

Microplastics and Female Reproduction: Comprehensive Review of Hormonal and Fertility Alterations

Precious Ojo

Indigo Research; preciousdara.ojo@gmail.com

Mentor: Dr. Barbara Biney

ABSTRACT: Microplastics (MPs) and nanoplastics (NPs) are ubiquitous pollutants derived from plastic degradation. These plastics emerged as silent disruptors of human health. While early studies centered on environmental contamination, recent research reveals alarming links between MP/NP exposure and female reproductive health. This paper synthesizes evidence from studies published between 2019 and 2025, sourced from PubMed and Google Scholar, to evaluate how MPs and NPs influence female fertility, hormonal regulation, and reproductive diseases. Findings indicate that MPs are now being detected in human placental tissue and ovarian follicles, follicular fluid, confirming the ability to cross biological barriers. Exposure has been associated with hormonal dysregulation (altered FSH, LH, and estradiol levels), ovarian fibrosis, reduced oocyte quality, precocious puberty, and uterine fibroids. Moreover, co-exposure to MPs and plastic additives such as DEHP exacerbates endocrine disruption via the TGF- β 1/Smad3 pathway, mirroring PCOS-like changes. These outcomes suggest that MPs are not passive pollutants but active agents of reproductive dysfunction. The study underscores an urgent need for more toxicological research, detection, and monitoring of MPs and NPs.

KEYWORDS: Biomedical and Health Sciences, Cell, Organ and Systems Physiology, Earth and Environmental Science, Microplastics, Reproductive Health, Female Fertility.

■ Introduction

Global plastic production reached 4.9 billion tons, which entered the environment in 2015. By 2025, this amount will have doubled, leading to 11 billion tons of plastic polluting the earth.¹ The increasing presence of plastic and the degradation of its products in the environment has become an important global emergency. Although the use of plastics has greatly increased convenience, it has also led to the environmental release of a significant amount of plastic waste, which has accumulated in ecosystems. Right now, only 9% of the 460 million tons of plastic items produced in 2019 were recycled, and by 2060, this number is predicted to rise to 1.2 billion tons.²

From the process of plastic degradation, particles of plastic on a micrometric scale are created. This is commonly known as microplastics. Microplastics (MPs) are typically defined as synthetic plastic particles ranging from 5 mm to 1 μ m in size, and nanoplastics with a diameter smaller than 1 μ m.³ “They are hydrophobic, synthetic, solid, or polymer matrices with a regular or irregular shape⁴ (fibers, fragments, spheres, beads, films, scales, pellets, and foam) and colors depending on the original form of the large plastics from which they derive.”^{3,4} Their compositions mainly include polyethylene (PE), polyethylene terephthalate (PET), polypropylene (PP), polystyrene (PS), and polyvinyl chloride (PVC).⁵ These MPs are classified into two major types; the first is the primary, which arose from intentional products, smaller in size and mostly rounded in shape. Primary MPs are specifically produced by humans both for their abrasive properties and to improve the stability of certain products; consequently, MPs are intentionally added to cosmetics, toothpaste, household products, detergents, and

paint formulas.³ The other type is the secondary, which originates from the fragmentation of larger particles and is usually irregular in shape. Secondary MPs are also derived from the fragmentation of plastic wastes created from thermal destruction, UV radiation, photodegradation, and biodegradation.³ These MP particles are found everywhere in all natural environments, including water, sewage, air, soil, and the human food chain.¹

Data have shown that humans can ingest up to 5g of MPs per week via multiple routes of exposure.⁶ MPs can enter the human body via three primary routes: oral (consumption), inhalation (breathing), and dermal exposure (skin contact). Of these, oral ingestion is the main route of human exposure to MPs.⁵ Given the ubiquity of MPs in the environment, people inevitably inhale or ingest them daily. MPs have been found in human feces, which is strong proof that they were consumed through the food chain and exposed to the stomach.⁷ Therefore, evaluating the risks of exposure to MPs is an urgent public health priority.⁸ Recent studies have detected microplastics in various human organs and tissues, including the placenta of pregnant women, liver, lung tissues, penis, blood, urine, amniotic fluid, and the human brain.¹ Several studies show that MPs smaller than 10 μ m can enter cells and cause serious health problems, including oxidative stress and damage to cells, inflammation, immune issues, neurotoxicity, reprotoxicity, and carcinogenicity.³

Furthermore, MPs, due to their hydrophobic surface, act as a Trojan horse for other types of notoriously toxic environmental contaminants (such as bisphenols, phthalates, and heavy metals) and can bind to them through processes of bioaccu-

mulation and biomagnification, causing additional harm to living organisms.³ These substances have been associated with various health issues in humans, such as epigenetic changes, reproductive toxicity, and cancer.⁷

These developments raised urgent questions about the long-term impacts of MPs and NPs on female reproductive health, especially in areas such as hormonal regulation, fertility, and transgenerational health. It is within this critical context that this paper synthesizes current research on the impact of microplastics (MPs) and nanoplastics (NPs) on female reproductive health, examining their potential roles in hormonal disruption, abnormal puberty onset, infertility, pregnancy complications, uterine fibroids, and reproductive cancers. This paper provides a comprehensive overview of how MPs and NPs may influence female reproductive biology at multiple stages by merging findings across molecular and hormonal pathways. Moreover, despite this growing body of evidence, the unique risks MPs pose to female reproductive health remain under-researched and overlooked in global health discourse. Addressing this gap requires urgent investment in gender-specific toxicology research and proactive public health strategies.

■ Methods

An electronic literature search was performed across Google Scholar and PubMed for material published between 2019 and 2025. The search terms used were: microplastics, nanoplastics, microplastics and reproductive health, effects of microplastics on reproductive health, microplastics and the ovaries, microplastics in human health, microplastics and female reproductive health, microplastics and female reproductive hormones, plastic pollution and human health, microplastics estradiol, microplastics estriol, microplastics and puberty, microplastics fibroids, microplastics and PCOS, microplastics female fertility, microplastics estriol, microplastics and FSH, LH, polystyrene and female reproductive health.

Each publication was reviewed individually, focusing on the aim of the study and specifically on microplastics and female reproductive health. There were no language restrictions.

■ Results

The Global Decline of Fertility amid Plastic Pollution:

Over the past decades, global fertility rates have declined sharply. Since the 1960s, global total fertility rates (TFR) have begun to fall, and according to the United Nations, two-thirds of the global population currently live in countries with fertility rates below replacement levels. Increasing attention is being drawn to the role of environmental toxins, especially microplastics (MPs), in this global trend, even though social, economic, and medical factors are equally important contributors.⁹ As plastic production continues to rise exponentially, human exposure to MPs and nanoplastics (NPs) has become unavoidable due to them infiltrating air, water, food, and ultimately the human body. Microplastics and nanoplastics have become a great concern for public health experts due to their evident prominence in impacting the reproductive system. Predictably, both male and female reproductive systems

are susceptible to environmental and lifestyle-related damage. However, research has historically focused more on male fertility, largely due to the easier accessibility of sperm for analysis compared to oocytes.⁹

Research on reduced fertilization and failure of offspring to thrive due to micro and nano-plastics was first done primarily on different marine animal models like *Tigriopus japonicus* and *Daphnia magna*. This showed a visible decrease in fecundity, survival, growth, development, feeding capability, and reproduction capacity of offspring. Testing also revealed that the toxicity of MPs-NPs in egg follicles made them remain unfertilized and fail to develop into new offspring. Moreover, it was observed that the MPs induced trans-generational effects on offspring, affecting their phenotypes.⁴

Controlled laboratory studies on mice further demonstrate the reproductive toxicity of microplastics at the mammalian level. In one study, male and female mice were exposed to polystyrene microplastics (PS-MPs) via oral gavage over 44 days. Post-mating analysis revealed that PS-MPs accumulated significantly in both the testes and ovaries, with a higher degree in ovarian tissue ($p < 0.05$), confirming their ability to cross biological barriers. The fertility outcomes in this study showed that in females, this accumulation was associated with reduced ovary-to-body weight ratios and a lower number of embryos.⁵ Both sexes exhibited reduced pregnancy rates and fewer embryos post-mating, indicating compromised fertility. When neither was exposed to MP, the pregnancy rate was 89%; when only the female was exposed, the pregnancy rate was 38%; when only the male was exposed, the rate was 78%; and when both were exposed, the pregnancy rate was 75%. Meaning that exposure to PS-MPs reduced the pregnancy rate by up to ~57% when only the female was exposed. This was the most significant reduction across all groups; a summary of these findings is provided in Appendix 1, Table 1.⁵

Several studies focus specifically on correlations between MP exposure and female fertility disruption. One systematic review (Sharma *et al.*) analyzed 12 studies (totaling 234 subjects) and confirmed the presence of MPs ranging from 2.1 to 100 μm in human placental and fetal tissues. The findings showed correlations of MP levels with indicators of reduced fetal growth, including low birth weight and altered gestational age. This suggested broader reproductive implications, though direct fertility data remain limited.² Another breakthrough study showed that co-exposure to MPs and DEHP triggers PCOS-like ovarian damage via activating the TGF- β 1/Smad3 pathway. This pathway is normally inhibited, preventing fibrosis, oxidative stress, and hormone imbalance. The study also found serious molecular damage via proteomics, including barrier dysfunction and metabolic disruption. This makes it a key contribution to understanding how plastic pollution may harm female fertility.⁸

These are clear indicators of reduced fertility. These findings suggest that MPs may affect ovarian function and have a negative impact on ovarian capacity, oocyte maturation, and oocyte quality through endocrine disruption, oxidative stress, inflammation, and ultimately affect reproductive success.⁴

The Hormonal Cycle and Microplastics Disruption:

Figure 1 illustrates the normal female reproductive cycle regulated by the hypothalamic–pituitary–ovarian (HPO) axis, highlighting the coordinated roles of follicle-stimulating hormone (FSH), luteinizing hormone (LH), estrogen, and progesterone across the follicular, ovulatory, and luteal phases. Within this axis, estradiol (E2) drives follicular growth and ovulation, estrone (E1) contributes to baseline estrogen balance, estriol (E3) supports pregnancy and placental function, while progesterone stabilizes the luteal phase and prepares the endometrium for implantation. Disruption of estradiol, estriol, or progesterone synthesis impairs gonadotropin signaling, folliculogenesis, luteal function, endometrial receptivity, and pregnancy maintenance, linking hormonal imbalance to infertility, PCOS, and adverse reproductive outcomes.

One of the first ways MP and NP impact female reproductive health is through the disruption of these major hormones: FSH, LH, estrogen, and progesterone. Each of these has a specific role in regulating the menstrual cycle and reproductive success in females. Research has shown that MP exposure led to increased FSH and LH levels in mouse models when exposed to polystyrene microplastics.⁵ This suggests MPs can cause reduced ovarian reserve, decreased fertility, and a lower chance of a successful pregnancy, potentially leading to conditions like Polycystic Ovary Syndrome (PCOS) and ovarian failure.

Furthermore, a study, detailed in Appendix 1, Table 1, on human adrenal carcinoma cells (H295R) found that exposure to 50 nM of PS-MNPs at 100 µg/mL for 48 hours reduced estriol (E3) levels by ~37.5%, with no significant changes to estrogen and androgen receptor activity. Since adequate estriol is necessary for placental and uterine function, this drop suggests that PS-MNPs may disrupt steroidogenesis and trigger oxidative stress, with potential consequences for reproductive and fetal health during pregnancy, particularly when compared to normal physiological estrogen dynamics illustrated in Figure 1.¹⁰ Another study on female Sprague-Dawley rats utilized doses of 0.5 mg/day of PS-MPs, found that co-exposure to polystyrene microplastics (MPs) and DEHP (a plastic additive) lowered estradiol (E2) while raising testosterone levels, a hormonal profile consistent with PCOS-like ovarian damage via the TGF-β1/Smad3 pathway.⁸ Relative to the normal cycle shown in Figure 1, where rising estradiol drives follicular maturation and ovulation, those reduced levels are insufficient to support ovulation, thereby promoting follicular atresia and infertility.

In addition, there is also growing evidence that microplastics may accelerate pubertal onset in females.¹¹ Precocious puberty (PP), the unusually early onset of sexual development, is a prevalent pediatric endocrine disorder affecting children's growth and sexual development with documented links to environmental pollutants, including microplastics. Observational research found that early MP exposure activated pubertal signaling in the brain: microglial and astrocytic activation increased GnRH expression in the hypothalamus, pushing puberty forward.¹² As illustrated in Figure 2, GnRH is the primary neuroendocrine trigger for HPA axis activation, driv-

ing downstream estrogen and progesterone secretion, which connects MP-induced neural changes directly to accelerated development.

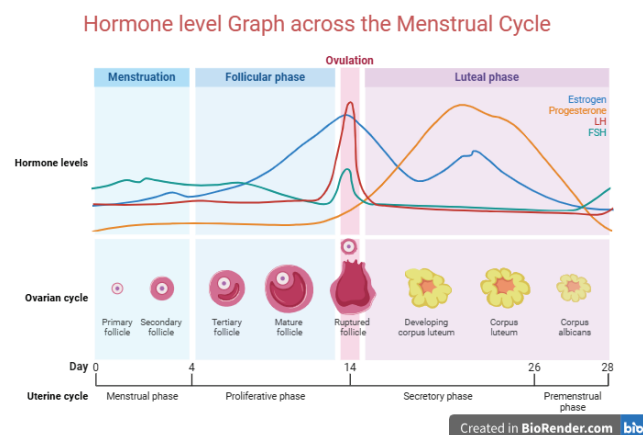


Figure 1: Hormone Level Graph Across the Menstrual Cycle. Created in BioRender.com. The LH surge at day 14 triggers ovulation, marking the shift from the follicular to the luteal phase. Rising progesterone after ovulation reflects corpus luteum formation and prepares the uterine lining for implantation.

In a human case-control study of 14 girls, those with central precocious puberty (CPP) had significantly higher MP concentrations in their blood serum compared to healthy controls (68.19 ± 26.49 mg/kg vs. 36.94 ± 13.22 mg/kg). The dominant polymers were polyethylene (PE), CPP: 28.22%; Control: 31.62%, and polyvinyl chloride (PVC), CPP: 62.67%; Control: 58.14%. Hormonal analysis revealed a negative correlation between MPs and progesterone levels, alongside a positive correlation with dehydroepiandrosterone sulfate (DHEAS), indicating that MPs may disrupt steroid balance during critical developmental windows.¹² Under normal physiology, progesterone rises in the luteal phase to prepare the endometrium for implantation (Figure 1); the observed reduction with MP exposure suggests impaired reproductive function and increased infertility risk. Complementary animal experiments confirmed these findings. Young female mice exposed to PE (3 mg/day) and PVC (6 mg/day) showed advanced vaginal opening, a biological marker of early puberty. Brain tissue analysis confirmed elevated hypothalamic GnRH expression and glial cell activation, as seen in Figure 2. These findings provide some of the first experimental evidence that PE and PVC specifically can drive premature puberty by disrupting ovarian hormones and overstimulating the HPG axis. Early puberty carries long-term risks for pediatric endocrine health, including infertility, metabolic disease, and reproductive cancers.

As women move into adulthood, microplastics can further impact the reproductive system by inducing and intensifying pathological dysregulated hormonal states like Polycystic Ovarian Syndrome (PCOS), one of the most common endocrine disorders in reproductive-age women, defined by cystic follicles, hyperandrogenism, and hormonal dysregulation. One study examined the effects of co-exposure to polystyrene microplastics (MPs) and di-2-ethylhexyl phthalate (DEHP), a widely used plastic additive, finding PCOS-like phenotypes

in animal models. Key findings include the visible disruption of the ovarian morphology, particularly the formation of cystic follicles and elevated follicular atresia. Exposed female Sprague-Dawley rats in the MP + low-DEHP and MP + high-DEHP groups developed approximately 64 and 58 atretic follicles, respectively, compared to only ~25–30 in saline and MP-only controls. Ovarian fibrosis was also observed, with excess connective tissue damaging normal ovarian structure. Hormonally, the exposed groups showed roughly a 20% drop in serum estradiol and a concurrent 21–29% rise in testosterone, producing a significant downregulation of the E2/T ratio by up to 29%. This hormonal profile aligns with hyperandrogenism, a defining trait of PCOS.⁸

Furthermore, the study found that microplastics and DEHP together activated the TGF- β 1/Smad3 pathway. This pathway is an intracellular serine/threonine kinase signaling cascade that drives fibrosis and inflammation. It is activated by the binding of TGF- β 1 to cell surface receptors and is downregulated by inhibitory proteins such as Smad7 or targeted chemical compounds. When dysregulated, this pathway promotes excessive extracellular matrix deposition and ovarian tissue remodeling, contributing to follicular loss and impaired ovarian function.¹³ This signaling pathway increased fibrosis (scarring), quantified by an elevated collagen volume fraction of over 40% in the MP + H-DEHP group, and led to higher oxidative stress in the ovary. When researchers blocked Smad3 with the compound called SIS3, the detrimental effects were significantly reversed. Hormone levels improved, the E2/T ratio by ~42% (from ~5.0 to ~7.1) following SIS3 treatment, returning it to the Saline control level of 7.0, and tissue damage was reduced, the collagen volume fraction by ~25% (from 40% to ~30%).⁸ This shows that TGF- β 1/Smad3 plays a central role in how pollutants harm reproductive health.

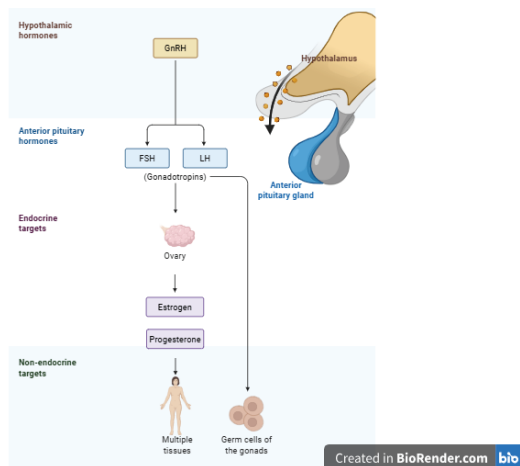


Figure 2: The Hypothalamic-Pituitary-Gonadal (HPG) Axis and its role in hormone release. Created in BioRender.com. GnRH from the hypothalamus stimulates the pituitary to release FSH and LH, which in turn drive ovarian production of estrogen and progesterone. These hormones act on multiple tissues, coordinating the body's reproductive response.

Pathological and Developmental Risks of Microplastic Exposure: Uterine Fibroids and Cervical Cancer:

Emerging evidence suggests that microplastics have a dual pathological effect on female reproductive tissues. They can

stimulate abnormal tissue proliferation (hyperplasia, tumor formation) while also damaging healthy reproductive structures through inflammation, oxidative stress, and endocrine disruption. A 2025 case-control study of 40 fibroid patients and 40 healthy controls provided direct evidence of MPs in uterine fibroid tissue.¹⁴ Uterine fibroids (UFs) are benign smooth muscle tumors of the uterus that affect an estimated 70–80% of women of reproductive age globally, often leading to infertility, heavy bleeding, and chronic pain.¹⁵

The results of the study showed that MP concentrations were highest in fibroid tissue, followed by patient myometrium, and lowest in healthy controls. Polyethylene (PE) dominated, and its exposure was linked to a threefold increased fibroid risk (OR = 3.327, $p = 0.012$).¹⁴ This suggests not all MP types carry equal risk, with PE posing a particular concern. While genetics and hormones are established contributors to fibroid development, these findings add environmental pollutants to the list of plausible risk factors.

Regarding cervical cancer, a preliminary cross-sectional study found that MPs may promote pathological growth, inflammation, and potentially carcinogenic processes in reproductive tissues. A study done in China analyzed blood, cancerous cervical tissue, and adjacent paracancerous tissue from 26 cervical cancer patients using Raman spectroscopy. Thirteen microplastic (MP) types were identified, with polyethylene (PE), polypropylene (PP), and PE-co-PP as the dominant polymers. MP abundance followed the pattern: blood > cancerous tissue > paracancerous tissue, with significantly higher diversity in tumors than in adjacent tissue ($p < 0.05$). Although these findings are significant, further research comparing cancerous vs non-cancerous populations is needed to further investigate the association between microplastics and cancerous tissue.

The pathogenesis of cervical cancer and fibroids from microplastics may be due to the upregulation of amino sugar and nucleotide sugar metabolism pathways. These pathways may support abnormal cell and fibroid growth. In addition, microplastics may cause a downregulation of cofactor biosynthesis and platelet activation pathways, leading to decreased cell repair or detox, allowing fibroids to thrive and poor healing or inflammation control, worsening fibroid symptoms.¹⁴

Extracorporeal Therapeutic Apheresis in Microplastic Removal:

Until very recently, there were no established medical interventions capable of actively removing microplastics (MPs) or nanoplastics (NPs) from the human body. Research on the biological impact of these particles has grown rapidly, but therapeutic strategies have lagged, largely because of the microscopic size of MPs/NPs and their ability to accumulate in deep tissues. Other than detection, the extraction has been a major impediment.

In 2025, Bornstein and colleagues conducted the first human study exploring the possibility of using extracorporeal therapeutic apheresis (TA) as a method for eliminating microplastic- and nanoplastic-like particles circulating in the blood plasma. As shown in Figure 3, this process involves blood extraction via venous access. The plasma is separated from blood

cells using a porous membrane, then filtered through adsorption columns or membranes designed to capture pathogenic or toxic components before the blood components are reinfused into the patient. The study recruited 21 patients diagnosed with post-infectious myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), all of whom underwent at least two sessions of double-filtration apheresis (INUSpheres).¹⁶

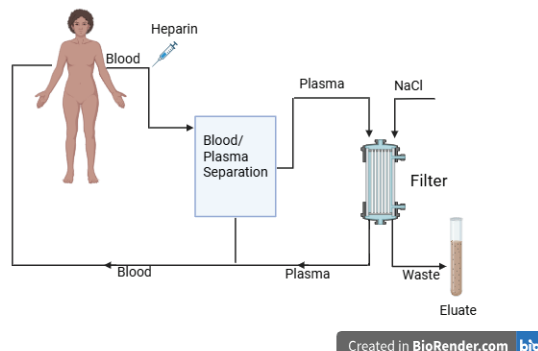


Figure 3: Schematic representation of the extracorporeal therapeutic apheresis process used for microplastic and nanoplastic removal. Blood is withdrawn from a large vein and circulated through an apheresis system, where a semipermeable membrane separates plasma from cellular components. The isolated plasma is then directed through specialized filtration columns that selectively remove targeted molecules like microplastics. Once filtration is complete, the remaining plasma and blood cells are returned to the patient via a second venous line. The filtered waste, or eluate, may contain retained microplastic and nanoplastic particles. Created in BioRender.com.

Analysis of the concentrated eluate, which is the waste fraction collected after filtration, revealed that 14 polymer-like substances, including polyamide 6 and polyurethane, were identified using attenuated total reflectance Fourier-transform infrared (ATR-FT-IR) spectroscopy. Figure 3 contextualizes this workflow, showing how the eluate fraction is isolated and analyzed for polymer content. The polymer signatures matched up to 67.5% for polyamide 6 and 35.3% for polyurethane. This suggests that apheresis may be capable of physically trapping and removing certain microplastic residues from human circulation. However, the study did not quantify total polymer concentrations removed, nor did it measure blood levels before and after treatment.

The study authors cautioned that these findings are preliminary and novel. Larger patient cohorts and more extensive clinical trials are still needed. Future work should use quantitative analytical tools like pyrolysis–gas chromatography–mass spectrometry (Py-GC/MS) to measure microplastic levels in blood plasma both before and after therapy, and within the eluate fractions collected during apheresis. Tracking these levels across multiple treatment cycles would help determine how consistently and effectively apheresis removes MNPs from circulation.

Despite these limitations, the findings are considered groundbreaking because no prior human intervention had ever demonstrated active removal of microplastic-like materials from the bloodstream. The absence of such approaches historically stems from some major challenges, which include the invisibility and chemical resilience of microplastics in biological systems, the lack of analytical tools sensitive enough to

detect microplastics at nanoscales, and uncertainty over their clinical relevance and effect until recent evidence confirmed microplastic presence in blood and reproductive tissues.

Polyamide and polyurethane, which are the same polymer types detected in the apheresis eluates, have also been identified in female reproductive tissues, including the placenta and the uterus.¹⁷ Although the 2025 apheresis study did not quantify how much of these polymers were removed from circulation, the overlap in polymer classes is scientifically meaningful. It suggests that apheresis is capable of capturing some of the microplastics accumulating in reproductive organs. This raises the possibility that, with further refinement, extracorporeal therapeutic apheresis could one day be adapted to target the specific polymer types most frequently detected in female reproductive systems.

However, achieving this goal requires future studies that measure blood microplastic levels before and after apheresis, track polymer concentrations across multiple treatment cycles, answer the question of whether removal improves with repeated sessions, and directly compare the polymer profiles found in circulation with those accumulating in reproductive tissues. These future steps are essential because they determine whether removing specific polymer types from the bloodstream can reduce the very same microplastics that accumulate in female reproductive tissues, thereby directly addressing the sex-specific risks highlighted in this study.

■ Discussion

The collective findings reviewed in this paper demonstrate that microplastics and nanoplastics are not inert environmental contaminants but biologically active agents capable of disrupting female reproductive health through endocrine imbalance, oxidative stress, inflammatory signaling, and scarring of reproductive tissues. Evidence from animal models, *in vitro* systems, and emerging human studies consistently links microplastic exposure to altered hormonal regulation. In addition, exposure to microplastics is correlated with impaired ovarian function, abnormal pubertal timing, and increased risk of uterine fibroids and cervical cancer. Despite growing evidence of these effects, significant challenges remain in accurately characterizing human exposure. Currently, there are no standardized clinical blood tests for microplastics; however, the techniques mentioned earlier, such as Raman spectroscopy and Fourier-transform infrared (FTIR) analysis, demonstrate the initial methodology of detection.⁷ Simultaneously, the recent discovery and use of therapeutic apheresis represents a promising strategy for addressing internal microplastic accumulation.

These methodologies are helpful steps in the right direction in addressing this problem. The female reproductive system is highly sensitive to microplastic exposure, and even minor fluctuations in FSH, LH, estradiol, or progesterone can cascade into significant physiological disruption. This sensitivity, along with increasing plastic contamination in the environment, makes for a compounding effect and a public health concern. Ultimately, prevention remains the most practical and sustainable approach to this problem. On an individual level, using

high-quality water filters, avoiding single-use plastics, and choosing fresh foods over heavily packaged alternatives are a few of the many solutions to the problem. Additional individual practices to reduce microplastics exposure include limiting shellfish consumption due to high levels of bioaccumulation in the shellfish.¹⁸

Community-level interventions are also critical, and implementing recycling programs in communities that do not currently have them is an initial step. In areas where recycling systems are already in place, more advanced recycling methods such as chemical pyrolysis can also be utilized. Chemical pyrolysis uses heat in the absence of oxygen to break plastics down into their basic molecular components.¹⁹ This approach is often more effective than traditional mechanical recycling, which typically involves shredding and melting plastic into pellets and can reduce material quality over time. Lastly, using policy to decrease the manufacturing of plastics is another solution. For example, the EU Single-Use Plastics Directive in 2021 banned the manufacture and sale of single-use plastic items across all 27 EU member states and thus directly eliminated the entire category of plastics from sale.²⁰ Ultimately, combating the pollutants of microplastics necessitates a shift from personal lifestyle adjustments to strict source-control legislation. Future global health frameworks must prioritize toxicological standards to protect the unique sensitivities of the female reproductive system and ensure that the documented decline in global fertility is not exacerbated by unregulated plastic pollution.

The collective findings reviewed in this paper demonstrate that microplastics and nanoplastics are not inert environmental contaminants but biologically active agents capable of disrupting female reproductive health through endocrine imbalance, oxidative stress, inflammatory signaling, and scarring of reproductive tissues. Evidence from animal models, *in vitro* systems, and emerging human studies consistently links microplastic exposure to altered hormonal regulation, impaired ovarian function, abnormal pubertal timing, and increased risk of gynecological pathologies, including uterine fibroids and cervical cancer.

Limitations & Future Work:

The strength of current conclusions is constrained by a number of limitations, despite the mounting evidence that microplastics are linked to the reproductive health of females. First, there is still a lack of human data, as most research uses invasive, non-scalable sampling or small sample sizes. It is not possible to accurately measure microplastics or differentiate particle toxicity from chemical additives using detection techniques like Raman and FTIR spectroscopy. Standardized worldwide measurement protocols and longitudinal cohorts will be necessary to close these gaps. Furthermore, comprehending the practical effects of long-term, low-level environmental exposure over a person's lifetime requires going beyond the high-dose laboratory models used to determine biological thresholds.

Table 1: Summary of Experimental Studies on Microplastic Exposure and Female Reproductive Outcomes.

Study Type	Polymer Type	Experimental Dose	Target Tissue	Key Reproductive Outcome	Proposed Mechanism
Animal (Mouse) ⁵	Polystyrene (PS)	Oral gavage (44 days)	Ovary	Reduced the ovary-to-body weight ratio; decreased pregnancy rate by up to 57%	Oxidative stress and endocrine disruption
Human (Placental) ²	Mixed Microplastics (2.1–100 µm)	Environmental exposure	Placenta and fetal tissue	Altered gestational age and reduced fetal growth	Barrier penetration and inflammation
Animal (Mice) ⁵	Polystyrene (PS)	Oral gavage (44 days)	ovary	Decreased Pregnancy rate (up to 57%)	Oxidative stress; Endocrine disruption
<i>In vitro</i> (H295R cells) ¹⁰	Polystyrene (50 nm)	100 µg/mL (48 hours)	Adrenal steroidogenic cells	Reduced estril levels by approximately 37.5%	Disrupted steroidogenesis and oxidative stress
Animal (female Sprague-Dawley rats) ⁸	Polystyrene + DEHP	Low and high DEHP co-exposure	Ovary	PCOS-like phenotype; reduced estradiol; increased testosterone	TGF-β1/Smad3 activation and fibrosis
Human Uterus: (40 fibroid patients; 40 healthy controls) ¹⁴	Polyethylene (PE)	Environmental exposure	Uterine fibroid tissue	3x higher risk of fibroids	Metabolic dysregulation ²¹ nd tissue prolif ² at ²³
Human (Cervical) ²¹	PE, PP, and PE-co-PP	Environmental exposure	Cervical tissue	Higher microplastic diversity in tumor tissue	Inflammation and carcinogenic signaling
Human (Puberty Study) ¹¹	PE and PVC	Environmental exposure	Blood serum	Reduced progesterone; increased DHEAS	HPG axis disruption
Animal (Juvenile Mouse) ¹¹	PE and PVC	3 to 6 mg per day	Hypothalamus and ovary	Accelerated pubertal onset	GnRH overstimulation

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During the preparation of this work, the author used Google Gemini in order to brainstorm research topics and key terms, etc. After using this tool, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

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■ Author

Precious Ojo is a gap-year student from Nigeria. She is deeply passionate about women's health and biomedical science. She is fascinated by understanding reproductive health, uncovering the mechanisms behind different diseases, and exploring how to improve women's well-being. She plans to major in biology and minor in global health to pursue these interests.