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THE IMPACT OF MICROPLASTICS ON MALE FERTILITY

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ABSTRACT

Male infertility represents a growing global health concern, accounting for more than half of all infertility cases, while its environmental determinants remain insufficiently explored. Over recent decades, a marked decline in semen quality has coincided with the rapid expansion of plastic production and the widespread presence of microplastics and nanoplastics in the environment. This narrative review aims to synthesize current evidence on the impact of micro- and nanoplastic exposure on the male reproductive system, identify key mechanisms of reproductive toxicity, and highlight gaps requiring further investigation. A comprehensive literature search was conducted using PubMed, Scopus, Web of Science, and Google Scholar, focusing on experimental, epidemiological, and review studies addressing microplastics, male fertility, and reproductive toxicity. Available data indicate that microplastics can accumulate in testicular tissue and adversely affect spermatogenesis through multiple pathways, including disruption of the blood–testis barrier, oxidative stress, chronic inflammation, endocrine dysregulation, and structural damage to Sertoli and Leydig cells. These alterations are consistently associated with impaired sperm concentration, motility, morphology, and hormonal balance in experimental models. Although human data remain limited, emerging evidence suggests that lifelong, low-dose exposure may contribute to declining male reproductive potential. The review also discusses preventive strategies and emerging therapeutic approaches aimed at mitigating microplastic-induced reproductive damage. Collectively, current findings support microplastics as a relevant and potentially modifiable environmental risk factor for male infertility.

KEYWORDS

Microplastics, Male Fertility, Spermatogenesis, Environmental Exposure, Endocrine Disruption, Sperm Quality

CITATION

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Introduction

Infertility is one of the biggest health problems worldwide, affecting approximately 186 million people. More than half of all infertility cases are attributable to male factors, and although male infertility has a substantial impact on reproductive health, this issue remains underrecognized (Inhorn & Patrizio, 2015). Based on long-term epidemiological data and successive editions of the WHO Laboratory Manual for the Examination and Processing of Human Semen, a clear decline in male semen quality can be observed. Notably, the reference value for normal sperm concentration decreased from 60 × 10^6/ml in the 1980 edition to 15 × 10^6/ml in the 2010 edition, remaining at a comparable level in later revisions (Table 1) (World Health Organization, 1999, 2021).

Table. 1. Comparison between chosen variables between 4th (1999) and 6th (2021) edition of WHO manuals for the examination of human semen.

Reference values	4th edition (World Health Organization, 1999)	6th edition (World Health Organization, 2021)
Volume	≥ 2.0 ml	≥ 1.4 ml
Sperm concentration	≥ 20*10^6/ml	≥ 16*10^6/ml
Total sperm number	≥ 40*10^6/ejaculation	≥ 39*10^6/ejaculation
Total motility	≥ 50%	≥ 42%
Normal morphology	≥ 14%	≥ 4%
Vitality	≥ 75%	≥ 54%

Currently, the correspondence between the long-term decline in semen quality and the global expansion of plastics, particularly microplastics, has prompted investigation into microplastic exposure as a possible contributing factor. The effects of microplastics on the male reproductive system vary according to particle size. These materials can accumulate within testicular tissue, where they may disturb the organization of seminiferous structures, interfere with germ-cell development, and alter the normal functioning of somatic cells such as Sertoli and Leydig cells. Together, these effects can impair the process of spermatogenesis and ultimately compromise sperm quality (Marcelino et al., 2022; Volsa et al., 2025). Given the pervasive occurrence of plastic-derived particles in the environment, there is a clear need to better understand how exposure to MPs/NPs affects male fertility. In this review, we aim to synthesize current data on the effects of microplastics on the male reproductive system, outline key knowledge gaps that require further investigation, and discuss the biological pathways through which microplastics may exert their reproductive toxicity. In addition to summarizing toxicological mechanisms, our work also addresses potential strategies to reduce or counteract exposure-related effects.

Methodology

This narrative review brings together current evidence on the potential effects of microplastics and nanoplastics on male fertility. Relevant publications were identified through literature searches in PubMed, Scopus, Web of Science, and Google Scholar. Search queries combined keywords and Medical Subject Headings (MeSH) related to microplastics, nanoplastics, male fertility, spermatogenesis, sperm quality, testicular toxicity, endocrine disruption, oxidative stress, and reproductive toxicity. The review included original research articles, experimental studies conducted *in vivo* and *in vitro*, epidemiological studies, as well as relevant review papers, primarily published in English. Most of the analyzed literature originated from the last 15 years, although earlier studies were also considered when they provided important background on plastic chemistry, classification, or exposure pathways. No geographical limitations were applied during the

selection process. Studies were eligible for inclusion if they investigated the impact of microplastics or nanoplastics on the male reproductive system. Reported outcomes included alterations in sperm concentration, motility, and morphology, changes in hormone levels, testicular structure, oxidative stress, inflammatory responses, and overall fertility parameters. To provide broader context, publications addressing exposure routes, physicochemical characteristics of microplastics, and potential preventive or therapeutic approaches were also taken into account. Studies unrelated to male reproductive health or focused exclusively on female fertility were excluded. An initial screening of titles and abstracts was followed by full-text evaluation of selected articles. Data were collected qualitatively, with emphasis on study design, particle characteristics, exposure conditions, measured biological endpoints, and principal findings. Due to substantial variability in experimental models, exposure protocols, and outcome measures, a quantitative meta-analysis was not feasible. Instead, the available evidence was synthesized narratively to highlight recurring observations, proposed mechanisms of toxicity, and gaps in the existing literature. Overall, this review integrates data from environmental science, toxicology, and reproductive biology to clarify how microplastics may contribute to impaired male fertility and to outline possible strategies aimed at reducing or mitigating their adverse effects.

Definition, Classification and Physicochemical Characteristics of Microplastics

Plastics are substances created by polymerization of specific monomers into high molecular weight polymers, which can be shaped by flow into desired products. Eventually many of these products degrade into plastic particles that are smaller in size and pollute the environment. Depending on the size they can be divided into nanoplastics (NPs) smaller than 1 μm, microplastics (MPs) ranging in size between 1 μm and 5 mm, mesoplastics (between 5 and 25 mm) and macroplastics larger than 25 mm (Boctor et al., 2025; Hartmann et al., 2019).

Microplastics can vary among themselves by shape, namely fibers, beads, films, disks and irregular fragments. Researchers have found that some of them are more harmful than others, specifically fibers and beads being more damaging to living organisms, but the direct association between the shape and human health still remains unclear (Lamoree et al., 2025; Rani, 2024).

There are many polymers that are used in the production process of plastic products, with the exact mix of polymers and additives varying depending on the aimed physical and chemical properties, as well as their purpose. The additives may comprise thermal and photo- stabilizers, pigments, antioxidants, plasticizers, flame retardants as well as others, consequently contaminating synthetic goods well before entering the ecosystem with the MPs (Hahladakis et al., 2018). In addition to this the chemical composition of degrading MPs may differ under varying conditions such as temperature and UV light (Andrady, 2011). Moreover, MPs have been found to have an affinity to all types of contaminants from the surroundings, including heavy metals (Figure 1) (Rochman et al., 2014).

Sources and Environmental Origin of Microplastics

Depending on the source MPs can be either intentionally produced and put into products, which are called ‘primary MPs’, or be a product of decomposition of improperly disposed larger plastics by the environmental factors (e.g. photodegradation, thermooxidative degradation, hydrolysis) and be called ‘secondary MPs’ (Andrady, 2011; Browne et al., 2011). Primary MPs are most commonly found in domestic and industrial cleaning products, beauty and personal care products, fertilizers, clothing, construction materials and medicines, while secondary MPs derive mostly from municipal debris (An et al., 2020).

Routes of Human Exposure to Microplastics

The human body can be exposed to MPs through various ways. The most common is inhalation of the molecules suspended in the air (Doroftei et al., 2025). The concentration of the MPs indoors has been proven to be higher than outside, especially in the settled dust (0,3-1,5 particles m3- vs. 0,4-56,5 particles m3), but the overall daily dose varies between studies due to the differences between methodologies and other factors (Vasse & Melgert, 2024). Some synthetic materials have been observed to release MPs to the air through friction, especially all types of vehicle tires which are believed to generate a staggering 6 000 000 tons of waste a year worldwide (Rani, 2024). Nevertheless, inhaled particles are believed to cause damage only in the respiratory tract with little evidence of translocation of the particles throughout the body.

MPs can enter a human body through a gastrointestinal tract with contaminated water and food. Recent studies have shown a presence of these contaminants in foodstuffs such as seafood, eggs, meat and milk, as well as drinkable water (in particular packaged in plastic bottles), table salt, teabags and powdered baby formulas (in the latter most likely coming from both metal and cardboard packs) (Boctor et al., 2025; Prata et al., 2020). Among fruits, pears (*Pyrus communis*) and apples (*Malus domestica*) have been found to contain

the highest concentration of MPs in them, with a shocking estimation of daily intake of 462 000 particles for adults and 1 410 000 particles per kilogram for children (sic!) (Boctor et al., 2025). On the contrary another study estimated an average ingestion of 39 000 – 52 000 particles of MPs a year per person (Prata et al., 2020). Despite this fact some researchers consider settling of dust on plates and plastic containers a more considerable source of MPs than the concentration in the food itself (Catarino et al., 2018; Rist et al., 2018). After consumption the MP particles are presumed to be either internalized by the gut M-cells or by the enterocytes via endocytosis (Prata et al., 2020).

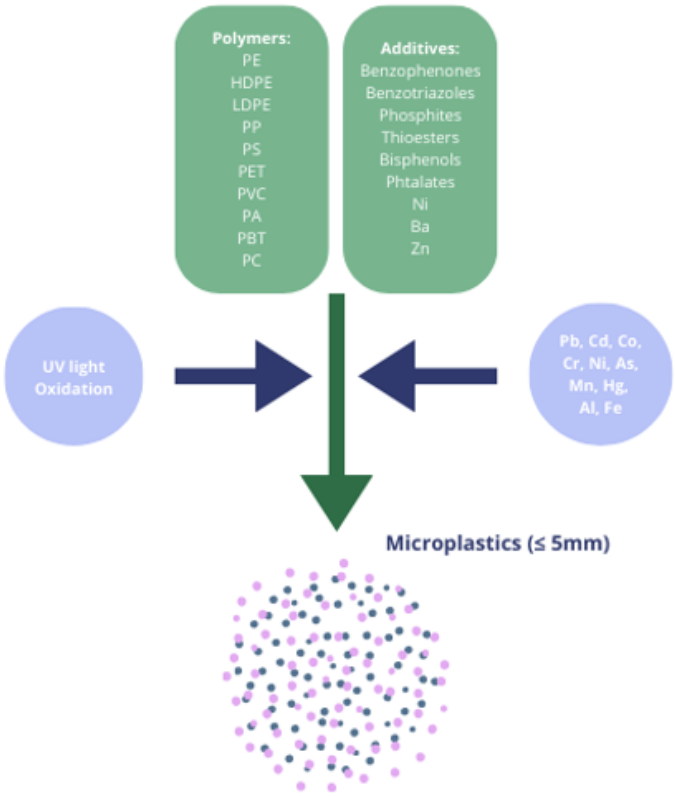

Fig. 1. *From plastic to microplastic. PE- Polyethylene, HDPE- High density polyethylene, LDPE- Low density polyethylene, PP- Polypropylene, PS- Polystyrene, PET- Polyethylene terephthalate, PVC- Polyvinylchloride, PA- Polyamide, PBT- Polybutylene terephthalate, PC- Polycarbonate, Ni- Nickel, Ba- Barium, Zn- Zinc, Pb- Lead, Cd- Cadmium, Co- Cobalt, Cr- Chromium, As- Arsenic, Mn- Manganese, Hg- Mercury, Al- Aluminum, Fe- Iron*

MPs have also been found in nasal irrigators, intravenous infusion sets and menstruation products namely tampons and pads (Doroftei et al., 2025). Dermal exposure is also taken into consideration because of monomers and additives (bisphenol A and phthalates) but it isn't regarded as a significant route of MPs absorption as others (Figure 2) (Prata et al., 2020).

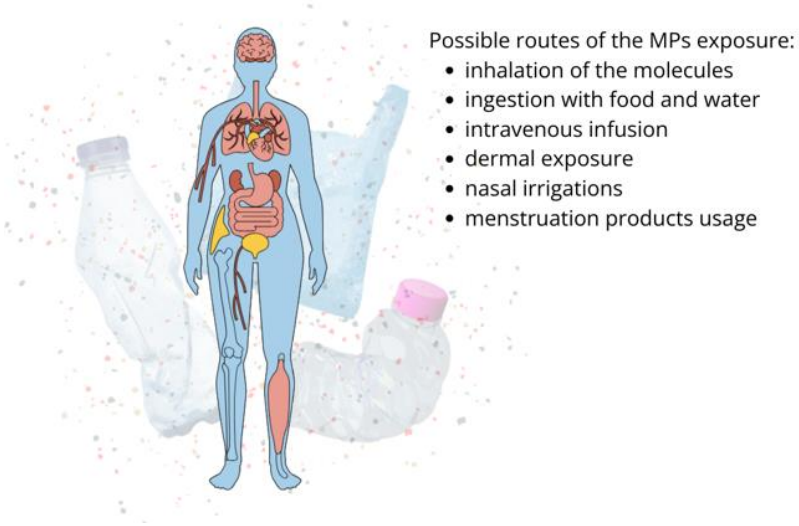

Fig. 2. *Possible routes of the MPs exposure.*

Mechanisms of Gonadal and Sperm Toxicity

Although there are numerous well-recognized factors why semen parameters are deteriorating, such as chronic illnesses, obesity, unbalanced diet and exposure to unfavorable environmental conditions, microplastics have emerged as a crucial issue when debating male reproductive health (Volsa et al., 2025). MPs are being thoroughly researched in order to understand their impact on male fertility. Due to ethical constraints, the majority of the studies are conducted on rodents, especially mice, which poses a challenge for the direct extrapolation of the findings to human physiological conditions (Jeon et al., 2024). Moreover, there are many different polymers identified that may vary in their toxic influence as well as bio-cumulative effects, and further research is necessary to fully establish their role in pathogenesis of male infertility. While the exact mechanisms underlying microplastic-induced disruption of male gonadal function remain incompletely understood, MPs have been linked to reduced semen quality, including lower sperm concentration and motility (Volsa et al., 2025; Zhang et al., 2024).

In vivo studies showed that MPs impair spermatogenesis and gonadal development in both prenatal and postnatal periods. They disturb these processes through multiple ways, including blood-testis barrier (BTB) disruption, chronic inflammatory state, oxidative stress, genetic and structural damage to Sertoli and Leydig cells, hormonal imbalance and endocrine disruption (Figure 3) (Zenclussen et al., 2025; Zhao et al., 2023).

Blood-testis barrier disruption

BTB, established by Sertoli cells, is necessary in order to maintain immune tolerance and effective spermatogenesis. Some MPs have been proved to alter this barrier by degrading tight junction proteins such as occludin, claudin, and ZO-2. These alterations disturb the germ cell microenvironment, leading to decreased testosterone levels and reduced sperm production (Zenclussen et al., 2025; Zhao et al., 2023).

Inflammation and oxidative stress pathways

Long-term MP exposure induces a chronic inflammatory response in testis mediated by dysfunctional cytokines. Studies show that these polymers can significantly influence levels of several cytokines such as IL-6, IL-10, TNF-α, TGF-β1, and TGF-β3 (Jeon et al., 2024; Zhao et al., 2023). Activating these immune responses can enhance the activity of oxidative stress pathways and as a consequence, cause mitochondrial damage. It particularly disrupts the electron transport chain, resulting in impaired ATP production and a decrease in mitochondrial membrane potential (Volsa et al., 2025). Additionally, longer exposures were connected with notable increases in inflammatory and oxidative stress responses, whereas no significant changes were observed at higher doses, which may indicate potential threshold effect (Jeon et al., 2024).

Structural damage

According to some studies, even short-term exposure to microplastics has been shown to affect cellular physiological processes, cell morphology, and proliferative capacity (Jeon et al., 2024). Histopathological and ultrastructural studies have shown disorganization of the seminiferous epithelium, cellular vacuolization, and widening of interstitial spaces. MPs induce mitochondrial swelling, disrupted cristae, plasma membrane damage, and nuclear abnormalities in spermatogenic cells, reflecting impaired cellular integrity and energy

metabolism. These changes are often accompanied by disrupted cell cycle progression and reduced proliferative capacity, ultimately compromising sperm production and quality (Zhao et al., 2023).

Hormonal imbalance

Experimental studies have further demonstrated that MP exposure disrupts the hypothalamic–pituitary–testicular (HPT) axis, leading to altered gonadotropin signaling and impaired steroidogenesis. In murine models, prenatal and postnatal exposure to polystyrene microplastics resulted in decreased testosterone and inhibin B levels, increased follicle-stimulating hormone (FSH) concentrations, and a reduced testosterone-to-luteinizing hormone (LH) ratio, despite unchanged absolute LH levels. These hormonal alterations were accompanied by delayed puberty onset. MP exposure was associated with downregulation of androgen receptor activity, disruption of hormone-mediated signaling pathways, inhibition of cell-cycle progression, and suppression of the Hippo signaling pathway, which plays a critical role in testicular development, Sertoli cell proliferation, and regulation of gonadotropin secretion (Zhao et al., 2023).

Endocrine disruptors

Micro- and nanoplastics can act as vesicles for endocrine-disrupting chemicals (EDCs). Common EDCs include bisphenol A, phthalates, and polybrominated diphenyl ethers. Due to their physicochemical properties, they can mimic or interfere with natural hormones, influencing endocrine signaling and reproductive functions. MPs can act as 'molecular sponges', enabling these chemicals to accumulate in tissues and amplify reproductive toxicity (Volsa et al., 2025; Zenclussen et al., 2025).

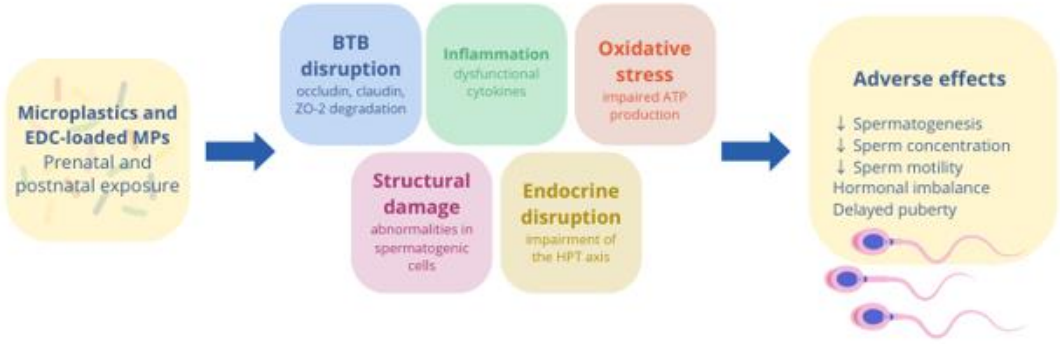

Fig. 3. *Suggested mechanisms of gonadal toxicity of MPs.*

How can we address the problem of microplastics?

The growing evidence of microplastics in water, food and environment has created the need to address and mitigate this concern. Current strategies focus on reducing the exposure to microplastics through both technological and behavioural approaches. Advanced filtration methods such as ultrafiltration, nanofiltration, and reverse osmosis, have proven effective in removing microplastic particles from drinking water, while new biotechnological tools, including plastic-degrading enzymes such as PETase and microorganisms like *Ideonella sakaiensis*, accelerate the breakdown of polymers in the environment. Simple everyday changes in a daily routine, such as using filtered water, avoiding plastic kitchenware, and choosing glass or metal storage containers, help reduce secondary microplastic contamination (Jahedi & Jaafarzadeh Haghighi Fard, 2025).

Indoor spaces can be major sources of microplastic inhalation, mainly due to fibers released from synthetic textiles and furnishings. Using High Efficiency Particulate Air (HEPA) filters, ventilating regularly, and vacuuming with HEPA devices reduces airborne particle levels (Tang, 2025). In a broader perspective, strict government policies on plastic production and waste management, as well as public campaigns encouraging reduced plastic consumption, may play an important role in decreasing microplastic pollution (OECD, 2024).

Therapeutic options

Although limiting environmental exposure remains the primary strategy for reducing the adverse effects of microplastics on human health, including male fertility, recent studies have increasingly focused on

therapeutic options that can counter the damage to the reproductive system caused by microplastics. One study described an innovative approach using extracellular vesicles containing the SKAP2 protein (mEVs-SKAP2) to repair PTFE-induced damage to sperm. PTFE (polytetrafluoroethylene) is a common source of microplastic particles, which can be released when Teflon coatings become damaged. Extracellular vesicles deliver SKAP2 protein to sperm cells, which restores the disrupted actin cytoskeleton structure and corrects defects in sperm morphology and motility. In mice exposed to PTFE, treatment with mEVs-SKAP2 significantly improved sperm quality and restored fertility, while co-incubation with sperm from infertile men exposed to PTFE improved sperm motility (Gan et al., 2025).

Similar studies have shown the positive effects of supplementation with a postbiotic containing *Lactobacillus brevis* GKJOY on damage caused by polystyrene microplastics in male rats. GKJOY supplementation significantly reduced oxidative stress and inflammation, restored hormonal balance in the hypothalamus, pituitary gland, and gonads, and improved semen parameters (Hwang et al., 2025). This effect of L. brevis GKJOY may be a promising treatment for male infertility caused by microplastics.

Conclusions

This review, which focuses on examining the impact of small plastic particles on male fertility, after reviewing available scientific sources, confirms the unhealthy and toxic impact on male fertility by them.

Plastic fibres- one of the smallest type of plastic waste are ubiquitous in the environment. People can find Micro- and Nanoplastic in food, drinking water or in air pollution, which are even more dangerous for health indoors, where those pollution accumulate than outdoors.

MPs are characterized by features that contribute to their extraordinary toxicity. Small sizes allow plastic particles to migrate and deposit throughout the human body. Chemical composition, such as Polyethylene or Polystyrene and contamination, such as heavy metals destroy cells' cytoskeleton and therefore tissues and organs functions. (Fig.1)

Scientific studies show a clearly negative impact of MPs on male reproductive system. This conclusion forces us to more quality education, health-promoting behaviors, changes at the production level or even tightening of the law. Such actions will help reduce Microplastic circulation in the environment but they can not eliminate it completely. That's why scientists work on ideas, which will help repair negative effects of MPs in the human body, such as supplementation with Lactobacillus brevis GKJOY or SKAP2 protein. All these activities provide a chance to prevent the decline in sperm quality and increase male fertility.

Beyond the general conclusions drawn from the reviewed studies, it is important to emphasize that the biological effects of microplastics are highly complex and cannot be explained by a single mechanism. Their impact on the male reproductive system depends not only on particle size or concentration, but also on polymer type, surface characteristics, and the ability to bind other environmental contaminants, such as heavy metals and endocrine-disrupting chemicals. This diversity of microplastic particles makes direct comparisons between studies difficult and represents a major challenge for reliable risk assessment.

Another important issue is the limited availability of human data. Although animal models have provided valuable insights into the potential mechanisms of toxicity, they do not fully reflect real-life human exposure, which is typically chronic, low-dose, and involves mixtures of different microplastic types. Moreover, differences in metabolism, reproductive physiology, and exposure duration further restrict the direct translation of experimental findings to clinical practice. For this reason, future research should increasingly focus on well-designed human studies, including biomonitoring approaches and long-term observational analyses.

From a broader perspective, the ubiquitous presence of microplastics in everyday environments suggests that male reproductive health may be affected not by short-term exposure, but by continuous contact throughout life. This highlights the importance of preventive measures aimed at limiting environmental contamination and reducing individual exposure wherever possible. At the same time, the emerging therapeutic strategies discussed in this review, including microbiota-based interventions and targeted molecular approaches, provide an interesting direction for future research, although their clinical applicability still requires further confirmation.

Taken together, the evidence presented in this review supports the view that microplastics constitute a relevant and potentially modifiable environmental factor contributing to impaired male fertility. Addressing this issue will require a coordinated effort combining advances in environmental science, toxicology, reproductive medicine, and public health policy, with the ultimate goal of protecting male reproductive health in the context of increasing global plastic pollution.

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