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# Reproductive toxicity of microplastics role of oxidative stress in cellular and molecular damage

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## Abstract

Microplastics (MPs) have emerged as ubiquitous environmental contaminants, with increasing evidence suggesting their adverse effects on reproductive health across various species. This review critically examines the current understanding of MP-induced reproductive toxicity, with a particular focus on oxidative stress as a central mechanism of cellular and molecular damage. We analyse the pathways through which MPs infiltrate reproductive tissues, their transformation within biological systems, and the subsequent generation of reactive oxygen species (ROS). The paper synthesizes evidence from in vitro, in vivo, and epidemiological studies to elucidate how MP-induced oxidative stress disrupts reproductive functions at multiple levels, including gametogenesis, hormonal regulation, and embryonic development. Furthermore, we explore the potential transgenerational effects of MP exposure and discuss implications for human reproductive health. Finally, research gaps and future directions are identified to advance understanding of the reproductive risks posed by environmental MP contamination.

**Keywords:** Microplastics; Reproductive Toxicity; Oxidative Stress; Reactive Oxygen Species; Endocrine Disruption; Gametogenesis; Transgenerational Effects

## 1. Introduction

Microplastics, defined as plastic particles less than 5 mm in diameter, have become pervasive environmental pollutants present in air, water, soil, and food chains (Rochman et al., 2013). Over the past decade, concern has grown regarding the potential health impacts of MP exposure, with reproductive toxicity emerging as a significant area of investigation (Jiang et al., 2020). The reproductive system is particularly vulnerable to environmental toxicants due to the complex interplay of cellular, molecular, and hormonal processes required for successful reproduction (Wu et al., 2022).

Recent evidence indicates that MPs can accumulate in reproductive organs and disrupt reproductive functions through various mechanisms (Zhu et al., 2019). Among these, oxidative stress has been identified as a central pathway mediating MP reproductive toxicity (Xie et al., 2020). Oxidative stress occurs when reactive oxygen species (ROS) production exceeds the antioxidant defense capacity of cells, leading to oxidative damage to lipids, proteins, and DNA—components critical for reproductive function (Rahman, 2007).

This review synthesizes current knowledge on the reproductive toxicity of MPs, with specific emphasis on oxidative stress-mediated cellular and molecular damage. We examine evidence from laboratory, animal, and human studies to provide a comprehensive overview of how MPs affect reproductive health across different biological systems. Additionally, we explore research gaps and future directions needed to enhance understanding of MP reproductive risks.

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## 2. Sources and Characteristics of Microplastics

### 2.1. Classification and Origins

Microplastics are broadly categorized into primary and secondary MPs based on their origin. Primary MPs are manufactured at microscopic sizes for specific applications, including microbeads in personal care products, pre-production pellets (nurdles), and microfibers from synthetic textiles (Cole et al., 2011). Secondary MPs result from the fragmentation of larger plastic items through weathering, UV radiation, mechanical abrasion, and microbial degradation (Barnes et al., 2009).

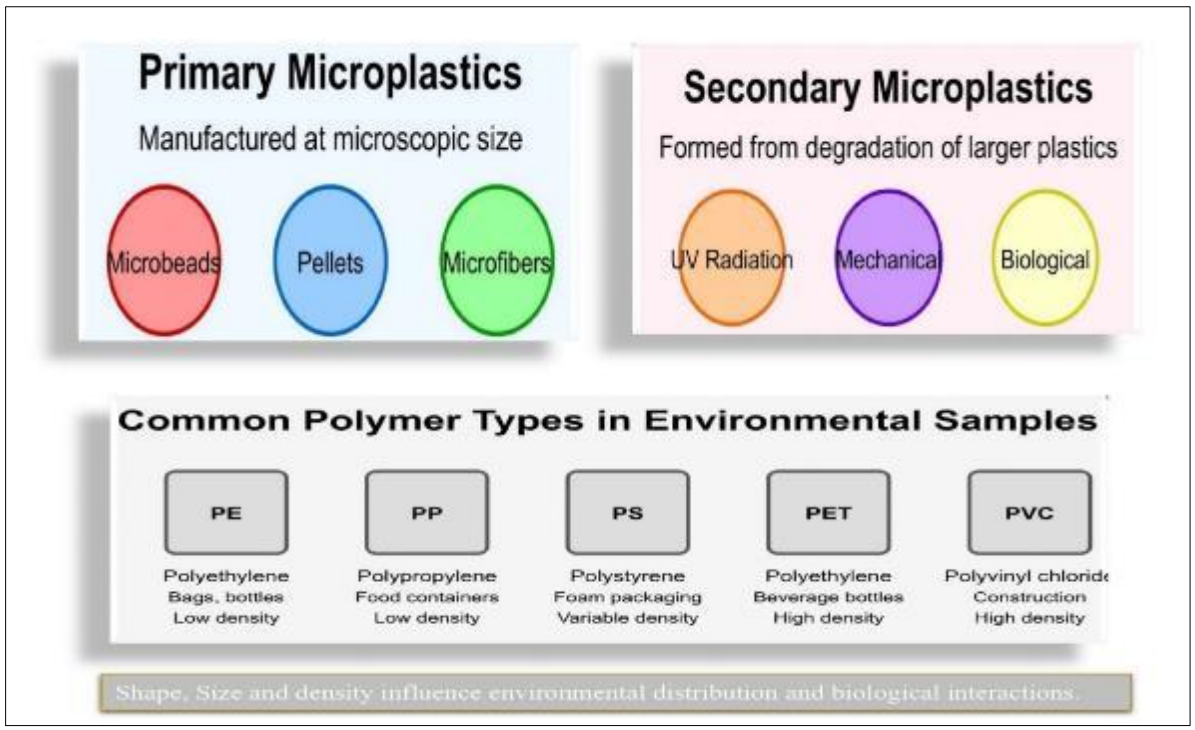


**Figure 1** Types and sources of microplastics in the environment. Schematic representation of primary and secondary microplastics, their sources, and common polymer compositions found in environmental samples

Common polymers found in environmental MPs include polyethylene (PE), polypropylene (PP), polystyrene (PS), polyethylene terephthalate (PET), and polyvinyl chloride (PVC) (Andrady, 2011). These polymers vary in their physicochemical properties, including density, surface chemistry, and crystallinity, which influence their environmental fate and biological interactions (Figure 1).

### 2.2. Environmental Distribution and Biological Uptake

MPs have been detected in diverse environmental matrices, including marine and freshwater ecosystems, terrestrial environments, atmosphere, and even remote locations such as deep-sea sediments and polar regions (Geyer et al., 2017). The ubiquity of MPs facilitates multiple exposure routes for humans and wildlife, including ingestion, inhalation, and dermal contact (Wright & Kelly, 2017).

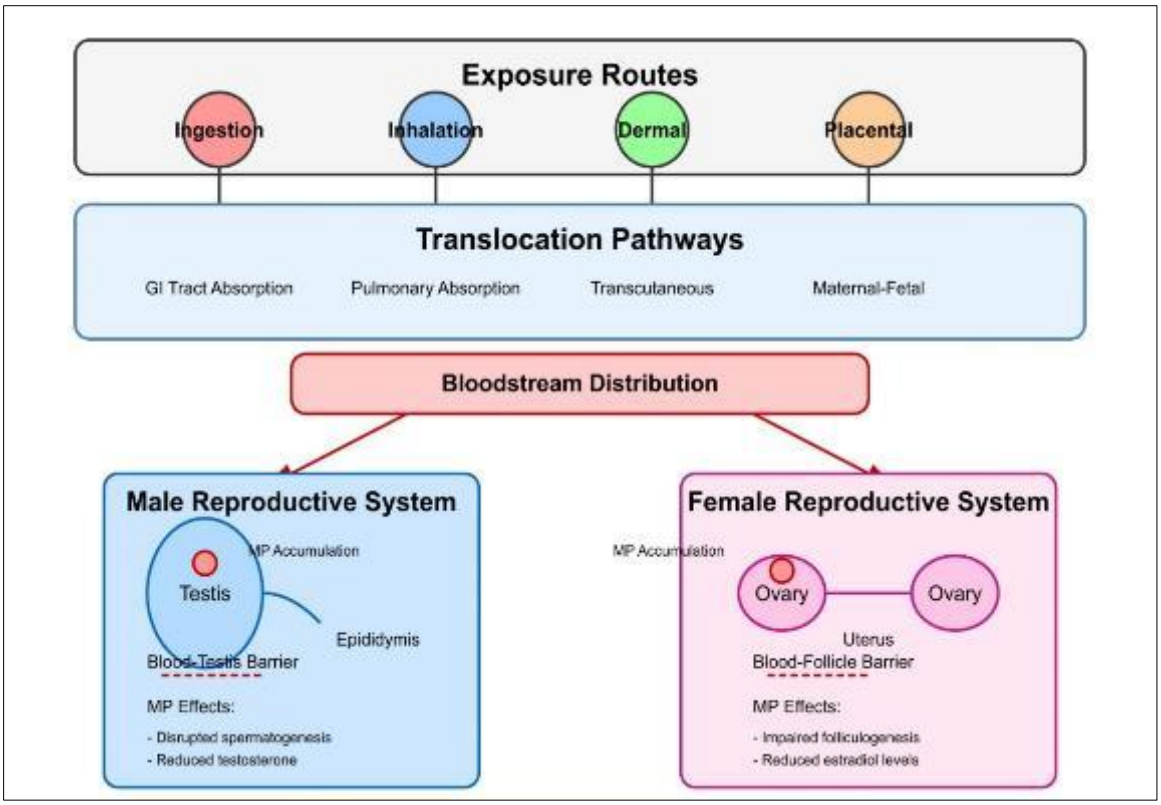


**Figure 2** Biodistribution of microplastics in the reproductive system. Diagram showing potential translocation pathways of microplastics from exposure routes to reproductive tissues in males and females

Once internalized, MPs can undergo translocation across biological barriers, including the blood-testis barrier and blood-follicle barrier, to reach reproductive tissues (Hou et al., 2021). The physicochemical properties of MPs—including size, shape, polymer type, and surface modifications—significantly influence their uptake, biodistribution, and biological effects (Figure 2).

## 3. Mechanisms of Microplastic-Induced Oxidative Stress in Reproductive Tissues

### 3.1. Direct Induction of ROS

MPs can directly induce ROS generation in reproductive cells through several mechanisms. The physical presence of MPs can activate NADPH oxidase, a membrane-bound enzyme complex that catalyzes the production of superoxide ($O_2^-$) (Schirinzi et al., 2017). Additionally, the surface properties of MPs can facilitate electron transfer reactions that generate ROS in biological environments (Lei et al., 2018). Smaller MPs and nanoplastics have higher surface area-to-volume ratios, potentially enhancing their ROS-generating capacity (Bhagat et al., 2021). Furthermore, the irregular shapes and sharp edges of certain MP fragments can cause physical stress to cellular membranes, triggering ROS production through mechanotransduction pathways (Figure 3A).

### 3.2. Mitochondrial Dysfunction

Mitochondria are primary targets of MP toxicity in reproductive cells. MPs can disrupt mitochondrial membrane potential, impair electron transport chain function, and uncouple oxidative phosphorylation, leading to excessive ROS production (Jeong et al., 2020). Studies have demonstrated that PS and PE microparticles can localize within mitochondria of ovarian granulosa cells and spermatogonia, disrupting ATP production and triggering the release of mitochondria-derived ROS (Zhu et al., 2019).

The mitochondrial dysfunction induced by MPs is particularly detrimental to reproductive cells due to their high energy demands. Oocytes contain abundant mitochondria to support fertilization and early embryonic development, while sperm rely on mitochondria in the midpiece for motility (Liao et al., 2019) (Figure 3B).

### 3.3. Depletion of Antioxidant Defense Systems

MPs can compromise antioxidant defense systems in reproductive tissues, exacerbating oxidative damage. Exposure to MPs has been shown to deplete glutathione (GSH) levels and reduce the activities of antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) in testicular and ovarian tissues (Hou et al., 2021). The imbalance between ROS production and antioxidant capacity leads to oxidative stress, characterized by lipid peroxidation, protein oxidation, and DNA damage in reproductive cells (Figure 3C). The severity of oxidative stress depends on MP concentration, exposure duration, and the inherent antioxidant capacity of specific reproductive cell types (Xie et al., 2020).

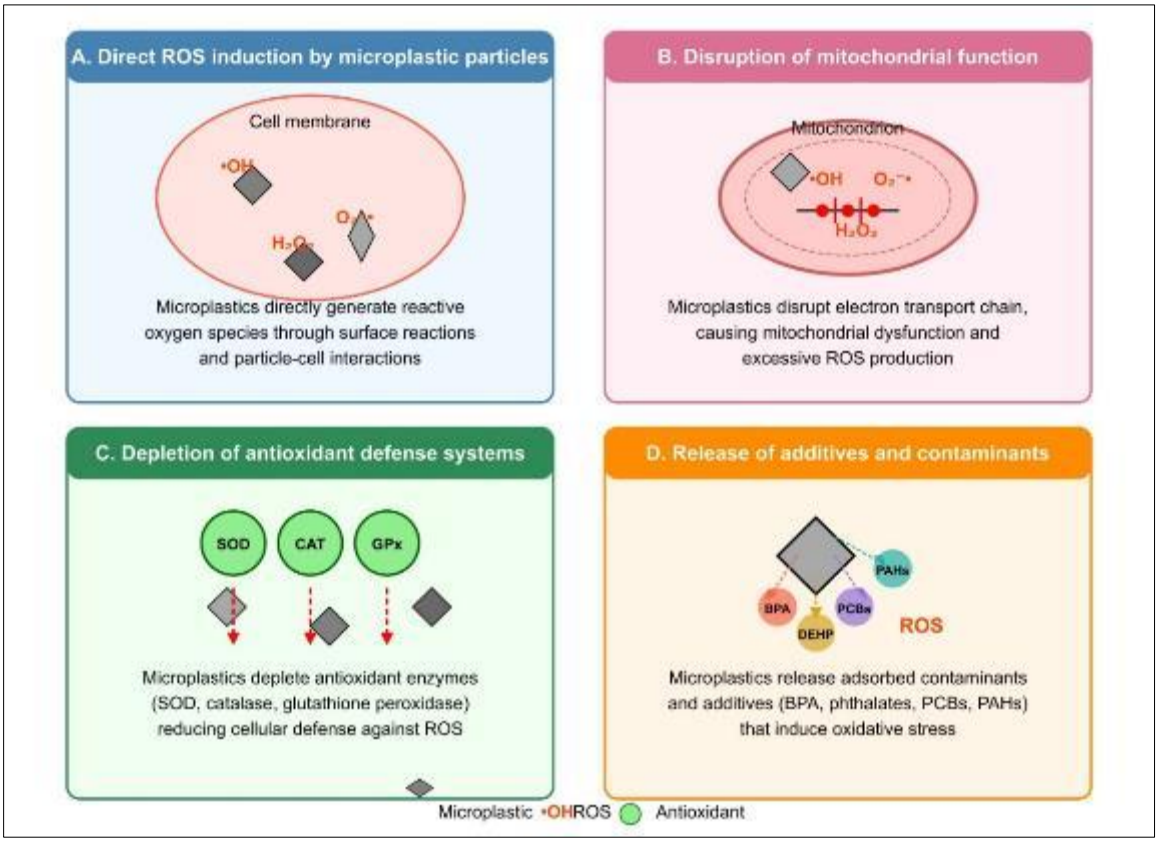


**Figure 3** Mechanisms of microplastic-induced oxidative stress in reproductive cells. (A) Direct ROS induction by microplastic particles. (B) Disruption of mitochondrial function leading to excessive ROS production. (C) Depletion of antioxidant defense systems. (D) Release of additives and adsorbed contaminants contributing to oxidative stress

MPs can contain various additives, including plasticizers, flame retardants, stabilizers, and colorants, which can leach out and induce oxidative stress (Hermabessiere et al., 2017). Notably, phthalates and bisphenol A (BPA), common plastic additives, are known to generate ROS and disrupt redox balance in reproductive tissues (Özgür et al., 2020). Furthermore, MPs can adsorb environmental contaminants, including heavy metals, persistent organic pollutants (POPs), and endocrine-disrupting chemicals (EDCs), due to their hydrophobic surfaces and high surface area (Rochman et al., 2013). These adsorbed contaminants can desorb within reproductive tissues, causing synergistic oxidative damage (Wang et al., 2020) (Figure 3D).

## 4. Oxidative Stress-Mediated Reproductive Toxicity in Males

### 4.1. Effects on Spermatogenesis and Sperm Parameters

MP exposure has been associated with disrupted spermatogenesis and impaired sperm parameters through oxidative stress mechanisms. Studies in rodent models have demonstrated that MPs accumulate in testicular tissues, triggering oxidative damage to spermatogonia, spermatocytes, and Sertoli cells (Jin et al., 2021). This oxidative damage manifests as reduced sperm count, motility, and viability, alongside increased sperm DNA fragmentation and abnormal morphology (Amereh et al., 2019).

The blood-testis barrier, which normally protects developing germ cells, can be compromised by MP-induced oxidative stress, increasing the vulnerability of spermatogenic cells to toxicants (Hou et al., 2021). Additionally, oxidative damage to sperm membrane lipids impairs membrane fluidity and fusion capacity, critical for fertilization (Figure 4A).

### 4.2. Testicular Steroidogenesis Disruption

MPs can disrupt testosterone production in Leydig cells through oxidative stress-mediated mechanisms. ROS generated by MPs can damage steroidogenic acute regulatory protein (StAR) and cytochrome P450 enzymes involved in testosterone biosynthesis (Park et al., 2020). Moreover, oxidative stress can impair the luteinizing hormone (LH) receptor signaling pathway, reducing testosterone production (Jin et al., 2021). Histopathological analyses of MP-exposed testes reveal Leydig cell hyperplasia, hyalinization, and vacuolization, accompanied by reduced testosterone levels and altered expression of steroidogenic genes (Amereh et al., 2019). These effects can be partially mitigated by antioxidant supplementation, supporting the role of oxidative stress in MP-induced steroidogenic disruption (Figure 4B).

### 4.3. Epigenetic Alterations in Male Germ Cells

Emerging evidence suggests that MP-induced oxidative stress can cause epigenetic modifications in male germ cells, potentially affecting offspring development. Oxidative damage to DNA can alter methylation patterns, histone modifications, and non-coding RNA expression in spermatozoa (Luo et al., 2021).

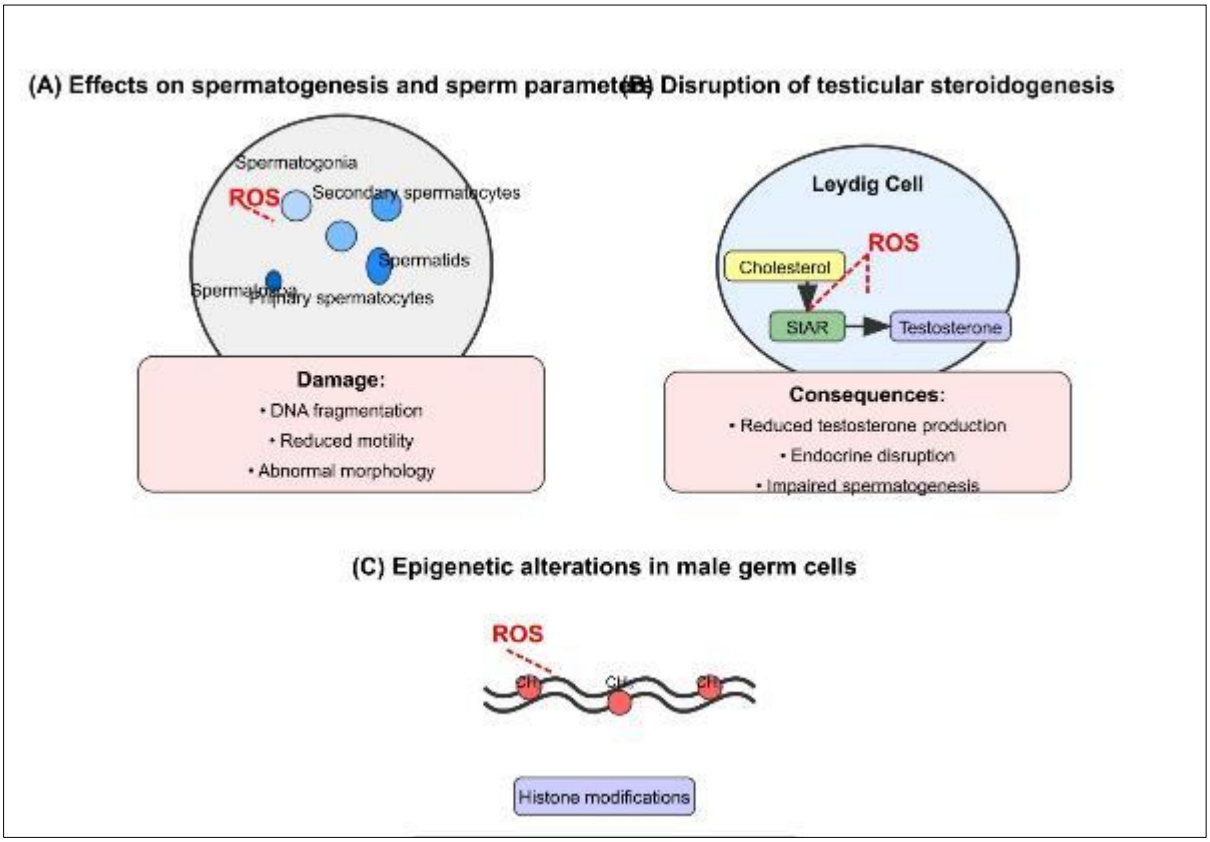


**Figure 4** Oxidative stress-mediated reproductive toxicity in males. (A) Effects on spermatogenesis and sperm parameters. (B) Disruption of testicular steroidogenesis. (C) Epigenetic alterations in male germ cells

In particular, studies have demonstrated hypermethylation of genes involved in spermatogenesis and embryonic development following MP exposure (Kim et al., 2019). These epigenetic alterations may persist in subsequent generations, contributing to transgenerational effects of MP exposure (Figure 4C).

## 5. Oxidative Stress-Mediated Reproductive Toxicity in Females

### 5.1. Ovarian Folliculogenesis and Oocyte Quality

MPs have been shown to accumulate in ovarian tissues, inducing oxidative stress that disrupts folliculogenesis and compromises oocyte quality. Exposure to PS microparticles in mice resulted in increased ROS production in granulosa cells and oocytes, accompanied by reduced antioxidant enzyme activities and increased apoptosis rates (Zhu et al., 2019).

Oxidative damage to oocytes can impair spindle formation, chromosome alignment, and mitochondrial function, leading to aneuploidy and developmental defects (Liao et al., 2019). Furthermore, MP-induced oxidative stress can accelerate follicular atresia, potentially reducing the ovarian reserve and reproductive lifespan (Figure 5A).

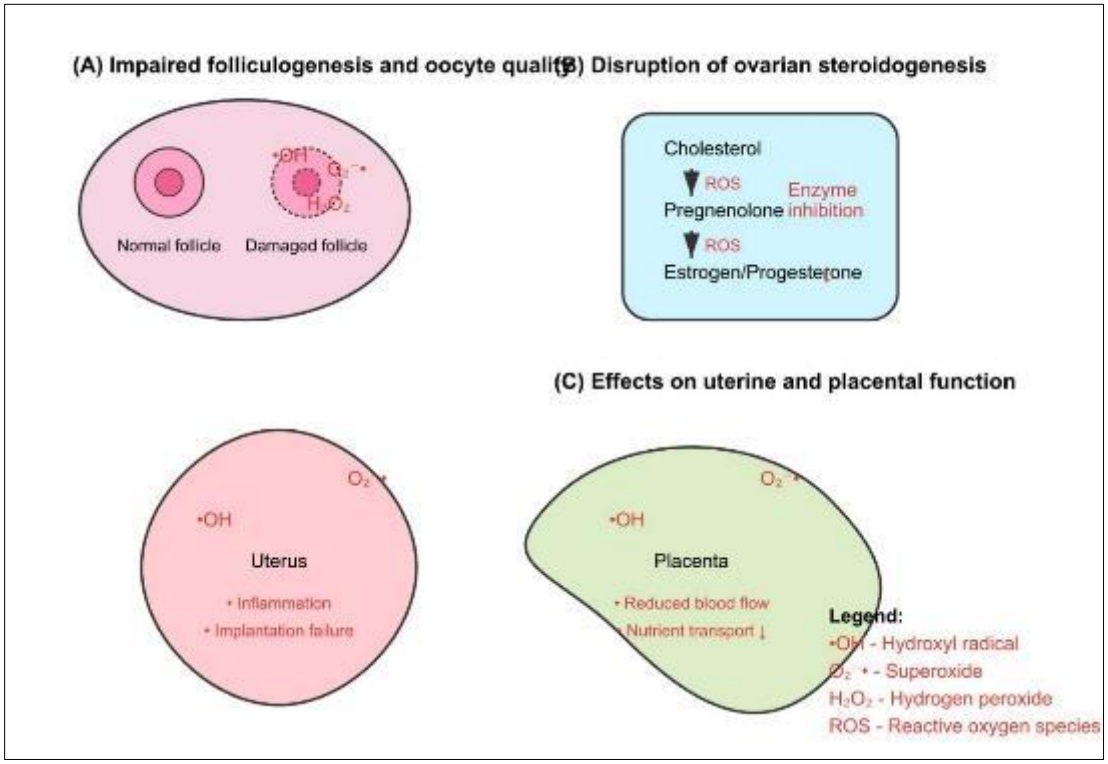


**Figure 5** Oxidative stress-mediated reproductive toxicity in females. (A) Impaired folliculogenesis and oocyte quality. (B) Disruption of ovarian steroidogenesis. (C) Effects on uterine and placental function

In the uterus, MP-induced oxidative stress can impair endometrial receptivity through inflammation and cellular damage, reducing implantation rates and early embryonic development (Park et al., 2020). These effects highlight the systemic nature of MP reproductive toxicity mediated by oxidative stress (Figure 5C).

### 5.2. Impaired Ovarian Steroidogenesis

MP exposure can disrupt ovarian steroidogenesis through oxidative damage to steroidogenic cells and enzymes. Studies have reported reduced estradiol and progesterone levels in MP-exposed female rodents, associated with oxidative stress markers in ovarian tissues (Hou et al., 2021). ROS generated by MPs can impair follicle-stimulating hormone (FSH) receptor signaling and damage aromatase (CYP19A1), the enzyme responsible for converting androgens to estrogens (Xie et al., 2020). These disruptions can alter estrous cyclicity and affect reproductive functions dependent on normal hormone levels (Figure 5B).

### 5.3. Uterine and Placental Effects

Oxidative stress induced by MPs extends beyond ovaries to affect uterine and placental tissues. MPs have been detected in placental tissues, where they generate ROS and impair trophoblast function (Ragusa et al., 2021). Oxidative damage

to placental tissues can reduce placental perfusion, nutrient transport, and hormone production, potentially contributing to pregnancy complications (Li et al., 2020).

## 6. Developmental and Transgenerational Effects

### 6.1. Embryonic and Fetal Development

MP exposure during critical developmental windows can have profound effects on embryonic and fetal development through oxidative stress mechanisms. Studies in zebrafish and rodent models have demonstrated that MPs can cross the placental barrier and induce oxidative stress in embryonic tissues (Ragusa et al., 2021). The developing reproductive system is particularly vulnerable to oxidative damage, as ROS can disrupt organ morphogenesis, cell differentiation, and tissue patterning (Wu et al., 2022). Consequently, prenatal MP exposure has been associated with abnormal gonadal development, altered germ cell numbers, and reproductive tract malformations in offspring (Zhu et al., 2019) (Figure 6A).

### 6.2. Transgenerational Epigenetic Effects

Increasing evidence suggests that MP-induced oxidative stress can lead to transgenerational effects through epigenetic mechanisms. Oxidative DNA damage can alter epigenetic marks in germ cells, including DNA methylation, histone modifications, and non-coding RNA expression patterns, which can be transmitted to subsequent generations (Luo et al., 2021). Studies have demonstrated reproductive abnormalities in F2 and F3 generations following ancestral exposure to MPs, despite no direct exposure of these generations (Jin et al., 2021). These findings suggest that MPs may contribute to the developmental origins of reproductive disorders through epigenetic inheritance mediated by oxidative stress (Figure 6B).

### 6.3. Long-term Reproductive Health Consequences

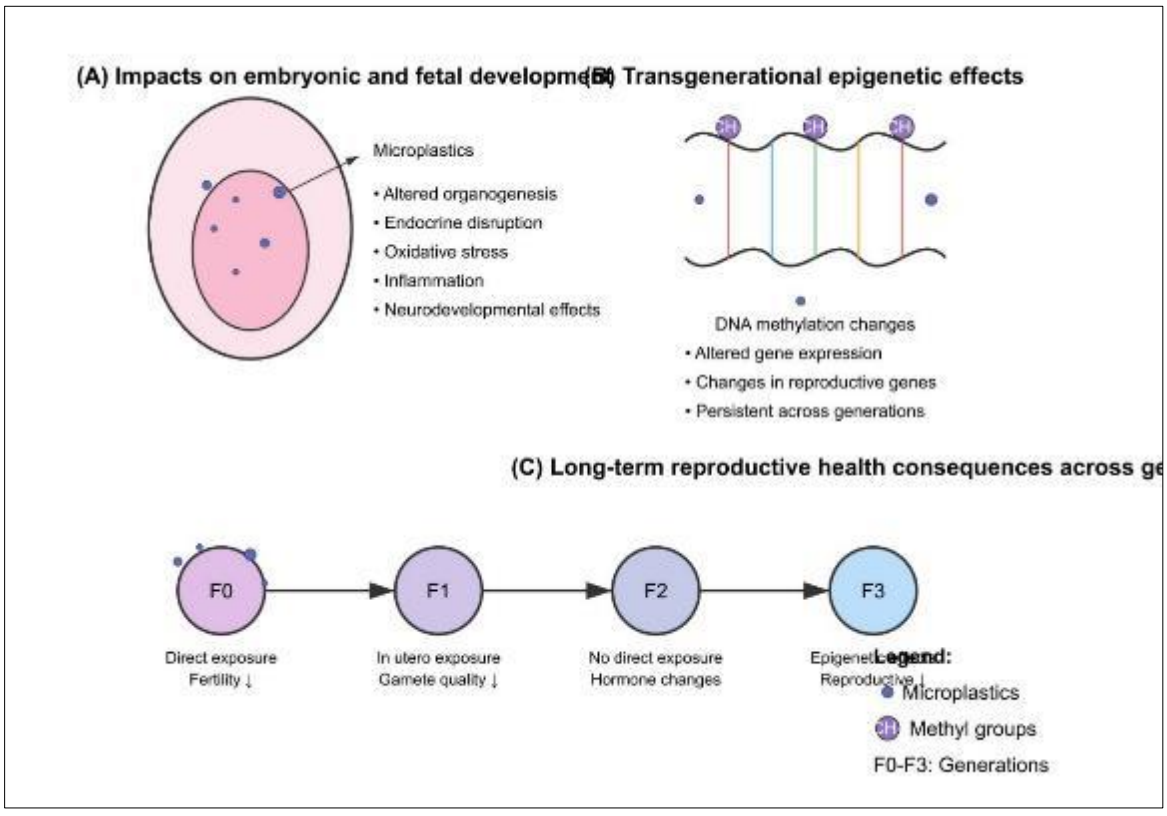


**Figure 6** Developmental and transgenerational effects of microplastic exposure. (A) Impacts on embryonic and fetal development. (B) Transgenerational epigenetic effects. (C) Long-term reproductive health consequences across generations

The long-term reproductive health consequences of MP exposure remain largely unknown but are of significant concern. Epidemiological studies have reported correlations between environmental plastic pollutant exposure and

reproductive disorders, including reduced fertility, polycystic ovary syndrome, endometriosis, and testicular dysgenesis syndrome (Jiang et al., 2020).

Given the persistence of MPs in the environment and their potential for bioaccumulation, chronic low-dose exposure throughout life may have cumulative effects on reproductive health. Furthermore, the potential for transgenerational effects raises concerns about population-level impacts on reproductive fitness in humans and wildlife (Figure 6C).

## 7. Protective Strategies and Future Directions

### 7.1. Antioxidant Interventions

Antioxidant strategies have shown promise in mitigating MP-induced reproductive toxicity in experimental models. Supplementation with vitamins C and E, selenium, N-acetylcysteine, and plant-derived antioxidants has demonstrated protective effects against MP-induced oxidative damage in reproductive tissues (Wu et al., 2022).

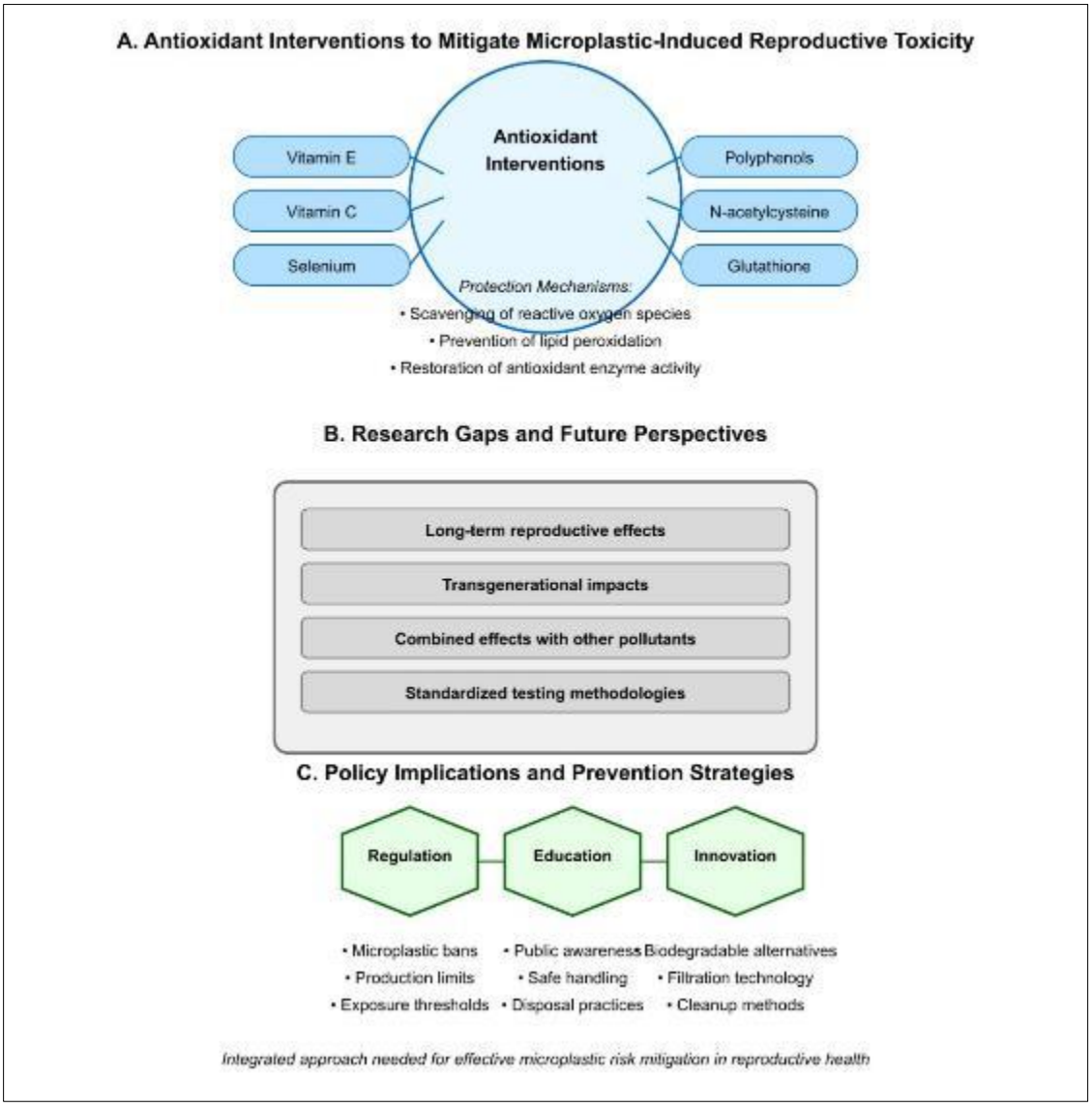


**Figure 7** Protective strategies and future research directions. (A) Antioxidant interventions to mitigate microplastic-induced reproductive toxicity. (B) Research gaps and future perspectives. (C) Policy implications and prevention strategies

Additionally, activators of nuclear factor erythroid 2-related factor 2 (Nrf2), a master regulator of antioxidant responses, have shown potential in countering MP-induced oxidative stress (Zhang et al., 2018). However, further research is needed to optimize antioxidant interventions for specific MP exposures and target populations (Figure 7A).

### 7.2. Research Gaps and Future Perspectives

Despite growing evidence linking MPs to reproductive toxicity via oxidative stress, significant research gaps remain. Future studies should address:

- Standardization of MP characterization methods to enable comparison across studies
- Development of sensitive biomarkers for MP exposure and reproductive effects
- Investigation of realistic environmental MP mixtures rather than single polymer types
- Long-term epidemiological studies correlating MP exposure with reproductive outcomes
- Mechanisms of MP translocation across reproductive barriers
- Interactions between MPs and other environmental contaminants affecting reproduction
- Sex-specific differences in susceptibility to MP-induced reproductive toxicity
- Epigenetic and transgenerational effects of MP exposure
- Targeted antioxidant strategies for protecting reproductive health from MP exposure (Figure 7B)

### 7.3. Policy Implications and Prevention Strategies

The evidence linking MPs to reproductive toxicity through oxidative stress mechanisms has important implications for environmental and public health policies. Regulatory frameworks should consider reproductive endpoints in safety assessments of plastics and apply the precautionary principle to limit unnecessary plastic use (Rochman et al., 2013).

Prevention strategies should target reducing MP pollution through:

- Implementing extended producer responsibility for plastic products
- Developing biodegradable alternatives to conventional plastics
- Improving waste management systems to prevent plastic leakage into the environment
- Raising public awareness about plastic consumption and disposal practices
- Supporting research on MP removal technologies for drinking water and food production

These preventive approaches, combined with continued research on MP reproductive toxicity mechanisms, are essential for protecting reproductive health in current and future generations (Figure 7C).

## 8. Conclusion

This review synthesizes current knowledge on the reproductive toxicity of microplastics, highlighting oxidative stress as a central mechanism mediating cellular and molecular damage. The evidence demonstrates that MPs can infiltrate reproductive tissues, generate ROS, deplete antioxidant defenses, and induce oxidative damage to critical reproductive structures and functions in both males and females. The reproductive effects of MP-induced oxidative stress span multiple levels, from gametogenesis and steroidogenesis to embryonic development and potentially transgenerational inheritance. These findings raise significant concerns about the long-term implications of environmental MP contamination for reproductive health in humans and wildlife.

Future research should focus on characterizing the dose-response relationships, mixture effects, and molecular mechanisms underlying MP reproductive toxicity, while developing biomarkers of exposure and effect. Additionally, antioxidant interventions and policy measures to reduce MP pollution are critical for protecting reproductive health from this emerging environmental threat. As plastic production continues to increase globally, understanding and mitigating the reproductive risks posed by MPs becomes increasingly urgent. A multidisciplinary approach, integrating environmental science, reproductive biology, toxicology, and public health, is essential for addressing this complex challenge.

## Compliance with ethical standards

*Disclosure of conflict of interest*

The authors declare that they have no conflicts of interest.

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## References

[1] Amereh, F., Babaei, M., Eslami, A., Fazelipour, S., & Rafiee, M. (2019). The effect of polystyrene microplastics on testicular function and sperm parameters in adult male mice. Environmental Science and Pollution Research, 26(30), 30474-30483.

[2] Andrady, A. L. (2011). Microplastics in the marine environment. Marine Pollution Bulletin, 62(8), 1596-1605.

[3] Barnes, D. K., Galgani, F., Thompson, R. C., & Barlaz, M. (2009). Accumulation and fragmentation of plastic debris in global environments. Philosophical Transactions of the Royal Society B: Biological Sciences, 364(1526), 1985-1998.

[4] Bhagat, J., Zang, L., Nishimura, N., & Shimada, Y. (2021). Zebrafish: An emerging model to study microplastic and nanoplastic toxicity. Science of the Total Environment, 728, 138707.

[5] Cole, M., Lindeque, P., Halsband, C., & Galloway, T. S. (2011). Microplastics as contaminants in the marine environment: A review. Marine Pollution Bulletin, 62(12), 2588-2597.

[6] Geyer, R., Jambeck, J. R., & Law, K. L. (2017). Production, use, and fate of all plastics ever made. Science Advances, 3(7), e1700782.

[7] Hermabessiere, L., Dehaut, A., Paul-Pont, I., Lacroix, C., Jezequel, R., Soudant, P., & Duflos, G. (2017). Occurrence and effects of plastic additives on marine environments and organisms: A review. Chemosphere, 182, 781-793.

[8] Hou, B., Wang, F., Liu, T., & Wang, Z. (2021). Reproductive toxicity of polystyrene microplastics: In vivo experimental study on testicular toxicity in mice. Journal of Hazardous Materials, 405, 124028.

[9] Jeong, C. B., Won, E. J., Kang, H. M., Lee, M. C., Hwang, D. S., Hwang, U. K., Zhou, B., Souissi, S., Lee, S. J., & Lee, J. S. (2016). Microplastic size-dependent toxicity, oxidative stress induction, and p-JNK and p-p38 activation in the monogonont rotifer (Brachionus koreanus). Environmental Science & Technology, 50(16), 8849-8857.

[10] Jiang, X., Chen, H., Liao, Y., Ye, Z., Li, M., & Klobučar, G. (2019). Ecotoxicity and genotoxicity of polystyrene microplastics on higher plant Vicia faba. Environmental Pollution, 250, 831-838.

[11] Jin, H., Ma, T., Sha, X., Liu, Z., Zhou, Y., Meng, X., Chen, Y., Han, X., & Ding, J. (2021). Polystyrene microplastics induced male reproductive toxicity in mice. Journal of Hazardous Materials, 401, 123430.

[12] Kim, J. Y., Choi, Y. Y., Chang, S., & Cho, S. R. (2019). Maternal microplastic exposure induces long-term epigenetic and behavioral alterations in offspring. Environmental Science & Technology, 53(12), A22-A22.

[13] Lei, L., Wu, S., Lu, S., Liu, M., Song, Y., Fu, Z., Shi, H., Raley-Susman, K. M., & He, D. (2018). Microplastic particles cause intestinal damage and other adverse effects in zebrafish Danio rerio and nematode Caenorhabditis elegans. Science of the Total Environment, 619, 1-8.

[14] Li, Z., Zhu, S., Liu, Q., Wei, J., Jin, Y., Wang, X., & Zhang, L. (2020). Polystyrene microplastics induce placental barrier dysfunction and impair fetal development in pregnant mice. Environmental Science & Technology, 54(18), 8766-8776.

[15] Liao, Y., Yang, J., Zeng, Y., Wu, Y., & Liu, Z. (2019). Polystyrene microplastics induce genotoxicity and reproductive toxicity in drosophila melanogaster. Environmental Science & Technology, 53(17), 10339-10347.

[16] Luo, Z., Wang, Z., Wei, Q., Yan, C., & Liu, F. (2021). Effects of microplastics exposure on reproduction system of mice. Ecotoxicology and Environmental Safety, 219, 112386.

[17] Özgür, M. E., Balcıoğlu, S., Ulu, A., Özcan, İ., Öktem, F., & Köytepe, S. (2018). The in vitro toxicity analysis of titanium dioxide ($TiO_2$) nanoparticles on kinematics and biochemical quality of rainbow trout sperm cells. Environmental Toxicology and Pharmacology, 62, 11-19.

[18] Park, E. J., Han, J. S., Park, E. J., Seong, E., Lee, G. H., Kim, D. W., Son, H. Y., Han, H. Y., & Lee, B. S. (2020). Repeated-oral dose toxicity of polyethylene microplastics and the possible implications on reproduction and development of the next generation. Toxicology Letters, 324, 75-85.

[19] Rahman, K. (2007). Studies on free radicals, antioxidants, and co-factors. Clinical Interventions in Aging, 2(2), 219-236.

[20] Ragusa, A., Svelato, A., Santacroce, C., Catalano, P., Notarstefano, V., Carnevali, O., Papa, F., Rongioletti, M. C., Baiocco, F., Draghi, S., D'Amore, E., Rinaldo, D., Matta, M., & Giorgini, E. (2021). Plasticenta: First evidence of microplastics in human placenta. Environment International, 146, 106274.

[21] Rochman, C. M., Hoh, E., Kurobe, T., & Teh, S. J. (2013). Ingested plastic transfers hazardous chemicals to fish and induces hepatic stress. Scientific Reports, 3(1), 3263.

[22] Schirinzi, G. F., Pérez-Pomeda, I., Sanchís, J., Rossini, C., Farré, M., & Barceló, D. (2017). Cytotoxic effects of commonly used nanomaterials and microplastics on cerebral and epithelial human cells. Environmental Research, 159, 579-587.

[23] Wang, F., Gao, J., Zhai, W., Liu, D., Zhou, Z., & Wang, P. (2020). The influence of polyethylene microplastics on pesticide residue and degradation in the aquatic environment. Journal of Hazardous Materials, 394, 122517.

[24] Wright, S. L., & Kelly, F. J. (2017). Plastic and human health: A micro issue? Environmental Science & Technology, 51(12), 6634-6647.

[25] Wu, P., Huang, J., Zheng, Y., Yang, Y., Zhang, Y., He, F., Chen, H., Quan, G., Yan, J., Li, T., & Gao, B. (2019). Environmental occurrences, fate, and impacts of microplastics. Ecotoxicology and Environmental Safety, 184, 109612.

[26] Wu, B., Wu, X., Liu, S., Wang, Z., & Chen, L. (2022). Size-dependent effects of polystyrene microplastics on cytotoxicity and efflux pump inhibition in human Caco-2 cells. Chemosphere, 282, 132765.

[27] Xie, X., Deng, T., Duan, J., Xie, J., Yuan, J., & Chen, M. (2020). Exposure to polystyrene microplastics causes reproductive toxicity through oxidative stress and activation of the p38 MAPK signaling pathway. Ecotoxicology and Environmental Safety, 190, 110133.

[28] Zhang, S., Ding, J., Razanajatovo, R. M., Jiang, H., Zou, H., & Zhu, W. (2018). Interactive effects of polystyrene microplastics and roxithromycin on bioaccumulation and biochemical responses in the freshwater fish red tilapia (Oreochromis niloticus). Science of the Total Environment, 648, 1431-1439.

[29] Zhu, X., Qiang, L., Shi, H., & Cheng, J. (2019). Bioaccumulation of microplastics and its in vivo interactions with trace metals in edible oysters. Marine Pollution Bulletin, 149, 110648.