IBD outcomes (surgery, hospitalization, steroid use, treatment escalation and obstruction), as well as metabolic endpoints (weight, HbA1c and lipids) and inflammatory markers when available.

Nevertheless, the study’s limitations are substantial. Most evidence was observational (12/13 studies), with a risk of

confounding by indication, selection bias, and residual confounding. Exposure and outcomes were frequently derived from administrative codes, and dose, duration, adherence and exposure were inconsistently captured. Pooling was constrained, because only 3 studies contributed event-count data, while 1 key study reported aHRs only. The difference in effect measures and follow-up windows further limited comparability. Several pooled outcomes differed substantially across studies. This probably reflects real differences between cohorts, including UC vs. CD, metabolic comorbidity, the choice of comparator group, and background IBD treatment. Publication bias could not be robustly assessed because fewer than 10 studies contributed to the analyses. Moreover, objective activity, endoscopy, safety, and subgroup effects were sparsely reported. Therefore, our findings should be interpreted cautiously and viewed as hypothesis-generating.

Emerging evidence suggests that GLP-1RAs exert dual immunometabolic effects relevant to IBD pathogenesis. Beyond weight loss and glycemic control, they may dampen immune activation, reduce proinflammatory cytokines, and support epithelial integrity and permeability. GLP-1RA treatment may also favor microbiota profiles linked to mucosal health. These signals are not proof of disease control in IBD [37]. Future studies should prioritize adequately powered RCTs with standardized endpoints, including CRP, fecal calprotectin, endoscopic, histologic and patient-reported outcomes. Designs should stratify UC versus CD, metabolic strata, background biologic therapy, and phenotype, with harmonized flare and hospitalization definitions. Safety requires systematic capture of discontinuation, rare events, and obstruction or ileus. Notably, the ongoing COMMIT-UC (NCT06937086) and COMMIT-CD (NCT06937099) trials, Phase 3 RCTs assessing the combination of mirikizumab and tirzepatide in patients with IBD and obesity, represent an important step toward understanding how GLP-1RAs and biologics may work together to influence both inflammation and metabolism [38-39]. These trials are crucial to clarify the clinical role of GLP-1RAs in IBD treatment and to identify which patients are most likely to benefit.

In conclusion, this systematic review and meta-analysis synthesizes emerging evidence on GLP-1RAs in IBD by integrating clinical and metabolic outcomes. The most consistent finding is clinically meaningful weight loss in IBD patients receiving GLP-1RAs. Pooled observational data also suggest a possible reduction in IBD-related surgery, whereas other clinical outcomes remain uncertain and heterogeneous. Measures of objective inflammatory activity are mixed and do not consistently demonstrate improvement. At present, GLP-1RAs should be viewed primarily as metabolic therapy for IBD patients who meet established indications, and any disease-modifying benefit remains unconfirmed. Rigorous, adequately powered RCTs with standardized IBD endpoints and robust safety reporting are needed to clarify their role in clinical practice.

Acknowledgment

Declaration of AI use: Synthesia AI was used to assist title and abstract screening. All the decisions regarding eligibility of the included studies, data extraction, analyses, interpretation, and final manuscript approval were performed by the authors

Summary Box

What is already known:

- Obesity is increasingly common among patients with inflammatory bowel disease (IBD) and may influence disease course and treatment response
- Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are effective therapies for obesity and type 2 diabetes, with emerging evidence suggesting possible anti-inflammatory and gut-barrier effects

What the new findings are:

- In this systematic review of 13 studies, including 74,513 patients with IBD, GLP-1RA exposure was associated with consistent weight reduction across included cohorts
- In pooled observational data, GLP-1RA exposure was associated with lower odds of IBD-related surgery, while hospitalization, corticosteroid use, advanced therapy escalation and intestinal obstruction were not significantly affected

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Supplementary material

Supplementary Table 1 PRISMA 2020 Checklist

Section and topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review	Page-1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist	
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge	Page-2
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses	Page-2
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses	Page-3
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted	Page-3
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used	Supplementary Appendix
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process	Page 3
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process	Page 3
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect	Page 3
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information	Page 3
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process	Page 3
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results	Page 3
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5))	NA
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions	Page 3
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses	NA
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used	Page 3

(Contd...)

Supplementary Table 1 (Continued)

Section and Topic	Item #	Checklist item	Location where item is reported
METHODS			
Reporting bias assessment	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression)	NA
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results	NA
	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases)	NA
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome	NA
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram	Page 4 Page 13
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded	NA
Study characteristics	17	Cite each included study and present its characteristics	Page 14-15
Risk of bias in studies	18	Present assessments of risk of bias for each included study	Page 17
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots	Page 18
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies	Page 4
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect	Page 18
	20c	Present results of all investigations of possible causes of heterogeneity among study results	NA
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results	NA
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed	NA
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed	NA
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence	Page 8
	23b	Discuss any limitations of the evidence included in the review	Page 8-9
	23c	Discuss any limitations of the review processes used	Page 8-9
	23d	Discuss implications of the results for practice, policy, and future research	Page 9
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered	Page 3
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared	Page 3
	24c	Describe and explain any amendments to information provided at registration or in the protocol	NA
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review	Page 10
Competing interests	26	Declare any competing interests of review authors	Page 10
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review	Page 10

Supplementary Table 2 IBD outcomes

Author (Year)	Steroid dependency	Advanced therapy initiation	IBD-related surgery	IBD-related hospitalization	Ileus/intestinal obstruction	C-reactive protein (CRP)
Anderson <i>et al</i> , 2024 [21]	N.A.	N.A.	N.A.	Before GLP-1RA - 10/100 (10%) After GLP-1RA - 10/100 (10%) P=1.00	N.A.	Before GLP-1RA - Mean=12.92 mg/dL After GLP-1RA - Mean=6.38 mg/dL, P=0.005
Belinchón <i>et al</i> , 2024 [25]	N.A.	N.A.	N.A.	N.A.	N.A.	Before GLP-1RA - Mean=0.45 mg/dL (IQR: 0.4–0.78) After GLP-1RA - Mean=0 mg/dL (IQR: 0–1.5) P=0.66
Clarke <i>et al</i> , 2025 [26]	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Desai <i>et al</i> , 2024 (1) [18]	CD: Oral steroid use GLP-1RA- 361/1058 (34.1%) Control - 313/1058 (29.5%) aHR=1.10 [0.95–1.28] P=0.19 Intravenous steroid use GLP-1RA- 125/1058 (11.8%) Control - 116/1058 (10.9%) aHR=1.04 [0.80–1.34] P=0.75 UC: Oral steroid use GLP-1RA- 331/1010 (32.7%) Control - 284/1010 (28.1%) aHR=1.21 [0.96–1.31] P=0.14 Intravenous steroid use GLP-1RA- 119/1010 (11.7%) Control - 93/1010 (9.2%) aHR=1.04 [0.92–1.59] P=0.15	CD: GLP-1RA- 50/1058 (4.8%) Control- 39/1058 (5.1%) aHR=1.31, [0.86–2.00] P=0.19 UC: GLP-1RA- 31/1010 (3.6%) Control- 36/1010 (4.2%) aHR=0.82, [0.51–1.33] P=0.43	CD: GLP-1RA- 34/1058 (3.2%) Control- 58/1058 (5.4%) aHR=0.55, [0.36–0.84] P=0.005 UC: GLP-1RA- 10/1010 (1.0%) Control- 15/1010 (1.48%) aHR=0.70, [0.14–0.97] P=0.03	CD: GLP-1RA - 125/1058 (11.8%) Control - 116/1058 (10.9%) aHR=1.04 [0.80–1.34] P=0.75 UC: GLP-1RA - 119/1010 (11.7%) Control - 93/1010 (9.2%) aHR=1.04[0.92–1.59] P=0.15	CD: GLP-1RA- 27/871 (3.1%) Control- 31/861 (3.6%) aOR=0.85, [0.50–1.45] P=0.56 UC: GLP-1RA- 13/924 (1.4%) Control- 20/924 (2.2%) aOR=0.64, [0.31–1.29] P=0.21	N.A

(Contd...)

Supplementary Table 2 (Continued)

Author (Year)	Steroid dependency	Advanced therapy initiation	IBD-related surgery	IBD-related hospitalization	Ileus/intestinal obstruction	C-reactive protein (CRP)
Desai <i>et al</i> , 2024 (2) [28]	Oral steroid use GLP-1RA- 38/142 (26.7%) Control- 44/142 (30.9%) aOR=0.81, [0.48–1.36] P=0.43 Intravenous steroid use GLP-1RA- 10/142 (7.0%) Control- 14/142 (9.8%) aOR=0.69, [0.29–1.61] P=0.39	GLP-1RA- 10/142 (-10.0%) Control- 10/142 (-10.3%) aOR=1.03, [0.41–2.60] P=0.94	GLP-1RA- 0/142 (0%) Control- 10/169 (5.9%) aOR=Not estimable	GLP-1RA- 17/142 (11.9%) Control- 39/142 (27.4%) aOR=0.35, [0.19–0.67] P=0.001	N.A.	N.A.
Gorelik <i>et al</i> , 2024 [16]	All IBD: aHR=0.66 [0.48–0.99], CD: aHR=0.66 [0.41–1.08], UC: aHR=0.75 [0.42–1.35]	All IBD: aHR=0.82 [0.59–1.11] CD: aHR=0.92 [0.61–1.39], UC: aHR=0.70 [0.37–1.33]	All IBD: aHR=0.84 [0.47–1.50] CD: aHR=0.94 [0.47–1.91], UC: aHR=0.74 [0.27–2.07]	All IBD: aHR=0.74 [0.61–0.91] CD: aHR=0.82 [0.65–1.05], UC: aHR=0.63 [0.45–0.90]	N.A.	N.A.
Herfarth <i>et al</i> , 2024 [17]	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Levine <i>et al</i> , 2024 [23]	Pre-GLP-1RA- 50/224 (22.3%) Post-GLP-1RA- 41/224 (18.3%) P=0.27	Pre-GLP-1RA- 36/224 (16.1%) Post-GLP-1RA- 28/224 (12.5%) P=0.34	Pre-GLP-1RA- 3/224 (1.3%) Post-GLP-1RA- 4/224 (1.8%) P=1.00	Pre-GLP-1RA- 20/224 (8.9%) Post-GLP-1RA- 17/224 (7.6%) P=0.70	N.A.	Pre-GLP-1RA- Mean 6.9 mg/L (IQR 3.1–11.4) Post-GLP-1RA- Mean 5.5 mg/L (IQR 2.8–11.6) P=0.45
Nielsen <i>et al</i> , 2024 [40]	N.A.	N.A.	N.A.	N.A.	aHR=0.57, [0.36–0.88] Subgroup: Diabetes: aHR=0.59 [0.32–1.10] No diabetes: aHR=0.48 [0.25–0.93] CD: aHR=0.74 [0.42–1.30] UC: aHR=0.42 [0.21–0.86]	N.A.
Pham <i>et al</i> , 2024 [27]	Total IBD - 4/36 [11.1%], CD- 3/21 [14.3%], UC- 1/15 [6.7%]	Total IBD - 5/36 [13.9%], CD- 4/21 [19.0%], UC- 1/15 [6.7%]	Total IBD - 0/36 (0%)	Total IBD- 2/36 (5.6%), CD- 2/21 [9.5%], UC- 0/15 [0%]	N.A.	N.A.

(Contd...)

Supplementary Table 2 (Continued)

Author (Year)	Steroid dependency	Advanced therapy initiation	IBD-related surgery	IBD-related hospitalization	Ileus/intestinal obstruction	C-reactive protein (CRP)
St-Pierre <i>et al</i> , 2024 [24]	0/36 [0%]	N.A.	N.A.	N.A.	N.A.	Pre-GLP-1RA-Mean=3 mg/L (IQR 3–6.5 mg/L), Post-GLP-1RA-Mean 3 mg/L (IQR 3–6.5 mg/L), P=0.2081
Villumsen <i>et al</i> , 2021 [41]	GLP-1RA/ DPP-4i- 47.3 (133 events/2813 PY) Non-users- 94.5 (1238 events/13,104 PY) aIRR: 0.54 [0.41–0.70]	GLP-1RA/ DPP-4i- 6.9 (29 events/4183 PY) Non-users- 11.4 (213 events/18,737 PY) aIRR: 0.56 [0.32–1.00]	GLP-1RA/DPP-4i- 26.4 (97 events/3675 PY) Non-users- 34.0 (593 events/17,456 PY) aIRR: 0.79 [0.57–1.09]	GLP-1RA/ DPP-4i - 61.6 (178 events/2889 PY) Non-users- 103.0 (1445 events/14,024 PY) aIRR: 0.73 [0.58–0.91]	N.A.	N.A.
Sehgal <i>et al</i> , 2025 [22]	N.A.	N.A.	N.A.	N.A.	N.A.	Pre-GLP-1RA-mean (SD)= 10.1 mg/dL (16.6) Post GLP-1RA-mean (SD)= 3.0 mg/dL (6.3) P<0.01, n=71

Author (Year)	Fecal calprotectin	IBD exacerbation	Clinical severity score	Endoscopic score	Time point
Anderson <i>et al</i> , 2024 [21]	N.A.	1/174 therapeutic trials (0.57%)	CD (HBI): Before- Mean=3.52 After- Mean=3.18 P=0.61 UC (MMS): Before- Mean=1.61 After- Mean=1.54 P=0.67	SES-CD: Before - Mean=3.10 After - Mean=2.60 P=0.35 MES: Before - Mean=0.67 After - Mean=0.00 P=0.16	1 year before and 1 year after GLP-1RA initiation
Belinchón <i>et al</i> , 2024 [25]	Before GLP-1RA - Mean=40.9 µg/g (IQR: 13.77–79.88) After GLP-1RA - Mean=32.7 µg/g (IQR: 13.8–50.98) P=0.376	2/15 patients	CD (HBI): Before- Mean=3 (IQR: 1–5) After- Mean=4 (IQR: 2.75–4.25) P=0.388 UC (PMS): Before- Mean=1.29 (IQR: 0–1) After- Mean=1.00 (IQR: 1–2.23) P=0.089	N.A.	6 months

(Contd...)

Supplementary Table 2 (Continued)

Author (Year)	Steroid dependency	Advanced therapy initiation	IBD-related surgery	IBD-related hospitalization	Ileus/intestinal obstruction	C-reactive protein (CRP)
Clarke <i>et al</i> , 2025 [26]	N.A.	Before GLP-1RA - 29/175 (16.6%) After GLP-1RA - 23/175 (13.1%)	N.A.	N.A.	1 year before and 1 year after GLP-1RA initiation	
Desai <i>et al</i> , 2024 (1) [18]	CD: GLP-1RA- Mean=260 µg/g Control- Mean=324 µg/g P=0.29 UC: GLP-1RA- Mean=324.7 µg/g Control- Mean=369.7 µg/g P=0.67	N.A.	N.A.	N.A.	3 years	
Desai <i>et al</i> , 2024 (2) [28]	N.A.	N.A.	N.A.	N.A.	1 year	
Gorelik <i>et al</i> , 2024 [16]	N.A.	N.A.	N.A.	N.A.	Median follow up 6 years	
Herfarth <i>et al</i> , 2024 [17]	No difference in Calprotectin during placebo or liraglutide therapy	N.A.	N.A.	N.A.	6 weeks	
Levine <i>et al</i> , 2024 [23]	N.A.	Pre-GLP-1RA- 77/224 (34.4%) Post-GLP-1RA- 68/224 (30.4%) P=0.36	N.A.	N.A.	1 year before and 1 year after GLP-1RA initiation	
Nielsen <i>et al</i> , 2024 [40]	N.A.	N.A.	N.A.	N.A.	Median follow up 6 years	
Pham <i>et al</i> , 2024 [27]	N.A.	Total IBD- 7/36 (19.4%) CD- 6/21 [28.6%], UC- 1/15 [6.7%]	N.A.	N.A.	1 year	
St-Pierre <i>et al</i> , 2024 [24]	Pre-GLP-1RA <250 mcg/g, n=8 Post-GLP-1RA <250 mcg/g, n=3	N.A.	N.A.	N.A.	Median follow up 450 days	
Villumsen <i>et al</i> , 2021 [41]	N.A.	N.A.	N.A.	N.A.	N.A.	
Sehgal <i>et al</i> , 2025 [22]	Pre-GLP-1RA- mean (SD)= 824.9 mg/g (2112.7), Post GLP-1RA- mean (SD)= 234.7 mg/g (530.8), P=0.13, n=32	1/244 (0.5%)	N.A.	N.A.	24 weeks	

aHR, adjusted hazard ratio; aIRR, adjusted incidence rate ratio; aOR, adjusted odds ratio; CD, Crohn's disease; CRP, C-reactive protein; DPP-4i, dipeptidyl peptidase-4 inhibitor; GLP-1RA, glucagon-like peptide-1 receptor agonist; HBI, Harvey-Bradshaw Index; IBD, inflammatory bowel disease; IQR, interquartile range; MES, Mayo endoscopic subscore; MMS, modified Mayo score; N.A., not available; PMS, partial Mayo score; PY, person-years; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; UC, ulcerative colitis.

Supplementary Table 3 Metabolic outcomes

Author (Year)	Weight change (%)	Glycemic control (HbA1c%)	Change in Lipids
Anderson <i>et al</i> , 2024 [21]	All GLP-1RAs: -3.9% (SD 7.7) Dulaglutide: -3.5% (SD 7.4) Exenatide: -0.59% (SD 4.4) Liraglutide: -2.9% (SD 4.1) Semaglutide: -5.3% (SD 8.0)	N.A	N.A
Belinchón <i>et al</i> , 2024 [25]	Baseline: 92.17 kg (IQR: 83.42–94.60) 6 months: 81.95 kg (IQR: 76.62–87.67) P=0.002	Baseline: 6.2% (IQR: 5.5–7.8) 6 months: 5.9% (IQR: 5.3–7.58) P=0.14	Total cholesterol: Baseline: 193 mg/dL (IQR: 158–225) 6 months: 148 mg/dL (IQR: 137–195) P=0.10 LDL cholesterol : Baseline: 99 mg/dL (IQR: 84–141) 6 months: 78 mg/dL (IQR: 58–126) P=0.58 HDL cholesterol : Baseline: 54 mg/dL (IQR: 50.5–62.5) 6 months: 42 mg/dL (IQR: 41–52) P=0.20 Triglycerides: Baseline: 139 mg/dL (IQR: 97–197) 6 months: 127 mg/dL (IQR: 103–200) P=0.58
Clarke <i>et al</i> , 2025 [26]	Mean (TWL%) : 8.72% (SD 8.98) Mean change in BMI : -3.26 kg/m ² (SD 3.60) Weight loss ≥5% by agent: Semaglutide: 57/75 (76.0%) Liraglutide: 20/34 (58.82%) Tirzepatide: 7/13 (53.85%) Dulaglutide: 22/52 (42.31%) P=0.02	N.A	N.A
Desai <i>et al</i> , 2024 (1) [18]	Semaglutide: mean BMI=34.0 kg/m ² Control: mean BMI=34.1 kg/m ² , P=0.87	Semaglutide: mean=6.8% Control: mean=7.0%, P=0.12	N.A
Desai <i>et al</i> , 2024 (2) [28]	Mean TBW change: IBD: -16±13 pounds Non-IBD: -15±11.2 pounds P=0.24 Semaglutide vs. liraglutide: P=0.04 Tirzepatide vs. semaglutide: P=0.01 Semaglutide vs. AOMs: Phentermine-topiramate: P<0.001 Bupropion-naltrexone: P<0.001 Orlistat: P=0.007	N.A	N.A
Gorelik <i>et al</i> , 2024 [16]	N.A	N.A	N.A
Herfarth <i>et al</i> , 2024[17]	N.A	N.A	N.A

(Contd...)

Supplementary Table 3 (Continued)

Author (Year)	Weight change (%)	Glycemic control (HbA1c%)	Change in Lipids
Levine <i>et al</i> , 2024 [23]	Median BMI change (IBD cohort): −1.9 kg/m ² , P<0.01 Median BMI change (non-IBD controls): −1.7 kg/m ² , P<0.01	Mean HbA1c change (IBD cohort): −0.3%, P=0.01 Mean HbA1c change (non-IBD controls): −0.3%, P=0.03	IBD Cohort: Total cholesterol (n=34): • Baseline: 169 mg/dL (IQR: 130–189) • 12 months: 177 mg/dL (IQR: 151–207) • Change: +8 mg/dL, P<0.01 LDL (n=33): • Baseline: 79 mg/dL (IQR: 58–108) • 12 months: 89 mg/dL (IQR: 69–111) • Change: +10 mg/dL, P<0.01 HDL (n=34): • Baseline: 52 mg/dL (IQR: 40–60) • 12 months: 58 mg/dL (IQR: 44–66) • Change: +6 mg/dL, P=0.03 Non-IBD controls : Total cholesterol (n=31): 153 -> 151 mg/dL, P=0.85 LDL (n=28): 71 -> 69 mg/dL, P=0.20 HDL (n=31): 45 -> 46 mg/dL, P=0.69
Nielsen <i>et al</i> , 2024 [40]	N.A	N.A	N.A
Pham <i>et al</i> , 2024 [27]	%TBWL at 6 months: IBD group: 25.4% ± 6.7 Control group: 28.6% ± 5.9 P=0.03	N.A	N.A
St-Pierre <i>et al</i> , 2024 [24]	Median BMI change: 34.0 (IQR 31.0–38.2) to 31.0 (IQR 29.0–36.1) P<0.001 Median total body weight (TBW) change: Decreased by 8.15 kg (IQR −15.9 to −2.2 kg) P<0.001	Mean baseline: 5.45% (IQR 5.3–5.7) Mean change: −0.2% (IQR −0.4 to 0.0), P=0.0536	Total cholesterol: Baseline: 190.5 mg/dL (IQR 157.8–215) Follow up: 160 mg/dL P=0.0634 LDL: Baseline: 102.5 mg/dL (IQR 75.3–121.8) Follow up: 90.5 mg/dL (IQR 76.8–111.8) P=0.1859 HDL: Baseline: 55.5 mg/dL (IQR 46.3–69.8) Follow up: 49 mg/dL (IQR 44.5–53.5) P=0.0427 Triglycerides: Baseline: 116.5 mg/dL (IQR 80.5–165) Follow up: 88.5 mg/dL (IQR 67.5–109.3) P=0.4618
Villumsen <i>et al</i> , 2021 [41]	N.A	N.A	N.A
Sehgal <i>et al</i> , 2025 [22]	Mean weight before GLP-1RA: 102 kg (SD 26.4) Mean weight at 12–24 weeks: 97.6 kg (SD 28.0) Mean difference: −4.4 kg Mean % weight loss: 5.2% (SD 5.0) P<0.01 Mean % weight loss by agent (overall): Dulaglutide: 3.8% (SD 3.6) Semaglutide: 6.2% (SD 5.4) Liraglutide: 4.5% (SD 4.5) Exenatide: 1.5% (SD 1.3) Tirzepatide: 4.0% (SD 1.6)	N.A	N.A

AOMs, anti-obesity medications; BMI, body mass index; GLP-1RA, glucagon-like peptide-1 receptor agonist; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; IBD, inflammatory bowel disease; IQR, interquartile range; LDL, low-density lipoprotein; N.A., not available; SD, standard deviation; TBW, total body weight; TBWL, total body weight loss; TWL, total weight loss

Full search strategy

Search algorithm

PUBMED

RESULTS-71> last search 30/06/2025
("Inflammatory Bowel Diseases"[MeSH Terms]
OR "inflammatory bowel disease"[All Fields]
OR "inflammatory bowel diseases"[All Fields]
OR "colitis, ulcerative"[MeSH Terms]
OR "ulcerative colitis"[All Fields]
OR ("ulcerative"[All Fields] AND "colitis"[All Fields])
OR ("Crohn Disease"[MeSH Terms]
OR "crohn disease"[All Fields]
OR ("crohn"[All Fields] AND "disease"[All Fields])
OR ("crohn's disease"[All Fields]))
AND
("Glucagon-Like Peptide 1 Receptor Agonists"[MeSH
Terms]
OR "glucagon like peptide 1 receptor agonists"[All Fields]
OR "glp-1 receptor agonist"[All Fields]
OR "glucagon like peptide 1"[MeSH Terms]
OR dulaglutide[All Fields]
OR dulaglutide[Supplementary Concept]
OR exenatide[MeSH Terms]
OR exenatide[All Fields]
OR liraglutide[MeSH Terms]
OR liraglutide[All Fields]
OR liraglutide[Supplementary Concept]
OR semaglutide[All Fields]
OR semaglutide[Supplementary Concept]
OR tirzepatide[MeSH Terms]
OR tirzepatide[All Fields]
OR tirzepatide[Supplementary Concept])

SCOPUS-483 RESULTS

TITLE-ABS-KEY (crohn) AND TITLE-ABS-KEY
(disease)) OR TITLE-ABS-KEY ("crohn's disease")) AND
(TITLE-ABS-KEY ("glucagon like peptide 1 receptor agonist")
OR TITLE-ABS-KEY ("glucagon like peptide 1 receptor
agonists") OR TITLE-ABS-KEY ("glp-1 receptor agonist")
OR TITLE-ABS-KEY ("glp-1 receptor agonists") OR TITLE-
ABS-KEY ("glucagon like peptide 1") OR TITLE-ABS-KEY
(dulaglutide) OR TITLE-ABS-KEY (exenatide) OR TITLE-
ABS-KEY (liraglutide) OR TITLE-ABS-KEY (semaglutide)
OR TITLE-ABS-KEY (tirzepatide))

COCHRANE LIBRARY -12 RESULTS

- #1 inflammatory bowel disease
- #2 inflammatory bowel diseases
- #3 ulcerative colitis
- #4 crohn's disease
- #5 crohn disease
- #6 #1 OR #2 OR #3 OR #4 OR #5
- #7 glucagon like receptor 1 agonist
- #8 glp-1 ra
- #9 glp-1
- #10 glp-1/gip
- #11 dulaglutide
- #12 semaglutide
- #13 exenatide
- #14 tirzepatide
- #15 liraglutide
- #16 #7 OR #8 OR #9 OR #10 OR #11 OR #12 OR #13 OR
#14 OR #15
- #17 #6 AND #16