Obesity Pillars 19 (2026) 100297

Contents lists available at ScienceDirect

Obesity Pillars

journal homepage: www.journals.elsevier.com/obesity-pillars

Obesity and pelvic organ prolapse: Review

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ARTICLE INFO

Keywords:
Obesity
Pelvic organ prolapse
Quality of life
Collagen
Epigenetics

ABSTRACT

Introduction: Obesity is a major public health challenge with substantial effects on women's health and quality of life. Pelvic organ prolapse (POP), characterized by the descent of pelvic organs into the vaginal canal, is influenced by both obesity-related mechanical forces and molecular alterations. This review aimed to evaluate the relationship between obesity and POP, with particular emphasis on etiopathogenesis, clinical consequences, and implications for prevention and treatment strategies.

Methods: This was a narrative review of the literature examining the relationship between obesity and pelvic organ prolapse. A literature search was performed using PubMed, Scopus, and Google Scholar databases. Relevant studies addressing obesity, pelvic organ prolapse, extracellular matrix remodeling, genetic susceptibility, epigenetic mechanisms, inflammation, and surgical outcomes were reviewed and synthesized. The available evidence was evaluated with respect to mechanical loading, connective tissue alterations, molecular pathways, and clinical implications.

Results: Increased intra-abdominal pressure resulting from visceral adiposity was identified as a major mechanical factor contributing to pelvic floor weakening. In addition, obesity was found to influence extracellular matrix remodeling through altered collagen metabolism, adipokine imbalance, chronic inflammation, and epigenetic modifications. These mechanisms may contribute to the development and progression of POP, complicate surgical management, and increase the risk of recurrence. Current evidence also suggests that genetic susceptibility and connective tissue disorders may amplify the effects of obesity on pelvic floor dysfunction.

Conclusions: Obesity represents both an important risk factor and a therapeutic challenge in pelvic organ prolapse, adversely affecting women's quality of life. Understanding the interaction between mechanical, molecular, genetic, and epigenetic mechanisms may facilitate individualized prevention and management strategies for women with obesity. Further clinical and translational studies are needed to clarify these mechanisms and support the development of personalized approaches to POP prevention and treatment.

1. Introduction

Obesity is one of the most important public health challenges worldwide. This situation plays a role in the etiopathogenesis of diseases and also emerges as a problem that can complicate treatment. When considered in terms of work, time, and cost calculation, it is evident that preventing obesity, diagnosing and treating obesity, comorbidities, and the extra burden during surgery are the main problems.

The accumulation of fat tissue in the abdominal region can have numerous effects under the molecular and mechanical categories. Pelvic organ prolapse is a common condition characterized by the downward displacement of pelvic organs, such as the uterus, bladder, and rectum, towards the vaginal canal. Although many risk factors contribute to the pathophysiology of this condition, a chronic increase in IAP is one of the most significant factors contributing to its development. Obesity, chronic cough, heavy lifting, constipation, and especially increased IOP during recurrent pregnancies can create mechanical loads on the pelvic floor muscles and connective tissues, causing these structures to weaken over time and prolapse to develop [1,2]. This increase in pressure, particularly by straining the support of the levator ani muscle group and endopelvic fascia, facilitates the pelvic organs' shift in the direction of gravity.

2. Methods

This was a narrative review of the literature examining the relationship between obesity and pelvic organ prolapse. A literature search was conducted using PubMed, Scopus, and Google Scholar databases.

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https://doi.org/10.1016/j.obpill.2026.100297
Received 25 June 2026; Received in revised form 7 July 2026; Accepted 7 July 2026
Available online 11 July 2026

The review focused on studies addressing obesity, pelvic organ prolapse, extracellular matrix remodeling, genetic susceptibility, epigenetic mechanisms, inflammation, and surgical outcomes. Original articles and relevant review papers published in English were considered. The objective was to summarize current evidence regarding the mechanical, molecular, genetic, and clinical links between obesity and pelvic organ prolapse.

3. Discussions

Clinical studies have demonstrated that increased mechanical loading contributes to stretching and weakening of the pelvic floor support structures. Experimental and imaging studies have shown that excessive strain on the levator ani muscles and connective tissues may contribute to pelvic floor dysfunction and pelvic organ prolapse [3]. In this context, strategies aimed at reducing ICP are essential both in the prevention and treatment of POP. Lifestyle changes (weight loss, smoking cessation, pelvic floor exercises) may reduce the risk of developing POP. In addition, ICP control should be considered an essential element in patients planned for surgical treatment to prevent postoperative recurrence [4]. Obesity, together with increased body mass index (BMI), has been identified as a significant risk factor for the development of pelvic organ prolapse. In individuals with obesity, increased visceral fat tissue leads to increased intra-abdominal pressure (IAP), placing a chronic mechanical load on the pelvic floor structures. This leads to overdistension and weakening of the pelvic support tissues over time. Several epidemiological studies have reported an association between increasing BMI and a higher prevalence of POP symptoms [5, 6]. In addition, obesity is associated with complications in POP surgery rates and increases the risk of recurrence after surgery. Therefore, weight control plays an essential role in preventive strategies for women at risk of POP. Collagen tissue diseases, especially connective tissue disorders such as Ehlers-Danlos syndrome, are an important predisposing factor for the development of POP by directly affecting the pelvic floor support structures. In these diseases, the deterioration in the synthesis and structure of type I and III collagen reduces the elasticity and durability of the connective tissues. As a result, the pelvic organs cannot be adequately supported even under normal pressure, and prolapse may develop [7]. Histological studies have demonstrated irregularities in collagen fiber organization and a decrease in collagen density in patients with POP [8]. These data highlight the role of genetic and structural factors in the pathogenesis of POP and emphasize the importance of individualizing treatment and follow-up strategies. Pelvic organ prolapse is a multifactorial condition that develops as a result of mechanical and structural weakness of the pelvic floor structures. Although obesity is a significant risk factor for the development of POP, it has been recognized that this relationship cannot be explained solely by increased mechanical load. Molecular biology studies in recent years have demonstrated that obesity has direct effects on collagen synthesis, extracellular matrix (ECM) structure, and connective tissue integrity through both genetic and epigenetic pathways [9]. These effects are particularly evident in fibroblast activity, the collagen type I/III ratio, and the expression of ECM remodeling proteins.

Individuals with obesity, changes in the genetic expression of collagen proteins, one of the basic building blocks of connective tissue, have been detected Alterations in COL1A1 and COL3A1 expression have been reported in women with POP, and several studies suggest that genetic variants may contribute to these findings [10]. Systemic inflammation associated with obesity disrupts the transcriptional regulation of these genes, reducing the flexibility of connective tissue and compromising its structural integrity. In addition, adipokines (especially leptin and adiponectin) may affect collagen balance in the ECM by modulating the expression of these genes at the fibroblast level [11].

At the epigenetic level, DNA methylation and histone modifications are essential mechanisms that determine the effects of obesity on connective tissue. Hypermethylation of ECM-related genes has been proposed as a potential mechanism that may reduce gene expression and contribute to connective tissue alterations associated with POP [12]. Chronic inflammation and oxidative stress associated with obesity can alter these methylation patterns by activating epigenetic regulators, such as DNA methyltransferases (DNMTs) and histone deacetylases (HDACs). This reduces the repair capacity of connective tissue, facilitating the development of POP. MicroRNAs (miRNAs) are short RNA molecules that regulate gene expression at the posttranscriptional level. Some obesity-associated miRNAs (e.g., miR-29a, miR-21, and miR-181) can directly affect collagen synthesis [13]. The miR-29 family has been implicated in ECM regulation, and obesity-associated alterations in miRNA expression may influence collagen synthesis and tissue remodeling. In addition, obesity-associated alterations in miRNA expression may influence fibroblast activity and extracellular matrix remodeling through pathways involving TGF-β signaling [13].

Increased levels of adipokines (especially leptin, resistin, and TNF-α) due to obesity may suppress or unregulatedly activate signaling pathways that play a crucial role in connective tissue regeneration, such as TGF-β (transforming growth factor-beta). TGF-β signaling typically promotes collagen production and tissue remodeling. Dysregulation of this pathway may impair fibroblast function and extracellular matrix remodeling [14]. These molecular derangements may reduce the flexibility and load-bearing capacity of pelvic connective tissue and may contribute to the development of POP.

Finally, genetic predisposition to develop POP in some individuals may work synergistically with obesity. For example, polymorphisms in genes encoding extracellular matrix components, including **COL1A1**, have been associated with susceptibility to pelvic organ prolapse. However, the interaction between these genetic variants and obesity has not yet been fully established and requires further investigation [15]. Such gene-environment interactions have been proposed as factors that may influence susceptibility to POP and disease severity. These findings underscore the significance of genetic analyses and epigenetic profiling in the pathogenesis of POP, paving the way for the development of personalized prevention and treatment strategies.

Although patients are examined and classified in the same way as normal individuals, excessive fat storage in the vaginal walls and fat accumulation at the vulvar level may make it difficult to evaluate the pelvic examination [16]. If obesity is considered a significant predisposing etiological factor or a challenging treatment factor for the patient, it may be beneficial to lose weight and adjust lifestyle standards [17]. Although obesity is not always the primary factor, it should be evaluated in detail during anamnesis in cases such as hysterectomy, difficult delivery, and incontinence accompanied by multiparity [18].

Information should also be provided about additional complications that may occur secondary to surgery, such as the risk of embolism after surgery. Vascular insufficiency, inflammatory tissue, and inadequate oxygenation also impose an additional burden [19]. At the cellular level, tissue fibrosis is a complex and dynamic process involving multiple cell types, including macrophages and fibroblasts. Crown-like structures that drive inflammation and fibrosis in obesity have emerged as important targets for single-cell and spatial transcriptomic analyses. Kohda et al. identified novel cell-to-cell interactions between macrophages and fibroblasts in adipose tissue from diet-induced obese mice, particularly during the fibrotic phase. In a **mouse model of diet-induced obesity**, Kohda et al. identified novel cell-to-cell interactions between macrophages and fibroblasts in adipose tissue during the fibrotic phase [20]. Their findings highlighted the role of the macrophage-inducible C-type lectin–oncostatin M axis in obesity-induced adipose tissue fibrosis and provided mechanistic insights into the relationship between obesity, inflammation, and tissue remodeling. Although these findings are derived from an experimental animal model, they may contribute to understanding biological pathways potentially relevant to pelvic floor disorders.

Limitations: This review has several limitations. First, it was conducted as a **narrative review** rather than a systematic review; therefore,

a predefined systematic review methodology was not applied, and a formal assessment of study quality or risk of bias was not performed. Consequently, publication bias and selective reporting within the reviewed literature may have influenced the interpretation of the findings. In addition, the current evidence regarding the genetic and epigenetic mechanisms linking obesity and pelvic organ prolapse remains limited and heterogeneous, with many proposed pathways requiring further validation in well-designed clinical studies. Some mechanistic evidence is also derived from experimental and animal studies, which may limit the direct applicability of these findings to humans. Despite these limitations, this review provides an integrated overview of the current evidence and highlights important directions for future clinical and translational research.

4. Conclusion

Obesity contributes to the development and progression of pelvic organ prolapse through a complex interplay of mechanical loading, chronic inflammation, extracellular matrix remodeling, and genetic and epigenetic mechanisms. Recognizing these interconnected pathways may facilitate earlier identification of women with obesity who are at increased risk of pelvic floor dysfunction and support more individualized prevention and management strategies. Although current evidence highlights the importance of these mechanisms, further well-designed clinical and translational studies are needed to clarify their interactions and to improve risk stratification, prevention, and therapeutic approaches for pelvic organ prolapse.

Consent to participate

Not applicable.

Take-home messages

- Obesity contributes to pelvic organ prolapse through interconnected mechanical, inflammatory, genetic, and extracellular matrix mechanisms.
- Individualized prevention and management strategies for women with obesity may improve pelvic floor outcomes.
- Further clinical and translational research is needed to clarify genetic and epigenetic mechanisms and guide personalized treatment.

Author contributions

Belma Gözde Özdemir conceived the study, designed the review framework, performed the literature search, evaluated and synthesized the scientific evidence, drafted the manuscript, critically revised the content, and approved the final version for publication.

Author disclosures

The author declares no conflicts of interest.

Ethical review

“Ethical approval was not required because this study is a narrative review based exclusively on previously published literature."

Ethics approval

Not applicable.

Written consent for publication

Not applicable.

Availability of data and material

Not applicable.

Code availability (software application or custom code)

Not applicable.

Declaration of use of artificial intelligence

A statement has been added indicating that generative artificial intelligence was used only for language editing and manuscript refinement, while the author reviewed, verified, and takes full responsibility for the final content of the manuscript.

Source of funding

No external funding was received for this work.

Conflicts of interest/competing interests

No.

Acknowledgements

None.

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Lead Author Statement: Dr. Belma Gözde Özdemir served as the Lead Author and provided the conceptual leadership for this review. She developed the scientific rationale and scope of the manuscript, identified the key themes to be addressed, synthesized the available literature, interpreted the evidence, and supervised the overall preparation and revision of the article.

Professional Relationship Between Authors: This manuscript was prepared by a single author; therefore, no inter-author professional relationships apply.

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