Frontiers of Environmental Science & Engineering
2025, 19(6): 79 https://doi.org/10.1007/s11783-025-1999-2

REVIEW ARTICLE

Recent advances in microplastics research: impacts on mammalian sperm and reproductive health

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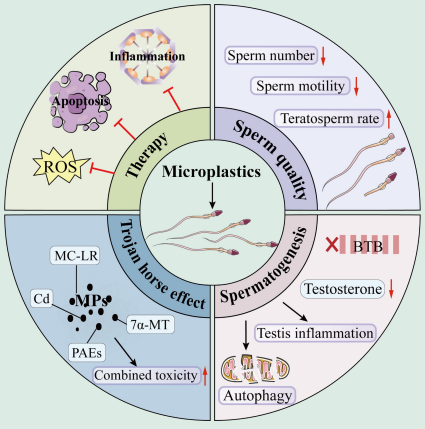
HIGHLIGHTS

- MPs exposure significantly impacts male spermatogenesis and sperm quality.
- Combined MPs and pollutant exposure may increase reproductive toxicity.
- MPs can impair gamete quality and directly affect offspring development.



ABSTRACT: Formed via the physical, chemical, and biodegradation of plastic products, Microplastics (MPs) are plastic fragments smaller than 5 mm. As a notable contributor to environmental pollution, MPs have gained significant attention owing to their wide application and potential toxicity. MPs have been reported to accumulate in mammalian reproductive organs, adversely affecting male sperm quality, and pose a serious threat to male fertility. Therefore, it is important to understand how MPs exposure impacts sperm and the male reproductive system. This manuscript reviews the research progress of MPs exposure on sperm toxicity from three aspects: the effect of MPs on spermatogenesis and sperm quality, the 'Trojan horse effect' and cross-generational effect of MPs. The findings indicated a significant correlation between MPs exposure and reduced sperm quality reduction as well as abnormal spermatogenesis. Additionally, the study highlights the 'Trojan horse effect' and cross-generation toxicity of MPs in mammals. This manuscript also reviews current treatment approaches for MPs exposure, providing a valuable theoretical foundation for future scientific research and clinical interventions. In summary, this review emphasizes that MPs can impair male reproductive health through mechanisms such as inflammatory responses, hormonal disruption, and sperm toxicity. By consolidating current evidence, this work lays a foundation for future research to further investigate the molecular pathways and long-term effects of MPs on male fertility.

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Article history: Received 3 December 2024, Revised 2 March 2025, Accepted 24 March 2025, Available online 15 April 2025

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KEYWORDS: Microplastics, Male fertility, Sperm quality, Spermatogenesis, Toxicity, Cross-generational

1 Introduction

The growing plastics industry and the increasing demand for plastic products in daily life have led to the widespread production, use, and disposal of plastic materials. By 2025, global plastic production is projected to reach approximately 590 million metric tons. However, due to inefficient recycling, millions of tons of plastic accumulate in the environment every year (Li et al., 2024a). The plastic wastes can be degraded by various physical, chemical, and biological processes into smaller plastic particles (Hu et al., 2024). Microplastics (MPs) are plastic particles smaller than 5mm in size (Zhou et al., 2024). The most common microplastic polymers found in the environment include high-density and low-density polyethylene (HD/LD-PE), polyethylene terephthalate (PET), polypropylene (PP), polystyrene (PS) and polyvinyl chloride (PVC) (Wagner et al., 2014). There are two main kinds of MPs: primary and secondary. Primary MPs enter the environment through various sources—for example, product use and unintentional loss from spills during manufacturing or transport. While secondary MPs result from the breakdown of larger plastic materials. Since MPs are non-biodegradable, both types accumulate and persist in the environment once released (D'Angelo and Meccariello, 2021). Furthermore, their small size and resistance to biodegradation make them easily absorbed by organisms, leading to bioaccumulation in the body. MPs can be transmitted through the food chain from lower to higher trophic levels, ultimately posing a health risk to humans. Human exposure to MPs primarily occurs through ingestion, inhalation, and skin contact (Prata et al., 2020). Notably, recent studies suggest that the MPs may adversely affect the reproductive systems of both sexes due to their accumulation in the reproductive organs of mammals (He and Yin, 2024). In men, exposure to MPs has been linked to testicular and sperm structural abnormalities, reduced sperm motility, and endocrine disorders (He and Yin, 2024). For these reasons, MPs, have garnered widespread public attention as an emerging pollutant.

Infertility is reported to affect approximately 17% of couples worldwide, with male factors contributing to about 50% of cases. Notably, half of these cases are classified as idiopathic, highlighting the gaps in understanding male reproductive physiology (Barrachina et al., 2023). Data indicates that the average sperm number in men declined by 62% globally between 1973 and 2018. Exposure to environmental toxic substances plays a key role in sperm decline (Zhang et al., 2023b). Therefore, it is the need of the hour to understand and mitigate the impact of MPs exposure on mammalian sperm and the male reproductive system. This article reviews the research progress on MPs-induced sperm toxicity from three key aspects: the effect of MPs on spermatogenesis and sperm quality, the Trojan horse effect, and the cross-generational impact of MPs. The review aims to provide a foundation for further research in this area.

2 Effect of MPs on spermatogenesis

Azoospermia or oligozoospermia are the primary characteristics of male infertility, both resulting from abnormal spermatogenesis, which results in sperm deformity and weakness (Krausz and Riera-Escamilla, 2018). Successful spermatogenesis is essential for maintaining normal male fertility. However, several factors, such as obesity, genetic abnormalities, environmental chemicals, and lifestyle choices can interfere with this process (Neto et al., 2016). Particularly, exposure to bisphenol A, nonylphenol, and phthalates—widely studied environmental chemicals commonly found in plastics, personal care products, and industrial applications—has been reported to disrupt spermatogenesis, leading to male reproductive disorders (Jenardhanan et al., 2016). In recent years, several studies have demonstrated that polystyrene microplastics (PS-MPs) induce reproductive toxicity in male mice. For instance, Wen et al. reported that PS-MPs cause sperm damage by primarily disrupting pyruvate metabolism and thyroid hormone metabolism. Their study was the first to highlight the molecular mechanism of PS-MPs in spermatogenesis damage in male mammals, providing valuable mechanisms into the precise impact of PS-MPs on male reproduction (Wen et al., 2024).

The blood-testis barrier (BTB) plays a crucial role in maintaining the homeostasis of the seminiferous tubule microenvironment, which is essential for spermatogenesis. Previous studies have shown that

BTB damage can impair spermatogenesis, resulting in male infertility and diseases such as oligospermia, asthenospermia, and teratozoospermia, as it is essential for maintaining the biochemical environment necessary to support spermatogenesis (Ma et al., 2024). Additionally, most studies on male animals have demonstrated that MPs can disrupt the BTB by triggering MAPK/Nrf2, p38 MAPK, and Nrf2/HO-1/NF-B signaling pathways via reactive oxygen species (ROS)-related mechanisms. This disruption negatively impacts testicular health, reducing sperm quality, and ultimately leading to male infertility (Li et al., 2021; Yuan et al., 2022). Furthermore, a mouse study demonstrated that MPs can compromise BTB integrity through mTORC1 and mTORC2 imbalance, mediated by ROS (Wei et al., 2021).

Testosterone plays a crucial role in maintaining the structural integrity of BTB (Liang et al., 2024). The biosynthesis and secretion of testosterone are regulated by multiple transcription factors. Particularly, the expression levels of steroid acute regulatory protein (StAR) and Cytochrome P450 side-chain cleavage enzyme(P450scc), serve as key indicators for evaluating the reproductive toxicity of PS-MPs. Wen et al. reported that StAR expression, as well as the transcription level of P450scc, was significantly reduced in the 0.1 mg/d PS-MP exposure group. They suggested that the apoptosis of interstitial cells may disrupt testosterone synthesis by lowering StAR levels, ultimately leading to spermatogenesis disorders (Wen et al., 2023). Meanwhile, Zhang et al. demonstrated that the adsorption of Polystyrene Microplastics (PA-MPs) reduced the bioavailability of testosterone, leading to decreased sperm quality. These findings provided new insights into the comprehensive toxicity mechanisms of MPs in male mammals. They also highlighted the potential impact of plastic on reproductive health, suggesting that selecting plastics with lower hormone and molecule adsorption properties can alleviate the complex reproductive toxicity associated with MPs (Zhang et al., 2024b). Additionally, Liu et al. (2023) discovered that PS-MPs can penetrate Leydig cells directly and speculated that this may suppress testosterone production by impairing Leydig cell function, contributing to reproductive dysfunction. Meanwhile, Jin et al. (2022) demonstrated that prolonged exposure to PS-MPs leads to male reproductive toxicity and a decline in testosterone levels by LH-mediated downregulation of the LHR/cAMP/PKA/StAR pathway. In another study, they observed that MPs can infiltrate three types of testicular cells (germ cells, Leydig cells, and Sertoli cells), accumulate in the testes, and subsequently cause spermatogenesis disorders, reduce testosterone secretion, induce testicular inflammation, and impair the BTB (Jin et al., 2021).

The BTB is maintained by Sertoli cells and their quantity plays a crucial role in normal spermatogenesis progression. These cells provide essential nutritional and immune support for developing germ cells (Cai et al., 2023). Gao et al. (2023) demonstrated that exposure to MPs led to a reduction in the Sertoli number. Transmission electron microscopy revealed significant damage to cell junctions and severe edema in Sertoli cells, indicating BTB disruption in the experimental group. This finding was consistent with previous studies, suggesting that MPs migrating into the testis can destroy BTB.

Testis undergoes active cell division and metabolism, making it particularly susceptible to toxins (Ma et al., 2023). Several studies have demonstrated that PS-MPs induce testicular inflammation. Hou et al. (2021) reported that exposure to PS-MPs significantly upregulated the expression of pro-inflammatory molecule NF-κB and interleukin (IL)-1 and IL-6 inflammatory factors while downregulating the anti-inflammatory molecules Nrf2/HO-1. These findings suggest that PS-MPs exposure results in abnormal sperm quality in mice, primarily through the Nrf2/HO-1/NF-κB signaling pathway. These disruptions may contribute to spermatogenesis defects by promoting inflammation and apoptosis in testicular tissue, ultimately causing reproductive system damage. Similarly, Xie et al. (2020) reported that PS-MPs enhanced the expression of inflammatory factors such as IL-6, IL-1β, and TNFα in mouse testis tissue. Lin et al. (2024a) examined the impact of four different sizes of micro/nanoplastics (MNPs) in a mouse model through oral exposure, further supporting the fact that MNPs induce testicular inflammation by regulating macrophages. Hu et al. (2024) identified testicular cell apoptosis, BTB damage, decreased testosterone levels, and testicular inflammation as the key events contributing to spermatogenesis disorders and male reproductive toxicity. Furthermore, they integrated meta-analysis into adverse outcome pathway analysis and demonstrated that nearly a third of the included studies showed that MPs can enter and accumulate in the male reproductive system regardless of their size (20 nm to 10 μm) or duration of exposure (0.5 h to 35 d). Their findings also suggested that MPs exposure altered the morphology of testicular cells (Hu et al., 2024). A recent study also reported that PS-MPs can induce inflammatory responses by directly penetrating the BTB and infiltrating supporting cells. This heightened inflammation disrupts cell physiology and

function, leading to reproductive dysfunction. However, further research is necessary to gain a more precise understanding of underlying mechanisms and interactions (Jeon et al., 2024).

Testicular tissue exhibits a unique energy production pattern due to its special structure (Cai et al., 2023). Spermatogenesis, sperm motility, and fertilization require significant energy, primarily generated by mitochondria. Thus, any damage to mitochondria can significantly impair sperm function. Liu et al. examined the mitochondrial structure and function following PS-MPs exposure and found that 5 μm PS-MPs not only destroyed the GC-2 cells' mitochondrial structure but also reduced ATP levels and membrane potential, compromised mitochondrial genome integrity, and disturbed mitochondrial dynamic homeostasis imbalance, ultimately increasing mitophagy (Liu et al., 2022a). Furthermore, they determined that ROS may be a contributing factor to mitochondrial structure damage and autophagy (Liu et al., 2022b). These studies highlighted the mitochondrial toxicity of PS-MPs and contributed valuable scientific data toward understanding the mechanism of PS-MPs on sperm damage. Succinate dehydrogenase (SDH) and lactate dehydrogenase (LDH) play essential roles in sperm development and energy metabolism. Xie et al. (2020) observed a decline in LDH and SDH following PS-MPs exposure, suggesting that MPs may lead to insufficient energy supply in sperm cells. This energy deficiency could impair spermatogenesis, reduce the quality and quantity of mature sperm, and even cause morphological abnormalities, ultimately contributing to reproductive toxicity in mice. The study attributed these effects to MPs-induced oxidative stress and the activation of p38 and JNK MAPK pathways (Xie et al., 2020). Recent studies have demonstrated that by regulating the Sirt1-Pgc1α signaling pathway in male mice, PS-MPs impair mitochondrial function and disrupt spermatogenesis. These findings offer novel insights into the potential mechanism underlying PS-MPs-triggered reproductive toxicity (Fig. 1) (Jin et al., 2025).

3 Effect of MPs on sperm quality

Male fertility is commonly evaluated based on sperm quality, with sperm number and deformity index serving as key parameters (Xie et al., 2020). Wei et al. (2022) observed a significant decrease in sperm number in mice and a marked increase in sperm deformity after exposure to PS-MPs for 30 and 44 d. These findings suggest that PS-MPs may impair spermatogenesis in mice through testicular damage, leading to reduced sperm number and quality. Other experiments in this area have yielded similar results. Jin et al. (2021) observed that 28 d of continuous exposure to 0.5, 4, and 10 μm PS-MPs caused a decline in sperm motility and quality in male mice across all three size groups. Furthermore, the incidence of abnormal sperm also increased, with observed deformities, including acrosome loss, microcephaly, headless sperm, cervical folding, and tailless sperm. Similarly, in another mouse study, Hou et al. (2021) revealed a significant drop in epididymis viable sperm number after PS-MPs exposure. They identified three types of malformations: double tail deformity, unhooked deformity, and neck swelling deformity. Additionally, they found that higher exposure dosage correlated with increased sperm abnormalities. Lin et al. (2024a) observed that MNPs within the same size range impaired sperm motility in an oral exposure mouse model, leading to a reduction in progressive motility, linear motility, and linear velocity of sperm motility. Notably, the effect was most pronounced in the smaller size ranges (25–30 nm and 1–5 μm). Zhang et al. (2023b) exposed male mice to PS-MPs for five weeks and observed a significant decrease in sperm number and viability compared to the mice in the control group, along with an increased sperm deformity rate. Other abnormalities included sperm head enlargement, microcephaly, unhooked heads, irregular heads, and double-headed sperm.

Fang et al. (2024) also reported similar findings, demonstrating that PS-MPs exposure significantly increased the apoptosis rate in spermatogonial cells, decreased mitochondrial membrane potential, induced mitochondrial morphological damage, and elevated mitochondrial ROS levels. Overall, their study demonstrated that PS-MPs reduce sperm quality by activating mitochondrial oxidative stress and apoptosis in spermatogonia, offering novel insights into MPs-induced reproductive toxicity. Meanwhile, Li et al. (2024c) reported that polyethylene terephthalate microplastics (PET-MPs) induced different degrees of testicular tissue pathological damage and reduced sperm quality via oxidative stress and activation of the p38 signaling pathway. These results highlight the crucial role of oxidative stress mechanism and p38 signaling pathway in MPs-induced reproductive toxicity, warranting further investigation in future studies. Lin et al. (2024b) were the first to provide direct evidence that PS-MPs present in masks may induce reproductive toxicity in mammalian sperm, with high-dose exposure significantly reducing sperm motility. Similarly, Muhammad et al. investigated the

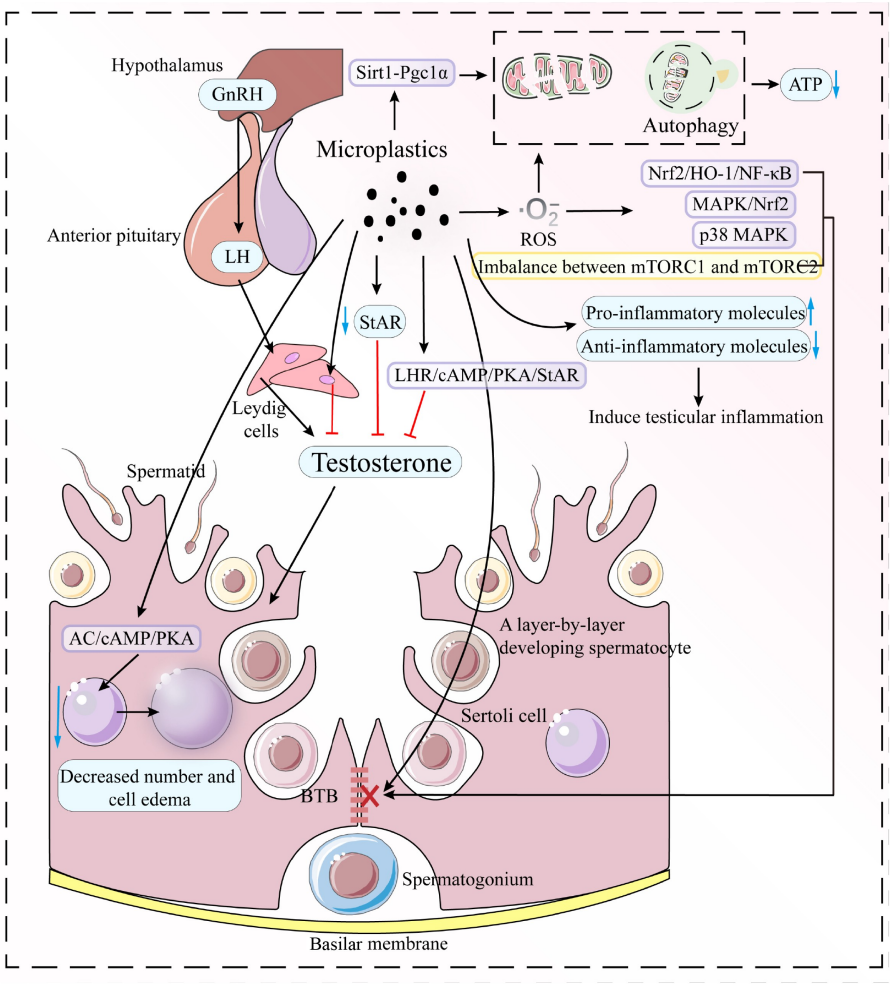

Fig. 1 The effect of MPs on spermatogenesis.

effects of five different concentrations of PS-MPs on rat testis tissue. Their study demonstrated that PS-MPs notably decreased antioxidant enzyme activity and total protein content while increasing lipid peroxidation and ROS levels. Additionally, sperm number, motility, and survival rate were significantly reduced. Thus, their studies suggest that PS-MPs may induce potential damage to rat testis in a dose-dependent manner through oxidative stress. Interestingly, higher doses of 10 μm PS-MPs had more pronounced effects on rats, suggesting a focal point for future research (Ijaz et al., 2021). Additionally, Gao et al. (2024) demonstrated that both low-dose MPs exposure and a high-fat diet (HFD) significantly reduced sperm quality and offspring number, with their combined effect enhancing these adverse outcomes. The study offers valuable insights into the impact of low-dose MPs exposure on the reproductive system, particularly in the context of metabolic disorders. Their findings serve as an early warning of the reproductive toxicity resulting from co-exposure to MPs and obesity, further expanding the understanding of the reproductive hazards associated with MPs.

Recent studies have made significant progress in understanding the reproductive toxicity of MPs in humans. Zhao et al. (2023) detected the presence of MPs in human testis and semen, demonstrating for the first time that MPs contaminate the human male reproductive system. This report provided crucial information and foundational results for assessing the potential risks of MPs to human health. By analyzing semen samples from 40 participants undergoing pre-marital health assessment in Jinan, China. Li et al. (2024) recently conducted an early study to detect MPs in semen samples from a general population cohort. The findings of this study confirmed the presence of MPs in semen, even in individuals without occupational exposure. The authors identified PS, polyethylene (PE),

and PVC exposure as the most prevalent MPs and highlighted their varying associations with progressive sperm motility. Therefore, there is an urgent need to understand how MPs affect the male reproductive system in real-world scenarios to facilitate more in-depth studies in the future. It is also the need of the hour to conduct more robust and multifaceted studies to validate these results and gain a more comprehensive understanding of MPs on human reproductive health.

Sperm DNA is a crucial determinant of embryonic development, with the DNA fragmentation index (DFI) serving as a key indicator of sperm DNA integrity. DFI is widely used in clinical settings to evaluate male fertility (Zhang et al., 2022). A traditional Chinese medicine formulation, Yishen Tongluo prescription, is known for its potential benefits in improving renal function and alleviating oxidative stress. Recent studies have determined its precise mechanism in inhibiting DNA damage through the PI3K/Akt pathway and secreted protein acidic and cysteine rich gene (Zhang et al., 2023a). These findings provide a novel direction for using traditional Chinese medicine in the prevention and repair of MPs-induced reproductive system damage. In recent times, there has been significant research on the effects of PS-MPs on DNA fragmentation in aquatic organisms. For instance, PS-MPs have been demonstrated to reduce the sperm motility of *Paracentrotus lividus* and induce sperm DNA fragmentation through ROS production (Mottola et al., 2024). However, data on the impact of MPs in mammals remains relatively scarce. Future research should focus on this area, particularly on elucidating the underlying molecular mechanisms (Table 1).

4 The ‘Trojan horse effect’ and cross-generational effect of MPs

In the natural environment, mammals are exposed to various combinations of MPs, yet the combined exposure of MPs and other substances is often overlooked in accumulation research. Beyond their inherent toxicity, MPs can interact with environmental molecules/particles, absorbing them due to their hydrophobicity and high surface-to-volume ratio. Acting as carriers, they can transport and release them to new sites and organisms—a phenomenon known as the ‘Trojan Horse effect’ (Hassine et al., 2023). Toxic chemicals or endocrine-disrupting compounds, either as plastic additives or substances, can easily enter the human body, acting as agonists or antagonists for various hormone receptors, and inducing endocrine toxicity (Ullah et al., 2022). Given the complexity of real-life exposure environments, predicting the impact of a single substance on human health remains challenging. Zhang et al. (2023b) were the first to evaluate the combined effects of MPs and cadmium (Cd) on rat testes. Their findings revealed that co-exposure caused greater reproductive damage than Cd or PS-MPs alone. This study further elucidated that co-exposure of MPs and Cd can lead to iron overload in the testis of mice, mediated by miR-199a-5p/HIF-1α. This overload may induce testicular pathological damage through ferroptosis in mice, thereby reducing sperm quality and testosterone levels, ultimately contributing to male reproductive toxicity in mice.

In another study, two different exposure modes were applied to prevent the early adsorption of Cd by MPs. The results indicated that the simultaneous exposure of MPs and Cd caused more severe effects than MPs alone, but was less harmful than Cd alone. This suggests that the differing interactions between the two substances in rats may influence their toxicity. Upon initial contact in the gastrointestinal tract of animals, MPs can adsorb Cd, reducing its bioavailability through the ‘Trojan horse effect’ (Venditti et al., 2023). Liu et al. (2023) also found that combined PS-MPs and MC-LR (microcystin-LR) also led to a similar effect on the reproductive system of male mice. The PS-MPs were found to adsorb MC-LR in water and transport it to the Leydig cells, where they inhibited testosterone synthesis by downregulating StAR and steroid synthase expression. Furthermore, the co-exposure of the two aggravated the reproductive toxicity in male mice. The study also reported that MPs contaminated with phthalates (PAEs) can accumulate in the testis, altering testicular weight and impairing sperm physiologic function by decreasing sperm number and motility. Additionally, PAEs-contaminated MPs can also cause oxidative stress in the testis and disrupt sperm function (Deng et al., 2021). Further studies are needed to elucidate the molecular mechanisms underlying coexposure-induced male reproductive dysfunction.

Numerous studies have demonstrated that MPs can act as carriers of aquatic pollutants, potentially amplifying their toxic effects. For instance, Rong et al. (2024) observed in animal experiments that co-exposure to PS-MPs and 7α-methyltestosterone over the long-term can disrupt steroid hormone production. Disruptions in steroid hormone synthesis can also delay gonadal development, leading to various pathological conditions. This ultimately results in decreased sperm motility, exacerbates reproductive system damage in zebrafish, and increases toxicity to subsequent generations. Studies on aquatic organisms have revealed that PS-MPs can be transmitted through the food chain and accumulate in organisms. Given that

Table 1 Details of animal experiment articles included in this review

Species	Type of study	MPs size and type	Contact ways	Exposure frequency and / or duration	Exposure dose	Impact	Reference
Kunming mice	*in vivo*	0.1, 1 μm; bPS-MPs	Oral gavage	5 d a week 5 weeks	100 mg/(kg·d)	Testicular pathological damage, sperm quality and testosterone levels decreased; the co-exposure of PS-MPs and Cd aggravated reproductive damage.	Zhang et al., 2023b
C57BL/6 mice	*in vivo*; *in vitro*	5 μm; PS-MPs	Oral gavage	every day for 60 d	40 mg/(kg·d)	PS-MPs impair spermatogenesis mainly by affecting pyruvate metabolism and thyroid hormone metabolism.	Wen et al., 2024
Wistar rats	*in vivo*	0.5 μm; PS-MPs	Drink water containing PS-MPs	every day for 90 d	0.015, 0.15, 1.5 mg/d	PS-MPs exposure may lead to the destruction of BTB integrity and spermatogenic cell apoptosis by activating the MAPK-Nrf2 pathway.	Li et al., 2021
BALB/C mice	*in vivo*; *in vitro*	4, 10 μm; PS-MPs	Gavage	once per day 28 d	20, 40 mg/kg	PS-MPs disrupt the blood-testis barrier integrity through ROS-Mediated imbalance of mTORC1 and mTORC2.	Wei et al., 2021
Kunming mice	*in vivo*	10 μm; PS-MPs	Oral gavage	35 d	0.01, 0.1, 1.0 mg/d	Sperm number and motility decreased, the rate of sperm deformity increased, testicular injury and testosterone levels decreased.	Wen et al., 2023
ICR mice	*in vivo*; *in vitro*	75 μm (PA, PE, PP, PMMA, PS, PC, PVC, PET) and 5 μm (PA, PP, PMMA)	Gavage	once per day 28 d	0.1, 0.5 mg/d PA-MPs; 0.1, 0.5 mg/d PMMA-MPs	PA-MPs adsorption reduced testosterone bioavailability and caused sperm quality to decline.	Zhang et al., 2024b
BALB/C mice	*in vivo*; *in vitro*	4 μm; PS-MPs	Oral gavage	once per day 28 d	100 μL, 45 mg/(kgžbw)	Single exposure can destroy the testicular structure, increase tissue apoptosis level, and reduce sperm quality. The toxic effect of co-exposure was enhanced	Liu et al., 2023
BALB/C mice	*in vivo*; *in vitro*	0.5, 4, 10 μm; PS-MPs	Drink water containing PS-MPs	180 d	100, 1000 μg/L	PS-MPs caused changes in testicular morphology, decreased LH and FSH, decreased sperm motility and increased abnormal rate, and reduced testosterone levels by downregulating the LHR / cAMP / PKA / StAR pathway.	Jin et al., 2022
BALB/C mice	*in vivo*; *in vitro*	0.5, 4, 10 μm; PS-MPs	Oral gavage	once per day 28 d	100 μL (10 mg/mL)	MPs can infiltrate three types of testicular cells and induce spermatogenesis disorders, decreased testosterone secretion, testicular inflammation and BTB damage.	Jin et al., 2021
CD-1 mice	*in vivo*	0.5, 5 μm; PS-MPs	Drink water diluted with PS-MPs	12 weeks	1 mg/L	The number of supporting cells decreased and the area of interstitial cells decreased. Decreased sperm motility and quality	Gao et al., 2023
ICR mice	*in vivo*	5 μm PS-MPs	Drink water diluted with PS-MPs	35 d	0.6–0.7 μg/d; 6–7 μg/d; 60–70 μg/d	The abnormal sperm quality caused by PS-MPs exposure is closely related to Nrf2 / HO-1 / NF-κB pathway.	Hou et al., 2021
BALB/C mice	*in vivo*	5.0–5.9 μm MPS	Oral gavage	once per day 42 d	0.25 mL; 0.01, 0.1, 1 mg/d	Induce oxidative stress and activate p38 and JNK MAPK pathways; sperm quality, testosterone production, SDH and LDH activity decreased.	Xie et al., 2020
C57BL/6J mice	*in vivo*	25–30 nm, 1–5 μm, 20–27 μm, 125–150 μm; MNPs	Oral gavage	21 d	0.1·mg /d per mouse	The impact of MNPs on sperm motility varied with particle size.	Lin et al., 2024a
C57BL/6 mice	*in vivo*; *in vitro*	5 μm; PS-MPs	Oral gavage	28 and 56 d	10, 30 mg/L	The increase in the duration of PS-MPs exposure is associated with a decrease in sperm number, normal morphology, and motility.	Jeon et al., 2024
GC-2 cells	in vitro	5 μm PS-MPs	Direct exposure	24h	0, 50, 100, 200, 400, 800 μg/mL	Mitochondrial damage, reduced ATP content, decreased mitochondrial membrane potential, and destruction of mitochondrial genome integrity.	Liu et al., 2022a
ICR mice	*in vivo*	5 μm PS-MPs	Drink water diluted with PS-MPs	35 d; 71 d; 106 d	60–70 μg/d	Mitochondrial membrane potential decreased, mitophagy, ATP content decreased. Mitochondrial damage may be caused by oxidative stress.	Liu et al., 2022b
BALB/c mice	*in vivo*; *in vitro*	0.5, 4, 10 μm; PS-MPs	Drink water containing PS-MPs	28 d	10 mg/mL; 100 μL	PS-MPs induced spermatogenesis disorder via disrupting mitochondrial function through the regulation of the Sirt1-Pgc1α signaling pathway.	Jin et al., 2025

(*Continued*)

Species	Type of study	MPs size and type	Contact ways	Exposure frequency and / or duration	Exposure dose	Impact	Reference
C57BL/6 mice	*in vivo*	5.0–5.9 μm PS-MPs	Oral gavage	once per day 20 d	0.1 mg/d, 0.2 mL of the PS-MPs diluent	Oxidative stress is induced and serum hormone levels are reduced. As a result, sperm quality and number decrease.	Wei et al., 2022
C57BL/6 mice	*in vivo*; *in vitro*	5 μm PS-MPs	Oral gavage	once per day four weeks	0.25, 0.5, 1 mg/d	Trigger a decrease in sperm quality through the activation of mitochondrial oxidative stress and apoptosis in spermatogonia.	Fang et al., 2024
BALB/c mice	*in vivo*	1 μm; PET-MPs	Gavage	42 d	0.01, 0.1, 1 mg/d	Oxidative stress and p38 signaling pathway may play an important role in PET-MPs-induced sperm quality reduction, testicular pathological changes and spermatogenic cell apoptosis.	Li et al., 2024c
C57BL/6J mice	*in vivo*	0.5 μm Mask MP	Oral gavage	21 d	0.1, 1 mg/d per mouse	impair sperm motility; interference with testicular spermatogenesis, oxidative stress, Immune and metabolic disorders.	Lin et al., 2024b
Sprague Dawley rats	*in vivo*	10 μm; PS-MPs	Oral gavage	60 d	2, 20, 200, 2000 μg/L of PS-MPs	Testosterone levels, sperm number, and motility decreased. Oxidative stress and dose-dependent potential damage to rat testis.	2021
C57 mice	*in vivo*	PS-MPs	Exposed by drinking water	12 weeks	25–30 μg/(kg·d)	Exposure to low-dose MPs or HFD feeding can reduce sperm quality and offspring numbers, and the combination of the two is more effective.	Gao et al., 2024
CD1 mice	*in vivo*	90 nm; PS-MPs	Gavage	60 d	1 mg/d	Damage sperm DNA.	Zhang et al., 2023a
Wistar rats	*in vivo*	PS-MPs	Drinking water for Cd and oral gavage for MPs	30 d	0.1 mg/d (1.5×10^6 particles/d)	The effect of simultaneous treatment of MPs and Cd was more serious than that of MPs alone, and less harmful than that of Cd alone.	Venditti et al., 2023
CD1 mice	*in vivo*	0.4–5 μm; PE-MPs	Oral gavage	30 d	100 mg/kg	MPs contaminated with PAE enhanced reproductive toxicity.	Deng et al., 2021

mammals may be exposed to PS-MPs through the ingestion of contaminated aquatic products or drinking water, their potential toxic effects on the mammalian reproductive system warrant further study. This research direction could offer crucial scientific insights for a comprehensive assessment of the reproductive health risks posed by MPs pollution to higher organisms. A recent multicenter study examined the presence of various MPs in human semen and urine and their impact on sperm quality. The findings indicated that an increase in the number of types of MPs significantly correlated with reduced sperm concentration, total sperm number, progressive motility, non-progressive motility, and normal morphology. Different MPs exhibit different toxicity mechanisms and bioaccumulation effects. Consequently, simultaneous exposure to multiple MPs in humans may lead to a synergistic effect, exacerbating reproductive system damage. Future microplastic mixed exposure research should focus on elucidating the potential mechanisms underlying MPs mixed exposure and its impact on male reproductive toxicity (Zhang et al., 2024a).

Reproductive development is closely linked to gamete quality, and MPs can adversely impact this quality, resulting in compromised offspring development. Recent studies have also emphasized the trans-generational effects of MPs, revealing that after being transferred from parental mice to offspring, they contribute to metabolic disorders, reproductive abnormalities, immune system deficiencies, and neurodevelopmental disorders (Liang et al., 2024). These findings offer new insights into the risks posed by MPs on mammalian fertility. However, the trans-generational toxic effects of MPs in mammals remain insufficiently understood, thereby necessitating further in-depth research.

5 Intervention strategies and treatment prospects of male reproductive damage caused by MPs exposure

Building on the previous in-depth discussion on the

relationship between MPs exposure and sperm toxicity, it is evident that MPs can accumulate in the reproductive organs through the "Trojan horse effect", and might also cause cross-generational toxicity, significantly impacting male fertility. Therefore, exploring effective prevention and treatment strategies is particularly important. In recent years, numerous studies have revealed that various natural compounds, such as Kaempferide (Ijaz et al., 2024), Rhamnetin (Hamza et al., 2023), Pinostrobin (Ijaz et al., 2023a), Ginkgetin (Akbar and Ijaz, 2024), Chrysoeriol (Ijaz et al., 2023b), and Astilbin (Rizwan et al., 2023) exhibit significant therapeutic potential against MPs-induced testicular injury. These compounds exert their effects through five similar mechanisms, including anti-oxidant, anti-inflammatory, anti-apoptotic, androgenic properties, and the regulation of steroidogenic enzyme expression (Fig. 2). Specifically, they mitigate MPs-induced oxidative stress by activating the Nrf-2 pathway, enhancing the activities of Catalase(CAT), Glutathione Reductase(GSR), Superoxide Dismutase (SOD), and Glutathione Peroxidase(GPx), and reducing ROS and Malondialdehyde (MDA) levels. Their anti-inflammatory properties are evidenced by the suppression of key inflammatory markers, including NF-κB, IL-1β, TNF-α, IL-6, and Cyclooxygenase-2 (COX-2). Additionally, these compounds exhibit anti-apoptotic effects by downregulating BCL2-Associated X Protein (Bax) and Caspase-3 expression while upregulating the anti-apoptotic protein B cell lymphoma 2 (Bcl-2). Through their androgenic activity, they restore hypothalamic-pituitary-testicular axis function, normalize follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels, and promote spermatogenesis, while also increasing germ cell numbers and alleviating MPs-induced testicular damage. Finally, they enhance testosterone synthesis by upregulating steroidogenic enzymes, such as 17β-hydroxysteroid dehydrogenase (HSD), StAR, and 3β-HSD, thereby regulating steroidogenesis. Notably, Zhang et al. reported a novel mechanism linking PS-MPs-induced sperm quality decline with gut microbiota and suggested that the activation of the IL-17A signaling pathway, driven by intestinal flora imbalance,

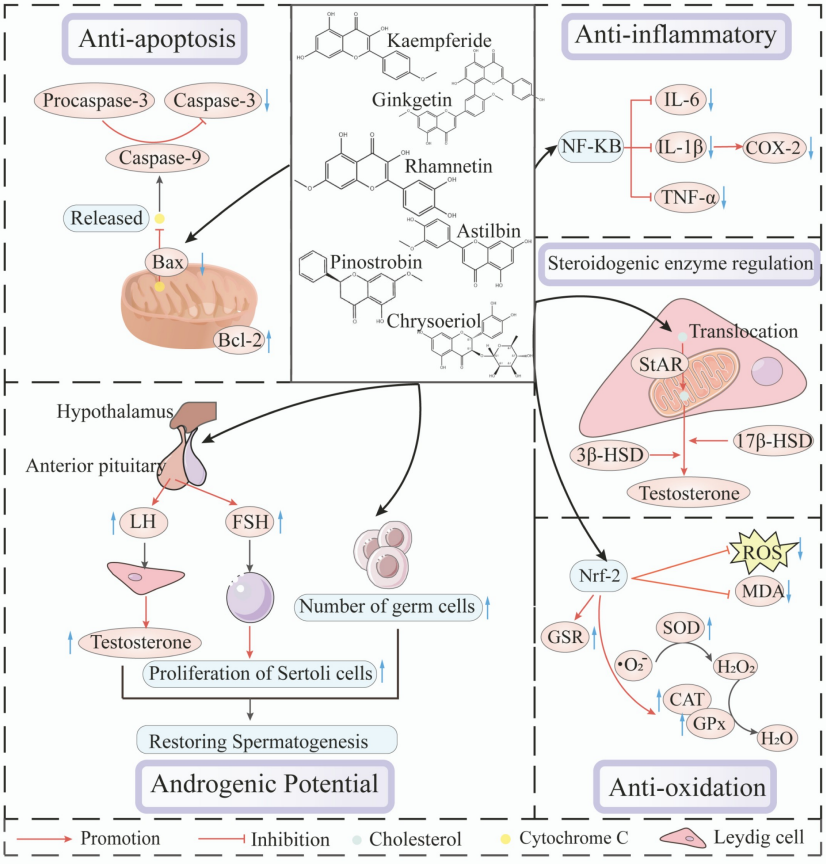

Fig. 2 Common mechanisms of drugs in reversing MPs-induced damage.

may play a crucial role in the process. The use of probiotic supplements may help mitigate sperm quality decline and testicular tissue inflammation caused by PS-MPs (Zhang et al., 2023c). These findings not only offer diverse treatment strategies for male reproductive damage caused by MPs but also establish an important foundation for developing personalized treatment approaches in the future.

6 Discussion and summary

MPs have become a significant environmental concern and their detection in human serum has introduced a new avenue for investigating their potential health effects. The widespread presence of these pollutants, raises concerns, particularly regarding their impact on male reproductive health. This review highlights numerous studies supporting the notion that MPs may induce reproductive toxicity in mammals through various mechanisms, ultimately affecting fertility. In summary, MPs exposure has been significantly associated with sperm quality decline and abnormal spermatogenesis, highlighting the 'Trojan horse effect' and cross-generational toxicity of MPs in mammals. Meanwhile, current intervention strategies and treatment advancements for MPs exposure offer a crucial theoretical foundation for subsequent in-depth research and potential clinical applications. Future research will be essential to gain a deeper understanding of these effects and to develop essential strategies to minimize the potential risks linked with MPs exposure.

However, the existing research has certain limitations. Most experimental toxicology models use particle types, shapes, sizes, concentrations, and exposure durations that are different from real-world human exposure. Additionally, they often fail to account for the impact of the composition, size, and shape of MPs on their reproductive toxicity. Moreover, data on the effects of degradable plastics on the reproductive system remain limited. Future studies should further investigate how these factors contribute to the reproductive toxicity of MPs to mammals, with a particular focus on the potential effects of degradable plastics. This will enable a more comprehensive assessment of their risks to reproductive health. Meanwhile, given the current research scale, many animal models have limited exposure durations, making it difficult to accurately represent the chronic or cumulative toxic effects of MPs on human organs and disease development. Furthermore, significant cross-species differences between humans and animals emphasize the need for comprehensive clinical and large-scale epidemiological studies for evaluating the possible reproductive toxicity of MPs to humans.

List of Abbreviations

Abbreviation	Definition
MPs	Microplastics
HD/LD-PE	High density and low density polyethylene
PET	Polyethylene terephthalate
PP	Polypropylene
PS	Polystyrene
PVC	Polyvinyl chloride
PS-MPs	Polystyrene microplastics
BTB	Blood-Testis Barrier
ROS	Phthalate Microplastics
StAR	Steroid Acute Regulatory Protein
P450scc	Cytochrome P450 side-chain cleavage enzyme
PA-MPs	Polystyrene Microplastics
IL-1	Interleukin-1
MNPs	Micro/Nanoplastics
SDH	Succinate Dehydrogenase
LDH	Lactate Dehydrogenase
PET-MPs	Polyethylene terephthalate microplastics
HFD	High-Fat Diet
PE	Polyethylene
DFI	Dna Fragmentation Index
Cd	Cadmium
MC-LR	Microcystin-LR
PAEs	Phthalates
KFD	Kaempferid
PE-MPs	polyethylene microplastics
Abbreviation	Definition
MPs	Microplastics
BTB	Blood-Testis Barrier
ROS	Reactive Oxygen Species
StAR	Steroid Acute Regulatory Protein
PS-MPs	Polystyrene Microplastics
IL-1	Interleukin-1
MNPs	Micro/Nanoplastics
SDH	Succinate Dehydrogenase
LDH	Lactate Dehydrogenase

HFD	High-Fat Diet
DFI	Dna Fragmentation Index
Cd	Cadmium
MC-LR	Microcystin-LR
PAEs	Phthalates
PMF	Plastic Mulch
CAT	Catalase
GSR	Glutathione Reductase
SOD	Superoxide Dismutase
GPx	Glutathione Peroxidase
MDA	Malondialdehyde
COX-2	Cyclooxygenase-2
Bax	BCL2-Associated X Protein
Bcl-2	B cell lymphoma 2
FSH	Follicle-stimulating hormone
LH	Luteinizing hormone
HSD	Hydroxysteroid dehydrogenase

CRediT Authorship Contribution Statement All authors contributed to the study conception and design. SHF: substantial contributions to conception and design; final approval of the version to be published. ZLL:substantial contributions to conception and design; agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. WN and JPP: article management and manuscript writing; drafting the article or revising it critically for important intellectual content. WLR, LXR and HJJ: manuscript editing.

Conflict of Interests The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Acknowledgements The Article was supported by the Gansu Youth Science and Technology Fund, China (No. 24JRRA319); the Lanzhou Science and Technology Development Guiding Plan Project, China (No. 2024-9-111); the Gansu Provincial Key R&D Program, China (No. 22YF7FA084). And the authors would like to thank all the reviewers who participated in the review and MJEditor for its linguistic assistance during the preparation of this manuscript.

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