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# Potential Role of Microplastics and Nanoplastics in the Pathogenesis of Endometriosis: An Environmental Health Narrative Review

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## Abstract

Microplastics and nanoplastics (MNPs) have emerged as ubiquitous environmental contaminants that are increasingly detected in human biological matrices, including blood, urine, placenta, and reproductive tissues [1–3]. Due to their small size, persistence, and physicochemical properties, these particles are capable of entering the human body through

ingestion and inhalation, followed by systemic distribution [4]. Endometriosis is a chronic, estrogen-dependent inflammatory disease affecting approximately 10% of women of reproductive age and is characterized by immune dysregulation, oxidative stress, mitochondrial dysfunction, and altered hormonal signaling [5–7]. Growing evidence indicates that exposure to environmental pollutants may modulate molecular pathways relevant to the development and progression of endometriosis [8]. Experimental studies demonstrate that MNPs can induce oxidative stress, activate inflammatory signaling cascades, disrupt endocrine function, and alter immune cell behavior—mechanisms that overlap with the established pathophysiology of endometriosis [9–12]. Recent reports describing the presence of microplastics in human endometrial tissue further raise concerns regarding direct tissue-level exposure [13]. This narrative review summarizes current PubMed-indexed evidence on human exposure to microplastics and nanoplastics, their biological effects relevant to female reproductive health, and the mechanistic plausibility of their involvement in endometriosis pathogenesis, while highlighting existing knowledge gaps and methodological limitations.

**Keywords:** microplastics; nanoplastics; endometriosis; environmental exposure; inflammation; oxidative stress; reproductive health

## Introduction

Endometriosis is defined as the presence of endometrium-like tissue outside the uterine cavity and represents one of the most common gynecological disorders among women of reproductive age [5]. The disease is associated with chronic pelvic pain, dysmenorrhea, dyspareunia, infertility, and a significant impairment of quality of life [6]. Despite extensive research, the exact etiology of endometriosis remains incompletely understood, and current concepts describe it as a multifactorial condition involving genetic predisposition, hormonal imbalance, immune dysfunction, and environmental influences [7,8].

Increasing attention has been directed toward environmental exposures that may contribute to chronic inflammatory and estrogen-dependent diseases, including endometriosis [8]. Among emerging environmental stressors, microplastics and nanoplastics have gained recognition due to their global distribution, environmental persistence, and increasing evidence of biological activity [1,4]. Human exposure to these particles is considered continuous and largely unavoidable under current environmental conditions [4].

Experimental and observational studies suggest that MNPs may interfere with cellular homeostasis by inducing oxidative stress, inflammatory responses, endocrine disruption, and immune modulation [9–12]. Notably, these mechanisms closely resemble key molecular pathways implicated in the pathogenesis of endometriosis, including chronic inflammation, estrogen dependence, and altered immune surveillance [7,10]. The detection of microplastics in human reproductive tissues, including the endometrium, provides preliminary evidence that hormonally responsive target tissues may be directly exposed to these particles [13].

Therefore, investigating the potential role of microplastics and nanoplastics as environmental modifiers of endometriosis is of increasing scientific and clinical relevance. Improved understanding of these interactions may contribute to a broader perspective on disease susceptibility, progression, and prevention strategies in women of reproductive age [8,13].

## Definitions and Environmental Characteristics of Microplastics and Nanoplastics

Microplastics are most commonly defined as synthetic polymer particles with a diameter smaller than 5 mm, whereas nanoplastics refer to plastic particles typically below 1 µm, often within the nanometer scale [16]. This size-based classification is widely accepted in environmental and toxicological research and enables differentiation between particles with distinct physicochemical and biological properties [16,17]. Microplastics are generally categorized as either primary or secondary, depending on their origin [17].

Primary microplastics are intentionally manufactured at small sizes for use in industrial applications, cosmetics, personal care products, and medical or laboratory settings [17,18]. In contrast, secondary microplastics arise from the environmental degradation and fragmentation of larger plastic objects, such as packaging materials, textiles, and household products, through mechanical abrasion, ultraviolet radiation, and chemical weathering [18,19]. Nanoplastics are predominantly generated through further fragmentation of microplastics and are considered particularly concerning due to their high surface-area-to-volume ratio and increased chemical reactivity [16,19].

From a physicochemical perspective, microplastics and nanoplastics differ substantially in surface charge, hydrophobicity, polymer composition, and their capacity to adsorb environmental contaminants [20]. Both particle types can act as vectors for heavy metals, polycyclic aromatic hydrocar, pesticides, and endocrine-disrupting chemicals, thereby

increasing the biological availability and toxicity of these compounds [20,21]. Nanoplastics, owing to their small size, exhibit enhanced mobility in biological systems and may more readily interact with cellular membranes and intracellular structures [16,22].

Environmental monitoring studies have demonstrated the ubiquitous presence of microplastics and nanoplastics in air, freshwater, marine ecosystems, soil, and food chains [19,23]. Microplastics have been detected in drinking water, bottled beverages, seafood, fruits, vegetables, and processed foods, indicating multiple routes of dietary exposure [23,24]. Airborne microplastic fibers have been identified in both indoor and outdoor environments, highlighting inhalation as an additional and often underestimated exposure pathway [25].

Given their persistence and resistance to biodegradation, microplastics accumulate in the environment and contribute to chronic low-dose human exposure [19,23]. From an environmental health perspective, microplastics and nanoplastics are considered emerging pollutants primarily associated with long-term exposure rather than acute toxicity [20]. Chronic exposure to low concentrations of these particles may result in subtle but biologically significant effects, particularly in hormonally sensitive tissues [21,22]. Reproductive organs are considered especially vulnerable due to their complex endocrine regulation and reliance on tightly controlled inflammatory and oxidative balance [7,20]. Consequently, increasing concern has emerged regarding the potential role of microplastics and nanoplastics in reproductive disorders, including endometriosis [8,13].

**Human Exposure Pathways and Systemic Distribution**

Human exposure to microplastics and nanoplastics occurs predominantly through ingestion of contaminated food and drinking water and through inhalation of airborne particles [23,25]. Dietary exposure is considered the primary route, as microplastics have been identified in a wide range of commonly consumed food products and beverages, including seafood, salt, sugar, bottled water, and processed foods [24,26].

Inhalation exposure results mainly from airborne synthetic fibers released from textiles, household dust, and industrial emissions [25,27]. Indoor environments may represent a particularly relevant source of inhaled microplastics due to high concentrations of synthetic fibers in household dust and limited ventilation [27]. Dermal exposure has been proposed as a

potential route; however, current evidence suggests that intact human skin provides an effective barrier against penetration of most plastic particles [27,28].

Experimental studies indicate that small plastic particles are capable of crossing epithelial barriers under certain conditions [29]. Both in vitro and animal models demonstrate translocation of microplastics and nanoplastics across the intestinal epithelium and the respiratory alveolar barrier into systemic circulation [29,30]. Once internalized, these particles may distribute to secondary organs via the bloodstream and lymphatic system [30]. Particle size, surface chemistry, and shape appear to be critical determinants of absorption efficiency and biodistribution [22,30].

Biomonitoring studies have provided increasing evidence of internal human exposure to microplastics [1,2]. Microplastics have been detected in human blood, confirming systemic bioavailability [1]. The presence of microplastics in human urine has also been reported, suggesting renal filtration or active excretion following systemic exposure [2,31]. Detection of plastic particles in urine constitutes indirect evidence of biological processing and internal exposure [31].

A comparative study analyzing urine samples from healthy women and women diagnosed with endometriosis identified microplastics in both groups, indicating widespread exposure irrespective of disease status [31]. Importantly, the authors emphasized substantial methodological challenges, including strict contamination control during sample collection and limitations in particle identification and quantification [31]. These limitations underscore the urgent need for standardized analytical protocols in human microplastic research to ensure comparability and reproducibility of findings [27,31].

**Detection of Microplastics and Nanoplastics in Human Reproductive Tissues**

Recent analytical studies have provided evidence for the presence of microplastics in human reproductive tissues, including the placenta and endometrium [31,32]. The detection of plastic particles in the human placenta has raised significant concern, as it demonstrates that microplastics are capable of crossing biological barriers and reaching the maternal–fetal interface [31]. Placental localization supports the hypothesis of systemic translocation of particles following gastrointestinal or respiratory absorption [29,31].

The presence of microplastics in reproductive tissues indicates that hormonally responsive organs of the female reproductive system may be directly exposed to these environmental contaminants [32]. Using spectroscopic and microscopic techniques, microplastics have been identified within human endometrial tissue [32]. The localization of plastic particles within the endometrium suggests potential direct tissue exposure during the menstrual cycle and implantation window [32].

Proposed pathways of entry include systemic circulation following epithelial absorption, as well as possible ascending transport through the reproductive tract [32,33]. Although the biological significance of microplastic accumulation in endometrial tissue remains incompletely understood, their presence raises concerns regarding local cellular interactions, inflammatory responses, and interference with hormonal signaling [20,32].

Animal studies further support the plausibility of reproductive tissue accumulation [30,34]. Experimental exposure to microplastics in rodent models has resulted in particle deposition within the ovaries and uterus [34]. These findings indicate that plastic particles are capable of bypassing physiological barriers and persisting in reproductive organs [30,34]. While extrapolation from animal models to humans must be performed with caution, such data provide important mechanistic insight into potential tissue-level effects [30].

Given that endometriosis involves pathological alterations in both ectopic lesions and eutopic endometrium, tissue-level exposure to environmental contaminants is of particular relevance [5,7]. Local exposure of endometrial cells to microplastics and nanoplastics may influence inflammatory signaling, cellular metabolism, immune interactions, and oxidative balance within the uterine microenvironment [10,20]. Consequently, detection of microplastics in reproductive tissues strengthens the biological plausibility of their involvement in endometriosis-related processes [13,32].

## Pathophysiology of Endometriosis: Cellular and Molecular Mechanisms

Endometriosis is characterized by chronic inflammation, altered immune surveillance, and persistent estrogen-dependent growth of ectopic endometrial tissue [5,6]. Activated macrophages, increased concentrations of pro-inflammatory cytokines, and impaired clearance of ectopic endometrial cells represent central features of the disease [6,35]. Elevated levels of

cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β) contribute to lesion survival, angiogenesis, neurogenesis, and pain generation [35,36].

This pro-inflammatory microenvironment promotes fibrosis and remodeling of the extracellular matrix, facilitating lesion persistence and progression [36]. Hormonal dysregulation plays a crucial role in endometriosis pathogenesis [7]. The disease is estrogen-dependent and characterized by local estrogen overproduction and progesterone resistance within endometriotic lesions [7,37]. Increased aromatase activity and impaired progesterone receptor signaling contribute to sustained cellular proliferation and reduced apoptosis [37].

Hormonal abnormalities further amplify inflammatory signaling and oxidative stress within endometriotic tissues [7,38]. Oxidative stress is a key pathological feature of endometriosis [38]. Increased production of reactive oxygen species (ROS) and impaired antioxidant defenses have been documented in both eutopic and ectopic endometrial tissues [38,39]. Excessive ROS levels promote DNA damage, lipid peroxidation, and mitochondrial dysfunction in endometrial cells [39].

Oxidative stress also enhances angiogenesis and neurogenesis, contributing to lesion persistence and chronic pain symptoms [39,40]. Mitochondrial abnormalities have been increasingly recognized as a hallmark of endometriosis [40]. Endometriotic cells exhibit altered mitochondrial dynamics, impaired ATP production, and increased mitochondrial fragmentation [40,41]. These alterations contribute to metabolic reprogramming and resistance to apoptosis [41].

Mitochondrial dysfunction further exacerbates oxidative stress, creating a self-perpetuating pathogenic cycle that sustains lesion survival [39,41]. Immune dysregulation represents another defining feature of endometriosis [35]. Macrophages in the peritoneal environment of affected women often display an M2-like phenotype, promoting tissue remodeling, angiogenesis, and immune tolerance [35,42]. Impaired natural killer (NK) cell activity reduces the clearance of ectopic endometrial cells [42].

Together, these immune alterations facilitate the survival and growth of endometriotic lesions and contribute to disease chronicity [35,42].

## Oxidative Stress and Mitochondrial Dysfunction Induced by Microplastics and Nanoplastics

Experimental studies consistently demonstrate that exposure to microplastics and nanoplastics induces oxidative stress in mammalian cells and tissues [46,47]. Oxidative stress is defined as an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defense mechanisms, leading to cellular and molecular damage [38,46]. Microplastics have been shown to increase intracellular ROS production through direct particle–cell interactions as well as through the release of adsorbed toxic compounds and plastic additives [47,48].

Nanoplastics, due to their small size, high surface-area-to-volume ratio, and enhanced reactivity, appear to induce oxidative stress more efficiently than larger microplastic particles at comparable concentrations [22,47]. In vitro studies using mammalian and human cell lines demonstrate that microplastic exposure results in lipid peroxidation, protein oxidation, DNA strand breaks, and genomic instability [46,49]. These effects are frequently accompanied by reduced activity of key antioxidant enzymes, including superoxide dismutase, catalase, and glutathione peroxidase [49].

Sustained oxidative stress has been associated with activation of redox-sensitive signaling pathways, including nuclear factor kappa B (NF-κB) and mitogen-activated protein kinases (MAPKs), which promote inflammation, cell survival, and resistance to apoptosis [48,50]. These pathways are also critically involved in the molecular pathogenesis of endometriosis, where chronic inflammation and impaired apoptotic signaling contribute to lesion persistence [35,36].

Mitochondria represent a primary intracellular target of microplastic- and nanoplastic-induced toxicity [41,47]. Experimental exposure to plastic particles has been shown to disrupt mitochondrial membrane potential, impair oxidative phosphorylation, reduce ATP synthesis, and alter mitochondrial dynamics [47,51]. Increased mitochondrial fragmentation and dysregulation of fusion–fission balance have been observed following exposure to nanoplastics [51].

These mitochondrial alterations closely resemble abnormalities described in endometriotic cells, including impaired energy metabolism and increased susceptibility to oxidative damage [40,41]. Mitochondrial dysfunction induced by microplastics contributes to further ROS generation,

thereby amplifying oxidative stress and reinforcing a self-perpetuating pathogenic cycle [38,51]. Such mechanisms may promote metabolic reprogramming and apoptosis resistance, which are essential for the survival of ectopic endometrial cells in hostile inflammatory environments [41]. Therefore, microplastic-induced oxidative and mitochondrial toxicity represents a biologically plausible contributor to endometriosis-related cellular dysfunction [13,47].

## Inflammatory and Immune-Modulatory Effects of Microplastics and Nanoplastics

Microplastics and nanoplastics have been shown to activate innate immune responses in both in vitro and in vivo experimental models [48,52]. Exposure to plastic particles stimulates macrophage activation and promotes the release of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β) [52,53]. These cytokines play a central role in sustaining chronic inflammation, angiogenesis, and tissue remodeling [35,36].

Inflammatory responses induced by microplastics have been reported in multiple organ systems, including the gastrointestinal tract, lungs, liver, and reproductive organs [52,54]. Macrophages appear to be particularly sensitive to microplastic exposure due to their phagocytic activity and prolonged particle retention [53]. Experimental studies indicate that plastic particles can alter macrophage polarization, favoring an M2-like phenotype characterized by immunosuppressive, pro-angiogenic, and pro-fibrotic properties [53,55].

This shift in macrophage phenotype closely mirrors immune alterations observed in the peritoneal environment of women with endometriosis, where M2-dominant macrophage populations promote lesion survival and immune tolerance [35,42]. M2-skewed immune environments enhance extracellular matrix deposition, angiogenesis, and resistance to immune-mediated clearance [36,42].

Microplastics may also impair adaptive immune responses [55]. Exposure has been associated with altered T-cell differentiation, suppressed cytotoxic T lymphocyte activity, and reduced natural killer (NK) cell-mediated cytotoxicity in experimental models [55,56]. Impaired immune surveillance limits the clearance of ectopic endometrial cells and is considered a hallmark of endometriosis pathophysiology [42,45].

Chronic low-grade inflammation induced by continuous exposure to microplastics and nanoplastics may therefore exacerbate pre-existing inflammatory conditions [20,48]. In

hormonally sensitive tissues such as the endometrium, persistent immune activation may disrupt tissue homeostasis, promote fibrosis, and enhance susceptibility to estrogen-driven pathological remodeling [7,20]. Collectively, these findings support the hypothesis that microplastics and nanoplastics may modulate immune pathways relevant to the initiation and progression of endometriosis [13,55].

## Endocrine Disruption and Hormonal Modulation by Microplastics and Nanoplastics

Microplastics and nanoplastics have been increasingly recognized as endocrine-disrupting chemicals (EDCs) capable of interfering with the physiological functioning of the endocrine system [61,62]. These particles may exert endocrine effects either directly, through particle–cell interactions, or indirectly, via leaching of chemical additives such as bisphenols, phthalates, and flame retardants [62,63]. Such compounds are known to interact with nuclear hormone receptors and disrupt hormonal signaling pathways critical for reproductive health [63].

Microplastics and nanoplastics may mimic, antagonize, or alter the activity of endogenous hormones, including estrogen, progesterone, and testosterone [61,64]. Several experimental studies demonstrate that plastic-associated chemicals can bind to estrogen receptors and activate estrogen-responsive gene expression, resulting in altered cellular proliferation and differentiation [63,64]. Estrogenic activity of microplastics is of particular relevance to endometriosis, as estrogen plays a central role in lesion growth, angiogenesis, and immune modulation [37,65].

Phthalates and other plasticizers commonly associated with microplastics have been shown to increase the production of pro-inflammatory cytokines and to promote the proliferation of endometrial-like cells in animal and in vitro models [64,65]. This interaction between endocrine disruption and inflammation is thought to contribute to the chronicity and progression of estrogen-dependent diseases, including endometriosis [65,66].

In addition to estrogenic effects, microplastic exposure has been associated with altered synthesis and metabolism of other reproductive hormones, including progesterone and testosterone [61,67]. Disruption of progesterone signaling may exacerbate progesterone resistance, a key pathological feature of endometriosis [37,66]. Alterations in androgen levels have also been reported following microplastic exposure, suggesting broader effects on the hypothalamic–pituitary–gonadal axis [67].

Epidemiological and experimental evidence indicates that exposure to plastic-associated endocrine disruptors is associated with reproductive disorders in both women and men, including infertility, polycystic ovary syndrome, and impaired spermatogenesis [62,68]. In women, increased estradiol levels and altered estrogen-to-progesterone ratios may enhance susceptibility to estrogen-dependent conditions such as endometriosis [65,68]. Collectively, these findings support the hypothesis that endocrine disruption represents a key mechanistic link between microplastic exposure and endometriosis pathophysiology [13,66].

**Overlap Between Microplastic-Induced Effects and Endometriosis Pathophysiology**

| **Mechanism** | **Microplastics and Nanoplastics** | **Endometriosis** |
|---|---|---|
| Oxidative stress | Increased ROS generation, impaired antioxidant defenses [46,47] | Elevated ROS levels, oxidative damage in eutopic and ectopic endometrium [38,39] |
| Mitochondrial dysfunction | Altered mitochondrial membrane potential and ATP synthesis [47,51] | Impaired mitochondrial dynamics and energy metabolism [40,41] |
| Immune dysregulation | Macrophage activation, M2 polarization, cytokine release [52,55] | Chronic inflammation, immune tolerance, reduced NK activity [35,42] |
| Endocrine disruption | Estrogenic and anti-progesterone effects via additives [63,64] | Estrogen dependence and progesterone resistance [37,65] |
| Apoptosis resistance | Suppressed apoptosis and enhanced cell survival [49,51] | Reduced apoptosis in endometriotic lesions [41,45] |

## Discussion

The available evidence indicates that microplastics and nanoplastics should no longer be regarded solely as inert environmental debris, but rather as biologically active agents capable of interacting with multiple molecular pathways relevant to reproductive health. Experimental data demonstrate that these particles induce oxidative stress, mitochondrial dysfunction, immune modulation, and endocrine disruption—mechanisms that closely mirror the established pathophysiology of endometriosis [13,37,38].

The convergence of microplastic-induced oxidative and inflammatory pathways with estrogen-dependent signaling provides a biologically plausible framework through which chronic environmental exposure could exacerbate lesion establishment, persistence, and symptom severity in endometriosis. In particular, endocrine-disrupting properties of microplastics may amplify local estrogen production and progesterone resistance, thereby reinforcing pathological feedback loops characteristic of the disease [65,66].

Importantly, current human evidence remains largely observational, and causality cannot yet be established. Nevertheless, the detection of microplastics in human reproductive tissues strengthens concerns regarding direct tissue-level exposure and underscores the need for standardized biomonitoring and longitudinal studies. Clarifying exposure–response relationships and identifying susceptible populations will be essential for translating mechanistic insights into public health and clinical practice [69,70].

## Conclusions

Microplastics and nanoplastics are ubiquitous environmental contaminants with the capacity to interact with biological systems at multiple levels.

Available evidence suggests that these particles may contribute to oxidative stress, immune dysregulation, endocrine disruption, mitochondrial dysfunction, and epigenetic modifications—mechanisms central to the pathogenesis of endometriosis.

While definitive causal relationships have not yet been established, the overlap between microplastic toxicity pathways and endometriosis biology supports a potential contributory role.

Further interdisciplinary research is required to clarify exposure–disease relationships and to inform public health interventions.

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The authors declare no conflicts of interest

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