Comparative Effectiveness of GLP-1 RAs, SGLT2 Inhibitors, and Metformin in Type 2 Diabetes

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Abstract

Background: Insulin-sparing therapies are central to contemporary type 2 diabetes (T2D) care, yet comparative HbAIc effects across GLP-1 receptor agonists (GLP-1 RAs), SGLT2 inhibitors, and metformin remain variably reported.

Methods: Following PRISMA 2020, we searched major databases (January 2010—February 2025) for randomized and real-world studies in adults with T2D reporting baseline and change in HbA Ic.

Arm-level data were pooled as mean differences (MD, %) using random-effects models; heterogeneity was assessed with Q and P. Prespecified analyses included class-level pooling, duration and baseline-HbA Ic subgroups, dose-response, and sensitivity checks.

Results: Fifteen studies met criteria for qualitative and quantitative synthesis. GLP-1 RAs achieved greater HbAIc reduction than active comparators (pooled MD =~ -0.53%, 95% CI = — 0.65 to —0.42; moderate heterogeneity). SGLT2 inhibitors lowered HbAIc versus placebo (pooled

MD = —0.52%, 95% Cl ~ —0.61 to —0.43; moderate heterogeneity) with dose-dependent and durable effects. Metformin extended-release and immediate-release were glycaemically equivalent over 24 weeks. A pragmatic head-to-head study suggested semaglutide reduced HbA 1c more than dapagliflozin, especially in insulin-deficient phenotypes, but variance was insufficient for pooling. Sensitivity analyses (RCT-only, exclusion of open-label/higher-risk studies, fixed-effects) supported the main findings.

Conclusions: Among insulin-sparing options, GLP-1 RAs provide the largest HbA1Ic reduction, SGLT2 inhibitors offer durable, moderate benefit versus placebo, and metformin XR is non-inferior to IR with better tolerability. Early phenotype-guided signals favor GLP-1 RAs in insulin- deficient patients.

Keywords: GLP-1 receptor agonists; SGLT2 inhibitors; metformin; HbA 1c; type 2 diabetes; insulin-sparing; systematic review; meta-analysis; precision medicine.

Introduction

Type 2 diabetes mellitus (T2DM) has emerged as a major global public-health challenge, driven by sedentary lifestyles, aging, and urbanization. In China alone, prevalence reached 116 million adults (~ 10.9 %) in 2019 and is projected to rise to 140 million by 2030 (Huang et al.,2021)Despite broader access to glucose-lowering therapies, fewer than 20 % of patients achieve HbAlc < 7 %, exposing them to micro- and macro-vascular complications. East-Asian T2DM typically reflects impaired B-cell function rather than obesity-driven insulin resistance, meaning modest weight loss (= 5 %) can substantially improve glycaemic control.

While insulin remains the most potent hypoglycaemic agent, it is limited by hypoglycaemia, weight gain, and injection burden. Consequently, international consensus guidelines from the American Diabetes Association (ADA) and European Association for the Study of Diabetes (EASD) advocate individualized, insulin-sparing pharmacotherapy that optimizes HbAlc while considering weight, adherence, and cardiovascular outcomes (Davies et al., 2022).

2.2 Therapeutic Overview

GLP-I Receptor Agonists

GLP-1 RAs mimic the incretin hormone GLP-1, enhancing glucose-dependent insulin secretion, suppressing glucagon, delaying gastric emptying, and reducing appetite (Home et al., 2018).These actions yield clinically meaningful HbAIc reductions (~ 1.2 — 1.7 %), accompanied by weight loss and minimal hypoglycaemia risk. Semaglutide and dulaglutide, long-acting once-weekly analogues, show consistent superiority to older comparators. In the SUSTAIN China trial, semaglutide 0.5 mg and 1.0 mg achieved HbA Ic reductions of —1.4 % and —1.7 %, compared with

—0.9 % for sitagliptin (Ji et al., 2021).Similarly, dulaglutide 1.5 mg produced —1.48 % versus —0.9% with glimepiride among East-Asian patients (Shi et al., 2020). The LEAD-3 study confirmed long-term liraglutide efficacy (—0.9 % to —1.1 % over two years) versus —0.6 % for glimepiride (Garber et al., 2011).

Real-world findings echo trial data. A UK cohort reported greater HbA Ic reductions for liraglutide than for basal insulin at 3 and 12 months (Forst et al., 2017).Emerging precision-medicine evidence indicates inter-individual variability: semaglutide yielded HbA 1c reductions of 13.4 mmol/mol in insulin-deficient (SIDD) patients versus 4.7 mmol/mol for dapagliflozin (Garber et al., 2011).These results highlight GLP-1 RAs' durable glucose control, weight benefit,

and cardiovascular protection, reinforcing their role as preferred second-line, insulin-sparing agents.

SGLT2 Inhibitors

SGLT2 inhibitors reduce plasma glucose by blocking renal glucose re-absorption, producing glycosuria, mild diuresis, and caloric loss. This insulin-independent mechanism leads to modest yet sustained HbAIc reductions (~ 0.7 — 1.0 %) and secondary benefits such as weight and blood- pressure reduction.

In a placebo-controlled RCT, canagliflozin 100 mg and 300 mg reduced HbAlc by —0.85 % and —1.06 % at 26 weeks, maintained over 52 weeks (Stenléf et al., 2013).Dapagliflozin 2.5-10 mg showed dose-dependent HbAlIc reductions (—0.65 % to —0.82 %) sustained through 102 weeks (Garber et al., 2011).The EMPA-REG MONO extension confirmed durability at 76 weeks with — 0.78 % to —0.89 % mean reductions (Garber et al., 201 1).Real-world Turkish data reported HbAlc drops of —1.18 % to —1.54 % with empagliflozin 10-25 mg added to metformin + gliclazide (Htoo et al., 2024). Meta-analytic pooling suggests a faster onset of HbAlc decline compared with sitagliptin or glimepiride (Cahn et al., 2019).

Together, these findings position SGLT2 inhibitors as durable, insulin-independent glucose-lowering drugs that confer metabolic and cardio renal benefits, suitable for combination or monotherapy when weight control and cardiovascular protection are prioritized.

Metformin

Metformin remains the first-line therapy and metabolic backbone in T2DM. It inhibits hepatic gluconeogenesis and enhances insulin sensitivity. Both immediate-release (IR) and extended-release (XR) forms achieve similar HbAlc reductions (~ —0.9 %), but XR improves gastrointestinal tolerability and once-daily adherence (Ye et al., 2018).Despite newer agents,metformin continues to complement GLP-1 RAs and SGLT2 inhibitors in insulin-sparing combination regimens, extending durability of glycaemic control.

2.3 Research Gap

Although these three classes have well-established efficacy, direct head-to-head comparisons remain inconsistent. Heterogeneity in study populations, treatment duration, and background therapy complicates relative efficacy assessment. Some trials favour GLP-1 RAs for the largest HbA Ic reductions, whereas others demonstrate parity between SGLT2 inhibitors and metformin add-ons (Ma et al., 2023; Zhang et al., 2025). Moreover, regional studies often lack

generalizability, and previous reviews seldom integrated both randomized and real-world data. Consequently, there is a clear need for a quantitative synthesis of comparative effectiveness among these insulin-sparing strategies using standardized statistical evaluation.

2.4 Objective

This systematic review and meta-analysis aims to compare the HbA1c-lowering effectiveness of GLP-1 receptor agonists, SGLT2 inhibitors, and metformin in adults with T2DM, focusing on their insulin-sparing potential, treatment durability, and real-world applicability. The study consolidates data from peer-reviewed randomized and observational studies published between 2011 and 2025.

2.5 Significance

By unifying recent evidence, this analysis provides evidence-based guidance for individualized pharmacologic therapy in T2DM. Determining which insulin-sparing class yields the most durable HbAle reduction will aid clinicians in tailoring regimens that balance efficacy, safety, and adherence. Furthermore, the findings may inform future ADA/EASD consensus updates and support the optimization of global diabetes-care strategies.

3. Methods

3.1 Study Design and Registration

This systematic review and meta-analysis adhered to PRISMA 2020 guidance. The protocol prespecified a comparative evaluation of glycaemic outcomes for glucagon-like peptide-1 receptor agonists (GLP-1 RAs), sodium—glucose co-transporter-2 inhibitors (SGLT2i), and metformin in adults with type 2 diabetes mellitus (T2DM). Only human, English-language studies were considered. Because all data were extracted from published sources, ethics approval and informed consent were not required.

3.2 Data Sources and Search Strategy

We systematically searched PubMed/MEDLINE, Embase, Scopus, Web of Science, ClinicalTrials.gov, and the Cochrane Library from January 2010 through February 2025. The strategy combined MeSH terms and free-text keywords for “type 2 diabetes,” “HbAIc,” and drug- or class-specific terms (“GLP-1 receptor agonist,” “semaglutide,” “liraglutide, “dulaglutide, “exenatide,” “SGLT2_ inhibitor,” “dapagliflozin,” “canagliflozin,” “empagliflozin, ” “metformin”), together with study-design terms (“randomized,” “real-world,” “observational,” “comparative effectiveness’’). Search strings were adapted to each database. We hand-searched

grey literature, clinical trial registries, and reference lists of relevant reviews and trials to identify additional studies.

3.3 Eligibility Criteria

Eligible studies were randomized controlled trials or real-world cohort studies published in peer-reviewed journals between 2010 and 2024 that enrolled adults with T2DM. Interventions included GLP-1 RA monotherapy or combinations (liraglutide, semaglutide, dulaglutide, exenatide), SGLT2 inhibitors (canagliflozin, dapagliflozin, empagliflozin), or metformin (immediate-release or extended-release). Acceptable comparators were placebo, sitagliptin, glimepiride, insulin glargine, basal insulin, or metformin. Studies were required to report baseline HbAlc and a quantitative change from baseline in HbAlc (mean + SD or least-squares mean + SE). We excluded paediatric or type | diabetes populations; publications without extractable numerical HbA Ic data (including those reporting only slopes or qualitative outcomes); non-peer-reviewed abstracts, reviews, editorials, or conference proceedings; and studies that combined multiple glucose-lowering agents without separate arm-level reporting.

3.4 Study Selection

The search identified 1,472 records from databases and 38 from manual sources. After removing 392 duplicates, 1,118 titles and abstracts were screened, of which 934 were excluded as non-human, non-English, non-primary research, or unrelated to HbAlc outcomes. We assessed 184 full-text articles for eligibility and excluded 169 because they lacked usable HbAlc data, had follow-up shorter than 12 weeks, were non-comparative, or did not provide sufficient statistics for pooling. The final dataset comprised 15 studies included in both the qualitative synthesis and the quantitative meta-analysis. Discrepancies at any stage were resolved by discussion and consensus.

3.5 Data Extraction

Using a standardized form, we extracted author, year, country/region, study design, sample size per arm, background therapy, intervention and comparator (drug, class, and dose), treatment duration, baseline HbA Ic (mean + SD), and change in HbAIc (mean + SD, or LS mean + SE with conversions as needed). Where HbAlc was reported in mmol/mol, we converted to percentage using HbAIc (%) = 0.09148 x mmol/mol + 2.152. For multi-arm trials with a shared comparator,

we treated each dose as a separate comparison and split the comparator sample size evenly to avoid double counting. Trials providing only endpoint means were recorded but preferentially we used change-from-baseline data; where necessary, endpoint and change data were mixed and flagged for sensitivity analysis. Studies reporting only slopes or without arm-level variance (e.g., modelling analyses) were retained for narrative context but were not pooled.

3.6 Quality Assessment

Randomized trials were appraised with the Cochrane Risk of Bias 2 tool across randomization, allocation concealment, blinding, incomplete outcome data, and selective reporting. Observational studies were evaluated with the Newcastle—Ottawa Scale (selection, comparability, and outcome).

Two reviewers performed assessments independently and reconciled differences by consensus. We summarized study-level judgments and used them to inform sensitivity analyses (e.g., excluding open-label or higher-risk studies).

3.7 Statistical Analysis

The primary effect size was the mean difference (MD) in HbAlc (%) between intervention and comparator. When only LS means and SEs were available, SEs were converted to SDs using SD = SE x Vn. We pooled effects using a DerSimonian—Laird random-effects model to accommodate between-study heterogeneity and reported 95% confidence intervals. Statistical heterogeneity was quantified with Q and I statistics, and we also inspected the consistency of effects across studies.

Prespecified subgroup analyses evaluated drug class, treatment duration (< 24 vs > 24 weeks), baseline HbA Ic strata, region, background therapy, and study design (RCT vs real-world). Dose-response was explored within classes where multiple fixed doses were available (e.g., semaglutide 0.5 vs 1.0 mg; canagliflozin 100 vs 300 mg; dapagliflozin 2.5/5/10 mg). Sensitivity analyses included leave-one-out influence diagnostics, exclusion of open-label and higher-risk studies, RCT-only pooling, and comparison of random- and fixed-effects models. Publication bias was planned via funnel plots and Egger's test for comparisons with =10 studies; where that threshold was not met, we reported this as a limitation. Analyses were conducted in Review Manager 5.4 and R (Meta and metaphor), with two-sided a = 0.05.

3.8 Ethical Considerations

This review synthesized previously published data without individual patient identifiers and did

not require institutional review board approval. All procedures complied with best practices for secondary data use and transparent reporting.

4. Results

4.1 Study selection

A systematic search of PubMed, Embase, Scopus, Web of Science, ClinicalTrials.gov and the Cochrane Library identified 1,472 database records and 38 additional records from manual searches (trial registries and reference lists). After removal of 392 duplicates, 1,118 unique titles/abstracts were screened. 934 records were excluded for being non human, non English, and reviews or unrelated to HbAlc outcomes. 184 full texts were assessed for eligibility; 169 were excluded because they lacked extractable HbA Ic data, had short duration (<12 weeks), used non comparative designs or lacked statistical information. This left 15 studies meeting the inclusion criteria; 11 studies (covering 5 GLP 1 receptor agonist comparisons, 5 SGLT2 inhibitor comparisons and | metformin formulation comparison) had sufficient numerical data for quantitative pooling, while other trials contributed to narrative synthesis.

4.2 Characteristics of included studies

key features of the included trials, including design, sample size,

interventions, baseline HbAlc and key remarks. The majority were randomised controlled trials

5. Discussion

5.1 Principal findings

Drawing on contemporary randomized and real-world evidence, our synthesis shows that GLP-1 receptor agonists (GLP-1 RAs) yield larger HbAlc reductions than active comparators (e.g., sitagliptin, glimepiride, titrated basal insulin) over ~24—30 weeks, with additional benefits on weight and a low risk of hypoglycaemia. This is exemplified by semaglutide and dulaglutide outperforming sitagliptin, glimepiride and insulin glargine in East-Asian and international cohorts (Araki et al., 2015; Garber et al., 2011; Huang et al., 2021). SGLT2 inhibitors demonstrate moderate, dose-dependent HbA Ic lowering versus placebo that is durable to 52-102 weeks and accompanied by weight and blood-pressure reductions (Garber et al., 2011, 2011; Wilding et al., 2013).In direct comparison, semaglutide reduced HbAlc more than dapagliflozin in a pragmatic, cluster-stratified study, especially among patients with insulin-deficient phenotypes, underscoring heterogeneity of response (Garber et al., 2011). For metformin, XR and IR formulations achieved similar glycaemic efficacy, with XR conferring tolerability and

adherence advantages (Aggarwal et al., 2018).Real-world data echo trial findings, with liraglutide outperforming basal insulin for HbA Ic and weight at multiple time points (Overbeek et al., 2018).

5.2 Interpretation in context of prior literature

Our findings are consistent with the therapeutic hierarchy implied by guideline trends: GLP-1 RAs often provide the greatest HbAlc lowering with weight loss and minimal hypoglycaemia risk, aligning with semaglutide's and dulaglutide's superiority over DPP-4 inhibitors, sulphonylureas, and basal insulin in pivotal programs (Araki et al., 2015; Garber et al., 2011; Zhang et al., 2025).SGLT2 inhibitors' effects appear modest but steady and durable, with additional cardio-renal benefits increasingly valued in current practice (Garber et al., 2011, 2011; Wilding et al., 2013). The head-to-head signal favoring semaglutide over dapagliflozin—particularly in insulin- deficient clusters—fits the emerging precision-medicine narrative in T2D (Garber et al., 2011).

Meanwhile, metformin's parity between XR and IR for HbAlc reduction while improving GI tolerability with XR supports its continued role as a backbone therapy (Aggarwal et al., 2018).

Longer-term liraglutide data further reinforce the durability of GLP-1 RA effects (Garber et al., 2011)

5.3 Insulin-sparing strategies and clinical implications

Collectively, the evidence supports prioritizing insulin-sparing regimens that pair glycaemic efficacy with weight loss and low hypoglycaemia risk. For patients inadequately controlled on metformin, GLP-1 RAs are compelling when weight management and robust HbAIc reduction are key goals; SGLT2 inhibitors are attractive when cardiorenal risk reduction and durability are prioritized. The real-world advantage of liraglutide over basal insulin suggests that, in appropriate patients, advancing to a GLP-1 RA may defer or minimize insulin exposure while improving outcomes (Overbeek et al., 2018). For initiation or optimization of metformin, XR may enhance adherence without sacrificing glycaemic control (Aggarwal et al., 2018).

5.4 Precision-medicine insights

The cluster-stratified comparison indicates that insulin-deficient phenotypes (SIDD) respond more strongly to GLP-1 RAs than to SGLT2 inhibitors, while insulin-resistant phenotypes (SIRD) show smaller differentials (Garber et al., 2011). In East-Asian populations—where B-cell dysfunction is prominent—this observation dovetails with GLP-1 RA efficacy seen in

semaglutide and dulaglutide trials (Araki et al., 2015; Zhang et al., 2025). Such stratification could inform agent selection beyond one-size-fits-all algorithms, especially when aiming to spare or delay insulin.

5.5 Safety, tolerability, and adherence considerations

Safety profiles were consistent with class expectations. GLP-1 RAs were associated with gastrointestinal adverse events but low hypoglycaemia risk, and weight loss was a common favorable (Garber et al., 2011, 2011). SGLT2 inhibitors were linked to weight and BP reductions, with known risks such as genital mycotic infections not systematically captured in our HbAlc- focused extraction (Garber et al., 2011; Wilding et al., 2013). Metformin XR enhanced gastrointestinal tolerability and once-daily adherence without compromising efficacy (Aggarwal et al., 2018). These tolerability profiles have practical implications for persistence and real-world effectiveness.

5.6 Strengths

This review uses a structured, PRISMA-guided approach with explicit extraction of arm-level HbAlc changes, standardized unit conversions, and pre-specified subgroup and sensitivity analyses. By incorporating both randomized and real-world evidence, we reflect practice-relevant effectiveness while maintaining internal validity through risk-of-bias appraisal (RoB 2 and NOS). The dataset spans short- and longer-term follow-up and includes dose-response contrasts for several agents, adding granularity to comparative effectiveness.

5.7 Limitations

Several constraints warrant caution. First, the number of pool able studies per comparison was modest, limiting precision for some class-level estimates and precluding formal publication-bias testing in most contrasts ($k < 10$). Second, heterogeneity arose from differences in baseline HbA Ic, duration, background therapies (e.g., metformin + sulphonylurea), geography (East-Asia-heavy in some trials), and study design (blinded RCTs vs open-label vs observational). Third, some reports provided least-squares means without SDs or lacked variance around head-to-head differences, constraining pooling (e.g., the semaglutide—-dapagliflozin pragmatic trial) and necessitating narrative synthesis in places (Garber et al., 2011). Fourth, a few studies had short follow-up (e.g.,12 weeks) or open-label designs introducing performance bias (Araki et al., 2015), though sensitivity analyses generally supported the main signals. Finally, our scope centered on HbA Ic,

so cardiovascular-renal outcomes, hypoglycaemia, and adherence/cost-effectiveness were not synthesized quantitatively here, despite their clinical importance.

5.8 Clinical implications

For adults with T2D not at glycaemic targets on metformin, GLP-1 RAs should be considered when the priority is maximizing HbA Ic reduction and weight loss with low hypoglycaemia risk. SGLT2 inhibitors are compelling for durable glycaemic control with metabolic and potential cardio-renal advantages, especially when weight and BP lowering are desired. Metformin XR may improve treatment persistence relative to IR without sacrificing efficacy. Where available, phenotype-informed selection—e.g., favoring GLP-1 RAs in insulin-deficient clusters—may further optimize outcomes and delay insulin escalation (Garber et al., 2011).

5.9 Future research

Three priorities emerge. First, adequately powered head-to-head RCTs of GLP-1 RA vs SGLT2 inhibitor with phenotypic stratification (SIDD/SIRD and beyond) and arm-level variance reporting are needed to enable robust pooling. Second, longer-term comparative studies (=104 weeks) should integrate hard outcomes (e.g., cardiovascular/renal events), hypoglycaemia, weight trajectories, and treatment persistence alongside HbAlc. Third, implementation and cost-effectiveness research in East-Asian and resource-constrained settings is essential, given regional pathophysiology and access considerations highlighted in the background (Zhang et al., 2025).

6. Conclusion

This systematic review and meta-analysis integrates randomized and real-world evidence to compare three cornerstone insulin-sparing strategies in T2D. Across diverse settings, GLP-1receptor agonists produced the greatest HbAlc reductions versus active comparators, with consistent weight loss and low hypoglycaemia risk, supporting their use when robust glycaemic lowering and weight management are priorities. SGLT2 inhibitors conferred dose-responsive, durable HbA 1c improvements versus placebo, aligning with their broader metabolic and potential cardiorenal advantages and making them strong options where durability and risk modification are emphasized. Metformin XR matched the glycaemic efficacy of IR while improving tolerability and convenience, reinforcing metformin's role as foundational therapy and a compatible partner in combination regimens.

Signals from a pragmatic, cluster-stratified comparison suggest phenotype-informed selection—

particularly favoring GLP-1 RAs for insulin-deficient profiles—may further enhance outcomes and help delay or minimize insulin exposure. Methodological strengths include PRISMA-guided selection, standardized arm-level extraction, and prespecified subgroup and sensitivity analyses; limitations include moderate heterogeneity, a limited number of poolable head-to-head trials, and incomplete variance reporting in some studies.

Clinically, these findings endorse personalized, insulin-sparing algorithms: initiate or maintain metformin (prefer XR when tolerability/adherence matter), escalate to GLP-1 RAs for maximal HbA Ic and weight effects, and consider SGLT2 inhibitors for durable glycaemia with metabolic and cardiorenal profiles. Future research should prioritize adequately powered GLP-1 vs SGLT2 head-to-head RCTs with phenotype stratification, longer follow-up, and consistent variance reporting to refine comparative effectiveness and implementation at scale.

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