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Is There an Effect of Glucagon-like Peptide-1 (GLP-1) Receptor Agonists on Endometriosis?

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The clinical approach to gynecological endocrinopathies has been significantly altered by the introduction of glucagon-like peptide-1 (GLP-1) receptor agonists (1). Although these agents were initially developed for type 2 diabetes and obesity, they now have a central role in the management of reproductive dysfunctions like polycystic ovary syndrome (PCOS) (1). Clinicians routinely trust their proven ability to reverse insulin resistance, drive weight loss, and improve cardiovascular risk profiles (2). But when their applications broaden into gynecology, a novel and compelling target emerges: the pathophysiology and medical management of endometriosis.

It might initially seem to be contradictory to associate weight-loss medications with endometriosis. An inverse relationship between body mass index (BMI) and the risk of developing the disease has been revealed by epidemiological evidence consistently pointing to an "adiposity paradox" (3). The theoretical risk of retrograde menstruation and, consequently, symptomatic endometriosis is lower because patients with higher BMIs frequently experience anovulation and oligomenorrhea (3). However, the complex metabolic nature of the condition is usually overlooked by this inverse correlation. Endometriosis is an estrogen-dependent, systemic, inflammatory disorder that is deeply connected to immunometabolic stress, rather than being a strictly localized pelvic disease.

For endometriosis, the real promise of GLP-1 receptor agonists lies in their strong anti-inflammatory effects, not in their weight reduction effects. Recent molecular data have highlighted a direct link between incretin hormones and the peritoneal microenvironment. Notably, in one study, GLP-1 concentrations, along with other metabolic mediators such as glucagon and ghrelin, were substantially lower in the peritoneal fluid of women with endometriosis (4). Moreover, this local lack of GLP-1 was strongly correlated with elevated expression of proteases, such as CD10, and pro-inflammatory markers in peritoneal macrophages (4). These macrophages clear retrograde endometrial debris in a healthy environment. However, their immunomodulatory capacity fails in a GLP-1-deficient state. This dysfunction sustains chronic inflammation, promotes impaired cellular recycling, and ultimately allows ectopic lesions to survive and proliferate.

Independent of weight loss, targeted pharmacological supplementation with GLP-1 agonists could yield specific clinical benefits, as GLP-1 deficiency appears to facilitate this inflammatory pathogenesis. Although current evidence for these agonists in gynecology is heavily skewed toward PCOS (1,2), their well-documented ability to regulate cellular metabolism and dampen systemic inflammation makes them excellent candidates for disrupting the fibrotic processes underlying endometriosis. In addition, long-term hormonal suppression, often relied upon by routine endometriosis management, can paradoxically cause weight gain and metabolic disruption. These adverse effects may be counteracted by co-administration of a GLP-1 agonist, providing a disease-modifying synergistic benefit directly at the peritoneal level.

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As a result, a highly promising therapy for endometriosis lies in the overlap between metabolic endocrinology and pelvic inflammation. Robust, randomized controlled trials are urgently needed to assess the direct impact of GLP-1 receptor agonists on peritoneal macrophage function, lesion progression, and endometriosis-associated pain. Unraveling this relationship may shift our clinical focus from merely hormonal symptom suppression to active correction of the immunological dysregulation at the heart of the disease.

Competing Interests
None declared.

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