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Review Article

Ozempic (Semaglutide): Beyond Diabetes-An Emerging Role in Weight Loss

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Obesity and type 2 diabetes mellitus (T2DM) are among the most prevalent chronic metabolic disorders worldwide and they are associated with increased morbidity, mortality, and healthcare costs. Semaglutide, marketed as Ozempic for the treatment of Type2 DM and Wegovy for chronic weight management, is a long-acting glucagon-like peptide-1 receptor agonist (GLP-1 RA) that has transformed the management of metabolic disease. Initially developed to improve glycaemic control in patients with Type2 DM, semaglutide has demonstrated remarkable efficacy in promoting clinically significant and sustained weight loss, leading to its expanded role beyond diabetes management. Its mechanisms include glucose-dependent stimulation of insulin secretion, suppression of glucagon release, delayed gastric emptying, and central regulation of appetite through hypothalamic GLP-1 receptors, resulting in reduced energy intake and improved satiety. This review summarizes the current evidence regarding the pharmacology, mechanism of action, pharmacokinetic profile, clinical efficacy, safety, and emerging therapeutic applications of semaglutide in weight management. Major clinical evidence from the SUSTAIN and STEP trial programmes consistently demonstrates superior reductions in glycated haemoglobin (HbA1c), body weight, waist circumference, and cardiometabolic risk factors compared with placebo and several active comparators. Furthermore, findings from the SELECT cardiovascular outcomes trial suggest that semaglutide significantly reduces major adverse cardiovascular events in individuals with overweight or obesity, even in the absence of diabetes, highlighting its broader cardiometabolic benefits. Although gastrointestinal adverse effects such as nausea, vomiting, diarrhoea, and constipation are the most frequently reported limitations, semaglutide is generally well tolerated, with an acceptable long-term safety profile when used under appropriate clinical supervision. In addition to its established benefits in diabetes and obesity, emerging evidence suggests potential therapeutic roles for semaglutide in non-alcoholic fatty liver disease, chronic kidney disease, cardiovascular risk reduction, and other obesity-related comorbidities. Ongoing research is also investigating its utility in several metabolic and neurodegenerative disorders. Overall, semaglutide represents a major advancement in metabolic medicine by bridging diabetes treatment and obesity management. Future long-term studies focusing on durability of weight loss, cost-effectiveness, safety, and real-world clinical outcomes will further define its place in precision medicine and comprehensive chronic disease management.

Keywords: Semaglutide; Ozempic; GLP-1 receptor agonist; Obesity; Weight loss; Type 2 diabetes mellitus; STEP trials; SUSTAIN trials; Cardiovascular outcomes; Metabolic disease.

INTRODUCTION

Obesity and type 2 diabetes mellitus (T2DM) are among the most prevalent chronic non-communicable diseases worldwide and represent major public health challenges. The prevalence of both conditions has increased dramatically over the past few decades because of urbanization, sedentary lifestyles, unhealthy dietary habits, and genetic predisposition. Obesity is now recognized as a chronic, relapsing, multifactorial disease characterized by excessive adipose tissue accumulation that adversely affects metabolic, cardiovascular, renal, and musculoskeletal health. It significantly increases the risk of developing Type 2 DM, hypertension, dyslipidaemia, coronary artery disease, stroke, obstructive sleep apnoea, non-

alcoholic fatty liver disease (NAFLD), chronic kidney disease (CKD), and several types of cancer. Consequently, obesity contributes substantially to healthcare expenditure, reduced quality of life, and premature mortality worldwide (1–5). Lifestyle interventions, including dietary modification, increased physical activity, and behavioural therapy, remain the cornerstone of obesity management. However, these approaches often result in only modest weight loss, and long-term maintenance remains difficult because of adaptive physiological mechanisms that promote weight regain (6–8). These limitations have driven the search for more effective pharmacological therapies. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have emerged as one of the most significant therapeutic advances in the management of metabolic disorders. GLP-1 is an endogenous incretin hormone secreted by intestinal L-cells in response to food intake. It enhances glucose-dependent insulin secretion, suppresses glucagon release, delays gastric emptying, and acts on the hypothalamus to reduce appetite and increase satiety. These combined actions improve glycaemic control while simultaneously reducing energy intake and promoting weight loss (9–12). The favourable metabolic profile of GLP-1 receptor agonists has transformed the treatment paradigm for type 2 DM and has expanded their role into obesity management. Semaglutide, a long-acting GLP-1 receptor agonist developed by Novo Nordisk, represents one of the most potent agents within this drug class. Structural modifications, including amino acid substitution and acylation with a fatty diacid side chain, confer resistance to degradation by dipeptidyl peptidase-4 (DPP-4) and allow strong albumin binding, resulting in an extended half-life of approximately one week. This pharmacokinetic profile enables convenient once-weekly subcutaneous administration. Semaglutide is marketed as **Ozempic** for the treatment of type 2 diabetes mellitus, **Wegovy** for chronic weight management, and **Rybelsus** as the first orally administered GLP-1 receptor agonist for glycaemic control (13–16). Initially approved for improving glycaemic control in adults with type 2 DM, semaglutide rapidly gained attention because of its remarkable ability to induce clinically meaningful and sustained weight loss. Large-scale clinical trials consistently demonstrated reductions in glycated haemoglobin (HbA1c), body weight, waist circumference, blood pressure, and several cardiometabolic risk factors. Unlike conventional anti-obesity medications, semaglutide addresses multiple pathophysiological mechanisms involved in obesity by regulating appetite, reducing food cravings, improving insulin sensitivity, and decreasing caloric intake. These properties have established semaglutide as a promising therapeutic option for patients with obesity irrespective of diabetic status (17–20). The clinical efficacy of semaglutide has been extensively evaluated through the SUSTAIN and STEP clinical trial programmes. The SUSTAIN trials primarily investigated the effectiveness of semaglutide in patients with type 2 DM and consistently demonstrated superior reductions in HbA1c and body weight compared with placebo and other glucose-lowering therapies, while maintaining an acceptable safety profile (21–26). Building upon these findings, the Semaglutide Treatment Effect in People with Obesity (STEP) programme evaluated semaglutide 2.4 mg once weekly in adults with overweight or obesity, both with and without diabetes. Across STEP 1, STEP 2, STEP 3, STEP 4, STEP 5, and STEP 8, semaglutide produced unprecedented weight reductions of approximately 10–17% of baseline body weight, along with improvements in blood pressure, lipid profile, physical functioning, and health-related quality of life. Most adverse events were gastrointestinal in nature and were generally mild to moderate in severity (27–33). More recently, evidence has suggested that the therapeutic benefits of semaglutide extend beyond glycaemic control and weight reduction. The SELECT cardiovascular outcomes trial demonstrated that semaglutide significantly reduced the incidence of major adverse cardiovascular events in adults with overweight or obesity and established cardiovascular disease, even in individuals without diabetes. These findings support the concept that semaglutide exerts cardioprotective effects independent of glucose lowering and has broadened its potential clinical applications (34–36). Emerging evidence also indicates possible benefits in metabolic dysfunction-associated steatotic liver disease (formerly NAFLD), chronic kidney disease, heart failure, obstructive sleep apnoea, and other obesity-related disorders, although further long-term studies are required to confirm these effects (37–40). Despite its considerable therapeutic

promise, semaglutide is associated with several challenges, including gastrointestinal adverse effects such as nausea, vomiting, diarrhoea, and constipation, high treatment costs, limited accessibility in many countries, and weight regain following treatment discontinuation. These factors highlight the importance of appropriate patient selection, long-term monitoring, and comprehensive lifestyle modification to maximise therapeutic outcomes (41–43). Ongoing investigations continue to explore higher-dose formulations, combination therapies, and additional indications that may further expand the role of semaglutide in metabolic medicine (44,45). Therefore, this review aims to comprehensively summarise the current evidence regarding Ozempic (semaglutide), with particular emphasis on its pharmacology, mechanism of action, pharmacokinetics, clinical efficacy, safety profile, and expanding role in obesity management. Furthermore, this review discusses evidence from major clinical trials, including the STEP, SUSTAIN, and SELECT programmes, while highlighting emerging therapeutic applications and future research directions. By integrating current evidence, this review seeks to provide healthcare professionals with a comprehensive understanding of semaglutide as a transformative therapy that extends beyond diabetes management and is reshaping the treatment landscape of obesity and related metabolic disorders.

Burden of Diabetes Mellitus:

Diabetes mellitus is one of the fastest-growing chronic metabolic disorders worldwide and represents a major global health concern. Type 2 diabetes mellitus (T2DM) accounts for approximately 90–95% of all diabetes cases and is characterized by insulin resistance, progressive β-cell dysfunction, and chronic hyperglycaemia. The increasing prevalence of obesity, physical inactivity, unhealthy dietary habits, and population ageing has contributed substantially to the rising incidence of type 2 DM. Persistent hyperglycaemia is associated with both microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy, and macrovascular complications such as myocardial infarction, stroke, and peripheral arterial disease, resulting in increased morbidity, mortality, and healthcare expenditure (1–5). Despite advances in glucose-lowering therapies, achieving durable glycaemic control remains challenging for many patients because of progressive disease, poor adherence, hypoglycaemia risk, and treatment-related weight gain. Consequently, current treatment strategies emphasize not only glycaemic control but also weight reduction, cardiovascular protection, renal preservation, and improvement in overall metabolic health. Recent clinical guidelines therefore recommend the preferential use of GLP-1 receptor agonists, particularly semaglutide, in patients with type 2 DM and obesity because of their demonstrated benefits on glycaemic control, body weight, and cardiovascular outcomes (6–9).

Burden of Obesity:

Obesity is now recognized as a chronic, progressive, and relapsing disease rather than merely a consequence of excessive caloric intake. According to the World Health Organization, the prevalence of overweight and obesity has increased dramatically during the past five decades and continues to rise globally. Obesity results from complex interactions among genetic, environmental, behavioural, hormonal, and socioeconomic factors that disrupt long-term energy homeostasis (10–13). Excess adiposity contributes significantly to insulin resistance, chronic low-grade inflammation, dyslipidaemia, hypertension, and endothelial dysfunction, thereby increasing the risk of type 2 DM, cardiovascular disease, chronic kidney disease, metabolic dysfunction-associated steatotic liver disease (MASLD), obstructive sleep apnoea, osteoarthritis, infertility, and several malignancies. In addition to its physical consequences, obesity adversely affects mental health, quality of life, and productivity while imposing substantial economic burdens on healthcare systems. Lifestyle interventions remain the first-line approach; however, long-term maintenance of weight loss is difficult because adaptive physiological responses promote increased appetite and reduced energy expenditure after weight reduction. These limitations have accelerated the development of effective anti-obesity pharmacotherapies, among which semaglutide has demonstrated unprecedented efficacy (14–18).

Evolution Of GLP-1 Receptor Agonists:

The discovery of glucagon-like peptide-1 (GLP-1) revolutionized the treatment of type 2 DM and obesity. GLP-1 is an endogenous incretin hormone secreted by intestinal L-cells following nutrient ingestion. It stimulates glucose-dependent insulin secretion, suppresses glucagon release, delays gastric emptying, and promotes satiety. However, native GLP-1 has an extremely short plasma half-life of approximately 1–2 minutes because of rapid degradation by the enzyme dipeptidyl peptidase-4 (DPP-4), limiting its therapeutic application (19–21). The first clinically available GLP-1 receptor agonist was exenatide, followed by liraglutide, lixisenatide, dulaglutide, albiglutide, and semaglutide. Progressive structural modifications improved resistance to DPP-4 degradation, enhanced albumin binding, prolonged half-life, and enabled once-daily or once-weekly administration. Semaglutide represents a second-generation GLP-1 receptor agonist with superior pharmacokinetic properties, potent glucose-lowering activity, and exceptional weight-loss efficacy. Clinical evidence from the SUSTAIN programme established semaglutide as one of the most effective agents for glycaemic control in type 2 DM, whereas the STEP programme demonstrated clinically meaningful weight reductions of approximately 15–17% in adults with obesity. More recently, the SELECT cardiovascular outcomes trial confirmed that semaglutide also reduces major adverse cardiovascular events in individuals with overweight or obesity, extending its therapeutic role beyond diabetes management (22–30).

Mechanism of Action Of Semaglutide (OZEMPIC):

Semaglutide is a long-acting glucagon-like peptide-1 receptor agonist designed to mimic the physiological actions of endogenous GLP-1 while resisting enzymatic degradation by DPP-4. Structural modifications, including substitution of amino acids and attachment of a C18 fatty diacid side chain, allow reversible albumin binding and prolong the elimination half-life to approximately one week, enabling convenient once-weekly administration (31–33).

Semaglutide exerts its therapeutic effects through multiple complementary mechanisms:

- **Pancreas:** stimulates glucose-dependent insulin secretion from pancreatic β-cells while suppressing glucagon secretion from α-cells, thereby improving glycaemic control with a low risk of hypoglycaemia.
- **Central nervous system:** activates GLP-1 receptors within the hypothalamus and brainstem to enhance satiety, reduce hunger, suppress food cravings, and decrease overall caloric intake.
- **Gastrointestinal tract:** delays gastric emptying, prolonging postprandial fullness and reducing postprandial glucose excursions.
- **Peripheral tissues:** improves insulin sensitivity, reduces hepatic glucose production, and contributes to favourable metabolic adaptations associated with sustained weight reduction.

Beyond glucose lowering and appetite regulation, semaglutide improves several cardiometabolic risk factors, including blood pressure, lipid profile, inflammatory markers, and waist circumference. Evidence from the SELECT trial further suggests that semaglutide reduces cardiovascular events through mechanisms extending beyond glycaemic control alone, including weight reduction, improved endothelial function, anti-inflammatory effects, and favourable haemodynamic changes. These pleiotropic actions have established semaglutide as an important therapeutic option for patients with obesity and obesity-related metabolic disorders (34–45).

Pharmacokinetics and Pharmacodynamics of Semaglutide:

Pharmacokinetics

semaglutide is a long-acting glucagon-like peptide-1 receptor agonist (GLP-1 RA) developed to overcome the limitations of native GLP-1, which is rapidly degraded by dipeptidyl peptidase-4 (DPP-4). Structural modifications, including amino acid substitution and attachment of a C18 fatty diacid side chain, promote reversible albumin binding and confer resistance to DPP-4 degradation, resulting in a prolonged elimination half-life of approximately seven days. This pharmacokinetic property enables convenient once-weekly subcutaneous administration and improves treatment adherence (31–33). Following subcutaneous injection, semaglutide is

slowly absorbed, with peak plasma concentrations reached within 24–72 hours. Bioavailability is approximately 89%, and steady-state concentrations are achieved after 4–5 weeks of once-weekly dosing. Semaglutide is extensively bound to plasma albumin (>99%), which minimizes renal clearance and prolongs systemic exposure. The drug undergoes proteolytic degradation and β-oxidation, with metabolites eliminated through both urine and faeces. Dose adjustment is generally unnecessary in patients with mild to moderate renal or hepatic impairment (31–35). The oral formulation (Rybelsus®) incorporates sodium N-(8-[2-hydroxybenzoyl] amino) caprylate (SNAC), an absorption enhancer that protects semaglutide from gastric degradation and facilitates gastrointestinal absorption. Although oral bioavailability is relatively low, clinically effective plasma concentrations can still be achieved with appropriate dosing (35,36).

Pharmacodynamics

Semaglutide selectively activates GLP-1 receptors, producing multiple glucose-dependent physiological effects. It stimulates insulin secretion, suppresses glucagon release, delays gastric emptying, enhances satiety, and reduces appetite. Because insulin secretion is glucose-dependent, the risk of hypoglycaemia remains low when semaglutide is used as monotherapy. These pharmacodynamic properties contribute to significant reductions in HbA1c, body weight, blood pressure, waist circumference, and other cardiometabolic risk factors (9,31–36).

Clinical Evidence Supporting Semaglutide:

The Sustain Clinical Trial Programme

The SUSTAIN programme evaluated once-weekly semaglutide in patients with type 2 diabetes mellitus. Across multiple randomized controlled trials, semaglutide consistently demonstrated greater reductions in HbA1c, fasting plasma glucose, and body weight than placebo and active comparators including sitagliptin, insulin glargine, exenatide extended-release, and dulaglutide. The SUSTAIN-6 cardiovascular outcomes trial further demonstrated a significant reduction in major adverse cardiovascular events, establishing semaglutide as an effective therapy for both glycaemic control and cardiovascular risk reduction (21–26).

The Step Clinical Trial Programme

The STEP (Semaglutide Treatment Effect in People with Obesity) programme investigated semaglutide 2.4 mg in adults with overweight or obesity, with or without diabetes. STEP 1 demonstrated a mean body-weight reduction approaching 15% compared with placebo when combined with lifestyle intervention. STEP 2 confirmed efficacy in patients with type 2 diabetes, whereas STEP 3 and STEP 4 demonstrated sustained weight reduction when semaglutide was combined with intensive behavioural therapy and continued long-term treatment. STEP 5 showed maintenance of clinically significant weight loss over two years, while STEP 8 demonstrated superior efficacy compared with liraglutide. Collectively, these studies established semaglutide as one of the most effective pharmacological treatments currently available for chronic weight management (1–8,27–33).

The Select Trial

The SELECT trial marked a major advancement in obesity management by demonstrating that semaglutide significantly reduced major adverse cardiovascular events in adults with overweight or obesity and established cardiovascular disease, even in the absence of diabetes. These findings indicate that the cardiovascular benefits of semaglutide extend beyond glycaemic control and are likely mediated through improvements in body weight, inflammation, endothelial function, blood pressure, and lipid metabolism (34–36).

Safety Profile and Adverse Effects:

Semaglutide is generally well tolerated, with gastrointestinal adverse effects representing the most frequently reported events. Nausea, vomiting, diarrhoea, constipation, abdominal discomfort, and reduced appetite are usually mild to moderate in severity and occur primarily during dose escalation. Gradual dose titration substantially improves treatment tolerability (14,41). Less common adverse effects include gallbladder disease, pancreatitis, dehydration, and transient worsening of diabetic

retinopathy in patients with rapidly improving glycaemic control. Because semaglutide delays gastric emptying, caution should be exercised in patients with severe gastrointestinal disorders. It is contraindicated in individuals with a personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia syndrome type 2 (MEN2). Overall, long-term clinical trials have demonstrated an acceptable safety profile with favourable benefit-risk balance (14,21,41–43).

Emerging Therapeutic Applications Beyond Diabetes:

Recent research indicates that semaglutide possesses therapeutic potential extending beyond diabetes and obesity. Clinical studies have demonstrated beneficial effects in metabolic dysfunction-associated steatotic liver disease (MASLD), chronic kidney disease, heart failure with preserved ejection fraction, polycystic ovary syndrome, obstructive sleep apnoea, and cardiovascular disease. Ongoing investigations are evaluating possible neuroprotective effects in Alzheimer's disease and Parkinson's disease owing to the anti-inflammatory and neuroprotective properties of GLP-1 receptor agonists. These emerging applications suggest that semaglutide may become a cornerstone therapy for multiple chronic metabolic disorders (36–45).

FUTURE PERSPECTIVES:

Future research should focus on determining the long-term durability of weight loss, minimizing weight regain after treatment discontinuation, improving affordability and accessibility, and identifying patient populations most likely to benefit from therapy. Combination treatment strategies involving semaglutide and newer agents such as dual GLP-1/GIP receptor agonists are also under investigation and may produce even greater improvements in metabolic health. Real-world evidence and pharmacoeconomic analyses will further define the role of semaglutide in routine clinical practice (42–45).

CONCLUSION:

Semaglutide has transformed the management of both type 2 diabetes mellitus and obesity through its potent glucose-lowering and weight-reducing effects. Extensive evidence from the SUSTAIN, STEP, and SELECT clinical trial programmes demonstrates significant improvements in glycaemic control, sustained weight loss, cardiovascular outcomes, and overall metabolic health. Beyond diabetes, semaglutide shows considerable promise in the treatment of obesity-related complications, including cardiovascular disease, chronic kidney disease, and metabolic dysfunction-associated steatotic liver disease. Although gastrointestinal adverse effects, treatment cost, and weight regain following discontinuation remain important considerations, the overall benefit-risk profile strongly supports its expanding clinical use. As ongoing research continues to explore additional therapeutic indications and combination treatment strategies, semaglutide is expected to remain one of the most influential advances in metabolic medicine and an integral component of future precision healthcare.

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