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Open Access REVIEW

Semaglutide As a Multiorgan Therapeutic Agent: Evidence for Cardiovascular, Renal, and Hepatic Benefits

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ABSTRACT

Semaglutide, a long-acting glucagon-like peptide-1 receptor agonist (GLP-1 RA), is being used as a cardiometabolic treatment instead of a medication to reduce blood sugar. Major adverse cardiovascular events have decreased, diabetic kidney disease has progressed more slowly, obesity-related heart failure with preserved ejection fraction (HFpEF) symptoms have improved, and metabolic dysfunction-associated steatohepatitis (MASH) has improved histologically, according to large randomized trials. The pharmacology, suggested mechanisms, important clinical data, and safety profile of semaglutide across several organ systems are summarized in this review, which also identifies any gaps.

Keywords: Semaglutide; GLP-1 receptor agonist; cardiovascular outcomes; chronic kidney disease; MASH; safety; pharmacovigilance

INTRODUCTION

Semaglutide is a once-weekly GLP-1 RA that was modified from the natural incretin hormone to withstand enzymatic breakdown and stay in the bloodstream for roughly a week [1]. In the cardiovascular safety experiment SUSTAIN-6, it was initially licensed for type 2 diabetic mellitus (T2DM) and surprisingly demonstrated fewer cardiovascular events than placebo [2]· Then, higher doses were created for long-term weight control, resulting in an average weight loss of about 15% in overweight or obese adults [3]· Since then, outcome trials have demonstrated benefit in individuals with diabetic chronic kidney disease (CKD) [5], MASH with fibrosis [6], and obesity and preexisting cardiovascular disease without diabetes [4]· The cardiovascular-kidney-metabolic (CKM) syndrome theory, which views obesity, diabetes, CKD, and cardiovascular disease as a single interconnected disease process, aligns with these findings [7]. Additionally, NAFLD has been reclassified as metabolic dysfunction-associated steatotic liver disease (MASLD), whereas NASH has been dubbed as MASH [8].

Pharmacology and Molecular Basis of Semaglutide

After meals, intestinal L-cells release native GLP-1, which dipeptidyl peptidase-4 (DPP-4) breaks down in a matter of minutes [9]. Although semaglutide and human GLP-1 have roughly 94% sequence identity, there are three significant differences [1]:

- The substitution of α-aminoisobutyric acid for alanine at position 8 prevents DPP-4 cleavage.
- A spacer connects the C18 fatty diacid chain at lysine 26, allowing for robust albumin binding and a half-life of roughly one week.

• The substitution of arginine for lysine at position 34, allowing for a single acylation site. G-protein-coupled GLP-1 receptors are present in the kidney, gastrointestinal tract, brain (brainstem and hypothalamus), pancreatic islets, and vasculature [9][10]. Activation increases intracellular cyclic AMP, which results in decreased hunger, slower stomach emptying, glucagon suppression, and glucose-dependent insulin secretion [9].

Many of the organ-protective benefits listed below begin with weight loss, which is caused by these central and peripheral mechanisms. For T2DM, an oral formulation that is co-formulated with an absorption enhancer is also offered [11].

Mechanisms of Cardiovascular Protection

Semaglutide's cardiovascular benefits are probably caused by multiple overlapping routes rather than a single mechanism [9][10]

• Effects on weight and metabolism: long-term weight loss, reduced blood pressure, a healthier lipid profile, and improved glycemic control all lessen the total cardiometabolic burden [3][12]

• Anti-atherosclerotic and anti-inflammatory effects: semaglutide and liraglutide reduced the formation of lesions in mice models of atherosclerosis in a way that was not entirely explained by decreasing cholesterol and altering inflammatory pathways [13]

• Direct vascular effects: reduced systemic inflammation and enhanced endothelial function have been suggested; however, GLP-1 receptor expression in the heart and arteries is restricted, and the precise targets are still up for debate [9][10].

The splitting of event curves in SELECT started early, prior to the achievement of maximal weight loss, indicating that mechanisms other than weight reduction are involved [4]. The proportional contributions are still up for debate, and this is still a theory.

Figure 1 gives an overview of the proposed pathways linking semaglutide to cardiac, renal and hepatic effects.

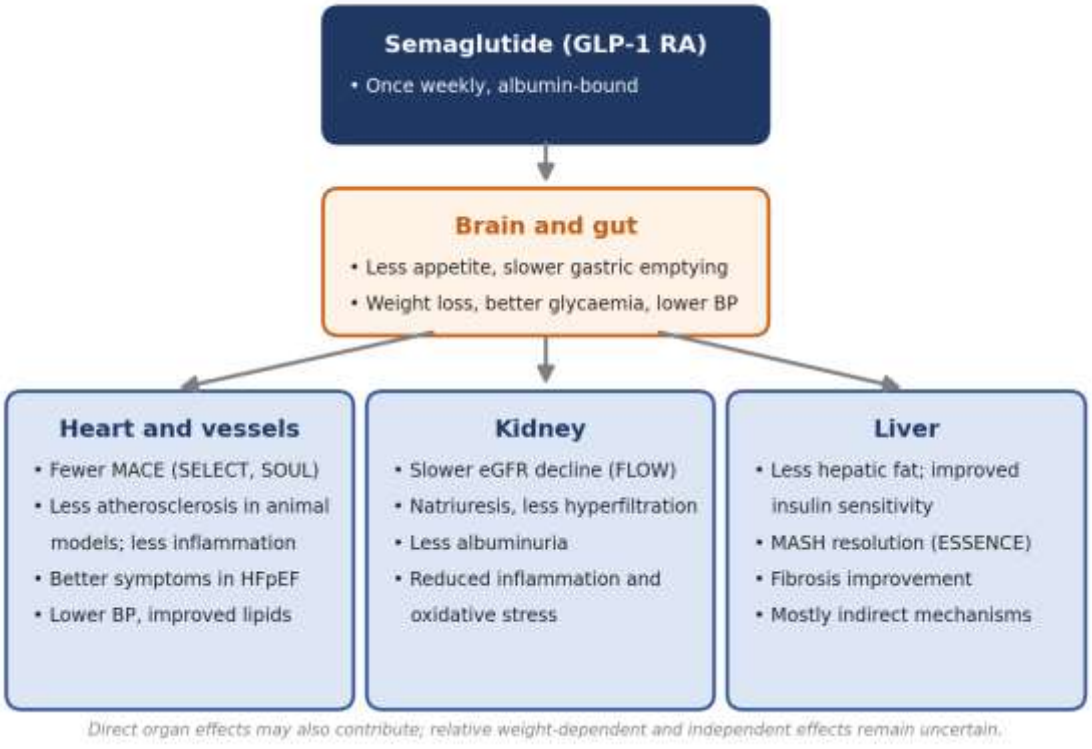


Fig 1. Overview of proposed pathways by which semaglutide influences the heart, kidney and liver.

Clinical Evidence in Atherosclerotic Cardiovascular Disease

3,297 T2DM individuals at high cardiovascular risk were randomized in SUSTAIN-6. Although complications from diabetic retinopathy were more common, subcutaneous semaglutide reduced the composite of cardiovascular death, non-fatal myocardial infarction, or non-fatal stroke (hazard ratio [HR] 0.74) [2]. For the same outcome (HR 0.79), PIONEER 6 demonstrated that oral semaglutide was not inferior to a placebo [11]. 17,604 adults 45 years of age or older who had established cardiovascular disease and were overweight or obese but did not have diabetes were included in SELECT. Semaglutide 2.4 mg decreased major adverse

cardiovascular events (MACE) from 8.0% to 6.5% (HR 0.80) throughout a mean follow-up of almost 40 months [4]. Long-term weight loss was sustained [12]. With a MACE decrease of almost 14% (HR 0.86), SOUL expanded the evidence to include oral semaglutide in high-risk T2DM [14].

These findings fit into the larger body of class evidence. A meta-analysis of GLP-1 RA studies in T2DM revealed lower rates of MACE, all-cause mortality, and renal outcomes [17]. Previous trials with liraglutide and dulaglutide also decreased MACE [15][16]. Table 1 summarises the principal semaglutide trials.

Table 1. Key randomised trials of semaglutide beyond glycaemic control

Trial	Population	Principal finding	Ref.
SUSTAIN-6	T2DM, high CV risk (n=3,297)	MACE lower with semaglutide (HR 0.74); more retinopathy complications	[2]
PIONEER 6	T2DM, high CV risk (n=3,183)	Oral semaglutide non-inferior for MACE (HR 0.79)	[11]
SELECT	Obesity and CVD, no diabetes (n=17,604)	MACE 6.5% vs 8.0% (HR 0.80)	[4]
SOUL	T2DM with CVD and/or CKD	Oral semaglutide lowered MACE (HR 0.86)	[14]
STEP-HFpEF	HFpEF and obesity (n=529)	Better KCCQ symptom score and 6-minute walk distance; greater weight loss	[18]
FLOW	T2DM with CKD (n=3,533)	24% lower risk of the primary kidney composite (HR 0.76)	[5]
ESSENCE	MASH with F2-F3 fibrosis	Higher rates of MASH resolution and fibrosis improvement (part 1)	[6]

CV, cardiovascular; CVD, cardiovascular disease; HR, hazard ratio; KCCQ, Kansas City Cardiomyopathy Questionnaire; MACE, major adverse cardiovascular events.

Heart Failure with Preserved Ejection Fraction

Increased epicardial fat, increased blood volume, and systemic inflammation all contribute to stiff, congested ventricles, and obesity is a primary cause of HFpEF [18]. Semaglutide 2.4 mg or a placebo was administered for 52 weeks to 529 HFpEF patients with a body mass index of 30 kg/m² or higher in STEP-HFpEF. Semaglutide increased weight reduction, improved the 6-minute walk distance, and provided bigger improvements in the Kansas City Cardiomyopathy Questionnaire clinical summary score [18].

Although weight loss was less than in individuals without diabetes, STEP-HFpEF DM replicated the strategy in individuals with T2DM and found comparable clinical and functional improvements [19]. These studies did not demonstrate a decrease in heart failure hospitalization or mortality on their own because they were focused on symptoms and function.

Figure 3 shows the main STEP-HFpEF findings.

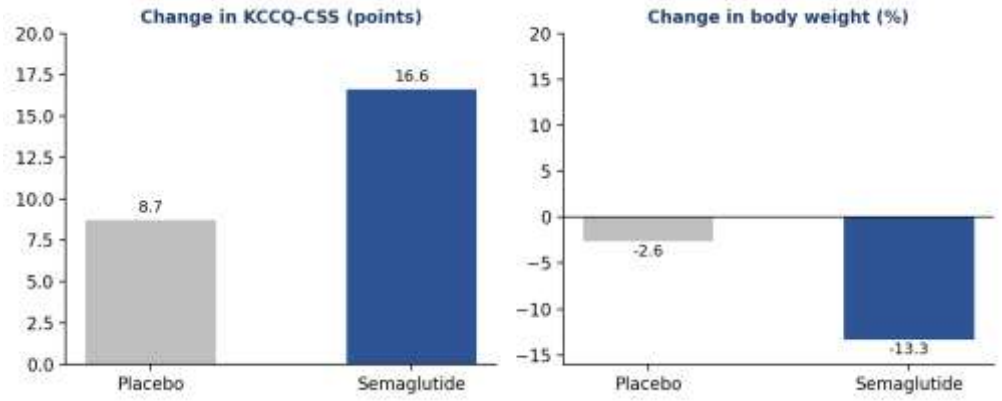


Fig 3. STEP-HFpEF at 52 weeks: change in KCCQ clinical summary score (KCCQ-CSS) and body weight with semaglutide 2.4 mg versus placebo.

Mechanisms of Renal Protection

In the kidney, GLP-1 receptors are mostly expressed in the glomerular structures and vasculature, with minimal expression in the proximal tubular cells. Renal mechanisms that are suggested include:

• Indirect effects: glomerular hyperfiltration and damage are lessened by weight loss, decreased blood pressure, and better glycemic management

· • Natriuresis: GLP-1 RAs increase sodium excretion, in part by decreasing the proximal tubule's sodium-hydrogen exchanger NHE3's activity

• Antioxidant and anti-inflammatory effects: decreased oxidative stress and inflammatory signalling in kidney tissue [20]. Liraglutide lowered a composite renal outcome in the LEADER trial, mostly due to a decrease in

new-onset persistent macroalbuminuria [21].

Clinical Evidence in Chronic Kidney Disease

The first GLP-1 RA renal outcomes trial was called FLOW. 3,533 adults with T2DM and CKD were enrolled; the majority were previously on renin-angiotensin system blockage. Semaglutide 1.0 mg decreased the primary composite of death from kidney or cardiovascular causes by 24% (HR 0.76), a prolonged decline in estimated glomerular filtration rate (eGFR) of at least 50%, or kidney failure. Due to efficacy, the trial was terminated early [5].

Secondary findings revealed reduced risks of major cardiovascular events and all-cause mortality, as well as a slower annual drop in eGFR [5]. The best combination with SGLT2 inhibitors and finerenone still needs to be determined in practice because FLOW was carried out in a population where a minority utilized SGLT2 inhibitors.

Liver: Mechanism and Evidence in Masld and Mash

The benefits of semaglutide for the liver are believed to be mostly indirect because hepatocyte GLP-1 receptor expression is modest and currently under discussion. They probably represent decreased systemic inflammation, decreased hepatic fat storage, increased insulin sensitivity, and weight loss [10][22].

Clinical evidence has evolved over time:

• Smaller GLP-1 RA research called LEAN (liraglutide) found that more patients experienced NASH resolution than those who received a placebo [23].

• Phase 2 semaglutide trial: 59% of patients with NASH resolved with semaglutide 0.4 mg daily, compared to 17% with a placebo; nevertheless, the improvement in fibrosis stage was not statistically significant [22].

• Compensated cirrhosis: once cirrhosis is established, there may be little benefit, according to a phase 2 trial that demonstrated no meaningful effect on fibrosis regression or NASH resolution [24].

• ESSENCE (phase 3, part 1): in MASH with stage 2 or 3 fibrosis, fibrosis improved without increasing steatohepatitis in 36.8% against 22.4% and steatohepatitis cleared in 62.9% on semaglutide 2.4 mg versus 34.3% on placebo [6].

In MASH with fibrosis, the thyroid hormone receptor-beta agonist resmetirom has also demonstrated histological benefit [25]. The impact of semaglutide on long-term liver outcomes, such as decompensation, transplantation, and liver-related death, is still to be determined in the later stages of ESSENCE because to the lack of head-to-head data [6].

Fig 4. compares the histological response rates in the phase 2 and phase 3 trials, and Table 2 summarises the proposed mechanisms in each organ.

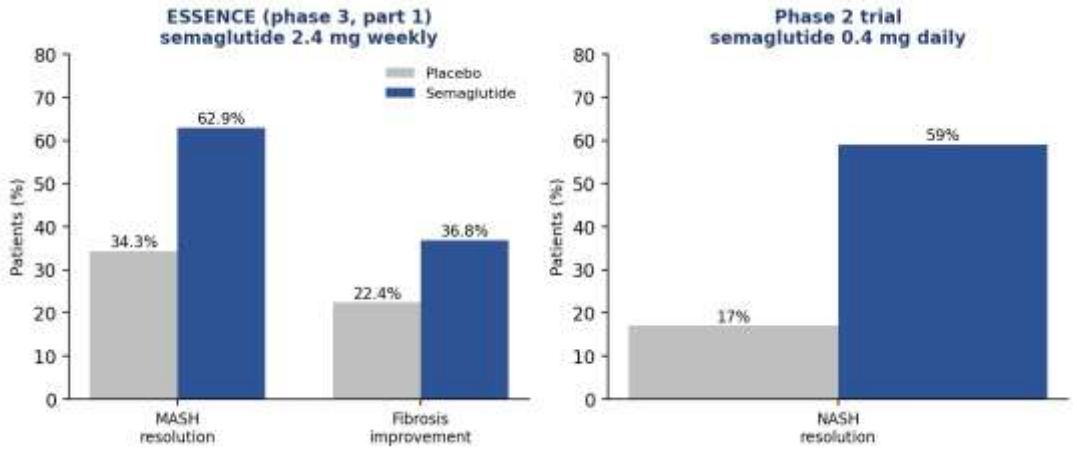


Fig 4. Histological response rates in the phase 2 semaglutide trial and part 1 of the phase 3 ESSENCE trial in MASH.

Table 2. Proposed mechanisms of semaglutide by organ system

Organ	Proposed mechanisms	Supporting evidence	Ref.
Heart and vessels	Weight loss; lower blood pressure and lipids; less inflammation; possible direct vascular effects	Early event-curve separation in SELECT; less atherosclerosis in mouse models	[4][13]
Kidney	Less glomerular hyperfiltration; natriuresis via NHE3; anti-inflammatory and antioxidant effects; blood pressure and glucose control	Slower eGFR decline in FLOW; less macroalbuminuria with liraglutide in LEADER	[5][20] [21]

Liver	Weight loss; better insulin sensitivity; less hepatic fat; limited direct hepatocyte GLP-1 receptor action	MASH resolution and fibrosis improvement in ESSENCE	[6][10]

eGFR, estimated glomerular filtration rate; NHE3, sodium-hydrogen exchanger 3.

Safety and Tolerability

The most frequent consequences are gastrointestinal. In STEP 1, semaglutide was more likely than placebo to cause nausea, diarrhoea, vomiting, and constipation. These side effects were primarily mild to moderate and occurred during dose escalation; around 4.5% of patients stopped their therapy as a result [3]. When compared to other weight-loss medication combinations, observational data indicates that GLP-1 RAs used for weight loss are associated with increased risks of pancreatitis, gastroparesis, and bowel obstruction [26].

Other safety considerations include:

• **Gallbladder disease:** GLP-1 RAs increase the risk of cholelithiasis and cholecystitis, especially in cases of fast weight reduction [3].

• **Diabetic retinopathy:** SUSTAIN-6 patients with pre-existing retinopathy had an increase of retinopathy problems, particularly with rapid glucose lowering [2].

• **Naion:** Individuals prescribed semaglutide had an increased risk of non-arthritic anterior ischemic optic neuropathy, according to a retrospective cohort study [27]. This signal has been examined by regulators, and the current label language needs to be modified.

• **Thyroid C-cell tumours:** Patients with a personal or family history of medullary thyroid carcinoma or MEN 2 should not use semaglutide; a boxed warning based on rodent research applies [1].

• **Hypoglycaemia:** Combining semaglutide with insulin or sulfonylureas increases the risk [2].

• **Peri-procedural aspiration:** guidelines on medication management prior to anesthesia or sedation have been motivated by delayed gastric emptying. Local advice ought to be adhered to.

Table 3 summarises the main safety issues and practical points.

Table 3. Main safety considerations with semaglutide

Adverse effect	Evidence	Practical consideration	Ref.
Nausea, vomiting, diarrhoea, constipation	Common in STEP 1; mostly mild to moderate during dose escalation	Gradual titration, small meals, adequate hydration	[3]
Pancreatitis, gastroparesis, bowel obstruction	Higher risk than with another weight-loss drug combination in observational data	Evaluate promptly if severe or persistent abdominal pain	[26]
Gallbladder disease	More frequent, especially with rapid weight loss	Counsel on biliary symptoms	[3]
Diabetic retinopathy complications	Excess seen in SUSTAIN-6	Eye screening in people with diabetes, especially with existing retinopathy	[2]
NAION	Higher risk in a retrospective cohort study	Report sudden vision loss; check current label wording	[27]
Thyroid C-cell tumours	Rodent data; boxed warning	Avoid with personal or family history of medullary thyroid carcinoma or MEN 2	[1]
Hypoglycaemia	Risk rises with insulin or sulfonylureas	Consider reducing the partner drug dose	[2]

NAION, non-arteritic anterior ischaemic optic neuropathy; MEN 2, multiple endocrine neoplasia type 2.

LIMITATIONS, FUTURE DIRECTIONS

Weight regains: Patients regain roughly two-thirds of their weight loss within a year of stopping semaglutide, and some cardiometabolic gains are reversed [28]. Treatment continuation sustains weight loss [29]. This lends credence to the idea that obesity is a chronic condition that calls for ongoing treatment.

Other emerging uses: Trials for sleep apnea, peripheral vascular disease, and neurodegeneration are ongoing, and semaglutide 2.4 mg improved pain and function in obese with knee osteoarthritis [30].

Open questions: The extent to which the benefit is weight-dependent, outcomes other than MASH histology, effects in individuals with advanced chronic kidney disease, long-term safety in real-world populations, and cost and access, particularly in low- and middle-income countries, are some of the major gaps. Tirzepatide and combination peptides, two more recent medicines that are being compared, have the potential to alter the landscape.

Role of the pharmacist: Through pharmacovigilance systems, clinical pharmacists can assist with patient selection, dose escalation counselling, side-effect management, peri-procedural medication guidance, and adverse drug reaction reporting.

CONCLUSION

Semaglutide has now been shown to reduce cardiovascular risk, protect the kidneys in diabetic CKD, improve symptoms in obesity-related HFpEF, and improve histology in MASH. Although gastrointestinal side effects are the most prevalent, it is generally well tolerated; however, it requires cautious patient selection and monitoring for less common safety signals.

Table 4 outlines a practical monitoring framework for pharmacists.

Table 4. Pharmacist checklist for patients on semaglutide

Stage	Key actions
Before starting	Confirm indication and contraindications (medullary thyroid carcinoma, MEN 2, pregnancy); review insulin and sulfonylurea use; check kidney function and, in diabetes, retinal status
Dose escalation	Follow the labelled titration schedule; advise small meals and hydration; manage nausea and constipation
During therapy	Monitor weight, glycaemia, kidney function and GI symptoms; watch for biliary and pancreatic symptoms; encourage adequate protein intake and physical activity
Special situations	Give peri-procedural advice according to local anaesthesia guidance; advise on stopping before planned pregnancy as per the label; counsel on dehydration risk during illness, especially in CKD
Reporting	Report suspected adverse drug reactions to the national pharmacovigilance system (for example PvPI in India)

CONFLICT OF INTEREST

Authors declare for none conflict of interest.

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