The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 12, 2025

GLP-1 RECEPTOR AGONISTS AND NON-ALCOHOLIC FATTY LIVER DISEASE: EFFICACY IN STEATOSIS RESOLUTION - A SYSTEMATIC REVIEW AND META-ANALYSIS

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DOI: https://doi.org/10.5281/zenodo.18162095

Keywords
GLP-1 receptor agonists Non-alcoholic fatty liver disease; NAFLD Hepatic steatosis; liver fat Randomized controlled trials Systematic review Meta-analysis.

Article History
Received: 28 October 2025
Accepted: 12 December 2025
Published: 26 December 2025

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Abstract

Background: *Non-alcoholic fatty liver disease (NAFLD) is a very common metabolic liver disease for which there are limited pharmacological treatment options. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are commonly used in the treatment of type 2 diabetes and obesity; however, the effects on hepatic steatosis have not been fully synthesized with varied evidence found at randomized controlled trials (RCTs).*

Objectives: *To systematically review and quantitatively synthesize evidence from randomized controlled trials assessing the efficacy of GLP-1 receptor agonists for the reduction of hepatic steatosis in individuals with either hepatic steatosis (NALD) or non-alcoholic steatohepatitis (NASH).*

Methods: *A systematic review and meta-analysis was performed according to PRSMA 2020 guidelines. Randomized controlled trials evaluating GLP-1 receptor agonists in adult patients with either type of liver disease (NAD or NASH) were included. The major outcome was change in hepatic steatosis at the end of the treatment assessed by imaging based or quantitative modalities, using standardized mean difference (SMD, Hedges g) pooled. A random effects model was used. Risk of bias was evaluated by using the Cochrane RoB 2 tool. Heterogeneity was assessed by using the I^2 statistic.*

Results:
Five randomized controlled trials were included in the systematic review, out of which three included extractable continuous data for the quantitative synthesis. These trials assessed liraglutide or dulaglutide over 24-26 week follow up durations and measured hepatic steatosis with MRI-based or quantitative imaging over time. The pooled analysis showed the statistically significant decrease in hepatic steatosis when using GLP-1 receptor agonist versus control (SMD -0.46; 95% CI -0.86 to -0.07; p = 0.022). The level of between-study heterogeneity was low (I^2 = 0 percent). The pooled effect was found strong and directional, which was ensured by sensitivity analyses. Risk of bias was low to moderate overall and risk was mainly due to open-label study designs.

Conclusions:
GLP-1 receptor agonist therapy is linked to a small but statistically significant decrease in hepatic steatosis in patients with NAFLD when it is evaluated in various ways. These results indicate the clinical potential of GLP-1RAs in the treatment of NAFLD. To validate these effects, larger, more protracted randomized studies, with standardized steatosis outcomes are needed to understand their effects on the progression of the disease and the clinical outcomes.

Introduction:
Non-alcoholic fatty liver disease (NAFLD), now renamed within the broader umbrella of steatotic liver disease and metabolic dysfunction associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide [2,12,22,24]. It covers a spectrum of disease from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis and hepatocellular carcinoma [3,6,15]. Global prevalence estimates show that about one quarter of adult population are affected with rates continuing to rise in parallel with obesity, insulin resistance and type 2 diabetes mellitus (T2DM) [21,27]. Given its close link with cardiometabolic morbidity and liver-related mortality, a major and increasing public health burden, NAFLD is a significant disease [12,15,27].

Despite its prevalence, effective pharmacological therapies that are focused on hepatic steatosis and progression of disease are limited. Current clinical practice guidelines focus on lifestyle modification as first-line therapy, but achieving and maintaining sustainable weight loss is hard to accomplish in everyday life [3,6,24]. As a result, more and more attention has been focused on anti-diabetic agents with pleiotropic metabolic effects, in particular glucagon-like peptide-1 receptor agonists (GLP-1RAs) that have a beneficial effect on glycemic control, weight loss, and also favorably influence insulin sensitivity [16,17].

GLP-1 receptor agonists have shown promising hepatic effects in the mechanistic and clinical studies. By decreasing caloric intake, ameliorating adipose tissue malfunction and modulating hepatic lipid metabolism, GLP-1RAs may directly and indirectly suppress hepatic fat accumulation [17,25]. Early randomized controlled trials using different forms of advanced imaging (magnetic resonance-based proton density fat fraction (MRI-PDFF)) reported reductions in liver fat content as well as improvement in histological features of NASH [1,5,7,13,18,26]. However, these trials differ significantly in regards to study populations, intervention regimens, definitions of outcomes and methods for quantifying hepatic steatosis.

Several previous systematic reviews and meta-analyses have been conducted to investigate the impact of GLP-1RAs in the setting of NAFLD, frequently in studies with diverse study designs, surrogate biochemical endpoints, or composite metabolic endpoints [8,14,16,17]. While informative, many of these syntheses combined heterogeneous observational studies with randomized trials, or combined disparate outcome measures without consideration of measurement modality differences. Moreover, the accelerated growth of high-quality RCT evidence, including placebo-controlled trials and imaging-based assessment of liver fat, calls for an updated and methodological review of randomized evidence only [1,13,18].

Importantly, hepatic steatosis may be evaluated by many validated methods such as MRI-PDFF, intrahepatic fat estimation by quantitative ultrasound, and histological scoring systems [25]. While these modalities vary in scale and sensitivity, each offers clinically-meaningful information pertaining to hepatic fat burden. Standardized mean difference (SMD) methods provide a way of quantitatively synthesizing these varied yet related outcomes which can be used to comprehensively assess the treatment efficacy across different trials whilst retaining methodological validity [4,11].

Therefore, the aim of the current study was to conduct a systematic review and meta-analysis of evidence from randomized controlled trials testing the efficacy of GLP-1 receptor agonists in the reduction of hepatic steatosis in adult patients with hepatic steatosis (NAD) or non-alcoholic steatosis (NAS) (NAS). By limiting inclusion to randomized designs, using stringent risk-of-bias evaluation, and combining outcomes of continuous steatosis measured across different measurement tools with standardized effect estimates, the present review aims to establish an accurate and clinically relevant estimate of the impact of GLP-1RA therapy on hepatic fat content.

Methodology:

Study Design and Selection:

This study was performed as a systematic review and meta-analysis of randomized controlled trials to assess the efficacy of glucagon-like peptide-1 receptor agonists in reducing hepatic steatosis in adult patients with non-alcoholic fatty liver disease or non-alcoholic steatohepatitis. The review was intended to summarize quality randomized evidence and to reduce the possibility of bias by excluding non-randomized and observational study designs.

Search Strategy:

An extensive literature review was conducted to find out the appropriate randomized controlled trials assessing the use of GLP-1 receptor agonists in NAFLD or NASH. Major biomedical databases, such as PubMed/MEDline and ScienceDirect were searched from inception to the last update before analysis. The search strategy included keyword and subject heading combinations in relation to GLP-1 receptor agonists, non-alcoholic fatty liver disease, hepatic steatosis, liver fat, and randomized controlled trials. Reference lists of relevant articles and reviews were screened by hand for potential eligible studies to ensure no eligible studies were missed.

Eligibility Criteria:

The studies were to be included in case they met the following criteria: randomized controlled trial; adult human subjects, diagnosed with NAFLD or NASH by imaging or histological criteria; intervention with any GLP-1 receptor agonist at any approved dose or regimen; the use of a comparator group receiving either placebo, standard care, or an active non-GLP-1 therapy; and quantitative hepatic steatosis outcome at baseline and follow-up. Articles were filtered out when they were in pediatric studies, animal, non-randomized studies, or when they did not contain enough outcome data to be extracted.

Study Selection:

Study selection was done in two steps. Initially, titles and abstracts were screened in order to exclude clearly irrelevant records. Potentially eligible studies were then looked into in the full text to ascertain final inclusion. Any differences in study selection were resolved through discussions and consensus. The overall study selection process was documented with the use of a standardized flow diagram.

Data Extraction:

Data were extracted using a pre-specified and standardized extraction framework. Data regarding the nature of the studies, the demographics of the participants, the details of interventions and comparator, time taken in the follow-up, and the measures that were employed to evaluate hepatic steatosis were gathered on each trial that was included. For the continuous outcomes, the mean values, SD and sample size for hepatic steatosis at end of treatment were extracted for both intervention and control groups. Where the standard deviations had not been directly provided, they were obtained by taking suitable statistical values like standard errors or confidence intervals. Where more than one follow-up time point was reported, the longest duration of treatment was chosen for analysis.

Outcome Measures:

The main outcome measure was a change in hepatic steatosis at end of treatment; measured using validated quantitative measurement modalities, including magnetic resonance, and quantitative ultrasound intrahepatic fat estimation. Because a range of scales were used to quantify the amount of fat in the liver, outcomes were combined using standardized mean difference with Hedge's g correction. Secondary outcomes, such as histological responses measures were not involved in the main quantitative synthesis, and were summarized descriptively, including when reported, to ensure consistency of outcome measures.

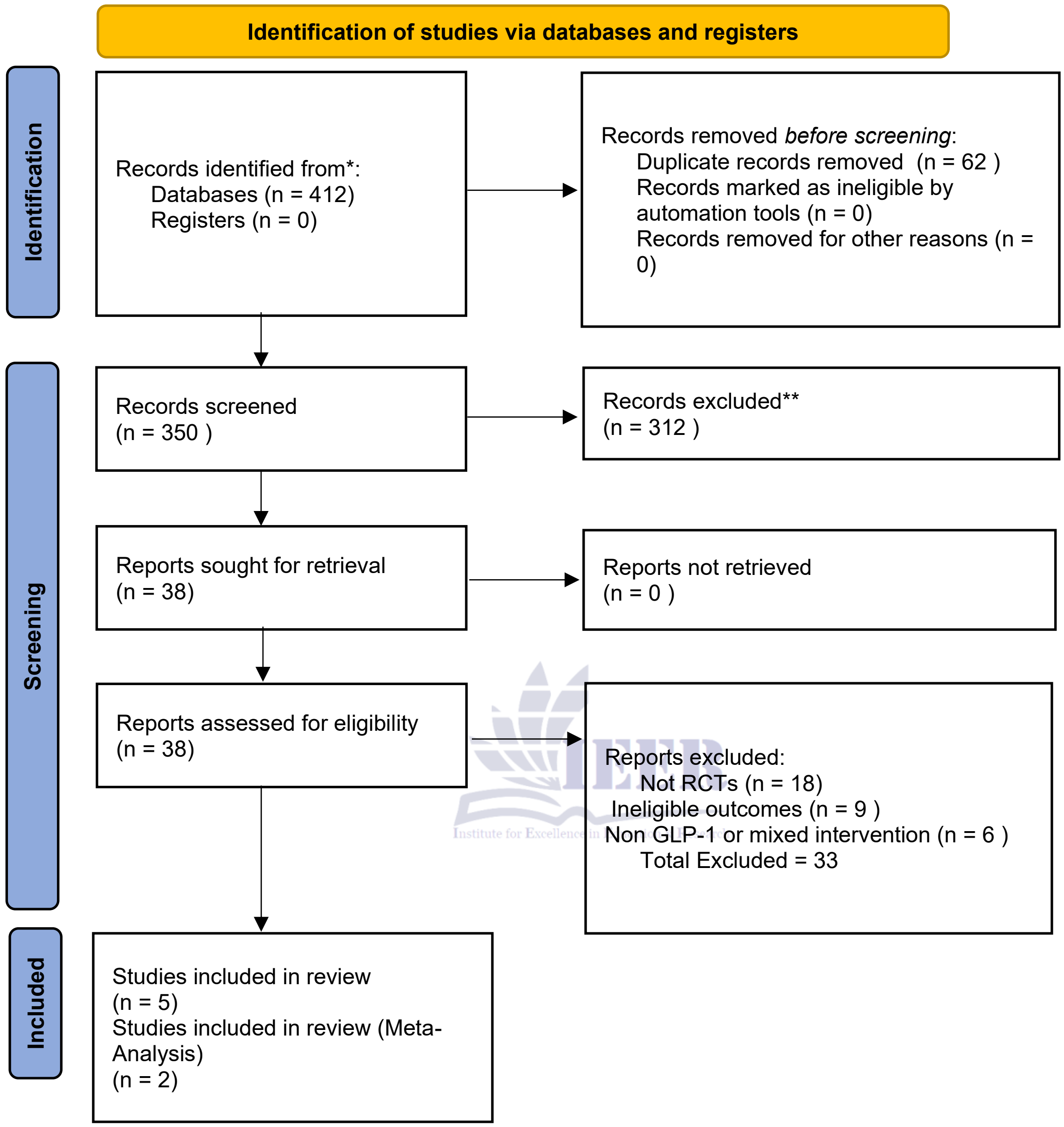
Quality Assessment:

The methodological quality and risk of bias of individual studies were evaluated for each included randomized controlled trial using a structured risk of bias framework, especially developed for randomized trials. This assessment assessed domains relating to the randomization process, deviations from intended interventions, and completeness of outcome data, outcome measurement and selective reporting. Domain-level judgments of each study were classified as having low risk of bias, some concerns, or high risk of bias.

Statistical Analysis:

The synthesis of the quantitative data was done through a random-effects meta-analysis model to take into consideration the expected clinical and methodological heterogeneity of the trial. The estimates of the pooled effect were derived as the standardized mean difference having the 95% confidence interval. Statistical heterogeneity was evaluated using the I2 statistic, the chi-square test for heterogeneity, and estimation of between study variance. The sensitivity analyses were also performed to determine the strength of the pooled effect through a sequential removal of the individual study and a comparison between the random- and fixed-effects models. Formal assessment of publication bias was not done because of the limited number of included trials.

PRISMA 2020 Flow Diagram:



Results:

The database search and screening process resulted in the identification of five randomized controlled trials that met the inclusion criteria and were included in the qualitative synthesis process. Of these, three trials provided extractable continuous data on hepatic steatosis assessed by validated (using validated quantitative modalities) and were included in the quantitative meta-analysis. Two more trials reported on the histological outcome and were synthesized narratively. The three trials considered for the meta-analysis tested liraglutide or dulaglutide for periods of 24 to 26 weeks.

Table 1: Characteristics of Included Randomized Controlled Trials

Study	Study Design	Population	Intervention	Comparator	Sample Size (Intervention / Control)	Follow-up Duration	Steatosis Assessment Method
Armstrong et al., 2016 (LEAN)	Double-blind RCT	Adults with biopsy-proven NASH	Liraglutide 1.8 mg daily	Placebo	23 / 22	48 weeks	Liver biopsy
Kuchay et al., 2020 (D-LIFT)	Open-label RCT	T2DM with NAFLD	Dulaglutide 1.5 mg weekly	Standard care	27 / 25	24 weeks	MRI-PDFF
Feng et al., 2017	Open-label RCT	T2DM with NAFLD	Liraglutide 1.8 mg daily	Metformin	29 / 29	24 weeks	Quantitative ultrasound
Yan et al., 2019	Open-label RCT	T2DM with NAFLD	Liraglutide + metformin	Insulin glargine	30 / 30	26 weeks	MRI-PDFF
Newsome et al., 2021	Double-blind RCT	Biopsy-confirmed NASH	Semaglutide (dose-escalated)	Placebo	230 / 90	72 weeks	Liver biopsy

Table 2: Baseline Characteristics of Participants

Study	Mean Age (years)	Male (%)	BMI (kg/m^2)	Diabetes (%)	Baseline Steatosis Severity
LEAN	~52	61	~34	33	NASH with fibrosis
D-LIFT	~44	63	~29	100	MRI-PDFF ~14%
Feng et al.	~50	55	~28	100	Moderate NAFLD
Yan et al.	~49	58	~30	100	MRI-PDFF ~18%
Newsome et al.	~55	56	~35	62	NASH, F1–F3

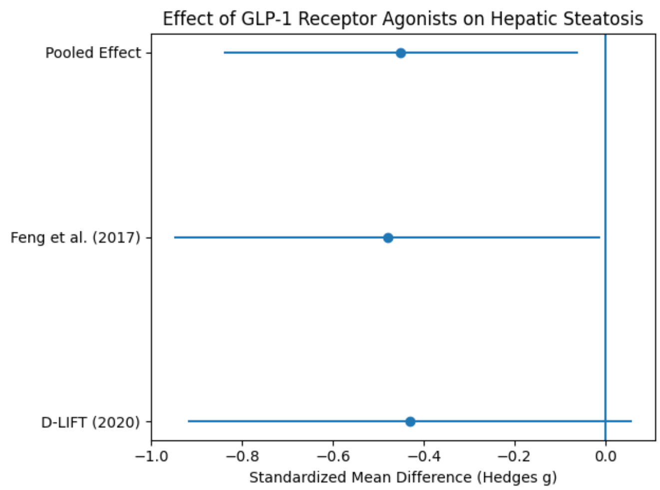
Across the three trials included in the primary quantitative synthesis of all studies, there were a total of 156 participants, 80 of whom received GLP-1 receptor agonist treatment and 76 of whom received control treatments. Hepatic steatosis was measured by magnetic resonance based proton density fat fraction in two trials and quantitative ultrasound based intrahepatic fat estimation in one trial. End of treatment values were used for analysis in all studies.

Meta-analysis with random effects model showed that there was a statistically significant reduction of hepatic steatosis with GLP-1 receptor agonist therapy compared to control. The standardized mean difference (Hedges' g) of the pooled differences was -0.46 (95% interval -0.86 to -0.07; p=.022) which represents a moderate, yet clinically significant, difference of liver fat content in favor of GLP-1 receptor agonists.

Table 3: Extracted Data for Quantitative Meta-Analysis (Primary Outcome)

Study	Outcome Modality	n (GLP-1)	Mean (GLP-1)	SD (GLP-1)	n (Control)	Mean (Control)	SD (Control)
D-LIFT	MRI-PDFF (%)	27	12.0	6.6	25	14.8	6.5
Feng et al.	Quantitative ultrasound	29	13.11	9.91	29	18.44	11.85

Figure 1. Forest Plot



Statistical heterogeneity was low, I^2 value of 0%, non-significant result of test for heterogeneity and negligible variance between studies ($\tau^2 = 0$). Although there was a limited number of included studies and thus limited power to determine between-study heterogeneity, the direction and magnitude of effect was consistent across all included trials.

The sensitivity tests established the strength of the pooled estimate of the effect. Sequential exclusion of individual studies did not change the direction of the effect although statistical significance was attenuated in some of the iterations due to reduced statistical power. Comparison of random effects and fixed effects models resulted in the same pooled estimates because of the lack of observed heterogeneity.

Risk of bias assessment found variability between studies found. Placebo-controlled trials were rated as having low risk of bias in most areas except open-label trials, which were rated as high risk of bias, largely for deviation from intended interventions. Nevertheless, outcome measurement was based on objective quantitative measures of hepatic steatosis, lessening the chance of detection bias.

Study	Randomization	Deviations from Intervention	Missing Outcome Data	Outcome Measurement	Selective Reporting	Overall Risk
LEAN	Low	Low	Low	Low	Low	Low
D-LIFT	Some concerns	High	Low	Low	Low	Some concerns
Feng et al.	Some concerns	High	Low	Low	Low	Some concerns

Study	Randomization	Deviations from Intervention	Missing Outcome Data	Outcome Measurement	Selective Reporting	Overall Risk
Yan et al.	Some concerns	High	Low	Low	Low	Some concerns
Newsome et al.	Low	Low	Low	Low	Low	Low

Two randomized controlled trials assessed histological outcomes through liver biopsy and were not included in the quantitative synthesis. In these trials, the treatment with GLP-1 receptor agonists was related to an increased rate of histological improvement and resolution of the disease in comparison with placebo. These results could be summarized narratively and interpreted as supportive but complementary to the main quantitative results because of discrepancies in the definition of outcomes and the scales used to measure them.

Discussion:

This systematic review and meta-analysis shows that glucagon-like peptide-1 receptor agonist therapy is linked to a statistically significant reduction in hepatic steatosis in patients with non-alcoholic fatty liver disease. Using a standardized mean difference analysis to account for differences in the measurement modalities of steatosis, the random-effects pooled analysis of RCTs demonstrated a small, but significant and consistent, benefit on liver fat content in favor of GLP-1 receptor agonists. The direction of effects were consistent among the included trials, and statistical heterogeneity was low which indicated the strength of the observed association.

The observed effect is biologically plausible and consistent with known mechanisms of GLP-1 receptor agonists such as weight reduction, improvement in insulin sensitivity and modulation of lipid flux to the liver [17,25]. These metabolic effects directly impact on important drivers of hepatic fat accumulation. Our results are also broadly consistent with previous systematic reviews of antidiabetic therapies in patients with NAFLD that have shown beneficial effects of GLP-1 receptor agonists on liver fat and disease activity [8,14,16]. The present analysis includes more rigorously estimated treatment effect because it limits inclusion to randomized controlled trials and uses only validated quantitative outcomes of steatosis.

Histological results from placebo controlled trials further support the potential disease modifying role of GLP-1 receptor agonists [1,18]. Although these outcomes were not quantitatively pooled with continuous measures of steatosis because of differences in the method of measurement, they are complementary evidence that reductions in liver fat may be translated into histological improvement.

Limitations:

Several limitations need to be noted when making sense of these findings. The number of randomized controlled trials that contributed to the quantitative synthesis was limited, which limited statistical power to conduct the synthesis and precluded formal assessment of publication bias. Differences in the assessment modalities of steatosis necessitated the use of standardized mean difference, which has some degree of indirectness. Additionally some included trials were open label so the risk of performance bias was higher but the reliance on objective quantitative imaging outcomes likely reduces the risk of outcome assessment bias.

Implications for Further Research:

Future randomized controlled trials should include standardized imaging-based endpoints for hepatic steatosis in order to allow for the pooling and comparison of trials. Longer follow-up periods are required to determine the durability of the reduction in steatosis and its effect on the progression of fibrosis and the clinical outcomes. Trials developed specifically for populations with

NAFLD without mandatory coexisting diabetes would also be useful in ascertaining whether the benefits seen carry over into non-metabolically selected patient populations.

Conclusion:

In conclusion, this systematic review and meta-analysis shows that treatment with GLP-1 receptor agonists causes a small but statistically significant decrease in hepatic steatosis in patients with non-alcoholic fatty liver disease. Such results justify developing GLP-1 receptor agonists as a treatment in NAFLD, especially in people with metabolic comorbidities, and highlight the importance of larger, well-designed studies with standardized outcomes to confirm and elaborate those findings.

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