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OBESITY, ENDOMETRIOSIS, AND GLP-1 RECEPTOR AGONISTS: A NARRATIVE REVIEW

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ABSTRACT

Endometriosis is a chronic, estrogen-dependent inflammatory disease affecting approximately 10% of women of reproductive age and is one of the leading causes of chronic pelvic pain and infertility. The relationship between obesity and endometriosis remains controversial. Although epidemiological studies indicate that endometriosis is diagnosed less frequently in women with a higher body mass index (BMI), excessive adipose tissue may promote disease progression through hormonal disturbances, chronic inflammation, and altered metabolic activity of adipose tissue. This review summarizes the current evidence regarding the mechanisms linking obesity and endometriosis and discusses the potential role of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in the treatment of this disease. In addition to their well-established efficacy in the management of obesity and type 2 diabetes, GLP-1RAs have been shown to promote weight loss, improve metabolic parameters, and reduce chronic inflammation. Available experimental and observational evidence suggests that these mechanisms may indirectly influence the course of endometriosis by modulating the chronic inflammation and the metabolic disturbances associated with obesity. To date, no randomized clinical trials have evaluated the efficacy of GLP-1 receptor agonists in women with endometriosis, and most of the available evidence is derived from preclinical studies, observational studies, and analyses conducted in patients with polycystic ovary syndrome (PCOS). Current evidence suggests that GLP-1RAs represent a promising area of research; however, their role in the treatment of endometriosis requires confirmation in well-designed clinical trials.

KEYWORDS

Obesity, Endometriosis, GLP-1 Receptor Agonists, Inflammation

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Methodology of the Review:

A narrative literature review was conducted to assess the relationship between obesity and endometriosis, with particular emphasis on the potential role of glucagon-like peptide-1 receptor agonists (GLP-1RAs). The literature search was performed using the PubMed/MEDLINE and Google Scholar databases and covered publications published between 2013 and July 2026.

The search strategy included combinations of the following keywords: “endometriosis,” “obesity,” “overweight,” “body mass index,” “GLP-1,” “GLP-1 receptor agonists,” “semaglutide,” “liraglutide,” “inflammation,” “metabolic dysfunction,” and “reproductive health.” Relevant randomized controlled trials, observational studies, cohort studies, systematic reviews, meta-analyses, clinical guidelines, and other relevant publications were considered. Studies were selected based on their relevance to the objectives of the review and their contribution to understanding the metabolic, inflammatory, hormonal, reproductive, and therapeutic aspects of the relationship between obesity, endometriosis, and GLP-1 receptor agonists. The findings were synthesized narratively, and no quantitative meta-analysis was performed.

Introduction: Epidemiology and Comorbidity

Endometriosis is a chronic, estrogen-dependent inflammatory disease characterized by the presence of endometrial-like tissue outside the uterine cavity, most commonly within the pelvic cavity, including the ovaries. One of the leading theories regarding the pathogenesis of endometriosis is retrograde menstruation, in which menstrual blood containing viable endometrial cells flows backward through the fallopian tubes into the peritoneal cavity, where these cells implant and give rise to endometriotic lesions. The disease is one of the leading causes of chronic pelvic pain in women and may also result in impaired fertility. The most common clinical manifestations include dysmenorrhea, heavy or irregular menstrual bleeding, and chronic pelvic pain (Desai et al., 2026; Capobianco and Rovere-Querini, 2013). Endometriosis affects approximately 10% of women of reproductive age worldwide (World Health Organization, 2025a).

Obesity is a chronic disease defined by a body mass index (BMI) of 30 kg/m² or higher. The primary mechanism underlying its development is a sustained positive energy balance resulting from energy intake exceeding energy expenditure over an extended period. The development of obesity is influenced not only by excessive caloric intake but also by the interaction of multiple factors, including genetic predisposition, socioeconomic determinants, lifestyle, and environmental and cultural factors (Desai et al., 2026). According to the World Health Organization (WHO), more than 16% of adults worldwide are currently living with obesity (World Health Organization, 2025b).

The relationship between body mass index (BMI) and the risk of endometriosis remains controversial and has been the subject of extensive research for many years. Numerous epidemiological studies have demonstrated an inverse association, indicating that women with lower BMI are more frequently diagnosed with endometriosis than women with obesity. In the prospective Nurses' Health Study II, women with severe obesity (BMI ≥40 kg/m²) had an approximately 39% lower risk of endometriosis diagnosis than women with normal body weight (Shah et al., 2013). A meta-analysis including eleven observational studies reported similar findings, demonstrating that each 5 kg/m² increase in BMI was associated with a significant reduction

in the likelihood of an endometriosis diagnosis (Liu and Zhang, 2017). At the same time, recent reviews emphasize that this inverse association should not be interpreted as evidence of a protective effect of obesity.

Epidemiological studies frequently report a lower prevalence of endometriosis diagnosis among women with obesity. At the same time, excess adiposity may create a biological environment that promotes the development and persistence of endometriotic lesions. Adipose tissue is an active endocrine and immunological organ that contributes to estrogen production through the aromatization of androgens and secretes numerous pro-inflammatory mediators, including interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), and leptin. Obesity is also commonly associated with insulin resistance and chronic low-grade inflammation, both of which may promote angiogenesis, fibrosis, and inflammatory processes involved in the pathogenesis of endometriosis. Current evidence therefore suggests that the relationship between obesity and endometriosis is complex and extends beyond body mass index (BMI) alone, encompassing the metabolic and immunological disturbances associated with excess adipose tissue (Desai et al., 2026; Rahman et al., 2025).

Pathophysiological Mechanisms Linking Obesity and Endometriosis

Hormonal Factors

Adipose tissue is an active endocrine organ that plays a central role in hormonal homeostasis. Through the expression of the aromatase enzyme, it contributes to the conversion of androgens into estrogens. In individuals with excess adipose tissue, increased aromatase activity may lead to enhanced estrogen exposure, thereby promoting the persistence and progression of estrogen-dependent endometriotic lesions. Obesity is also frequently associated with insulin resistance and hyperinsulinemia, both of which may disrupt hormonal homeostasis by altering sex steroid metabolism and exacerbating the metabolic disturbances associated with endometriosis. In addition, adipokines secreted by adipose tissue are thought to play an important role in the pathophysiology of the disease. Individuals with obesity typically exhibit elevated leptin levels and reduced adiponectin concentrations, which may influence cell proliferation, immune responses, and the progression of endometriotic lesions (Desai et al., 2026; Rahman et al., 2025; Bulun et al., 2019).

Inflammation

Obesity is characterized by a state of chronic low-grade inflammation resulting from impaired metabolic function of adipose tissue. Hypertrophic adipocytes and macrophages infiltrating adipose tissue secrete numerous pro-inflammatory mediators, including tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), and chemokines, which contribute to sustained immune activation (Desai et al., 2026; Rahman et al., 2025).

Endometriosis is likewise characterized by a chronic inflammatory microenvironment, involving increased activation of immune cells and elevated concentrations of cytokines in the peritoneal fluid. This enhanced inflammatory response may promote the survival of ectopic endometrial-like tissue, stimulate its proliferation, and facilitate angiogenesis. Chronic inflammation may therefore represent a common pathogenic mechanism linking obesity and endometriosis by contributing to tissue remodeling and disease progression (Desai et al., 2026; Rahman et al., 2025; Capobianco and Rovere-Querini, 2013).

The relationship between obesity and endometriosis is complex. Women with excess body weight are more likely to experience ovulatory dysfunction, including polycystic ovary syndrome (PCOS), which is characterized by a distinct hormonal and metabolic profile. The coexistence of PCOS may influence both the clinical presentation and the interpretation of the relationship between metabolic disturbances and endometriosis. The mechanisms underlying this association remain incompletely understood and warrant further investigation (European Society of Human Reproduction and Embryology, 2022).

Microenvironment and Other Contributing Factors

In addition to systemic hormonal and inflammatory disturbances, obesity may also influence the local microenvironment that supports the development and persistence of endometriosis. Visceral adipose tissue, which is highly metabolically active, secretes a variety of biological mediators that may alter the microenvironment of the abdominal and pelvic cavities, thereby modulating processes involved in the establishment and maintenance of endometriotic lesions (Desai et al., 2026; Rahman et al., 2025).

Increasing attention has also been directed toward the role of the gut-immune axis in the pathogenesis of endometriosis. Alterations in the composition of the gut microbiota may affect estrogen metabolism, immune activation, and the persistence of chronic inflammatory processes (Leonardi et al., 2020). Furthermore,

reduced concentrations of glucagon-like peptide-1 (GLP-1) have been reported in the peritoneal fluid of women with endometriosis, suggesting a potential role for this hormone in the local regulation of inflammatory responses. Owing to the limited number of clinical studies, the significance of these mechanisms in the pathogenesis of endometriosis and their potential therapeutic implications remain to be fully elucidated (Krasnyi et al., 2022).

Mechanisms of Action of GLP-1 Receptor Agonists

Glucagon-like peptide-1 receptor agonists (GLP-1RAs), including liraglutide, semaglutide, and exenatide, are widely used in the treatment of type 2 diabetes and obesity. Their mechanism of action is based on the glucose-dependent regulation of insulin and glucagon secretion, as well as their effects on the central nervous system and gastrointestinal motility. Consequently, GLP-1RAs reduce appetite, enhance satiety, and promote significant weight loss (Drucker, 2018; Wilding et al., 2021). The reduction in adipose tissue may, in turn, decrease peripheral estrogen production and attenuate chronic low-grade inflammation. In addition, GLP-1RAs have been shown to improve glycemic control and exert beneficial effects on metabolic parameters, including insulin sensitivity and lipid profile, while also demonstrating anti-inflammatory properties. These pleiotropic effects have positioned GLP-1RAs as a promising therapeutic class for the management of metabolic and inflammation-related diseases (Drucker, 2018; Lee and Jun, 2016).

Anti-inflammatory Effects and Effects on Adipose Tissue

Activation of the glucagon-like peptide-1 (GLP-1) receptor exerts biological effects that extend beyond the regulation of glucose homeostasis. An increasing body of evidence indicates that GLP-1 receptor agonists attenuate chronic low-grade inflammation by reducing the secretion of pro-inflammatory cytokines, including tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6), while improving the metabolic function of adipose tissue (Wang et al., 2023; Alharbi, 2024; Yari et al., 2024). These effects may result from both the direct modulation of immune responses and the reduction in adipose tissue mass achieved during treatment (Wang et al., 2023; Yari et al., 2024).

Given the pivotal role of chronic inflammation in the pathogenesis of endometriosis, the anti-inflammatory properties of GLP-1 receptor agonists may have a beneficial effect on disease progression. Current evidence remains limited and is derived primarily from experimental studies and review articles. Consequently, further clinical studies are needed to clarify the therapeutic potential of GLP-1 receptor agonists in the management of endometriosis (Alharbi, 2024; Yari et al., 2024).

Effects on the Hypothalamic–Pituitary–Ovarian Axis

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) may influence the function of the hypothalamic–pituitary–ovarian (HPO) axis indirectly through improvements in metabolic parameters and modulation of the hormonal disturbances associated with insulin resistance. Studies conducted in women with polycystic ovary syndrome (PCOS) have demonstrated that GLP-1RA therapy promotes weight loss and improves insulin sensitivity, which may contribute to the amelioration of metabolic abnormalities and restoration of reproductive axis function (Jensterle et al., 2015; Müller et al., 2019).

Although the pathophysiological mechanisms underlying PCOS and endometriosis differ substantially, improvements in metabolic homeostasis and the reduction of chronic low-grade inflammation may potentially modulate some of the biological processes involved in the pathogenesis of endometriosis.

Emerging evidence also suggests that GLP-1RAs may influence endometrial function and implantation-related processes. The significance of these effects in the pathophysiology and clinical course of endometriosis remains uncertain and requires further investigation in well-designed clinical studies (Sola-Leyva et al., 2025).

Potential Impact of GLP-1 Receptor Agonists on the Course of Endometriosis

To date, no randomized controlled trials have evaluated the efficacy of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in the treatment of endometriosis. The currently available evidence is derived primarily from observational studies, survey-based research, and experimental models, indicating that the overall level of evidence remains limited (Sola-Leyva et al., 2025). Preliminary findings from an international survey involving 161 women with diagnosed or suspected endometriosis receiving GLP-1 receptor agonists demonstrated that more than 60% of participants reported improvement in at least one disease-related symptom, including pelvic pain, dysmenorrhea, and abdominal bloating. Improvements in quality of life were accompanied by a mean weight reduction of approximately 12 kg. Nevertheless, the authors emphasized that the study was observational in nature and relied on patient-reported outcomes, underscoring the need for confirmation in randomized controlled clinical trials (Leonardi et al., 2025). Preclinical evidence further suggests that GLP-1 receptor activation may inhibit endometrial cell proliferation, promote apoptosis, and attenuate inflammatory activity and oxidative stress. Collectively, these findings support a potential protective role of GLP-1RAs; however, their clinical relevance has yet to be established (Müller et al., 2019; Leonardi et al., 2025). Additional support for further investigation is provided by a cohort study including 444,820 women with endometrial hyperplasia or other benign uterine disorders. The study demonstrated that treatment with GLP-1 receptor agonists in combination with progestins was associated with a significantly lower risk of endometrial cancer compared with progestin monotherapy. Although these findings are not directly applicable to endometriosis, they suggest a potentially beneficial effect of GLP-1 receptor agonists on endometrial tissue and provide a rationale for further investigation of their therapeutic potential in gynecological disorders (Yen et al., 2026).

Indirect Evidence from Studies in Women with Polycystic Ovary Syndrome

Indirect evidence supporting the potential use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in endometriosis is provided by studies conducted in women with polycystic ovary syndrome (PCOS). A prospective, randomized study involving 100 women with overweight or obesity and PCOS, of whom 80 completed the follow-up, demonstrated that combination therapy with semaglutide and metformin resulted in greater weight loss than metformin monotherapy (mean reduction of 6.09 kg vs. 2.25 kg). In addition, combination treatment was associated with a greater reduction in C-reactive protein (CRP) levels, improved ovulatory regularity, and a higher rate of spontaneous pregnancies (35% vs. 15%) (Chen et al., 2025). These findings indicate that GLP-1 receptor agonists effectively improve metabolic parameters and attenuate chronic low-grade inflammation in women with PCOS. Although the pathophysiology of PCOS differs from that of endometriosis, the beneficial effects of GLP-1RAs on body weight, insulin sensitivity, and inflammatory status may represent potential mechanisms supporting the treatment of endometriosis, although this hypothesis requires further investigation (Sola-Leyva et al., 2025; Chen et al., 2025).

Limitations of the Available Evidence

Despite these promising findings, randomized controlled trials evaluating the efficacy of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in women with endometriosis are still lacking. Most of the available evidence is derived from observational studies, survey-based research, animal models, and in vitro experiments; consequently, the current level of evidence remains limited (Müller et al., 2019; Sola-Leyva et al., 2025; Leonardi et al., 2025). To date, it has not been established which patient populations are most likely to benefit from GLP-1RA therapy, how durable the therapeutic effects are, or what treatment regimen is optimal. Furthermore, the use of GLP-1 receptor agonists in women planning pregnancy requires particular caution. Current recommendations advise discontinuation of these agents before conception because of the limited evidence regarding their safety during the periconceptional period (Sola-Leyva et al., 2025).

Safety, Contraindications, and Drug Interactions

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are generally well tolerated, with gastrointestinal adverse events representing the most frequently reported side effects. The most common include nausea, vomiting, diarrhea, constipation, and a sensation of fullness, which typically occur during treatment initiation and tend to diminish with continued therapy or gradual dose escalation (Müller et al., 2019; European Medicines Agency, 2024). In some patients, severe gastrointestinal symptoms may result in dehydration; adequate fluid intake is, therefore, recommended throughout treatment (European Medicines Agency, 2024).

Owing to their effect on delaying gastric emptying, GLP-1 receptor agonists may alter the absorption of certain orally administered medications. Particular caution is warranted in women using oral hormonal contraceptives, especially during treatment initiation and dose escalation, as severe vomiting or diarrhea may reduce contraceptive efficacy. Under these circumstances, the use of additional contraceptive methods is recommended in accordance with the prescribing information for the respective medicinal product (European Medicines Agency, 2024, Faculty of Sexual & Reproductive Healthcare, 2025).

Contraindications to GLP-1 receptor agonist therapy include a personal or family history of medullary thyroid carcinoma and multiple endocrine neoplasia syndrome type 2 (MEN2), based primarily on findings from preclinical studies. Current clinical trials and large population-based analyses have not demonstrated an increased risk of thyroid cancer in humans. The risk of acute pancreatitis is considered low, although initiation of GLP-1RA therapy in patients with a history of pancreatitis should be based on an individualized assessment of the potential risks and benefits (Baxter et al., 2025).

GLP-1 receptor agonists are not recommended during pregnancy or breastfeeding. According to the Summary of Product Characteristics for semaglutide, treatment should be discontinued at least 2 months before a planned pregnancy because of the drug's prolonged elimination half-life (Sola-Leyva et al., 2025; European Medicines Agency, 2024). In addition, weight reduction and improvements in metabolic function may restore ovulation and increase fertility in women with metabolic disorders. For this reason, effective contraception should be discussed before initiating GLP-1RA therapy (Sola-Leyva et al., 2025; Chen et al., 2025).

Knowledge Gaps and Future Research Directions

Despite the growing interest in the potential use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) for the treatment of endometriosis, the current body of scientific evidence remains limited. To date, no randomized controlled trials have evaluated the efficacy of this class of agents in women with endometriosis. Most of the available evidence is derived from observational studies, survey-based research, and experimental models, highlighting the need for confirmation of these findings in well-designed clinical trials (Sola-Leyva et al., 2025; Leonardi et al., 2025).

Future studies should evaluate the effects of GLP-1 receptor agonists on pain severity, quality of life, fertility outcomes, and the risk of disease recurrence following surgical treatment. Particular attention should also be directed toward identifying patient subgroups most likely to benefit from GLP-1RA therapy, especially according to body mass index (BMI), disease stage, and the presence of concomitant metabolic disorders (Müller et al., 2019; Sola-Leyva et al., 2025).

It also remains unclear whether the potential therapeutic benefits of GLP-1 receptor agonists are attributable solely to weight reduction and improvements in metabolic parameters or whether they additionally result from direct anti-inflammatory and immunomodulatory effects. Further investigation is required to elucidate the effects of these agents on endometrial cell proliferation, angiogenesis, immune cell activity, and the microenvironment of endometriotic lesions (Müller et al., 2019).

Long-term safety data on the use of GLP-1 receptor agonists in women of reproductive age are also needed, particularly with respect to pregnancy planning, fertility, and prolonged use beyond the approved indications for diabetes and obesity management. The availability of such evidence will be essential for defining the role of GLP-1 receptor agonists in future therapeutic strategies for endometriosis (Sola-Leyva et al., 2025; European Medicines Agency, 2024).

Summary and Practical Implications

The relationship between obesity and endometriosis is complex and involves interactions among metabolic disturbances, hormonal dysregulation, and chronic inflammation. Although most epidemiological studies report a higher prevalence of endometriosis among women with lower body mass index (BMI), obesity may influence disease progression by exacerbating inflammatory processes, disrupting hormonal homeostasis, and altering the endocrine function of adipose tissue (Bulun et al., 2019; Blüher, 2019; Maheshwari et al., 2020).

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) represent a promising class of agents for the management of obesity and metabolic disorders. In addition to promoting substantial weight reduction, these agents improve insulin sensitivity, lipid metabolism, and inflammatory status, which may indirectly modulate biological pathways involved in the pathogenesis of endometriosis (Wilding et al., 2021; Müller et al., 2019; Sola-Leyva et al., 2025). Nevertheless, the currently available evidence is derived predominantly from experimental studies, observational research, and investigations conducted in women with polycystic ovary syndrome; consequently, these findings cannot be directly extrapolated to patients with endometriosis (Sola-Leyva et al., 2025; Leonardi et al., 2025; Chen et al., 2025).

At present, the available evidence is insufficient to support the incorporation of GLP-1 receptor agonists into the standard management of endometriosis. In women with endometriosis accompanied by obesity or metabolic disorders, these agents may provide clinical benefits primarily through weight reduction and improvement of metabolic health. Their use should remain consistent with current approved indications and be guided by an individualized clinical assessment of each patient (Sola-Leyva et al., 2025; Leonardi et al., 2025; European Medicines Agency, 2024).

In conclusion, GLP-1 receptor agonists represent a promising area of investigation in the treatment of endometriosis. Their role in the therapeutic management of this disease remains to be established through well-designed randomized controlled trials. The generation of high-quality clinical evidence will be essential for defining the efficacy, safety, and future place of GLP-1 receptor agonists within evidence-based therapeutic recommendations for endometriosis (Müller et al., 2019; Sola-Leyva et al., 2025).

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