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Anti-inflammatory effects of tirzepatide: a systematic review and meta-analysis

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Abstract

Tirzepatide, a dual GIP and GLP-1 receptor agonist, has shown significant metabolic benefits and weight reduction, but its anti-inflammatory effects have been less studied. This study was conducted in accordance with PRISMA guidelines, including observational (cohort) studies and randomized clinical trials that evaluated tirzepatide use and reported percentage changes in high-sensitivity C-reactive protein (hsCRP) and interleukin-6 (IL-6). A random-effects model was used. Seven randomized clinical trials and one observational study were included (six studies were eligible for meta-analysis). Compared to placebo, tirzepatide reduced hsCRP (mean difference [MD]: −32.9; 95% confidence interval [CI]: −33.6 to −32.2; I^2=15.3%) and IL-6 (MD: −17.8; 95% CI: −24.3 to −11.3; I^2 = 1.6%). Levels of hsCRP were significantly reduced with tirzepatide at 15 mg (MD: −32.9; 95% CI: −33.6 to −32.2; I^2=4.4%), 10 mg (MD: −33.9; 95% CI: −50.3 to −17.6; I^2=41.8%), and 5 mg (MD: −20.3; 95% CI: −35.2 to −5.3; I^2=0%). Similarly, IL-6 levels were significantly reduced with tirzepatide at 5 mg (MD: −18.8; 95% CI: −32.9 to −4.6; I^2=17.2%), 10 mg (MD: −17.9; 95% CI: −28.2 to −7.7; I^2=2.1%), and 15 mg (MD: −16.8; 95% CI: −31.1 to −2.6; I^2=47.4%). This study demonstrated that tirzepatide use is associated with a significant reduction in inflammatory markers, regardless of the population studied or treatment regimen.

Keywords Tirzepatide · Inflammation · C-reactive protein, interleukin 6

Abbreviations

T2DM	Type 2 diabetes mellitus
GLP-1RAs	Glucagon-like peptide-1 receptor agonists
GIP	Glucose-dependent insulinotropic peptide
hsCRP	High-sensitivity C-reactive protein
IL	Interleukin
MD	Mean difference
CI	Confidence intervals
SE	Standard error
MASLD	Metabolic dysfunction-associated steatotic liver disease
MCP-1	Monocyte chemoattractant protein-1

1 Introduction

The global public health landscape continues to be severely impacted by the closely Linked epidemics of obesity and type 2 diabetes mellitus (T2DM). The age-standardized prevalence of obesity among adults has increased in most countries between 1990 and 2022 [1]. According to the World Health Organization, in 2022, 43% of adults aged 18 years or older Worldwide were classified as overweight, and 16% were living with obesity [2]. In line with these findings, a recent meta-analysis reported that the global prevalence of overweight and obesity, irrespective of a country's economic status, was 37.0% [3]. Similarly, in 2022, approximately 828 million adults were living with T2DM, representing a rise of 630 million cases since 1990 [4]. Moreover,

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the 2025 edition of the International Diabetes Federation (IDF) Diabetes Atlas reports that 11.1% of adults aged 20 to 79 are affected by T2DM [5] In this context, the integration of weight-loss agents with lifestyle interventions such as diet and exercise gains considerable clinical importance.

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are associated with a variety of favorable metabolic effects and have been shown to induce substantial weight loss [6]. Importantly, this drug class has proven effective in reducing cardiovascular event rates in individuals with T2DM, obesity, or chronic kidney disease who have existing cardiovascular conditions or are at increased cardiovascular risk [7–10]. Tirzepatide, the most recently approved GLP-1-based medicine, a dual agonist of the two primary human incretins: GLP-1 and glucose-dependent insulinotropic peptide (GIP) [11]. Notably, tirzepatide has demonstrated superior clinical efficacy in terms of weight loss, metabolic control, and blood pressure reduction compared to the selective GLP-1RAs such as dulaglutide and semaglutide [12].

Chronic inflammation significantly contributes to the pathogenesis of atherosclerosis and its cardiovascular outcomes [13]. Similarly, elevated inflammatory markers are predictive of the development and progression of T2DM and its complications [14]. – [15] As a result, the anti-inflammatory effects of GLP-1-based medicines may constitute a complementary pathway underlying their cardiovascular protective effects. This is supported by findings from a prior meta-analysis, which revealed notable reductions in multiple inflammatory biomarkers following treatment with selective GLP-1RAs [16]. – [17] These studies did not include trials with tirzepatide. In light of the aforementioned evidence, the main objective of this study was to conduct a systematic review and meta-analysis evaluating the anti-inflammatory effects of tirzepatide.

2 Materials and methods

2.1 Data extraction and quality assessment

This study followed the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) for reporting systematic reviews [18]. It was registered in PROSPERO (CRD420251059211). A comprehensive Literature search was conducted to identify clinical trials involving tirzepatide published up to May 22, 2025. Two independent reviewers performed the search across several databases, including PubMed/MEDLINE, Scielo, Latindex, and Lilacs. The literature search was conducted using the PubMed/MEDLINE, SciELO, Latindex, and LILACS databases. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords related to tirzepatide and inflammatory markers, including C-reactive protein (hsCRP), cytokines, and interleukins (ILs). Boolean operators ("AND", "OR") were used to combine terms and refine results. The exact search strings for each database are as follows: a)

PubMed/MEDLINE search string: ("tirzepatide"[All Fields] OR "LY3298176"[All Fields]) AND("inflammation"[MeSHTerms]OR"inflammation"[All Fields] OR "C-Reactive Protein"[MeSH Terms] OR "hsCRP"[All Fields] OR "cytokines"[MeSH Terms] OR "cytokines"[All Fields] OR "interleukins"[MeSH Terms] OR "interleukin"[All Fields]); b) SciELO, Latindex, and LILACS search terms: tirzepatide OR LY3298176 AND inflammation OR hsCRP OR cytokines OR IL. Additionally, a grey literature search was conducted using sources such as the ClinicalTrials.gov website, international conference presentations, and information available on the drug manufacturer's official website (Eli Lilly). A snowballing strategy was also employed to identify further relevant studies. Only studies involving human participants were considered, and no language restrictions were applied during the search process.

The inclusion criteria for this study were as follows: (a) experimental or observational studies (retrospective or prospective cohorts) conducted in humans; (b) studies investigating the anti-inflammatory effects of tirzepatide, specifically focusing on hsCRP, IL-6, and IL-1 levels, with clinical trials comparing these effects to either a placebo or a control group; and (c) studies with a follow-up duration of at least 3 months. The choice of this time horizon was based on robust evidence indicating that approximately 3 months (≥12 weeks) is a reasonable follow-up period to detect stable and clinically meaningful changes in hsCRP across various interventions, including statins, diet, and bariatric surgery [19–21]. Studies were excluded if they consisted of expert opinions, reviews, cross-sectional studies, case-control studies, or case series.

Potential risks of bias were assessed for all included studies using the Cochrane tool designed for this purpose [22]. – [23] For non-randomized studies, the ROBINS-I tool evaluates bias across seven domains: confounding bias; selection bias in participant recruitment; bias in the classification of interventions; bias due to deviations from the intended intervention; bias arising from missing data; measurement bias of outcomes; and bias in the selection of the reported result. Each domain was rated as "serious," "moderate," or "low," based on the authors' judgment. For randomized clinical trials, the RoB-2 tool assesses bias in five domains: bias from randomization; bias due to deviations from the planned intervention; bias due to missing outcome

data; bias in outcome measurement; and bias in the selection of reported outcomes. Each domain was rated as "high risk," "low risk," or "some concerns". An overall risk rating was assigned to each study.

Initially, we conducted a qualitative analysis of all studies that fulfilled the inclusion and exclusion criteria. Subsequently, a quantitative analysis (meta-analysis) was performed using studies that provided the necessary data for implementation. The reduction in inflammatory markers was evaluated after once-weekly subcutaneous administration of tirzepatide at doses of 5, 10, or 15 mg.

2.2 Statistical analysis

The effect of tirzepatide treatment on the percentage reduction in hsCRP and IL-6 levels was calculated. Effect sizes were reported as mean differences (MDs) with their corresponding 95% confidence intervals (95% CIs). When summary or dispersion measures were reported as values other than means and standard deviations, conversion methods recommended in the literature were employed [24]. Additionally, the I^2 statistic was calculated to assess heterogeneity and inconsistency across studies. A random-effects model was selected due to variations in the populations studied and follow-up durations, as well as the elevated heterogeneity observed in some subgroups (doses). To compare mean effects between subgroups, a Z-test was employed. Statistical significance was defined as a p-value of 0.05 (two-tailed). The analysis was performed using R statistical software (version 3.5.1) [25].

2.3 Sensitivity analysis

Sensitivity analysis was conducted by replicating the meta-analysis results while excluding one study at a time. If the results remain consistent in terms of effect direction, magnitude, and statistical significance, this suggests that the findings are robust.

2.4 Analysis of publication bias

A funnel plot using the standard error (SE) of the MD was created, and Egger's regression intercept tests were performed.

3 Results

The database search initially identified 244 potentially relevant articles. After screening titles and abstracts, 202 studies were excluded because they were duplicate publications or did not align with the objectives of this study. Of the remaining 42 articles, 34 were further excluded following full-text review, primarily because the exposure or event of interest was not reported. The study selection process is summarized in the flowchart shown in Fig. 1.

One observational study, two abstract communications (containing information from three studies), and four randomized clinical trials were included in the qualitative analysis [26–31]. Of these, six studies provided the inflammatory marker data necessary for quantitative analysis through meta-analysis. The inflammatory markers reported and subsequently evaluated were hsCRP and IL-6; no data were available for IL-1. The main characteristics of the included studies are summarized in Table 1, and their quality assessment is presented in Fig. 2.

The only observational study included in this systematic review was a retrospective cohort study conducted among 54 Japanese patients with T2DM and metabolic dysfunction-associated steatotic liver disease (MASLD) [31]. The study aimed to evaluate the effects of switching from a selective GLP-1RA to tirzepatide. Six months after the switch, a significant reduction in hsCRP levels was observed (from 3 ± 0.85 mg/L to 2.24 ± 0.97 mg/L, $p < 0.001$), suggesting that tirzepatide may have an anti-inflammatory effect. Notably, the therapeutic benefits did not differ according to the specific GLP-1RA previously used (semaglutide or dulaglutide). This study was excluded from the quantitative analysis due to the lack of dispersion measures for the percentage reduction in hsCRP and the absence of a control group.

The other study included in the systematic review but excluded from the meta-analysis was a report by Bhatt et al. [27] In this case, hsCRP concentrations were measured at baseline and after 52 weeks in 1,995 fasting participants from the SURPASS-4 trial, distributed as follows: 329, 328, and 338 participants received 5, 10, and 15 mg of once-weekly subcutaneous tirzepatide, respectively, while 1,000 participants received insulin glargine. After 52 weeks of treatment, the hsCRP levels decreased by 38.0%, 44.2%, and 47.8% in the 5, 10, and 15 mg tirzepatide groups, respectively, compared to a 0.6% increase in the insulin glargine group. Interestingly, the reduction in hsCRP levels among the pooled tirzepatide groups correlated with a reduction in body mass index. This study was excluded from the quantitative analysis due to the absence of dispersion measures associated with the percentage reduction in inflammatory markers.

Of the studies included in the meta-analysis, two enrolled patients with T2DM, two focused on patients with obstructive sleep apnea (OSA), with or without positive airway pressure treatment; one involved overweight or obese individuals without T2DM; and one included patients with both obesity and heart failure. The quantitative analysis showed that once-weekly subcutaneous administration

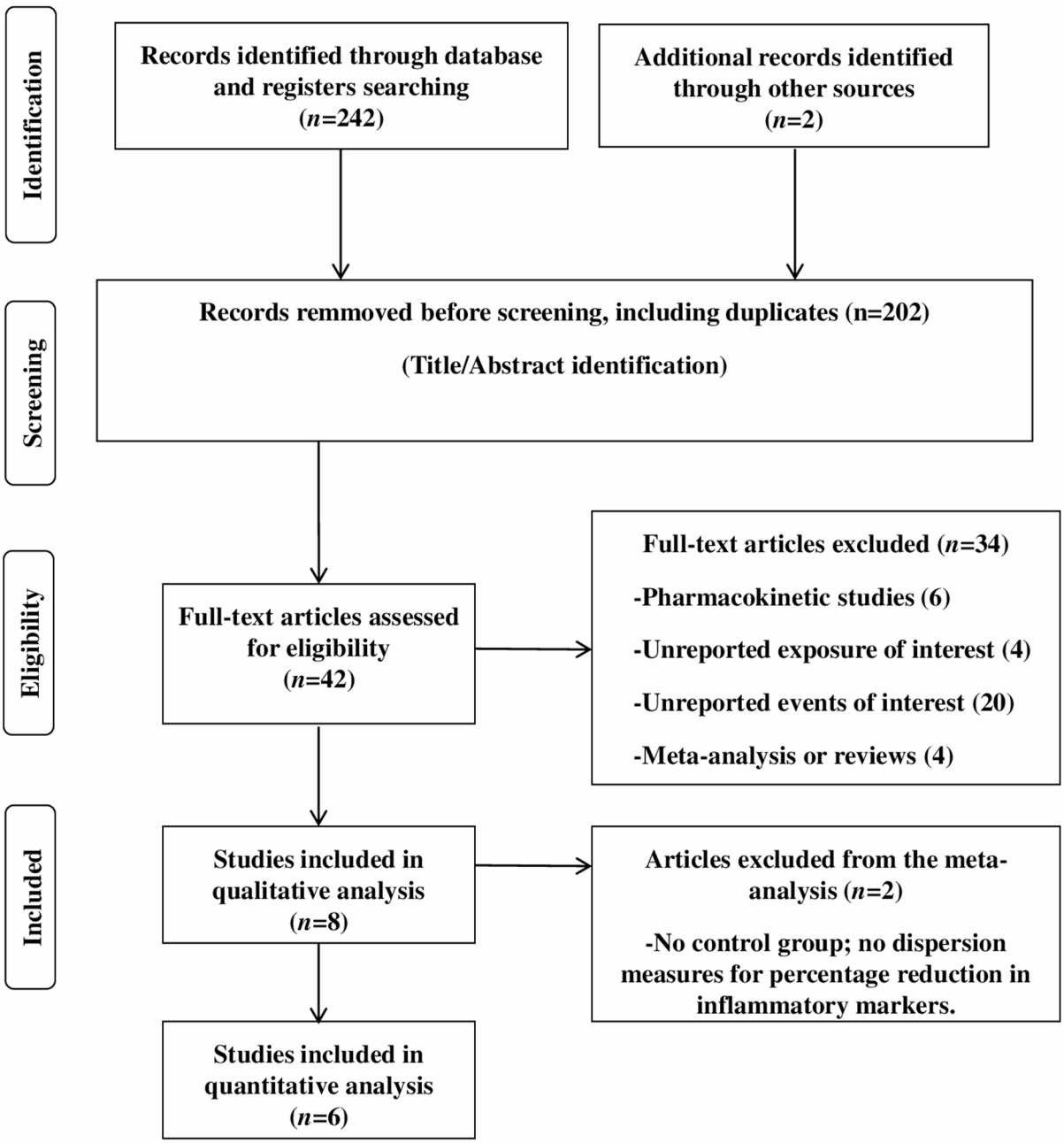

Fig. 1 Flow diagram of the study screening process

of tirzepatide, at any dose, significantly reduced levels of hsCRP (MD −32.9; 95% CI −33.6 to −32.2; I^2 = 15.3%) and IL-6 (MD −17.8; 95% CI −24.3 to −11.3; I^2 = 1.6%) compared to placebo. The use of 15 mg of tirzepatide was associated with significantly lower hsCRP levels compared to placebo (MD −32.9; 95% CI −33.6 to −32.2, I^2=4.4%). Likewise, although fewer studies evaluated these doses, similar effects were observed with 5 mg (MD −20.3; 95% CI −35.2 to −5.3, I^2=0%) and 10 mg (MD −33.9; 95% CI −50.3 to −17.6, I^2=41.8%) of tirzepatide (Fig. 3).

Table 1 Characteristics of studies included in the systematic review

Study (year)	Design	*n*	Population and therapeutic regimen evaluated	Inflammatory parameters evaluated	Follow-up (months)
Wilson et al. (2022)[26]	Post hoc analysis of RCT	318	Patients with T2DM inadequately controlled with diet and exercise alone or with stable metformin therapy, and a BMI between 23 and 50 kg/m². Mean age: 57.2 years; mean BMI: 32.6 kg/m²; 52.8% male. Tirzepatide 1, 5, 10, and 15 mg sc once weekly vs. placebo vs. dulaglutide.	hsCRP and IL-6	6
Bhatt et al. (2023)[27]	Post-hoc analysis of RCT*	1995	Patients with T2DM and established CVdisease or at high risk for it. Mean age: 63.6 years; mean BMI: 32.6 kg/m²; 62% male. Tirzepatide 5, 10, and 15 mg sc once weekly vs. insulin glargine.	hsCRP	12
SURMOUNT-OSA Trial I (2024)[28]	RCT	234	Patients with moderate-to-severe OSA and obesity who were not receiving positive airway pressure treatment. Mean age: 47.9 years; mean BMI: 39.1 kg/m²; 67.1% male. Tirzepatide (up to 15 mg sc once weekly) vs. placebo.	hsCRP	12
SURMOUNT-OSA Trial II (2024)[28]	RCT	235	Patients with moderate-to-severe OSA and obesity who were receiving positive airway pressure treatment. Mean age: 51.7 years; mean BMI: 38.7 kg/m²; 72.3% male. Tirzepatide (up to 15 mg sc once weekly) vs. placebo.	hsCRP	12
Sattar et al. Trial I, (2024)[29]	Post hoc analysis of RCT**	400	Patients with a BMI of 30 kg/m² or higher, or 27 kg/m² or higher with at least one weight-related complication, excluding T2DM. Mean age: 44.9 years; mean BMI: 38 kg/m²; 32.5% male. Tirzepatide 5, 10, and 15 mg sc once weekly vs. placebo.	hsCRP and IL-6	18
Sattar et al. Trial II, (2024)[29]	Post hoc analysis of RCT***	300	Patients with T2DM, a BMI of 27 kg/m² or higher, and an HbA1c between 7% and 10%. Mean age: 54.2 years; mean BMI: 36.1 kg/m²; 49% male. Tirzepatide 10 and 15 mg sc once weekly vs. placebo.	hsCRP and IL-6	18
SUMMIT (2025)[30]	RCT	731	Patients with obesity and heart failure with an ejection fraction of at least 50%. Mean age: 65.2 years; mean BMI: 38.3 kg/m²; 46.2% male. Tirzepatide (up to 15 mg sc once weekly) vs. placebo.	hsCRP	12
Okuma et al. (2025)[31]	Retrospective cohort	54	Japanese patients with T2DM and MASLD switching from a GLP-1RA to tirzepatide (5 mg sc once weekly). Mean age: 57.2 years; mean BMI: 26.8 kg/m²; 53.7% male.	hsCRP	6

BMI body mass index; *CV* cardiovascular; *GLP-1RA* Glucagon-like peptide-1 receptor agonists; *hsCRP* High-sensitivity C-reactive protein; *IL* interleukin; *MASLD* Metabolic dysfunction-associated steatotic liver disease; *OSA* obstructive sleep apnea; *RCT* Randomized clinical trial; *sc* subcutaneously; *T2DM* type 2 diabetes mellitus*SURPASS-4 trial.**SURMOUNT-1 trial. ***SURMOUNT-2 trial

In addition, the available evidence suggests that once-weekly subcutaneous administration of tirzepatide at doses of 5 mg (MD −18.8; 95% CI −32.9 to −4.6, I^2 =17.2%), 10 mg (MD −17.9; 95% CI −28.2 to −7.7, I^2=2.1%), and 15 mg (MD −16.8; 95% CI −31.1 to −2.6, I^2=47.4%), compared to placebo, is associated with a reduction in IL-6 levels (Fig. 4).

The funnel plots of standard error versus MD for hsCRP and IL-6 levels did not suggest the presence of publication bias (Supplementary Figs. 1 and 2). Similarly, Egger's regression intercept tests yielded p-values of 0.70 and 0.47 for the hsCRP and IL-6 analyses, respectively, further indicating no evidence of publication bias.

The sensitivity analysis demonstrated consistent directionality and magnitude of the overall results when each trial was excluded sequentially **(**Figs. 5 and 6**).**

4 Discussion

In this systematic review and meta-analysis, we found that treatment with tirzepatide was associated with reductions in hsCRP and IL-6 levels. Although a previous meta-analysis demonstrated reductions in hsCRP with GLP-1-based medicines in patients with OSA [32], to our knowledge, this is

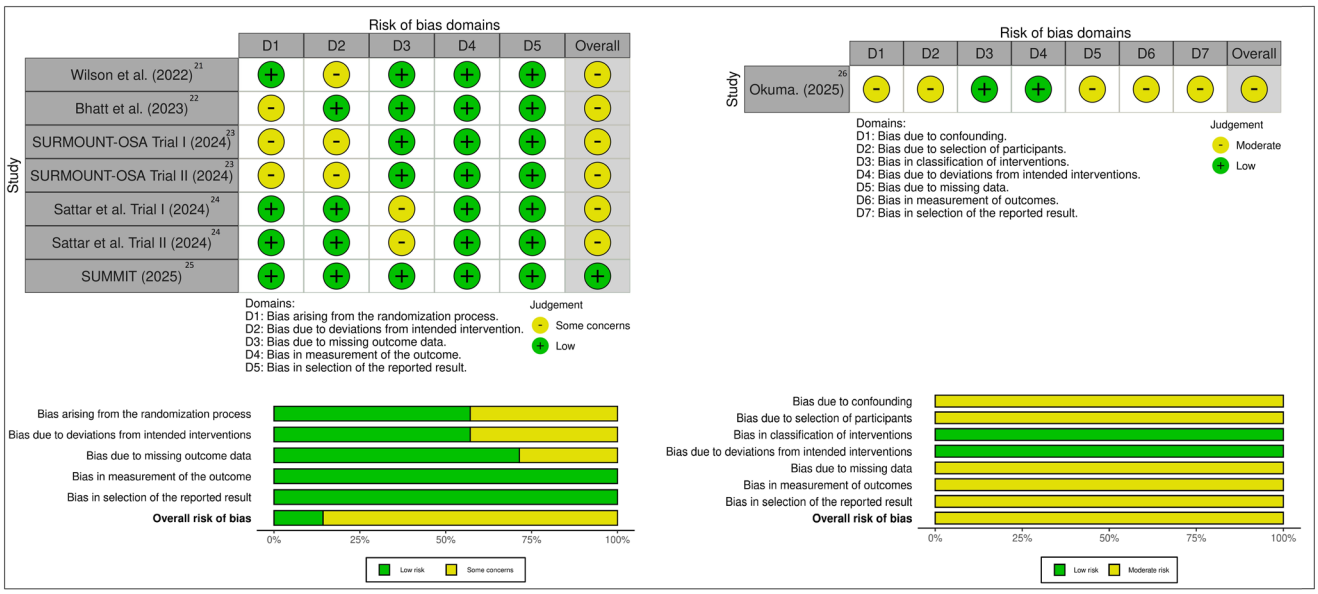

Fig. 2 Summary and evaluation of individual bias of included studies

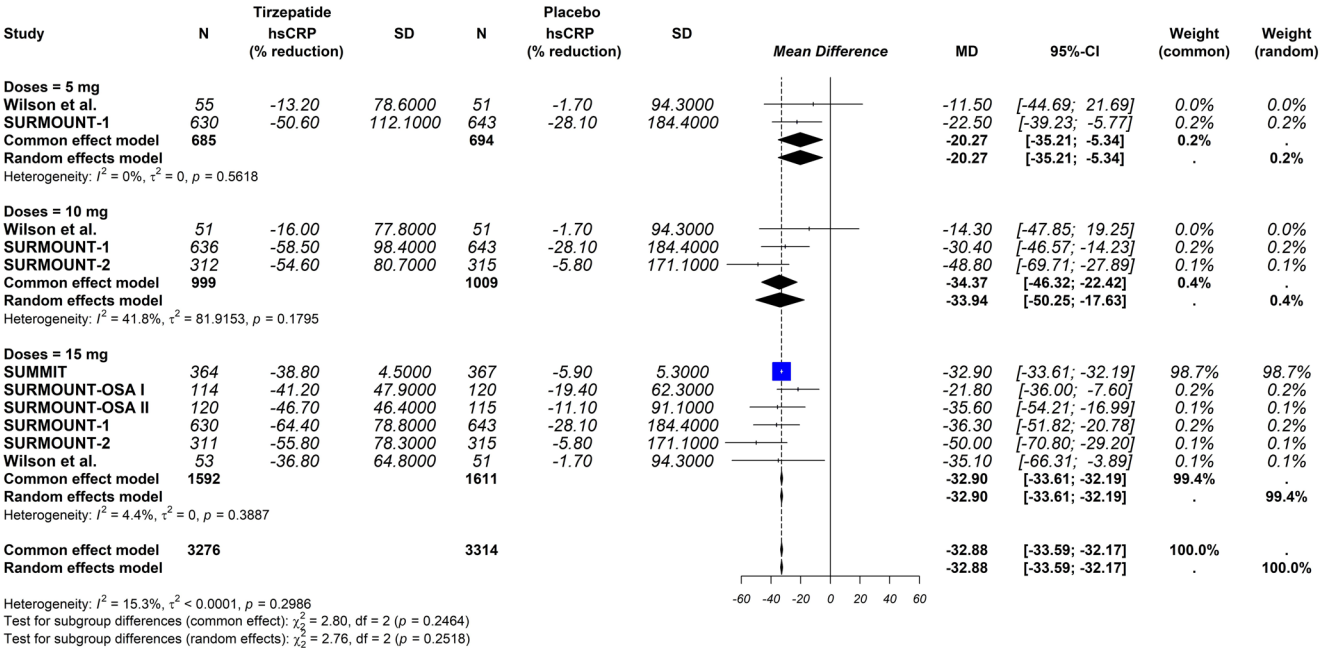

Fig. 3 Percentage reduction in high sensitivity C-reactive protein (hsCRP) levels with tirzepatide therapy. Random-effects model, mean difference (MD), 95% confidence intervals (CI) and statistical I^2

the first meta-analysis to specifically evaluate the effect of tirzepatide on inflammatory markers.

Tirzepatide is the first dual peptide agonist developed for the treatment of T2DM, acting on both the GIP and GLP-1 receptors. GIP plays a regulatory role in gastrointestinal and metabolic processes by inhibiting gastric secretions, ENHANCING INSULIN SECRETION, and exerting insulin-mimetic effects on adipose tissue, specifically through the suppression of lipolysis and the promotion of lipogenesis [33]. Likewise, GLP-1 contributes to glucose homeostasis and weight reduction by enhancing insulin secretion, suppressing glucagon release, delaying gastric emptying, and promoting satiety [34]. Clinical trials in individuals with T2DM have shown that tirzepatide produces greater improvements in key health indicators—including body weight, HbA1c levels, lipid profiles, and blood pressure—compared

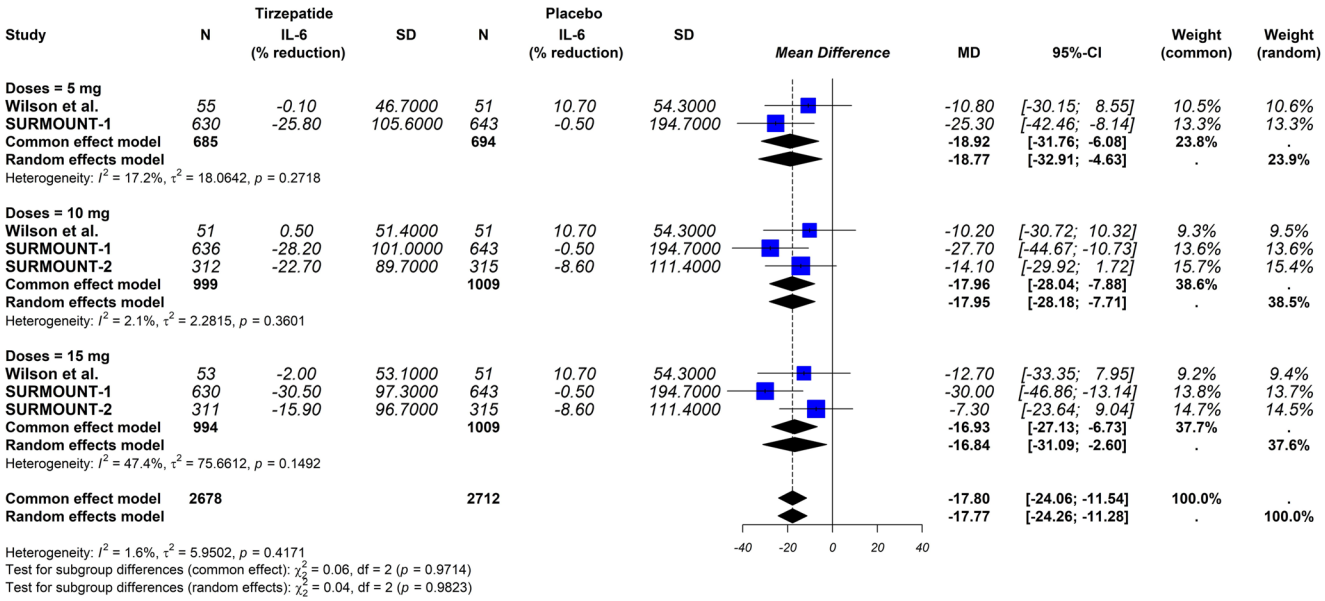

Fig. 4 Percentage reduction in interleukin (IL)−6 levels with tirzepatide therapy Random-effects model, mean difference (MD), 95% confidence intervals (CI) and statistical I^2

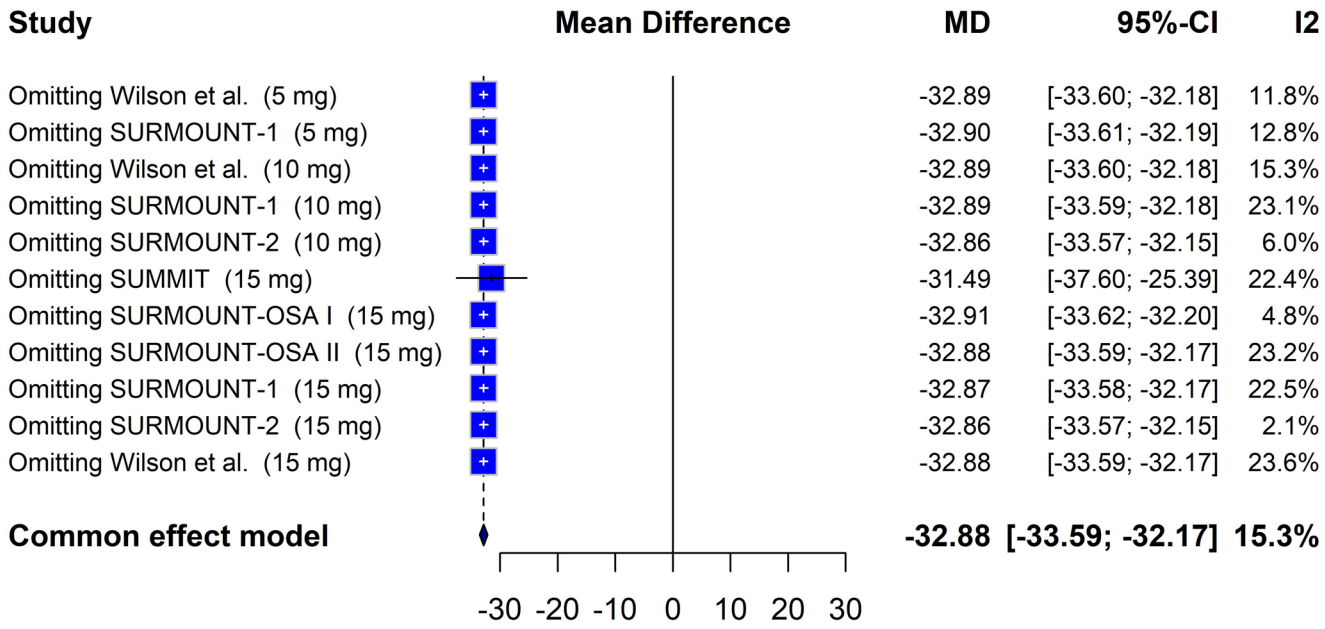

Fig. 5 Sensitivity analysis of studies including high-sensitivity C-reactive protein (hsCRP)

to selective GLP-1RAs such as dulaglutide and semaglutide [35]. In addition to its positive effects on metabolic parameters and traditional cardiovascular risk factors, tirzepatide—like the selective GLP-1RAs—has also been suggested to exert additional beneficial pathophysiological effects, including anti-inflammatory properties [36].

Animal studies have demonstrated an association between GIP receptor activity and both adipose tissue inflammation and low-grade systemic inflammation, as well as the modulatory effects of tirzepatide on these inflammatory processes [37–39]. In humans, Gögebakan et al. demonstrated that the nutrient-induced gut hormone GIP may initiate adipose tissue inflammation by promoting crosstalk between adipocytes and macrophages, involving monocyte chemoattractant protein-1 (MCP-1), a proinflammatory chemokine implicated in T2DM and cardiovascular disease [40].

CRP is primarily produced by the liver in response to proinflammatory cytokines, particularly IL-6, with additional

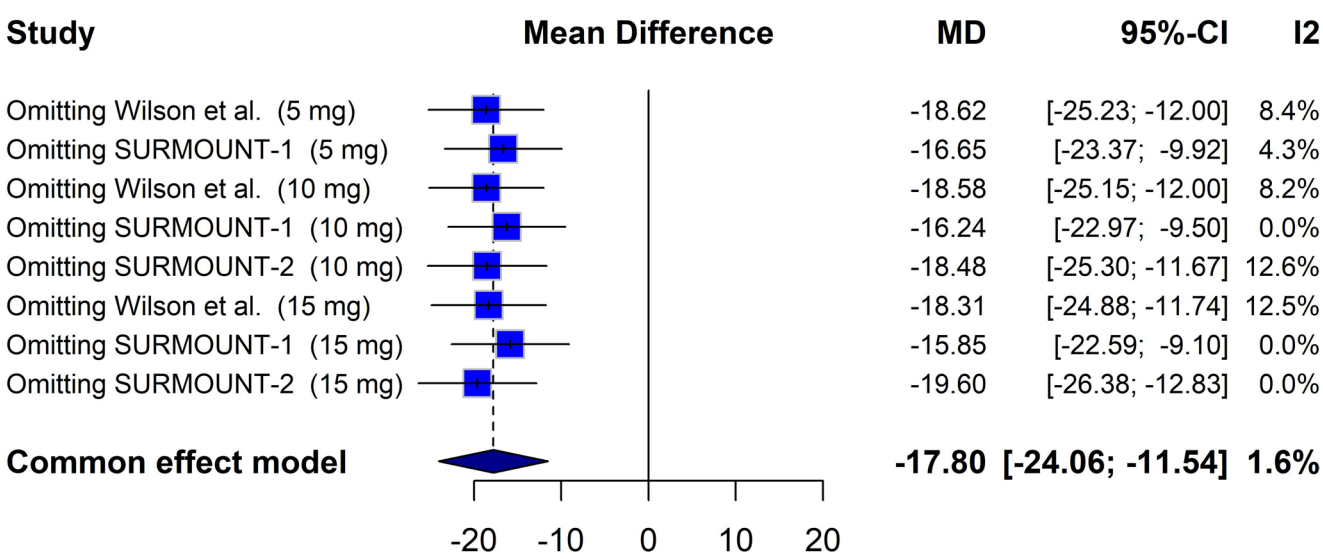

Fig. 6 Sensitivity analysis of studies including interleukin (IL)−6

contributions from IL-1β, IL-17, and TNF-α [41]. Elevated circulating hsCRP, IL-1, and IL-6 levels are widely recognized as a surrogate markers of systemic inflammation and have been consistently associated with increased cardiovascular risk in epidemiological studies [42]. – [43] This underscores the role of inflammation as a central mechanism in atherogenesis and cardiovascular events. Importantly, various pharmacological agents evaluated in the context of cardiovascular prevention have demonstrated dual benefits—reducing hsCRP levels and lowering the incidence of major cardiovascular events. These agents include statins, colchicine, IL-1β receptor antagonists, and bempedoic acid, highlighting the potential clinical relevance of targeting inflammatory pathways alongside traditional risk factors [44–47].

Emerging evidence indicates that the selective GLP-1RAs may exhibit a similar dual effect, combining anti-inflammatory properties with cardiovascular protective benefits [8–10, 16]. – [17] However, current evidence regarding the anti-inflammatory effects of tirzepatide remains limited, and to date, no completed clinical trials have specifically assessed its impact on cardiovascular outcomes.

Our analysis included studies conducted in diverse populations, with a predominant focus on individuals with obesity and T2DM. Specifically, 50% of the included studies used T2DM as an explicit inclusion criterion [26]. – [27, 29, 31] In terms of obesity, 7 out of the 8 studies analyzed populations with a body mass index within the obesity range (≥30 kg/m²) [26–30], with the only exception being the Japanese cohort reported by Okuma et al. [28] This is particularly relevant, as obesity induces chronic systemic inflammation, which can contribute to the development of insulin resistance, β-cell dysfunction, and ultimately T2DM [48]. Furthermore, this persistent inflammatory state plays a central role in the progression of T2DM and its long-term complications, including cardiovascular disease, MASLD, diabetic retinopathy, cardiovascular disease, and diabetic nephropathy.

Additionally, certain studies included in this meta-analysis evaluated specific subgroups, such as patients with heart failure [30], MASLD [31], or obstructive sleep apnea [28], thereby broadening the clinical context in which tirzepatide's effects were assessed. Notably, the reduction in hsCRP levels observed with tirzepatide in patients with heart failure was comparable to the 43.5% decrease reported in the STEP-HFpEF trial with semaglutide [49].

Our study demonstrated an average reduction in hsCRP levels of approximately 33% in the overall analysis, regardless of the tirzepatide dose administered. This reduction compared to placebo is comparable to that reported with semaglutide (37.8%) [8] and somewhat greater than the reduction observed with liraglutide (18.2%) in obese patients at high cardiovascular risk, without or with T2DM, respectively [50]. Only a few studies evaluating tirzepatide have assessed IL-6 levels. Overall, the reduction observed appears to be smaller than that seen for hsCRP, likely influenced by the study conducted by Wilson et al. [31], which reported virtually no effect on this inflammatory marker.

Finally, this meta-analysis is primarily based on the evaluation of hsCRP as an inflammatory marker. hsCRP is currently recognized as the most extensively studied biomarker of inflammation, with robust epidemiological and clinical evidence linking it to the risk of cardiovascular events in both primary and secondary prevention settings [51]. However, its prognostic value should be interpreted with caution due to its nonspecific nature and its role as an indirect marker

rather than a direct causal factor in disease processes. The incremental contribution of hsCRP to cardiovascular risk prediction models remains limited, and its routine integration into clinical decision-making algorithms remains controversial among major guidelines [52]. Importantly, given the wide range of conditions that can lead to elevated hsCRP levels, marginal increases may be difficult to interpret and should not be considered in isolation but rather within the broader clinical context. Therefore, the development of new, more specific inflammatory biomarkers is likely needed for routine clinical use, particularly as tools for prognostic assessment or to predict therapeutic response.

This meta-analysis has several limitations. First, clinical heterogeneity was evident due to variations in population characteristics and differing follow-up durations across studies. Additionally, statistical heterogeneity was substantial; however, sensitivity analyses confirmed the robustness of the overall findings. Second, the absence of reported data in the original studies prevented the analysis of additional inflammatory markers. Third, the inability to account for concomitant therapies and comorbidities that may influence inflammatory status represents another limitation. Fourth, some of the included studies (e.g., Bhatt et al., Sattar et al.) were post hoc analyses of randomized controlled trials. While the primary studies benefit from rigorous design, post hoc analyses are inherently subject to limitations such as selective outcome reporting and residual confounding. Therefore, their findings should be interpreted with caution, as they may introduce bias into our synthesis. Fifth, this meta-analysis did not include searches of other databases, such as Embase and Cochrane CENTRAL, which could have increased the risk of missing relevant studies and introduced potential selection bias. Finally, an important limitation of this meta-analysis is the small number of included studies, which limits the power of formal tests for publication bias. Although we performed visual inspection of the funnel plot and Egger's regression test, these methods are known to be unreliable when fewer than 10 studies are included. Therefore, the assessment of publication bias in our analysis should be interpreted with caution.

5 Conclusion

This systematic review and meta-analysis demonstrated a significant anti-inflammatory effect associated with tirzepatide use. However, given that our analysis was limited to two inflammatory markers and included a relatively small number of studies, these conclusions should be interpreted with caution and confirmed by future research. If the cardiovascular benefits of tirzepatide are validated in ongoing clinical trials, the inflammatory pathway may emerge as an additional pathophysiological mechanism—complementary to the drug's favorable effects on glycemic control, lipid metabolism, body weight, and blood pressure—that could contribute to its overall cardiometabolic profile.

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Author contributions WM and JPN participated in the conception and design of the research. WM, LB and PT participated in the data collection. The interpretation of the data and the statistical analysis was done by WM and ML. WM, LB, JPN and JPF drafted the manuscript. All authors performed a critical review of the final document. All authors have read and agreed to the published version of the manuscript.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Ethical approval This article is based on previously conducted studies and does not contain any studies with human participants or animals performed by any of the authors.

Conflict of interest WM and ML have served as a speaker from Novo Nordisk. JPF has the following disclosures: Research support (Akero, Altimmune, Boehringer Ingelheim, 89bio, Eli Lilly, Janssen, Madrigal, Merck, NorthSea Therapeutics, Novartis, Novo Nordisk, Oramed, Pfizer, Sanofi, and Shionogi), advisory boards and consulting (Akero, Altimmune, Boehringer Ingelheim, 89bio, Eli Lilly, Gilead, Intercept, Merck, Novo Nordisk, Pfizer, Sanofi), speaker bureau (Eli Lilly), and employee and stockholder (Biomea Fusion, Inc.). The rest of the authors have no conflicts to declare. The relationships disclosed by the authors did not influence the design, conduct, or interpretation of the data presented in this study.

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