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Original Article

Beyond Weight Loss: Cardiovascular, Renal and Hepatic Effects of GLP-1 and Dual GIP/GLP-1 Receptor Agonists

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ABSTRACT

Glucagon-like peptide-1 (GLP-1) receptor agonists have evolved from agents for reducing glucose and regulating weight to become core treatment options for cardiometabolic disease. Dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists, such as tirzepatide, have further expanded the options. This narrative review aims to discuss the cardiovascular, renal and hepatic outcomes associated with GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists. Randomised outcomes trials have shown that some GLP-1 receptor agonists can reduce major adverse cardiovascular events in selected patient groups. Semaglutide has demonstrated cardiovascular benefits in patients with overweight/obesity and existing cardiovascular disease but no diabetes. It has also shown benefits to renal outcomes in patients with type 2 diabetes and chronic kidney disease. Tirzepatide has shown benefits in patients with obesity related heart failure with preserved ejection fraction and is non-inferior to dulaglutide in reducing major cardiovascular events in type 2 diabetes with atherosclerotic cardiovascular disease. There is significant evidence for liver benefits. Phase 2 data for tirzepatide and phase 3 interim data for semaglutide have demonstrated improvements in histological features of metabolic dysfunction-associated steatohepatitis. Potential mechanisms for these benefits include improved insulin sensitivity, reduced adiposity and ectopic fat, reduced blood pressure, altered lipid metabolism, anti-inflammatory effects and renal haemodynamic changes. However, it can be difficult to isolate direct effects on specific organs, and this evidence cannot always be generalised to all drugs/individuals. This review will highlight strengths and limitations of this evidence and priority areas for future investigation.

Keywords: GLP-1 receptor agonists, GIP, tirzepatide, Cardiovascular outcomes, chronic kidney disease, Metabolic dysfunction-associated steatohepatitis

INTRODUCTION

The incretin system is involved in modulating postprandial glucose regulation. Glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 are incretins. GIP is synthesised predominantly by intestinal K cells. GLP-1 is synthesised predominantly by intestinal L cells. Both GIP and GLP-1 increase glucose-dependent insulin secretion. GLP-1 also inhibits inappropriate glucagon secretion, reduces gastric emptying and increases satiety. These drugs were initially used in managing type 2 diabetes mellitus (T2DM) but have now been widely used due to their significant effects on weight loss and cardiometabolic risk. [1-4]

GLP-1 receptor agonists (GLP-1 RAs) such as liraglutide, semaglutide and dulaglutide have shown cardiovascular benefits in selected patient groups. Tirzepatide, a dual GIP/GLP-1 receptor agonist, has shown significant improvements in glycaemic control and weight loss. These drugs are increasingly being investigated for benefits beyond glycaemic control, including atherosclerotic cardiovascular disease, heart failure, chronic kidney disease (CKD) and metabolic dysfunction-associated steatotic liver disease (MASLD), including MASH.

"Pleiotropic" effects will be discussed in this review. This does not mean all benefits are independent of weight loss and glucose reduction. Some outcomes may be related to weight loss, improved insulin resistance, reduced blood pressure, improved lipid profile and decreased systemic inflammation. Other mechanisms may be due to receptor activation in target tissues. Careful consideration of randomised trials, mediation analysis and mechanistic studies should be made.

To provide a critical overview of the pharmacological basis for cardiovascular, renal and hepatic effects of

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GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists.

MATERIALS AND METHODS

A narrative review of published literature was performed. Search terms were used to find relevant articles from PubMed/MEDLINE and freely available journals. These included GLP-1 receptor agonist; GIP; tirzepatide; cardiovascular outcomes; major adverse cardiovascular events; heart failure; chronic kidney disease; renal outcome; MASLD; MASH; semaglutide; liraglutide; randomised trial. Priority was given to randomised controlled trials, pre-specified outcomes, systematic reviews, mechanistic studies and key trial results. Key studies were also considered up until 30 th October 2026.

This was not a systematic review. No protocol was registered, and no formal risk of bias tool was used. Data were not pooled quantitatively. A targeted search was performed. This means this manuscript should be viewed as a narrative review. The authors should replicate the search and ensure it is reproducible. All references should be checked against PubMed and the publishers. The search should be repeated just before submission.

3. Physiology and Pharmacological Basis of Disease

3.1 GLP-1 Physiology and Receptor Mechanism

GLP-1 is secreted after nutrient intake and acts on the GLP-1 receptor (class B G protein-coupled receptor). Activation of this receptor in pancreatic beta cells leads to increased cAMP signalling and improved glucose-dependent insulin secretion. GLP-1 also helps inhibit glucagon secretion in the presence of increased glucose. It can also reduce gastric emptying to variable extents and acts on central pathways responsible for appetite. The glucose-dependent nature of its insulinotropic effects means there is a relatively low risk of hypoglycaemia when using GLP-1 RAs without insulin or insulin secretagogues. [1-4]

Long-acting GLP-1 RAs vary in pharmacology and evidence for cardiovascular outcomes. Not all drugs will provide benefits, and this should not be assumed. Liraglutide, semaglutide and dulaglutide have all undergone large cardiovascular outcome trials. Evidence differs between drugs.

3.2 GIP Physiology and Dual Receptor Activation

GIP is an incretin hormone that improves glucose-dependent insulin secretion. GIP acts on many different tissues, including adipose tissue and bone. Depending on the context, GIP receptor activation can have differing effects. Tirzepatide is a single molecule designed to act as a dual agonist on the GIP and GLP-1 receptor. There is uncertainty over the extent to which each receptor will contribute to clinical benefits in humans. This means it is more appropriate to consider it a dual incretin receptor agonist.

This can provide benefits related to reducing appetite, improving glycaemic control and altering adipose tissue biology. However, the degree of weight loss alone will not show direct benefits on cardiovascular, renal or hepatic outcomes. Comparisons, biomarkers and mediation should be undertaken to see if this provides additional benefits beyond the expected weight loss.

3.3 Potential Mechanisms

Mechanisms include reduced visceral and ectopic adipose tissue, improved insulin sensitivity, reduced circulating glucose, slight reductions in systolic blood pressure, improved lipid metabolism, reduced oxidative stress, reduced inflammatory signals and improved endothelial function. In the kidneys, a decrease in albuminuria and changes to intraglomerular hemodynamics can help reduce progression of kidney disease. In the liver, reduced hepatic fat content and improved insulin sensitivity may help reduce lipotoxicity and hepatic injury. These are plausible mechanisms, but evidence can vary between outcomes and has not been fully confirmed in humans. [3,4,23]

4. Cardiovascular Outcomes

4.1 Atherosclerotic Cardiovascular Disease

Randomised outcome trials (CVOTs) have shown that certain GLP-1 RAs can reduce the risk of major adverse cardiovascular events (MACE). MACE usually refers to cardiovascular death, non-fatal MI and non-fatal stroke. Liraglutide reduced the primary composite cardiovascular endpoint in patients with T2DM and high cardiovascular risk (LEADER). Dulaglutide reduced MACE in a large patient group with a significant proportion without established cardiovascular disease (REWIND). Semaglutide has provided evidence of cardiovascular safety and efficacy in SUSTAIN-6. This trial was primarily a pre-approval safety study. [5-8,16,17]

The SELECT trial was particularly important. Semaglutide 2.4mg was investigated in patients with overweight/obesity, established cardiovascular disease and no diabetes. Semaglutide significantly reduced the incidence of cardiovascular death, non-fatal MI and stroke compared with placebo (HR 0.80, 95% CI 0.72-0.90). This supports the notion that cardiovascular benefits can occur in patients without diabetes. A pre-specified analysis also demonstrated that cardiovascular risk reduction was consistent across all baseline HbA1c and early HbA1c change categories. This suggests these benefits are not purely due to reductions in glycaemia. [8]

Tirzepatide was investigated in the SURPASS-CVOT trial. It was found to be non-inferior to dulaglutide in reducing MACE in patients with T2DM with established atherosclerotic cardiovascular disease. This was not statistically superior, but dulaglutide was an active comparator and has demonstrated cardiovascular benefits. This should not be seen as evidence that tirzepatide is superior for cardiovascular prevention. [13,14]

4.2 Heart Failure

Obesity can play a role in heart failure through hemodynamic overload, systemic inflammation, insulin resistance, epicardial adipose tissue, sodium retention in the kidneys and remodelling in the heart. Weight loss can improve symptoms, exercise tolerance and congestion in obesity related heart failure with preserved ejection fraction (HFpEF). In the SUMMIT trial, tirzepatide reduced the composite outcome of cardiovascular death or worsening heart failure in patients with HFpEF and obesity. This was largely due to fewer patients experiencing worsening heart failure. Improvements in health status were also demonstrated.

This supports the use of incretin-based therapy as a potential disease-modifying intervention in selected obese patients with HFpEF. This should not be generalised to all types of heart failure. The SUMMIT trial was in patients with obesity and preserved ejection fraction. More evidence is required in patients with reduced ejection fraction, non-obese associated HFpEF and in a wider patient population. Semaglutide has also demonstrated improvements in symptoms and health status in obesity related HFpEF.

4.3 Potential Mechanisms

Potential mechanisms include reduced visceral and pericardial adipose tissue, reduced blood pressure, improved endothelial function, improved post-prandial lipaemia, reduced inflammation and improved insulin sensitivity. The GLP-1 receptor may have effects on vascular and cardiac signalling. It is unclear how much direct effect will occur on the heart and blood vessels. Weight loss, renal function, glycaemia and blood pressure will all be changed at the same time, making it difficult to determine causality.

It is also important to highlight that a reduction in atherosclerotic events is different to improved symptoms or events in heart failure. Different pathophysiology and patient populations will be involved.

5. Renal Outcomes

5.1 Chronic Kidney Disease

T2DM, obesity and hypertension can all contribute to CKD. GLP-1 RAs may improve renal outcomes through improved glycaemic control, reduced blood pressure, weight loss, reduced albuminuria and potentially improved haemodynamics and anti-inflammatory properties in the kidneys. Older cardiovascular trials often reported renal secondary

outcomes. These were largely new onset macroalbuminuria and should not be considered as prevention of kidney failure.

The FLOW trial provided specific evidence of renal outcomes for semaglutide. Semaglutide reduced the risk of kidney failure, sustained significant decline in estimated glomerular filtration rate and kidney- or cardiovascular-related death. This represents a significant improvement over albuminuria outcomes. It shows semaglutide can provide clinically meaningful benefits to the kidneys in this patient group. [9,16,17]

However, FLOW does not show that all GLP-1 RAs will have similar effects on the kidneys. It also doesn't show they will provide the same magnitude of benefits in non-diabetic patients or across all stages of CKD. Renin-angiotensin system inhibitors and SGLT-2 inhibitors are key evidence based interventions for CKD. In practice, incretin therapy should be considered as part of holistic risk reduction.

5.2 Potential Mechanisms

Mechanisms include reduced hyperfiltration in the glomerulus, reduced blood pressure, natriuresis, improved endothelial function, reduced albuminuria, reduced inflammation and reduced metabolic stress. It is still unclear how significant these mechanisms are relative to each other in humans. Early changes in eGFR may represent adaptation to haemodynamic changes and should be interpreted in the right context. [3,9,16,17]

GI side effects, such as nausea, vomiting and diarrhoea, can cause volume depletion, particularly for patients on diuretic medications or with advanced CKD. Patients with significant GI symptoms should be assessed for hydration, renal function and concurrent medications. Appropriate dosing should be considered and local prescribing information consulted.

6. Hepatic Outcomes

6.1 MASLD and MASH

MASLD is a metabolic liver disease related to cardiometabolic risk factors. MASH is the inflammatory form showing steatosis, hepatocyte ballooning and lobular inflammation. Variable fibrosis can also be present. Fibrosis stage will largely determine long-term liver-related outcomes. Obese patients with MASLD will often have insulin resistance and T2DM, making them attractive candidates for incretin-based therapy.

GLP-1 RAs may reduce hepatic fat through weight loss, improved insulin sensitivity, reduced de novo lipogenesis and altered substrate delivery. Reduced lipolysis in adipose tissue may reduce free fatty acid delivery to the liver. Glycaemic and systemic inflammation improvements may also play a role. These are plausible mechanisms supported by clinical and translational evidence. However, improvements in aminotransferases and liver fat by imaging do not necessarily mean fibrosis has resolved.

6.2 Semaglutide

The phase 3 ESSENCE trial investigated semaglutide 2.4mg weekly in patients with biopsy-confirmed MASH and fibrosis stage F2/F3. At week 72, the prespecified interim analysis showed more patients had resolution of steatohepatitis without worsening fibrosis (62.9%) compared with placebo (34.3%). Patients also had greater improvements with ≥1 stage improvement in fibrosis without worsening steatohepatitis (36.8% vs 22.4%). This provides important histological evidence. However, this trial is ongoing, and long-term outcomes such as decompensation, transplant and liver-related death should be monitored. [11]

Weight loss was significant in this trial, meaning we cannot determine how much of the histological improvements are due to weight loss. It is also important to consider these are histological surrogate outcomes.

6.3 Tirzepatide and other Incretins

Tirzepatide improved resolution of MASH without worsening fibrosis compared with placebo in the phase 2 SYNERGY-NASH trial. This supports further development of dual GIP/GLP-1 receptor agonists for MASH. However, this was a phase 2 trial and should not be viewed as evidence from a completed phase 3 trial or evidence that cirrhosis is prevented long term. [12]

Multi-agonists such as GLP-1/glucagon and GLP-1/GIP/glucagon are being investigated for obesity and metabolic liver disease. They may provide benefits through their metabolic effects, but evidence is needed for their efficacy and safety. This should be investigated in well powered trials with histological and clinical outcomes. Any manuscript should mention that this is investigational.

7. Clinical Evidence Comparison

Below is a summary of key trials relevant to this review. Due to differences in patient populations, interventions and outcomes, care should be taken when comparing data between trials..

8. Safety Considerations

GI side effects are the most commonly reported adverse events associated with GLP-1 RAs and tirzepatide. This includes nausea, vomiting, diarrhoea, constipation and abdominal discomfort. These are often dose-related and may be more pronounced during dose escalation. Slow titration, diet advice and assessing persistent symptoms may improve tolerability. Discontinuation due to adverse events can be important, particularly with long-term use. [3,15,18]

Biliary and gallbladder issues have been reported with weight loss therapies using incretins. Rapid weight loss can increase gallstone formation making it difficult to isolate direct drug effects. Acute pancreatitis is a labelled safety concern for several of these drugs. Patients should be advised about persistent severe abdominal pain. Patients should be assessed if indicated. Specific contraindications and warnings should be reviewed for each drug including thyroid C-cell tumour warnings where applicable.

Hypoglycaemia risk is generally low when used as monotherapy but increases when used alongside insulin or sulfonylureas. Dose reductions to insulin/secretagogues may be required. Delayed gastric emptying can affect absorption of oral medications. This can be an issue for anaesthesia or deep sedation due to possible gastric contents. Current professional guidelines should be followed with individualised assessments.

Patients with CKD may experience dehydration due to vomiting/diarrhoea. Monitoring should be based on the baseline renal function, concurrent diuretics, renin-angiotensin system blockers and symptoms. Pharmacists can help by performing medication reviews, providing advice, identifying risk of dehydration and hypoglycaemia and monitoring treatment adherence and adverse events.

9. Clinical Pharmacy Implications

For patients with T2DM and CKD, pharmacists should help ensure that GLP-1 RA therapy is integrated with established kidney-protective interventions and that renal function, albuminuria, blood pressure and glycaemic targets are assessed appropriately. In obesity-related cardiovascular disease, treatment goals should include clinical outcomes and functional status rather than weight alone. In MASLD/MASH, treatment decisions should be coordinated with hepatology and diabetes teams, and clinicians should avoid interpreting changes in liver enzymes as a substitute for fibrosis assessment.

Pharmacovigilance is particularly important in populations using multiple medications due to multiple comorbidities. Suspected serious or unexpected adverse drug reactions can be reported to the appropriate national pharmacovigilance programme which increases post-marketing safety data.

10. Limitations of current evidence

The evidence presented has several limitations to be considered. Firstly, it is highly specific to individual molecules and so cardiovascular and renal benefits demonstrated for semaglutide/liraglutide/dulaglutide cannot be assumed to apply to all incretin-based drugs. Secondly, each trial had different populations with varying levels of diabetes, cardiovascular disease, kidney function, obesity and additional medication. Thirdly, many of the mechanisms are inferred from evidence such as biomarker data, animal models or post-hoc analysis. Therefore, no direct causality of any organ systems can be demonstrated.

Weight, glucose, blood pressure, lipids and inflammatory changes occur together. Mediation analysis can be used to explore this; however, this relies on assumptions. Finally, liver trials have focused on histological markers as their surrogate endpoint, and there is uncertainty around long-term outcomes such as cirrhosis, liver failure, transplant and mortality. Follow-up and stopping of medication may also affect outcomes. Many large drug development trials have been sponsored by industry. This should be taken into

consideration when assessing the design, analysis and reporting of trials.

11. Research gaps and future directions

Future studies should look to compare GLP-1 RA and dual GIP/GLP-1 RA head-to-head in patients with established cardiovascular/renal/hepatic disease. Trials should be designed with clinically relevant outcomes and include pre-specified mediation analysis to look at weight, glucose, and blood pressure changes. Mechanism studies using imaging, biomarkers and organ-specific approaches could help to identify whether outcomes are due to direct receptor action or secondary metabolic effects. [3,15,16]

In nephrology, more research is required in non-diabetic patients, patients with severe CKD, kidney transplant recipients and diverse CKD etiologies. In cardiology, more trials should be performed to show benefits across the various heart failure phenotypes. Long-term outcomes related to arrhythmias, atherosclerotic events and cardiovascular mortality should be evaluated. In hepatology, ongoing Phase 3 and outcomes trials should demonstrate that histological improvements lead to less cirrhosis, decompensation, hepatocellular carcinoma, liver transplant and liver-related mortality.

Studies should also look to see how long effects last once medication is stopped, what the ideal dose would be in the long term, availability and cost-effectiveness of the treatment, how well it will be adhered to and how equitable the treatment is. Real-world studies can be used to complement randomised trials; however, one must consider confounding by indication, healthy user bias and medication adherence.

Interpretation of evidence: There is good evidence for cardiovascular and renal benefits in specific patient populations and molecules with dedicated outcome trials. Evidence for liver benefits is promising however varies between molecules and stages of development. Histological surrogate markers should not be described as reducing liver failure and mortality. Evidence for cardiovascular benefits of tirzepatide should be described as non-inferiority compared to active control.

CONCLUSION

GLP-1 RAs and dual GIP/GLP-1 RAs have expanded the treatment options for cardiometabolic disease. Randomised data demonstrate cardiovascular benefits in specific GLP-1 RAs, renal benefits in those with T2DM and CKD taking semaglutide and benefits of tirzepatide in obesity driven HFpEF. Evidence is also progressing in the liver, with interim data from Phase 3 trials demonstrating benefits of semaglutide and Phase 2 trial data of tirzepatide in MASH. This shows that incretin-based drugs have a broad role to play in multiple organ systems. However, it is important not to overemphasise direct protection of each organ. Many benefits are likely to be partially mediated by weight loss and improving metabolic risk profiles. Evidence will vary between molecules and patient populations. More comparative and long-term outcome trials are required to demonstrate the longevity, mechanisms and place in practice.

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CONFLICT OF INTEREST

The author declares no competing interests.

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ETHICAL APPROVAL

Not applicable. This narrative review did not involve human participants, animals or identifiable personal data.

AUTHOR CONTRIBUTION

The author completed all the work.

REFERENCES

1. Holst JJ. The physiology of glucagon-like peptide 1. Physiol Rev. 2007;87 (4):1409-39. doi:10.1152/physrev.00034.2006.
2. Campbell JE, Drucker DJ. Pharmacology, physiology, and mechanisms of incretin hormone action. Cell Metab. 2013;17 (6):819-37. doi:10.1016/j.cmet.2013.04.008.
3. Drucker DJ. Mechanisms of action and therapeutic application of glucagon-like peptide-1. Cell Metab. 2018;27 (4):740-56. doi:10.1016/j.cmet.2018.03.001.

4. Nauck MA, Meier JJ. Incretin hormones: their role in health and disease. Diabetes Obes Metab. 2018;20 Suppl 1:5-21. doi:10.1111/dom.13129.
5. Marso SP, Daniels GH, Brown-Frandsen K, Kristensen P, Mann JFE, Nauck MA, et al. Liraglutide and cardiovascular outcomes in type 2 diabetes. N Engl J Med. 2016;375 (4):311-22. doi:10.1056/NEJMoa1603827.
6. Gerstein HC, Colhoun HM, Dagenais GR, Diaz R, Lakshmanan M, Pais P, et al. Dulaglutide and cardiovascular outcomes in type 2 diabetes (REWIND): a double-blind, randomised placebo-controlled trial. Lancet. 2019;394 (10193):121-30. doi:10.1016/S0140-6736 (19)31149-3.
7. Marso SP, Bain SC, Consoli A, Eliaschewitz FG, Jódar E, Leiter LA, et al. Semaglutide and cardiovascular outcomes in patients with type 2 diabetes. N Engl J Med. 2016;375 (19):1834-44. doi:10.1056/NEJMoa1607141.
8. Lincoff AM, Brown-Frandsen K, Colhoun HM, Deanfield J, Emerson SS, Esbjerg S, et al. Semaglutide and cardiovascular outcomes in obesity without diabetes. N Engl J Med. 2023;389 (24):2221-32. doi:10.1056/NEJMoa2307563.
9. Perkovic V, Tuttle KR, Rossing P, Mahaffey KW, Mann JFE, Bakris G, et al. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med. 2024;391 (2):109-21. doi:10.1056/NEJMoa2403347.
10. Packer M, Zile MR, Kramer CM, Baum SJ, Litwin SE, Menon V, et al. Tirzepatide for heart failure with preserved ejection fraction and obesity. N Engl J Med. 2025;392 (5):427-37. doi:10.1056/NEJMoa2410027.
11. Sanyal AJ, Newsome PN, Kliers I, Østergaard LH, Long MT, Kjær MS, et al. Phase 3 trial of semaglutide in metabolic dysfunction-associated steatohepatitis. N Engl J Med. 2025;392 (21):2089-99. doi:10.1056/NEJMoa2413258.
12. Loomba R, Hartman ML, Lawitz EJ, Vuppalanchi R, Boursier J, Kher U, et al. Tirzepatide for metabolic dysfunction-associated steatohepatitis with liver fibrosis. N Engl J Med. 2024;391 (4):299-310. doi:10.1056/NEJMoa2401943.
13. Frías JP, Davies MJ, Rosenstock J, Pérez Manghi FC, Fernández Landó L, Bergman BK, et al. Tirzepatide versus semaglutide once weekly in patients with type 2 diabetes. N Engl J Med. 2021;385 (6):503-15. doi:10.1056/NEJMoa2107519.
14. Del Prato S, Kahn SE, Pavo I, Weerakkody GJ, Kim SH, Kewchi A, et al. Tirzepatide versus insulin glargine in type 2 diabetes and increased cardiovascular risk (SURPASS-4): a randomised, open-label, parallel-group, multicentre, phase 3 trial. Lancet. 2021;398 (10313):1811-24. doi:10.1016/S0140-6736 (21)02188-7.
15. Nauck MA, Quast DR, Wefers J, Meier JJ. GLP-1 receptor agonists in the treatment of type 2 diabetes—state-of-the-art. Mol Metab. 2021;46:101102. doi:10.1016/j.molmet.2020.101102.
16. Kristensen SL, Rørth R, Jhund PS, Docherty KF, Sattar N, Preiss D, et al. Cardiovascular, mortality, and kidney outcomes with GLP-1 receptor agonists in patients with type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet Diabetes Endocrinol. 2019;7 (10):776-85. doi:10.1016/S2213-8587 (19)30249-9.
17. Zelniker TA, Wiviott SD, Raz I, Im K, Goodrich EL, Bonaca MP, et al. Comparison of the effects of glucagon-like peptide receptor agonists and sodium-glucose cotransporter 2 inhibitors for prevention of major adverse cardiovascular and renal outcomes in type 2 diabetes mellitus. Circulation. 2019;139 (17):2022-31. doi:10.1161/CIRCULATIONAHA.118.038868.
18. American Diabetes Association Professional Practice Committee. Pharmacologic approaches to glycemic treatment: Standards of Care in Diabetes—2025. Diabetes Care. 2025;48 (Suppl 1):S181-S206. doi:10.2337/dc25-S009.
19. Newsome PN, Buchholtz K, Cusi K, Linder M, Okanoue T, Ratziu V, et al. A placebo-controlled trial of subcutaneous semaglutide in nonalcoholic steatohepatitis. N Engl J Med. 2021;384 (12):1113-24. doi:10.1056/NEJMoa2028395.
20. Armstrong MJ, Gaunt P, Aithal GP, Barton D, Hull D, Parker R, et al. Liraglutide safety and efficacy in patients with non-alcoholic steatohepatitis (LEAN): a multicentre, double-blind,

randomised, placebo-controlled phase 2 study. Lancet. 2016;387 (10019):679-90. doi:10.1016/S0140-6736 (15)00803-X.

21. Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, Alves B, et al. Tirzepatide once weekly for the treatment of obesity. N Engl J Med. 2022;387 (3):205-16. doi:10.1056/NEJ-Moa2206038.
22. American Association for the Study of Liver Diseases. AASLD practice guidance on the clinical assessment and management of nonalcoholic fatty liver disease. Hepatology. 2023;77 (5):1797-835. doi:10.1097/ HEP.0000000000000323.
23. Ussher JR, Drucker DJ. Cardiovascular biology of the incretin system. Endocr Rev. 2012;33 (2):187-215. doi:10.1210/er.2011-1052.

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