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ANTI-INFLAMMATORY DIETARY PATTERNS IN ENDOMETRIOSIS: MECHANISMS, CLINICAL EVIDENCE, AND PERSPECTIVES FOR FUTURE RESEARCH

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ABSTRACT

Purpose: The aim of this article was to provide an in-depth review of the current literature and research on the role of anti-inflammatory dietary patterns in the treatment of endometriosis.
Methodology: A thorough and extensive review of the literature was conducted using various scientific databases, such as PubMed, Google Scholar and Scopus. During the selection process, particular attention was paid to clinical trials, systematic reviews and meta-analyses concerning dietary patterns, specific nutrients and bioactive anti-inflammatory compounds in relation to the risk, severity of symptoms and progression of endometriosis.
Findings: Evidence indicates that anti-inflammatory diets may help manage endometriosis symptoms. However, the few available clinical studies are varied and often low in quality. There is also a lack of long-term randomized controlled trials with standardized protocols and biomarker endpoints, making it difficult to confirm causality or create clinical guidelines.
Conclusion: Anti-inflammatory diets may help reduce endometriosis symptoms by lowering inflammation, affecting oestrogen metabolism, and regulating the gut microbiome—factors central to disease progression. While observational studies and small trials suggest benefits from diets rich in whole foods and low in red meat and trans fats, more advanced clinical trials are needed. The authors highlight the importance of further research using biomarkers and personalized nutrition to create evidence-based dietary guidelines. Future studies should assess diet alongside inflammation, hormone, and microbiome profiles to support tailored nutritional strategies for endometriosis treatment.

KEYWORDS

Endometriosis, Anti-Inflammatory Diet, Mediterranean Diet, Omega-3 Fatty AIDS, Estrogen Metabolizm, Inflammation, Gut Microbiome, Dysmenorrhea

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1. Introduction

Burden of endometriosis

Endometriosis is a chronic condition in which the lining of the womb (endometrium) grows and develops outside the uterine cavity. This misplaced lining undergoes changes during the menstrual cycle similar to those occurring in the lining inside the uterine cavity. Endometriosis is most common in women of reproductive age (6–10%), and in women with pelvic pain syndrome or infertility, the prevalence can be as high as 50%. [1] Endometriosis lesions can be found in various locations within the abdominal cavity; lesions on the ovaries, fallopian tubes and the uterus itself are relatively common (endometrial lesions are found on the outer surface of the uterus). Endometrial cells can also be found in the bladder, intestines and uterosacral ligaments – depending on their location and severity, they are associated with varying degrees of pain. The most characteristic symptoms associated with endometriosis include pelvic pain, painful sexual intercourse, painful urination or bowel movements, and painful, prolonged and heavy periods. The pain stems from the fact that endometrial tissue located in an abnormal site proliferates and sheds during the menstrual cycle but cannot be expelled from the body. This leads to chronic inflammation, adhesions and endometriomas. Many patients with endometriosis also struggle with infertility, as the condition can negatively affect the anatomy of the reproductive organs or the environment necessary for fertilisation and embryo implantation. Beyond the physical symptoms, endometriosis can have a significant impact on quality of life, disrupting daily activities, work, relationships and emotional well-being due to persistent pain and uncertainty regarding fertility.

Limitations of hormonal and surgical management

Although both hormonal and surgical treatments are widely used to manage endometriosis, each of these approaches has significant limitations. [1] Hormonal therapies, such as oral contraceptives, progestogens and GnRH analogues, are primarily aimed at alleviating symptoms by lowering oestrogen levels and reducing menstrual bleeding. However, these therapies do not eliminate endometriosis lesions, may cause side effects (e.g. mood swings, bone loss, intermenstrual bleeding), and symptoms often recur after treatment is stopped. Surgical treatment, including excision or ablation of endometriosis lesions, may in some cases provide symptom relief and improve fertility, but is associated with risks such as surgical complications, the formation of adhesions and recurrence of the disease. Furthermore, repeated surgery may increase the risk of reduced ovarian reserve and adversely affect fertility. Consequently, neither hormonal nor surgical therapy provides a definitive cure, and individualised, multidisciplinary treatment is essential.[2]

Growing interest in dietary modification

In recent years, there has been growing scientific and clinical interest in the role of dietary changes as a complementary approach to the treatment of endometriosis. This interest stems from emerging evidence suggesting that specific dietary patterns and nutrients may influence inflammation, immune system function and hormonal balance – key factors associated with the pathophysiology of endometriosis. [2] Observational studies have shown that a diet rich in fruit, vegetables, omega-3 fatty acids and whole grains, and low in red meat, alcohol and trans fats, may be associated with a reduced risk of developing or experiencing worsening symptoms related to endometriosis. Although robust randomised controlled trials remain limited, many patients seek dietary interventions as a non-pharmacological strategy for pain relief and improving quality of life. As a result, nutritional counselling is increasingly recognised as a valuable complement to conventional pharmacological and surgical treatments, although further research is needed to establish evidence-based guidelines.

2. Pathophysiology of Endometriosis

The pathophysiology of endometriosis is multifactorial and has not yet been fully elucidated. Currently, three forms of endometriosis can be distinguished: ovarian, peritoneal and deep-seated. Several theories have been proposed, including the retrograde menstruation theory, according to which cells of the endometrium travel backwards into the pelvic cavity; the theory of spread via the vascular and lymphatic systems, suggesting spread to distant sites via blood or lymph vessels; the theory of peritoneal metaplasia, in which peritoneal cells transform into tissue similar to the endometrium; and the 'immune deficiency' theory, suggesting a dysfunction of the peritoneal immune system, specifically its phagocytic and cytotoxic activity. [1] Furthermore, it is believed that genetic and epigenetic factors influence individual susceptibility. It is likely that a combination of these mechanisms underlies the development and progression of endometriosis. [3]

This section sets up the biological plausibility for diet intervention.

Elevated cytokines (IL-6, TNF-α, IL-1β)

Elevated levels of cytokines such as interleukin-6 (IL-6), tumour necrosis factor alpha (TNF-α) and interleukin-1 beta (IL-1β) play a key role in the inflammatory pathogenesis of endometriosis. In women affected by this disease, peritoneal macrophages secrete excessive amounts of these cytokines, which promotes the development of chronic inflammation in the microenvironment. At the level of biological mechanisms, these cytokines promote the proliferation and survival of ectopic endometrial cells, enhance cell adhesion and promote angiogenesis through the activation of the NF-κB and JAK/STAT signalling pathways. They also increase the expression of growth factors (e.g. VEGF), matrix metalloproteinases and adhesion molecules, which promotes tissue invasion and the establishment of pathological changes. [3] Clinically elevated cytokine levels correlate with pain, disease progression and infertility, and their dysregulation underlies immune dysfunction, including, among other things, reduced NK cell activity and impaired clearance of ectopic cells. Therapies targeting these cytokines and their signalling pathways may represent a promising treatment option for endometriosis.

Prostaglandins (PGE2)

Elevated levels of prostaglandins, including PGE2 and PGF, are found in endometriosis lesions compared with healthy tissue. These prostaglandins, particularly PGE2, play a significant role in the pathogenesis and manifestation of endometriosis symptoms through their involvement in inflammatory processes. From a mechanistic perspective, prostaglandins released from endometriotic implants into the peritoneal fluid and blood may increase uterine smooth muscle tone and reduce uterine perfusion, leading to pain and dysmenorrhoea. Furthermore, these effects may disrupt fallopian tube motility and the implantation

process, whilst increased prostaglandin activity in the ovary may lead to corpus luteum dysfunction, increasing the risk of infertility. Clinically, these findings highlight the importance of prostaglandin-induced inflammation in the development of pain and infertility in endometriosis, suggesting inhibitors of their synthesis as a potential treatment strategy. [4]

COX-2 overexpression

Increased expression of COX-2 (cyclooxygenase-2) plays an important role in the pathogenesis of endometriosis, as it enhances the production of prostaglandin E2 (PGE2), which is responsible for pain, inflammation and cell proliferation. High levels of COX-2 in disease foci increase local PGE2 synthesis, which promotes chronic inflammation and stimulates aromatase activity, leading to increased oestrogen production. This mechanism creates a positive feedback loop that supports the development of lesions and limits the effectiveness of the immune response. Clinically, this manifests as symptoms such as pelvic pain and dysmenorrhoea, which justifies the use of NSAIDs and COX-2 inhibitors as treatments for patients affected by this condition. [3]

Oxidative stress

Oxidative stress plays a key role in the inflammatory pathogenesis of endometriosis, resulting from an imbalance between reactive oxygen species (ROS) and antioxidant defence mechanisms. This phenomenon is often associated with the breakdown of red blood cells and the activation of macrophages in the peritoneal cavity, leading to increased ROS production. These molecules act as inflammatory mediators, promoting cell proliferation and the release of pro-inflammatory cytokines, growth factors and angiogenesis. Excess iron further exacerbates ROS generation, intensifying tissue damage and the formation of adhesions. High levels of oxidative stress markers in women with endometriosis correlate with disease severity and symptoms such as pelvic pain and infertility, indicating its significance both in the pathogenesis and as a potential therapeutic target in the treatment of endometriosis. [5]

NF-κB activation

The activation of NF-κB plays a significant role in the development of inflammation in endometriosis. Increased expression of this factor in peritoneal macrophages and stromal cells leads to heightened production of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and factors that promote angiogenesis, such as VEGF. Together with increased ROS levels, this contributes to the maintenance of chronic inflammation, tissue damage and pain. Clinically, this translates into the persistence of pathological changes, immune dysfunction and resistance to apoptosis, which is associated with chronic pain and infertility. Targeting NF-κB represents a promising direction for the treatment of endometriosis. [3]

Estrogen–inflammation feedback loop

The feedback loop between oestrogens and inflammation plays a significant role in the development and progression of endometriosis, linking elevated oestrogen levels with chronic local inflammation. Increased expression of aromatase (CYP19A1) leads to enhanced oestrogen synthesis and activation of ERα and ERβ receptors, which promotes cell proliferation and the development of pathological lesions. A hyper-oestrogenic environment stimulates COX-2 expression and prostaglandin production, whilst NF-κB activation enhances the inflammatory response by increasing cytokine production and the influx of immune cells. This mechanism perpetuates both inflammatory and oestrogenic processes, contributing to pain and disease progression. From a clinical perspective, this highlights the need for therapies that act simultaneously on both pathways. [6]

Gut microbiome dysbiosis

Disruptions to the gut microbiome are increasingly linked to the development of inflammation in endometriosis. Changes in the composition of the microbiota can lead to increased intestinal permeability and the entry of microbial products, such as lipopolysaccharides (LPS), into the bloodstream, which activates the immune system and intensifies the production of pro-inflammatory cytokines. Elevated levels of these cytokines are also observed in the peritoneal fluid of patients with endometriosis. Chronic inflammation promotes the adhesion, invasion and survival of endometrial cells outside the uterine cavity. From a clinical perspective, this points to the potential of therapies targeting the gut microbiome, such as probiotics and dietary interventions, and highlights the role of the gut-vaginal axis in the course of this disease.[3]

3.What Is an Anti-Inflammatory Diet?

An **anti-inflammatory diet** is a way of eating that focuses on reducing chronic inflammation in the body—a process linked to many health problems, including heart disease, diabetes, obesity, and some autoimmune conditions. This dietary approach emphasizes whole, minimally processed foods, healthy fats, and a variety of plant-based nutrients, while limiting foods known to promote inflammation, such as red meat, processed foods, and added sugars. Several dietary patterns and principles are commonly associated with anti-inflammatory effects:

Mediterranean diet

This traditional dietary pattern from the countries bordering the Mediterranean Sea emphasises plant-based foods such as fruit, vegetables, whole grains, pulses, nuts and seeds, with olive oil as the main source of fat. It includes moderate amounts of fish, poultry and fermented dairy products, whilst limiting red and processed meat. The Mediterranean diet is rich in healthy fats, antioxidants and minimally processed foods, and moderate amounts of wine may be consumed with meals; it is associated with improved heart health and metabolism. [7]

Plant-based diets

These diets are based on plant-based foods — fruit, vegetables, whole grains, pulses, nuts and seeds. They may be vegetarian (including dairy and/or eggs) or vegan (excluding all animal products). Plant-based diets are generally high in fibre and low in saturated fat, and following them is associated with a lower risk of heart disease and diabetes. However, they can also lead to reduced intake of certain nutrients, such as vitamin B12, calcium and iodine, so careful planning or the use of supplements is important. [8]

High omega-3 / low omega-6 ratio

Eating more omega-3 fatty acids (from sources such as oily fish, flaxseed and walnuts) and limiting your intake of oils rich in omega-6 (such as corn, soya and sunflower oil) helps to reduce inflammation. Omega-3 fatty acids help reduce inflammation, whilst an excess of omega-6 can exacerbate inflammatory processes. Maintaining this balance can reduce the risk of chronic diseases and support overall health. [9]

Low red meat intake

Reducing red meat intake is a key component of anti-inflammatory diets, which are associated with reduced inflammation and improved fertility. Excessive consumption of red and processed meat promotes an increase in inflammatory markers, whereas limiting or consuming it in moderation has a beneficial effect on health. Anti-inflammatory diets are based mainly on vegetables, fruit, whole grains, nuts, seeds and fish. The accumulated evidence suggests that reducing red meat intake promotes a less pro-inflammatory diet. [10]

High fiber intake

Fibre from fruit, vegetables, whole grains and pulses promotes gut health by supporting a balanced microbiome and producing short-chain fatty acids with anti-inflammatory properties. Increased fibre intake is associated with reduced inflammation, a better immune response and a lower risk of metabolic diseases. [11]

Polyphenol-rich foods

Foods rich in polyphenols — such as blueberries, grapes, green tea, coffee, soya, oats and pistachios — offer significant anti-inflammatory and antioxidant benefits, as well as positive effects on cardiovascular and metabolic health. It is recommended to regularly include these foods in the diet to prevent or treat obesity, type 2 diabetes, cardiovascular diseases and neurodegenerative disorders, as they help reduce inflammation, improve glucose and lipid metabolism, and protect against oxidative stress. In general, a variety of polyphenol-rich foods should form part of a healthy, anti-inflammatory diet. [12]

Low ultra-processed foods

Anti-inflammatory diets recommend limiting the consumption of ultra-processed foods (UPFs), as a high intake of these foods promotes chronic inflammation and increases the risk of conditions such as diabetes, heart disease and cancer. Diets such as the Mediterranean and Nordic diets are based on natural or minimally processed foods, including vegetables, fruit, whole grains, fish and pulses, whilst limiting red meat and UPFs. Limiting processed foods helps to reduce the intake of pro-inflammatory compounds and increase the intake of anti-inflammatory compounds, such as fibre, vitamins and polyphenols. Health guidelines emphasise the importance of this approach for reducing inflammation and improving overall health. [13]

Potential low-FODMAP overlap

The low-FODMAP diet, which involves limiting fermentable carbohydrates, can help reduce gastrointestinal symptoms such as bloating and pain, particularly in people with IBS or endometriosis. Although it is not a typical anti-inflammatory diet, it shares certain characteristics with it, such as limiting processed foods and carefully selecting plant-based ingredients. However, it is more restrictive and should not

be followed long-term, unlike anti-inflammatory diets, which focus on a balanced and nutritious way of eating. Combining elements of both diets may be beneficial, but requires supervision by a specialist to avoid deficiencies. [14]

In summary, the anti-inflammatory diet is not a single, standardised dietary model or a rigid plan, but rather refers to a set of principles and various dietary patterns, such as the Mediterranean diet or the plant-based diet. What they have in common is an emphasis on consuming wholesome, minimally processed foods rich in nutrients, including vegetables, fruit, fibre and polyphenols, as well as healthy fats, whilst limiting red meat and highly processed foods. Although individual approaches may differ in their specific recommendations, their main aim is to reduce inflammation, support overall health and lower the risk of chronic diseases. This approach is flexible and can be adapted to individual needs, for example through modifications such as a low-FODMAP diet for people with gut sensitivity.

4.Mechanistic Links Between Diet and Endometriosis

Omega-3 Fatty Acids

Omega-3 fatty acids are being extensively studied for their potential role in modulating inflammatory processes, including those associated with endometriosis. Polyunsaturated fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), exhibit anti-inflammatory effects by regulating the synthesis of eicosanoids and other inflammatory mediators. A key mechanism of their action is the reduction in the production of pro-inflammatory prostaglandins derived from arachidonic acid, which play a significant role in pain, inflammation and the progression of pathological changes. By incorporating into cell membranes, omega-3 fatty acids compete with arachidonic acid for enzymatic reactions occurring in the cyclooxygenase and lipoxygenase pathways, which leads to a reduction in the synthesis of pro-inflammatory prostaglandins and leukotrienes, thereby alleviating the inflammatory response.

The authors of the meta-analysis entitled ‘The effect of omega-3 polyunsaturated fatty acids on endometriosis’ assessed the significance of these compounds in the course of the disease. The authors concluded that omega-3 fatty acids may contribute to reducing inflammation in patients with endometriosis, mainly by lowering levels of pro-inflammatory cytokines such as TNF-α, IL-6 and IL-1. The results obtained demonstrate promising anti-inflammatory effects, which require further verification in subsequent studies. [15]

Available clinical studies suggest that increasing the intake of omega-3 fatty acids, particularly in the form of fish oil, may alleviate the symptoms of painful periods in women with endometriosis. This effect is linked to their anti-inflammatory properties and their ability to reduce the production of prostaglandins, which are responsible for triggering pain. It has also been shown that supplementation can reduce the intensity of pain and limit the need for painkillers. [16]

The anti-inflammatory effects of omega-3 fatty acids, combined with their role in regulating prostaglandin synthesis, may help to alleviate symptoms and potentially influence the course of the disease in women with endometriosis. [17]

Fiber and Estrogen Metabolism

Dietary fibre plays a significant role in regulating oestrogen metabolism and thus influences the pathophysiology of endometriosis, which is an oestrogen-dependent condition. Its action involves, amongst other things, increasing the excretion of oestrogens in the intestine by binding them, which limits their reabsorption and promotes their elimination from the body via the stools, leading to a reduction in their concentration in the blood. [18] This process contributes to a reduction in blood estrogen levels, potentially decreasing estrogen-stimulated proliferation of ectopic endometrial tissue.

One of the observational studies cited in this paper showed that a higher intake of dietary fibre was associated with a more than 40% lower risk of endometriosis (odds ratio 0.588; $p = 0.041$). [18]

It has been shown that increased dietary fibre intake can reduce blood oestrogen levels by approximately 10–25%, which may help to reduce the risk of developing endometriosis and alleviate its symptoms. Furthermore, studies indicate that a higher intake of fibre, particularly of plant origin, is associated with a lower risk of developing this condition. [19]

At the same time, dietary fibre may modulate the enterohepatic circulation of oestrogens by influencing the composition and activity of the gut microbiota, including a reduction in the abundance and enzymatic activity of β-glucuronidase-producing bacteria. This enzyme is responsible for the deconjugation of oestrogens in the intestine, which enables their reabsorption into the bloodstream. [20] Reducing its activity promotes increased excretion of oestrogens, which may contribute to alleviating the hyperoestrogenic microenvironment characteristic of endometriosis.

In summary, these mechanisms provide a biologically plausible link between a high-fibre diet and a potential reduction in disease activity, although direct clinical evidence remains limited.

Polyphenols

Dietary polyphenols, such as resveratrol, curcumin and epigallocatechin gallate (EGCG), have attracted interest due to their potential therapeutic use in endometriosis, stemming from their potent anti-inflammatory and anti-proliferative properties. [21]

The key mechanism of their action involves the inhibition of nuclear factor kappa B (NF-κB), which is the main transcription factor regulating the expression of pro-inflammatory cytokines, adhesion molecules and enzymes such as COX-2, levels of which are elevated in endometriosis lesions. Inhibition of the NF-κB pathway may lead to a reduction in inflammation, which contributes to the persistence of pathological changes and pain. [22]

Furthermore, polyphenols exhibit anti-angiogenic properties by reducing levels of the vascular endothelial growth factor (VEGF) and other mediators of neovascularisation, thereby limiting the blood supply necessary for the formation and maintenance of ectopic endometrial tissue. [23]

Furthermore, resveratrol, curcumin and EGCG have demonstrated, in laboratory and animal studies, the ability to inhibit the proliferation of and induce apoptosis in endometriosis cells, which translates into a reduction in their invasiveness. [23]

It is worth noting that the results of in vitro studies and animal models indicate that EGCG and its derivatives (pro-EGCG) may reduce the number, size and volume of endometriosis lesions, as well as limit the fibrotic process by modulating various molecular factors and signalling pathways. These findings provide a basis for considering the use of plant extracts, including those derived from green tea, in clinical trials for the treatment of endometriosis. It should be noted, however, that the available data are limited, which highlights the need for further research in this area. [24]

In conclusion, these mechanisms suggest the potential of polyphenol-rich diets and appropriately selected supplementation as an adjunct to controlling disease progression; however, sufficient clinical evidence is still lacking.

Gut Microbiome

The gut microbiome has been recognised as a key factor linking diet to endometriosis, as it influences oestrogen metabolism, the integrity of the intestinal barrier and the functioning of the immune system.

A central concept is the ‘estrobolome’, which refers to the set of genes found in the gut microbiome that are responsible for oestrogen metabolism. It includes, amongst others, genes of bacteria producing enzymes such as β-glucuronidase and β-glucosidase, which remove conjugating groups from oestrogens, enabling their reabsorption into the bloodstream and contributing to a hyperoestrogenic state, which may promote the development of endometrial lesions. The estrobolome therefore plays a vital role in maintaining hormonal balance by regulating oestrogen metabolism. Changes in the microbiome, resulting for example from diet, can influence oestrogen levels throughout the body. At the same time, an imbalance in the microbiota (dysbiosis) can lead to increased intestinal permeability, allowing bacterial toxins such as lipopolysaccharides (LPS) to enter the bloodstream. Consequently, this may trigger low-grade chronic inflammation and activate immune mechanisms associated with the development of endometriosis. An abnormal composition of the gut microbiome (dysbiosis) may affect the functioning of the estrobolome, causing fluctuations in oestrogen levels. This is particularly significant in conditions such as endometriosis, where excessive activity of the estrobolome may exacerbate hyperoestrogenism, promote inflammation and contribute to disease progression. [25]

Furthermore, the gut microbiota plays a vital role in regulating the immune system, influencing the balance between pro-inflammatory and immunoregulatory responses, including T-cell differentiation and cytokine production. [26] Through the interaction of these mechanisms, the gut microbiome acts as a key link between diet and the inflammatory and hormonal background observed in endometriosis; however, causal relationships and potential therapeutic applications require further investigation. [27]

Red Meat and Trans Fats

A high intake of red meat and trans fats is associated with increased inflammation, which may contribute to the development and progression of endometriosis. [28]

In particular, the consumption of red meat — especially processed and high-fat varieties — correlates with increased levels of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6) and tumour necrosis factor alpha (TNF-α). These markers are also characteristic of the inflammatory environment associated with endometrial changes. [29,30]

Similarly, trans fatty acids — found mainly in highly processed foods — may exacerbate systemic inflammation by increasing oxidative stress and activating pro-inflammatory signalling pathways, such as nuclear factor kappa B (NF-κB). [31]

Studies confirm these mechanistic relationships, indicating that higher intake of red meat and trans fats is associated with an increased risk of endometriosis, whilst limiting their consumption may have a protective effect. [32]

Although the available data do not allow for unequivocal confirmation of a cause-and-effect relationship, the observed association between these dietary components and the severity of inflammation and the risk of disease suggests that they may act as modifiable factors in the treatment of endometriosis.

5. Clinical Evidence: What Do Human Studies Show?

Human studies suggest that diet may influence the severity of endometriosis symptoms; however, the available clinical evidence remains preliminary and methodologically inconsistent. The most reliable data come from prospective cohort studies, which primarily address the risk of developing the condition rather than therapeutic effects. The Nurses' Health Study II demonstrated that higher intake of long-chain omega-3 fatty acids was associated with a lower risk of developing endometriosis, whilst higher intake of trans fats correlated with an increased risk. These findings highlight the importance of the quality of dietary fats as a potential factor in the pathogenesis of the disease. [33,34] Case-control studies and smaller cohort studies also suggest that greater adherence to the Mediterranean diet or generally healthier dietary patterns may be associated with a lower risk of developing endometriosis or reduced severity of pain symptoms. It should be emphasised, however, that such studies are subject to significant limitations, such as recall bias, the possibility of reverse causality, the influence of uncontrolled confounding factors, or selection bias. An additional difficulty is the often delayed diagnosis of the disease and the fact that dietary habits may change after the first symptoms have already appeared. [35,36] Data from intervention studies are even scarcer: small randomised and quasi-experimental trials on omega-3 supplementation and the use of antioxidants suggest a possible improvement in pain and inflammatory markers, but the results remain inconsistent. These studies involve small groups of participants, are characterised by a significant placebo effect, and the interventions themselves vary in terms of dose, composition and duration. For example, a randomised placebo-controlled trial involving adolescents and young adults did not demonstrate a clear advantage of fish oil over placebo in reducing pain. In contrast, studies on antioxidants suggest a reduction in chronic pelvic pain and a decrease in the levels of certain inflammatory mediators. [37,38] The evidence suggesting a beneficial effect of the Mediterranean diet is promising; however, it is based mainly on observational or uncontrolled studies, rather than on well-designed randomised trials with sufficient statistical power. This limits the ability to draw definitive conclusions regarding a cause-and-effect relationship. [35,36] In the scientific literature, the most commonly assessed endpoint is pain intensity, whilst disease progression is rarely analysed in the context of dietary interventions in humans. Furthermore, data on fertility are largely limited or indirect in nature, making it difficult to assess whether changes in diet affect the course of the disease, rather than merely the subjective perception of symptoms. [39] Generally speaking, the main advantages of human studies include: their biological validity, the reproducibility of results across different types of studies, and the low risk associated with dietary changes. At the same time, the interpretation of these data is hampered by a number of limitations, such as the heterogeneity of the study populations, the lack of uniform diagnostic criteria, short follow-up periods, insufficient control of concomitant hormonal or surgical treatment, as well as the lack of a clear definition of 'anti-inflammatory' diets or dietary patterns based on the Mediterranean diet. [36]

6. Gaps in the Literature

Despite growing interest in the role of diet in the course of endometriosis, significant gaps in the available literature limit the reliability of existing findings and highlight the need for further research. In particular, there is a lack of well-designed, long-term randomised controlled trials that would assess the impact of specific dietary interventions on key clinical outcomes, such as pain severity, disease progression or fertility. [40, 36] A significant proportion of the available evidence is based on observational studies and short-term interventions, which often employ imprecise methods of dietary assessment based on self-reporting, making them susceptible to recall bias and incorrect data classification. [41] Furthermore, there is no uniform definition or standard protocol for an 'anti-inflammatory' diet in the context of endometriosis, which leads to considerable variability between studies and hinders the comparison and replication of the results obtained. [36] Another significant limitation is the reliance primarily on subjective endpoints, whilst relatively few

studies utilise objective biomarker-based indicators, such as inflammatory cytokines, hormonal profiles or markers of oxidative stress, which could help to elucidate the mechanisms underlying the observed associations. [18] Furthermore, the complex relationships between diet, the gut microbiome and endometriosis are still not fully understood, even though they may play a significant role in oestrogen metabolism and the regulation of the immune system. [42] Finally, a growing body of evidence suggests that individual differences in metabolic, hormonal and microbiological profiles may shape the body's response to diet, underscoring the importance of a personalised approach to nutrition. [40] Addressing these gaps will be crucial for the further development of this field, enabling a transition from observed correlations to a better understanding of the mechanisms involved and the formulation of dietary recommendations based on sound scientific evidence.

7. Clinical Implications

From a clinical perspective, current evidence suggests that dietary changes may serve as a supportive measure in the treatment of endometriosis, but should not replace standard pharmacological or surgical methods. Although these forms of therapy remain the cornerstone of management, a growing body of evidence suggests that an anti-inflammatory diet may help alleviate symptoms – particularly pain – and improve patients' quality of life. [40,36] Importantly, dietary interventions are generally safe, widely accessible and may offer additional metabolic and general health benefits, making them a valuable component of supportive therapy. Observational studies and a few intervention studies indicate that a higher intake of omega-3 fatty acids, fruit, vegetables and fibre, combined with a reduction in red meat and trans fats, may be associated with lower levels of inflammation and reduced symptom severity. [33,41] Given the heterogeneity and limited quality of the available data, dietary recommendations should be formulated with caution and tailored to the individual. Current evidence suggests that a practical approach may involve following a Mediterranean or plant-based diet, based on unprocessed foods, rich in antioxidants and healthy fats, whilst limiting highly processed foods and pro-inflammatory ingredients. [36] In summary, although no clear cause-and-effect relationships have yet been demonstrated, dietary modification appears to be a promising and low-risk adjunctive measure which, when used alongside standard treatment, may support a comprehensive management approach to endometriosis.

8. Future Research Directions

Future research into the role of diet in endometriosis should go beyond the analysis of observational associations alone and focus on well-designed intervention studies based on an understanding of the underlying mechanisms. Above all, randomised controlled trials (RCTs) are needed, utilising standardised dietary models — such as the Mediterranean diet — to assess their impact on key clinical outcomes, including pain severity, disease progression and fertility. [36,40] These studies should include objective endpoints, including through long-term monitoring of inflammatory and hormonal biomarkers, such as cytokine, prostaglandin and oestrogen metabolite profiles, which will allow for a better understanding of the underlying mechanisms. [18] Furthermore, the use of multi-omic approaches — encompassing genomics, metabolomics and microbiome analysis — may provide more detailed insights into the complex relationships between diet, the body's metabolism and the pathophysiology of endometriosis. Given the heterogeneous nature of this disease, future studies should employ stratified approaches that allow for the identification of patient subgroups based on inflammatory phenotype, hormonal profile or microbiome composition. This will enable the identification of individuals most likely to benefit from specific dietary interventions. Finally, analysing the potential synergistic effects of combining dietary interventions with standard hormonal treatment may contribute to the development of more comprehensive and personalised therapeutic strategies. Such an approach will be crucial for the development of this field towards precision nutrition and the creation of dietary recommendations based on robust scientific evidence in the treatment of endometriosis.

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