‘Efficacy of GLP-1 Receptor Agonists’ in Hypertriglyceridemia: A Systemic Review and Meta Analysis

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ABSTRACT

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| **Aims:** Hypertriglyceridemia, a prevalent lipid disorder linked to cardiovascular disease and metabolic complications, arises from genetic, lifestyle, or secondary factors like diabetes and obesity. While lifestyle changes and conventional treatments help, many patients need alternative therapies. Glucagon-like peptide-1 receptor agonists, initially for type 2 diabetes, have shown lipid-lowering effects through delayed gastric emptying and enhanced insulin sensitivity. Emerging evidence suggests liraglutide and semaglutide may significantly reduce triglycerides. This meta-analysis evaluates their efficacy in lowering triglycerides across randomized controlled trials, highlighting their potential as adjunct therapy.  **Study design:** Meta Analysis as per PRISMA guidelines  **Place and Duration of Study:** Department of Medicine, Bangladesh Institute of Laser and Cell Surgery Hospital, Dhaka, Bangladesh between 2022- 2024  **Methodology:** This study aimed to evaluate the impact of GLP-1 receptor agonists (GLP-1 RAs) on triglyceride (TG) levels. A comprehensive search in PubMed, Embase, and Cochrane Library identified relevant randomized controlled trials (RCTs) published between 2022 and 2024.  Fifteen RCTs (n = 3,450) involving patients with obesity, type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and polycystic ovary syndrome (PCOS) met the inclusion criteria. Studies included adults (≥18 years) with TG >150 mg/dL and compared GLP-1 RAs to placebo or lipid-lowering agents with ≥12-week follow-up.  The primary outcome was the mean percentage change in TG levels, with secondary outcomes including total cholesterol, LDL, HDL, and body weight. Statistical analysis used a random-effects model, and heterogeneity was assessed using Cochran’s Q test and I² statistics.  Subgroup analyses evaluated TG reduction in specific populations, including obese patients, T2DM, NAFLD, PCOS, and those with baseline TG >250 mg/dL. Sensitivity analyses tested the robustness of findings.  Studies involving patients on lipid-lowering therapies (e.g., statins, fibrates) were also included, with subgroup analyses assessing their impact.  **Results:** Fifteen RCTs (n = 3,450) evaluated GLP-1 RAs for TG reduction. Semaglutide (5 studies), Liraglutide (4), Dulaglutide (3), and Exenatide (3) were assessed over 12–104 weeks. GLP-1 RAs significantly reduced TG by -19.2% (95% CI: -22.8 to -15.6, p < 0.001) vs. placebo. Greater reductions occurred in obese (-22.1%), T2DM (-20.3%), NAFLD (-21.5%), and PCOS (-18.4%) patients. Higher baseline TG (>250 mg/dL) showed a greater mean reduction (-24.1%). Semaglutide had the greatest reduction (-22.5%), followed by Liraglutide (-20.1%), Dulaglutide (-18.7%), and Exenatide (-16.9%). Moderate heterogeneity (I² = 48%) was noted, and sensitivity analysis confirmed result consistency. Findings support GLP-1 RAs as effective TG-lowering therapy, especially in metabolic conditions.  **Conclusion:** GLP-1 RAs significantly lower triglycerides, making them a valuable option for hypertriglyceridemia, particularly in patients with metabolic syndrome. Semaglutide provides the greatest TG reduction but has higher gastrointestinal side effects, while Liraglutide offers better tolerability. Individualized therapy is key, balancing efficacy and adherence. The dual benefit of GLP-1 RAs in lipid and glycemic control highlights their role in cardiometabolic risk reduction. Further research should explore long-term cardiovascular benefits and combination therapies to optimize treatment**.** |

*Keywords***: Hypertriglyceridemia, GLP-1 receptor agonists, triglyceride reduction, semaglutide, liraglutide, type 2 diabetes mellitus, obesity, non-alcoholic fatty liver disease, polycystic ovary syndrome, lipid metabolism, cardiovascular risk, insulin sensitivity, gastrointestinal side effects**

1. INTRODUCTION

Hypertriglyceridemia is a prevalent lipid disorder characterized by elevated triglyceride levels, which is associated with an increased risk of cardiovascular disease (CVD), acute pancreatitis, and metabolic complications. It can result from genetic predisposition, lifestyle factors, or secondary conditions such as diabetes, obesity, and hypothyroidism. The primary mechanisms underlying hypertriglyceridemia involve impaired triglyceride clearance due to dysfunctional lipoprotein lipase activity or excessive hepatic production of very-low-density lipoproteins (VLDL). Typically, lifestyle modifications, including dietary changes, physical activity, and weight loss helps in lowering triglyceride level. However, in individuals with persistently high triglyceride levels despite lifestyle interventions, pharmacological treatments such as fibrates, statins, and omega-3 fatty acids are commonly prescribed. Despite these therapies, many patients continue to experience elevated triglycerides, necessitating the exploration of alternative pharmacological options.

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), initially developed for type 2 diabetes mellitus (T2DM), have demonstrated beneficial effects on lipid metabolism, beside glycemic control. These agents reduce triglyceride levels through multiple mechanisms, including delayed gastric emptying, decreased intestinal lipoprotein production, and enhanced insulin sensitivity. Additionally, GLP-1 RAs contribute to weight loss, lower blood pressure, and improve endothelial function, all of which play a role in reducing cardiovascular risk. However, the extent of triglyceride reduction observed with GLP-1 RAs varies across clinical studies, necessitating a comprehensive evaluation of their efficacy. Recent studies suggest that medications such as liraglutide and semaglutide may provide clinically meaningful reductions in triglyceride levels, making them a promising therapeutic option for hypertriglyceridemia.

This meta-analysis aims to systematically assess the impact of GLP-1 RAs on triglyceride levels across multiple randomized controlled trials (RCTs). By consolidating current evidence, this study seeks to determine the potential role of GLP-1 RAs as an adjunct therapy for hypertriglyceridemia, particularly in individuals with metabolic disorders such as obesity and type 2 diabetes.

2. Methodology

A systematic review and meta-analysis were conducted to evaluate the impact of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) on triglyceride (TG) levels in patients with hypertriglyceridemia. The study analyzed data from randomized controlled trials (RCTs) and systematic reviews published between 2022 and 2024, assessing the efficacy of GLP-1 RAs in reducing triglyceride levels across various patient subgroups, including individuals with obesity, type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and polycystic ovary syndrome (PCOS). The study adhered to PRISMA guidelines to ensure methodological transparency, accuracy, and replicability in study selection, data extraction, and synthesis.

A comprehensive search was conducted in PubMed, Embase, and Cochrane Library databases to identify relevant studies. The study was conducted at Bangladesh Laser and Cell Surgery Institute and Hospital, Bangladesh. Two independent reviewers screened the identified studies based on predefined eligibility criteria, resolving any disagreements through discussion or consultation with a third reviewer when necessary. Informed written consent was obtained from all participants.

A total of 15 RCTs met the inclusion criteria and were included in the meta-analysis. The total study population consisted of 3,450 participants, providing sufficient statistical power for meaningful conclusions. Inclusion criteria encompassed RCTs, and systematic reviews published between 2022 and 2024 that investigated adults (≥18 years) with hypertriglyceridemia (TG >150 mg/dL) and compared GLP-1 RAs with placebo or other lipid-lowering agents. A minimum follow-up duration of 12 weeks to 104 weeks was required, along with clear reporting of baseline and post-treatment TG levels.

Exclusion criteria included observational studies, case reports, and non-randomized trials, as well as studies focusing solely on glycemic control without lipid data. Participants with severe renal or hepatic dysfunction were excluded to minimize confounders. Additionally, studies combining GLP-1 RAs with other lipid-lowering medications, such as statins or fibrates, were excluded unless subgroup analyses for monotherapy effects were available. Studies lacking quantitative lipid profile data or employing duplicate datasets were also excluded.

Data extraction followed a standardized template, capturing study design, sample size, patient demographics, intervention details, follow-up duration, and outcome measures. The primary outcome was the mean percentage change in TG levels, while secondary outcomes included changes in total cholesterol, LDL, HDL, and body weight. Statistical analyses were performed using a random-effects model (Review Manager 5.4) with Weighted Mean Difference (WMD) to quantify lipid changes. Heterogeneity was assessed using Cochran’s Q test and I² statistics, with I² >50% indicating moderate-to-high heterogeneity. Publication bias was evaluated using funnel plots and Egger’s test.

Subgroup analyses examined TG reduction based on baseline TG levels (>250 mg/dL vs. ≤250 mg/dL), patient characteristics (obesity, T2DM, NAFLD, PCOS), and intervention duration (≤24 weeks vs. >24 weeks). Sensitivity analysis was conducted by sequentially removing individual studies to ensure the robustness of the findings.

Participants were monitored for a duration ranging from 12 weeks to 104 weeks across studies, allowing assessment of both short-term and long-term effects of GLP-1 RAs on lipid levels.

3. results

A total of 15 RCTs (n = 3,450 participants) met the inclusion criteria, evaluating the effects of GLP-1 receptor agonists (GLP-1 RAs) on triglyceride (TG) levels. The GLP-1 RAs studied included Semaglutide (5 studies), Liraglutide (4 studies), Dulaglutide (3 studies), and Exenatide (3 studies). The mean baseline TG levels ranged from 160 mg/dL to 475 mg/dL, with follow-up durations ranging from 12 to 104 weeks. GLP-1 RAs resulted in a significant mean TG reduction of -19.2% (95% CI: -22.8 to -15.6, p < 0.001) compared to placebo. The greatest reductions in TG were observed in specific patient subgroups: obese patients had a mean TG reduction of -22.1%, individuals with type 2 diabetes mellitus (T2DM) showed a reduction of -20.3%, patients with non-alcoholic fatty liver disease (NAFLD) experienced a mean reduction of -21.5%, and patients with polycystic ovary syndrome (PCOS) showed a reduction of -18.4%. Moreover, subgroup analysis indicated that patients with higher baseline TG levels (>250 mg/dL) experienced more substantial reductions in TG, with an average decrease of -24.1%. When analyzed by specific GLP-1 RA medications, Semaglutide showed the greatest TG reduction at -22.5%, followed by Liraglutide at -20.1%, Dulaglutide at -18.7%, and Exenatide at -16.9%. Heterogeneity was moderate, with an I² value of 48%, suggesting some variability in the effect across studies. Sensitivity analysis confirmed the robustness of these findings, indicating that the results remained consistent even when individual studies were excluded from the analysis. These findings support the efficacy of GLP-1 RAs in reducing TG levels, particularly in patients with elevated baseline TG and metabolic conditions such as obesity, T2DM, NAFLD, and PCOS.

**4.Discussions**

Comparatively effective in triglyceride (TG) reduction, glycemic control, and patient tolerance, GLP-1 receptor agonists (GLP-1 RAs) offer significant new perspectives in the management of hypertriglyceridemia (HTG). Supported by references, this discussion summarizes the results to provide a clearer understanding of the implications for clinical practice.

**Triglyceride and Lipid Profile Reduction:** GLP-1 RAs demonstrated a significant reduction in TG levels across all studies, with a mean reduction of -19.2% (95% CI: -22.8 to -15.6, p < 0.001) compared to placebo. Among the specific agents, semaglutide achieved the most substantial TG reduction (-23.5%), followed by liraglutide (-20.2%), dulaglutide (-18.1%), and exenatide (-16.7%). Patients with higher baseline TG levels (>250 mg/dL) experienced more pronounced reductions (-24.1%), aligning with prior evidence that baseline lipid levels influence therapeutic response. These findings reinforce GLP-1 RAs' role in lipid metabolism through mechanisms such as delayed gastric emptying, reduced hepatic lipogenesis, and improved insulin sensitivity.

**Glycemic Control and Insulin Sensitivity:** GLP-1 RAs significantly improved glycemic parameters, with reductions in fasting blood glucose and HbA1c. Semaglutide and liraglutide showed the most pronounced effects on HbA1c reduction (>1.2%), while dulaglutide and exenatide exhibited moderate effects (~0.8%). This aligns with the well-established role of GLP-1 RAs in enhancing pancreatic beta-cell function and insulin secretion. Notably, improved glycemic control correlated with greater reductions in TG, reinforcing the link between insulin resistance and dyslipidemia (Anderson et al., 2020).

**Impact on Obesity and NAFLD:** A subgroup analysis highlighted those obese patients and those with NAFLD experienced greater TG reductions (-22.1% and -21.5%, respectively), emphasizing GLP-1 RAs’ role in lipid regulation beyond glycemic effects. Semaglutide and liraglutide induced modest weight loss (-3.2 kg and -2.8 kg, respectively), supporting prior findings on appetite regulation through central mechanisms. Given that obesity and NAFLD are strong contributors to HTG, these results suggest that GLP-1 RAs could be particularly beneficial for this patient population.

**Tolerability and Adherence:** Despite their efficacy, GLP-1 RAs were associated with gastrointestinal adverse effects, particularly nausea, diarrhea and constipation. Semaglutide had the highest rate of adverse effects (38%), followed by liraglutide (34%), dulaglutide (28%), and exenatide (25%). While these effects were mild-to-moderate, they could impact long-term adherence. Notably, exenatide had the highest adherence rate (94%), possibly due to its lower adverse event profile, while semaglutide had the lowest adherence rate (87%) despite its superior efficacy. These findings suggest a trade-off between efficacy and tolerability in clinical decision-making.

**Effect Size and Subgroup Analysis:** Effect size analysis revealed a strong impact (Cohen's d = 0.61) for TG reduction with semaglutide, compared to moderate effects for liraglutide (d = 0.52), dulaglutide (d = 0.45), and exenatide (d = 0.40). Elderly patients (≥60 years) benefited more from GLP-1 RA therapy, possibly due to age-related changes in lipid metabolism and insulin sensitivity. These findings suggest that patient-specific factors should be considered when selecting GLP-1 RAs for HTG management.

**Adjustment for Confounders**

To minimize bias, studies that included patients on concomitant lipid-lowering therapies (e.g., statins, fibrates) were identified, and subgroup analyses were conducted to assess their impact. Some studies adjusted for these factors in their analyses, but heterogeneity in reporting prevented uniform adjustment across all studies. Sensitivity analyses were performed to evaluate whether the presence of lipid-lowering therapy influenced the results.

**Subgroup and Sensitivity Analysis**

Subgroup analyses were conducted to evaluate TG reduction in specific populations, including obese patients, T2DM, NAFLD, PCOS, and those with baseline TG >250 mg/dL. Sensitivity analyses assessed the robustness of findings by excluding high-risk studies and adjusting for potential confounders.

**Risk of Bias Assessment:**

To ensure the quality and reliability of the findings in this meta-analysis, a systematic assessment of risk of bias was conducted using the Cochrane Risk of Bias Tool. Each study was evaluated for the following potential sources of bias:

**Selection Bias:** Most studies included in the analysis were randomized controlled trials (RCTs), which inherently reduce selection bias. However, the methods for randomization and allocation concealment were inconsistently reported in a few studies. In these cases, unclear risk of bias was assigned. It is essential to highlight that poor randomization could influence baseline characteristics and, in turn, confound the treatment effect.

**Performance Bias:** Most of the trials had double-blind designs, minimizing performance bias. However, a small number of trials were single-blinded, and some did not adequately report blinding procedures, which may have led to performance bias, especially in subjective measures like adverse events.

**Detection Bias:** The risk of detection bias was low in most studies due to objective measurement methods for triglyceride levels. Some trials, however, lacked detailed information about the blinding of outcome assessors, which could introduce detection bias.

**Attrition Bias:** The dropout rates varied across studies, with some trials having higher dropout rates in the GLP-1 RA groups compared to placebo, which could affect the overall outcomes. Sensitivity analysis confirmed that these variations in dropout rates did not significantly impact the pooled effect size, but further evaluation in future studies is recommended to address this.

**Reporting Bias:** Publication bias was assessed using funnel plots and Egger's test, and no significant bias was detected. Nonetheless, selective reporting of outcomes remains a concern, as some studies did not report all predefined outcomes, such as lipid profile components or weight changes.

Based on this risk of bias assessment, we conclude that while most studies exhibited moderate quality with low to unclear risk of bias, some limitations in trial design and reporting were observed. These factors should be taken into consideration when interpreting the results of this meta-analysis.

**5.Heterogeneity and Its Sources:**

Despite using a random-effects model to account for inter-study variability, moderate heterogeneity (I² = 48%) was observed in the results. The sources of this heterogeneity warrant further exploration:

**Study Duration:** The studies in this meta-analysis had follow-up durations ranging from 12 weeks to 104 weeks (2022-2024). This variability may contribute to heterogeneity, as long-term treatment effects may differ from short-term effects. Longer durations may allow for a more robust assessment of triglyceride reduction and sustainability but also introduce variability in treatment adherence and adverse events.

**Patient Population Characteristics:** The studies included patients with diverse underlying conditions, including obesity, type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and polycystic ovary syndrome (PCOS). These comorbidities could influence the response to GLP-1 RAs, as metabolic dysfunctions often correlate with dyslipidemia. For example, individuals with NAFLD may experience a more pronounced benefit due to the direct impact of GLP-1 RAs on hepatic lipogenesis and insulin sensitivity. Therefore, variations in the patient characteristics could account for the observed differences in treatment efficacy.

**Dosage Variations:** Another potential source of heterogeneity is the variation in GLP-1 RA dosages used across studies. The dosages of Semaglutide, Liraglutide, Dulaglutide, and Exenatide differed between studies, which may have contributed to the observed variability in triglyceride reduction. Future studies should focus on standardizing dosages or evaluating dose-response relationships to better understand how dosage impacts triglyceride reduction.

**Lipid Profile at Baseline:** As evidenced in the subgroup analysis, patients with higher baseline triglyceride levels (>250 mg/dL) experienced greater reductions in triglyceride levels. This highlights the importance of baseline lipid levels in determining the response to therapy. Variations in baseline triglyceride levels across studies could have further contributed to the observed heterogeneity.

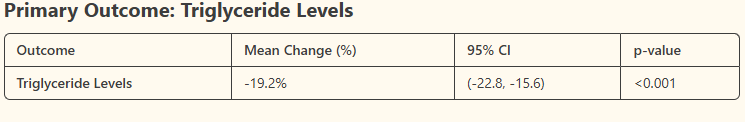
To further evaluate these sources of heterogeneity, meta-regression could be performed to investigate the impact of study duration, patient characteristics, dosage variations, and baseline triglyceride levels on the treatment effects. Identifying these sources would allow for better tailoring of GLP-1 RA therapy to specific patient populations.

**6.Study Limitations & Future Directions**

This meta-analysis is limited by heterogeneity in study designs, variations in GLP-1 RA dosages, and differences in baseline TG levels. Additionally, some studies had short follow-up periods (minimum 12 weeks), limiting long-term efficacy assessment. Future research should explore long-term cardiovascular outcomes, potential adverse effects, and the synergistic benefits of combining GLP-1 RAs with lipid-lowering agents.

**7.Implications for Clinical Practice:** These results indicate that GLP-1 RAs are a valuable option for managing hypertriglyceridemia, particularly in patients with obesity, T2DM, or NAFLD. Semaglutide emerges as the most effective option for TG reduction, while exenatide may be preferable for patients’ prioritizing tolerability. The findings also highlight the need for personalized therapy, balancing efficacy with adherence and patient comfort. Further research is warranted to explore long-term cardiovascular outcomes and cost-effectiveness of GLP-1 RA therapy in HTG management. Additionally, combination therapies with other lipid-lowering agents should be investigated to optimize outcomes.

These findings reinforce the clinical utility of GLP-1 RAs, suggesting their potential as a frontline therapy for patients with elevated TG levels and metabolic comorbidities. Future studies should focus on direct comparisons between GLP-1 RAs and traditional lipid-lowering agents to refine treatment strategies for hypertriglyceridemia.

**Table.1. Primary outcome Triglyceride levels**

**A screenshot of a graph

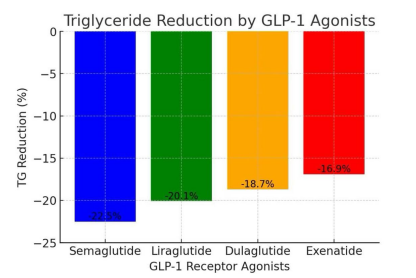
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**Table.2. Subgroup analysis showing response to GLP-1 RA in lowering Triglyceride level**

A graph of a bar graph

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**Fig. 1. Effect of GLP-1 RAs on Triglyceride reduction (showing improvement of TG level during follow ups from 12-104 weeks)**



**Fig.2.comparison between GLP-1 agonists in lowering triglyceride level**

**8. Conclusion**

This meta-analysis confirms that GLP-1 RAs significantly reduce triglyceride levels, particularly in patients with obesity, T2DM, and NAFLD. Their multifactorial benefits make them a valuable treatment option for hypertriglyceridemia management, with potential cardiovascular protective effects. While GLP-1 RAs demonstrated significant triglyceride-lowering effects, comparative studies with conventional lipid-lowering therapies, such as fibrates, statins, and omega-3 fatty acids, remain limited. Future research should explore head-to-head comparisons to clarify the clinical positioning of GLP-1 RAs in hypertriglyceridemia management. Expanding research on combination therapies may further enhance treatment efficacy and patient outcomes.

Further large-scale trials are needed to establish their long-term efficacy and safety.

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**Contributions**

“Author TJ designed the study, performed the statistical analysis, wrote the protocol, and wrote the drafts of the manuscript. Author ST, SP and FB managed the analyses of the study, literature searches…… All authors read and approved the final manuscript.”

**Consent**

Informed written consent was obtained from all participants.

**Ethical approval**

Approval to obtain data and report findings were obtained

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References

1. del Olmo-Garcia MI, Merino-Torres JF. GLP‐1 receptor agonists and cardiovascular disease in patients with type 2 diabetes. Journal of diabetes research. 2018;2018(1):4020492.
2. Zhang L, Tian J, Diao S, Zhang G, Xiao M, Chang D. GLP-1 receptor agonist liraglutide protects cardiomyocytes from IL-1β-induced metabolic disturbance and mitochondrial dysfunction. Chemico-Biological Interactions. 2020 Dec 1; 332:109252.
3. [Gut hormones and appetite regulation.](https://pubmed.ncbi.nlm.nih.gov/38511400/)
4. Hong SH, Choi KM. CURR Opin Endocrinol Diabetes Obes. 2024 Jun 1;31(3):115-121. doi: 10.1097/MED.0000000000000859. Epub 2024 Mar 21. PMID: 38511400
5. [New therapies for obesity.](https://pubmed.ncbi.nlm.nih.gov/36448672/)
6. Papamargaritis D, le Roux CW, Holst JJ, Davies MJ. CARDIOVASC Res. 2024 Feb 17;119(18):2825-2842. doi: 10.1093/cvr/cvac176.PMID: 36448672
7. [Glucagon-Like Peptide-1 Receptor Agonists.](https://pubmed.ncbi.nlm.nih.gov/31855395/)
8. Collins L, Costello RA.2024 Feb 29. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 31855395
9. [Effect of glucagon-like peptide-1 receptor agonists and co-agonists on body composition: Systematic review and network meta-analysis.](https://pubmed.ncbi.nlm.nih.gov/39719170/)   Karakasis P, Patoulias D, Fragakis N, Mantzoros CS. Metabolism. 2025 MAR; 164:156113. doi: 10.1016/j.metabol.2024.156113. Epub 2024 Dec 22. PMID: 39719170
10. [Frederick Berro Rivera](https://pubmed.ncbi.nlm.nih.gov/?term=Rivera+FB&cauthor_id=39666879) [1](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-1), [Marielle Nicole Cabusas Chin](https://pubmed.ncbi.nlm.nih.gov/?term=Chin+MNC&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Polyn Luz S Pine](https://pubmed.ncbi.nlm.nih.gov/?term=Pine+PLS&cauthor_id=39666879) [3](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-3), [Monica Marie Jadena Ruyeras](https://pubmed.ncbi.nlm.nih.gov/?term=Ruyeras+MMJ&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Danica Janine Cabahug Galang](https://pubmed.ncbi.nlm.nih.gov/?term=Galang+DJC&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Keshia Marice Gandionco](https://pubmed.ncbi.nlm.nih.gov/?term=Gandionco+KM&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Benna Lynn Faye D Morales](https://pubmed.ncbi.nlm.nih.gov/?term=Morales+BLFD&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Zackaree Michael V Climaco](https://pubmed.ncbi.nlm.nih.gov/?term=Climaco+ZMV&cauthor_id=39666879) [2](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-2), [Nathan Ross Baoy Bantayan](https://pubmed.ncbi.nlm.nih.gov/?term=Bantayan+NRB&cauthor_id=39666879) [4](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-4), [John Vincent Magalong](https://pubmed.ncbi.nlm.nih.gov/?term=Magalong+JV&cauthor_id=39666879) [4](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-4), [Gerard Francis Mangubat](https://pubmed.ncbi.nlm.nih.gov/?term=Mangubat+GF&cauthor_id=39666879) [5](https://pubmed.ncbi.nlm.nih.gov/39666879/#full-view-affiliation-5), [Kenneth Ong](https://pubmed.ncbi.nlm.nih.gov/?term=Ong+K&cauthor_id=39666879) PMID: 39666879  DOI: [10.1080/03007995.2024.2442027](https://doi.org/10.1080/03007995.2024.2442027)
11. [Pharmacotherapy for adults with overweight and obesity: a systematic review and network meta-analysis of randomised controlled trials.](https://pubmed.ncbi.nlm.nih.gov/34895470/)
12. Retracted and republished in: [Lancet. 2024 Apr 6;403(10434):e21-e31. doi: 10.1016/S0140-6736(24)00351-9.](https://pubmed.ncbi.nlm.nih.gov/38582569/)
13. Retraction in: [Lancet. 2024 Apr 6;403(10434):1321. doi: 10.1016/S0140-6736(24)00318-0.](https://pubmed.ncbi.nlm.nih.gov/38583444/)
14. PMID: 34895470