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Meta-Analysis of the Efficacy of Different Glucagon-Like Peptide-1 Receptor Agonists in Patients with Type 2 Diabetes Mellitus and Comorbid Non-Alcoholic Fatty Liver Disease

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Abstract: This meta-analysis aimed to evaluate the efficacy of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) in patients with type 2 diabetes mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD) and to explore potential sources of heterogeneity. PubMed, Web of Science, Embase, and the Cochrane Library were searched from inception to August 2026 for randomised controlled trials (RCTs), and meta-analyses were performed using RevMan 5.3 and Stata 17.0, with subgroup analyses and GRADE assessment. Eight RCTs involving 602 patients were included. Compared with control interventions, GLP-1 RAs significantly reduced body mass index (MD −1.43 kg/m²), alanine aminotransferase (MD −8.11 U/L), aspartate aminotransferase (MD −4.23 U/L), fasting plasma glucose (MD −0.24 mmol/L), and 2-h postprandial glucose (MD −1.95 mmol/L), but had no significant effects on body weight, lipid profiles, HbA1c, or HOMA-IR ($P > 0.05$). Subgroup analyses suggested that exenatide was associated with greater reductions in liver enzymes than liraglutide; treatment durations of ≥24 weeks were associated with greater improvements in HbA1c and HOMA-IR; patients with BMI ≥29 kg/m² showed significant HOMA-IR reduction; and background therapy was associated with reductions in total cholesterol and triglycerides, whereas BMI and ALT improved more markedly without concomitant background therapy. In conclusion, GLP-1 RAs may improve BMI, liver enzyme levels, and glycaemic control in patients with T2DM and NAFLD, although their overall effects on lipid profiles and insulin resistance remain uncertain; treatment effects may vary according to the GLP-1 RA used, treatment duration, BMI, and concomitant therapy, and further well-designed RCTs are needed.

1. Introduction

Type 2 diabetes mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD), now termed metabolic dysfunction-associated steatotic liver disease (MASLD), frequently coexist[1]. The

global pooled prevalence of NAFLD in T2DM is 65.04%, with 31.55% progressing to steatohepatitis and 35.54% having clinically significant fibrosis[2]. Insulin resistance is a shared driver: it promotes hepatic triglyceride accumulation, while MASLD-associated inflammation and liver-derived insulin resistance worsen glycaemic control, forming a vicious cycle[3-6].

Current pharmacological options for MASLD remain limited. The 2024 EASL/EASD/EASO guidelines emphasise lifestyle intervention and optimisation of metabolic comorbidities[7]. Resmetirom and subcutaneous semaglutide are approved for MASH with moderate-to-advanced fibrosis, but traditional glucose-lowering agents have limited hepatic benefit and are constrained by weight regain, adverse effects, and lack of standardised regimens[8-9]. Therapies that simultaneously improve glycaemia, weight, and liver outcomes are therefore needed[10]. GLP-1 RAs have attracted attention for these multifaceted benefits[11-12].GLP-1 RAs improve T2DM with MASLD through multiple mechanisms. They suppress appetite, delay gastric emptying, and enhance glucose-dependent insulin secretion, thereby improving glycaemic control[13-14]. In the liver, they reduce fat accumulation, inflammation, oxidative stress, and insulin resistance[5, 15-16]. These multi-target actions address shared upstream drivers—excess adiposity and insulin resistance—offering dual metabolic and hepatic benefits[17-18].

Previous meta-analyses have often pooled patients with T2DM and non-T2DM populations or examined each condition separately, limiting their applicability to T2DM complicated by NAFLD/MASLD. Recent high-quality RCTs have also not been fully incorporated. This meta-analysis therefore assessed the efficacy of different GLP-1 RAs in patients with T2DM and NAFLD/MASLD to inform precision pharmacotherapy.

2. Methods

This study was designed, conducted, and reported in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [19]. The protocol for this systematic review has been registered in the PROSPERO database (Registration ID: CRD420261531164).

2.1 Inclusion and Exclusion Criteria

Inclusion criteria were defined using PICOS: (P) adults aged ≥18 years with confirmed T2DM (WHO or ADA criteria) and MASLD diagnosed by imaging or histopathology; no restrictions on sex, ethnicity, or disease duration. (I) GLP-1 RAs (e.g., liraglutide, semaglutide, dulaglutide, exenatide), with or without standard therapy. (C) placebo, standard therapy, or lifestyle intervention. (O) valid quantitative data for at least one of: BMI/body weight; ALT/AST; TC/TG/LDL-C/HDL-C; FPG/2-h PPG/insulin/HbA1c/HOMA-IR. (S) RCTs.

Exclusion criteria were: non-T2DM populations; hepatic steatosis from other causes, cirrhosis, hepatocellular carcinoma, or liver transplantation; severe infection, malignancy, end-stage renal disease, pregnancy/lactation, or other conditions affecting outcome assessment; duplicate publication, insufficient or erroneous data; and non-RCT designs.

2.2 Literature Search Strategy

PubMed, Web of Science, Embase, and the Cochrane Library were searched from inception to August 15, 2026. Controlled vocabulary and free-text terms (title/abstract) were combined, and search strings were tailored to each database (Supplementary Table S1).The specific search terms were as follows:(("Non-alcoholic Fatty Liver Disease"[MeSH] OR "NAFLD"[tiab] OR "NASH"[tiab] OR "MAFLD"[tiab] OR "metabolic dysfunction-associated steatotic liver

disease"[tiab] OR "non-alcoholic steatohepatitis"[tiab] OR ((("fatty liver"[tiab] OR "hepatic steatosis"[tiab]) AND ("non-alcoholic"[tiab] OR "nonalcoholic"[tiab]))) AND ("Diabetes Mellitus, Type 2"[MeSH] OR "Type 2 Diabetes"[tiab] OR "T2DM"[tiab] OR "NIDDM"[tiab] OR ("diabet*"[tiab] AND ("type 2"[tiab] OR "type II"[tiab] OR "adult onset"[tiab] OR "non-insulin dependent"[tiab]))) AND ("Glucagon-Like Peptide-1 Receptor Agonists"[MeSH] OR "GLP-1 Receptor Agonist"[tiab] OR "GLP-1RA"[tiab] OR "Glucagon-Like Peptide 1"[tiab] OR "Incretin Mimetics"[tiab] OR "Liraglutide"[tiab] OR "Victoza"[tiab] OR "Semaglutide"[tiab] OR "Ozempic"[tiab] OR "Rybelsus"[tiab] OR "Wegovy"[tiab] OR "Dulaglutide"[tiab] OR "Trulicity"[tiab] OR "Exenatide"[tiab] OR "Byetta"[tiab] OR "Bydureon"[tiab] OR "Lixisenatide"[tiab] OR "Adlyxin"[tiab] OR "Tirzepatide"[tiab] OR "Mounjaro"[tiab] OR "Retatrutide"[tiab] OR "Orforglipron"[tiab] OR "Albiglutide"[tiab] OR "Efinopegdutide"[tiab]).

2.3 Literature Selection and Data Extraction

Records were imported into EndNote X9, and duplicates were removed by software detection and manual screening. Two researchers (YJ and YR) independently screened titles and abstracts against the eligibility criteria, excluding reviews, animal studies, conference abstracts, non-RCTs, and irrelevant records. Full texts of potentially eligible studies were then screened against the inclusion and exclusion criteria. Disagreements were resolved by discussion or third-party adjudication. Reasons for exclusion at each stage were recorded, and a PRISMA flow diagram was constructed.

Two researchers independently extracted data using a standardised Excel form: (1) study characteristics (first author, year, country); (2) baseline data (sample size, sex, age, BMI, NAFLD/MASLD diagnostic method); (3) intervention details (GLP-1 RA type, dose, frequency, duration); (4) control regimen; and (5) outcomes (post-intervention mean ± SD). When only standard errors (SEs) were reported, they were converted to SDs using the Cochrane Handbook formula[20]. Studies reporting only medians with interquartile ranges (IQRs) or change values were excluded. Attempts were made to obtain missing data from corresponding authors.

2.4 Risk of Bias Assessment of Included Studies

The Cochrane Risk of Bias tool was used to assess included studies [20]. Domains included random sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, incomplete outcome data, selective reporting, and other bias. Each domain was judged as low, high, or unclear risk. Two researchers independently assessed all studies and cross-checked the results.

2.5 Statistical Analysis

Data were synthesised using RevMan 5.3. For continuous outcomes, mean difference (MD) or standardised mean difference (SMD) with 95% CIs was used when units or measurement methods differed. Statistical significance was set at $P < 0.05$. Heterogeneity was assessed using Cochran's Q test and the I^2 statistic. A fixed-effects model was used when $P \geq 0.10$ and $I^2 \leq 50\%$; otherwise, a random-effects model was applied. Subgroup and leave-one-out sensitivity analyses explored sources of heterogeneity. Stata 17.0 was used to generate funnel plots and perform Egger's test for publication bias.

3. Results

3.1 Literature Selection Process and Results

The initial search retrieved 2,429 records. After duplicates were removed using EndNote X9, 1,341 records remained. Title and abstract screening excluded 1,231 records. The full texts of the remaining 110 records were reviewed, and 102 were excluded because of ineligible populations, non-RCT design, inappropriate interventions, unsuitable or unavailable outcomes, duplicate publication, or no-treatment control. Finally, 8 RCTs (602 patients) were included. The screening process is shown in Figure 1.

3.2 Baseline Characteristics of Included Studies

Eight RCTs (602 patients) were included (references 21-28); their characteristics are summarised in Table 1. All studies were conducted in China and published between 2013 and 2020, and all enrolled patients with T2DM and NAFLD. Mean age ranged from 42 to 58 years, and the proportion of female participants ranged from 27.6% to 56.7%. Specific inclusion criteria included newly diagnosed T2DM without prior glucose-lowering therapy (Lin 2020), obesity with markedly elevated liver enzymes (Shao 2014), and inadequate glycaemic control on metformin monotherapy (Guo 2020; Yan 2019; Zhang 2020). Baseline characteristics were comparable between groups in each study.

Control interventions included metformin (Fan 2013; Feng 2017; Tian 2018), insulin (Shao 2014; Lin 2020; Yan 2019), pioglitazone (Zhang 2020), or placebo (Guo 2020). All experimental groups received GLP-1 RAs: exenatide in three studies (Fan 2013; Lin 2020; Shao 2014) and liraglutide in five (Feng 2017; Guo 2020; Tian 2018; Yan 2019; Zhang 2020). Two studies (Guo 2020; Yan 2019) co-administered a stable dose of metformin. Exenatide doses ranged from 5 to 10 μg twice daily, and liraglutide from 0.6 to 1.8 mg/d. Treatment duration ranged from 12 to 26 weeks. Detailed data are shown in Table 1.

3.3 Risk of Bias

The Cochrane risk-of-bias tool was used to assess the eight included RCTs (Figure 2). All studies were rated low risk for random sequence generation, selective reporting, and other bias. Allocation concealment was low risk in three studies (37.5%) and unclear in five (62.5%). Performance bias was high risk in all studies. Detection bias was low risk in two studies (25.0%) and unclear in six (75.0%). Incomplete outcome data were low risk in seven studies (87.5%), with only Fan 2013 rated high risk.

Overall, methodological quality was moderate. The main risks were blinding, allocation concealment, and outcome assessment reporting. Future studies should improve methodological reporting transparency.

3.4 Meta-analysis Results

3.4.1 Effects of Different GLP-1 RAs on Anthropometric Measures

Eight studies (602 participants) were included [21-28]. Heterogeneity was substantial for both BMI($P < 0.00001$, $I^2 = 90\%$) and body weight($P < 0.00001$, $I^2 = 88\%$), so a random-effects model was used. GLP-1 RAs significantly reduced BMI (MD -1.43 kg/m^2, 95% CI -2.52 to -0.34, $P = 0.010$; Figure 3A) but did not significantly reduce body weight (MD −3.28 kg, 95% CI −7.36 to 0.81, $P = 0.12$; Figure 3B).

3.4.2 Effects of Different GLP-1 RAs on Liver Enzymes and Hepatic Injury

Seven studies (542 participants) were included [21-27]. Both ALT ($P < 0.0001$, $I^2 = 81\%$) and AST ($P = 0.006$, $I^2 = 67\%$) showed high heterogeneity, and a random-effects model was used. Compared with controls, GLP-1 RAs significantly reduced ALT (MD −8.11 U/L, 95% CI −14.43 to −1.78, $P = 0.01$; Figure 3C) and AST (MD −4.23 U/L, 95% CI −7.30 to −1.15, $P = 0.007$; Figure 3D).

3.4.3 Effects of Different GLP-1 RAs on Lipid Metabolism Profiles

TC was reported in 7 studies (21-23, 25-28; 531 participants), TG in 8 studies (21-28; 602 participants), and HDL-C and LDL-C in 6 studies each (21-23, 26-28; 471 participants). Heterogeneity was moderate for TC ($P = 0.23$, $I^2 = 26\%$) and HDL-C ($P = 0.09$, $I^2 = 47\%$), high for TG ($P = 0.005$, $I^2 = 65\%$), and negligible for LDL-C($P = 0.97$, $I^2 = 0\%$). Fixed-effects models were used for TC, HDL-C, and LDL-C, and a random-effects model for TG. GLP-1 RAs did not significantly affect TC (MD −0.08 mmol/L, 95% CI −0.24 to 0.08, $P = 0.33$; Figure 4A), TG (MD −0.14 mmol/L, 95% CI −0.29 to 0.02, $P = 0.09$; Figure 4B), HDL-C (MD 0.01 mmol/L, 95% CI −0.04 to 0.06, $P = 0.64$; Figure 4C), or LDL-C (MD −0.00 mmol/L, 95% CI −0.12 to 0.11, $P = 0.95$; Figure 4D).

3.4.4 Effects of Different GLP-1 RAs on Glycaemic Control and Insulin Resistance

FPG was reported in 7 studies (21-26, 28; 554 participants), 2-h PPG in 4 studies (22, 24, 26, 28; 375 participants), HbA1c in 8 studies (21-28; 602 participants), and HOMA-IR in 5 studies (22,23, 26-28; 413 participants). Heterogeneity was negligible for FPG ($P = 0.63$, $I^2 = 0\%$) and 2-h PPG ($P = 0.93$, $I^2 = 0\%$), but high for HbA1c ($P = 0.02$, $I^2 = 58\%$) and HOMA-IR ($P = 0.0004$, $I^2 = 80\%$). Fixed-effects models were used for FPG and 2-h PPG, and random-effects models for HbA1c and HOMA-IR. GLP-1 RAs significantly reduced FPG (MD −0.24 mmol/L, 95% CI −0.40 to −0.08, $P = 0.003$; Figure 5A) and 2-h PPG (MD −1.95 mmol/L, 95% CI −2.60 to −1.29, $P < 0.00001$; Figure 5B), but did not significantly reduce HbA1c (MD −0.16%, 95% CI −0.34 to 0.02, $P = 0.08$; Figure 5C) or HOMA-IR (MD −0.37, 95% CI −0.78 to 0.04, $P = 0.08$; Figure 5D).

3.5 Sensitivity Analysis

Leave-one-out sensitivity analyses were performed for outcomes with at least moderate heterogeneity (Table 2). Heterogeneity in TC, HDL-C, AST, TG, HbA1c, and HOMA-IR was mainly driven by individual studies: Yan et al.[27] for TC; Feng et al. [22] for HDL-C and AST; Guo et al.[23] for TG and HOMA-IR; and Lin et al. [24] for HbA1c. These studies differed in comparator drug, intervention dose, treatment duration, background therapy, or participant characteristics. After excluding the respective study, heterogeneity decreased markedly ($I^2 = 0\%$–30%), and the pooled effect remained non-significant for TC, HDL-C, TG, HbA1c, and HOMA-IR but remained significant for AST. For BMI, body weight, and ALT, sequential exclusion of each study did not substantially change effect direction or reduce I^2, suggesting that no single study drove the pooled estimates. Overall, the results were robust; heterogeneity was more likely multifactorial than attributable to one outlying study. Detailed estimates are shown in Table 2.

3.6 Subgroup Analysis

Subgroup analyses were performed for outcomes with substantial heterogeneity to explore potential sources related to GLP-1 RA type, mean age, BMI, treatment duration, and background

therapy. Detailed estimates are presented in Table 3. For BMI, significant reductions were observed in studies with mean age ≥50 years (MD −1.39 kg/m², 95% CI −2.15 to −0.64), treatment duration <24 weeks (MD −1.90 kg/m², 95% CI −3.58 to −0.21), and without background therapy (MD −1.61 kg/m², 95% CI −2.86 to −0.35), whereas neither the exenatide nor the liraglutide subgroup showed a significant effect. For body weight, no subgroup showed a significant effect, and heterogeneity was not explained by GLP-1 RA type, mean age, or treatment duration. For liver enzymes, exenatide was associated with significant reductions in ALT (MD −13.36 U/L, 95% CI −24.87 to −1.84) and AST (MD −6.78 U/L, 95% CI −10.53 to −3.03), whereas liraglutide showed no significant effect; ALT and AST reductions were also more marked in studies with mean age ≥50 years, treatment duration <24 weeks, and without background therapy. For lipids, TG and TC were significantly reduced in studies with background therapy (TG: MD −0.63 mmol/L, 95% CI −0.92 to −0.33; TC: MD −0.70 mmol/L, 95% CI −1.21 to −0.19), and TG was also reduced in studies with treatment duration ≥24 weeks (MD −0.38 mmol/L, 95% CI −0.69 to −0.07); no consistent subgroup effects were observed for HDL-C. For glycaemic outcomes, HbA1c was significantly reduced in studies with treatment duration ≥24 weeks (MD −0.35%, 95% CI −0.55 to −0.16), whereas HOMA-IR was significantly reduced in studies with BMI ≥29 kg/m² (MD −1.64, 95% CI −2.37 to −0.91), treatment duration ≥24 weeks (MD −1.46, 95% CI −2.12 to −0.81), and background therapy (MD −1.64, 95% CI −2.37 to −0.91). Overall, heterogeneity appeared to be multifactorial; background therapy and treatment duration were the most consistent contributors, whereas GLP-1 RA type, mean age, and BMI contributed to some outcomes. Given the small number of studies in each subgroup, these findings should be interpreted as exploratory.

3.7 Publication Bias

This study used Egger's linear regression test to quantitatively assess publication bias for each outcome, as shown in Figure 6. The analysis showed that, of the 12 outcomes evaluated, all except body weight had an Egger's test P value greater than 0.05—BMI ($P = 0.569$), ALT ($P = 0.437$), AST ($P = 0.667$), TG ($P = 0.101$), TC ($P = 0.368$), HDL-C ($P = 0.685$), LDL-C ($P = 0.797$), FPG ($P = 0.137$), 2-h PPG ($P = 0.314$), HbA1c ($P = 0.170$), and HOMA-IR ($P = 0.177$)—indicating no significant publication bias. However, the result for body weight ($P = 0.025$) suggests that some publication bias may be present for this outcome.

3.8 Certainty of Evidence

The GRADE framework was used to grade the certainty of evidence for each outcome; detailed results are shown in Table 4. The assessment rated the certainty of evidence for TC, HDL-C, LDL-C, FPG, and 2-h PPG as "High," indicating a high level of confidence in the effect estimates for these outcomes. The certainty of evidence for BMI, ALT, AST, TG, HbA1c, and HOMA-IR was rated as "Moderate," indicating reasonable confidence in their effect estimates, though it may have been downgraded because of the high between-study heterogeneity. In contrast, owing to potential publication bias ($P = 0.025$) and heterogeneity, the certainty of evidence for body weight was downgraded to "Low," suggesting a degree of uncertainty in the pooled result for body weight and indicating that further large-sample, high-quality RCTs are still needed to verify this finding.

4. Discussion

4.1 Study Overview

This study included eight RCTs involving 602 patients with T2DM and NAFLD and evaluated

the hepatic and metabolic benefits of different GLP-1 RAs. Unlike previous meta-analyses, this study strictly limited the study population to patients with T2DM and NAFLD/MASLD, excluding patients without T2DM and those with other types of diabetes, such as type 1 diabetes mellitus, thereby ensuring the clinical specificity of the effect estimates. The results showed that GLP-1 RAs significantly reduced BMI, ALT, AST, FPG, and 2-h PPG, but had no statistically significant effects on body weight, TC, TG, HDL-C, LDL-C, HbA1c, or HOMA-IR. Overall, GLP-1 RAs demonstrated relatively consistent clinical benefits in improving hepatic parenchymal injury and glycemic control; however, their effects on lipid metabolism and insulin resistance appeared to be substantially influenced by factors such as background glucose-lowering therapy, treatment duration, and patients' baseline characteristics.

4.2 Effects of Different GLP-1 RAs on Anthropometric Outcomes

GLP-1 RAs significantly reduced BMI but did not significantly reduce body weight (MD −3.28 kg, 95% CI −7.36 to 0.81, P=0.12). The BMI finding is consistent with previous meta-analyses, in which GLP-1 RAs reduced BMI compared with placebo (MD −2.07 kg/m²)[29], and both semaglutide and liraglutide produced significant BMI reductions (MD −4.26 and −1.66 kg/m², respectively) [30]. The null body-weight result may reflect the comparators used: metformin is generally weight-neutral, whereas pioglitazone can cause weight gain and may attenuate GLP-1 RA-associated weight loss [31-32]. Subgroup analyses further suggested that BMI reductions were more consistent in patients aged ≥50≥50 years, without background therapy, and with treatment duration <24 weeks, whereas neither exenatide nor liraglutide alone showed a significant effect, indicating that background therapy may be more relevant than agent type. Because the weight-reducing effects of GLP-1 RAs are dose- and time-dependent and differ among agents [33-35], the predominantly short-term exenatide or liraglutide regimens in the included studies may partly explain the non-significant body-weight effect.

4.3 Effects of Different GLP-1 RAs on Liver Enzymes

ALT and AST are commonly used biomarkers of hepatocellular injury [36]. In this meta-analysis, GLP-1 RAs significantly reduced ALT (MD −8.11 U/L, 95% CI −14.43 to −1.78) and AST (MD −4.23 U/L, 95% CI −7.30 to −1.15), suggesting that they may be associated with alleviation of hepatic injury in patients with T2DM and NAFLD. The AST estimate was robust in sensitivity analysis(after excluding Feng 2017, MD −5.67 U/L, 95% CI −8.00 to −3.33, P<0.00001), whereas ALT heterogeneity was not substantially reduced by excluding any single study, indicating multifactorial sources. Subgroup analyses suggested that ALT and AST reductions were more consistent with exenatide, mean age ≥50 years, treatment duration <24 weeks, and no background therapy, although these subgroup findings should be regarded as exploratory. These results are consistent with previous studies: semaglutide reduced ALT and AST [37], liraglutide reduced ALT [38], and liraglutide improved NASH histological scores and reduced ALT [39]. Mechanistically, GLP-1 RAs may lower liver enzymes by improving hepatic steatosis, attenuating insulin resistance, inhibiting inflammatory pathways such as NF-κB, and reducing oxidative stress [40-41].

4.4 Effects of Different GLP-1 RAs on Lipid Metabolism

Overall, GLP-1 RAs did not significantly affect TC, TG, HDL-C, or LDL-C. Sensitivity analyses showed that the heterogeneity for TC, TG, and HDL-C was mainly attributable to Yan et al. [27], Guo et al. [23], and Feng et al. [22], respectively; after excluding the corresponding study, heterogeneity decreased markedly, but the pooled effects remained non-significant (all P >0.05;

Table 2). LDL-C showed minimal heterogeneity $I^2 = 0\%$ and no significant difference. In subgroup analyses, TG and TC were significantly reduced in studies with background therapy (TG: MD −0.63 mmol/L, 95% CI −0.92 to −0.33; TC: MD −0.70 mmol/L, 95% CI −1.21 to −0.19), and TG was also reduced with treatment duration ≥24 weeks (MD −0.38 mmol/L, 95% CI −0.69 to −0.07), whereas no significant effects were observed without background therapy. These findings suggest that the lipid-modifying effects of GLP-1 RAs are limited overall and may depend on longer treatment and concomitant therapy. Previous meta-analyses have reported inconsistent lipid effects [42-47], possibly reflecting differences in comparator drugs, background lipid-lowering therapy, and lifestyle interventions.

4.5 Effects of Different GLP-1 RAs on Glycemic Control and Insulin Resistance

GLP-1 RAs significantly reduced FPG (MD −0.24 mmol/L, 95% CI −0.40 to −0.08) and 2-h PPG (MD −1.95 mmol/L, 95% CI −2.60 to −1.29), with negligible heterogeneity for both($I^2 = 0$), but did not significantly reduce HbA1c (MD −0.16%, 95% CI −0.34 to 0.02) or HOMA-IR (MD −0.37, 95% CI −0.78 to 0.04). The heterogeneity in HbA1c and HOMA-IR was mainly attributable to Liu et al. [24] and Guo et al. [23], respectively; after excluding the corresponding study, heterogeneity fell to 0%, but the pooled effects remained non-significant (Table 2). In subgroup analyses, HbA1c was significantly reduced with treatment duration ≥24 weeks (MD −0.35%, 95% CI −0.55 to −0.16), and HOMA-IR was significantly reduced in the BMI ≥29 kg/m², treatment duration ≥24 weeks, and background-therapy subgroups; however, these subgroups overlapped considerably and the findings should be regarded as exploratory (Table 3). These results indicate that the glucose-lowering effect of GLP-1 RAs is well established, whereas improvements in HbA1c and insulin resistance may require longer treatment and may be greater in patients with obesity receiving background therapy. Previous studies have reported similar glucose-lowering effects [48-49], and GLP-1 RAs act through glucose-dependent insulin secretion, glucagon suppression, delayed gastric emptying, and appetite suppression, with possible indirect improvement in insulin sensitivity [50-51]. The non-significant HbA1c and HOMA-IR findings may reflect the relatively short treatment durations, small sample sizes, and the metabolic effects of comparator drugs.

4.6 Safety of Different GLP-1 RAs in the Treatment of Patients with MASLD and T2DM

All eight RCTs reported adverse events, although reporting was inconsistent across studies. The most common GLP-1 RA-related events were mild-to-moderate gastrointestinal symptoms, including nausea, vomiting, diarrhoea, appetite suppression, and abdominal distension; these generally occurred early and were tolerated with continued treatment. Hypoglycaemia was uncommon, and no serious adverse events were attributed to GLP-1 RAs. Adverse events also occurred with comparators, including hypoglycaemia in insulin-treated patients and one case of heart failure in the pioglitazone control group [28]. The main limitations were small sample sizes, short intervention durations, mostly open-label designs, and incomplete or inconsistently defined adverse-event reporting. Large-scale, multicentre RCTs with standardised adverse-event monitoring are needed to clarify the long-term safety of GLP-1 RAs in patients with T2DM and NAFLD.

4.7 Limitations and Future Directions

This study has several limitations. Only 8 RCTs (602 participants) were included, limiting statistical power. All included studies were conducted in China, which may reduce generalisability. Most evidence concerned exenatide and liraglutide, with limited data on newer agents such as

semaglutide and dulaglutide. Comparators varied (placebo, metformin, pioglitazone, insulin) and have differing metabolic effects, introducing clinical heterogeneity.

Future research should include head-to-head RCTs comparing GLP-1 RAs; combination strategies with sodium-glucose cotransporter 2 (SGLT2) inhibitors, thyroid hormone receptor β (THR-β) agonists, and fibroblast growth factor 21 (FGF-21) analogues to target multiple pathogenic pathways; and individualised treatment based on metabolic phenotype, including insulin resistance, BMI, liver fibrosis stage, and T2DM duration.

5. Conclusions

In summary, GLP-1 RAs significantly improve BMI, ALT, AST, FPG, and 2-h PPG in patients with T2DM and NAFLD/MASLD, but have limited overall effects on body weight, lipid profiles, HbA1c, and HOMA-IR. Subgroup analyses showed that exenatide appeared to have a relative advantage in reducing liver enzyme levels, that a treatment duration of ≥24 weeks was associated with more pronounced improvements in HbA1c and HOMA-IR, and that patients with BMI ≥29 kg/m^2 appeared to derive greater benefits in terms of insulin sensitivity. Differences in efficacy may exist among different GLP-1 RAs, but these findings require confirmation in further head-to-head trials. In clinical practice, individualised GLP-1 RA treatment strategies should be developed according to patients' metabolic profiles, severity of liver disease, degree of obesity, and treatment tolerability.

Table 1 Basic characteristics of included studies.

Study	Source	Sample	Female (%)	Age (years)	Intervention	Comparison	Treatment duration (weeks)
Fan 2013	China	49/68	42.86	51.02	Exenatide 5 μg twice daily /1-4 week 10 μg twice daily/5-12 week	Metformin 0.5–1.0 g twice daily	12
Feng 2017	China	29/29	27.59	46.79	Liraglutide 0.6 mg/day/1week 1.2 mg/day/2 week 1.8 mg/day/3-24 week	Metformin 250 mg thrice daily/1 week 500 mg thrice daily/2 week 1000 mg twice daily/3-24 week	24
Guo 2020	Brazil	31/30	48.39	53.1	Liraglutide 0.6-1.8 mg/day + metformin 2000 mg/day	Metformin 2000 mg/day	26
Liu 2020	China	35/36	/	/	Exenatide 5–10 μg twice daily	Insulin glargine 0.1–0.3 U/kg/day	24
Shao 2014	China	30/30	50	43	Exenatide 5 μg twice daily/1-4 week 10 μg twice daily/5-12 week	Insulin aspart + insulin glargine	12
Tian 2018	China	52/75	40.38	58.5	Liraglutide 0.6–1.2 mg/day	Metformin 100–1500 mg/day	12
Yan 2019	China	24/24	29.17	43.1	Liraglutide 0.6-1.8 mg/day + metformin	Insulin glargine 0.2 U/kg/day + metformin	26
Zhang 2020	China	30/30	56.67	50.2	Liraglutide 0.6 mg/day/1week 1.2 mg/day/2-24 week	Pioglitazone 15 mg/day/1week 30 mg/day/2-24week	24

E, experimental group; C, control group. There were no significant differences in female ratio, average age, or disease duration between the experimental and control groups in each included study.

Table 2 Leave-one-out sensitivity analyses of identified heterogeneous sources.

Outcome	Heterogeneity source	Sensitivity analysis results			Robustness
		I^2/%	MD (95% CI)	*P* value	
AST	Feng 2017	30	-5.67 (-8.00, -3.33)	< 0.00001	Robust
TC	Yan 2019	0	-0.04 (-0.21, 0.12)	0.62	Robust
TG	Guo 2020	20	-0.04 (-0.14, 0.06)	0.41	Robust
HDL-C	Feng 2017	0	0.04 (-0.02, 0.09)	0.19	Robust

Outcome	Heterogeneity source	Sensitivity analysis results			Robustness
		I^2/%	MD (95% CI)	*P* value	
HbA1c	Lin 2020	0	-0.04 (-0.14,0.07)	0.51	Robust
HOMA-IR	Guo 2020	0	-0.02 (-0.14, 0.10)	0.78	Robust

MD, mean difference; CI, confidence interval; AST, Aspartate Aminotransferase; TC, total cholesterol; TG, Triglycerides; HDL-C, high-density lipoprotein cholesterol; HbA1c, Glycated Hemoglobin; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.

Table 3 Subgroup analyses of the high heterogeneity outcomes.

Outcome	Subject	Subgroup	Number of studies	I^2/%	MD (95% CI)	*P* value
BMI	GLP-1 RA type	exenatide group	3	95	-1.88 (-3.86, 0.10)	0.06
		liraglutide group	5	67	-1.09 (-2.25, 0.08)	0.07
	Average age	< 50 years old	3	93	-1.14 (-4.21, 1.93)	0.47
		≥ 50 years old	4	59	-1.39 (-2.15, -0.64)	0.0003
	Treatment duration	< 24 weeks	3	96	-1.90 (-3.58, -0.21)	0.03
		≥ 24 weeks	5	67	-1.05 (-2.43, 0.43)	0.14
	Background therapy	combination group	2	0	-0.71 (-2.11, 0.68)	0.32
		non-combination group	6	92	-1.61 (-2.86, -0.35)	0.01
Body weight	GLP-1 RA type	exenatide group	3	92	-4.80 (-11.64, 2.04)	0.17
		liraglutide group	5	70	-2.28 (-6.63, 2.08)	0.31
	Average age	< 50 years old	3	91	-2.83 (-12.68,7.02)	0.57
		≥ 50 years old	4	68	-3.35 (-7.17, 0.47)	0.09
	BMI	<29	5	71	-2.18 (-6.03, 1.67)	0.27
		≥29	3	83	-5.23 (-11.82, 1.37)	0.12
	Treatment duration	< 24 weeks	3	94	-4.29 (-11.21, 2.63)	0.22
		≥ 24 weeks	5	68	-2.57 (-7.23, 2.08)	0.28
ALT	GLP-1 RA type	exenatide group	3	87	-13.36 (-24.87, -1.84)	0.02
		liraglutide group	4	61	-4.44 (-10.41, 1.52)	0.14
	Average age	< 50 years old	3	92	-8.23(-25.98, 9.51)	0.36
		≥ 50 years old	3	59	-8.97 (-14.29, -3.64)	0.001
	BMI	<29	4	58	-7.54 (-12.91, -2.17)	0.006
		≥29	3	92	-9.04 (-25.19, 7.11)	0.27
	Treatment duration	< 24 weeks	3	79	-15.76 (-23.68, -7.85)	< 0.0001
		≥ 24 weeks	4	0	-2.09 (-6.06, 1.89)	0.3
	Background therapy	combination group	2	0	-2.23(-7.63, 3.18)	0.42
		non-combination group	5	83	-10.65(-18.16,-3.15)	0.005
AST	GLP-1 RA type	exenatide group	3	51	-6.78 (-10.53, -3.03)	0.0004
		liraglutide group	4	64	-2.13 (-6.29, 2.03)	0.32
	Average age	< 50 years old	3	87	-3.49 (-12.14, 5.16)	0.43
		≥ 50 years old	3	0	-5.44 (-7.63, -3.26)	< 0.00001
	BMI	<29	4	70	-3.58 (-7.18, 0.02)	0.0512
		≥29	3	68	-5.15(-11.80, 1.51)	0.13
	Treatment duration	< 24 weeks	3	34	-6.85 (-8.94, -4.75)	< 0.00001
		≥ 24 weeks	4	0	-0.53 (-3.25, 2.20)	0.7
	Background therapy	combination group	2	0	-1.70 (-6.81, 3.40)	0.51
		non-combination group	5	76	-4.87 (-8.58, -1.16)	0.01
TG	GLP-1 RA type	exenatide group	3	5	-0.03 (-0.12, 0.06)	0.53
		liraglutide group	5	75	-0.26(-0.56, 0.04)	0.09
	Average age	< 50 years old	3	48	-0.05 (-0.16, 0.05)	0.30
		≥ 50 years old	4	80	-0.12(-0.36, 0.12)	0.32
	BMI	<29	5	18	-0.03 (-0.13, 0.08)	0.62
		≥29	3	86	-0.40 (-0.94, 0.14)	0.15
	Treatment duration	< 24 weeks	3	0	-0.01 (-0.09,0.08)	0.91
		≥ 24 weeks	5	60	-0.38 (-0.69, -0.07)	0.02
	Background therapy	combination group	2	0	-0.63 (-0.92, -0.33)	< 0.0001
		non-combination group	6	0	-0.03 (-0.11, 0.04)	0.38
TC	GLP-1 RA type	exenatide group	2	0	-0.01(-0.22, 0.20)	0.93
		liraglutide group	5	42	-0.19 (-0.45, 0.07)	0.14
	Average age	< 50 years old	3	55	-0.23 (-0.61, 0.14)	0.22
		≥ 50 years old	4	10	-0.01 (-0.30,0.28)	0.96
	BMI	<29	4	0	0.00(-0.26, 0.27)	1.00

Outcome	Subject	Subgroup	Number of studies	I^2/%	MD (95% CI)	*P* value
		≥29	3	66	-0.38 (-0.93, 0.16)	0.17
	Treatment duration	< 24 weeks	3	0	0.00 (-0.19, 0.19)	0.98
		≥ 24 weeks	4	42	-0.31 (-0.63, 0.00)	0.0512
	Background therapy	combination group	2	0	-0.70 (-1.21,-0.19)	0.007
		non-combination group	5	0	-0.01 (-0.18,0.16)	0.9
HDL-C .	GLP-1 RA type	exenatide group	1	/	0.03 (-0.05, 0.11)	0.48
		liraglutide group	5	56	0.01 (-0.10,0.13)	0.83
	Average age	< 50 years old	2	47	-0.10 (-0.21, 0.01)	0.08
		≥ 50 years old	4	0	0.04(-0.02, 0.09)	0.17
	BMI	<29	4	65	0.01 (-0.09,0.10)	0.91
		≥29	2	0	0.04 (-0.12, 0.20)	0.66
	Treatment duration	< 24 weeks	2	0	0.02 (-0.03,0.08)	0.39
		≥ 24 weeks	4	65	0.03 (-0.15, 0.21)	0.78
	Background therapy	combination group	2	0	0.04(-0.12, 0.20)	0.66
		non-combination group	4	65	0.01(-0.09, 0.10)	0.91
HbA1c	GLP-1 RA type	exenatide group	3	85	-0.23 (-0.62, 0.16)	0.25
		liraglutide group	5	0	-0.06 (-0.22, 0.09)	0.43
	Average age	< 50 years old	3	0	-0.08 (-0.27, 0.11)	0.43
		≥ 50 years old	4	0	-0.02 (-0.14, 0.11)	0.79
	BMI	<29	5	74	-0.19 (-0.44, 0.06)	0.13
		≥29	3	0	-0.08 (-0.33, 0.17)	0.55
	Treatment duration	< 24 weeks	3	0	0.00 (-0.12,0.12)	0.98
		≥ 24 weeks	5	45	-0.35 (-0.55, -0.16)	0.0004
	Background therapy	combination group	2	0	-0.31 (-0.87, 0.25)	0.28
		non-combination group	6	68	-0.15 (-0.35, 0.05)	0.14
HOMA-IR	GLP-1 RA type	exenatide group	1	/	-0.01 (-0.18, 0.16)	0.91
		liraglutide group	4	85	-0.79 (-1.92, 0.34)	0.17
	Average age	< 50 years old	1	/	-0.71 (-2.60,1.18)	0.46
		≥ 50 years old	4	85	-0.36 (-0.79, 0.07)	0.10
	BMI	<29	3	0	-0.01 (-0.13, 0.11)	0.82
		≥29	2	8	-1.64 (-2.37, -0.91)	< 0.0001
	Treatment duration	< 24 weeks	2	0	-0.01 (-0.13, 0.11)	0.87
		≥ 24 weeks	3	12	-1.46 (-2.12, -0.81)	< 0.0001
	Background therapy	combination group	2	8	-1.64 (-2.37, -0.91)	< 0.0001
		non-combination group	3	0	-0.01 (-0.13, 0.11)	0.82

MD, mean difference; CI, confidence interval; BMI, Body Mass Index; ALT, Alanine Aminotransferase.

Table 4 Certainty of evidence.

Outcome	Riskofbias	Inconsistency	Indirectness	Imprecision	Publicationbias	MD (95%CI)	Certaintyofevidence
BMI	None	Very serious	None	None	None	-1.43 (-2.52, -0.34)	Moderate
Body weight	None	Very serious	None	None	Suspected	-3.28 (-7.36, 0.81）	Low
ALT	None	Very serious	None	None	None	-8.11 (-14.43, -1.78)	Moderate
AST	None	Serious	None	None	None	-4.23 (-7.30, -1.15)	Moderate
TG	None	Serious	None	None	None	-0.14 (-0.29, 0.02)	Moderate
TC	None	None	None	None	None	-0.08 (-0.24, 0.08)	High
HDL-C	None	None	None	None	None	0.01 (-0.04, 0.06)	High
LDL-C	None	None	None	None	None	-0.00 (-0.12,0.11)	High
FPG	None	None	None	None	None	-0.24 (-0.40, -0.08)	High
2-h PPG	None	None	None	None	None	-1.95 (-2.60, -1.29)	High
HbA1C	None	Serious	None	None	None	-0.16 (-0.34, 0.02)	Moderate
HOMA-IR	None	Very serious	None	None	None	-0.37 (-0.78,0.04)	Moderate

MD, mean difference; CI, confidence interval; BMI, Body Mass Index; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; TC, Total Cholesterol; TG, Triglyceride; HDL-C, High-density Lipoprotein Cholesterol; LDL-C, Low-density Lipoprotein Cholesterol; FPG, Fasting Plasma Glucose; 2-h PPG, 2-h Postprandial Plasma Glucose; HbA1c, Hemoglobin A1c; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance.

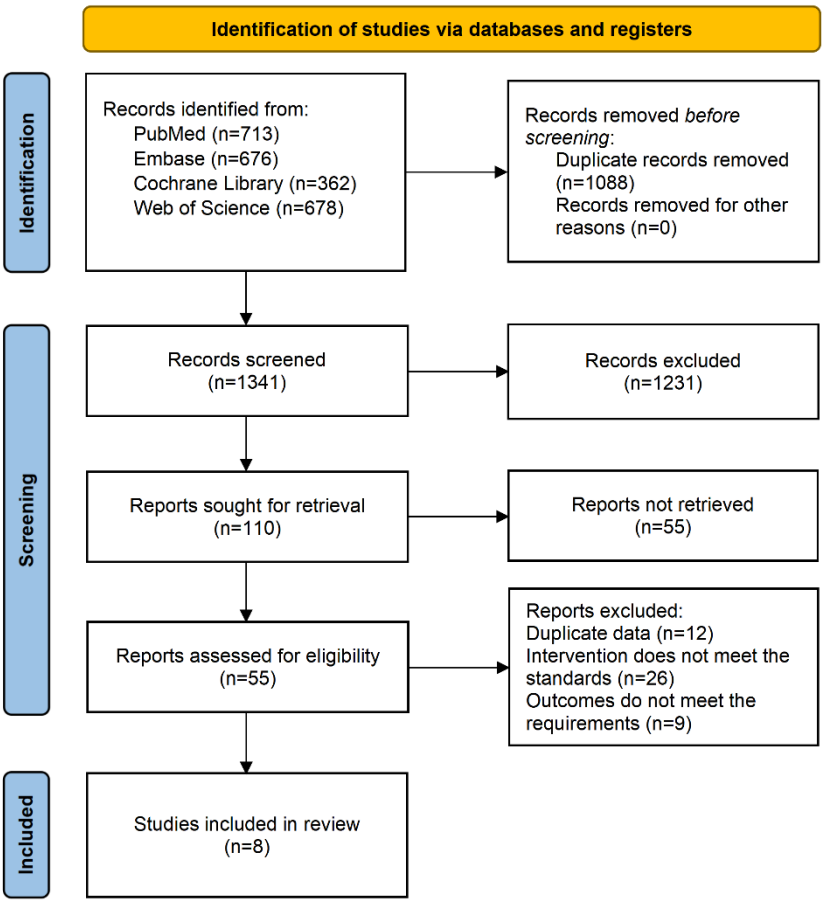
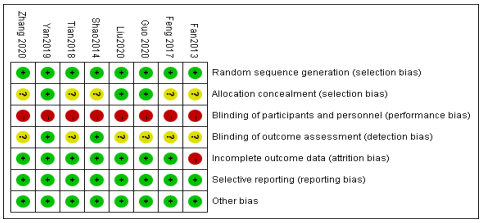


Figure 1. PRISMA flow diagram of the literature search and study selection process.

This figure illustrates the flow of information through the different phases of a systematic review. Records were identified from PubMed, Embase, Cochrane Library, and Web of Science databases. After removing duplicates and screening by title/abstract and full text, 8 randomized controlled trials were finally included in the meta-analysis.

(1) Risk of bias summary



(2) Risk of bias graph

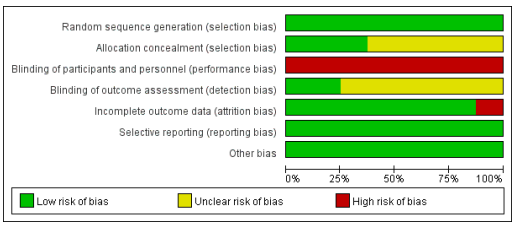
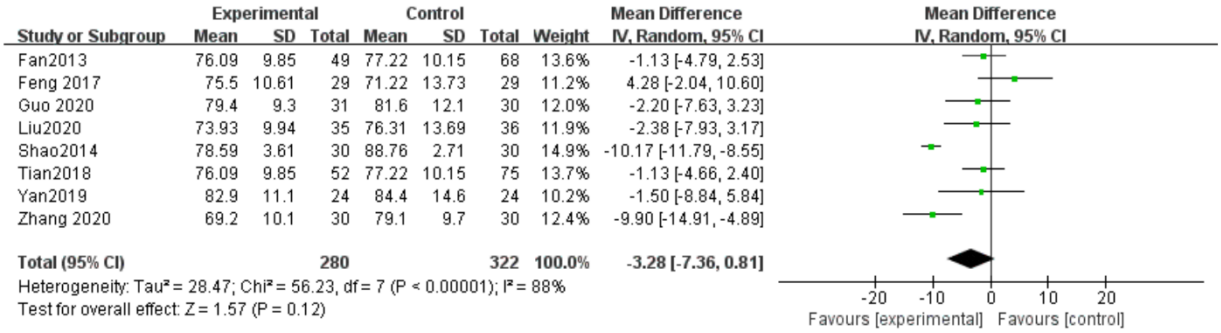


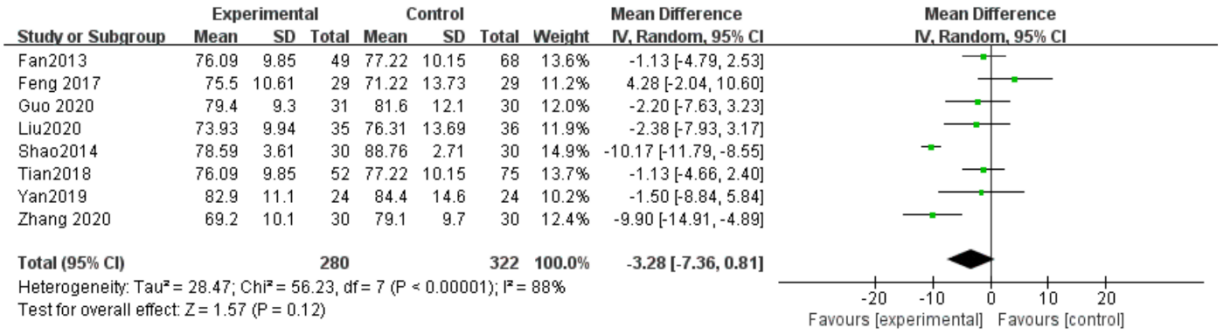
Figure 2. Risk of bias assessment of the included studies.

Quality assessment was performed using the Cochrane Risk of Bias tool. (A) Risk of bias summary: judgments about each risk of bias item for each included study (green indicates low risk, yellow indicates unclear risk, and red indicates high risk). (B) Risk of bias graph: review authors' judgments about each risk of bias item presented as percentages across all included studies.

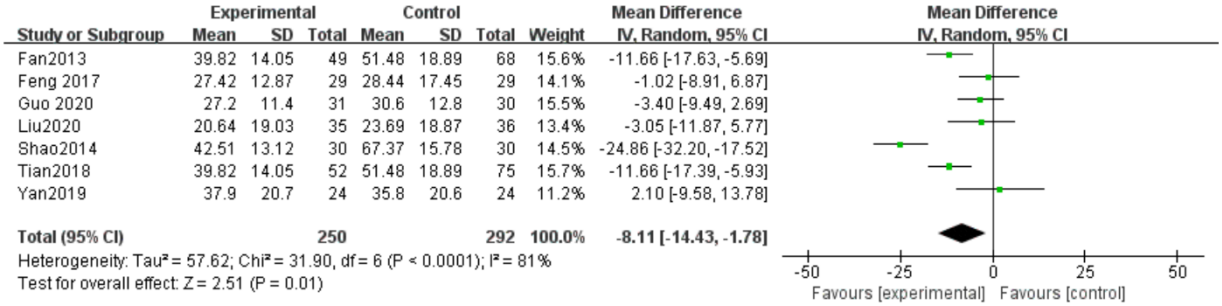
(A) BMI



(B) Body weight



(C) ALT



(D) AST

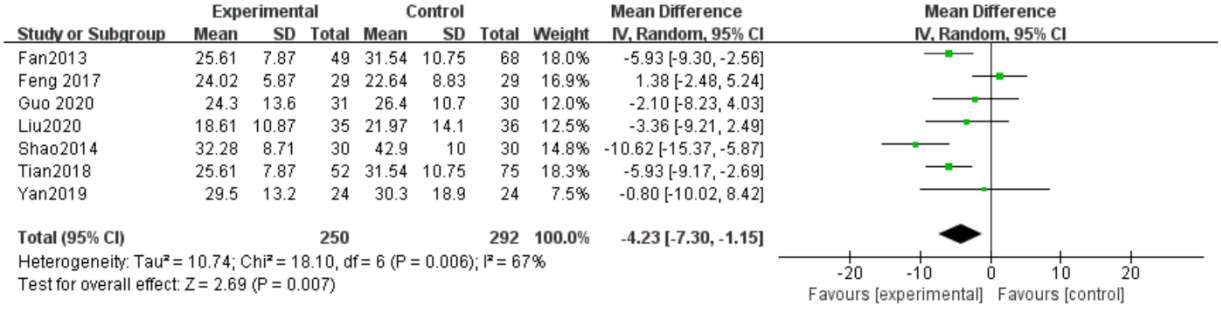
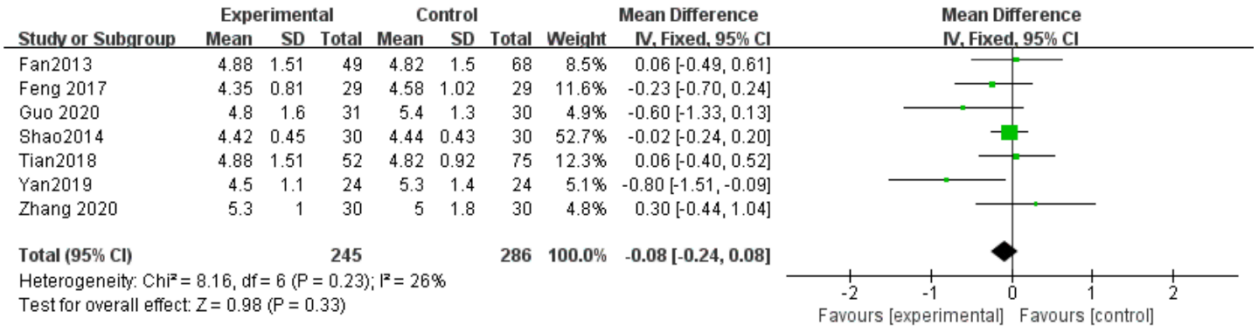


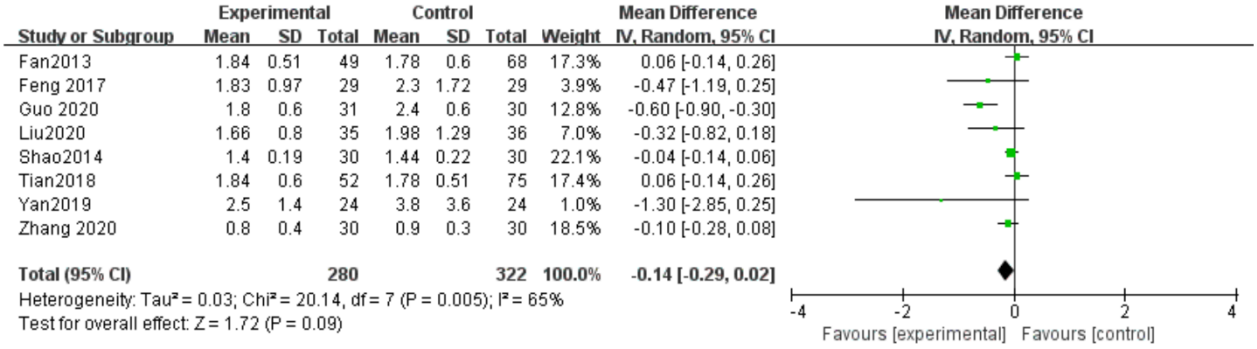
Figure 3. Forest Plots of the Effects of Different GLP-1 RAs on Anthropometric Measures and Liver Enzymes in Patients with T2DM and MASLD.

Forest plots detailing the mean difference and 95% confidence intervals (CIs) for (A) Body mass index (BMI), (B) Body weight ,(C) Alanine aminotransferase (ALT), and (D) Aspartate aminotransferase (AST). The size of the squares represents the weight of each study, and the black diamond represents the pooled overall effect.

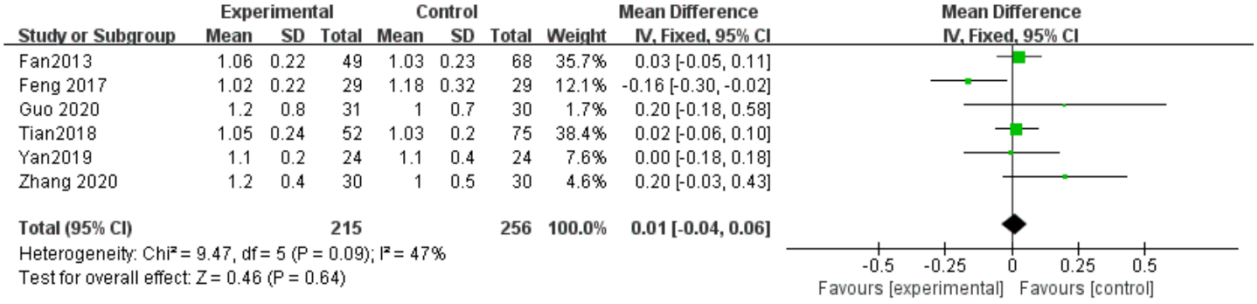
(A) TC



(B) TG



(C) HDL-C



(D) LDL-C

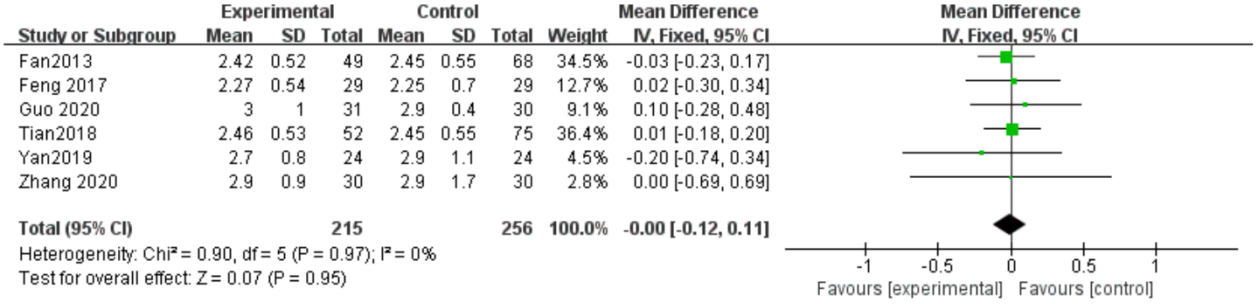
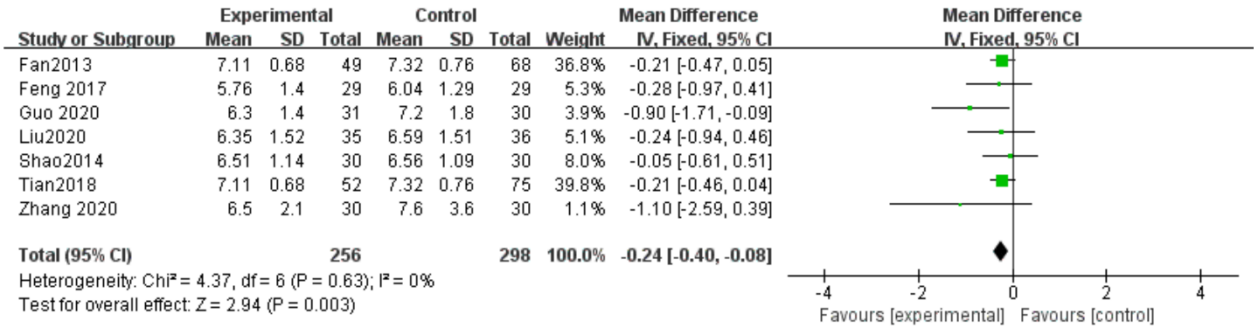


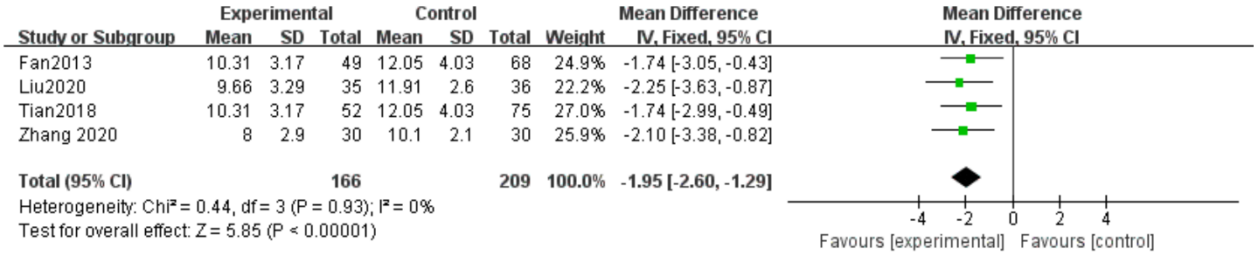
Figure 4. Forest plots of the effects of Different GLP-1 RAs on lipid profiles in patients with T2DM and MASLD.

Forest plots detailing the mean difference and 95% CIs for (A) Total cholesterol (TC), (B) Triglycerides (TG), (C) High-density lipoprotein cholesterol (HDL-C), and (D) Low-density lipoprotein cholesterol (LDL-C).

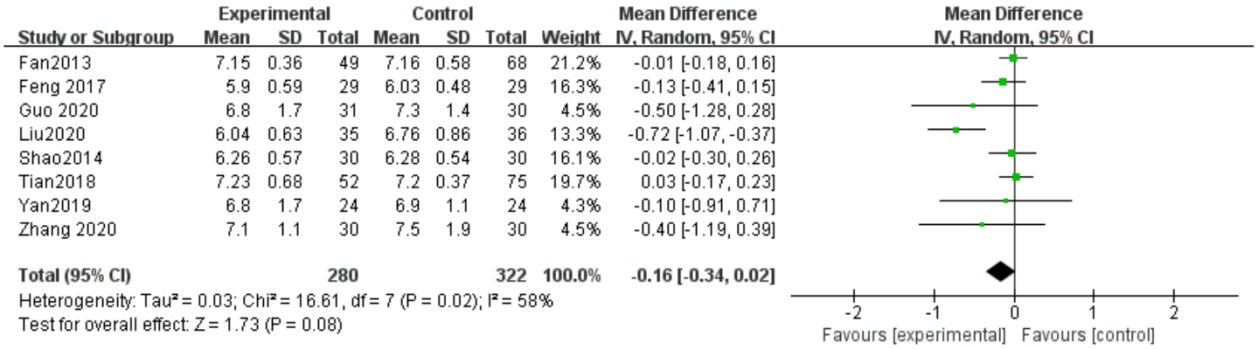
(A) FPG



(B) 2-h PPG



(C) HbA1c



(D) HOMA-IR

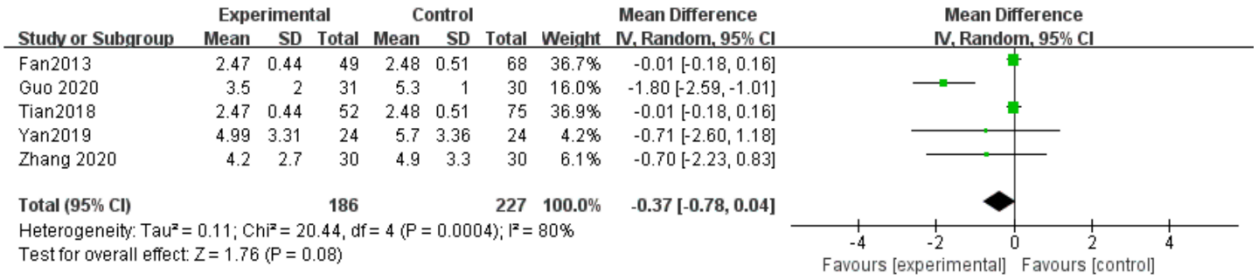


Figure 5. Forest plots of the effects of Different GLP-1 RAs on glycemic control and insulin resistance.

Forest plots detailing the mean difference and 95% CIs for (A) Fasting plasma glucose (FPG), (B) 2-h Postprandial Plasma Glucose (2-h PPG), (C) Glycated hemoglobin (HbA1c), and (D) Homeostatic Model Assessment for Insulin Resistance (HOMA-IR).

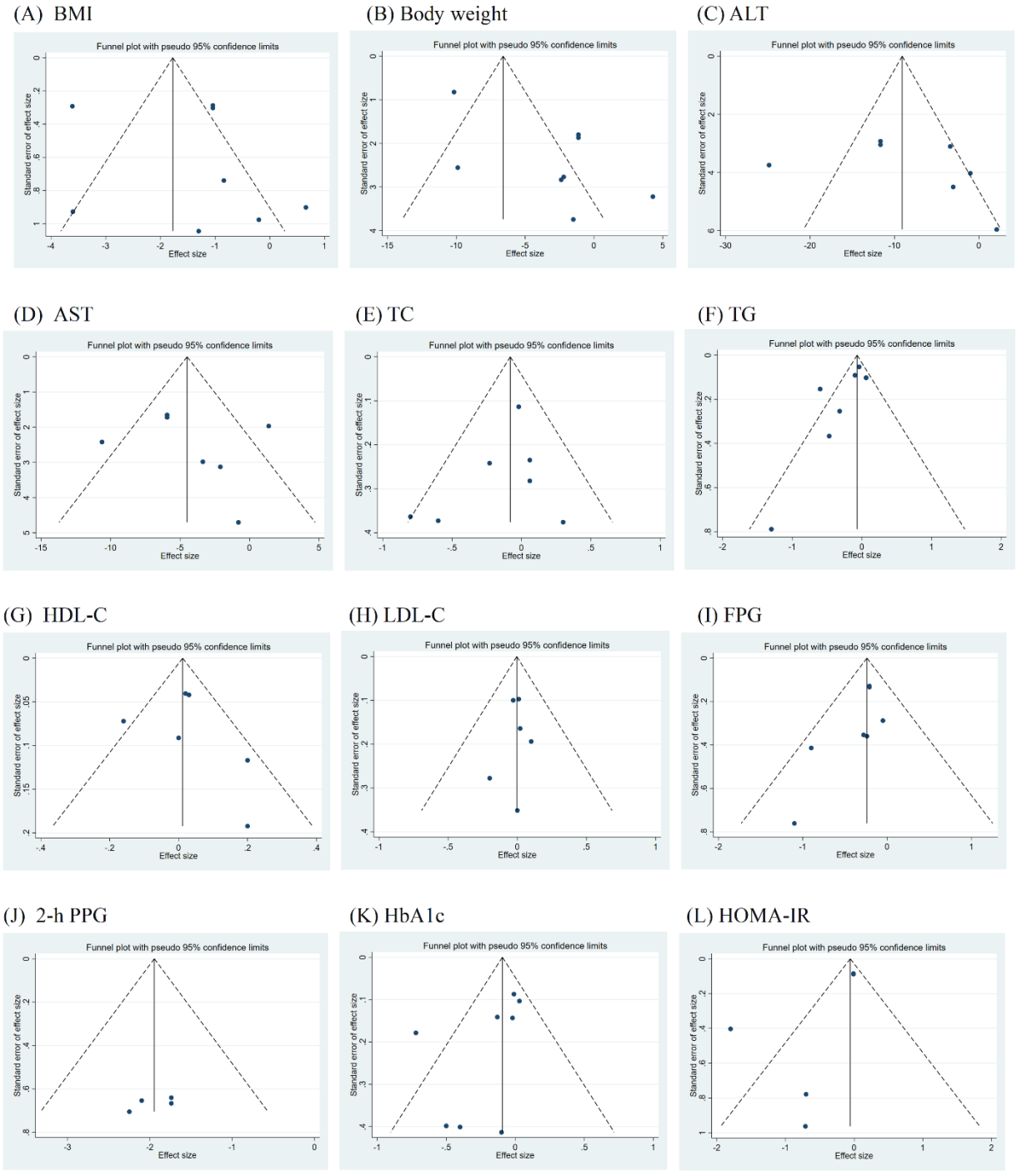


Figure 6. Funnel plots for assessing potential publication bias across clinical outcomes.

Funnel plots of standard error by effect size for various outcomes including Anthropometric Measures, liver enzymes, lipid and glycemic profiles. Each dot represents an individual study included in the meta-analysis, and the dashed lines indicate the pseudo 95% confidence limits. Symmetry of the scatter plot suggests a low probability of publication bias.

Availability of data and material

The original contributions from this study are included in the article/supplementary material. For

further inquiries, please contact the corresponding author.

Conflict of interest

The authors declare that they have no known financial interests or personal conflicts that could have affected the research work.

Authorship contribution statement

YJ: Writing-original draft, Methodology, Data curation, Formal analysis. YS: Writing-original draft, Data curation, Formal analysis. JC: Writing-review and editing, Conceptualization, Supervision.

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Appendix

Supplementary Table S1 The search strategies, filters, and limits for all databases.

Database	Search strategy	Search dates	Filters	Limits
PubMed	(("Non-alcoholic Fatty Liver Disease"[MeSH] OR "NAFLD"[tiab] OR "NASH"[tiab] OR "MAFLD"[tiab] OR "metabolic dysfunction-associated steatotic liver disease"[tiab] OR "non-alcoholic steatohepatitis"[tiab] OR ((("fatty liver"[tiab] OR "hepatic steatosis"[tiab]) AND ("non-alcoholic"[tiab] OR "nonalcoholic"[tiab])))) AND ("Diabetes Mellitus, Type 2"[MeSH] OR "Type 2 Diabetes"[tiab] OR "T2DM"[tiab] OR "NIDDM"[tiab] OR ("diabet*"[tiab] AND ("type 2"[tiab] OR "type II"[tiab] OR "adult onset"[tiab] OR "non-insulin dependent"[tiab])))) AND ("Glucagon-Like Peptide-1 Receptor Agonists"[MeSH] OR "GLP-1 Receptor Agonist"[tiab] OR "GLP-1RA"[tiab] OR "Glucagon-Like Peptide 1"[tiab] OR "Incretin Mimetics"[tiab] OR "Liraglutide"[tiab] OR "Victoza"[tiab] OR "Semaglutide"[tiab] OR "Ozempic"[tiab] OR "Rybelsus"[tiab] OR "Wegovy"[tiab] OR "Dulaglutide"[tiab] OR "Trulicity"[tiab] OR "Exenatide"[tiab] OR "Byetta"[tiab] OR "Bydureon"[tiab] OR "Lixisenatide"[tiab] OR "Adlyxin"[tiab] OR "Tirzepatide"[tiab] OR "Mounjaro"[tiab] OR "Retatrutide"[tiab] OR "Orforglipron"[tiab] OR "Albiglutide"[tiab] OR "Efinopegdutide"[tiab])	August 15, 2026	None	None
Embase	('non-alcoholic fatty liver'/exp OR 'nonalcoholic fatty liver':ti,ab,kw OR 'non-alcoholic fatty liver':ti,ab,kw OR 'non-alcoholic steatohepatitis':ti,ab,kw OR 'nonalcoholic steatohepatitis':ti,ab,kw OR nafld:ti,ab,kw OR nash:ti,ab,kw OR mafld:ti,ab,kw OR 'metabolic dysfunction-associated steatotic liver disease':ti,ab,kw OR 'fatty liver':ti,ab,kw OR 'hepatic steatosis':ti,ab,kw) AND ('diabetes mellitus, type 2'/exp OR 'non insulin dependent diabetes mellitus'/exp OR 'type 2 diabetes':ti,ab,kw OR 'type 2 diabetes mellitus':ti,ab,kw OR t2dm:ti,ab,kw OR niddm:ti,ab,kw OR 'non-insulin dependent diabetes':ti,ab,kw OR ((diabet* NEAR/2 ('type 2' OR 'type II')):ti,ab,kw)) AND ('glucagon-like peptide-1 receptor agonist'/exp OR 'glucagon like peptide 1 receptor agonist'/exp OR 'glp-1 receptor agonist':ti,ab,kw OR 'glp-1ra':ti,ab,kw OR 'glp-1':ti,ab,kw OR 'glucagon-like peptide 1':ti,ab,kw OR 'incretin mimetic*':ti,ab,kw OR liraglutide:ti,ab,kw OR victoza:ti,ab,kw OR saxenda:ti,ab,kw OR semaglutide:ti,ab,kw OR ozempic:ti,ab,kw OR rybelsus:ti,ab,kw OR wegovy:ti,ab,kw OR dulaglutide:ti,ab,kw OR trulicity:ti,ab,kw OR exenatide:ti,ab,kw OR byetta:ti,ab,kw OR bydureon:ti,ab,kw OR lixisenatide:ti,ab,kw OR adlyxin:ti,ab,kw OR lyxumia:ti,ab,kw OR albiglutide:ti,ab,kw OR tanzeum:ti,ab,kw OR eperzan:ti,ab,kw OR tirzepatide:ti,ab,kw OR mounjaro:ti,ab,kw OR retatrutide:ti,ab,kw OR orforglipron:ti,ab,kw OR cagrilintide:ti,ab,kw OR efinopegdutide:ti,ab,kw) AND ('randomized controlled trial'/exp OR 'crossover procedure'/de OR 'double-blind procedure'/de OR 'single-blind procedure'/de OR random*:ti,ab,kw OR factorial*:ti,ab,kw OR crossover*:ti,ab,kw OR placebo*:ti,ab,kw OR ((singl* OR doubl* OR trebl* OR tripl*) NEAR/3 (blind* OR mask*)):ti,ab,kw OR 'clinical trial':ti,ab,kw OR 'controlled trial':ti,ab,kw)	August 15, 2026	None	None
Cochrane Library	(Non alcoholic Fatty Liver Disease OR Nonalcoholic Fatty Liver OR Nonalcoholic Fatty Livers OR NAFLD OR Nonalcoholic Fatty Liver Disease OR Nonalcoholic Steatohepatitis OR Nonalcoholic Steatohepatitides OR Metabolic associated fatty liver disease OR MAFLD OR Metabolic dysfunction associated fatty liver disease OR Fatty Liver OR Steatohepatitis OR Steatohepatitides OR Steatosis of Liver OR Visceral Steatosis OR Visceral Steatoses OR Liver Steatosis OR Liver Steatoses OR NASH OR MASLD):ti,ab,kw AND (Type 2 Diabetes OR Type 2 Diabetes Mellitus OR T2DM OR NIDDM OR Non insulin dependent diabetes OR Adult onset diabetes OR Diabetes Mellitus Type 2):ti,ab,kw AND (GLP-1 receptor agonist OR GLP-1RA OR GLP-1 OR Glucagon-like peptide 1 OR Incretin mimetic OR Liraglutide OR Victoza OR Saxenda OR Semaglutide OR Ozempic OR Rybelsus OR Wegovy OR Dulaglutide OR Trulicity OR Exenatide OR Byetta OR Bydureon OR Lixisenatide OR Adlyxin OR Lyxumia OR Albiglutide OR Tanzeum OR Eperzan OR Tirzepatide OR Mounjaro OR Retatrutide OR Orforglipron OR Cagrilintide OR Efinopegdutide):ti,ab,kw	August 15, 2026	None	None
Web of Science	(TS=("Non-alcoholic Fatty Liver Disease" OR NAFLD OR NASH OR MAFLD OR "metabolic dysfunction-associated steatotic liver disease" OR ((("fatty liver" OR "hepatic steatosis") AND ("non-alcoholic" OR nonalcoholic)))) AND TS=("Type 2 Diabetes" OR T2DM OR NIDDM OR (diabet* AND ("type 2" OR "type II")))) AND TS=("GLP-1 Receptor Agonist" OR GLP-1RA OR Liraglutide OR Semaglutide OR Ozempic OR Dulaglutide OR Trulicity OR Exenatide OR Lixisenatide OR Tirzepatide OR Mounjaro OR Retatrutide OR Orforglipron OR Albiglutide OR Efinopegdutide OR "Glucagon-Like Peptide-1")	August 15, 20262026	None	None