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ORIGINAL RESEARCH

A Systematic Review and Meta-Analysis of Efficacy and Safety of Liraglutide in Patients with Type 2 Diabetes Mellitus

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Background: Obesity plays a pivotal and modifiable role in the development and progression of type 2 diabetes mellitus (T2DM). Clinicians increasingly use lower doses of liraglutide (1.2 mg and 1.8 mg) to achieve clinically meaningful weight loss while maintaining effective glycemic control in people with T2DM and obesity. In this meta-analysis, we compared the efficacy and safety of liraglutide 1.2 mg and 1.8 mg in this population.

Methods: We systematically searched PubMed, the Cochrane Central Register of Controlled Trials, LENS, ClinicalTrials.gov, and the Virtual Health Library (VHL) for randomized controlled trials published in English up to 30 September 2024. We included trials with 24–52 weeks of treatment that evaluated liraglutide at doses of 1.2 mg or 1.8 mg against placebo or glucose-lowering therapies (GLTs). Comparators included insulin, sulfonylureas, dipeptidyl peptidase-4 inhibitors (DPP-4i), sodium–glucose cotransporter-2 inhibitors (SGLT2i), other glucagon-like peptide-1 receptor agonists (GLP-1RAs), and oral antidiabetic drugs (OADs). We assessed changes in body weight and HbA1c as efficacy outcomes and evaluated the occurrence of nausea and vomiting as safety outcomes. Two reviewers independently extracted data and assessed study quality using PRISMA guidelines and the Cochrane Risk of Bias 2 tool.

Results: We included 25 RCTs comprising 10,593 participants. Liraglutide 1.2 mg (8 studies, 3,455 participants) produced a mean weight reduction of −1.24 kg versus GLTs, −0.75 kg versus placebo, and −2.46 kg versus OADs. Liraglutide 1.8 mg (22 studies, 8,259 participants) achieved significantly greater weight loss of −2.30 kg versus GLTs, −1.93 kg versus placebo, and −2.81 kg versus OADs. When compared with oral semaglutide, exenatide, dulaglutide, lixisenatide, and albiglutide, liraglutide showed comparable efficacy. For glycemic control, liraglutide 1.2 mg reduced HbA1c by −0.24% versus OADs, while liraglutide 1.8 mg reduced HbA1c by −0.26% versus GLTs. Liraglutide 1.2 mg showed a numerically lower incidence of nausea and similar rates of vomiting compared with other GLP-1RAs.

Conclusion: Liraglutide 1.2 mg and 1.8 mg doses improve weight and glycemic outcomes with a favourable safety profile, supporting its role as an effective therapeutic option for comprehensive management of T2DM with comorbid obesity.

Keywords: liraglutide, type 2 diabetes mellitus, GLP-1 receptor agonist, glycemic control, systematic review, meta-analysis

Introduction

Effective weight management plays a critical role in people with type 2 diabetes mellitus (T2DM) due to its significant impact on disease progression and associated comorbidities. Obesity is a well-established, modifiable risk factor contributing to both the onset and progression of T2DM. Clinical evidence suggests that weight reduction in the range of 5% to 10% can substantially improve metabolic outcomes in people with T2DM. This is particularly relevant in regions like South Asia, where the prevalence of T2DM and central obesity is remarkably high.[1]

Recognizing the strong association between obesity and T2DM, leading international guidelines—including those from the American Diabetes Association (ADA), the European Society of Cardiology (ESC), and the Lancet Obesity

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Commission - emphasize the importance of integrating obesity management into the overall treatment strategy for T2DM. Among the pharmacological interventions available, liraglutide, a glucagon-like peptide-1 receptor agonist (GLP-1RA), has gained considerable attention for its dual benefit in improving glycemic control and promoting weight loss.[2–4]

Studies indicate that lower doses of liraglutide can effectively facilitate weight reduction while maintaining glycemic benefits. The use of lower doses may provide an effective and more tolerable option, particularly in people with obesity-related T2DM characterized by visceral and ectopic fat accumulation, which may not always be reflected in overall body mass index (BMI). Additionally, lower doses have been associated with a reduced incidence of adverse effects, offering a favorable balance between weight loss and modest glucose-lowering effects, potentially improving patient adherence and tolerability.[5] Furthermore, utilizing lower doses and their biosimilars may help maintain liraglutide as a reasonably low-cost option by reducing medication expenses while preserving its clinical benefits, making it a more viable choice for long-term treatment.[6]

South Asians tend to develop T2DM at lower BMI thresholds and have relatively lower muscle mass and higher visceral adiposity compared to Western populations. Given these physiological differences, lower-dose GLP-1RAs therapy such as liraglutide 1.2 mg may be sufficient to achieve meaningful glycemic and weight-related benefits in this population. The potential for greater responsiveness to GLP-1RAs among Asians has been suggested in prior studies, but specific data on muscle mass preservation and optimal dosing in South Asians remain limited.[7] Moreover, the recent availability of generic formulations has made liraglutide more accessible, further supporting its use as a cost-effective option in low- and middle-income settings.[8,9] While several systematic reviews and meta-analyses have evaluated the efficacy of liraglutide, this study uniquely examines dose-specific effects of both liraglutide 1.2 mg and 1.8 mg in patients with T2DM, offering targeted insights relevant to clinical practice in the South Asian population.

This systematic review and meta-analysis aimed to evaluate the efficacy and safety of liraglutide 1.2 mg and liraglutide 1.8 mg compared with other therapeutic options in people with T2DM and comorbid obesity, providing insights into its role in effective weight management and overall treatment outcomes. This meta-analysis addresses a key evidence gap in existing literature, as no previous synthesis has comprehensively compared the dual effects of glycemic control and weight reduction between liraglutide 1.2 mg and 1.8 mg in patients with T2DM, thereby underscoring the novelty and clinical relevance of the present study.

Materials and Methods

Protocol and Reporting Guidelines

The meta-analysis was registered with PROSPERO under the registration ID CRD42024552647. A PRISMA flowchart [Figure 1] is used to illustrate the flow of information through different stages of the review process. By adhering to the PRISMA guidelines, we followed a structured approach in study selection, data extraction, quality assessment, data synthesis, and bias assessment, promoting transparency and robust reporting.[10]

Selection Criteria

Randomized clinical trials (RCTs) with the intervention arm involving Liraglutide at doses of 1.2 mg and / or 1.8 mg, compared against other Glucagon-Like Peptide-1 Receptor Agonists (GLP-1Ras), Insulins, and oral anti-diabetic drugs (OADs) [sodium-glucose co-transporter-2 (SGLT2) inhibitors, dipeptidyl peptidase-4 (DPP4) inhibitors, sulphonylureas (SU), or placebo were included in the analysis. The patient profile of interest included individuals with T2DM with or without atherosclerotic cardiovascular disease (ASCVD), obesity, non-alcoholic fatty liver disease (NAFLD), or chronic kidney disease (CKD), with T2DM being a mandatory criterion. The endpoints included change in weight, hemoglobin A1c (HbA1c), fasting plasma glucose (FPG), postprandial glucose (PPG). The duration of assessment included in the studies ranged from 24 to 52 weeks.

Studies were excluded if Liraglutide was used at doses other than 1.2 mg and 1.8 mg. Additionally, patients without T2DM but with ASCVD, Obesity, NAFLD, or CKD were also excluded from the review. Observational studies were excluded from the analysis to maintain the focus on RCTs meeting the specified criteria.

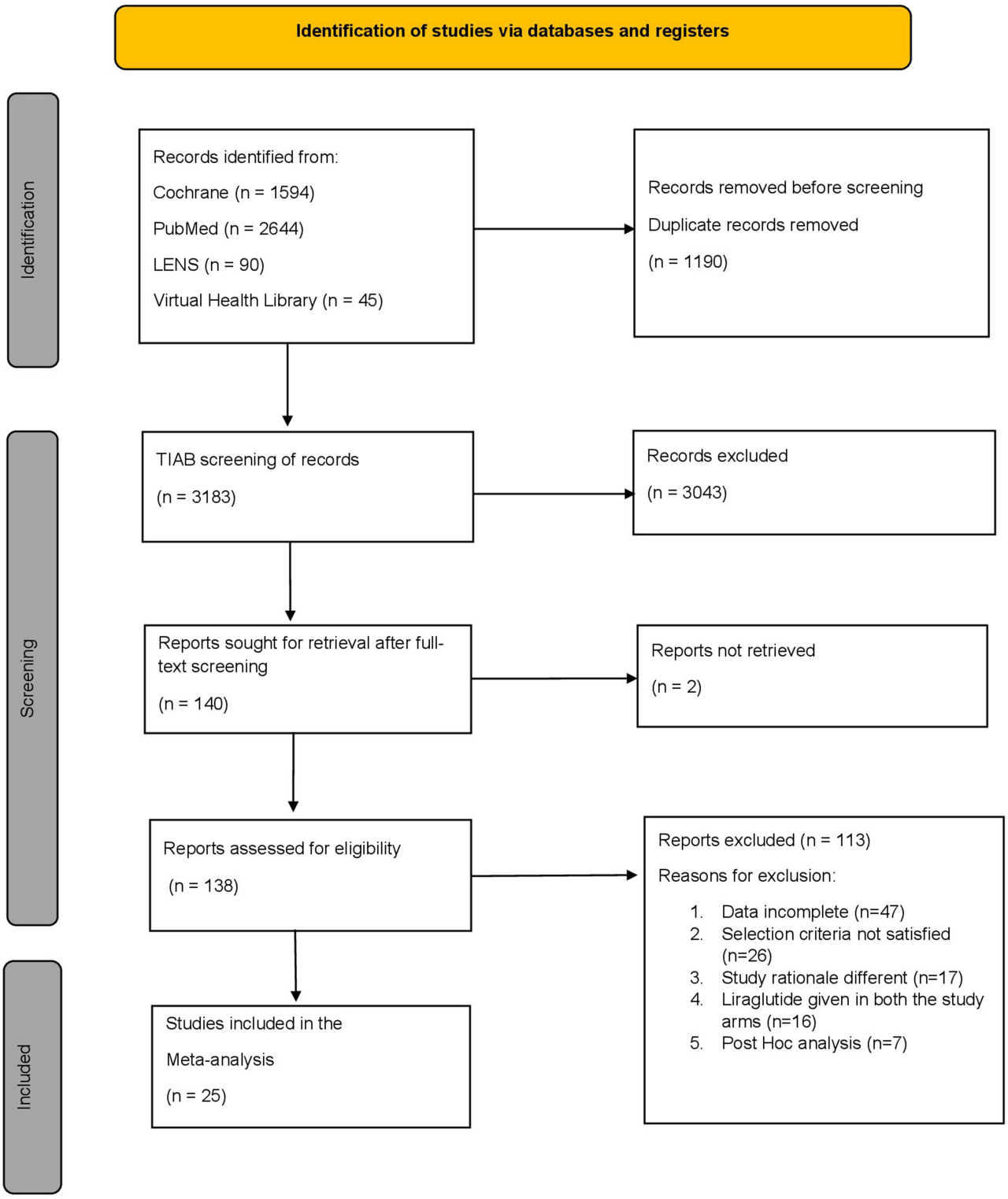

Figure 1 Identification of studies via databases and registers.

Search Strategy

The study conducted a comprehensive search for relevant literature from its inception until September 30, 2024, utilizing databases which included PubMed, Cochrane Central Register, LENS, and VHL. Although major databases such as Embase, Scopus, and Web of Science were not included due to institutional access limitations, the search was

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supplemented with extensive secondary strategies to minimize the risk of missing eligible studies. The search terms employed included "Liraglutide," "Victoza," "Saxenda," "Randomized controlled trial," "Controlled clinical trial," "Type 2 diabetes," "Obesity," and other pertinent keywords, with Boolean operators (AND, OR, NOT) used to further refine the search (Supplementary File 1). The extracted data included details such as the first author's name, Country, number of participants, comparator, age, gender, duration of diabetes, name of the journal, year of publication, treatment groups, dosage, and sample size. Manual searches of the reference list of included studies were also conducted to identify additional relevant literature.

Bias Assessment

The risk of bias for all randomized controlled trials was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool, focusing on the primary outcome of interest. This tool evaluates bias across five domains: the randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of the reported result. Two independent reviewers conducted the assessments, and any discrepancies were resolved through discussion to ensure consistency. The structured approach of the RoB 2 tool ensured a transparent and systematic evaluation of study quality. The detailed risk-of-bias assessments are presented in the ROB table (Figure 2A) and summarized in the ROB summary figure (Figure 2B). Most studies demonstrated low risk of bias across key domains, particularly in measurement of outcomes, missing outcome data, and randomization. Some concerns were mainly noted for deviations from intended interventions and selection of the reported result, with only a very small proportion rated as high risk.

Data Extraction and Assessment

The initial step involved a comprehensive literature search conducted by S.J., followed by independent manual screening of all potentially relevant studies by A.K.D. The full texts of eligible records were subsequently retrieved, and a thorough evaluation was performed by S.J. and S.K. based on pre-defined inclusion and exclusion criteria. Data extraction was carried out by S.J., A.K.D., and S.Y.C. using a standardized Microsoft Excel spreadsheet to systematically organize patient demographics and treatment characteristics for both intervention and comparator arms. Data quality validation was subsequently performed by S.K., ensuring accuracy and completeness of the extracted dataset. Any discrepancies identified during this process were resolved through collaborative review prior to formal analysis. The validated dataset was then entered into RevMan software version 8.1.1 by S.J., A.K.D., and S.Y.C. for comprehensive statistical analysis. Reasons for the exclusion of studies were documented in alignment with the PRISMA guidelines. In cases where trials randomized patients to different doses of the active intervention, only data pertaining to the specified doses of Liraglutide (1.2 mg and 1.8 mg) were utilized. A comprehensive account of the data abstraction process is available in the Appendix in the Supplement.

Outcome Analysis

The outcome measures were analyzed separately for both the doses of Liraglutide to assess the overall effect of each dose on the reduction of weight in (kgs) and glycemic parameters. The study quantitatively compared efficacy parameters like weight reduction (kg), reduction in HbA1c levels (mmol/mol and %), FPG levels (mg/dL), PPG levels (mg/dL) and safety parameters like GI adverse events such as nausea and vomiting from baseline to the end of the study between the Liraglutide intervention arm and the comparator arm [glucose lowering therapies (GLTs), OADs, insulin, other GLP-1RAs and placebo wherever applicable]. Heterogeneity was evaluated using the I^2 statistic and Cochran's Q test. I^2 values were interpreted following Cochrane thresholds (0–25%: low; 26–50%: moderate; 51–75%: substantial; >75%: considerable heterogeneity). For analyses with substantial or considerable heterogeneity (I^2 >10%), a random-effects model was applied to account for between-study variability.

Statistical Analysis

After identification of studies pertaining to the efficacy and safety of Liraglutide, data was extracted from the selected studies and represented qualitatively followed by quantitative analysis using forest plots. The Meta-analysis was performed using RevMan software 8.1.1 online version from the Cochrane website. With due consideration to

7A

Intention-to-treat	Unique ID	Study ID	Experimental	Comparator	Outcome	Weight	D1	D2	D3	D4	D5	Overall
	1	NCT02461589	Semaglutide	Liraglutide, Placebo	HbA1c reduction (primary), Body	1	+	+	+	+	+	+
	2	ChiCTR200003509 1	Liraglutide + Metformin	Placebo + Metformin	Reduction in IHCL (primary); SAT, VAT,	1	+	!	+	+	!	!
	3	NCT01617434	Liraglutide 1.8 mg/day + basal insulin ±	Placebo + basal insulin ± metformin	Change in HbA1c (primary), body	1	+	+	+	+	!	+
	4	NCT00518882	Liraglutide 1.8 mg QD	Exenatide 10 µg BID	HbA1c (primary); FPG, PPG, body weight, AEs	1	+	!	+	+	+	!
	5	NCT01029886	Liraglutide 1.8 mg QD	Exenatide 2 mg QW	HbA1c (primary); weight, FPG, lipids,	1	!	!	+	+	!	!
	6	NA	Semaglutide 1.0 mg once weekly	Liraglutide 1.2 mg once daily	HbA1c reduction (primary), body	1	+	!	+	+	!	!
	7	NCT01296412	Injectable strategy: Liraglutide 1.2–1.8	Oral strategy: Sitagliptin ± Glimepiride	HbA1c (primary); FPG, body weight, lipids,	1	+	!	+	+	!	!
	8	NCT01624259	Dulaglutide 1.5 mg once weekly	Liraglutide 1.8 mg once daily	Change in HbA1c (primary), FPG,	1	+	!	+	+	!	!
	9	NCT00294723	Liraglutide 1.2 mg or 1.8 mg once daily	Glimepiride 8 mg once daily	HbA1c (primary); FPG, weight, AEs, PROs	1	+	+	+	+	!	+
	10	NCT00294723	Liraglutide 1.2 mg or 1.8 mg QD	Glimepiride 8 mg QD	HbA1c, weight, FPG, AEs over 2 years	1	+	!	+	+	!	!
	11	NCT01272232	Liraglutide 3.0 mg once daily	Placebo + lifestyle	% weight loss, ≥5% weight loss, ≥10%	1	+	+	+	+	+	+
	12	NCT00318461	Liraglutide 0.6, 1.2, or 1.8 mg QD + metformin	Glimepiride 4 mg QD + metformin; Placebo +	HbA1c (primary); weight, FPG,	1	+	+	+	+	!	+
	13	NCT01128894	Liraglutide 1.8 mg once daily	Albiglutide 30→50 mg once weekly	HbA1c (primary); FPG, weight, AEs,	1	+	!	+	+	!	!
	14	NCT02863419	Oral semaglutide 14 mg once daily	Subcutaneous liraglutide 1.8 mg once daily,	Change in HbA1c (primary); body	1	+	+	+	+	+	+
	15	NCT02043054	Liraglutide 1.8 mg subcutaneously once	Sitagliptin 100 mg orally once daily	Primary: Change in PEDSR (diastolic	1	+	!	+	+	+	!
	16	NCT00333151	Liraglutide 1.2 mg or 1.8 mg + metformin +	Placebo + metformin + rosiglitazone	Primary: A1C reduction; Secondary:	1	+	+	+	+	!	!
	17	EudraCT 2012-001941-42	Liraglutide 1.8 mg SC once daily + multiple	Placebo + multiple daily insulin	HbA1c (primary); weight, insulin dose,	1	+	+	+	+	!	!
	18	NA	Liraglutide 1.2 mg once daily + oral	Saxagliptin 5 mg once daily or Vildagliptin 50 mg	HbA1c, FBG, P2BG, body weight, BMI,	1	-	!	+	+	+	-
	19	NCT01761318	Liraglutide 1.8 mg once daily subcutaneously	Placebo once daily subcutaneously	NA	1	!	+	+	+	!	!
	20	NCT02147925	Liraglutide 1.8 mg/day SC + metformin	Sitagliptin 100 mg/day PO or insulin glargine SC +	Primary: MRI-PDFF (IHL); Secondary: VAT,	1	+	+	+	+	!	!
	21	NCT01973231	Liraglutide 1.8 mg/day + metformin	Lixisenatide 20 mg/day + metformin	HbA1c (primary); FPG, SMPG, weight,	1	+	+	+	+	!	!
	22	NCT01919489	Liraglutide 1.8 mg/day (up-titrated) + usual	Insulin glargine (50–80% of inpatient dose) + usual	HbA1c (primary); weight, hypoglycemia,	1	+	+	+	+	!	!
	23	NCT02660047	Liraglutide 1.8 mg/day + standard care	Placebo + standard care	Primary: Cardiac function (reported	1	+	+	+	+	!	!
	24	ChiCTR180001986 4	Liraglutide 1.2 mg/day SC	Dapagliflozin 10 mg/day PO	HbA1c (primary); FPG, 2hPG, PPGE, SBP,	1	!	!	!	+	!	!
	25	NCT01392898	Liraglutide 1.8 mg/day + continued insulin	Continued standard insulin therapy	Body weight (primary); HbA1c,	1	+	+	+	+	+	+

Low risk
Some concerns
High risk

D1 Randomisation process
D2 Deviations from the intended
D3 Missing outcome data
D4 Measurement of the outcome
D5 Selection of the reported result

7B

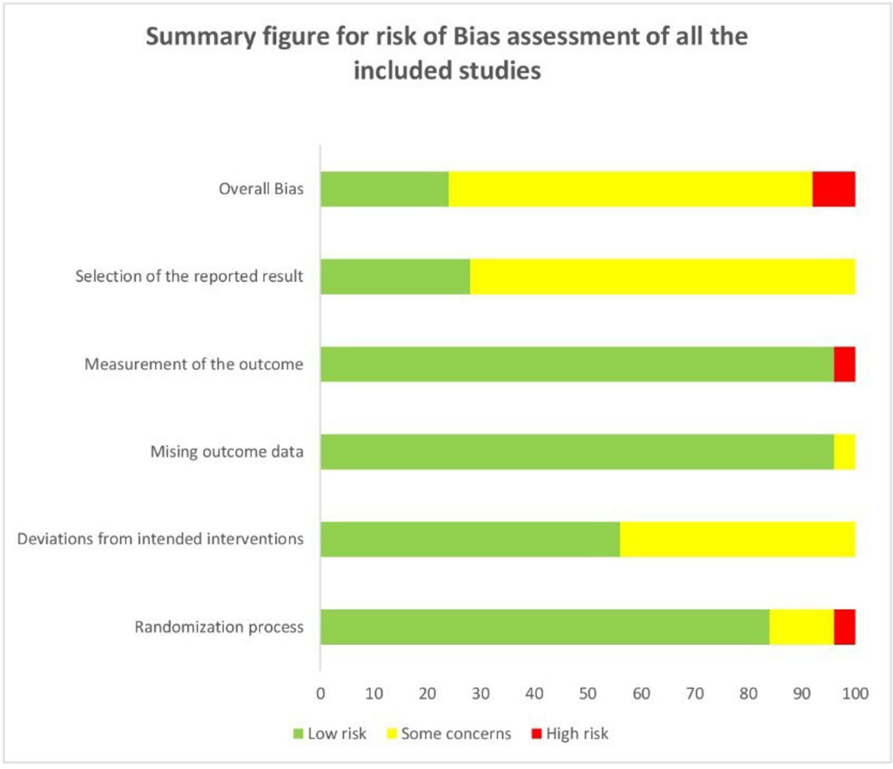

Figure 2 Liraglutide MA ROB. (**A**) Risk of Bias assessment Table of all the included studies. (**B**) Summary figure for risk of Bias assessment of all the included studies. **Notes**: Risk of bias assessment of all included studies evaluating liraglutide, conducted using the Cochrane Risk of Bias tool. Domains assessed include random sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, incomplete outcome data, selective reporting, and other sources of bias. Green (+): low risk; yellow (?): unclear risk; red (−): high risk.

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heterogeneity amongst the studies included, data was pooled using the Mantel–Haenszel (MH) equation utilizing random effects model. Publication bias was assessed visually using funnel plots, as formal statistical tests (eg, Egger's or Begg's) were not feasible due to the limited number of studies included. A leave-one-out (LOO) sensitivity analysis was performed for the primary outcome of weight reduction to evaluate the robustness of the pooled estimates. Subgroup analyses were performed by Liraglutide dose (1.2 mg and 1.8 mg) and comparator type (GLTs, OADs, insulin, other GLP-1RAs, and placebo) to evaluate consistency of efficacy and safety outcomes. Forest plots mentioning the Mean ± SD of the point estimates along with 95% CI of the factors responsible for weight reduction and glycemic efficacy of Liraglutide (1.2 mg and 1.8 mg) was plotted while Odds ratio (OR) was used to represent the safety parameters. A p value of < 0.05 was statistically significant for the analysis.

Results

Literature Selection

We identified 4373 studies from the initial database search. After removing duplicates (n=1190), 3183 studies were assessed. Following the title and abstract (TiAb) screening of these studies based on predefined criteria, 138 studies were selected for full text review from which 25 studies were identified as being eligible for inclusion. The entire process is detailed in the PRISMA flow diagram (Figure 1).

Included Studies

The studies were analyzed according to the inclusion criteria. The characteristics of the included studies for the meta-analysis are summarized in Table 1.[10–33]

Efficacy Outcomes

Among the comparator groups included in the study, OADs; SU (3 studies), DPP4i (3 studies), SGLT2i (1 study), GLTs included all the OADs along with insulin (3 studies), other GLP1-RAs (8 studies) and placebo (7 studies). Weight reduction, glycemic parameters, and gastrointestinal adverse drug reactions (GI ADRs) of liraglutide (1.2 mg and 1.8 mg) were compared within context of the endpoints and follow-up duration criteria. For the efficacy analyses, the line of significance was set at 0.

Weight Reduction

The weight reduction efficacy of Liraglutide 1.2 mg and 1.8 mg was assessed across the comparator groups. When comparing Liraglutide 1.2 mg to GLTs, OADs, and Placebo, the mean difference in weight reductions were −1.24 kg ([95% CI: −2.93, 0.46], $p = 0.15$), −2.46 kg ([95% CI: −4.32, −0.60], $p = 0.010$), and −0.75 kg ([95% CI: −1.44, −0.06], $p=0.03$) respectively. (Figure 3A–C) In subgroup analysis, Liraglutide 1.2 mg showed significant weight reduction compared to DPP-4 inhibitors (–5.10 kg [95% CI: –5.32, –4.88]; $p < 0.00001$). In contrast, when compared with Other GLP-1 RA (semaglutide), liraglutide 1.2mg resulted in significant less weight reduction (+3.80 kg [95% CI: 3.08, 4.52]; $p < 0.00001$). Additionally, there was a comparable reduction with sulfonylureas (–2.27 kg [95% CI: –4.88, 0.35]; $p = 0.09$) and SGLT2 inhibitors (–0.19 kg [95% CI: –0.51, 0.13]; $p = 0.24$) did not reach statistical significance (Figure 3D).

Liraglutide 1.8 mg exhibited significant reduction in weight when compared to other GLTs, with a mean difference of −2.30 kg ([95% CI: −3.51, −1.09], $p = 0.0002$) (Figure 3E), and against the placebo arm, with a mean difference of –1.93 kg ([95% CI: −2.23, −1.63], $p < 0.00001$). (Figure 3F) Additionally, Liraglutide 1.8 mg demonstrated statistically significant reductions in weight compared to OADs, with a mean difference of −2.81 kg ([95% CI: −4.53, −1.09], $p = 0.001$) (Figure 3G). Liraglutide 1.8 mg sub-group showed significant weight reduction versus insulin (mean difference: –5.37 kg [95% CI: –5.67, –5.07]; $p < 0.00001$), DPP-4 inhibitors (mean difference: –2.95 kg [95% CI: –4.36, –1.55]; $p < 0.0001$), and sulfonylureas (mean difference: –2.57 kg [95% CI: –4.99, –0.14]; $p = 0.04$). Comparison with other GLP-1 RAs demonstrated no statistically significant difference (mean difference: –0.48 kg [95% CI: –1.78, 0.83]; $p = 0.47$). (Figure 3H) Heterogeneity was observed across the studies, ranging from [I^2 = 0% to100%], and tau^2 values ranged from

Table 1 The Characteristics of the Included Studies for the Meta-Analysis

Sr No	Study	Name	Trial Design	Patient Population	Duration (Weeks)	Sample Size		Efficacy Endpoints				Safety Endpoints	
						Liraglutide	Comparator ARM	HbA1c	FPG	PPG	Weight	Nausea	Vomiting
1	A 26-{Week} {Randomized} {Controlled} {Trial} of {Semaglutide} {Once} {Daily} {Versus} {Liraglutide} and {Placebo} in {Patients} {With} {Type} 2 {Diabetes} {Suboptimally} {Controlled} on {Diet} and {Exercise} {With} or {Without} {Metformin}.	Desouza et al 2018[12]	Randomized, double blind trial	T2DM	26	1.2mg, 64; 1.8mg, 65	Semaglutide - 0.05mg, 64; Semaglutide - 0.1mg, 63; Semaglutide - 0.2mg, 65; Semaglutide - 0.3mg, 63; Semaglutide -flexible dose, 64; Placebo, 129	NO	NO	NO	NO	YES	YES
2	Liraglutide or insulin glargine treatments improves hepatic fat in obese patients with type 2 diabetes and nonalcoholic fatty liver disease in twenty-six weeks: {A} randomized placebo-controlled trial	Guo et al 2020[13]	Single-center, prospective, randomized placebo-controlled study	T2DM + NAFLD under metformin monotherapy >3 months	26	1.8mg, 32	Glargine 10IU, 32; Placebo, 32	YES	NO	NO	YES	NO	NO
3	The Effect of Liraglutide Versus Placebo When Added to Basal Insulin Analogues With or Without Metformin in Subjects With Type 2 Diabetes	Ahmann et al 2015[19]	Randomised controlled trial, double-blind, parallel-group study	T2DM	26	1.8mg, 226	Glargine 10IU, 225	NO	NO	NO	YES	NO	NO
4	Effect of Liraglutide or Exenatide Added to a Background Treatment of Metformin, Sulphonylurea or a Combination of Both on Glycaemic Control in Subjects With Type 2 Diabetes **(LEAD-6)**	Buse et al 2009[20]	Randomised, Open-label, Active comparator, Parallel-group, Multinational trial	T2DM + OAD dosage for >3 months	26 Weeks,; 52 weeks extension	1.8 mg, 232	Exenatide 10mcg, 232	NO	NO	NO	NO	YES	YES
5	Exenatide once weekly versus liraglutide once daily in patients with type 2 diabetes: a randomised, open-label study. **(DURATION 6)**	Buse et al 2013[21]	Open-label, randomised, parallel-group study	T2DM +OADs	26	1.8mg, 450	Exenatide 2mg, 461	YES	YES	NO	YES	YES	YES
6	Efficacy and Safety of Semaglutide 1.0 mg Once-weekly Versus Liraglutide 1.2 mg Once-daily as add-on to 1–3 Oral Anti-diabetic Drugs (OADs) in Subjects With Type 2 Diabetes **(SUSTAIN - 10)**	Capehorn et al 2020[22]	Randomized, multicentre, multinational, active-controlled, parallel-group, open-label, two-armed Phase 3b trial	T2DM	30	1.2mg, 287	Semaglutide 1.0mg, 290	YES	YES	NO	YES	YES	YES

(*Continued*)

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Table 1 (Continued).

Sr No	Study	Name	Trial Design	Patient Population	Duration (Weeks)	Sample Size		Efficacy Endpoints				Safety Endpoints	
						Liraglutide	Comparator ARM	HbA1c	FPG	PPG	Weight	Nausea	Vomiting
7	Efficacy and safety over 26 weeks of an oral treatment strategy including sitagliptin compared with an injectable treatment strategy with liraglutide in patients with type 2 diabetes mellitus inadequately controlled on metformin: a randomised clinical trial.	Charbonnel et al 2013[23]	Multinational, randomised, open label, active-controlled, parallel-arm study	T2DM + metformin >12 weeks	26	1.8mg, 327	Sitagliptin 100mg/day, 326	YES	NO	NO	YES	NO	NO
8	A Randomized, Open-Label, Parallel-Arm Study Comparing the Effect of Once-Weekly Dulaglutide With Once-Daily Liraglutide in Patients With Type 2 Diabetes **(AWARD-6)**	Dungan et al 2014[24]	Randomized, Parallel Assignment, Open label	T2DM	26	1.8mg, 300	Dulaglutide 1.2mg, 299	YES	YES	YES	YES	YES	YES
9	To Evaluate the Effect of Liraglutide Versus Glimepiride (Amaryl®) on Haemoglobin A1c **(LEAD 3)**	Garber et al 2009[25]	Randomized, double blind, parallel assignment trial	T2DM	52	1.2mg, 251; 1.8mg, 247	Glimipride 8mg, 248	YES	NO	NO	YES	NO	NO
10	Liraglutide, a once-daily human glucagon-like peptide 1 analogue, provides sustained improvements in glycaemic control and weight for 2 years as monotherapy compared with glimepiride in patients with type 2 diabetes	Garber et al 2011[26]	Active-control, parallel-group trial with an initial 52-week randomized, double-blind, double-dummy treat ment period followed by a 52-week open-label extension	T2DM +OADs	104	1.2mg, 251; 1.8mg, 247	Glimipride 8mg, 248	NO	YES	NO	NO	NO	NO
11	Effect of Liraglutide on Body Weight in Overweight or Obese Subjects With Type 2 Diabetes: A 56 Week Randomised, Double-blind, Placebo-controlled, Three Armed Parallel Group, Multi-centre, Multinational Trial With a 12 Week Observational Follow-up Period	Melanie et al 2015[1]	Randomized, double-blind, placebo-controlled, parallel-group trial	T2DM + metformin/ thiazolidinedione/ sulphonylurea	56	1.8mg, 211	Placebo, 212	YES	NO	NO	NO	NO	NO

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12	Liraglutide Effect and Action in Diabetes (LEAD-2): Effect on Glycaemic Control After Once Daily Administration of Liraglutide in Combination With Metformin Versus Metformin Monotherapy Versus Metformin and Glimepiride Combination Therapy in Subjects With Type 2 Diabetes **(LEAD 2)**	Nauck et al 2009[27]	Randomised, double-blinded, followed by an 18-month open-label extension.	T2DM + OAD dosage for >3 months	26, followed by 18-month extension. Total 2 years.	1.2mg, 242; 1.8mg, 242	Glimepiride 4mg, 242; Placebo, 121	YES	YES	NO	YES	NO	NO
13	A Randomized, Open-Label, Parallel-Group, Multicenter Study to Determine the Efficacy and Safety of Albiglutide as Compared With Liraglutide in Subjects With Type 2 Diabetes Mellitus - **(HARMONY 7)**	Pratley et al 2014[28]	Randomized, Open-Label, Parallel-Group, Multicenter Study	T2DM inadequately controlled with metformin	32	1.8mg, 419	Albiglutide 30mg 422	YES	YES	NO	YES	YES	YES
14	55-{OR}: {Oral} {Semaglutide} vs {Liraglutide} and {Placebo} in {T2D}: **(PIONEER 4)**	Richard et al 2019[29]	Randomised, double-blind, double-dummy, active-controlled, and placebo-controlled phase 3a trial	T2DM + metformin with or without an SGLT2 inhibitor	52	1.8mg, 284	Semaglutide 14 mg, 285; Placebo, 142	NO	YES	NO	YES	YES	YES
15	Impact of Liraglutide on Cardiac Function and Structure in Young Adults With Type 2 Diabetes: an Open-label, Randomised Active-comparator Trial	Webb et al 2020[30]	Single-centre, prospective, randomized, open-label, blinded endpoint active comparator trial.	T2DM	26	1.8mg, 38	Sitagliptin 100mg, 38	YES	NO	NO	YES	NO	NO
16	Efficacy and safety of the human glucagon-like peptide-1 analog liraglutide in combination with metformin and thiazolidinedione in patients with type 2 diabetes **(LEAD-4)**	Zinman et al 2009[31]	Randomized, double blind, parallel assignment trial	T2DM + OAD dosage for >3 months	26	1.2mg, 100; 1.8mg, 100	Placebo, 100	YES	NO	NO	YES	NO	NO
17	Liraglutide in people treated for type 2 diabetes with multiple daily insulin injections: randomised clinical trial **(MDI Liraglutide trial)**	Lind et al 2015[32]	Randomised, double blind, placebo controlled trial	T2DM + multiple daily insulin injections.	24	1.8mg, 58	Placebo, 55	YES	YES	NO	YES	NO	NO

(*Continued*)

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Table 1 (Continued).

Sr No	Study	Name	Trial Design	Patient Population	Duration (Weeks)	Sample Size		Efficacy Endpoints				Safety Endpoints	
						Liraglutide	Comparator ARM	HbA1c	FPG	PPG	Weight	Nausea	Vomiting
18	Efficacy and safety comparison of add-on therapy with liraglutide, saxagliptin and vildagliptin, all in combination with current conventional oral hypoglycemic agents therapy in poorly controlled {Chinese} type 2 diabetes.	C J Li et al 2014[33]	Randomized, open-label, parallel clinical trial	T2DM + metformin/ sulphonylurea for >6 months or <10 year	24	1.2mg, 68	Saxagliptin 5mg, 68; Vildagliptin 50mg, 67	YES	YES	NO	YES	NO	NO
19	Magnetic Resonance Assessment of Victoza Efficacy in the Regression of Cardiovascular Dysfunction In Type 2 Diabetes Mellitus	Bizino et al 2019[14]	Randomized, parallel assigned, quadruple masked	T2DM + metformin, and/or sulfonylurea derivative (SUD) and/or insulin.	26	1.8mg, 22	Placebo, 28	YES	NO	NO	YES	NO	NO
20	Efficacy of Liraglutide vs Sitagliptin vs Insulin Glargine Per Day on Liver Fat When Combined With Metformin in T2DM Subjects With Non-alcoholic Fatty-liver Disease	Jinhua et al 2019[15]	Open-label, active-controlled, parallel-group, multicenter trial	T2DM + NAFLD treated with metformin >3 months	26	1.8 mg, 24	Glargine 10IU, 24 Sitagliptin 100mg, 27	YES	YES	YES	YES	NO	NO
21	Efficacy and Safety of Liraglutide Versus Lixisenatide as add-on to Metformin in Subjects With Type 2 Diabetes	Nauck et al 2016[16]	Randomized, two armed, open-label, active-controlled, multicenter, multinational, parallel-group trial	T2DM + metformin >90 days	26	1.8mg, 202	Lixisenatide 20mcg, 202	NO	NO	NO	YES	YES	YES
22	A Randomized Controlled Trial Comparing the Safety and Efficacy of Liraglutide Versus Glargine Insulin for the Management of Patients With Type 2 Diabetes After Hospital Discharge	Pasquel et al 2021[17]	Randomized, Parallel Assignment, Open label	T2DM + OAD treatment	26	1.8mg 136	Glargine 10 IU, 137	NO	NO	NO	YES	NO	NO
23	A double-blind, placebo-controlled, randomised trial to assess the effect of liraglutide on ectopic fat accumulation in {South} {Asian} type 2 diabetes patients	Van Eyk et al 2019[11]	Prospective, randomised, double-blind, clinical trial.	T2DM	26	1.8 mg, 22	Placebo, 25	YES	NO	NO	YES	NO	NO

24	Efficacy and {Safety} of {Dapagliflozin} versus {Liraglutide} in {Patients} with {Overweight} or {Obesity} and {Type} 2 {Diabetes} {Mellitus}: {A} {Randomised} {Controlled} {Clinical} {Trial} in {Tianjin}, {China}.	Zhaohu et al 2022[18]	Single-centre, randomised, parallel, controlled clinical observational study	T2DM + metformin (≥1500 mg/d) alone or in combination with premixed insulin for ≥8 weeks	24	1.2 mg, 180	Dapagliflozin 10mg, 180	YES	YES	NO	YES	NO	NO
25	Liraglutide reverses pronounced insulin-associated weight gain, improves glycaemic control and decreases insulin dose in patients with type 2 diabetes: a 26 week, randomised clinical trial **(ELEGANT)**	Helena et al 2014[34]	Open-label, randomised, controlled clinical trial	T2D + insulin therapy (≥3and≤16 months), with concomitant documented weight gain of ≥4% body weight since initiation of insulin treatment	26	1.8 mg, 26	Insulin 6 IU, 24	YES	NO	NO	YES	NO	NO

Abbreviations: T2DM, Type 2 Diabetes Mellitus; NAFLD, Non-Alcoholic Fatty Liver Disease; OAD, Oral Anti-Diabetic Drug; HbA1c, Glycated Hemoglobin; FPG, Fasting Plasma Glucose; PPG, Postprandial Glucose; IU, International Units; mg, Milligrams; mcg, Micrograms; SGLT2, Sodium-Glucose Cotransporter-2; LEAD, Liraglutide Effect and Action in Diabetes; AWARD, Assessment of Weekly AdministRation of Dulaglutide; PIONEER, Peptide INsulIN secretion in diabetes Evaluation trial with oral semaglutide; MDI, Multiple Daily Insulin; ELEGANT, Efficacy of Liraglutide in reversinG wEight gain ANd improving glycaemic conTrol; SUD, Sulfonylurea Derivative.

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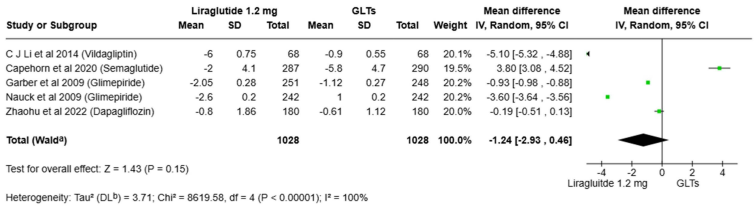

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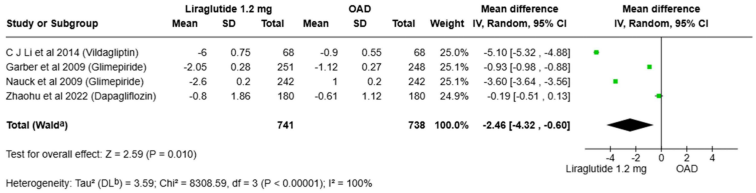

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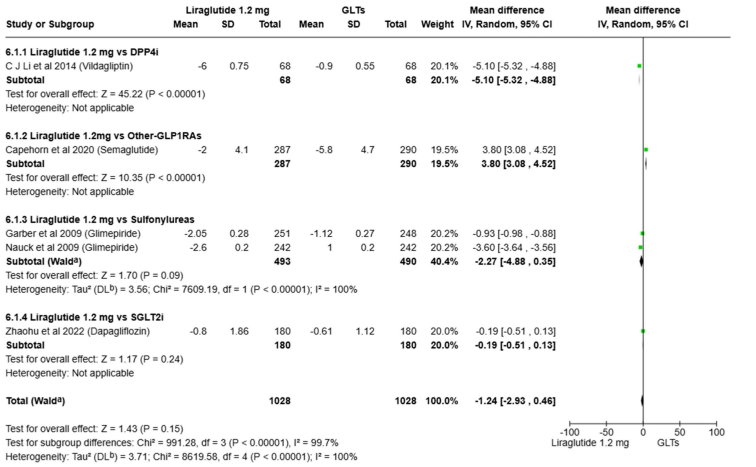

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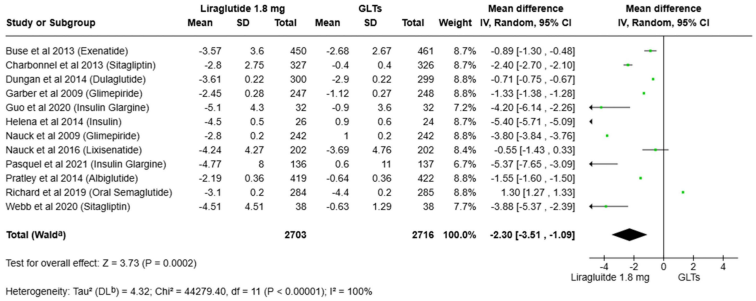

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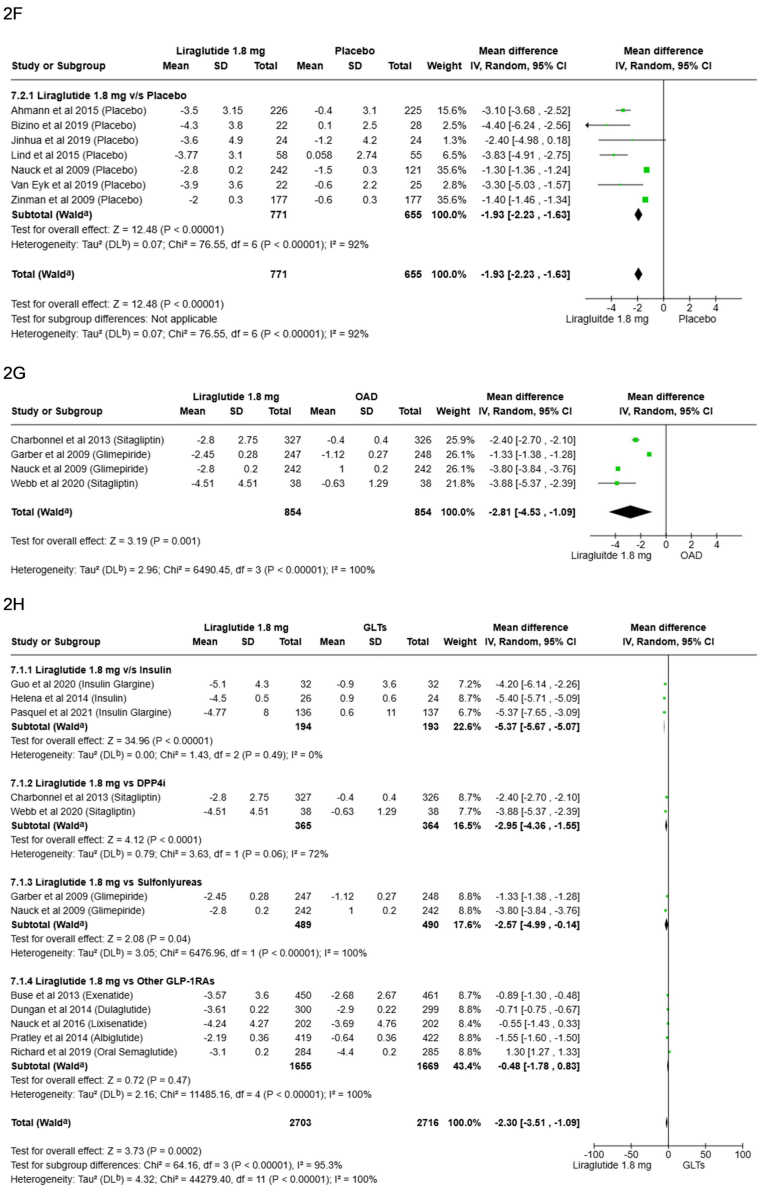

Figure 3 Liraglutide MA Forest Plots – Weight reduction. (**A**) Liraglutide 1.2 mg v/s GLTs, (**B**) Liraglutide 1.2 mg v/s OADs, (**C**) Liraglutide 1.2 mg v/s Placebo, (**D**) Liraglutide 1.2 mg (Subgroup analysis), (**E**) Liraglutide 1.8 mg v/s GLTs, (**F**) Liraglutide 1.8 mg v/s Placebo, (**G**) Liraglutide 1.8 mg v/s OAD, (**H**) Liraglutide 1.8mg (Subgroup analysis).
Note: Mean difference shown with 95% CI using random-effects model.
Abbreviations: GLTs, Glucose-lowering therapies (includes all OADs, insulin, and other GLP-1RAs); OADs, Oral antidiabetic drugs (SU, DPP4i, SGLT2i, Metformin); SU, Sulfonylureas; DPP4i, Dipeptidyl Peptidase-4 inhibitors; SGLT2i, Sodium–glucose cotransporter-2 inhibitors; GLP-1RA, Glucagon-like peptide-1 receptor agonist.

[0 to 3.7]. The reduction in weight observed with Liraglutide against all comparators in consideration is explained using a forest plot.

Glycemic Control

Liraglutide 1.2 mg demonstrated a comparable mean reduction in HbA1c levels relative to GLTs (mean difference: −0.09%, [95% CI: −0.28, 0.11], $p = 0.39$) (Figure 4A). Statistically significant mean reductions were observed compared to OADs (mean difference: −0.24%, [95% CI: −0.45, −0.03], $p = 0.02$) (Figure 4B) and against the placebo arm, with a (mean difference of –1.05%, [95% CI: −1.15, −0.95], $p < 0.00001$) (Figure 4C). In subgroup analyses, liraglutide 1.2 mg demonstrated significantly greater HbA1c reduction compared to DPP-4 inhibitors (mean difference: –0.27%; 95% CI: –0.32 to –0.22; $p < 0.00001$) and SGLT2 inhibitors (mean difference: –0.36%; 95% CI: –0.42 to –0.30; $p <$

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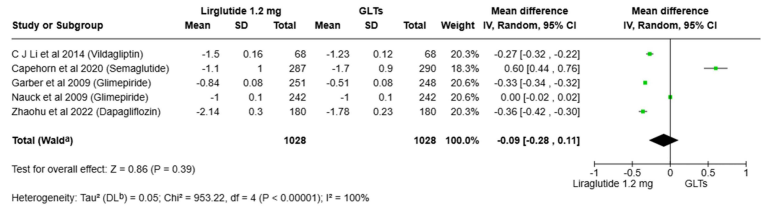
3A



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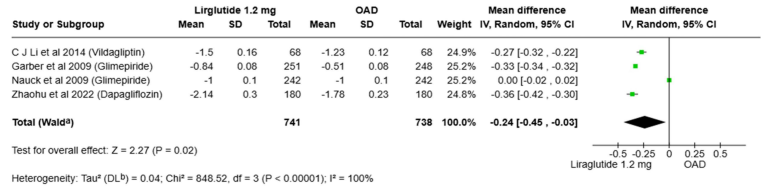

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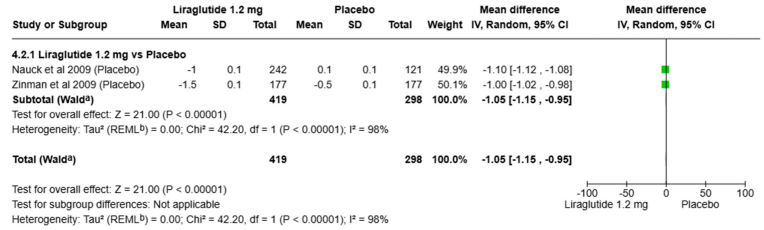

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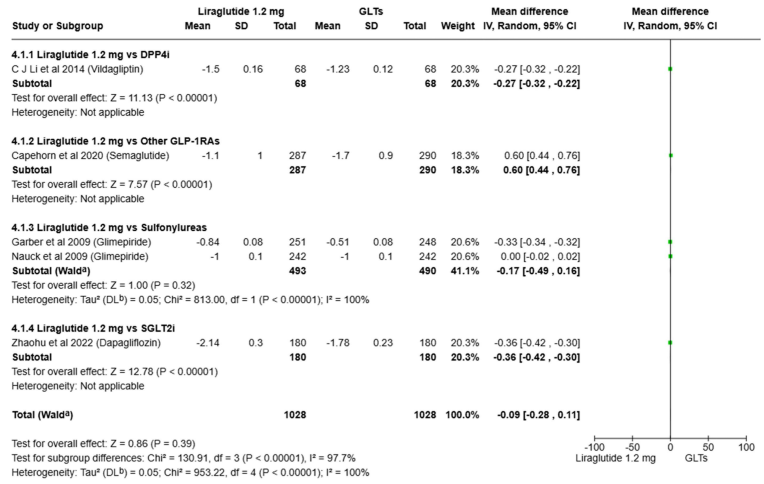

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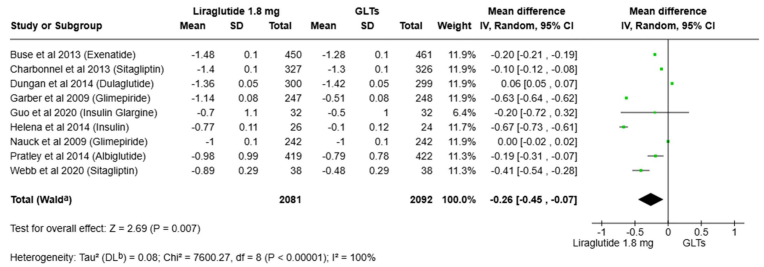

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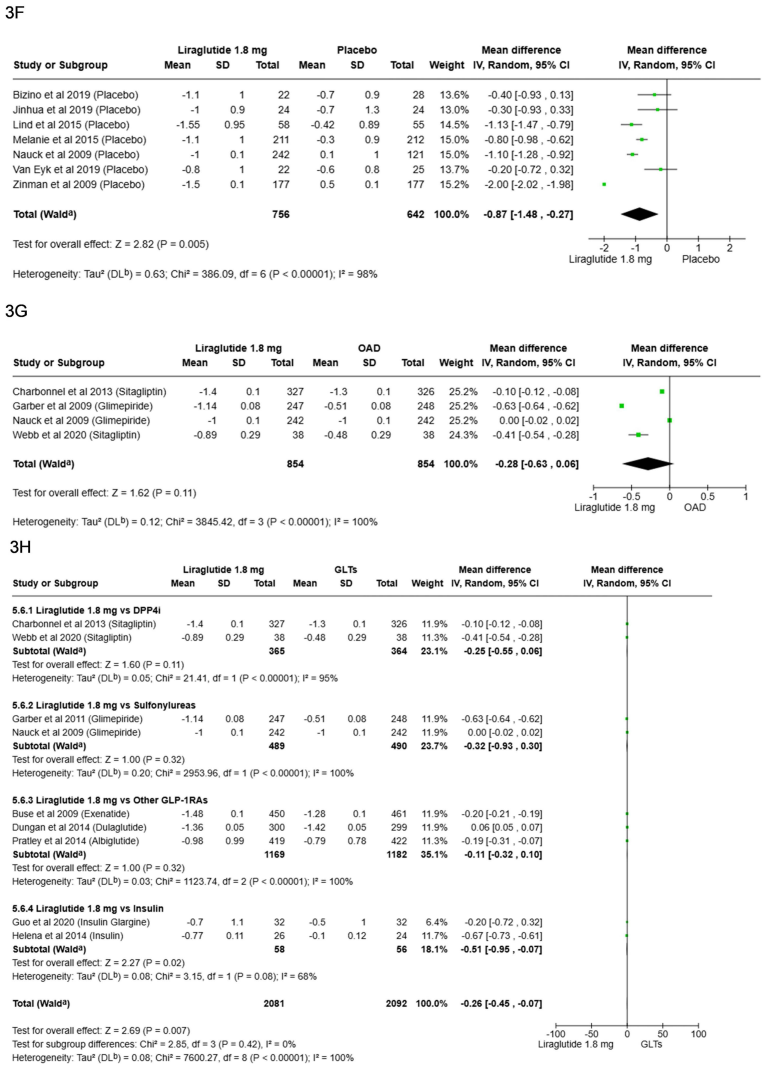

Figure 4 Liraglutide MA Forest Plots – Glycated Haemoglobin (HbA1c). (**A**) Liraglutide 1.2 mg v/s GLTs, (**B**) Liraglutide 1.2 mg v/s OAD, (**C**) Liraglutide 1.2 mg v/s Placebo, (**D**) Liraglutide 1.2 mg (Subgroup analysis), (**E**) Liraglutide 1.8 mg v/s GLTs, (**F**) Liraglutide 1.8 mg v/s Placebo, (**G**) Liraglutide 1.8 mg v/s OAD, (**H**) Liraglutide 1.8 mg (Subgroup analysis).

Note: Mean difference shown with 95% CI using random-effects model.

Abbreviations: GLTs, Glucose-lowering therapies (includes all OADs, insulin, and other GLP-1RAs); OADs, Oral antidiabetic drugs (SU, DPP4i, SGLT2i, Metformin); SU, Sulfonylureas; DPP4i, Dipeptidyl Peptidase-4 inhibitors; SGLT2i, Sodium–glucose cotransporter-2 inhibitors; GLP-1RA, Glucagon-like peptide-1 receptor agonist.

0.00001). However, in Other GLP-1RAs (semaglutide) demonstrated statistically significant HbA1c reduction than liraglutide 1.2 mg (mean difference: +0.60%; 95% CI: 0.44 to 0.76; $p < 0.00001$). The difference in HbA1c reduction between liraglutide 1.2 mg and sulfonylureas was not statistically significant (mean difference: –0.17%; 95% CI: –0.49 to 0.16; $p = 0.32$). (Figure 4D).

Liraglutide 1.8 mg demonstrated a significant reduction in HbA1c levels compared to both GLTs (mean difference: −0.26, [95% CI: −0.45, −0.07), *p = 0.007*) (Figure 4E) and against the placebo arm (mean difference: −0.87%, [95% CI: −1.48, −0.27], *p = 0.005*). (Figure 4F) The pooled effect size which compared Liraglutide 1.8 mg to OADs, exhibited a mean reduction of −0.28% (95% CI: −0.63, 0.06) in HbA1c levels, *p = 0.11*) (Figure 4G). Subgroup analysis demonstrated that Liraglutide 1.8 mg significantly reduced HbA1c compared to insulin (mean difference: –0.51% [95% CI: –0.95, –0.07]; $p = 0.02$), while differences versus DPP-4 inhibitors (mean difference: –0.25% [95% CI: –0.55, 0.06]; $p = 0.11$), sulfonylureas (mean difference: –0.32% [95% CI: –0.93, 0.30]; $p = 0.32$), and other GLP-1 RAs (mean difference: –0.11% [95% CI: –0.32, 0.10]; $p = 0.32$) were not statistically significant. (Figure 4H) Heterogeneity was observed across all the comparisons ranging from, [I^2 =68- 100%] and Tau^2 ranging from [0.04 to 0.75].

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In terms of FPG reduction (mg/dL), Liraglutide 1.2 mg versus GLTs showed a mean difference of 4.04 [95% CI: −4.38, 12.45] (Figure 5A), with a comparable *p*-value of *0.35*. Similarly, Liraglutide 1.2 mg versus OADs had a mean difference of −0.98 ([95% CI: −9.59, 7.64], $p = 0.82$) (Figure 5B) In subgroup analyses Liraglutide 1.2 mg significantly reduced FPG compared to DPP-4 inhibitors (–7.21 [95% CI: –8.36, –6.06]; $p < 0.00001$) and SGLT2 inhibitors (–2.16 [95% CI: –4.26, –0.06]; $p = 0.04$), a significantly smaller reduction in FPG with liraglutide 1.2 mg compared to sulfonylureas (+6.48 [95% CI: 4.77, 8.19]; $p < 0.00001$) and other GLP-1 RAs (+21.44 [95% CI: 14.65, 28.23]; $p < 0.00001$. (Figure 5C) In Liraglutide 1.8 mg versus GLTs demonstrated a comparable mean difference of –5.88 ([95% CI: −14.51], 2.74, $p=0.18$) and against the placebo arm, with a mean difference of –15.44 ([95% CI: −32.98, 2.10], $P=0.08$). (Figure 5D and E) Furthermore, in subgroup analysis liraglutide 1.8 mg demonstrated a significant reduction in FPG compared to sulfonylureas (mean difference: –10.81 [95% CI: –11.09, –10.53]; $p < 0.00001$). However, when compared with other GLP-1 RAs, results were mixed and overall non-significant (mean difference: –4.65 [95% CI: –14.18, 4.88]; $p = 0.34$), with high heterogeneity across studies.(Figure 5F) For PPG (mg/dL) analysis due to the availability of only two studies, the data of GLTs and Placebo were combined, Liraglutide 1.8 mg versus GLTs + placebo presented a mean difference of −12.90 ([95% CI: −48.25, 22.44], $p=0.47$) (Figure 5G). High heterogeneity was observed in all comparisons, with I^2 values ranging from 78% to 100% and Tau^2 values ranging from [57.20 to 557.54] and hence random effects model were used.

Safety Outcomes

Liraglutide 1.2 mg demonstrated significantly lower odds of nausea compared to other GLP-1RAs with an odds ratio of 0.66 ([95% CI: 0.44, 0.97], $p = 0.03$) and with no observed heterogeneity ($I^2 = 0\%$), (Figure 6A) Liraglutide 1.8 mg exhibited numerically lower incidence of nausea relative to other GLP-1RAs, with an odds ratio of 1.37 ([95% CI: 0.87, 2.16], $p = 0.17$) with substantial heterogeneity ($I^2 = 87\%$). (Figure 6C) For vomiting, Liraglutide 1.2 mg and 1.8 mg demonstrated no significant differences compared to other GLP-1RAs (OR 0.71 [95% CI: 0.41, 1.23], $p = 0.23$ and 1.13 [95% CI: 0.67, 1.91], $p = 0.64$) respectively. (Figure 6B and D). The safety assessment of Liraglutide 1.2 mg and 1.8 mg with respect to the GI ADRs, against the comparators, are represented using a forest plot.

Publication Bias

Funnel Plot for Weight Reduction

The funnel plot assessing the studies comparing the reduction of weight shows a relatively symmetrical distribution around the combined effect size suggesting no publication bias. The plot shows that most studies are clustered near the top, reflecting larger sample sizes and more precise estimates, with standard errors decreasing as sample sizes increase. The absence of conspicuous asymmetry reinforces the notion that publication bias is minimal for weight reduction (Figure 7A).

Funnel Plot for Glycemic Endpoints

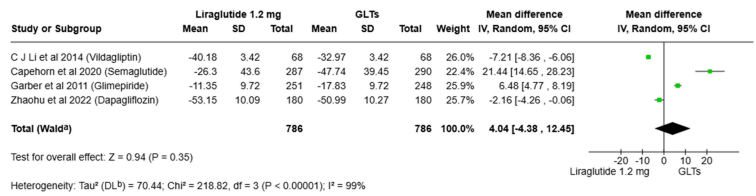
Similarly, the funnel plot for studies assessing glycemic endpoints shows a symmetrical distribution indicating no substantial publication bias. The presence of a few smaller studies with wider dispersion at the bottom of the funnel plot is typical and does not suggest asymmetry. Consequently, the glycemic outcomes are reliably represented by the available published data (Figure 7B).

Sensitivity Analysis

Sensitivity analysis using the LOO approach was conducted for the primary outcome of weight reduction to assess the stability of the pooled estimates for both liraglutide dose groups. For liraglutide 1.2 mg, the original analysis showed a non-significant reduction in weight compared with GLTs (mean difference: –1.24 kg, [95% CI –2.93 to 0.46]; $p = 0.15$). Following LOO analysis, the effect estimate remained comparable (–1.50 kg, [95% CI –3.40 to 0.39]; $p = 0.12$), indicating that the overall finding was not driven by any individual study (Supplementary Figure 1). Similarly, for liraglutide 1.8 mg, the original pooled analysis demonstrated a significant weight reduction relative to GLTs (mean difference: –2.30 kg, [95% CI –3.51 to –1.09]; $p = 0.0002$). After sequentially removing the study, effect size remained

robust (–2.17 kg, [95% CI –3.42 to –0.91]; $p = 0.0007$), confirming the consistency and reliability of the treatment effect (Supplementary Figure 2). Overall, the LOO sensitivity analyses support the robustness of the primary outcome findings for both liraglutide 1.2 mg and 1.8 mg.

4A



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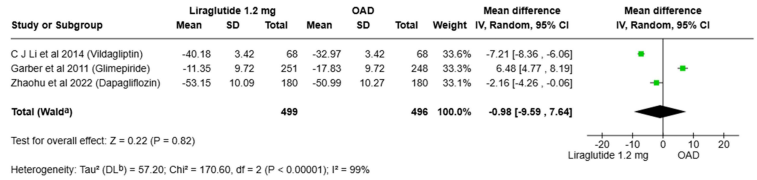

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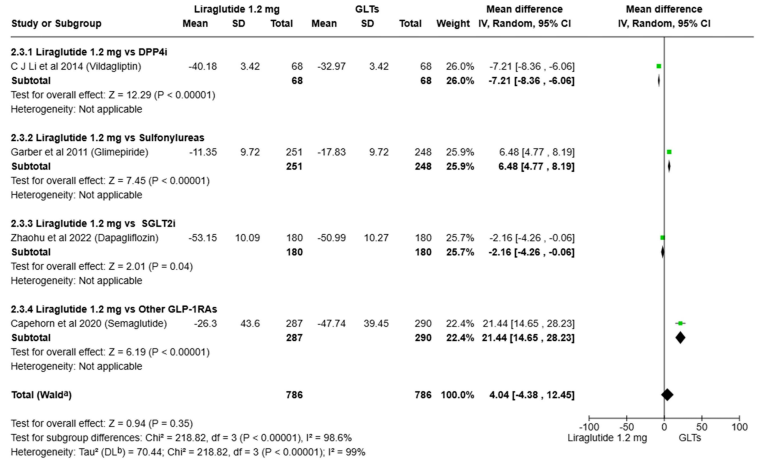

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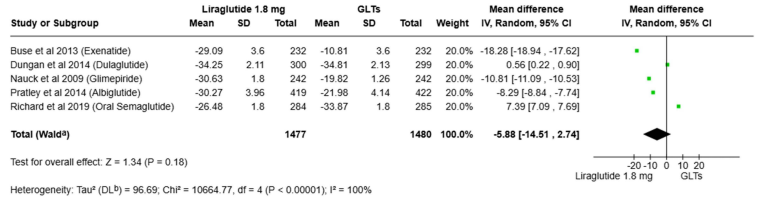

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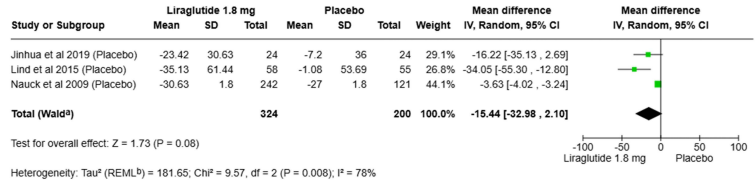

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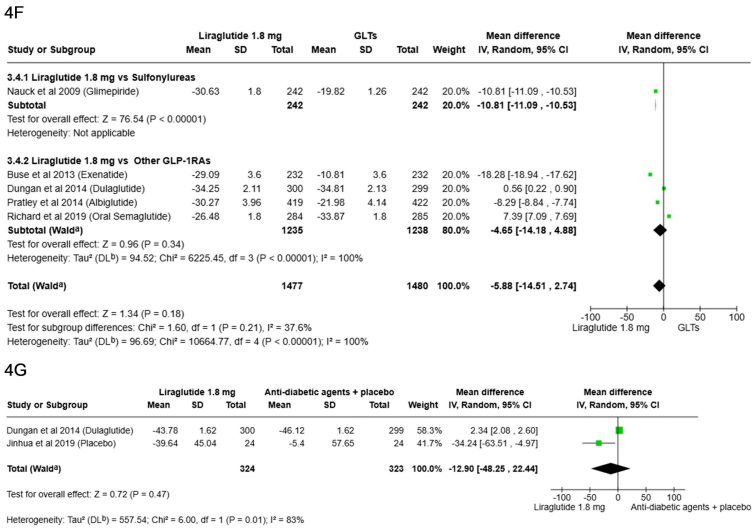


Figure 5 Liraglutide MA Forest Plots – Fasting Plasma Glucose (FPG) and Post Prandial Glucose (PPG). (**A**) Liraglutide 1.2 mg v/s GLTs, (**B**) Liraglutide 1.2 mg v/s OAD, (**C**) Liraglutide 1.2 mg (Subgroup analysis), (**D**) Liraglutide 1.8 mg v/s GLTs, (**E**) Liraglutide 1.8mg vs Placebo, (**F**) Liraglutide 1.8 mg (Subgroup analysis), (**G**) Liraglutide 1.8 mg v/s GLT + Placebo.

Note: Mean difference shown with 95% CI using random-effects model.

Abbreviations: GLTs, Glucose-lowering therapies (includes all OADs, insulin, and other GLP-1RAs); OADs, Oral antidiabetic drugs (SU, DPP4i, SGLT2i, Metformin); SU, Sulfonylureas; DPP4i, Dipeptidyl Peptidase-4 inhibitors; SGLT2i, Sodium–glucose cotransporter-2 inhibitors; GLP-1RA, Glucagon-like peptide-1 receptor agonist.

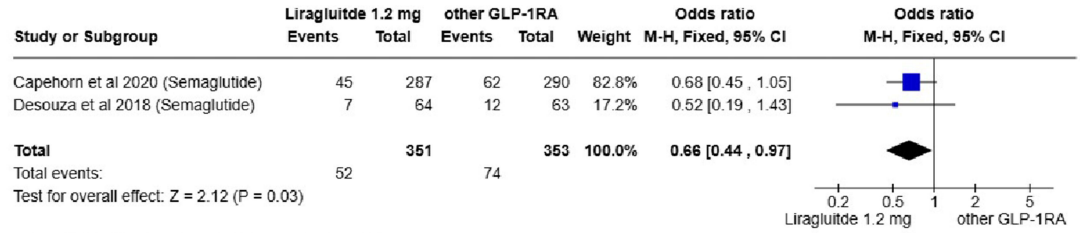
Discussion

This systematic review and meta-analysis demonstrates that low dose liraglutide at 1.2 mg leads to statistically significant reduction in weight –1.24 kg, 2.46 kg and −0.75 kg compared to GLTs, OADs and placebo while reductions relative to GLTs was comparable across weight and glycemic endpoints. However, the weight-reduction effect of liraglutide 1.2 mg was most evident in comparison with DPP-4 inhibitors, with a mean reduction of –5.10 kg, reinforcing its role in addressing weight-related metabolic concerns in type 2 diabetes. While the numerical differences versus sulfonylureas (–2.27 kg) and SGLT2 inhibitors (–0.19 kg) did not reach statistical significance, the directionality of effect still favored liraglutide, suggesting a potential advantage in clinical practice, especially for obese patients. Additionally, when compared to other GLP-1RAs, Liraglutide 1.8 mg showed a comparable reduction in weight (emphasizing its effectiveness in weight management within this class of medications. However, in several placebo studies, participants received concomitant GLTs, which may have attenuated the observed weight differences and led to an underestimation of the intervention's true effect. In terms of glycemic endpoints, Liraglutide 1.8 mg demonstrated significant reductions in HbA1c levels compared to GLTs ($p = 0.007$), and placebo ($p = 0.005$), respectively. Notably sub-group analysis revealed, a significant advantage was observed over insulin (mean difference: –0.51%; 95% CI: –0.95 to –0.07), and numerically greater reductions were seen compared to DPP-4 inhibitors, sulfonylureas, and other GLP-1 RAs, although these did not reach statistical significance individually In addition, Liraglutide 1.2 mg and 1.8 mg exhibited numerically lower incidence of nausea. For vomiting, Liraglutide 1.2 mg and 1.8 mg demonstrated no significant differences compared to other GLP-1RAs.

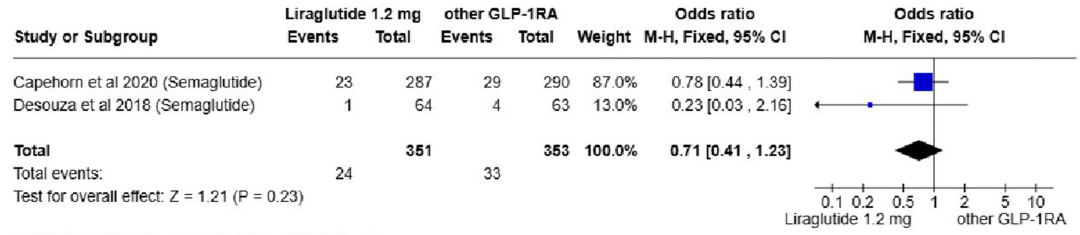
In diabetes management, addressing weight is critical as it plays a key role in disease progression and reducing the risk of complications such as hypertension, dyslipidemia, and cardiovascular conditions.[35–37] Extensive research has elucidated the effects of Liraglutide 3 mg on weight reduction in T2DM patients, yielding valuable insights into its therapeutic potential as an anti-obesity agent.[1,38] However, despite the proven efficacy of both 1.2 mg and 1.8 mg Liraglutide, there is a paucity of meta-analyses focusing on these doses, as recent meta-analyses address the utilization of 3 mg Liraglutide for weight management.[39,40]

In our meta-analysis, Liraglutide 1.8 mg resulted in a mean weight reduction of −2.81 kg compared to OADs, which is more substantial than the −2.56 kg reduction reported in a prior meta-analysis when OADs were used.[41] Our findings align with the substantial weight reduction observed with the lower doses of Liraglutide in Indian cohorts, where

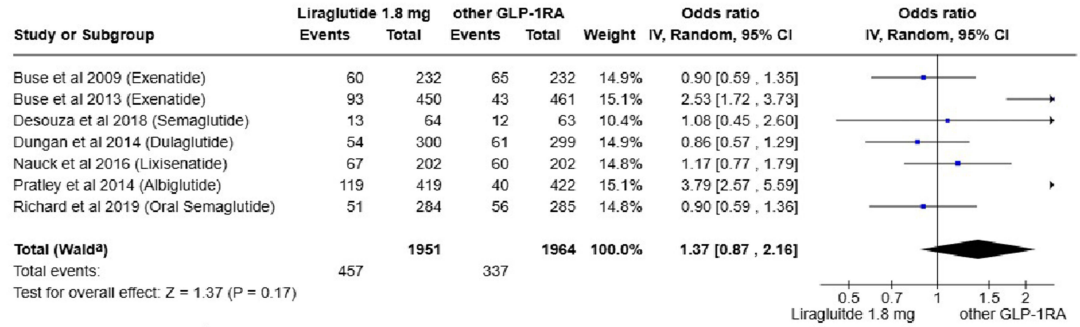
5A



5B



5C



5D

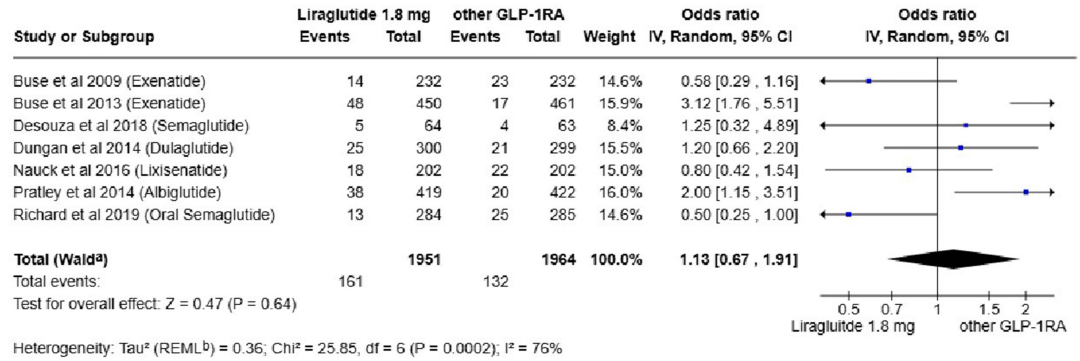


Figure 6 Liraglutide MA Forest Plots – Adverse Drug Reactions (ADR's). (**A**) Nausea – Liraglutide 1.2 mg v/s other GLP-1RA, (**B**) Vomiting – Liraglutide 1.2 mg v/s other GLP-1RA, (**C**) Nausea – Liraglutide 1.8 mg v/s other GLP-1RA, (**D**) Vomiting – Liraglutide 1.8 mg v/s other GLP-1RA.

Note: Mean difference shown with 95% CI using random-effects model.

Abbreviations: GLTs, Glucose-lowering therapies (includes all OADs, insulin, and other GLP-1RAs); OADs, Oral antidiabetic drugs (SU, DPP4i, SGLT2i, Metformin); SU, Sulfonylureas; DPP4i, Dipeptidyl Peptidase-4 inhibitors; SGLT2i, Sodium–glucose cotransporter-2 inhibitors; GLP-1RA, Glucagon-like peptide-1 receptor agonist.

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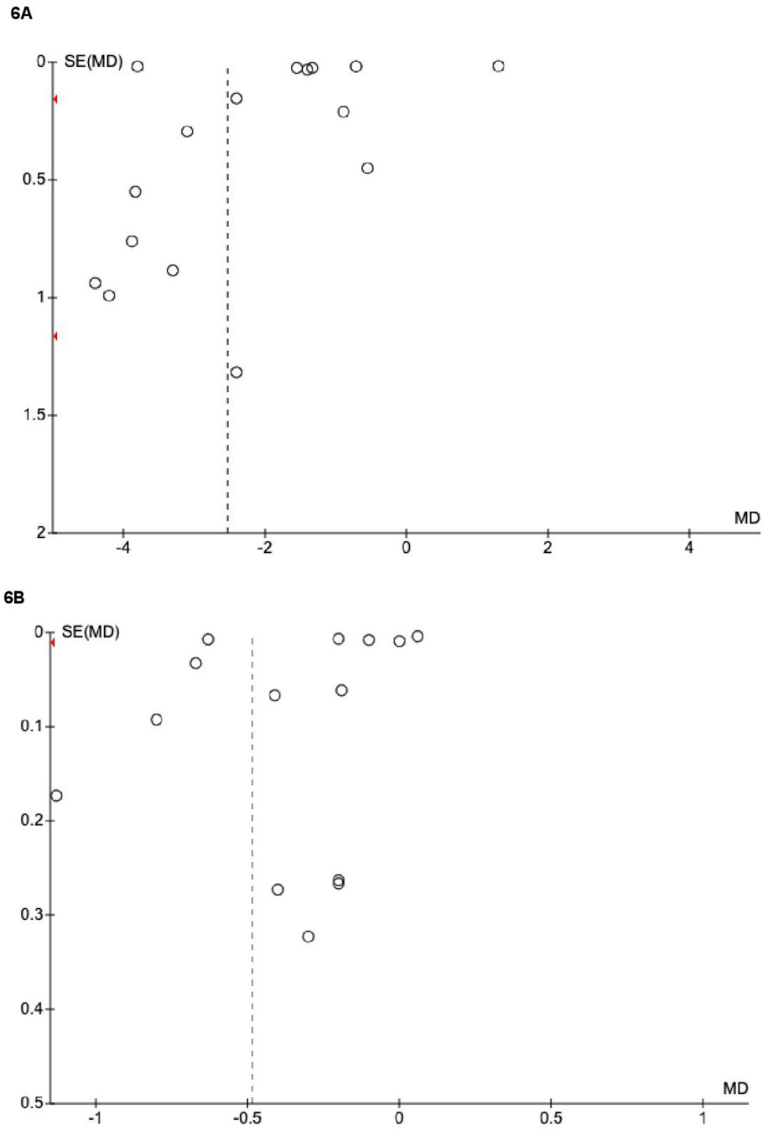

Figure 7 Liraglutide MA Forest Plots – Funnel plots. (**A**) Funnel plot for studies comparing Weight reduction, (**B**) Funnel plot for studies comparing HbA1c reduction.
Notes: Funnel plot assessing publication bias for studies comparing weight reduction with liraglutide versus glucose-lowering therapies (GLTs). Each circle represents an individual study. The vertical dashed line indicates the pooled mean difference (MD), and the y-axis shows the standard error [SE(MD)]. Symmetry suggests low risk of publication bias; asymmetry may indicate potential bias.

a 10.46% weight (kg) over nine months was reported.[42] Moreover, the SCALE trial indicated a mean weight loss of 4.7% with the 1.8 mg dose of Liraglutide, compared to 6.0% with the 3.0 mg dose, suggesting that the lower dose achieves nearly equivalent efficacy.[1] Collectively, these findings highlight the compelling evidence for the effectiveness of Liraglutide 1.8 mg in achieving sustained weight reduction in the South Asian demographic.

In the context of the ethnic minority phenotype of T2DM, a unique challenge arises due to the prevalence of lean fat body mass with abdominal obesity or adiposity in this population.[43–47] This distinctive body composition, characterized by a higher prevalence of subcutaneous abdominal fat despite a leaner overall body mass, presents a complex scenario for managing T2DM in Asian-Indian individuals.[48–51] The use of 3 mg Liraglutide has been widely beneficial and commonly employed in Western and Latin American populations with T2DM, who typically have higher baseline weights compared to their Asian-Indian counterparts.[52] Its efficacy in these phenotypes is well-established, demonstrating effectiveness in managing T2DM and promoting weight reduction.[53] Asians generally exhibit lower muscle mass compared to other ethnic groups. Consequently, significant weight loss in this population may result in a disproportionate reduction in

muscle mass, highlighting the risk of sarcopenia and related functional impairments.[54] Therefore, it is crucial to tailor weight loss interventions to preserve muscle mass in Asian population. Furthermore, if the higher dose of liraglutide (3 mg) were to be utilized in the Asian-Indian population, there is a notable concern regarding the potential for an increased incidence and severity of ADRs.[42,55,56] This risk is further compounded by the differences in drug metabolism and response patterns observed in Asian populations compared to Western populations, emphasizing the importance of tailoring treatment strategies to specific ethnic phenotypes to optimize efficacy and safety outcomes.[57–60]

Gastro Intestinal adverse effects with Liraglutide are transient and consistent with those typically associated with other GLP-1RAs, during the initial days of treatment.[5] Nausea and vomiting associated with Liraglutide are dose dependent.[61] The adherence with GLP1RAs is low, possibly due to the adverse events and costs.[62] Similarly, liraglutide is considered a more reasonable option over other GLP1RAs.[6] Therefore, it is plausible that utilizing lower doses and lower cost biosimilars GLP1RAs æ. However, it is important to contextualize these AEs within the broader clinical benefits of Liraglutide. Despite the higher incidence of nausea and vomiting, the significant weight reduction and improvements in glycemic control offered by Liraglutide 1.8 mg, provide a favorable overall benefit-risk profile.

The meta-analysis on the efficacy and safety of lower doses of Liraglutide provides substantial evidence for its use in weight reduction and glycemic control, particularly in people who are overweight or slightly obese such as Asian population where abdominal adiposity poses a significant challenge. By incorporating data from RCTs with varying durations and patient profiles, our study offers a unique and comprehensive analysis that sets it apart from existing meta-analyses and pooled analyses available. This meta-analysis suggests that clinicians may consider lower doses of liraglutide as pragmatic options to optimize both glycemic control and weight reduction, particularly in Asian and lean-fat phenotypes. This fills a critical gap, as no previous meta-analysis comprehensively covered the glycemic control and weight reduction properties of Liraglutide 1.2 mg compared to 1.8 mg in T2DM patients. The strength of our study lies in the rigorous methodology employed for selecting and evaluating eligible studies, ensuring a robust analysis of low dose Liraglutide' s effects over time. Limitations include high heterogeneity and the lack of meta-regression. Additionally, our search strategy did not include major databases such as Embase, Scopus, and Web of Science, which may have resulted in the omission of relevant studies and contributed to potential publication bias. Although we performed leave-one-out sensitivity analysis for key findings; however, this could not be applied to outcomes with fewer than five studies, which limited our ability to fully evaluate the robustness of those pooled estimates. However, our meta-analysis complements existing literature on the importance of using tailored diabetic management strategies in this specific demographic. This suggests that with proper management strategies, the therapeutic advantages of Liraglutide 1.8 mg can be maximized.

Conclusion

Liraglutide 1.8 mg consistently demonstrated significant reductions in both weight and HbA1c compared to various comparators, while Liraglutide 1.2 mg also showed notable efficacy with significant weight and HbA1c reductions compared to GLTs with lower incidence of some adverse events. This meta-analysis addresses a critical gap in the existing literature by providing a dose-specific evaluation of liraglutide at 1.2 mg and 1.8 mg, offering clinically meaningful insights. The findings are particularly relevant for South Asian populations, who may benefit from lower-dose regimens due to distinct metabolic profiles and potentially greater responsiveness to GLP-1 receptor agonists. These results support the need for individualized liraglutide dosing strategies to effectively optimize both glycemic control and weight management across diverse patient populations.

Data Sharing Statement

The data supporting the findings of this meta-analysis are derived from studies available in the public domain. All data extracted and analyzed during this study are included in the paper. The original datasets can be accessed from the respective publications cited in the reference list.

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Author Contributions

Conceptualization: S.J., A.K.D., K.K., S.K., S.Y.C., Methodology: S.J., A.K.D., S.Y.C., Investigation: S.J., A.K.D., S.Y.C., Data Curation: S.J., A.K.D., S.Y.C., Validation: S.K., Formal Analysis: S.J., S.Y.C., Software: S.J., S.Y.C., Resources: K.K., S.K., Project Administration: S.J., A.K.D., S.K., S.Y.C., Supervision: S.J., A.K.D., S.K., Visualization: S.J., S.Y.C., Writing – Original Draft: A.K.D., S.K., S.J., S.Y.C., Writing – Review & Editing: K.K., S.Y.C. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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