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Diet and Endometriosis: A Comprehensive Review of Current Evidence

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Abstract

Endometriosis is a chronic, estrogen-dependent inflammatory disease affecting 10% of women of reproductive age. Patients often experience symptoms such as pelvic pain, dysmenorrhea, gastrointestinal disturbances, and infertility. Conventional treatment methods, including hormonal therapy and surgery, offer limited relief and high recurrence rates. Dietary interventions have gained interest as potential adjuncts to symptom management. This study reviews current evidence on the impact of diet on the development, symptom severity, and progression of endometriosis, focusing on specific nutrients, dietary patterns, and elimination diets. A literature search was conducted using PubMed and Google Scholar for English-language, peer-reviewed studies published up to 2025. Keywords were “Endometriosis”, “Diet”, “Gluten-free”, “Vitamin D”, “Omega-3”, “FODMAP Diet”. Original research articles, randomized controlled trials, and systematic reviews (only if original data were also reported) were analysed. Anti-inflammatory, antioxidant-rich diets are associated with symptom reduction. Omega-3 fatty acids show protective effects, while high consumption of red meat and trans fat may increase the risk of endometriosis. Vitamin D shows potential benefits in experimental models, but clinical evidence remains inconclusive. Gluten-free and low-fermentable oligo-, di-, monosaccharide and polyol (FODMAP) diets may relieve

gastrointestinal symptoms in selected patients, but should be implemented with caution. Dairy products and dietary fiber may offer protective effects, though data remain inconsistent. While current evidence suggests that diet may influence endometriosis, most data remain observational. Further randomized controlled trials are needed to confirm causality and develop evidence-based dietary guidelines for endometriosis management.

Keywords: Endometriosis, Diet, Inflammation; Omega-3; Vitamin D; Antioxidants; FODMAP; Gluten-free

Introduction

Endometriosis is a chronic hormone-related inflammatory disease observed in one in 10 women of reproductive age. It is characterized by the presence of endometrial tissue outside of the uterine cavity. There are three subtypes of endometriosis: superficial, ovarian, and deep infiltrating [1–3]. Endometrium-like tissue is usually found in the pelvis minor, ovaries, fallopian tubes, uterosacral ligaments, and vesicouterine pouch [1]. The main symptom is chronic pain, but common complaints also include dysmenorrhea, dyspareunia, dysuria, dyschezia, infertility, fatigue, constipation, diarrhea, and bloating. These symptoms are often non-specific, which may delay diagnosis [4].

Various theories have been put forward to explain the pathophysiology and etiology of endometriosis. Immunological, genetic, endocrine, and inflammatory factors are all considered significant [5,6]. The „retrograde menstruation" or implantation theory remains the most widely accepted explanation, though alternative mechanisms, including coelomic metaplasia, hematologic or lymphatic spread, altered immune function, and gut dysbiosis, have also been suggested. Local estrogen production and a sustained pro-inflammatory environment are considered crucial in disease progression [7].

Treatment options for this disease are still limited. Conventional treatment strategies include hormonal therapy, surgical excision, pain management, and assisted reproductive techniques (ART). These approaches often provide only partial symptom relief. They also carry side effects, or are limited by high recurrence rates [7]. That is why almost half of patients with endometriosis implement self-management strategies such as diet modifications. Studies have suggested that specific dietary patterns or nutrients may modulate inflammation, immune responses, and hormonal regulation. It may influence endometriosis risk or symptom severity.

Diets rich in omega-3 fatty acids, antioxidants, and polyphenols, as well as a high intake of fruits, vegetables, and fiber, have been associated with pain reduction and improved quality of life. High consumption of red meat, trans fats, and processed foods has been reported to increase the risk of endometriosis [7]. Furthermore, specific dietary interventions, such as low-fermentable oligo-, di-, monosaccharide, and polyol (FODMAP) or gluten-free diets, have been investigated due to the overlapping symptoms of endometriosis, irritable bowel syndrome (IBS), and celiac disease [7].

Although current evidence is mainly observational and sometimes inconsistent, the biological plausibility of dietary influences on endometriosis is supported by biochemical pathways involving inflammation and estrogen metabolism. Dietary strategies represent a promising adjunct to conventional therapies and warrant further research through large-scale, high-quality randomized controlled trials.

This study aimed to review the current scientific evidence of the influence of diet on the development, progression, and symptomatology of endometriosis. The review sought to identify dietary components and nutritional interventions- such as specific nutrients, dietary patterns, and elimination diets- that may affect hormonal balance, inflammatory processes, oxidative stress, and gastrointestinal symptoms in women with endometriosis.

Materials and Methods

A literature review was conducted using the electronic databases (PubMed; Google Scholar) to identify relevant peer-reviewed studies published in English up to 2025. The search strategy combined Medical Subject Headings (MeSH) and free-text terms, including „Endometriosis", „Diet", „Gluten-free", „Diet, Gluten-free", „Antioxidants", „FODMAP Diet", „Vitamin D", „Fatty acids, Omega-3", and related synonyms. Boolean operators (AND, OR) were used to refine the search and include both clinical and experimental studies. Inclusion criteria were original research articles, randomized controlled trials, and systematic reviews (only if original data were also reported) focusing on the relationship between dietary factors and endometriosis incidence, symptom severity, or quality of life. Studies about nutritional interventions, supplementation, and anti-inflammatory or antioxidant mechanisms were also included. Non-English publications, case reports, and studies unrelated to dietary factors or without relevance to endometriosis were excluded.

1. Antioxidants

Chronic inflammation and oxidative stress play a crucial role in endometriosis pathophysiology; therefore, nutrients that are anti-inflammatory, antioxidant, and polyphenol-rich could favorably modulate these mechanisms. It has been reported that IL-1β may increase the proliferation of endometriosis cells and enhance the pro-inflammatory activity of IL-6 and IL-8, as well as NF-κB signaling. In endometriosis, increased levels of TNFα, COX-2-derived PGE2, and other cytokines are also observed [8–10]**.** Women with endometriosis have been shown to consume significantly fewer antioxidant-rich products than controls, with lower intakes of vitamins A, C, and E, zinc, and copper. A high-antioxidant diet for 4 months increased plasma concentrations of retinol, α-tocopherol, and ascorbate, enhanced the enzymatic activities of superoxide dismutase and glutathione peroxidase, and reduced oxidative stress markers, such as malondialdehyde and lipid hydroperoxides [11]. Clinical studies also suggest that adherence to the Mediterranean diet, rich in fruits, vegetables, plant-based fats, legumes, and polyphenols, is associated with decreased oxidative stress and significant reductions in endometriosis-related pain over 6 months (including dyspareunia and dyschezia). It could also help to improve the quality of life and long-term health of endometriosis patients; however, further studies are required [12]. Polyphenols (resveratrol, luteolin, genistein, quercetin, and curcumin) down-regulate NF-kappaB signaling.. They also suppress pro-inflammatory cytokine release, inhibit angiogenesis, and cell proliferation in ectopic lesions [3]. In rats with induced endometriosis, administration of quercetrin lowered serum E2 and TNF-α levels and also reduced the size of endometrial implants [13,14]. In a study by Signore et al., a quercetrin-based dietary supplement significantly decreased serum PGE2 levels and reduced the size of endometriosis foci after 3 months of supplementation in endometriosis patients [8,13].In a randomized, placebo-controlled clinical trial, 8 weeks of vitamins C and E (powerful antioxidants) supplementation was found to reduce pain in patients with endometriosis [3,15].These bioactive compounds may also act synergistically with omega-3 fatty acids and other anti-inflammatory nutrients to rebalance the immune microenvironment and attenuate nociceptive signaling. Although past findings seem promising, evidence remains heterogeneous, and there is a need for carefully and thoroughly designed randomized controlled trials to establish the efficacy, optimal dosage, and long-term safety of antioxidant and polyphenol-rich products in the management of endometriosis.

2. Gluten

Gluten could also influence endometriosis-related symptoms. Endometriosis patients often report gastrointestinal symptoms. They can experience constipation, diarrhea, bloating, and abdominal cramps, which can lead to misdiagnosis because of their association with irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), and celiac disease (CD) [16]. Chiaffarino et al., in their meta-analysis, highlight a two or more times higher risk of IBS in women with endometriosis compared to women without it [17]. Celiac disease and endometriosis are associated, and that is why a gluten-free diet (GFD) can help alleviate endometriosis-related symptoms. Although this type of diet is frequently suggested as a way to manage endometriosis-related symptoms, causality remains unproven. It is also crucial to note that because of the overlap among IBS, CD, and endometriosis, the gastrointestinal symptoms may be caused by IBS rather than endometriosis itself. Epidemiological data from the large-scale Nurses' Health Study II, which followed 81 861 premenopausal women for 24 years and identified 3,810 laparoscopically confirmed cases, showed no significant association between gluten intake and endometriosis risk after sensitivity analyses, suggesting that gluten is unlikely to be a strong etiological or symptom-atological factor [16,18]. The most frequently cited intervention, a prospective cohort study by Marziali et al., reported that 75% of women (156/207) experienced symptom improvement after 12 months on a gluten-free diet; however, there are essential limitations- none of the patients had been tested for CD before enrollment. Hence, it is possible that improvement was due to undiagnosed CD in some patients. In addition, 88 participants withdrew within 2-3 weeks due to gastrointestinal side effects, and only responders were included, thereby introducing considerable bias [7,16,19]. Notably, randomized, double-blind, placebo-controlled crossover study of 59 individuals with self-reported gluten sensitivity (with celiac disease ruled out beforehand) indicate that rapidly fermentable fructans (a subtype of FODMAPs present in grain-based foods) rather than gluten itself are more likely to trigger symptoms, which leads to a conclusion that FODMAP restriction may be more beneficial than gluten elimination in this patient population[16,20]. Moreover, gluten consumption is highly susceptible to placebo and nocebo effects, which have been demonstrated in recent placebo-controlled trials. Expectancy alone can significantly influence gastrointestinal symptom reporting after gluten consumption, further questioning the validity of uncontrolled finding[16,21]. It is also crucial to underline the potential consequences of following a gluten-free diet. Long-term adherence to GFD has been associated with impaired dietary quality, reduced fiber intake, adverse shifts in gut microbiome, increased risk of obesity and metabolic syndrome, and higher

food costs. A gluten-free diet is also more challenging and socially isolating than a regular diet [7]. Due to strict dietary restrictions, patients report a lower quality of life [22].
To conclude, current evidence does not support a causal role, and the efficacy of a gluten-free diet is unproven; therefore, a GFD for endometriosis patients should be discouraged unless there is a diagnosis of gluten disorder [7,16].

3. FODMAP

Emerging evidence suggests that a low-FODMAP diet may provide clinically meaningful improvement of gastrointestinal symptoms in women with endometriosis, who, as it was stated above, often experience bloating, diarrhea, constipation, and abdominal pain that are irrespective of bowel infiltration of the disease, as well as reduced quality of life in addition to gynecological complaints. In the EndoFOD trial, 60% of participants responded to a 28-day low-FODMAP intervention. The researchers found improvements in abdominal pain, bloating, stool consistency, and disease-specific quality of life [23].

Following a low FODMAP diet may result in lower fiber intake, which is associated with microbiome disturbances, a thinner intestinal mucus layer, and impaired barrier function. Low fiber intake results in reduced production of short-chain fatty acids (SCFA), which is a pro-inflammatory condition associated with an increased risk of irritable bowel disease, colon cancer, and other gastrointestinal disorders. The low-FODMAP diet should be implemented carefully with the guidance of nutrition specialists to avoid nutritional risks of a restrictive diet [7,24].
Despite promising short-term outcomes, strict adherence to the low-FODMAP diet carries potential risks, including adverse effects on the gut microbiota and nutritional adequacy. Therefore, this type of restrictive diet recommendation should be based on a positive medical diagnosis and implemented with care to minimize risks [7,24].

4. Vitamin D

Vitamin D has been discussed as a modulator of endometriosis, with experimental, observational, and interventional studies suggesting both biological plausibility and clinical relevance. Preclinical research has shown that vitamin D reduces endometriotic lesion growth by modulating key pathogenic pathways. Animal models have demonstrated a decrease in

lesion size or volume following vitamin D supplementation [25].In vitro studies reported suppressed expression of matrix metalloproteinases (MMP-2, MMP-9), reduced production of pro-inflammatory mediators including interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-17 (IL-17) and prostaglandin E2 (PGE2) and inhibition of angiogenesis through reduced expression of vascular endothelial growth factor-A (VEGF-A) genes and inhibition of the nuclear factor kappa B (NF-kappa B) signaling [25,26]. In one experimental study, Miyashita et al. incubated ectopic endometrial stromal cells (EESCs) with 1,25-(OH)2D3 and observed significantly lower IL-8 expression, cyclooxygenase-2 (COX-2), and PGE2 level[27]. Observational studies further indicate that women with endometriosis often present with significantly lower plasma and peritoneal [28,29] vitamin D levels, and deficiency has been associated with increased lesion size [30] and greater pain severity[25].

However, clinical data remain inconclusive. A randomized, double-blind trial involving 39 women aged 15-40 years who underwent laparoscopic treatment (with a VAS test score of 3 or more for dysmenorrhea and/or pelvic pain at second menses after laparoscopy) found no significant differences at 24 weeks after surgical treatment between weekly supplementation with 50 000 IU of vitamin D3 for 12 weeks and placebo in terms of pelvic pain (VAS 0,84 ± 1,74 vs. 0,68 ± 1,70, p=0,513) or dysmenorrhea (2,10 ± 2,33 vs. 2,73 ± 2,84, p=0,45) [31]. In contrast, other clinical studies cited in systematic reviews have reported improvement in pain scores among women with low baseline vitamin D levels. Mehdizadekhashi et al. found that vitamin D supplementation significantly decreased pelvic pain (β = -1,12; 95% CI: -2,1, -0,09; p = 0,03 compared with placebo [25,32].

In 2020, Qiu et al. associated low vitamin D levels with an increased risk of endometriosis and symptom severity [33].

A recent umbrella review of nutritional interventions also highlighted a mild protective association between higher dairy consumption- an essential source of vitamin D- and reduced endometriosis risk (total dairy: RR 0,874, 95% CI 0,81-0,95, p=0.001; high-fat dairy: RR 0,590, 95% CI 0,81-0,99, p=0.025) [34]. Collectively, these findings suggest that vitamin D may influence endometriosis through its immunomodulatory and anti-inflammatory properties; however, the clinical data are inconsistent, and the overall quality of evidence is low, underscoring the need for larger, well-designed randomized trials.

5. Dairy

As mentioned above, higher dairy intake is mildly protective against endometriosis. It might be due not only to vitamin D but also to the high calcium content in dairy products. They promote the down-regulation of growth-promoting factors (e.g., insulin-like growth factor I) and the up-regulation of growth-inhibitory modulators (e.g., transforming growth factor β). Dairy also contains other anti-inflammatory components that help decrease inflammatory markers such as tumor necrosis factor α (TNFα), IL-6, and reactive oxygen species (ROS), which could reduce the risk of endometriosis [34,35].

6. Meat

There have been many suggestions to avoid or significantly reduce red meat consumption because of the presence of estrogens due to the hormonal treatment of cattle in some countries (forbidden in all European countries). Red meat is also rich in heme-iron, which is potentially pro-oxidant and pro-inflammatory [7]. This point of view has been supported by a prospective cohort study by Yamamoto et al., who found a positive association between meat consumption and the risk of endometriosis. [36]. Other studies state that this association occurs only when at least seven servings of red meat per week are consumed [7,37].

The Nurses' Health Study II shows that consuming >2 red meat servings per day was associated with a 56% greater risk of developing endometriosis than consuming less than one serving per week [4,37].

It is also essential to consider other factors that may influence the data, such as excess weight and obesity among high meat consumers, which can cause low-grade inflammation and are associated with elevated estradiol levels [7]. To sum up, there is insufficient evidence to establish a causal role for red meat in the etiology of endometriosis.

7. Omega-3

Omega-3 polyunsaturated fatty acids (PUFA) have been increasingly recognized as potential modulators of endometriosis pathophysiology due to their anti-inflammatory and immunoregulatory properties. Biochemically, omega-3 fatty acids compete with arachidonic acid (an omega-6 fatty acid) for cyclooxygenase and lipooxygenase enzymes, thereby reducing the synthesis of pro-inflammatory prostaglandins (PGE2, PGF2α) and leukotrienes, while

enhancing the production of less inflammatory eicosanoids such as PGE3, ultimately reducing pelvic pain, uterine contractility, and dysmenorrhea [1,38]. Epidemiological studies, including the large prospective Nurses' Health Study II, presented that higher intake of long-chain omega-3 PUFAs was associated with a significantly reduced risk of laparoscopically confirmed endometriosis (women with the highest intake were 22% less likely to develop endometriosis compared to those with the lowest intake), whereas trans-fat intake increased the risk. [6,37,39–41]. In another study, an inverse correlation was observed between the consumption of saturated fatty acids, monounsaturated fatty acids, and trans fats and the risk of endometriosis [1,37]. However, in the Nurses' Health Study II, no association was found between monounsaturated fat intake and the risk of endometriosis [6,39]. Similarly, case-control and cross-sectional studies reported that women with higher consumption of fish oils or omega-3-rich foods had a lower prevalence of endometriosis compared with controls [1]. Another trial reported that the consumption of fish oil capsules and vitamin B12 reduced the severity of dysmenorrhea among affected women [5,42]. Systematic reviews state that omega-3 supplementation and an anti-inflammatory diet rich in PUFAs might improve quality of life and reduce pain [4,7]. Some trials reported no significant associations between fish intake or PUFA supplementation and decreased risk [1]. Current evidence therefore suggests a plausible protective role of omega-3 PUFAs in reducing inflammation, pain, and possibly disease progression in endometriosis. However, larger randomized controlled trials are required to confirm causality and establish optimal dietary or supplemental regimens.

8. Fiber

Dietary fiber could also influence the risk of endometriosis. It is because of its effects on estrogen metabolism and systemic inflammation. Fiber accelerates intestinal transit time. It increases fecal excretion of estrogens and reduces enterohepatic recirculation. This may lower circulating estrogen levels and decrease proliferation in ectopic endometrial tissue[3]. Fiber fermentation produces short-chain fatty acids, which are anti-inflammatory[7]. The hormone-dependent and inflammatory nature of endometriosis proves that fiber intake could reduce disease risk or symptom severity. A high-fiber diet is also rich in complex carbohydrates and has a low glycemic index. Insulin is known to stimulate endometrial proliferation, which is why a diet with a high glycemic index, causing rapid spikes in insulin levels, may be associated with an elevated endometriosis risk [5]. However, clinical data remain inconsistent. Trabert et al. were unable to determine whether a high-fiber diet has a significant

effect on endometriosis [5,37]. In contrast, Savaris et al. found that women with endometriosis consumed more fiber than controls, suggesting that higher fiber intake was associated with an increased risk of endometriosis [5,43]. In a case-control study, Parrazinni et al. state that women who consumed large amounts of green vegetables and fresh fruit had a lower risk of endometriosis [1,44]. Increased fruit fiber intake was associated with a lower risk of endometriosis, also in the study by Harris et al. [3,45]. There is insufficient evidence to recommend either increased or restricted fiber intake for endometriosis prevention or management of endometriosis.

Conclusions

Endometriosis is a chronic, estrogen-dependent, inflammatory disorder with multifactorial etiology involving hormonal, immunological, inflammatory, and genetic factors. Conventional strategies such as hormonal therapy, surgical excision, and pain management remain the core of endometriosis treatment; however, they are often associated with incomplete symptom relief, adverse effects, and high recurrence rates. Consequently, there has been an increase in the usage of self-management strategies, including diet modifications. Dietary interventions have gained increasing attention as potential adjunctive strategies to alleviate symptoms and improve the quality of life in affected women.

This review highlights that diet may influence endometriosis through several mechanisms, including modulation of systemic inflammation, oxidative stress, estrogen metabolism, and immune response. Diets rich in omega-3 PUFAs, antioxidants, and polyphenols, vegetables, fruits, and dairy products,can be beneficial for endometriosis patients by reducing pain intensity, inhibiting pro-inflammatory cytokines, and decreasing oxidative damage. On the contrary, high consumption of red meat and trans fats has been associated with an increased risk of endometriosis or worsening of symptoms. There is a biologically plausible relationship between vitamin D and endometriosis. Experimental studies support its anti-inflammatory and immunomodulatory effects, yet randomized clinical trials remain inconclusive. Furthermore, elimination diets such as gluten-free and low-FODMAP may alleviate gastrointestinal symptoms in selected patients, though they should be implemented cautiously under professional supervision to avoid nutritional deficiencies.

To conclude, current data suggest that adopting a balanced, anti-inflammatory diet appears beneficial for managing endometriosis symptoms. However, to date, the evidence base remains limited, inconsistent, and largely observational. Further large-scale, well-designed, randomized

controlled trials are required to clarify the causal relationship between dietary factors and endometriosis and to develop nutritional recommendations to complement conventional therapies.

Disclosure

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Conflicts of Interest

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