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Efficacy and safety of Glucagon-Like Peptide-1 receptor agonists in polycystic ovary syndrome: A systematic review and Meta-Analysis

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Abstract

Polycystic Ovary Syndrome (PCOS) affects an estimated 8–13% of reproductive-aged women and is frequently accompanied by obesity and insulin resistance, increasing cardiometabolic risk. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) may address these features, but comparative evidence in PCOS remains heterogeneous. We systematically searched PubMed, EMBASE, Scopus, Web of Science, and the Cochrane Library from inception to January 3, 2026 for comparative studies evaluating GLP-1RAs in women with PCOS. Random-effects meta-analyses were conducted using standardized mean differences (SMD) for continuous outcomes and risk ratios (RR) for adverse events. Heterogeneity was quantified using I^2. Risk of bias was assessed using ROBINS-I by independent reviewers with discrepancies resolved through discussion. Thirteen studies were included. GLP-1RAs improved abdominal girth (SMD −0.58; I^2=0%), BMI (SMD −1.14; I^2=92.5%), body weight change (SMD −1.24; I^2=93.4%), waist circumference (SMD −1.37; I^2=94.7%), glucose homeostasis (SMD −0.72; I^2=86.1%), and hormonal parameters overall (SMD −0.36; I^2=89.7%). The pooled effect for lipid profile was SMD −0.12 (p=0.060; I^2=53.1%). Safety analyses showed higher nausea (RR 2.20; I^2=17.2%) and diarrhea (RR 2.17; I^2=40.6%) with GLP-1RAs. In women with PCOS, GLP-1RAs were associated with improvements in anthropometric and glycemic outcomes, with gastrointestinal adverse events occurring more frequently.

Keywords: Glucagon-Like Pptide-1 receptor agonist, Meta-Analysis, Polycystic ovary syndrome, Systematic review

Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders in reproductive-aged women, affecting an estimated 8–13% depending on the diagnostic criteria used.[1] PCOS is frequently accompanied by excess adiposity and metabolic dysfunction: around half of patients are overweight or obese, and insulin resistance is common (often reported in up to 70%), contributing to impaired glucose tolerance, type 2 diabetes risk, and long-term cardiovascular risk.[2–4] Because these metabolic features interact with reproductive and hyperandrogenic manifestations, interventions that meaningfully reduce weight and improve glycemic physiology are central to real-world management.[2,4]

Current management often combines lifestyle intervention with agents such as metformin and symptom-directed therapies, but sustained weight reduction and consistent metabolic improvement remain challenging in routine practice.[5,6] Glucagon-like peptide-1 receptor agonists (GLP-1RAs) produce clinically meaningful weight loss and improve glucose regulation in broader metabolic populations, and they are increasingly considered for women with PCOS who have obesity and/or dysglycemia;[7] however, the comparative evidence in PCOS has been dispersed across trials using different GLP-1RA agents, comparators, and endpoints, and safety signals (particularly gastrointestinal adverse effects) require systematic synthesis alongside efficacy. Therefore, this systematic review and meta-analysis aimed to evaluate the efficacy and safety of GLP-1 receptor agonists in women with PCOS compared with placebo or active comparators.

Method

Protocol and reporting

This systematic review and meta-analysis was conducted using a prespecified methodology and is reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[8] We received no external

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fundings to conduct the following study.

Eligibility criteria

Studies were eligible if they: (1) enrolled women with a diagnosis of polycystic ovary syndrome (PCOS) based on recognized diagnostic criteria (e.g., Rotterdam, NIH, NICHD); (2) evaluated a glucagon-like peptide-1 receptor agonist (GLP-1RA) as the intervention (e.g., exenatide, liraglutide, semaglutide); (3) included a comparator group (placebo and/or active comparator such as metformin); and (4) reported extractable quantitative outcomes for efficacy and/or safety. Randomized controlled trials and controlled prospective clinical studies were included. Exclusion criteria comprised non-comparative before–after studies, reviews/editorials/protocols, animal or in vitro studies, and trials in which GLP-1RA was initiated only in the intervention arm alongside an additional medication not matched in the control arm.

Information sources and search strategy

A comprehensive electronic search was performed in PubMed, EMBASE, Scopus, Web of Science, and the Cochrane Library, from database inception to January 3, 2026. Search terms combined controlled vocabulary and free-text keywords relating to PCOS and GLP-1RAs, including terms such as "polycystic ovary syndrome," "PCOS," "glucagon-like peptide-1 receptor agonist," "GLP-1," "exenatide," "liraglutide," and "semaglutide," with appropriate Boolean operators and database-specific adaptations. Reference lists of included studies and relevant reviews were also screened to identify additional eligible studies.

Study selection

All retrieved records were exported to a reference management system and duplicates were removed. All reviewers independently screened titles and abstracts, followed by full-text assessment for eligibility. Disagreements were resolved by discussion and. Reasons for exclusion at the full-text stage were documented. The study selection process is summarized in the PRISMA flow diagram.

Data extraction

All reviewers independently extracted data using a standardized form. Extracted items included: study characteristics (author, year, design, setting/country, diagnostic criteria, intervention/comparator regimens, duration), participant baseline characteristics (sample size, age, weight, BMI), and outcome data. Efficacy outcomes included anthropometric measures (body weight, BMI, waist circumference, abdominal girth, waist-to-hip ratio), glucose-related outcomes (fasting glucose, 2-hour glucose, fasting insulin, 2-hour insulin, HOMA-IR), lipid profile parameters, and hormonal outcomes (e.g., total testosterone, free testosterone, SHBG, DHEAS, FAI as available). Safety outcomes included adverse events (e.g., nausea, vomiting, diarrhea, constipation, stomachache, bloating, dizziness, headache, insomnia). Where studies reported medians and interquartile ranges, these values were extracted as presented.

Risk of bias assessment

Risk of bias was assessed using the Risk of Bias In Non-randomized Studies of Interventions (ROBINS-I) tool. All reviewers performed the assessment independently, and any discrepancies were resolved through discussion until consensus was reached.

Effect measures and statistical synthesis

Meta-analyses were conducted using random-effects models to account for between-study heterogeneity. Continuous outcomes were synthesized as Standardized Mean Differences (SMDs) with 95% Confidence Intervals (CIs). Dichotomous safety outcomes were pooled as Risk Ratios (RRs) with 95% CIs. Statistical heterogeneity was quantified using the I^2 statistic, with corresponding heterogeneity tests where applicable. Pooled results are presented in forest plots for each outcome domain (Figures 2–10).

Publication bias and small-study effects were assessed when sufficient studies were available for an outcome (e.g., using funnel plots). Prespecified subgroup analyses and sensitivity analyses were performed when data permitted (e.g., by GLP-1RA agent or comparator type), and results were summarized descriptively if quantitative pooling was not appropriate.

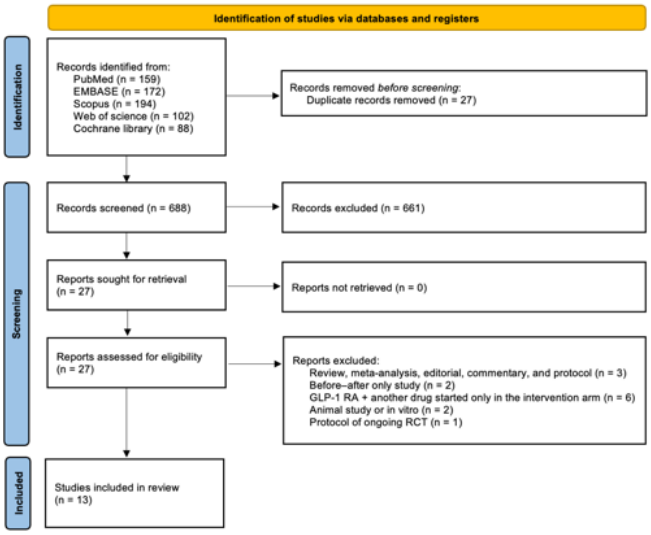

Figure 1. PRISMA flow chart for current systematic review of eligible studies

Results

Study selection

A total of 715 records were identified from databases (PubMed n=159, EMBASE n=172, Scopus n=194, Web of Science n=102, and Cochrane Library n=88). After removal of 27 duplicates, 688 records were screened, of which 661 were excluded. Twenty-seven reports were sought for retrieval and all 27 were retrieved and assessed for eligibility. Fourteen reports were excluded with reasons (review/meta-analysis/editorial/commentary/protocol n=3; before–after study n=2; GLP-1RA plus another drug started only in the intervention arm n=6; animal or in vitro study n=2; protocol of ongoing RCT n=1), leaving 13 studies included in the review (Figure 1).[9–21]

Study and participant characteristics

The included evidence base comprised randomized trials conducted in the USA, Denmark, Slovenia, and China, enrolling women with PCOS diagnosed primarily using the Rotterdam criteria (with additional trials using modified NIH 1990 or NICHD criteria). Interventions included exenatide (10 µg BID), liraglutide (1.2–3.0 mg QD), and semaglutide (0.5–1.0 mg weekly), compared with metformin (1000 mg BID) or placebo, with treatment durations ranging from 12 to 32 weeks. (Table 1).

Across studies, baseline demographics indicate a cohort largely characterized by overweight/obesity, with baseline BMI values spanning approximately the high-20s to low-40s kg/m^2 and baseline body weight ranging roughly from the low 70s to 120 kg, depending on the trial and setting, with baseline age generally in the late 20s to early 30s where reported (with some trials reporting age as not available in the baseline table). These baseline profiles align with the prespecified efficacy endpoints pooled in the meta-analyses (anthropometrics and cardiometabolic measures) and the safety endpoints summarized as adverse event rates.

Table 1. Characteristics and baseline demographics of included studies

Study (year)	Design	Country	PCOS diagnostic criteria	GLP-1RA vs Comparator (dose/frequency)	n (GLP-1RA / Control)	Duration	Baseline (GLP-1RA vs Control): Age (y); Weight (kg); BMI (kg/m^2)
Elkind-Hirsch (2008)	Open-label prospective RCT	USA	Rotterdam	Exenatide (10 µg BID) vs Metformin (1000 mg BID)	20 / 20	24 wks	Age: 28.2±1.1 vs 27.7±1.3
							Weight: 111.1±5.5 vs 109.1±5.0
							BMI: 39.9±1.5 vs 41.3±1.8
Elkind-Hirsch (2022)	Double-blind placebo-controlled RCT	USA	Modified NIH 1990	Liraglutide (3 mg QD) vs Placebo	44 / 23	32 wks	Age: 31.0±0.8 vs 32.0±1.1
							Weight: 111.0±2.8 vs 119.0±4.7
							BMI: 41.6±1.1 vs 43.9±1.7
		Denmark	Rotterdam		48 / 24	26 wks	Age: NR

Frossing (2018) †	Double-blind placebo-controlled RCT			Liraglutide (1.8 mg QD) vs Placebo			Weight: 94.2±15.4 vs 91.3±13.6 BMI: 33.3±5.1 vs 33.3±4.6
Jensterle (2014)	Open-label prospective RCT	Slovenia	NICHD	Liraglutide (1.2 mg QD) vs Metformin (1000 mg BID)	14-Nov	12 wks	Age: 31.5±6.4 vs 31.3±9.4 Weight: 108.9±15.1 vs 103.2±6.3 BMI: 39.3±4.2 vs 36.6±3.5
Jensterle (2015)	Open-label prospective RCT	Slovenia	Rotterdam	Liraglutide (1.2 mg QD) vs Metformin (1000 mg BID)	14 / 13	12 wks	Age: NR Weight: 102.8±16.3 vs 108.3±17.0 BMI: 36.7±5.6 vs 39.4±6.9
Jensterle (2015a)	Open-label prospective RCT	Slovenia	NICHD	Liraglutide (1.2 mg QD) vs Metformin (1000 mg BID)	14 / 14	12 wks	Age: 29.5±7.7 vs 25.3±5.2 Weight: 112.3±20.6 vs 105.3±20.1 BMI: 40.8±6.1 vs 38.2±7.0
Jensterle (2021)	Single-blind placebo-controlled RCT	Slovenia	Rotterdam	Semaglutide (0.5 mg weekly) vs Placebo	13 / 12	16 wks	Age: NR Weight: 100.8±11.8 vs 98.8±13.1 BMI: 36.8±3.9 vs 35.4±3.8
Jensterle (2022)	Single-blind placebo-controlled RCT	Slovenia	Rotterdam	Semaglutide (1 mg weekly) vs Placebo	9-Oct	12 wks	Age: NR Weight: 104.5 (89.8–111.8)* vs 99.5 (82.8–106)* BMI: 37.9 (33.2–41.4)* vs 31.0 (29.3–39.5)*
Li (2022)	Open-label prospective RCT	China	Rotterdam	Exenatide (10 µg BID) vs Metformin (1000 mg BID)	72 / 75	12 wks	Age: 28.19±3.96 vs 27.83±3.52 Weight: 73.72±11.43 vs 72.37±10.98 BMI: 29.07±3.92 vs 29.15±4.11
Liu (2017)	Open-label prospective RCT	China	Rotterdam	Exenatide (10 µg BID) vs Metformin (1000 mg BID)	78 / 80	24 wks	Age: 27.93±2.70 vs 27.69±3.80 Weight: 72.96±9.79 vs 70.39±4.57 BMI: 29.16±3.11 vs 28.29±1.86
Tao (2021)	Open-label prospective RCT	China	Rotterdam	Exenatide (10 µg BID) vs Metformin (1000 mg BID)	50 / 50	12 wks	Age: NR Weight: 80.00 (72.88–92.75)* vs 80.00 (74.70–86.00)*

							BMI: 30.99 (27.84–33.34)* vs 29.64 (28.23–33.14)*
Zheng (2017)	Open-label prospective RCT	China	Rotterdam	Exenatide (10 μg BID) vs Metformin (1000 mg BID)	41 / 41	12 wks	Age: 27.70±3.41 vs 28.16±3.92
							Weight: 72.58±11.29 vs 74.24±11.27
							BMI: 29.18±4.15 vs 29.00±4.10

Abbreviations: BID = twice daily; QD = once daily; wks = weeks; NR = not reported in the available table.
* Median (25th–75th percentile).† Consolidated as a single Denmark placebo-controlled liraglutide RCT entry to avoid duplicate counting.

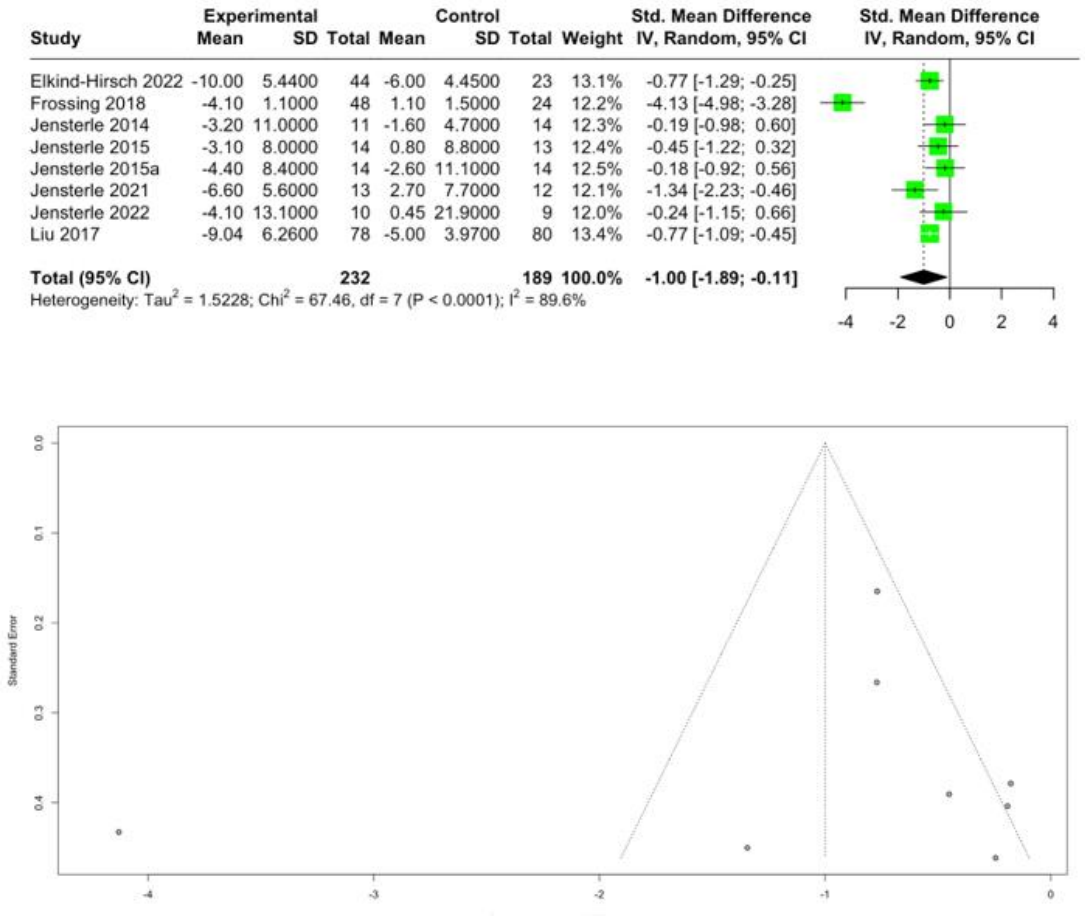

Figure 7. Meta-analysis of waist circumference in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

Efficacy outcomes

For anthropometric endpoints, GLP-1RA therapy was associated with pooled reductions in abdominal girth (SMD −0.58, 95% CI −0.84 to −0.32; p<0.001; I^2=0.0%) (Figure 2), BMI (SMD −1.14, 95% CI −1.80 to −0.47; p<0.001; I^2=92.5%) (Figure 3), body weight change (SMD −1.24, 95% CI −1.96 to −0.53; p<0.001; I^2=93.4%) (Figure 4), and waist circumference (SMD −1.37, 95% CI −2.32 to −0.41; p=0.005; I^2=94.7%) (Figure 7).

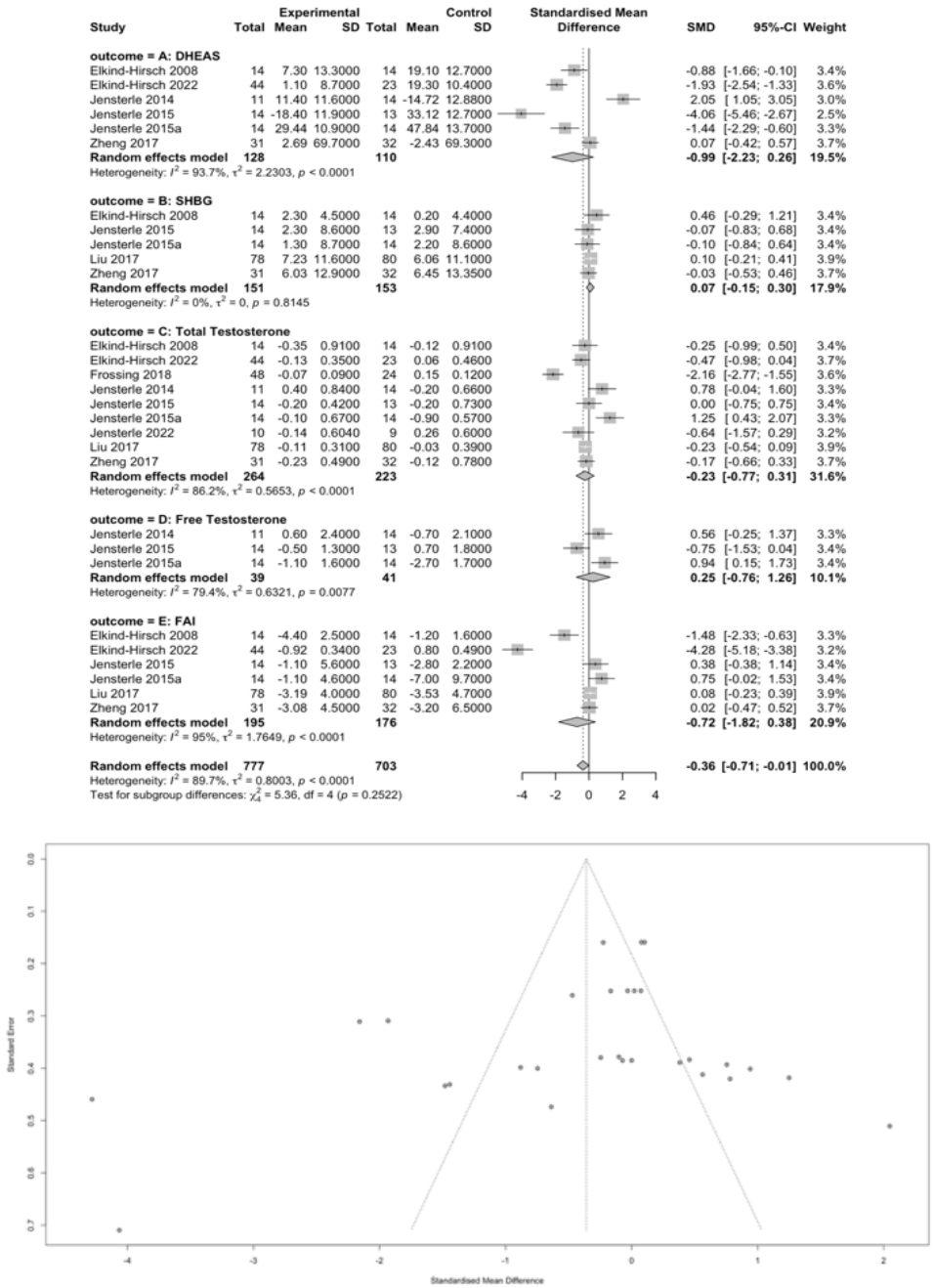

Figure 9. Meta-analysis of hormonal parameters in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

For metabolic and endocrine endpoints, the pooled effect for glucose homeostasis was SMD −0.72 (95% CI −0.96 to −0.48; p<0.001; I^2=86.1%) (Figure 5). The pooled effect for lipid profile was SMD −0.12 (95% CI −0.25 to 0.00; p=0.060; I^2=53.1%) (Figure 6). In the meta-analysis of waist circumference, GLP-1 receptor agonists were associated with a pooled standardized mean difference (SMD) of −1.37 (95% CI −2.32 to −0.41), with p = 0.005 and I^2 = 94.7% (Figure 8). The pooled effect for hormonal parameters was SMD −0.36 (95% CI −0.71 to −0.01; p=0.044; I^2=89.7%) (Figure 9).

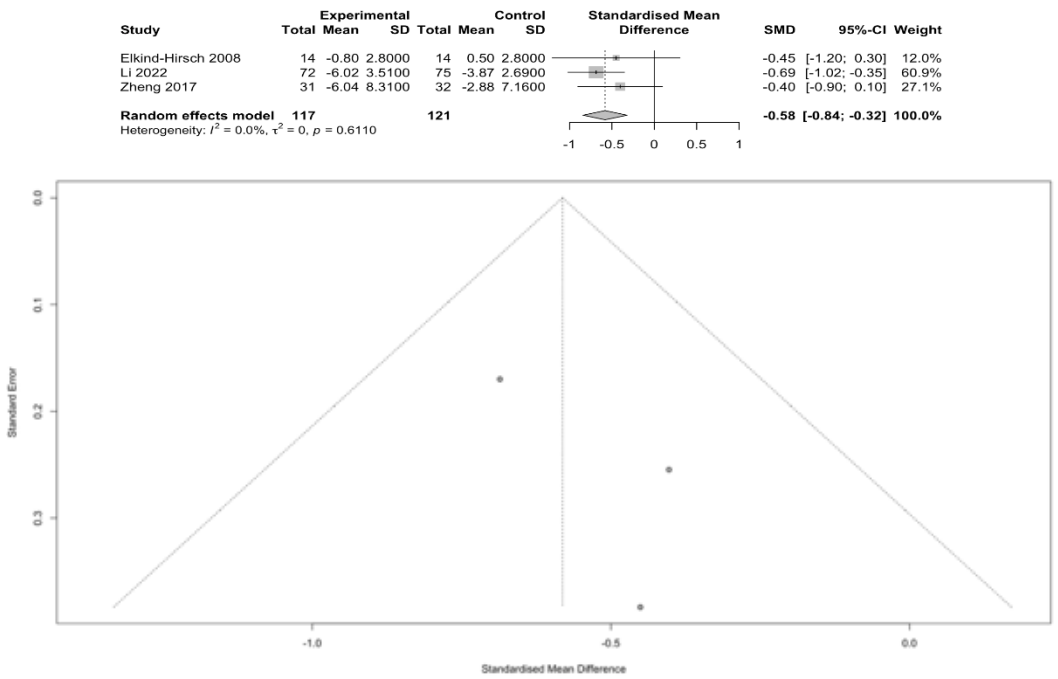

Figure 2. Meta-analysis of abdominal girth in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

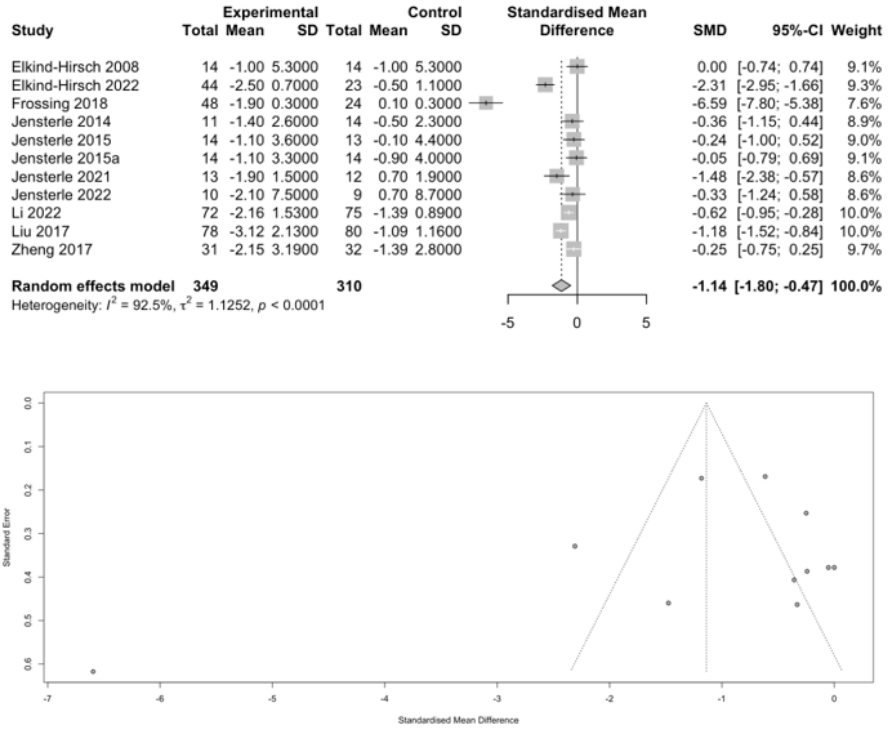

Figure 3. Meta-Analysis of body mass index in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

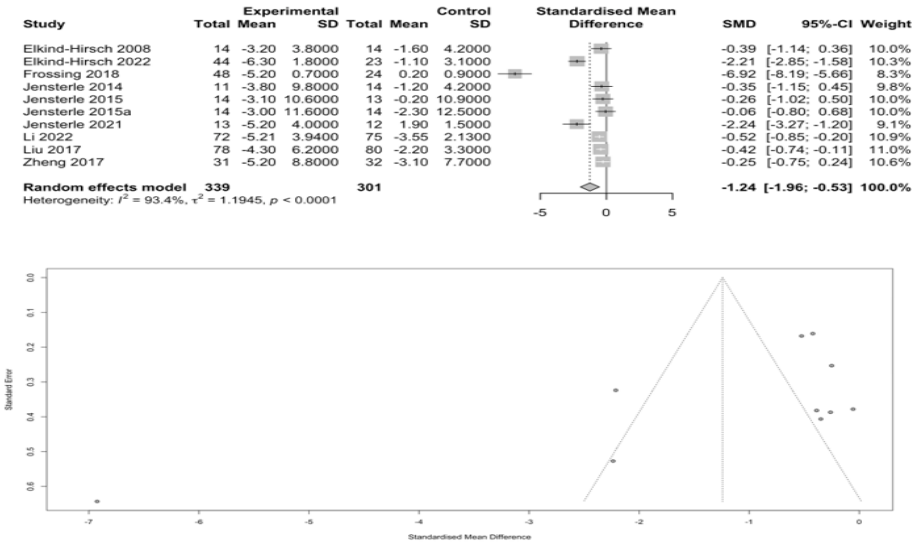

Figure 4. Meta-analysis of body weight change in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

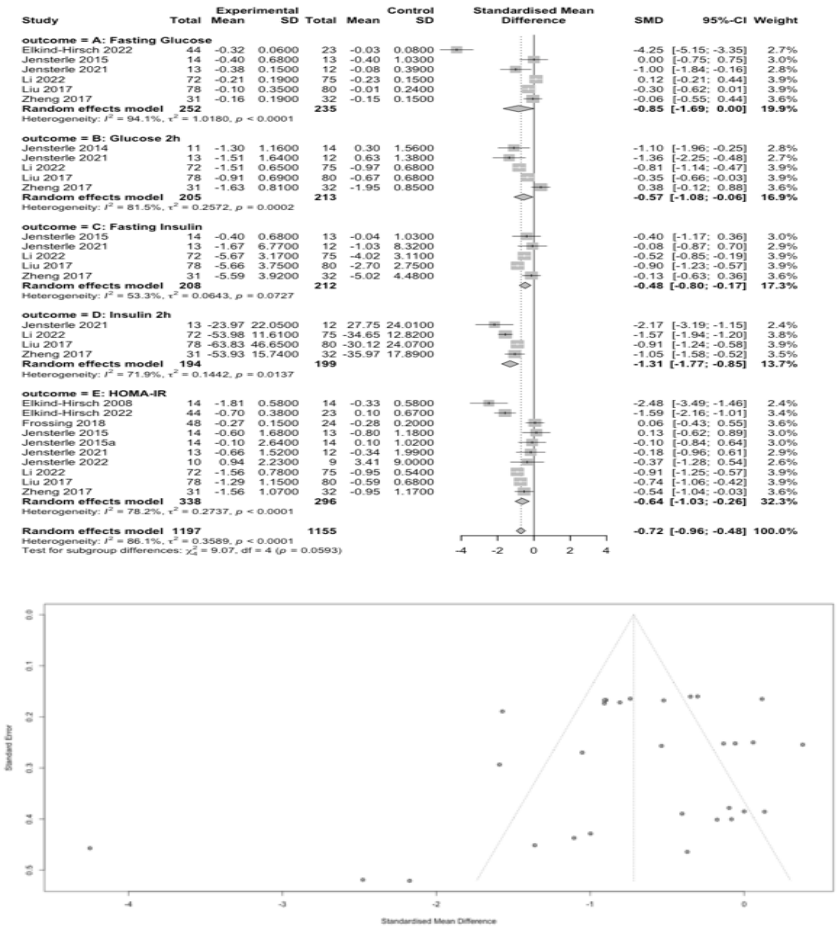

Figure 5. Meta-Analysis of glucose homeostasis in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

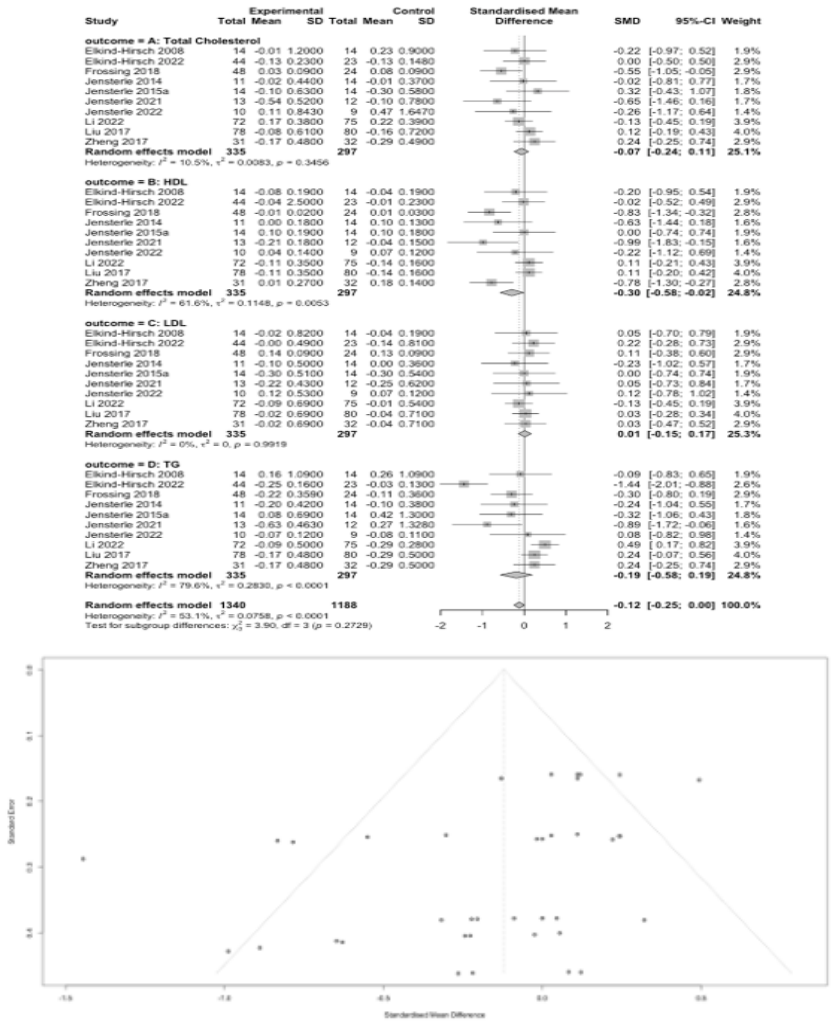

Figure 6. Meta-analysis of lipid profile in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

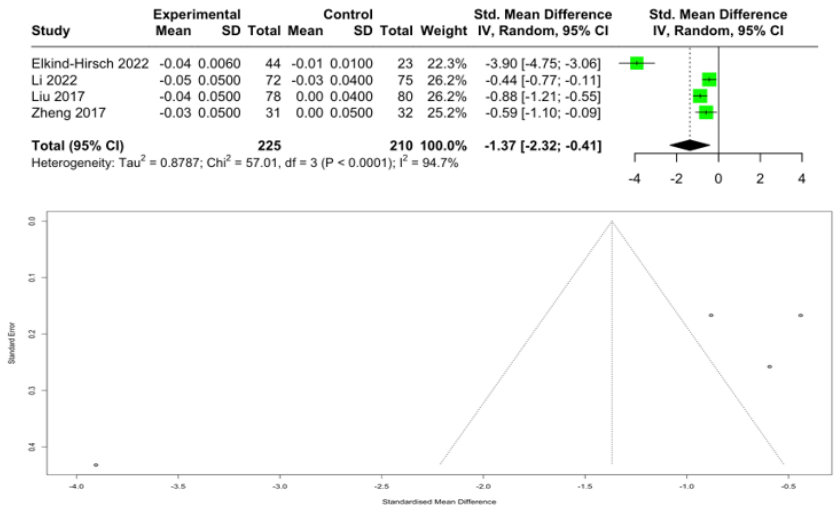

Figure 8. Meta-Analysis of waist-to-hip ratio in patients with polycystic ovary syndrome receiving GLP-1 receptor

agonists

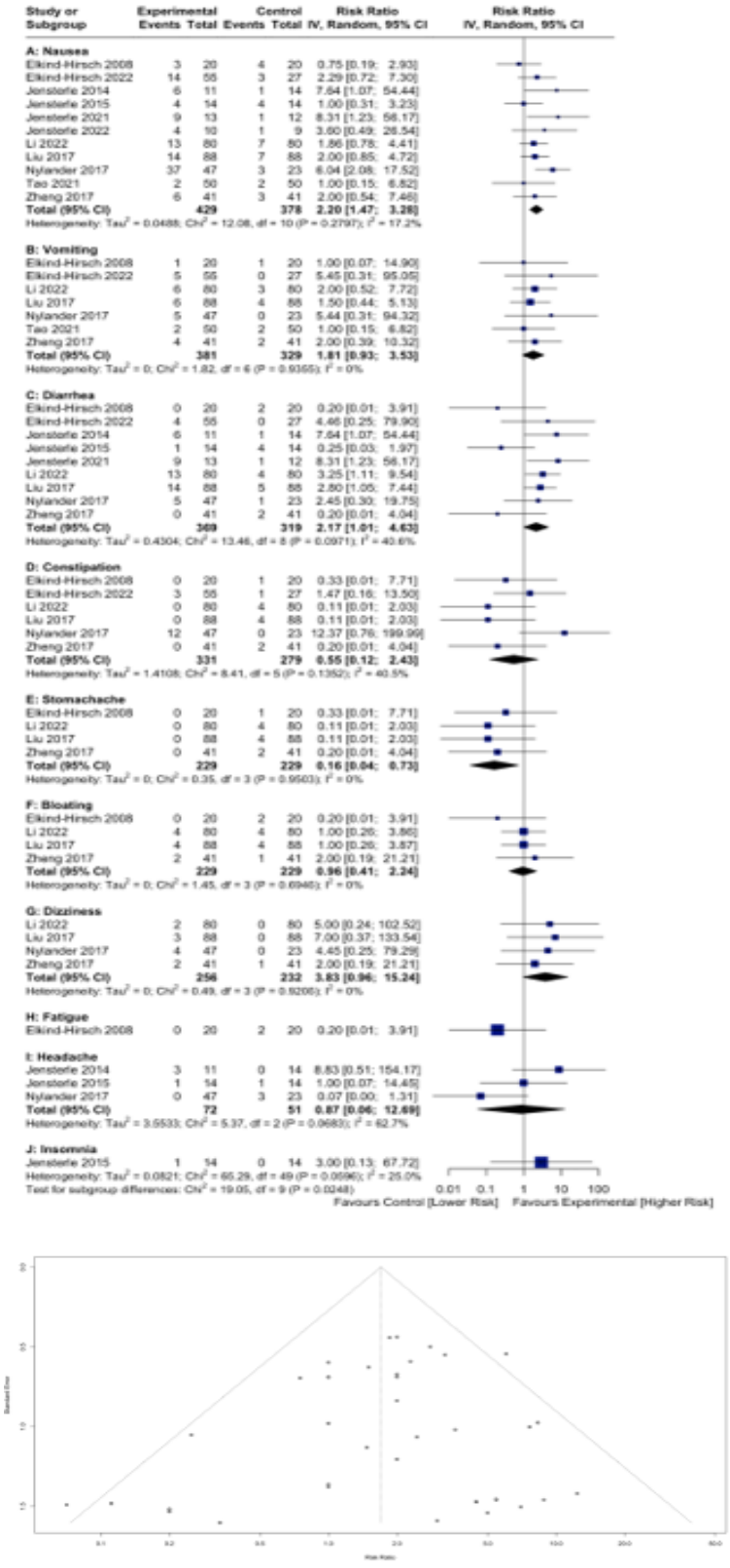

Figure 10. Meta-Analysis of adverse event rate in patients with polycystic ovary syndrome receiving GLP-1 receptor agonists

Safety outcomes

Adverse event rates were synthesized as pooled risk ratios (RRs). Nausea: RR 2.20 (95% CI 1.47 to 3.28; p<0.001; I^2=17.2%); vomiting: RR 1.81 (95% CI 0.93 to 3.53; p=0.081; I^2=0.0%); diarrhea: RR 2.17 (95% CI 1.01 to 4.63; p=0.046; I^2=40.6%); constipation: RR 0.55 (95% CI 0.12 to 2.43; p=0.436; I^2=40.5%); stomachache: RR 0.16 (95% CI 0.04 to 0.73; p=0.013; I^2=0.0%); bloating: RR 0.96 (95% CI 0.41 to 2.24; p=0.925; I^2=0.0%); dizziness: RR 3.83 (95% CI 0.96 to 15.24; p=0.057; I^2=0.0%); headache: RR 0.87 (95% CI 0.06 to 12.69; p=0.919; I^2=62.7%); insomnia: RR 3.00 (95% CI 0.13 to 67.72; p=0.491; I^2=25.0%) (Figure 10).

Risk of bias

The included studies risk of bias was of low to moderate risk of bias. Details of the scoring given to each included studies are presented in Figure 11.

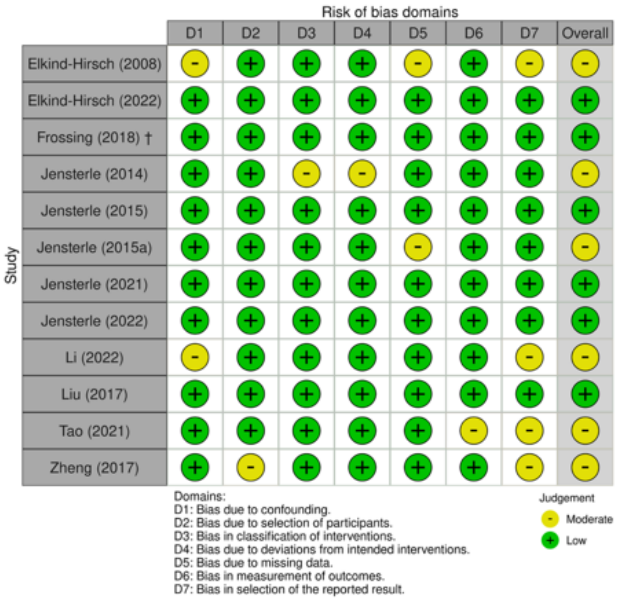

Figure 11. Risk of bias analysis of included studies using ROBINS-I

Discussion

Across controlled trials in women with PCOS, GLP-1 receptor agonists were associated with pooled improvements in key anthropometric and metabolic outcomes. In this synthesis, the pooled effect for body weight change (SMD −1.24) and BMI (SMD −1.14) was accompanied by improvements in measures of central adiposity, including waist circumference (SMD −1.37) and abdominal girth (SMD −0.58). Glycemic measures also improved overall (SMD −0.72 for glucose homeostasis), while the pooled effect for lipid parameters was smaller and not consistently significant (SMD −0.12). These findings are broadly consistent with prior randomized trials and earlier systematic reviews in PCOS that have reported weight reduction and better insulin-related outcomes with GLP-1RAs compared with placebo or metformin, with more variable effects on lipids and androgen-related markers.

The participant profiles across studies, predominantly young women with overweight or obesity, mirror the subgroup in which GLP-1RAs are most commonly considered in practice when weight and cardiometabolic risk are central treatment targets. Safety results were largely driven by gastrointestinal tolerability: nausea occurred more frequently with GLP-1RAs (RR 2.20), and diarrhea was also increased (RR 2.17), while several other adverse events showed no clear difference. These patterns are in line with prior PCOS and non-PCOS literature on GLP-1RAs, supporting the practical need for anticipatory counseling and symptom management (e.g., gradual up-titration when applicable, dietary adjustments, and coordination with metformin use if both are prescribed).[22,23]

Important variability across studies should be considered when applying these findings. Heterogeneity was substantial for several efficacy outcomes (often I^2 ~90% for weight-, BMI-, and waist-related endpoints), which is consistent with previous studies highlighting differences by agent (exenatide vs liraglutide vs semaglutide), dose, comparator (placebo vs metformin), baseline metabolic risk, and follow-up duration. Earlier PCOS trials have also suggested that GLP-1RA benefits may be more pronounced in higher-BMI or more insulin-resistant phenotypes, while endocrine outcomes can be smaller and less consistent, similar to the modest pooled hormonal effect observed here (SMD −0.36).[24]

Study limitations

This review has several limitations. Many pooled efficacy outcomes demonstrated substantial

between-study heterogeneity (often high I^2), reflecting differences in GLP-1RA agents and doses, comparators (metformin vs placebo), participant baseline characteristics (e.g., degree of obesity and metabolic risk), and treatment duration. Several trials were relatively small and short-term, limiting precision for some outcomes and restricting inference regarding longer-term efficacy and safety. Outcome reporting was not uniform across studies (including variations in units and reporting formats such as mean/SD vs median/IQR), which may have reduced the number of studies contributing to specific analyses. Finally, the inclusion of non-randomized or open-label designs in parts of the evidence base may increase susceptibility to bias despite structured risk-of-bias assessment.

Conclusion

In women with polycystic ovary syndrome, GLP-1 receptor agonists were associated with pooled improvements in anthropometric and glycemic outcomes, with more variable effects on lipid and hormonal parameters. Adverse events were predominantly gastrointestinal, with higher rates of nausea and diarrhea observed in pooled analyses. These findings support GLP-1 receptor agonists as a therapeutic option targeting weight and metabolic risk in PCOS, while emphasizing the importance of tolerability monitoring and the need for larger, longer-duration comparative trials with standardized outcome reporting to better define long-term benefit–risk.

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