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editorial-office@sciformat.ca

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GLP-1 RECEPTOR AGONISTS IN THE MANAGEMENT OF OBESE WOMEN WITH POLYCYSTIC OVARY SYNDROME: EFFECTS ON BODY WEIGHT, METABOLIC PROFILE, AND REPRODUCTIVE FUNCTION – A LITERATURE REVIEW

Patrycja Mularczyk *(Corresponding Author, Email: patrycja.mularczyk2000@gmail.com)*
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki, Poland
ORCID ID: 0009-0000-1733-7185

Nina Polek
Cardinal Stefan Wyszynski University in Warsaw, Warsaw, Poland
ORCID ID: 0009-0000-3233-5866

Małgorzata Sikorska
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki, Poland
ORCID ID: 0009-0006-3768-7338

Wojciech Łysoniewski
Szpital Specjalistyczny im. F. Ceynowy w Wejherowie, ul. dr. A. Jagalskiego 10, 84–200 Wejherowo, Poland
ORCID ID: 0009-0006-4850-6379

Artur Marcysiak
Mazowiecki Szpital Wojewódzki im. św. Jana Pawła II w Siedlcach, ul. Poniatowskiego 26, 08-110 Siedlce, Poland
ORCID ID: 0009-0002-8801-3836

Tomasz Mruzek
UCK WUM Centralny Szpital Kliniczny, ul. Banacha 1a, 02-097 Warszawa, Poland
ORCID ID: 0009-0002-3800-0172

Magdalena Kiełbasiewicz
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki, Poland
ORCID ID: 0009-0003-7055-0518

Karolina Zawadzka
Nowy Dwór Medical Center, ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki, Poland
ORCID ID: 0009-0005-8304-4993

ABSTRACT

Objective: This dissertation synthesizes contemporary evidence on glucagon-like peptide-1 (GLP-1) receptor agonists in women with polycystic ovary syndrome (PCOS) and obesity, with emphasis on body weight, metabolic parameters, and reproductive outcomes.
Methods: A narrative–systematic review (2012–2025) included randomized clinical trials, meta-analyses, and systematic and narrative reviews from PubMed, Embase, the Cochrane Library, Web of Science, and other databases. Studies of liraglutide, semaglutide, exenatide, and dulaglutide, as monotherapy or combined with metformin in women with PCOS, were analyzed with respect to anthropometric, metabolic, hormonal, and reproductive endpoints.
Results: GLP-1 receptor agonists generally achieved greater reductions in body weight, BMI, and waist circumference than lifestyle modification or metformin alone, alongside improvements in insulin sensitivity, glycaemic control, and selected lipid measures. Decreases in androgen levels, attenuation of clinical hyperandrogenism, and improved menstrual regularity, ovulation rates, and pregnancy rates were reported, particularly in obese, markedly insulin-resistant women. Adverse events were mainly transient gastrointestinal symptoms.
Conclusions: GLP-1 receptor agonists appear to be a promising adjunct to standard treatment of obese women with PCOS, enabling more effective weight loss, metabolic improvement, and potentially better cardiovascular prognosis, together with enhanced ovulatory and pregnancy outcomes. Nevertheless, the short duration of most studies, absence of safety data in pregnancy, and the requirement for contraception and wash out before conception highlight the need for long-term randomized trials evaluating efficacy, safety, and hard reproductive and cardiovascular endpoints.

KEYWORDS

Polycystic Ovary Syndrome; GLP-1 Receptor Agonists; Insulin Resistance; Hyperandrogenism; Reproductive Function; Obesity

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Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy among women of reproductive age (approximately 6-20%) and, according to the Rotterdam criteria, is defined by the presence of at least two of the following: oligo-ovulation or anovulation, clinical and/or biochemical hyperandrogenism and polycystic ovarian morphology on ultrasound. The syndrome is characterized by marked heterogeneity; however, the phenotype featuring visceral obesity is particularly metabolically adverse and is associated with an increased risk of type 2 diabetes, non-alcoholic fatty liver disease, metabolic syndrome and cardiovascular disease. Insulin resistance, which is often present even in women with normal body weight, represents a key pathophysiological mechanism that leads to hyperinsulinemia, aggravation of hyperandrogenism, and the establishment of a vicious cycle of reproductive and metabolic disturbances.

International guidelines emphasize that the cornerstone of management in women with PCOS is lifestyle modification—dietary change, increased physical activity, and weight reduction—because even moderate weight loss improves metabolic parameters, the androgen profile, and fertility. In practice, however, achieving and sustaining substantial long-term weight loss through behavioral interventions alone is difficult, and traditional insulin-sensitizing agents, such as metformin and thiazolidinediones, exert only a limited impact on body weight. In this context, there is increasing interest in pharmacological strategies for the management of obesity and insulin resistance in PCOS, including glucagon-like peptide-1 receptor agonists (GLP-1 RAs).

GLP-1 analogues are incretin GLP-1 receptor agonists that enhance glucose-dependent insulin secretion, inhibit glucagon secretion, slow gastric emptying, and, via central mechanisms, reduce appetite, thereby promoting clinically meaningful weight loss as well as improvements in glycaemic control, lipid profile, and

cardiovascular risk markers in type 2 diabetes and obesity (C. de Graaf et al., 2016). In studies involving women with PCOS, obesity and insulin resistance have emerged as appropriate therapeutic targets for GLP-1 receptor agonists. Treatment with GLP-1RAs produced greater reductions in body weight and waist circumference than lifestyle modification or metformin alone, accompanied by improvements in insulin sensitivity, lipid parameters, and, in many cases, androgen levels and reproductive function (H. Cena et al.,2020). In parallel, the field of GLP-1–based multi-receptor agonists (e.g. GLP-1/GIP and GLP-1/GIP/glucagon co- and tri-agonists) is rapidly expanding. In clinical and preclinical studies in obesity, these agents demonstrate greater weight-reducing efficacy and pleiotropic metabolic benefits compared with GLP-1 receptor agonists alone (M. A. Sánchez-Garrido et al., 2024).

Growing evidences indicate that GLP-1 receptor agonists may represent a promising adjunct to standard treatment of obesity-related PCOS. Their use aims to improve body-weight control, metabolic profile, and cardiovascular risk, and to increase ovulation rates, menstrual regularity, and the proportion of spontaneous and IVF-mediated pregnancies in obese women with PCOS (K. Elkind-Hirsch et al., 2022; Krzysztof Bednarz et al., 2022; M. Popoviciu et al., 2023). The primary objective of this thesis is to evaluate the role of glucagon-like peptide-1 (GLP-1) receptor analogues in the treatment of obesity in women with polycystic ovary syndrome (PCOS). Specifically, the thesis aims to analyse the effectiveness of these agents in reducing body weight and improving metabolic parameters, including insulin resistance, lipid profile, and arterial blood pressure. A key component is also the assessment of the potential impact of GLP-1 analogues on the hormonal profile and reproductive function, including androgen concentrations and the frequency of ovulatory cycles. Furthermore, the aim of the study is to compare the effects of GLP-1 analogue therapy with the current standard treatment of metabolic disturbances in PCOS, primarily metformin therapy.

Methodology

The present work is a narrative–systematic review examining the use of glucagon-like peptide-1 (GLP-1) receptor agonists in women with polycystic ovary syndrome (PCOS) and overweight/obesity, with an assessment of metabolic, hormonal, and selected reproductive outcomes. A systematic search of the PubMed/MEDLINE, Embase, Web of Science, and Cochrane Library databases was conducted up to 2025, including studies involving overweight or obese women with polycystic ovary syndrome (PCOS) treated with GLP-1 receptor agonists (GLP-1 RAs). Combinations of search terms were used: „polycystic ovary syndrome", „PCOS", „obesity", „overweight", „GLP-1 receptor agonist", „liraglutide", „semaglutide", „weight loss", „insulin resistance", „menstrual regularity", „fertility", „glycemia", „lipid profile".

Physiology Of GLP-1 and Pharmacology Of GLP-1 Analogs

Glucagon-like peptide-1 (GLP-1) is an incretin hormone produced mainly in L-cells of the distal intestine and in the central nervous system, and its circulating concentration increases several-fold, or even by more than an order of magnitude, within 10–30 minutes after meal ingestion. Nutrient stimuli (glucose, lipids, amino acids), together with neural and hormonal signals, trigger GLP-1 secretion, which is then rapidly inactivated by dipeptidyl peptidase-4 (DPP-4) (plasma half-life of approximately 2 minutes), so that a substantial proportion of its actions occurs locally, via paracrine mechanisms and neural pathways The principal stimulatory factors include glucose in the intestinal lumen (via the SGLT1 and GLUT2 transporters), long-chain fatty acids and monoacylglycerols (acting through the GPR40 and GPR119 receptors), and dietary amino acids and peptides. Neural signals, particularly cholinergic pathways, also play an important role, as do other gastrointestinal hormones, such as cholecystokinin and leptin (T. Müller et al.,2019). The GLP-1 receptor (GLP-1R) is a Gs protein-coupled receptor, whose activation leads predominantly to an increase in intracellular cAMP levels. The highest GLP-1R expression is observed in the pancreas, predominantly in β-cells and, to a lesser extent, in α-cells, which underlies its regulatory effects on insulin and glucagon secretion (M. Nauck et al.,2020). Significant expression is also observed within the central nervous system, notably in the nucleus of the solitary tract, the hypothalamus, and limbic structures, which participate in the regulation of appetite, food reward, and gastrointestinal motility (N. Alfaris et al.,2024). GLP-1 receptors are also present within the cardiovascular system, including cardiomyocytes, endothelial cells, and blood vessels, which may help to explain the cardioprotective effects observed with GLP-1R agonists (N. Sattar et al., 2021). GLP-1R expression is also detected in other metabolically active tissues—including the kidneys, lungs, gastrointestinal tract, and adipose tissue—where GLP-1 modulates lipid metabolism, inflammatory status, and adipocyte function (Brent A. McLean et al.,2020).

GLP-1 markedly enhances insulin secretion in a glucose-dependent manner, increases β-cell gene expression and inhibits their apoptosis, while simultaneously suppressing glucagon secretion from α-cells, ultimately leading to a reduction in hepatic glucose production (N. Sattar et al., 2021). This hormone markedly delays gastric emptying, thereby reducing postprandial glycemic excursions and enhancing the sensation of satiety; with chronic stimulation, this effect becomes partially attenuated, a phenomenon also observed with long-acting GLP-1R agonists (T. Müller et al.,2019). Through receptors located in the brainstem and hypothalamus, GLP-1 reduces appetite, decreases energy intake, and promotes body weight reduction while largely preserving overall energy expenditure. It also exerts a beneficial effect on lipid metabolism, reducing visceral and ectopic adipose tissue and attenuating inflammation within adipose tissue and other organs (Yan Xie et al., 2025).

Pharmacological GLP-1 receptor agonists have been engineered to increase resistance to degradation by DPP-4 and to prolong their half-life, which has enabled the development of formulations suitable for once-daily or once-weekly administration (M. Nauck et al., 2020). Among the most widely used agents are liraglutide, semaglutide, dulaglutide, and long-acting formulations of exenatide, which are effective in the treatment of type 2 diabetes and obesity (N. Sattar et al., 2021). High-dose obesity regimens (semaglutide 2.4 mg weekly and liraglutide 3.0 mg daily) produce approximately 10–15% body weight reduction in individuals without diabetes (J. J. Holst, 2024). A meta-analysis of seven cardiovascular outcome trials (56,004 patients) demonstrated a 12% reduction in major adverse cardiovascular events (MACE), a 12% decrease in all-cause mortality, and a 17% reduction in a composite kidney endpoint, with no increase in severe hypoglycemia, pancreatitis, or pancreatic cancer (S. Kristensen et al.,2019). The safety profile of GLP-1 receptor agonists is well established. The most frequent adverse events are nausea, vomiting, diarrhea, and constipation, which are dose- and titration-dependent; their incidence increases with higher doses and more complex agonists, but they are predominantly mild to moderate in severity (M. Nauck et al., 2022).

Pathophysiology of PCOS With a Focus on Obesity

In women with PCOS, obesity—particularly visceral adiposity—is more prevalent and exacerbates insulin resistance, hyperinsulinemia, and low-grade inflammation. Excess visceral adipose tissue promotes the secretion of pro-inflammatory adipokines and cytokines, which further impair insulin signaling. Insulin resistance leads to compensatory hyperinsulinemia that, in the presence of preserved ovarian MAPK pathway activity, enhances ovarian androgen steroidogenesis and reduces SHBG concentrations, thereby aggravating hyperandrogenemia. Hyperandrogenism, in turn, promotes visceral fat deposition, increases circulating free fatty acids, and alters muscle composition, thereby establishing a vicious cycle of insulin resistance–hyperinsulinemia–hyperandrogenism (M. A. Sánchez-Garrido et al., 2020; Han Zhao et al., 2023).

Polycystic ovary syndrome is characterized by accelerated gonadotropin-releasing hormone (GnRH) pulsatility, a predominance of luteinizing hormone (LH) over follicle-stimulating hormone (FSH) secretion, and elevated estrone levels, which collectively promote excessive stimulation of thecal cells and increased androgen production in the context of impaired follicular maturation (Pingping Su et al., 2025). Insulin acts at all levels of the hypothalamic–pituitary–ovarian (HPO) axis, augmenting ovarian responsiveness to LH and IGF-1 while maintaining mitogenic and steroidogenic activity despite underlying metabolic disturbances. Obesity and insulin resistance modify gonadotropin secretion by increasing the amplitude and frequency of LH pulses and enhancing the sensitivity of thecal cells to LH (Han Zhao et al., 2023). Neuroendocrine disturbances involving kisspeptin, KNDy neurons, and alterations in GABAergic signaling further stabilize abnormal GnRH pulsatility and chronic anovulation (Pingping Su et al., 2025).

PCOS, occurring on the background of insulin resistance, hyperandrogenism, and obesity, is associated with a high risk of metabolic disorders, including impaired glucose tolerance, type 2 diabetes mellitus, dyslipidemia, non-alcoholic fatty liver disease, and the metabolic syndrome. Chronic inflammation, oxidative stress, and atherogenic dyslipidemia translate into an elevated cardiovascular risk, encompassing hypertension, atherosclerosis, and cardiovascular events later in life (H. Cena et al., 2020). From a reproductive perspective, PCOS manifests as chronic anovulation, infertility, an increased rate of miscarriage and pregnancy complications (including gestational diabetes mellitus and gestational hypertension), as well as abnormal ovarian morphology and reduced oocyte quality (Sana Siddiqui et al., 2022).

In clinical studies of women with PCOS and obesity, GLP-1 receptor agonists (predominantly liraglutide) have produced significant weight loss, reductions in waist circumference, improvements in glycaemic parameters, and, in some reports, decreases in testosterone levels and improved menstrual cycle regularity. (H. Cena et al., 2020; Han Zhao et al., 2023). GLP-1 receptor agonists may also influence the hypothalamic–

pituitary–ovarian (HPO) axis by modulating LH secretion and may indirectly improve reproductive function through the normalization of body weight and insulin resistance. Their effects complement traditional therapies such as metformin, and combination treatment (metformin + GLP-1 RA) offers a comprehensive improvement in both metabolic and reproductive parameters in women with PCOS, particularly in those with pronounced visceral obesity and insulin resistance (H. Cena et al., 2020; Han Zhao et al., 2023).

Impact of GLP-1 Receptor Agonists on Body Weight in Obese Women with Polycystic Ovary Syndrome

Meta-analyses and systematic reviews of randomized trials involving liraglutide, exenatide, and semaglutide in women with PCOS consistently demonstrate significant reductions in BMI, body weight, waist circumference, and indices of central (abdominal) obesity (Shike Lin et al., 2025). In a meta-analysis of randomized controlled trials conducted in obese women with PCOS, treatment with GLP-1 receptor agonists (predominantly liraglutide and semaglutide), compared with placebo, resulted in:

- BMI: mean change of −2.42 kg/m² (95% CI −3.10 to −1.74);
- Waist circumference: mean change of −5.16 cm (95% CI −6.11 to −4.21) (Beatriz Austregésilo de Athayde De Hollanda Morais et al., 2024).

In a broader meta-analysis of randomized controlled trials (RCTs) including women with PCOS and comparing GLP-1 receptor agonists with metformin or placebo, the following results were obtained:

BMI: mean difference (MD) −1.59 kg/m² (95% CI −2.07 to −1.10)

Body weight: a significant reduction of several kilograms (with specific values varying across individual studies)

Waist circumference (WC): a significant decrease (negative MD, $p < 0.0001$)

Indices of body fat distribution: a significant reduction in waist-to-hip ratio (WHR) and abdominal girth (AG) (Shike Lin et al., 2025).

In a pilot study of obese women with PCOS, 12 weeks of liraglutide therapy resulted in:

- Body weight reduction of 3.1 ± 3.5 kg
- BMI reduction of 1.1 ± 1.26 kg/m²
- A significant decrease in waist circumference and in visceral adipose tissue (VAT) area as measured by DXA ($p = 0.015$) (M. Jensterle et al., 2015).

Systematic reviews and meta-analyses demonstrate that GLP-1 receptor agonists (GLP-1RAs) are more effective than metformin monotherapy in reducing body weight, BMI, and waist circumference in women with PCOS (K. Sridharan et al., 2025). In a meta-analysis of eight randomized controlled trials, GLP-1 receptor agonists (either as monotherapy or in combination with metformin) were associated with greater weight loss (weighted mean difference [WMD] −2.6 kg), as well as larger reductions in waist circumference (approximately −3.5 cm) and BMI compared with metformin alone (Xiaorui Lyu et al., 2021). Comparative analyses indicate that combining a GLP-1 receptor agonist with metformin further reduces body weight, BMI, and waist circumference, and improves HOMA-IR and glycaemic control compared with metformin monotherapy, without a significant increase in serious adverse events (Xiaorui Lyu et al., 2021.; Shike Lin et al., 2025).

The Effect of GLP-1 Receptor Agonists on The Metabolic Profile of Women With Polycystic Ovary Syndrome.

Insulin resistance and glycaemia

In a meta-analysis of 13 randomized controlled trials in women with PCOS, GLP-1 receptor agonists significantly improved markers of glucose metabolism. In analyses of fasting glucose and insulin, GLP-1RAs reduced fasting insulin concentrations and HOMA-IR, accompanied by a significant reduction in 2-h post-load glucose levels during the OGTT. Fasting insulin and HOMA-IR decreased to a statistically significant extent compared with metformin or placebo ($p < 0.0001$), indicating a marked improvement in insulin resistance (Shike Lin et al., 2025). A meta-analysis comparing GLP-1 receptor agonists with metformin demonstrated that GLP-1 RAs are more effective in improving insulin sensitivity, with a standardized mean difference for HOMA-IR of −0.40 (95% CI −0.74 to −0.06; $p = 0.02$) in favor of GLP-1Ras (Yiran Han et al., 2019). In another analysis of randomized controlled trials, GLP-1 receptor agonists reduced HOMA-IR even more markedly, with a standardized mean difference of −0.37 (95% CI −0.60 to −0.15; $p = 0.001$) compared with metformin (Rui-lin Ma et al., 2021). In studies where a GLP-1 receptor agonist was added to metformin, the

effect was more pronounced. In a meta-analysis of eight randomized controlled trials, combination therapy with a GLP-1RA plus metformin reduced:

- HOMA-IR: MD −1.58 (95% CI −2.10 to −1.06; $p < 0.0001$)
- Fasting plasma glucose: MD −0.30 mmol/L (95% CI −0.42 to −0.17; $p < 0.0001$)
- 2-h glucose during OGTT: MD −1.58 mmol/L (95% CI −2.10 to −1.06; $p < 0.0001$) compared with metformin monotherapy (Yuzi Zhao et al., 2025).

In a meta-analysis comparing GLP-1 receptor agonists with placebo in obese women with PCOS, no significant improvement in HOMA-IR was observed (MD −0.30; 95% CI −0.92 to 0.32; $p = 0.35$), despite a marked reduction in BMI and waist circumference (Beatriz Austregésilo de Athayde De Hollanda Morais et al., 2024).

Lipid profile and blood pressure

In a meta-analysis of randomized controlled trials comparing GLP-1 receptor agonists with placebo in obese women with PCOS, GLP-1 RAs significantly reduced:

- Triglycerides (TG): mean difference (MD) −0.20 mmol/L (95% CI −0.30 to −0.11; $p < 0.00001$)
- Total cholesterol: no significant change, MD −0.04 mmol/L (95% CI −0.10 to 0.01; $p = 0.15$) (Bassel H Hoteit et al., 2025).

In a broader meta-analysis of 13 randomized controlled trials including women with polycystic ovary syndrome, GLP-1 receptor agonists (compared with metformin or placebo) produced a significant reduction in triglycerides, while showing no consistent effect on total cholesterol, LDL-C, or HDL-C (a significant overall effect was observed only for triglycerides) (Melissa Frangie Machado et al., 2024).

In the meta-analysis comparing GLP-1 receptor agonists with metformin:

- For LDL-C, the standardized mean difference (SMD) was −0.47 (95% CI −1.03 to 0.09; $p = 0.10$), indicating a trend toward reduction that did not reach statistical significance.
- For HDL-C, the SMD was −0.09 (95% CI −0.98 to 0.81; $p = 0.85$), indicating no effect (Rui-lin Ma et al., 2021).

In a systematic review of clinical trials of GLP-1 receptor agonists in women with PCOS, a reduction in systolic blood pressure was observed in a subset of studies, typically in the range of approximately 2–6 mmHg, whereas other trials reported a neutral effect; no clearly significant differences were consistently demonstrated in pooled analyses (Yuzi Zhao et al., 2025).

The Impact of GLP-1 Analogues on Reproductive Function in Women with Polycystic Ovary Syndrome

Hormonal profile changes (androgens, SHBG, LH/FSH)

GLP-1 receptor agonists reduce androgen levels and increase sex hormone-binding globulin (SHBG), particularly when used in combination with metformin. In a network meta-analysis of insulin sensitizers, metformin plus a GLP-1 receptor agonist increased SHBG (weighted mean difference [WMD] +9.22; 95% credible interval [CrI] 5.46–12.98) and produced greater reductions in free testosterone and androstenedione than GLP-1 receptor agonist monotherapy (H. Cena et al., 2020). In the randomized controlled trial by Nylander et al., 26 weeks of liraglutide treatment increased SHBG and decreased free testosterone compared with placebo, with no changes in LH or FSH (M. Abdalla et al., 2021).

Menstrual cycles and ovulation

In a network meta-analysis of 27 randomized controlled trials, GLP-1 receptor agonists (used either as monotherapy or in combination with metformin) increased menstrual frequency and the proportion of patients with regular menstrual cycles compared with metformin, placebo, and other pharmacologic treatments. (M. A. Sánchez-Garrido et al., 2024). In a randomized controlled trial, liraglutide versus placebo improved the bleeding ratio and menstrual cycle regularity (Marine Monney et al., 2025). In a study of combined liraglutide and metformin therapy, 92% of women with PCOS regained regular menstrual cycles, accompanied by simultaneous improvements in LH, FSH, estradiol, progesterone, and testosterone levels (Marine Monney et al., 2025).

Pregnancy results and in vitro fertilization

Data are limited, but the overall trend appears favorable. Narrative and systematic reviews have associated GLP-1 receptor agonists—particularly liraglutide in combination with metformin—with higher pregnancy rates and improved IVF outcomes in obese, infertile women with PCOS, especially when used as preconception treatment before embryo transfer (Marine Monney et al., 2025; G. Papaetis et al., 2022). One of the studies cited in these reviews demonstrated a significantly higher rate of clinical pregnancies after

preconception treatment with liraglutide plus metformin compared with metformin alone in IVF/ET programs. (Marine Monney et al., 2025). However, robust data on live birth rates are scarce, and further randomized controlled trials are required.

Indirect and direct mechanisms

Indirect mechanisms:

Reduction in body weight, visceral obesity, and insulin resistance diminishes hyperinsulinemia, thereby decreasing ovarian stimulation for androgen production, increasing SHBG levels, and normalizing GnRH/LH pulsatility (Krzysztof Bednarz et al., 2022; H. Cena et al., 2020; K. Sridharan et al., 2025).

Direct mechanisms: GLP-1RAs may:

- Modulate the HPG axis – in rodent models, exendin-4 increases GnRH secretion and augments the preovulatory LH surge in the proestrous phase (Marine Monney et al., 2025; Bassel H Hoteit et al., 2025).
- Act directly on the ovary – in PCOS models, GLP-1 RAs reduce the expression of steroidogenic enzymes, lower testosterone levels, and improve ovarian morphology (fewer cystic follicles, more corpora lutea) (M. A. Sánchez-Garrido et al., 2024).
- Improve endometrial function – in animal studies, GLP-1 RAs reduce oxidative stress and fibrosis in the endometrium and enhance implantation potential (Shaima Rahim et al., 2025; K. Sridharan et al., 2025).

New Horizons: GLP-1 Multi-Agonists in Polycystic Ovary Syndrome

The development of unimolecular incretin-based multi-agonists opens a new perspective for the treatment of obesity-related PCOS, going beyond classical GLP-1RA monotherapies. In the recent study using two mouse models of PCOS, three multi-agonists were compared: GLP-1/estrogen (GLP-1/E), GLP-1/GIP, and GLP-1/GIP/glucagon, each evaluated against metformin. GLP-1/E showed the greatest improvement in the metabolic phenotype (body weight reduction, improved insulin resistance) and normalization of ovarian cyclicity in the ovulatory model, surpassing both other multi-agonists and metformin. Hypothalamic proteomic analysis revealed both shared and distinct pathways regulated by GLP-1/E in the two models, suggesting phenotype-dependent molecular signatures of PCOS and providing a rationale for therapy personalization. Importantly, GLP-1/E did not elicit direct estrogenic effects in the uterus (no uterotrophic activity), while preserving improvements in reproductive parameters (M. A. Sánchez-Garrido et al., 2024).

Safety and Practical Considerations For GLP-1 Receptor Agonist Use in Women with PCOS

Adverse event profile

The most common adverse events are mild to moderate gastrointestinal symptoms, including nausea, vomiting, diarrhea or constipation, and decreased appetite (Melissa Frangie Machado et al., 2024). A meta-analysis of RCTs in PCOS showed a significant increase in nausea ($p = 0.02$), vomiting ($p = 0.04$), and dizziness ($p = 0.03$), with no increase in diarrhoea, constipation, or bloating; some types of abdominal pain were even less frequent than in the control groups (Shike Lin et al., 2025). Compared with metformin, GLP-1 receptor agonists are associated with a higher incidence of nausea and headache (Yiran Han et al., 2019). Serious events (such as pancreatitis, intestinal obstruction, or gastroparesis) are reported only rarely, although pharmacovigilance studies and reviews highlight signals for pancreatitis and delayed gastric emptying and recommend monitoring. The risk of hypoglycaemia with monotherapy is very low (Marine Monney et al., 2025).

Contraindications

Reviews emphasize the need to exclude the following conditions before initiating GLP-1 receptor agonists:

- a history of pancreatitis**,**
- thyroid cancer, particularly medullary thyroid carcinoma, or suspected multiple endocrine neoplasia type 2 (MEN2),
- advanced diabetic retinopathy **(**Marine Monney et al., 2025; Areesha Moiz et al., 2025).

In addition, caution is advised in patients with severe gastrointestinal diseases (such as gastroparesis and severe inflammatory bowel disease) and in those with a history of severe hypersensitivity reactions, including anaphylaxis or severe injection-site reactions.

Preconception planning and discontinuation of GLP-1 receptor agonist therapy

Planned pregnancy in women using glucagon-like peptide-1 receptor agonists (GLP-1 RAs) requires scheduled discontinuation of the drug and transition to safer alternative therapies. Human data are increasingly available but remain limited, so current recommendations remain appropriately cautious. Due to the long elimination half-life:

- Semaglutide: t½ approximately 7 days → discontinue at least 35 days before conception.
- Tirzepatide: t½ approximately 5 days → discontinue 25–35 days before conception.
- Liraglutide: discontinue at least 3 days before conception (S. Price et al., 2025; Abdulrahman Saad Alfaiz, 2025).

Multiple guidelines and narrative reviews generally recommend discontinuing GLP-1 receptor agonists at least five elimination half-lives (approximately 6 weeks) before planned conception. Cohort studies and registry data do not demonstrate a clear increase in the risk of major congenital malformations following unintended early pregnancy exposure, but the evidence is observational and based on relatively small exposed samples, and therefore the findings must be interpreted with caution (K. Dao et al., 2024).

Discussion

Collected data confirm that GLP-1 receptor agonists represent a promising adjunct to therapy in obese women with PCOS; however, their exact position within treatment algorithms is still being defined. Meta-analyses of randomized controlled trials consistently demonstrate significant reductions in body weight, BMI, waist circumference, and measures of visceral adiposity, together with improvements in insulin resistance, aligning with the pathophysiological model of PCOS driven by visceral obesity and hyperinsulinaemia. Significant reductions in testosterone levels and improvements in hyperandrogenism markers indicate that the metabolic benefits of treatment also extend to the endocrine–reproductive domain. This observation is consistent with the concept of a "dual" action of GLP-1 receptor agonists (GLP-1 RAs): indirectly via weight loss and, potentially, directly through modulation of the hypothalamic–pituitary–gonadal axis. It should be emphasized, however, that most studies are based on small, single-centre cohorts, short follow-up periods, and predominantly obese women with PCOS, which markedly limits the generalizability of the findings, particularly to the lean PCOS phenotype. Heterogeneity in the diagnostic criteria used for PCOS and variability in GLP-1 RA dosing regimens and treatment durations hinder direct comparisons between studies. Although the safety profile of GLP-1 receptor agonists appears favorable, it is based predominantly on short-term data; evidence regarding rare, long-term adverse events and safety in the context of pregnancy planning remains insufficient. Another important limitation is the predominant focus on intermediate endpoints. Data on ovulation frequency, clinical pregnancy rates, live births, and long-term cardiovascular risk remain fragmentary, which hampers a comprehensive assessment of the impact of GLP-1 receptor agonists on reproductive and cardiometabolic prognosis. New multi-incretin agonists, despite promising preclinical findings, have not yet undergone systematic evaluation in women with PCOS. In this context, GLP-1 receptor agonists should be regarded as a highly promising yet still partially "experimental" option for the treatment of PCOS. Their use is pathophysiologically justified and supported by an increasing number of randomized controlled trials, but they still require further, well-designed long-term studies, particularly those incorporating hard reproductive and cardiovascular endpoints and explicitly addressing treatment safety in women planning pregnancy.

Conclusions

In women with PCOS and obesity, the use of GLP-1 receptor agonists results in a significant, clinically meaningful improvement in the metabolic profile, reduction in body weight, and favourable changes in reproductive function. Meta-analyses of randomized trials demonstrate clear reductions in BMI, body weight, waist circumference, waist-to-hip ratio (WHR), and visceral adipose tissue compared with placebo and often with metformin, reflecting a decrease in abdominal obesity – a key determinant of metabolic complications in PCOS. At the same time, improvement in insulin resistance is observed (decreases in HOMA-IR, fasting insulin, and glucose during OGTT), a reduction in triglycerides and, in some studies, favourable changes in the lipid profile and inflammatory markers.

At the endocrine level, GLP-1 receptor agonists are associated with reductions in free androgens (free testosterone and free androgen index) and increases in SHBG, consistent with mitigation of hyperandrogenism, one of the core pathophysiological axes of PCOS. Along with the improvement in metabolic parameters, this translates into a beneficial effect on reproductive function: a scoping review and meta-analyses report more

regular menses, higher ovulation rates, and an increased proportion of natural pregnancies, as well as improved IVF outcomes when GLP-1 receptor agonists (alone or in combination with metformin) are introduced in obese women with PCOS. Fertility-focused reviews emphasize that improvements in anthropometric parameters and insulin resistance are likely the principal mechanisms driving enhanced fertility, although an additional, indirect influence on the hypothalamic–pituitary–gonadal axis cannot be ruled out.

In summary, the available evidence indicates that, in women with PCOS and obesity, GLP-1 receptor agonists exert a dual, mutually interrelated effect: they markedly improve the metabolic profile and body weight and, through reducing insulin resistance and hyperandrogenism, promote normalization of menstrual cycles and ovulation and increase the likelihood of conception, while maintaining an acceptable safety profile in the preconception population.

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