NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

Quality in Sport. eISSN 2450-3118.

Journal Home Page
https://apcz.umk.pl/QS/index

GŁOWACKA, Aleksandra, KUREK, Aleksandra, DOMIŃCZAK, Dominika, IVANCHUK, Sofiia, GÓRECKI, Patryk, KAMINSKAYA, Anhelina, SAVCHAK, Tetiana, FERETYCKI, Hubert, SALAMA, Aladdin, and ABDULLA, Shafea. The Role of Vitamin D and Physical Activity in Endometriosis: Implications for Pain Relief, Physical Function and Reproductive Health – A Literature-Based Review. Quality in Sport. 2026;54:70766. eISSN 2450-3118. https://doi.org/10.12775/QS.2026.54.70766

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 12.04.2026. Revised: 20.04.2026. Accepted: 27.04.2026. Published: 29.04.2026.

The Role of Vitamin D and Physical Activity in Endometriosis: Implications for Pain Relief, Physical Function and Reproductive Health- A Literature-Based Review

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ABSTRACT

Purpose of research. The aim of this study was to evaluate the roles of vitamin D and physical activity in the management of endometriosis, with particular emphasis on their effects on pain reduction, physical function, and reproductive health in affected women. **Research materials and methods.** A literature-based review was conducted using major scientific databases, including PubMed, Scopus, and Web of Science. The analysis included original studies, randomized controlled trials, systematic reviews, and meta-analyses focusing on vitamin D status, supplementation, and physical activity interventions in women with endometriosis.

Basic results. Vitamin D exhibits immunomodulatory, anti-inflammatory, antiproliferative, and anti-angiogenic properties mediated through the vitamin D receptor (VDR), which is widely expressed in reproductive tissues. Lower serum levels of 25-hydroxyvitamin D may be associated with increased risk and severity of endometriosis; however, clinical evidence regarding pain reduction remains inconsistent.

Physical activity, including aerobic exercise and mind–body interventions such as yoga, has been shown to reduce pain, improve quality of life, and positively influence psychological well-being through anti-inflammatory and neuroendocrine mechanisms.

Emerging evidence suggests that optimizing vitamin D levels, combined with regular physical activity, may produce synergistic effects, particularly in improving physical performance, reducing inflammation, and supporting bone health.

Conclusions. Vitamin D and physical activity may serve as safe and effective complementary strategies in the management of endometriosis. Their integration into a personalized, multidisciplinary approach may improve patient outcomes; however, further high-quality studies are required to establish standardized clinical guidelines.

Keywords: endometriosis; vitamin D; physical activity; pelvic pain; reproductive health; lifestyle

1. Introduction

Endometriosis is a chronic, estrogen-dependent systemic inflammatory disease affecting approximately 10% of women of reproductive age worldwide [34]. It is characterized by the presence of endometrial-like tissue, including glands and stroma, outside the uterine cavity, most often in the pelvic area but also in extrapelvic sites. The disease presents with a wide range of clinical symptoms, including dysmenorrhea, chronic pelvic pain, dyspareunia, dyschezia and infertility, affecting approximately 30–50% of patients [4].

Due to the nonspecific nature of symptoms, the diagnosis of endometriosis is often delayed by 8–12 years, leading to increased healthcare burdens and a significant decline in patient's quality of life [35]. Despite extensive research, the precise pathogenesis of endometriosis remains only partly understood. Several theories have been proposed, including retrograde menstruation, coelomic metaplasia, and genetic and immunological factors [30]. Chronic inflammation and oxidative stress in the peritoneal environment play crucial roles, promoting the adhesion, invasion, and angiogenesis of ectopic lesions [4].

Current treatment strategies mainly rely on hormonal therapy to suppress ovulation and estrogen production, along with surgical removal of lesions. However, these approaches are often associated with side effects, limited long-term effectiveness, and a high recurrence rate [5]. Therefore, increasing attention has been directed toward complementary and lifestyle-based interventions that may support conventional treatment.

Vitamin D, a secosteroid hormone with pleiotropic effects, has emerged as a potential modulator of endometriosis due to its immunomodulatory, anti-inflammatory, and anti-proliferative properties [10, 32]. The vitamin D receptor (VDR) is widely expressed in reproductive tissues, and genetic research indicates that polymorphisms in VDR-related genes may affect susceptibility to endometriosis [6]. Additionally, observational and epidemiological studies indicate that lower serum levels of 25-hydroxyvitamin D may be associated with increased risk and greater severity of the disease [1,13].

Mechanistically, the active form of vitamin D ($1,25(OH)_2D_3$) may inhibit the production of pro-inflammatory cytokines, prostaglandins, and matrix metalloproteinases (MMP-2 and MMP-9), thereby potentially limiting lesion progression [14, 30]. However, current evidence on the clinical effectiveness of vitamin D supplementation remains inconsistent, as highlighted by recent systematic reviews [2,8,31].

In addition to nutritional and metabolic factors, physical activity has been identified as an important non-pharmacological approach in managing endometriosis. Regular exercise may reduce pain symptoms, improve physical function, and enhance quality of life through multiple

mechanisms, including anti-inflammatory effects, improved pelvic blood flow, and modulation of hormonal balance [13,16,26]. Additionally, contracting skeletal muscles act as an endocrine organ, releasing myokines with anti-inflammatory properties and stimulating β-endorphin secretion, which may increase pain tolerance and improve psychological well-being [27].
Emerging evidence indicates a possible interaction between vitamin D levels and physical activity. Exercise may influence serum vitamin D concentrations, while adequate vitamin D status could enhance physical performance and musculoskeletal health [23, 21]. Despite these findings, the combined effects of vitamin D and exercise in endometriosis remain poorly understood, highlighting a significant gap in current research.

The aim of this literature-based review is to evaluate the roles of vitamin D and physical activity in endometriosis, with particular emphasis on their effects on pain relief, physical function, and reproductive health, as well as to investigate potential synergistic mechanisms underlying their combined effects.

2. Research materials and methods

2.1 Search strategy

A comprehensive literature search was conducted across multiple electronic databases, including PubMed, MEDLINE, Scopus, Web of Science, Google Scholar, and The Cochrane Library. The search aimed to identify studies investigating the roles of vitamin D and physical activity in women diagnosed with endometriosis.

The screening process involved a stepwise evaluation of titles, abstracts, and full-text articles to ensure relevance to the research topic. To enhance the completeness of the review and minimize the risk of omitting relevant publications, the reference lists of all included studies were manually screened (snowballing method).

The search strategy was developed to capture both clinical and mechanistic studies examining the impact of vitamin D status and physical activity on endometriosis-related outcomes, including pain, inflammation, quality of life, and reproductive function.

2.2 Keywords

The literature search was performed using combinations of the following keywords and Medical Subject Headings (MeSH), connected with Boolean operators (AND, OR):
“endometriosis”, “vitamin D”, “25-hydroxyvitamin D”, “25(OH)D”, “vitamin D receptor”, “physical activity”, “exercise”, “pelvic pain”, “inflammation”, “reproductive health”, and “infertility”.

Search queries were adapted to the indexing systems and requirements of each database.

2.3 Inclusion criteria

Studies were included in the review if they met the following criteria:

- Articles published in English
- Studies involving human participants, particularly women of reproductive age with clinically or surgically confirmed endometriosis
- Original research articles, including randomized controlled trials (RCTs), cohort studies, and cross-sectional studies, as well as systematic reviews and meta-analyses
- Studies investigating the relationship between vitamin D status, supplementation, or physical activity and endometriosis-related outcomes (e.g., pain, inflammation, fertility, or quality of life)
- Publications published between 1990 and 2025, ensuring inclusion of both foundational and recent scientific evidence

2.4 Exclusion criteria

The following studies were excluded:

- Case reports and case series due to limited generalizability
- Non-peer-reviewed publications, including conference abstracts, editorials, letters to the editor, preprints, and unpublished manuscripts
- Studies unrelated to the scope of this review, including those focusing exclusively on primary dysmenorrhea without confirmed endometriosis
- Studies not addressing vitamin D, physical activity, or their interaction in the context of endometriosis

2.5 Data analysis

A qualitative synthesis of the selected studies was conducted. Extracted data included study characteristics (study design, sample size, population), type of intervention (vitamin D supplementation dosage, duration, form; type, frequency, and intensity of physical activity), and key clinical outcomes.

Primary outcomes of interest included pain severity (commonly assessed with the Visual Analog Scale, VAS), quality of life (e.g., SF-36, EHP-30 questionnaires), inflammatory markers, and reproductive outcomes (e.g., fertility indicators and pregnancy rates).

The findings were critically evaluated regarding methodological quality, consistency of results, and clinical relevance. Particular attention was given to discrepancies across studies, including differences in study design, population characteristics, and intervention protocols, as highlighted in recent systematic reviews [2,16,17].

3. Pathophysiology of endometriosis

The pathophysiology of endometriosis is complex, multifactorial, and not yet fully elucidated. Currently, endometriosis is no longer viewed solely as the ectopic presence of endometrial-like tissue but rather as a chronic, systemic, estrogen-dependent inflammatory disorder involving hormonal, immunological, and molecular dysregulation [4,34]. The development and progression of the disease result from intricate interactions between endocrine imbalance, immune dysfunction, and persistent inflammatory processes within the peritoneal environment [4,30].

3.1 Theories of ectopic lesion formation

Several hypotheses have been proposed to explain the origin of ectopic endometrial lesions.

The most widely accepted theory is retrograde menstruation (Sampson’s theory), which suggests that menstrual blood containing viable endometrial cells refluxes through the fallopian tubes into the peritoneal cavity [34]. Although this phenomenon occurs in the majority of women, only a subset develops endometriosis, indicating that additional factors—particularly immune dysfunction and genetic susceptibility—are required for implantation and survival of ectopic tissue [6,30].

The coelomic metaplasia theory proposes that peritoneal cells may transform into endometrial-like cells in response to hormonal and inflammatory stimuli [34]. The embryonic (Müllerian) theory suggests that endometriosis may arise from displaced Müllerian remnants during fetal development, explaining cases occurring before menarche or in atypical anatomical locations [34].

Additionally, lymphatic and hematogenous dissemination may account for distant lesions, such as those found in the lungs or brain [34]. The stem cell theory further supports the involvement of progenitor cells capable of migrating, differentiating, and contributing to ectopic lesion formation [4].

3.2 Hormonal dysregulation and progesterone resistance

Endometriosis is a strongly estrogen-dependent condition. Ectopic lesions exhibit increased local estradiol production due to overexpression of aromatase and steroidogenic acute regulatory protein (StAR), which promotes cellular proliferation and sustains inflammation [4,14].

Simultaneously, progesterone resistance represents a hallmark of the disease. This phenomenon is associated with reduced expression of progesterone receptor isoform B (PR-B) and epigenetic modifications, including promoter hypermethylation [4]. As a result, progesterone-mediated anti-inflammatory and anti-proliferative effects are impaired, leading to uncontrolled tissue growth and persistence of lesions [30].

3.3 Immune dysfunction and chronic inflammation

Impaired immune surveillance plays a critical role in the pathogenesis of endometriosis. Under physiological conditions, immune cells such as macrophages, natural killer (NK) cells, and T lymphocytes effectively eliminate ectopic endometrial cells from the peritoneal cavity. In women with endometriosis, this process is disrupted [4,30].

Key immunological alterations include:

- reduced cytotoxic activity of NK cells, facilitating survival of ectopic implants [30]
- activation of macrophages, which secrete pro-inflammatory cytokines such as IL-1, IL-6, IL-8, and TNF-α, promoting lesion growth and angiogenesis [4]

- activation of inflammatory signaling pathways, including NF-κB, leading to enhanced cell adhesion, proliferation, and resistance to apoptosis [4,14]

This persistent inflammatory environment contributes not only to disease progression but also to symptom severity.

3.4 Oxidative stress

Oxidative stress represents another important factor in the pathogenesis of endometriosis. An imbalance between reactive oxygen species (ROS) and antioxidant defense systems has been observed in the peritoneal fluid of affected women [4].

Elevated ROS levels may induce DNA damage, lipid peroxidation, and activation of pro-inflammatory signaling pathways, thereby facilitating the establishment and progression of ectopic lesions [30]. Furthermore, oxidative stress has been closely associated with both pain severity and infertility in endometriosis patients [4].

3.5 Angiogenesis and neurogenesis- mechanisms of pain

The survival and expansion of endometriotic lesions depend on the formation of new blood vessels. Angiogenesis is primarily mediated by vascular endothelial growth factor (VEGF), which is overexpressed in ectopic endometrial tissue [4,30].

In addition to vascularization, neurogenesis plays a crucial role in pain generation. Endometriotic lesions secrete nerve growth factor (NGF) and other neurotrophic factors, leading to increased innervation of ectopic tissue [30]. These newly formed nerve fibers are often hypersensitive, contributing to chronic pelvic pain and dysmenorrhea.

Over time, persistent peripheral stimulation may lead to central sensitization, a process in which pain perception becomes independent of lesion size or location. This mechanism may explain the frequently observed discrepancy between the extent of disease and symptom severity [30].

4. Role of vitamin D in endometriosis

Vitamin D, a secosteroid hormone with pleiotropic biological functions, plays a significant role in female reproductive health and has been increasingly investigated in the context of endometriosis [3,4,32]. Its active form, calcitriol ($1,25(OH)_2D_3$), exerts both genomic and non-

genomic effects through binding to the vitamin D receptor (VDR), which is widely expressed in reproductive tissues, including the endometrium, ovaries, and ectopic endometriotic lesions [3,6,12].

The presence of VDR and vitamin D-metabolizing enzymes within both eutopic and ectopic endometrial tissues suggests that vitamin D may act locally, influencing cellular proliferation, inflammatory responses, and immune regulation associated with the disease [4,14].

4.1 Molecular and cellular mechanisms

The biological effects of vitamin D in endometriosis are mediated through several key mechanisms.

Anti-inflammatory effects: Calcitriol suppresses the production of pro-inflammatory cytokines, including IL-1β, IL-6, IL-8, and TNF-α, thereby attenuating the chronic inflammatory state characteristic of endometriosis [4,14,28]. Additionally, vitamin D reduces prostaglandin synthesis (particularly PGE_2) through modulation of cyclooxygenase-2 (COX-2) and 15-hydroxyprostaglandin dehydrogenase (15-PGDH), which may contribute to pain reduction [14,28].

Anti-proliferative and pro-apoptotic properties: Experimental studies indicate that vitamin D inhibits proliferation and invasiveness of endometrial stromal cells (ESCs), induces apoptosis, and promotes cell cycle arrest [4,14]. These effects may limit the growth and persistence of ectopic lesions.

Anti-angiogenic activity: Vitamin D has been shown to downregulate the expression of vascular endothelial growth factor (VEGF) and matrix metalloproteinases (MMP-2 and MMP-9), thereby reducing angiogenesis and tissue invasion capacity of endometriotic lesions [4,14].

Immunomodulatory effects: Vitamin D modulates both innate and adaptive immune responses by influencing macrophages, T lymphocytes, and natural killer (NK) cells. This may enhance immune surveillance and facilitate the clearance of ectopic endometrial cells from the peritoneal cavity [3,4,6].

4.2 Vitamin D status and risk of endometriosis

A growing body of observational and epidemiological evidence suggests an association between serum 25-hydroxyvitamin D [25(OH)D] levels and the risk of endometriosis.

Several studies report an inverse relationship between vitamin D levels and disease occurrence, indicating that lower serum concentrations may be associated with increased risk and severity of endometriosis [1,13,5]. Population-based analyses, such as data from the NHANES cohort, have demonstrated a higher prevalence of endometriosis among women with lower vitamin D levels [13].

Similarly, Mendelian randomization analysis suggests a potential relationship between circulating 25(OH)D levels and susceptibility to endometriosis, although causality remains uncertain [1]. Some evidence indicates that vitamin D deficiency (e.g., <20 ng/mL) may significantly increase disease risk, whereas higher concentrations may exert a protective effect [4].

However, the available evidence is not entirely consistent. Certain studies have reported higher vitamin D levels in patients with endometriosis, possibly due to altered local metabolism, including increased expression of 1α-hydroxylase in ovarian tissue [4,8]. These discrepancies highlight the complexity of vitamin D metabolism and the need for further investigation.

4.3 Effects of vitamin D supplementation on clinical outcomes

The clinical impact of vitamin D supplementation in endometriosis remains inconclusive.

Some randomized controlled trials (RCTs) have demonstrated that high-dose vitamin D supplementation (e.g., 50,000 IU weekly or biweekly) may significantly reduce dysmenorrhea and chronic pelvic pain [2,29]. In addition, vitamin D supplementation has been associated with improvements in inflammatory biomarkers, such as high-sensitivity C-reactive protein (hs-CRP), and increased total antioxidant capacity (TAC) [4].

However, other studies and systematic reviews have failed to demonstrate a statistically significant advantage of vitamin D supplementation over placebo in reducing pain, particularly in postoperative settings [2,31]. Furthermore, recent analyses suggest that while vitamin D may

have beneficial effects on reproductive outcomes, including pregnancy rates, the available evidence remains limited and heterogeneous [9].

Overall, current findings indicate that vitamin D supplementation may have supportive, but not definitive, clinical benefits, and its effectiveness may depend on baseline vitamin D status, disease stage, and individual patient characteristics.

4.4 Evidence from Mendelian Randomization Studies

Recent Mendelian randomization (MR) studies provide important insights into the potential causal relationship between vitamin D and endometriosis.

Pan et al. [1] found no clear evidence that genetically determined serum 25(OH)D levels directly influence the overall risk of endometriosis. This suggests that associations observed in observational studies may be confounded by environmental or lifestyle factors.

Interestingly, subgroup analyses indicated a possible positive association between ovarian endometriosis and vitamin D levels, highlighting heterogeneity in disease mechanisms across different endometriosis phenotypes [1].

These findings underscore the complexity of the relationship between vitamin D and endometriosis and highlight the need for further well-designed studies to clarify causal pathways and clinical relevance.

5. Physical activity and symptom relief in endometriosis

Physical activity (PA) and structured exercise interventions are increasingly recognized as important components of non-pharmacological management strategies in women with endometriosis [25,27,35]. Although the available evidence remains heterogeneous due to differences in study design, intervention type, and outcome measures, a growing body of literature suggests that regular physical activity may positively influence both physical and psychological aspects of the disease [16,17,25].

5.1 Biological and analgesic mechanisms

The beneficial effects of physical activity in endometriosis are mediated through several interconnected biological mechanisms.

Anti-inflammatory effects (myokines): Contracting skeletal muscles function as an endocrine organ, releasing myokines such as interleukin-10 (IL-10), which exert systemic anti-inflammatory effects and may counteract the chronic inflammatory state associated with endometriosis [17,27]. Exercise also induces transient increases in cortisol and catecholamines, contributing to short-term suppression of inflammatory processes [17].

Hormonal regulation: Physical activity may influence hormonal balance by increasing levels of sex hormone-binding globulin (SHBG), thereby reducing the bioavailability of circulating estrogens, which are key drivers of endometriotic lesion growth [25,27]. Additionally, regular exercise improves insulin sensitivity and metabolic regulation, which may indirectly affect disease progression [25].

Pain modulation (neuroendocrine mechanisms): Exercise stimulates the release of β-endorphins and activates the endocannabinoid system, including increased levels of anandamide, leading to elevated pain thresholds and improved mood [27]. These mechanisms are particularly relevant in the context of chronic pelvic pain and central sensitization observed in endometriosis.

Improved pelvic circulation and muscle relaxation: Regular physical activity may enhance blood flow in the pelvic region, reduce muscle tension, and facilitate the clearance of inflammatory mediators, thereby contributing to symptom relief [25,26].

5.2 Effects on quality of life and clinical outcomes

Evidence from systematic reviews and meta-analyses indicates that exercise-based interventions can significantly improve multiple domains of quality of life (QoL) in women with endometriosis, including pain intensity, emotional well-being, and perceived control over symptoms [16,17,26].

Mind–body interventions (e.g., yoga): Evidence suggests that practices such as Hatha yoga may significantly reduce daily pain intensity, improve body awareness, and enhance psychological well-being in affected women [25,27].

Aerobic and relaxation-based exercise: Aerobic training (e.g., running, cycling) and progressive muscle relaxation techniques have been associated with reductions in dysmenorrhea, anxiety, and depressive symptoms [16,26].

Frequency and intensity of exercise: Studies suggest that regular physical activity—typically at least 150 minutes per week or ≥3 sessions weekly—is associated with reduced frequency and severity of pain episodes [17,25]. However, excessive or high-intensity exercise may exacerbate symptoms in some patients, highlighting the need for individualized exercise prescriptions [27].

5.3 Supportive role in pharmacological treatment and bone health

Physical activity may also play a supportive role in patients undergoing conventional hormonal therapy.

Reduction of treatment-related side effects: Exercise has been shown to mitigate certain adverse effects associated with pharmacological treatments, such as weight gain and hormonal imbalances (e.g., during danazol therapy) [25].

Bone health protection: Women treated with gonadotropin-releasing hormone (GnRH) agonists are at increased risk of decreased bone mineral density (BMD). Regular weight-bearing and aerobic exercise may help preserve or improve BMD, particularly in the femoral neck region [25,35].

5.4 Summary and clinical recommendations

Despite methodological limitations and variability across studies, the majority of available evidence supports a beneficial role of physical activity in the management of endometriosis symptoms [16,17,25].

Healthcare professionals should consider incorporating individualized physical activity recommendations into patient care, taking into account disease severity, pain levels, and overall physical condition. Moderate, regular exercise appears to be the most effective and sustainable approach, while overly intensive training should be avoided due to the potential risk of symptom exacerbation [27].

6. Combined effects of vitamin D and physical activity

The combination of vitamin D supplementation and regular physical activity may produce synergistic effects, potentially leading to greater therapeutic benefits than either intervention alone. Although direct evidence specifically addressing their combined impact in endometriosis remains limited, findings from related fields and mechanistic studies suggest significant potential for improving pain, inflammation, and overall physiological function [4,23,25].

6.1 Mechanisms of biological synergy

The synergistic interaction between vitamin D and physical activity appears to involve modulation of overlapping molecular and metabolic pathways.

Regulation of inflammatory and oxidative pathways: Both vitamin D and exercise independently exert anti-inflammatory effects, and their combination may enhance this response. Vitamin D suppresses pro-inflammatory cytokines such as TNF-α and IL-6, while physical activity induces anti-inflammatory myokines and improves antioxidant capacity [4,17,23,28]. This dual modulation may be particularly relevant in endometriosis, where chronic inflammation and oxidative stress are central to disease progression.

AKT/mTOR signaling pathway: Vitamin D and physical activity may influence the AKT/mTOR signaling pathway, which plays a key role in cellular metabolism, protein synthesis, and tissue repair [23]. Their combined activation may enhance regenerative processes and counteract tissue damage associated with chronic inflammation.

Myokine–vitamin D interaction: Exercise-induced myokines, such as IL-6 (released transiently during muscle contraction), may interact with vitamin D signaling pathways. Activation of the vitamin D receptor (VDR) may further modulate immune and metabolic responses, contributing to systemic homeostasis [3,17].

6.2 Effects on physical performance and body composition

Evidence from randomized controlled trials conducted in other populations indicates that combined interventions involving vitamin D supplementation and exercise produce superior outcomes in physical performance compared to either intervention alone.

Such combined approaches have been associated with improvements in muscle strength, functional capacity, and body composition, including reductions in body mass index (BMI) and enhanced metabolic efficiency [19,23]. Vitamin D supplementation may also potentiate the effects of aerobic and resistance training on muscle strength, gait speed, and overall physical fitness [19,20].

Improvements in balance and mobility have also been reported, which may be particularly relevant for women with endometriosis experiencing chronic pain and reduced physical function.

6.3 Bidirectional relationship: physical activity as a modulator of vitamin D status

The relationship between vitamin D and physical activity is bidirectional, with exercise influencing vitamin D metabolism and availability.

Mobilization of vitamin D stores: Acute and chronic physical activity may increase circulating levels of 25(OH)D through mobilization from adipose tissue and skeletal muscle stores [18].

Sunlight exposure: Outdoor physical activity is a major determinant of vitamin D synthesis, particularly through increased exposure to ultraviolet B (UVB) radiation [21,22].

Increased physiological demand: Athletes and physically active individuals may exhibit higher vitamin D requirements due to increased metabolic turnover and muscle activity, supporting the need for monitoring and potential supplementation [21,22].

6.4 Clinical implications for women with endometriosis

In the context of endometriosis, a multimodal approach combining vitamin D optimization and regular physical activity may offer clinically relevant benefits.

Potential combined effects include:

- Enhanced pain reduction, through synergistic modulation of inflammatory pathways and increased release of endogenous analgesics such as β-endorphins [17,28]
- Improved bone health, particularly important in patients undergoing GnRH agonist therapy, which is associated with decreased bone mineral density [25]

- Improved psychological well-being, including reduced anxiety and depressive symptoms, due to both neuroprotective effects of vitamin D and the mood-enhancing effects of physical activity [25,27]
- potential improvements in reproductive health outcomes, mediated by anti-inflammatory and hormonal regulatory mechanisms [3,9]

6.5 Limitations and research gaps

Despite promising theoretical and indirect evidence, high-quality studies directly assessing the combined effects of vitamin D and physical activity in endometriosis are limited. Existing research is characterized by heterogeneity in study design, variability in supplementation protocols, and differences in exercise interventions.

Future well-designed randomized controlled trials are needed to determine the extent of synergistic effects and to establish evidence-based clinical recommendations, including optimal dosing strategies for vitamin D and standardized exercise protocols.

7. Clinical implications

The available evidence suggests that lifestyle-based interventions, including vitamin D optimization and regular physical activity, may serve as valuable adjuncts to conventional treatment strategies in the management of endometriosis. Although these approaches are not substitutes for pharmacological or surgical interventions, their integration into clinical practice may contribute to improved symptom control, attenuation of inflammation, and enhanced overall patient well-being [2,17,25,35].

7.1 Monitoring and optimization of vitamin D status

Given the high prevalence of vitamin D deficiency among women with endometriosis and its potential association with increased inflammation, pain severity, and disease progression, regular assessment of serum 25-hydroxyvitamin D [25(OH)D] levels should be considered in clinical practice [1,4,13].

Vitamin D supplementation should be individualized and guided by baseline serum concentrations, with particular attention to patients presenting with deficiency (<20–30 ng/mL). Evidence suggests that correction of hypovitaminosis D may contribute to improvements in inflammatory markers and, in some cases, reduction of pain symptoms, although clinical outcomes remain heterogeneous [2,29].

From a clinical perspective, vitamin D supplementation appears most beneficial when incorporated into a personalized treatment strategy, particularly in patients with confirmed deficiency or those at increased risk of impaired bone health [3,4].

7.2 Prescription of tailored physical activity

Healthcare professionals should actively promote physical activity as a safe, accessible and cost-effective supportive intervention in endometriosis management [16,17,25]. Evidence from systematic reviews and meta-analyses indicates that structured exercise programs may reduce pain intensity, improve quality of life, and alleviate psychological symptoms such as anxiety and depression [16,26,27].

Particularly beneficial forms of activity include:

- Aerobic exercise (e.g., walking, cycling), which supports cardiovascular health and reduces systemic inflammation
- Mind–body interventions (e.g., yoga), which have demonstrated effectiveness in reducing chronic pelvic pain and improving emotional well-being
- Relaxation-based techniques (e.g., progressive muscle relaxation), which may decrease muscle tension and stress-related symptom exacerbation

Exercise prescriptions should be individualized, taking into account disease severity, pain levels, and patient preferences. Moderate-intensity, regular activity (e.g., ≥150 minutes per week) appears to be the most effective and sustainable approach, while excessive or high-intensity exercise should be avoided due to the potential risk of symptom exacerbation [17,25,27].

7.3 Integration into multimodal and personalized care

The management of endometriosis requires a comprehensive, multidisciplinary approach that integrates pharmacological, surgical, and lifestyle-based strategies. Incorporating vitamin D optimization and physical activity into standard care may enhance therapeutic outcomes through complementary and potentially synergistic mechanisms [4,23,25].

From a clinical standpoint, combined interventions may:

- Enhance pain management through modulation of inflammatory pathways and increased endogenous analgesic activity (e.g., β-endorphins) [17,28]
- Support bone health, particularly in patients treated with GnRH agonists, who are at increased risk of reduced bone mineral density [25,35]

- Improve psychological outcomes, including reductions in anxiety, depression, and perceived disease burden [25,27]
- Promote functional capacity and overall physical performance, which may be impaired in patients with chronic pelvic pain [23]

7.4 Patient education and empowerment

An important clinical implication is the role of patient education in improving adherence and long-term outcomes. Informing patients about the potential benefits of lifestyle interventions may enhance engagement in treatment and promote active participation in disease management. Encouraging sustainable lifestyle modifications, including regular physical activity and appropriate vitamin D supplementation, may improve patients' sense of control over the disease and contribute to long-term improvements in quality of life [25,35].

7.5 Limitations and considerations for clinical practice

Despite promising evidence, clinicians should be aware of the limitations of the current literature. The heterogeneity of study designs, variability in supplementation protocols, and differences in exercise interventions limit the ability to establish universal clinical guidelines.

Therefore, lifestyle interventions should be considered as supportive strategies within a personalized treatment framework rather than standalone therapies. Further high-quality randomized controlled trials are required to define optimal protocols and confirm long-term clinical benefits.

8. Conclusions

The present literature review indicates that both vitamin D and physical activity represent promising non-pharmacological strategies supporting the management of endometriosis. Despite heterogeneity in study design and outcome measures, the available evidence suggests clinically relevant benefits in modulating inflammation, reducing pain, and improving overall patient well-being [2,17,25].

8.1 Role of Vitamin D

Vitamin D functions as a potent immunomodulatory agent with anti-inflammatory, anti-proliferative, and anti-angiogenic properties that are highly relevant to the pathophysiology of endometriosis [3,4,14]. Mechanistic studies demonstrate that calcitriol may inhibit pro-

inflammatory cytokine production, suppress lesion growth, and modulate immune responses involved in disease progression [4,14,28].

However, clinical evidence remains inconsistent. While some randomized controlled trials report reductions in pain and inflammatory markers following supplementation, others fail to demonstrate significant benefits compared to placebo [2,29,31]. Notably, the effectiveness of vitamin D supplementation appears to depend on baseline vitamin D status, with the greatest potential benefits observed in patients with deficiency, which is common in women with endometriosis and may correlate with disease severity [1,13].

8.2 Role of Physical Activity

Regular physical activity has demonstrated beneficial effects across multiple dimensions of endometriosis, particularly in reducing pain and improving quality of life [16,17,26]. Interventions such as aerobic exercise, yoga, and relaxation-based techniques are associated with improvements in both physical and psychological outcomes.

These effects are mediated through anti-inflammatory pathways, including myokine release, as well as neuroendocrine mechanisms involving increased β-endorphin production, which contribute to elevated pain thresholds and improved mood [17,27]. Nevertheless, variability in exercise protocols and limited sample sizes restrict the ability to establish standardized clinical recommendations [16,17].

8.3 Clinical implications and future directions

A holistic, multimodal approach integrating conventional pharmacological treatment with vitamin D optimization and individualized physical activity may offer meaningful benefits in endometriosis management [4,23,25].

Such strategies may:

- improve symptom control and reduce chronic pain
- support bone mineral density (BMD), particularly in patients treated with GnRH agonists
- enhance psychological well-being and functional capacity
- contribute to improved reproductive health outcomes [3,9,25]

However, further high-quality, large-scale randomized controlled trials are required to:

- standardize vitamin D supplementation protocols (dose, duration, target levels)

- define optimal types, intensity, and frequency of physical activity
- evaluate the combined effects of these interventions in endometriosis populations

Future studies should incorporate validated outcome measures, such as the Visual Analog Scale (VAS) for pain assessment and standardized quality-of-life instruments (e.g., EHP-30), to improve comparability and clinical applicability of findings.

8.4 Final statement

In conclusion, despite existing gaps in the evidence, lifestyle modification through optimization of vitamin D status and regular physical activity represents a safe, accessible, and potentially effective component of comprehensive endometriosis care. When implemented within a personalized and multidisciplinary framework, these interventions may support reproductive health, alleviate chronic symptoms, and significantly improve patient's quality of life [4,25,35].

Disclosure

Supplementary Materials: Not applicable.

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All authors have read and agreed with the published version of the manuscript.

Funding Statement: The study did not receive special funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Conflict of Interest Statement: The authors declare no conflict of interest.

Acknowledgements: Not applicable.

AI Declaration: Declaration of the use of generative AI and AI-assisted technologies in the writing process. In preparing this work, the authors used ChatGPT for the purpose of assisting with grammar and readability. After using this tool, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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