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Microplastics and Human Health: Focus on the Reproductive System

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Abstract

Introduction:

Plastic pollution, especially in the form of microplastics (MPs), is a growing global health concern. Due to their small size and chemical properties, MPs can enter the human body via ingestion, inhalation, or dermal absorption. Beyond accumulating in organs such as the lungs, liver, and kidneys, MPs have also been found in reproductive tissues.

Aim:

The aim of this review is to evaluate current evidence on the effects of MPs on male and female reproductive systems.

Materials and Methods:

A literature review of 57 studies published between 2004 and 2025 was conducted. Of these, 41 were from 2020–2025 and 39 were original research. The selection included *in vivo* and *in vitro* experiments, as well as human observational studies addressing exposure, tissue accumulation, toxicity, and reproductive outcomes.

Results:

Microplastics have been detected in both male and female reproductive organs. In females, MPs were primarily found in the placenta, follicular fluid, and endometrial tissue, and were associated with hormonal imbalances, impaired folliculogenesis, reduced oocyte quality, and endometrial dysfunction. In males, MPs mainly affect sperm quality, leading to sperm DNA damage. Decreased testosterone levels and testicular abnormalities were also observed. *In vivo* studies have shown that the mechanism of MP-induced reproductive toxicity involves oxidative stress, inflammation, and hormonal disruption.

Conclusions:

Microplastics may pose a serious threat to reproductive health, as confirmed by numerous *in vivo* studies. Alarmingly, the amount of evidence in humans is also increasing. Therefore, further research is needed to assess the long-term effects and to develop effective preventive strategies.

Key words: Microplastics; Reproductive System; Endocrine Disruptors; Environmental Exposure; Oxidative Stress; Infertility

1. Introduction

Microplastics (MPs) are synthetic, water-insoluble particles ranging from 1 µm to 5 mm in size. The definition of nanoplastics remains under scientific debate. While some researchers define nanoplastics as particles between 1 µm (1000 nm) and 1 nm, others argue that a more accurate definition includes particles ranging from 100 nm to 1 nm [1]. Harmful plastic particles can be categorized into: primary microplastics - manufactured directly (e.g., from clothing fibers, industrial nanoparticles) and secondary microplastics - formed through the breakdown of larger plastic items [1,2].

Plastic is used globally in nearly all aspects of modern life and has gained immense popularity due to its versatility. Small modifications in its chemical structure can yield materials with very different properties. In 2023, global plastic production reached 400.3 million metric tons. Currently, plastic pollution is ubiquitous in our environment [1,3,4].

Interest in MPs' pollution has grown significantly over the past two decades. The term

"microplastic" was first introduced in 2004 by Thompson et al. in the paper titled *"Lost at Sea: Where Is All the Plastic?"* [5]. At the time, the idea that oceans contained microscopic plastic particles was entirely novel. Since then, the number of scientific publications on the topic has increased rapidly. For example, in a PubMed search, only one publication on MPs was recorded in both 2005 and 2006. By 2015, this number had increased to 96, and by 2024, it had surged to 4,823, demonstrating the exponential growth of scientific interest in this field.

Along with an increasing spread of plastic use, the pollution of MPs – the breakdown product of plastic itself – increases [6,7]. Due to its small size - less than 5 mm in diameter [6,8] – MPs particles can be easily spread through water and terrestrial environments [9,10]. MPs can enter and accumulate within various organisms through environmental exposure. Due to the process of biomagnification along the food chain, MPs ultimately concentrate on top-level predators, including humans, where they may disrupt physiological functions and pose significant health risks [6,8,11].

Initially, MPs raised concern primarily due to their environmental impact. However, only in the past decade have researchers begun to investigate their effects on human health. The findings are alarming. Exposure to MPs is associated with increased risk of diseases affecting the digestive, respiratory, cardiovascular, nervous, reproductive, immune, and endocrine systems [3,12,13,14,15]. MPs have even been found in human breast milk. More concerning are reports of MPs in meconium, indicating exposure begins in utero [16]. This paper focuses specifically on the effects of MPs on the reproductive system, given its crucial role in human survival and species continuation.

MPs vary in shape depending on their source and environmental exposure (sunlight, heat, water). Recent research shows that the two main shapes found in human excreta are microfibers (often from clothing) and films (also called sheets). MPs are typically transparent or colorless, although colored particles have been detected in human tissues [16].

MPs may contain or absorb harmful substances, such as phthalates and bisphenol A (BPA), which are known endocrine disruptors even at low concentrations [12,17]. In addition to their

direct toxicological effects, MPs can act as vectors for other harmful agents. Due to their large surface area and hydrophobic nature, they can transport heavy metals (e.g., arsenic) and persistent organic pollutants (POPs). Notably, MPs may also carry dangerous pathogens, facilitating the spread of diseases like those caused by SARS-CoV-2 and Helicobacter pylori [3,12,16].

Micro- and nanoplastics can enter the human body through three primary routes: ingestion, inhalation, and dermal contact [1,3,18,19]. The most significant source of MPs' exposure is food consumption. Seafood, particularly species such as *Mytilus edulis*, *Corbicula fluminae*, and tuna, raises the greatest concern due to high levels of MPs contamination. MPs have also been detected in beverages such as drinking water, white wine, milk, energy drinks, and soft drinks, as well as in food packaging materials (e.g., plastic cups and tea bags). Tap water has also been shown to contain MPs, with concentrations influenced by factors such as pipe material [3,12,19].

In the atmospheric air of large urban areas, the presence of MPs can reach thousands of particles per cubic meter. Major sources include industrial emissions, building materials, waste incineration, clothing fibers, and traffic-related particles. Indoor environments (e.g., offices, schools, gyms, and public transport) also accumulate MPs through infiltration (air exchange via windows and doors), transfer (through clothing and footwear), and penetration (through structural defects in buildings). Indoor plastic items (e.g., toys, furniture, textiles) further contribute to contamination. Studies have shown that indoor MPs levels often exceed those found outdoors, which is concerning given that people spend up to 70% of their time indoors [12,19,20].

Through inhalation, MPs can be absorbed into the respiratory system and may deposit deep within the lungs. Despite the presence of defense mechanisms such as mucociliary clearance, some particles persist and may trigger inflammation.

Dermal contact is the least studied exposure route. It can occur through wearing synthetic clothing or using cosmetics containing MPs. It is hypothesized that nanoplastics, due to their small size, may penetrate the skin barrier. Further research is needed to better understand this route of exposure [3,12,19].

2. The cytotoxic and genotoxic effects of microplastics

The cytotoxic and genotoxic effects of MPs have been increasingly studied, particularly in relation to particle characteristics such as shape, concentration, and duration of exposure. While genotoxicity has yet to be conclusively demonstrated *in vivo*, numerous *in vitro* studies have shown that exposure of fibroblasts and extracellular DNA to MPs can lead to alterations suggestive of potential genetic damage [21,22]. In an occupational context, a study involving 52 workers exposed to styrene (component of polystyrene (PS)) revealed that airborne concentrations and urinary metabolite levels exceeded acceptable thresholds. These individuals exhibited a significantly higher frequency of sister chromatid exchanges (SCEs) and DNA damage. Moreover, elevated microsomal epoxide hydrolase activity was observed, correlating with increased SCE frequency and suggesting that genetic polymorphisms may influence individual susceptibility to styrene-induced genotoxicity [23].

Further evidence indicates that MPs can cross the blood-brain barrier and accumulate in brain tissue, triggering various immune responses, including microglial activation and apoptosis [24]. Exposure to MPs has been associated with elevated levels of proinflammatory cytokines such as interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor alpha (TNF-α), contributing to a broader inflammatory response [25]. These immunological effects are often linked to oxidative stress, which activates signaling pathways such as NF-κB and the NLRP3 inflammasome, ultimately leading to increased expression of inflammatory mediators [26].

These mechanisms are also reflected in the reproductive system. Polyethylene (PE), the most commonly detected MPs in human follicular fluid (present in 86.4% of samples), has been shown to exert cytotoxic and potentially genotoxic effects on oocytes. In a murine model, PE exposure led to significantly increased levels of reactive oxygen species (ROS), indicative of oxidative stress, and a higher proportion of poor-quality oocytes. Concurrent RNA sequencing revealed the upregulation of inflammation-related genes (e.g., *Il10ra, Il1a, Il33, Tnfaip8l2*, and *Tnfrsf1b*), suggesting activation of inflammatory pathways in response to MPs presence [27].

A similar mechanism has been demonstrated in human dermal fibroblasts (Hs27) exposed to polystyrene nanoparticles (PNPs), where a rapid and dose-dependent increase in ROS levels was observed within 15–30 minutes of exposure. This oxidative burst was accompanied by clear signs of DNA damage, including increased frequencies of micronuclei, nuclear buds, and nucleoplasmic bridges [28]. These findings reinforce the role of oxidative stress as a central mechanism driving the genotoxic effects of both micro- and nanoplastics across various human cell types and biological systems.

In vivo studies have further confirmed these effects. For instance, research on mice exposed to polystyrene microplastics (PS-MPs) demonstrated significant reproductive toxicity in both sexes. In males, exposure reduced sperm viability, damaged spermatogenic cells, and increased sperm deformities. In females, PS-MPs exposure led to a decreased ovarian follicle count and reduced ovarian volume. These changes were associated with oxidative stress and hormonal imbalance, with females showing greater vulnerability to microplastic-induced toxicity [29].

Importantly, MPs including PE, PS, polypropylene (PP), and polyvinyl chloride (PVC) have been directly detected in human follicular fluid, confirming their presence within the ovarian microenvironment. Their accumulation was associated with impaired oocyte maturation and elevated ROS levels, indicating oxidative stress [30]. These findings underscore the cytotoxic and potentially genotoxic effects of MPs on female germ cells, highlighting the serious reproductive risks associated with environmental MPs exposure in humans.

3. The Impact of Microplastics on the Female Reproductive System

MPs can cross cellular membranes and have been detected in the placenta, follicular fluid, and endometrium, as confirmed by recent studies [31].

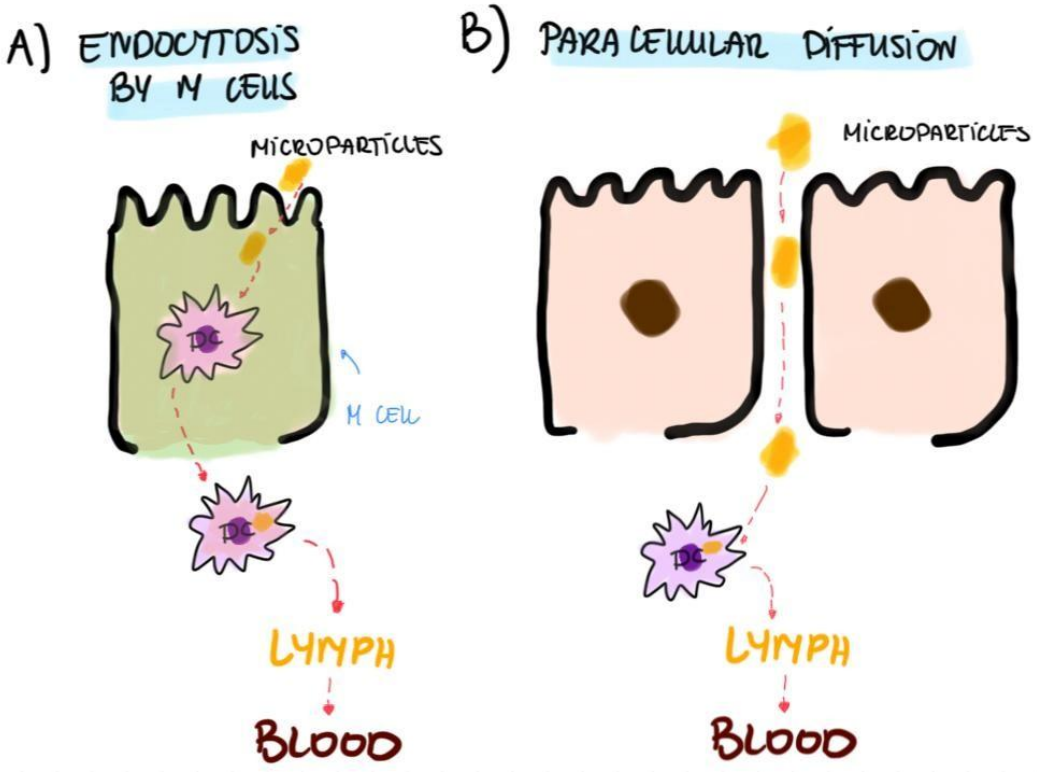
3.1 Placenta

A study conducted by Ragusa et al. (2024) evaluated placentas from healthy women who underwent spontaneous vaginal deliveries. The detection of MPs was confirmed in four placental samples, with a total of 12 fragments identified. These particles, ranging from 5 to 10 μm in size and exhibiting either spherical or irregular morphology, were distributed across

different placental regions: five on the fetal side, four on the maternal side, and three within the chorioamniotic membranes. Notably, only small sections of each placenta were analyzed, suggesting that the total MPs burden in the entire placenta may be significantly higher. Although the exact mechanism of MPs entry into the bloodstream and placenta remains unclear, it is hypothesized to involve either the respiratory or gastrointestinal tract. Proposed mechanisms of translocation from the gastrointestinal tract to the bloodstream include:

A) Endocytosis via M cells of the Peyer's patches epithelium, whereby M cells transfer MPs to dendritic cells.

B) Paracellular diffusion through intercellular spaces, which may widen during inflammatory states. MPs are subsequently captured by dendritic cells [32].



These particles are recognized by the immune system and, via dendritic cells, can reach the bloodstream. This mechanism may explain how MPs influence cellular signaling pathways in the placenta. MPs have the potential to disrupt immune tolerance during pregnancy, interfere with maternal-fetal communication, and negatively affect fetal growth factor signaling [32].

Recent research has also investigated the effects of MPs on the female reproductive system, yielding equally concerning results. Studies have shown that exposure to polystyrene MPs affects both female mice and their offspring [33,34]. Zhao et al. (2023) suggest that prenatal and postnatal exposure to PS-MPs leads to impaired testicular development and male infertility [35]. Furthermore, maternal exposure to MPs during pregnancy and lactation resulted in reduced birth weight, decreased postnatal body weight, and lowered fertility in female offspring [33]. These findings suggest that exposure to polystyrene MPs can adversely affect the reproductive health of female mice and the development of their progeny.

3.2 Follicular Fluid

Montano et al. (2025) selected 18 women undergoing assisted reproductive treatments from an initial pool of 80. Participants presented with various infertility-related conditions, including PCOS, diminished ovarian reserve, and advanced maternal age. The study aimed to detect MPs in follicular fluid and explore potential correlations between MPs accumulation and fertility biomarkers such as FSH, E2, and AMH. MPs were identified in 14 out of 18 samples. The study also highlighted similarities between the blood-follicle barrier and the placental barrier, supporting a shared mechanism of MPs and nanoplastic translocation [36].

MPs penetrate granulosa cells, which play a critical role in follicle and oocyte development. Rodent studies have shown that MPs accumulation in ovaries and granulosa cells impairs follicular growth and disrupts hormonal balance. Exposure to MPs results in decreased AMH and E2 levels, alongside elevated FSH levels, which contribute to irregular menstrual cycles and disrupted folliculogenesis [37].

Further effects include thinning of the granulosa layer in secondary follicles, reduced numbers of mature follicles, the formation of ovarian cysts, atretic follicles, and ovarian fibrosis [36,37]. MPs may also disrupt the hypothalamic-pituitary-ovarian axis, leading to aberrant hormonal signaling [38]. MPs exposure reduces ovarian mass and induces oxidative stress, as evidenced by reduced antioxidant enzyme activity and elevated lipid peroxidation levels. The ensuing inflammation further compromises ovarian function [31,36,38].

3.3 Endometrium

MPs affect the vascular system, particularly small uterine arteries, impairing endometrial development and disrupting embryo implantation processes. Chronic accumulation of MPs and nanoplastics in reproductive tissues may lead to various pathologies including premature puberty, menstrual irregularities, premature ovarian insufficiency, endometriosis, uterine fibroids, and miscarriage [36].

In a study by Sun et al. (2024), 13 types of MPs were detected in endometrial tissue samples, including PE, polyethylene terephthalate (PET), PS, PVC, PP, polyurethane (PU), ethylene acrylic acid (EAA), acrylate (ACR), flame retardants (FR), and butadiene rubber. Furthermore, a correlation was established between dietary habits and the concentration of MPs in the body [39].

Another study by Kim et al. (2025) analyzed MPs accumulation in human endometrial stromal cells (hESCs) and found that higher concentrations of PS, especially 0.1 μm, significantly reduced cell viability, exposed cells showed increased reactive oxygen species (ROS) production, MPs were localized in the cytoplasm and in some cases near the nucleus, and morphological changes suggestive of cell damage and inflammation were observed [40]. Micro- and nanoplastics may exert toxic effects on endometrial cells by impairing their physiological functions, inducing oxidative stress, and potentially disrupting embryo implantation or promoting the development of endometriosis.

4. The Male Reproductive System

According to statistics from 1940 to 1990, the quality of human semen has decreased – its concentration lowered from 113 x 10^6/ml to 66 x 10^6/ml [41]. The data may vary according to different statistics, but they all are consistent with the fact that the sperm concentration dropped to a percentage of its original value. The timing of such observations draws attention to MPs' increasing existence in the environment [8]. There is still a lack of human studies in such matter, but mice were a subject of research trials. In one study polystyrene PS-MPs exposure led to decreased sperm quality and testosterone levels in male mice [42,43]. Apart from MPs' direct influence on male mice, polystyrene nanoplastics (PS-NPs) reduced testicular weight, destroyed reproductive epithelium and reduced sperm count in male mice prenatally [44], but also decreased sperm motility [45].

MPs affect sperm's DNA, which is crucial for development of the reproductive system of male organisms [46]. This quality of sperm's DNA is expressed by DNA fragmentation index (DFI). This index is higher in infertile men – sperm DFI is used to assess male fertility. Studies showed that nanoplastics (NPs) exposure led to increased DFI in rats [8].

Research indicates that MPs negatively affect male mice reproductive function, including the induction of vacuolization in germ cells [47], reduction of testosterone levels [47,48], decrease in sperm count, and an increase in the proportion of morphologically abnormal sperm [47,48,49]. In their study, Wen et al. (2023) demonstrated that the use of an endoplasmic reticulum stress inhibitor alleviated testicular damage and restored testosterone levels almost to normal [48]. These findings provide hope that targeting endoplasmic reticulum stress could become a potential therapeutic strategy to counteract the harmful effects of MPs on the reproductive system. Molecular analyses have also shown that micro- and nanoplastic particles induce the expression of apoptosis-related proteins through activation of the p53 signaling pathway [42]. Importantly, it has been suggested that exposure to nanoplastics may lead not only to impaired fertility but even to infertility in men [50].

MPs can destroy the reproductive system by a few mechanisms – oxidative stress, disruption of the hypothalamic-pituitary-gonadal (HPG) axis and energy consumption [6,8,51]. Nanoparticles of MPs may interact with electron remittance of intracellular mediators, leading to redox dysfunction and appearance of reactive oxygen species (ROS) [52]. ROS may be toxic to embryos and oxidative stress itself may result in loss of fertilization ability of oocytes.

Disruption of HPG axis in male mice due to MPs toxicity manifests itself in decreased levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH) and testosterone (T) – again, there is lack of human-based studies. As similar results were obtained in studies with male rats, conclusion can be made that MPs disrupt steroid production pathway, which leads to imbalance of sex hormones and that can contribute to many reproductive pathologies such as delaying gonad maturation [6,51].

Microplastics (MPs) have been shown to exert toxic effects on the reproductive systems of various aquatic organisms, including oysters, medaka fish, and *Daphnia* [37,38]. In oysters, MPs were found to impair spermatogenesis by reducing energy availability through decreased activity of lactate dehydrogenase and succinate dehydrogenase, ultimately compromising sperm quality
[8].

Although animal studies have shown destructive effects of MPs on the reproductive system, which may lead to the assumption that MPs are equally harmful to humans, further research needs to be undertaken. The fact is that infertility is increasing every year and male factors are becoming more prominent. As mentioned, semen quality is declining and this phenomenon correlates with the modernization of today's world. The increasing consumption of MPs cannot be overlooked in this issue and must be investigated in the future.

5. Conclusions

MPs represent an increasingly concerning environmental contaminant that poses serious threats to human health, including the reproductive system. MPs can accumulate in reproductive organs, both male (testes) and female (placenta, follicular fluid). *In vivo* studies indicate that exposure to MPs leads to impaired fertility in both males and females - manifesting as changes in gamete quality, gonadal structure, and offspring development. Once MPs cross biological barriers, they exert cytotoxic effects by inducing oxidative stress, triggering inflammatory responses, and disrupting hormonal signaling pathways such as the hypothalamic–pituitary–gonadal (HPG) axis, as demonstrated primarily by *in vitro* and *in vivo* research. However, there is a lack of studies involving human subjects, which highlights the urgent need for further investigation into this emerging threat. In light of the global decline in fertility rates and the growing burden of plastic pollution, understanding and mitigating the impact of MPs on fertility should become a public health priority.

Disclosure Author's contribution

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