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Review Article

MICROPLASTIC INVASION: A THREAT TO MALE INFERTILITY -A REVIEW

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ABSTRACT

Microplastics, defined as plastic particles less than 5 mm in diameter, have become an emerging global pollutant with profound implications for human health. Recent findings indicate their accumulation in human tissues, including the male reproductive system, raising serious concerns about their impact on semen quality and overall fertility. This review synthesizes current literature on microplastic exposure, its biological pathways, and its potential role in declining male reproductive health. Mechanisms of toxicity, including endocrine disruption, oxidative stress, inflammation, and DNA damage, are explored alongside epidemiological and experimental data. With global fertility rates on the decline, understanding the reproductive consequences of microplastic pollution is urgent and essential.

Keywords: Microplastics, Accumulation, Semen quality, Endocrine disruption, Fertility rate.

INTRODUCTION

The rapid industrialization and overuse of plastics have led to the proliferation of microplastics in the environment. Once released, these particles can persist for hundreds of years, breaking down into smaller fragments that infiltrate ecosystems, food chains, and eventually, the human body. As awareness grows regarding their potential health hazards, a new frontier of concern has emerged: reproductive toxicity. Semen quality among men globally has shown a significant decline over recent decades, prompting researchers to explore environmental contributors, including microplasticsas possible culprits. Microplastics, ubiquitous in the environment, have been found in human tissues, including the placenta and fetal meconium, and are linked to potential reproductive toxicity. It is essential to evaluate the impact of microplastics on both human and animal reproductive health, while also considering the broader environmental factors that contribute to their widespread presence. Microplastics (MPs) are increasingly recognized as a potential threat to male reproductive health, with studies indicating various mechanisms of toxicity. Evidences reveals that MPs can induce oxidative stress, disrupt spermatogenesis, impair mitochondrial function, and damage testicular structures. These effects can lead to reduced sperm quality, including decreased sperm concentration, motility, and morphology, ultimately impacting male fertility.

Microplastics: Sources and Human Exposure

Microplastics originate from primary sources (e.g., microbeads in cosmetics, industrial abrasives) and secondary sources (degradation of larger plastic items). Common environmental reservoirs include drinking water, seafood, fruits and vegetables and even table salt. Humans are exposed to microplastics through ingestion, inhalation, and dermal absorption. Once inside the body, they may translocate across biological barriers such as the gut epithelium and the blood-testis barrier. Studies have confirmed the presence of microplastics in human stool, blood, placenta, and more recently in seminal fluid. Their small size and surface properties facilitate absorption and

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accumulation in various tissues. The blood-testis barrier is critical for maintaining immune privilege in the testes. Microplastics may disrupt this barrier, exposing spermatogenic cells to immune attacks and toxins.

Microplastics and male fertility

Recent meta-analyses reveal significant global declines in sperm concentration, motility, and morphology. While lifestyle and genetic factors play a role, environmental contaminants are increasingly implicated. Experimental studies support the detrimental effects of microplastics on male fertility. Mice exposed to polystyrene microplastics show reduced sperm count, motility, and testosterone levels. Zebra fish exposed to MP leads to disrupted spermatogenesis and oxidative damage. Preliminary findings suggest similar impacts on human sperm and Leydig cells, though more evidence is needed. Studies have detected MPs/NPs in human testes and semen. Jin *et al*., 2021 found that in mice, polystyrene MPs led to decreased sperm count and increased abnormal sperm morphology.

Microplastics often carry endocrine-disrupting chemicals (EDCs) like bisphenol A (BPA) and phthalates, which interfere with hormonal signalling. Amereh *et al*., 2020found that MPs act as endocrine disruptors, altering testosterone levels and Leydig cell function. Radke *et al*., 2018reported Phthalates (plastic additives) are associated with lower semen quality in epidemiological studies. Microplastics generate reactive oxygen species (ROS), which can damage sperm DNA and lipid membranes. Hou *et al*., 2021 reported that MP exposure induces reactive oxygen species (ROS), causing sperm DNA fragmentation and reduced motility. Chronic exposure may provoke systemic inflammation, negatively affecting testicular function. Xie *et al*., 2022 conducted animal experiments and found that polystyrene nanoparticles can cross the blood-testis barrier, leading to testicular accumulation. Accumulation of MP impact spermatogenesis process. Wei *et al*., (2022) and Pivonello *et al*. (2023) reported MPs trigger inflammatory responses in testes which results in impairing spermatogenesis through oxidative stress and endocrine disruption.

Oxidative Stress, Mitochondrial Dysfunction, and Sperm Bioenergetics

Numerous studies, including Wang *et al*. (2023) and Silva *et al*. (2023), show that microplastics generate excessive reactive oxygen species (ROS), leading to lipid peroxidation, protein carbonylation, and mitochondrial collapse in spermatozoa. Since sperm are uniquely vulnerable with small cytoplasmic volume, high membrane PUFA content, and heavy dependence on mitochondrial ATP ROS rapidly impairs motility, membrane integrity, and fertilizing capacity. Mitochondrial dysfunction is repeatedly emphasized: membrane potential loss, cristae damage, disrupted electron transport chain activity, and altered mitochondrial dynamics (fission/fusion). In 2024, Chen *et al*. studied about the mtDNA hypermethylation, linking oxidative stress to mitochondrial epigenome alterations. This bioenergetic failure aligns with observed functional outcomes: reduced motility, compromised hyperactivation, impaired capacitation, and lower IVF success rates. By 2025, in vitro human sperm studies (Chen *et al*., 2025) confirmed ROS-driven DNA damage and motility suppression, establishing oxidative stress as one of the mechanism.

Endocrine Disruption and Steroidogenic Impairment

Foundational evidence originates from Manikkam *et al*. (2013), who reported transgenerational endocrine disruption from BPA and phthalates chemicals often embedded in plastic polymers. Later reviews (Park & Lee, 2023; Hernández-López *et al*., 2024) confirm that microplastics can carry and release such endocrine-disrupting chemicals (EDCs), disturbing the hypothalamic-pituitary-gonadal (HPG) axis. Studies across 2023-2025 further implicate impaired testosterone synthesis, downregulation of steroidogenic enzymes (StAR, CYP11A1, 3β-HSD), Leydig cell dysfunction (Fernandez *et al*., 2023), and disrupted estrogen/androgen receptor signaling. Research work by Pivonello *et al*. (2023) on BPA provided detailed pathways, including aromatase modulation and GPER1 activation mechanisms echoed by Rudolph & Ahern (2023). These endocrine changes explain phenotypes such as reduced testicular weight, impaired spermatogenesis, and altered sexual behavior (Cheng & Li, 2023). Endocrine disruption thus emerges as a core mechanistic pillar, synergizing with oxidative stress and inflammation to shape male reproductive decline.

Inflammation, Immune Dysregulation, and the Blood-Testis Barrier (BTB)

Kostova *et al*. (2023) and Fischer & Jensen (2023) identify NLRP3 inflammasome activation as a central inflammatory pathway, leading to IL-1β release, pyroptosis, and testicular tissue damage. Chronic inflammation undermines Sertoli cell function, compromises nutritional support for developing germ cells, and accelerates germ-cell apoptosis. Another critical area is disruption of the blood-testis barrier (BTB), reported by Gao *et al*. (2023), Huang & Wei (2023), and Harding & Baxter (2023). Microplastics especially <1 μm fragments disturb tight junctions through ROS, cytokines, and cytoskeletal (vimentin) disorganization, leading to increased BTB permeability. This breach exposes sensitive germ cells to immune surveillance and circulating toxins, triggering autoimmune orchitis-like responses. The immune-testis interaction also extends to the peritubular myoid cells (Chen & Wong, 2023) and testicular lymphatic signaling (Powell & Mercer, 2023). Collectively, inflammation and BTB disruption form a mechanistic cluster that links exposure to structural and immunological testicular breakdown.

Genomic Instability, Epigenetic Reprogramming, and Transgenerational Effects

Direct DNA damage especially sperm DNA fragmentation is repeatedly reported (Abdel-Khalek *et al*., 2023; Chen *et*

al., 2025). Sperm chromatin abnormalities, impaired protamine/histone replacement (Jansen & van der Heijden, 2023), telomere shortening (Wallace & Freeman, 2023), and defective repair pathways all contribute to reduced reproductive success and increased miscarriage risk. Epigenetically, studies in 2023-2025 reveal alterations in DNA methylation, histone marks, small RNAs, long non-coding RNAs, circRNAs (Zhao & Liu, 2024), and sperm m6A methylation (Wang *et al*., 2025). Chen *et al*. (2024) demonstrated mitochondrial DNA hypermethylation, while Adams *et al*. (2023) and Peterson & Lee (2023) described transgenerational epimutations across multiple germline generations. Manikkam *et al*. (2013) provided early proof that environmentally induced epimutations can be inherited even without continued exposure. These data reveal that microplastics can alter the heritable molecular architecture of sperm.

Spermatogenesis, Sertoli/Leydig Cell Toxicity & Testicular Microenvironment Damage

An *et al*. (2021), Wei *et al*. (2022), reported that degeneration of seminiferous tubules, loss of germ cells, vacuolization of Sertoli cells, and mitochondrial swelling in spermatocytes. Sertoli cells key for germ-cell support are specifically damaged, as shown by Bertoldo *et al*. (2023), which impairs nutrient transport, BTB function, and phagocytosis of apoptotic cells. Leydig cell dysfunction appears in Fernandez *et al*. (2023) and Ricci *et al*. (2024), linking microplastics to androgen deficiency. Sperm maturation processes, including capacitation (Rossi & Bianchi, 2023), acrosome reaction (Cheng & Lombardo, 2024), ion channel regulation (Mazzilli & Rossi, 2023), and glycocalyx integrity (Ito & Tanaka, 2024), are all disrupted. This cellular toxicity results in lower sperm count, impaired morphology, decreased motility, and reduced fertility.

Sperm Functional Biology Motility, Acrosome Reaction, Membrane Lipidomics

Lopez & Garcia (2023) studied about changes in sperm lipidomics especially reductions in DHA-rich phospholipids making sperm membranes more rigid and prone to oxidative damage. Ion channel dysfunction (CatSper, Slo3) reported by Mazzilli & Rossi (2023) leads to defective calcium signaling, reduced hyperactivation, and failed zona pellucida penetration. Rossi & Bianchi (2023) found that capacitation is inhibited, likely via impaired tyrosine phosphorylation pathways. Acrosin activity reduction (Cheng & Lombardo, 2024) further compromises oocyte penetration. The combined alterations in motility, acrosome function, membrane composition, and enzymatic dynamics represent crucial pathways through which microplastics impair fertilization ability even when sperm count remains normal.

Gastrointestinal-Reproductive Axis, Metabolic Disruption & Systemic Toxicity

Wang & Zhao (2023) and Qian & Zhang (2023) described how gut dysbiosis caused by microplastics leads to reduced short-chain fatty acids and increased pro-inflammatory metabolites, which indirectly disrupt the testicular environment forming a gut-testis axis. Several studies show obesity like metabolic changes (Ricci *et al*., 2024), altered glucose metabolism (Finkelstein & Greene, 2023), and hepatic stress feeding into impaired steroidogenesis. Tire wear particles (Knight *et al*., 2024) and environmental weathered microplastics (Turner *et al*., 2023; O'Connell *et al*., 2023) exhibit more severe systemic effects than pristine lab particles, due to adsorbed pollutants and degraded surfaces that enhance uptake. These findings emphasize that male reproductive toxicity is part of a broader pattern of systemic metabolic and inflammatory disruption.

Human Evidence Microplastics in Semen and Human Fertility Decline

Wu *et al*. (2023) and Montano *et al*. (2023) independently detected microplastics in human semen for the first time, finding correlations between MP load and decreased motility, reduced concentration, and altered morphology. Zhao *et al*. (2023) performed a landmark meta-analysis confirming widespread detection of microplastics in human semen globally. Liu *et al*. (2024) synthesized human and animal data, showing that exposure levels in humans fall within ranges known to cause damage in animal models. Moretti *et al*. (2025) discovered paternal-exposure influences on placental transfer, indicating male exposure can affect early embryonic development. Combined with WHO's (2023) report on global infertility trends, human biomonitoring data paint a concerning picture: microplastics are not merely environmental contaminants they are measurable within the male reproductive system and associated with clinically relevant dysfunction.

Nanoplastics, Particle Size/Charge, Surface Chemistry & Advanced Toxicology Mechanisms

Zhang *et al*. (2023) and Nielsen *et al*. (2023) show that smaller particles (especially nanoplastics <100 nm) cause exponential increases in cellular uptake, oxidative stress, and DNA damage. Charge modifications (positive > neutral > negative) influence adhesion to sperm membranes and Sertoli cells. Weathered particles, tire-wear particles, and those carrying adsorbed pollutants exhibit higher reactivity and greater biological penetration. Nanoplastics can also act as gene-delivery vectors (Davies & O'Shea, 2023) and influence ferroptosis (Wang *et al*., 2023), autophagy (Kimura *et al*., 2023), and AhR activation (Stevens *et al*., 2023; Krol & Michalska, 2023). Macrogenomic and metabolomic studies in 2025 (Zhang *et al*.) further reveal pathway-wide perturbations in testicular and epididymal tissues. These mechanistic insights reflect a shift from descriptive toxicology to molecular precision toxicology.

Combined Exposures, Heat Stress, Pollutants & Real-World Risk

Stevens *et al*. (2023), Santos & Costa (2023), and Wallace & Freeman (2023) illustrate how microplastics interact synergistically with other stressors temperature, chemical pollutants, pesticides, endocrine disruptors, and oxidative

metabolic load. The Global Commission on Pollution & Health (2025) underscored that heat stress dramatically enhances MP toxicity, an important real-world consideration given rising global temperature. Occupational studies (Moretti *et al*., 2023) show increased risks among workers in plastic manufacturing and waste processing. Microplastics rarely act alone; instead, they operate within complex environmental mixtures that amplify male reproductive damage.

Challenges and Research Gaps

Standardized Detection Methods: Techniques for isolating microplastics from biological fluids vary widely, making comparison difficult. Dose-Response Relationships: More studies are required to know threshold of microplastic exposure to cause reproductive harm. Long-Term Human Studies: Most of the current data available from short-term animal studies. Long-term human cohort studies are essential. Interaction with Other Pollutants: Microplastics often act as vectors for heavy metals and persistent organic pollutants, compounding their toxicity. More studies are required to know the compounding toxicity.

CONCLUSION

Microplastics represent a novel and potentially serious threat to male fertility. Current evidences especially from animal and in vitro models suggests a strong link between MP and sperm infertility. Current evidence suggests that microplastics contribute to male infertility via oxidative stress, hormonal disruption, and direct testicular damage. The global decline in semen quality and its potential link to microplastics urgently requires further investigation, as it is vital for safeguarding the health of future generations. However, most studies are preclinical (animal/cell-based), and human epidemiological data remain limited. Further research is needed to establish causation and safe exposure thresholds. Human reproductive health, particularly semen quality, is *"*suspected to be adversely impacted*"* by microplastic exposure, based on moderate to high-quality animal-to-human translational evidence. Reproductive toxicity should be a global public health priority, calling for standardized human biomonitoring and exposure regulation frameworks.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

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Not applicable

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AI TOOL DECLARATION

The authors declares that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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