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Microplastics and Reproductive Dysfunction in Animals

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Abstract

Microplastics (MPs) are pervasive environmental contaminants increasingly detected across aquatic and terrestrial ecosystems. Growing experimental evidence indicates that MPs and nano plastics can translocate beyond the gastrointestinal tract and accumulate in reproductive tissues of animals. This review synthesizes current findings on microplastic-induced reproductive dysfunction, focusing on exposure pathways, mechanistic insights, and organ-level outcomes. Accumulated data from mammalian and aquatic models demonstrate that MPs induce oxidative stress, endocrine disruption, inflammation, and apoptosis, leading to impaired spermatogenesis, follicular depletion, altered hormone profiles, and reduced gamete quality. Developmental consequences include decreased fertilization success, embryonic abnormalities, and potential transgenerational effects. Although many studies rely on controlled laboratory exposures, the convergence of mechanistic and phenotypic evidence across species underscores the potential ecological and agricultural implications of chronic microplastic exposure. Standardized methodologies and environmentally relevant models are essential to clarify long-term reproductive risks in animals.

Keywords: Microplastics; Reproductive toxicity; Oxidative stress; Endocrine disruption; Transgenerational effects.

INTRODUCTION

Microplastics (MPs), defined as plastic particles ≤5 mm in size, originate either from the fragmentation of larger plastic debris or from primary microscopic manufacturing processes. Their persistence and widespread distribution across aquatic and terrestrial ecosystems have made them a significant emerging environmental contaminant (Dubey et al., 2022; Jewett et al., 2022). Due to extensive plastic production and insufficient waste management, MPs are now routinely detected in soil, freshwater, marine environments, and even atmospheric compartments. Animals are exposed primarily through ingestion, although inhalation and dermal contact may also contribute under specific environmental conditions (Bhuyan et al., 2022). The reproductive system is particularly vulnerable to environmental toxicants because it depends on tightly regulated endocrine signaling, redox balance, and cellular differentiation processes. Disruption at any stage, including gametogenesis, hormone synthesis, fertilization, or embryonic development, can compromise fertility, and long-term population sustainability. Increasing experimental evidence suggests that MPs and their smaller counterparts, nano plastics, are capable of translocating beyond the gastrointestinal tract and accumulating in reproductive tissues, thereby interfering with cellular and endocrine functions (Dubey et al., 2022; Yang et al., 2023). Microplastics may act as physical stressors and as carriers for adsorbed contaminants such as endocrine-disrupting chemicals and persistent organic pollutants

(Jewett et al., 2022). Mechanistic studies indicate that exposure can induce oxidative stress, inflammatory responses, apoptosis, and disruption of steroidogenic pathways, ultimately impairing reproductive integrity (Tang et al., 2023; Yang et al., 2023). This review synthesizes current evidence regarding microplastic-induced reproductive dysfunction in animals, focusing on exposure routes, mechanistic pathways, organ-level effects, developmental consequences, and existing knowledge gaps.

ENVIRONMENTAL SOURCES AND ANIMAL EXPOSURE

Microplastics enter animal systems not as isolated contaminants but as persistent and widely distributed environmental particles. Their distribution across ecosystems determines exposure intensity, duration, and biological relevance, creating continuous contact between animals and contaminated substrates.

Primary and Secondary Sources

Primary microplastics are intentionally manufactured microscopic particles used in industrial abrasives, cosmetics, and resin pellets. Secondary microplastics arise from the fragmentation of larger plastic materials through ultraviolet radiation, mechanical abrasion, and environmental weathering. Agricultural plastics, wastewater effluents, aquaculture materials, textile fibers, and degraded packaging waste are major contributors to environmental microplastic loading (Bhuyan et al., 2022; Dubey et al., 2022).

Aquatic Exposure

Aquatic ecosystems function as major sinks for microplastics, where particles accumulate in water columns, sediments, and benthic zones. Fish, mollusks, crustaceans, and zooplankton ingest microplastics either directly from surrounding water or indirectly through trophic transfer. Experimental evidence suggests that ingested particles may translocate from the gastrointestinal tract into systemic circulation and deposit in organs such as the liver and gonads (Subaramaniyam et al., 2023; Yang et al., 2023). In certain fish models, microplastics have been detected within ovarian and testicular tissues, indicating their capacity to cross biological barriers and potentially interfere directly with gametogenic processes (Yang et al., 2023).

Terrestrial Exposure

In terrestrial systems, soil contamination occurs through the application of sewage sludge, plastic mulching practices, irrigation with contaminated water, and atmospheric deposition. Grazing livestock and terrestrial wildlife may ingest microplastics through contaminated feed, forage, soil particles, and drinking water. Available reviews suggest that livestock species may chronically ingest low levels of microplastics, raising concerns regarding potential long-term subclinical reproductive impacts (Urli et al., 2023).

Feed and Water Contamination

Animal feed ingredients, particularly fishmeal and plant-based concentrates cultivated in contaminated soils, may contain measurable microplastic burdens. Drinking water has also been identified as an exposure pathway in both laboratory and farm environments (Bhuyan et al., 2022; Urli et al., 2023). Experimental rodent studies commonly model exposure through oral administration or contaminated drinking water to simulate ingestion-based pathways (Dubey et al., 2022; Tang et al., 2023).

Particle Characteristics Influencing Exposure

Particle size, morphology, and surface properties significantly influence biological interactions. Smaller particles, particularly those within the nanoplastic range, exhibit greater potential for epithelial penetration and systemic distribution. Fiber-shaped particles may demonstrate prolonged retention within gastrointestinal tissues compared to spherical particles. Surface chemistry and charge affect interactions with biological membranes and determine the adsorption capacity for heavy metals and endocrine-disrupting chemicals (Dubey et al., 2022; Jewett et al., 2022).

Internal Translocation and Gonadal Access

Experimental studies indicate that ingested microplastics may cross intestinal barriers and distribute to peripheral tissues,

including reproductive organs (Tang et al., 2023; Yang et al., 2023). In aquatic species, evidence supports movement from the gastrointestinal tract into systemic circulation and eventual deposition in gonadal tissues (Subaramaniyam et al., 2023). The capacity of smaller particles to bypass protective biological barriers, including the blood–testis and blood–ovary barriers, underlies their potential to disrupt reproductive physiology.

Maternal Transfer and Developmental Exposure

Nano plastics appear to have a higher probability of translocating across biological barriers compared to larger microplastic particles. Emerging evidence suggests the possibility of maternal–fetal transfer in mammalian models, raising concerns regarding exposure during critical windows of organogenesis and germ cell differentiation (Dubey et al., 2022; Yang et al., 2023). In summary, animal exposure to microplastics is multifaceted, chronic, and influenced by particle physicochemical characteristics. Understanding how these particles distribute within the body provides the foundation for examining the molecular and cellular mechanisms that drive reproductive dysfunction.

MECHANISMS OF REPRODUCTIVE TOXICITY

Microplastics exert reproductive toxicity not necessarily through intrinsic chemical reactivity but through their physical presence, surface properties, additive release, and capacity to transport co-contaminants. Reproductive tissues are particularly sensitive because successful gametogenesis and hormonal regulation depend on tightly controlled redox balance, endocrine signaling, and cellular homeostasis (Fig. 1)

Oxidative Stress and Mitochondrial Dysfunction

One of the most consistently reported mechanisms following microplastic exposure is the overproduction of reactive oxygen species (ROS) in both rodent and aquatic models (Dubey et al., 2022; Tang et al., 2023). Excess ROS damages sperm membranes, disrupts mitochondrial DNA, and reduces ATP synthesis required for sperm motility. In females, oxidative stress accelerates follicular atresia and impairs oocyte maturation. Suppression of antioxidant enzymes and increased lipid peroxidation have been observed in exposed animals, highlighting redox imbalance as a central pathway of toxicity (Dubey et al., 2022; Tang et al., 2023).

Endocrine Disruption and Steroidogenesis Interference

Microplastics can adsorb endocrine-disrupting chemicals such as bisphenols and phthalates, functioning as carriers of hormonally active compounds (Jewett et al., 2022). Experimental studies report altered testosterone, estradiol, and gonadotropin levels following exposure, indicating disruption of the hypothalamic–pituitary–gonadal axis (Tang et al., 2023). In fish, altered expression of steroidogenic enzymes has also been documented (Subaramaniyam et al., 2023). Such hormonal disturbances directly impair spermatogenesis, folliculogenesis, and reproductive cyclicity.

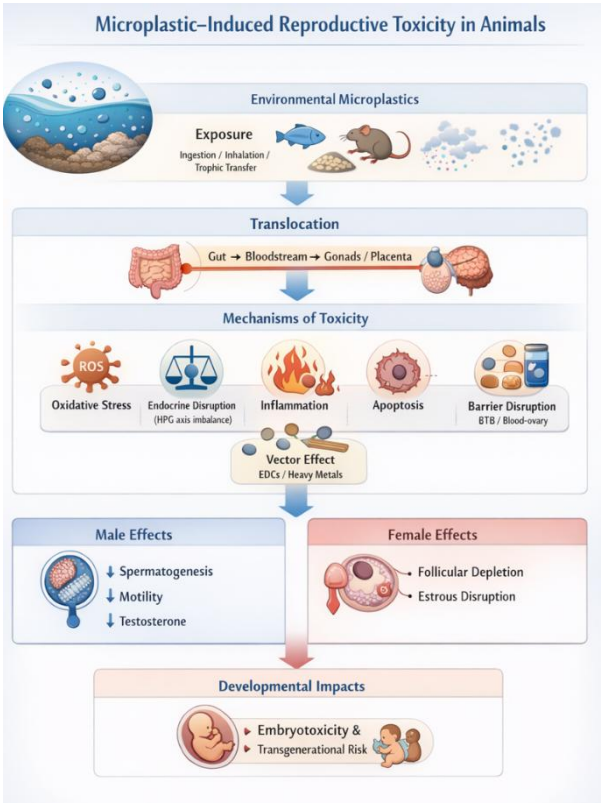

Fig. 1. Integrated Mechanistic Pathway of Microplastic-Induced Reproductive Toxicity Disruption of Biological Barriers

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Inflammation and Cellular Apoptosis

Chronic exposure to microplastics has been associated with inflammatory infiltration and increased expression of pro-inflammatory cytokines in reproductive tissues. Activation of apoptotic pathways leads to programmed cell death of germ cells, reducing sperm output and ovarian reserve (Tang et al., 2023; Yang et al., 2023). These structural and molecular alterations compromise overall reproductive capacity.

Smaller particles, particularly nano plastics, demonstrate greater ability to cross epithelial and vascular barriers (Dubey et al., 2022). Deposition of particles within gonadal tissues suggests potential compromise of protective structures such as the blood–testis and blood–ovary barriers, exposing germ cells to systemic stressors and inflammatory mediators.

Epigenetic and Transgenerational Effects

Emerging evidence indicates that microplastic exposure may alter gene expression and epigenetic regulation in reproductive tissues (Tang et al., 2023; Yang et al., 2023). Although multigenerational data remain limited, such modifications raise concerns regarding potential inheritance of reproductive dysfunction across generations.

Vector Effect and Synergistic Toxicity

Microplastics can bind heavy metals and hydrophobic pollutants, potentially amplifying toxicity through combined exposure (Jewett et al., 2022; Subaramaniyam et al., 2023). These interactions may intensify oxidative stress and endocrine disruption beyond the effects of particles alone. Collectively, these interconnected mechanisms oxidative imbalance, hormonal dysregulation, apoptosis, barrier disruption, and contaminant vectoring converge to impair spermatogenesis, folliculogenesis, steroidogenesis, and embryonic viability across animal species.

EFFECTS ON MALE REPRODUCTIVE FUNCTION

The male reproductive system has consistently emerged as a sensitive target of microplastic exposure in experimental animal models. Spermatogenesis is a highly coordinated and energy-dependent process, and disruption at structural, hormonal, or molecular levels can substantially impair fertility. Evidence from rodent and aquatic studies indicates that microplastics interfere with multiple components of male reproductive physiology (Fig. 2).

Testicular Histopathology

Chronic exposure to microplastics has been associated with structural alterations in testicular tissue. Experimental studies describe degeneration of seminiferous tubules, disorganization of the germinal epithelium, reduced tubular diameter, and thinning of spermatogenic layers following prolonged exposure (Tang et al., 2023). In some models, interstitial changes and Leydig cell alterations suggest involvement of endocrine pathways in addition to direct tissue stress. In fish, microplastic exposure has been linked to reduced spermatocyte density and increased apoptotic activity within gonadal tissue (Yang et al., 2023; Subaramaniyam et al., 2023). Such histological damage directly compromises sperm production.

Spermatogenesis Impairment

Decreased sperm count and concentration represent some of the most consistent findings in rodent exposure studies (Tang et al., 2023). Oxidative stress–induced DNA damage and activation of apoptotic pathways interfere with the progression of germ cells from spermatogonia to mature spermatozoa. In aquatic species, microplastic exposure has also been associated with reduced fertilization success and altered sperm morphology (Yang et al., 2023). Because spermatogenesis requires genomic integrity and sustained metabolic activity, oxidative and inflammatory stress significantly reduce its efficiency.

Sperm Quality Parameters

Microplastic exposure has been linked to reduced sperm motility, often attributed to mitochondrial dysfunction and impaired ATP production (Tang et al., 2023). Increased morphological abnormalities and elevated

DNA fragmentation indices suggest compromised chromatin integrity. Lipid peroxidation of sperm plasma membranes further reduces membrane fluidity and fertilization capacity (Dubey et al., 2022). These alterations collectively diminish sperm functionality even when sperm numbers are not profoundly reduced.

Hormonal Alterations

Altered reproductive hormone profiles have been reported following microplastic exposure. Decreased testosterone levels and disturbances in luteinizing and follicle-stimulating hormones indicate disruption of the hypothalamic–pituitary–gonadal axis (Tang et al., 2023). In fish, altered expression of genes involved in androgen synthesis has also been observed (Subaramaniyam et al., 2023). Hormonal imbalance not only impairs spermatogenesis but may also exacerbate structural testicular damage.

Barrier Integrity and Immunological Effects

Emerging evidence suggests that microplastics may compromise the integrity of the blood–testis barrier, exposing developing germ cells to systemic inflammatory mediators (Tang et al., 2023). Elevated oxidative stress markers and inflammatory responses within testicular tissue further contribute to reduced spermatogenic efficiency. The loss of immune privilege within the testicular environment may amplify germ cell apoptosis and long-term fertility impairment.

Dose and Particle Dependency

Particle size and exposure concentration appear to influence the severity of reproductive effects. Smaller particles, particularly nanoplastics, demonstrate greater systemic distribution and potential gonadal penetration (Dubey et al., 2022). Higher exposure doses generally correlate with more pronounced alterations in sperm parameters and hormone levels, although the effects of environmentally realistic chronic exposure require further investigation.

Integrative Perspective

Across rodent and aquatic models, male reproductive dysfunction associated with microplastic exposure includes structural testicular damage, reduced sperm quantity and quality, hormonal disruption, and oxidative and inflammatory stress. Although much of the evidence arises from controlled laboratory settings, the convergence of mechanistic and phenotypic findings across species strengthens the biological plausibility of microplastic-induced male infertility in animals.

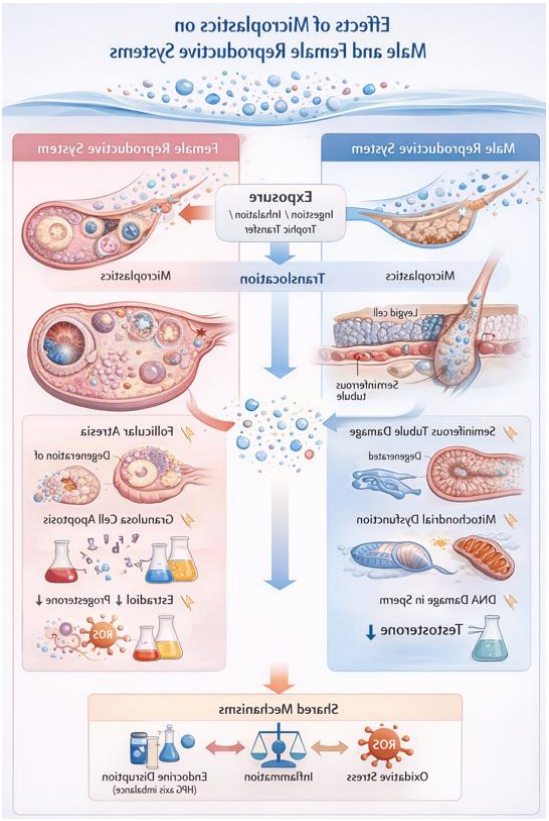

Fig. 2. Comparative Impact on Male vs Female Reproductive Systems

EFFECTS ON FEMALE REPRODUCTIVE FUNCTION

The female reproductive system is particularly sensitive to environmental stress because it relies on a finite ovarian reserve and tightly regulated hormonal cyclicity. In most mammals, ovarian follicles are non-renewable; therefore, damage to follicular populations represents a permanent reduction in reproductive potential. Experimental evidence indicates that microplastics interfere with multiple aspects of female reproductive physiology across animal models (Fig. 2).

Ovarian Histopathology and Follicular Dynamics

Rodent studies have reported reduced numbers of primordial and developing follicles

following chronic microplastic exposure, along with increased follicular atresia associated with oxidative stress and granulosa cell apoptosis (Tang et al., 2023). Structural alterations in ovarian tissue, including stromal disorganization and inflammatory infiltration, further indicate tissue-level damage. In aquatic species, exposure has been associated with oocyte degeneration and impaired vitellogenesis, suggesting disruption of normal gonadal maturation (Yang et al., 2023; Subaramaniyam et al., 2023). Such changes may reduce ovarian reserve and shorten reproductive lifespan.

Oxidative Stress in Ovarian Tissue

Elevated reactive oxygen species and increased lipid peroxidation have been documented in ovarian tissues following exposure (Tang et al., 2023). Suppression of antioxidant defenses disrupts the redox environment required for proper oocyte maturation. Because oocytes depend on mitochondrial integrity for energy production and meiotic progression, oxidative damage may impair cytoplasmic maturation, spindle formation, and chromosomal stability, thereby reducing fertilization competence.

Hormonal Imbalance and Steroidogenesis

Alterations in estradiol and progesterone levels have been reported in exposed animals, indicating disruption of ovarian steroidogenesis (Tang et al., 2023). In fish, changes in the expression of genes involved in estrogen synthesis have also been observed (Subaramaniyam et al., 2023). Such endocrine disturbances may impair ovulation, disrupt estrous cyclicity, and reduce overall reproductive efficiency by interfering with hypothalamic–pituitary–gonadal axis regulation.

Estrous Cycle and Reproductive Cyclicity

Microplastic exposure has been associated with irregular or prolonged estrous phases in experimental models (Tang et al., 2023). Disruption of cyclic hormonal signaling may decrease mating success and fecundity. Even modest alterations in cycle length or ovulatory timing can significantly affect conception probability over time.

Oocyte Quality and Developmental Competence

Evidence suggests that microplastic exposure may impair oocyte maturation and meiotic progression, accompanied by increased oxidative DNA damage in ovarian tissue (Yang et al., 2023). In aquatic species, reduced fertilization rates have been partially attributed to compromised oocyte quality (Subaramaniyam et al., 2023). Because gamete quality determines embryo viability, such alterations may propagate into early developmental stages.

Placental and Maternal–Fetal Considerations

Emerging data suggest that smaller plastic particles may cross biological barriers, raising concerns about potential maternal–fetal transfer in mammals (Dubey et al., 2022). Inflammatory responses within reproductive tissues may also affect uterine receptivity and implantation success. Although evidence remains limited, these findings suggest possible developmental exposure during gestation.

Integrative Perspective

Across mammalian and aquatic species, female reproductive effects associated with microplastic exposure include follicular depletion, increased ovarian apoptosis, hormonal dysregulation, impaired oocyte competence, and potential maternal–fetal exposure. Because ovarian follicles are finite, cumulative damage may have lasting consequences for reproductive lifespan. The mechanistic pattern observed in females parallels that in males, with oxidative stress and endocrine disruption emerging as central drivers of reproductive dysfunction.

EMBRYONIC DEVELOPMENT AND TRANSGENERATIONAL EFFECTS

Reproductive toxicity extends beyond gamete impairment to embryo viability and offspring health. In aquatic species, microplastic exposure has been associated with reduced fertilization rates, decreased hatchability, developmental delays, and increased embryonic mortality (Subaramaniyam et al., 2023; Yang et al., 2023). These outcomes are often linked to compromised gamete quality and elevated

oxidative stress in early developmental stages (Tang et al., 2023). Because embryonic tissues undergo rapid cell division and differentiation, even subtle redox disturbances may alter normal organogenesis. Smaller particles, particularly nanoplastics, demonstrate greater potential to cross biological barriers, raising concerns regarding placental transfer and in utero exposure in mammals (Dubey et al., 2022). Emerging evidence also suggests that microplastic exposure may influence gene expression and epigenetic regulation within reproductive pathways (Tang et al., 2023; Yang et al., 2023). Although multigenerational data remain limited, preliminary findings indicate possible alterations in reproductive parameters among offspring of exposed parents (Dubey et al., 2022). These observations suggest that microplastic-induced reproductive effects may extend beyond a single generation.

KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite increasing evidence of reproductive impairment, several limitations constrain current understanding. Many experimental studies employ exposure concentrations and particle types that may not accurately reflect environmental conditions (Jewett et al., 2022; Dubey et al., 2022). Standardized characterization of particle size, morphology, and chemical composition is needed to improve comparability across studies. Moreover, most mechanistic data derive from rodent and aquatic models, while livestock and wildlife species remain insufficiently investigated (Urli et al., 2023). Comprehensive multigenerational studies and environmentally relevant chronic exposure models are essential to clarify long-term reproductive risks. Further integration of molecular and systems-level approaches may strengthen mechanistic interpretation and improve risk assessment.

CONCLUSION

Microplastics are emerging environmental contaminants with demonstrated capacity to disrupt reproductive function in animals. Evidence across mammalian and aquatic models indicates impairment of spermatogenesis, folliculogenesis, hormonal regulation, and embryonic development, primarily mediated through oxidative stress, inflammation, and endocrine imbalance. Although much of the current evidence arises from controlled laboratory conditions, the consistency of mechanistic findings across species highlights potential ecological and agricultural implications. Continued research using realistic exposure models and comprehensive mechanistic analysis is necessary to define long-term reproductive risk and inform mitigation strategies.

STATEMENTS AND DECLARATIONS

Artificial Intelligence (AI) Statement

Artificial intelligence (AI) tools were used to assist in language refinement, structural organization, and formatting of the manuscript. All scientific content, interpretation of data, critical analysis, and final editorial decisions were performed by the authors. The authors take full responsibility for the accuracy, integrity, and originality of the work.

Conflict of Interest

The authors declare no conflict of interest.

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