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Microplastic pollution and fish reproduction: A critical review of molecular mechanisms, behavioral alterations, and transgenerational effects

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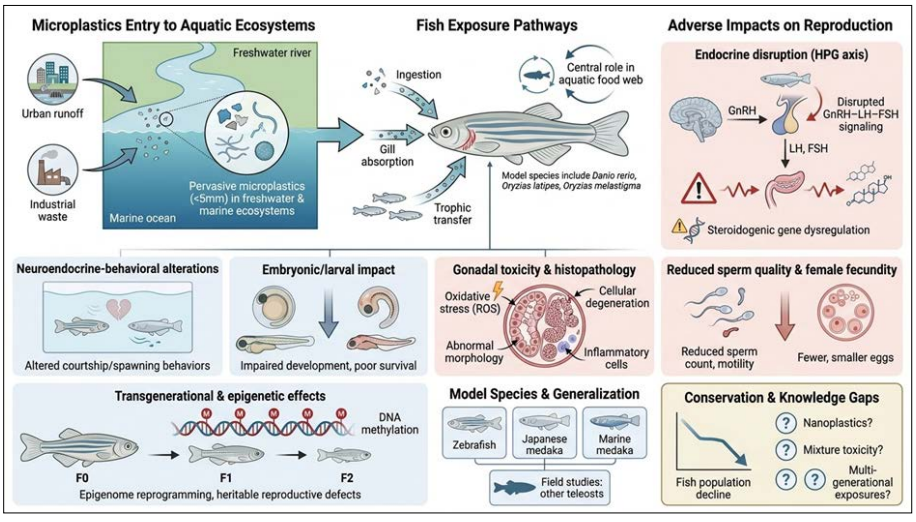
Abstract
Microplastics (MPs) — grit-sized (5 mm or smaller) plastic particles — have recently been identified as ubiquitous and global contaminants in both freshwater and marine ecosystems, contributing to the ongoing decline of biodiversity in aquatic environments. Fish, as key players in pelagic food webs, are most likely to be exposed to MPs through ingestion, gill absorption and trophic transfer. This review synthesizes the available evidence of how MPs affect fish reproduction across multiple mechanisms: (i) gonadal toxicity and histopathological changes, (ii) endocrine disruption at the hypothalamic–pituitary–gonadal (HPG) axis, (iii) sperm quality and female fecundity effects, (iv) embryonic development and larval survival effects, (v), neuroendocrine-mediated behavioral effects on courtship, mate choice, and spawning, and vi transgenerational/epigenetics. The main molecular mechanisms include: oxidative stress induced by increased production of reactive oxygen species (ROS), dysregulated expression of steroidogenic genes, inhibition of vitellogenin (VTG) synthesis, disruption of GnRH–LH–FSH signaling pathway and epigenetic changes such as DNA methylation alteration. Model species (e.g., zebrafish, Danio rerio, Japanese medaka, Oryzias latipes, marine medaka, Oryzias melastigma) have yielded mechanistic insight and field studies across the commercial and ecologically important teleosts bolster ecological relevance. Parental exposure to MPs effect transfer of reproductive damage across generations (F1 and possibly F2) via epigenome reprogramming in gametes, leading to serious population-level conservation implications. We identify major gaps in knowledge, specifically with regard to nanoplastics, mixture toxicity and multi-generational exposures at environmentally realistic concentrations, and outline areas where future research is needed to support or redirect regulatory frameworks. (Liu *et al*., 2024, Wang *et al*., 2023)



Keywords: Microplastics, fish reproduction, endocrine disruption, HPG Axis, transgenerational effects, gonadotoxicity, oxidative stress, sperm quality, epigenetics, aquatic toxicology

Introduction
Plastic pollution is one of the most serious environmental problems today, and it will be even intensifying in the twenty-first century. Global production of plastic has now exceeded 400 million metric tonnes per annum since commercial production started in the 1950s, with expectations for further exponential growth into the future. Much of this material finds its way into waterbodies because of improper waste disposal, urban runoff, and atmospheric deposition. After entering an aquatic environment, larger plastic debris breaks down through continuous physical abrasion, ultraviolet photodegradation and microbial attack into particles less than 5 mm — together called microplastics (MPs) — and eventually nanoplastics (NPs) below 1 µm.

Two main sources serve as the origin of MPs in aquatic systems. Some polymers are manufactured for being primary microplastics at a small scale which can be used in

cosmetics (microbeads), industrial abrasives, and textile fibres. Secondary MPs originate from in situ breakdown of macroplastic waste such as fishing gear, packaging and agricultural plastics. Among them, the major types of MPs detected in environmental matrices are polystyrene (PS), polyethylene (PE), polypropylene (PP) polyvinyl chloride(PVC), and polyethylene terephthalate(PET).

MP abundance in freshwater and marine environments varies over orders of magnitude, from < 1 particle l-1 for most open ocean surface waters to hundreds or thousands of particles l-1 in some river systems near urban population centres, and thus contributes substantially to the difficulty in performing inter-regional comparisons. Fish are exposed to MPs via several routes: direct ingestion of free-floating particles that mimic prey items; uptake through consumption of contaminated zooplankton or invertebrate prey; and for smaller particles (in the nano size range), across gill lamellae. After digestion, MPs could migrate across the gastrointestinal tract into systemic circulation and accumulate in different organs such as liver, kidney, brain and gonads.

Environmental disturbance is remarkably varied for fish reproduction. The reproductive axis — an exquisitely controlled neuroendocrine cascade from the hypothalamus to the pituitary and to the gonads — can be affected by chemical contaminants, physical stressors, or changes in nutritional state. Disruption of fish reproduction cascades through ecological pathways leading to reduced recruitment, compromised population fitness, skewed sex ratios and even population collapse. Through multiple interacting mechanisms including direct gonadotoxicity, endocrine disruption through leached plastic additives, oxidative stress, epigenetic reprogramming and neurobehavioural disruption impacting reproductive interactions, MPs pose a threat to fish reproduction. (Wang *et al*., 2021; Zhou *et al*., 2023) [3, 9]

Several lines of evidence from gonadal, endocrine, behavioural and transgenerational research in freshwater and marine fish contribute to recent speculation on the potential impact of MPs; however, given the rapid expansion of the literature on MP Ecotoxicology since around 2015, a mechanistic synthesis is timely. By evaluating laboratory evidence (especially from model species like zebrafish Danio rerio, Japanese medaka Oryzias latipes, marine medaka O. melastigma and fathead minnow Pimephales promelas) and also putting this literature into a broader ecological context based on data from wild populations, the present review seeks to fill this gap. We particularly focus on transgenerational effects and epigenetic mechanisms, which can raise concerns for population risk assessment.(Wang *et al*., 2021; Zhou *et al*., 2023) [3, 9]

Microplastic Sources, Physicochemical Properties, and Environmental Fate

1. Classification and Environmental Occurrence

MPs are categorized according to size (1 µm–5 mm), shape (fibres, fragments, beads, films, pellets), and polymer type. Fibres dominate many freshwater systems due primarily to washing of synthetic textiles. An important proportion of fragmented packaging polymers enters marine environments, while in agricultural soils, irrigation water and plastic mulch films constitute secondary sources by displacing MPs to adjacent aquatic systems.

Aquatic MP Particle concentrations are highly variable, ranging from 0.1–100 particles/m³ in marine surface waters to >10,000 particles/m³ near industrial and urban centres within riverine systems. Suspended sediment levels can occur at concentrations orders of magnitude greater than those occurring in the water column, which produces especially elevated risk for benthic-feeding and demersal fish such as salmonids or flatfish species that feed very close to or on the substrate.

2. Surface Chemistry and Contaminant Adsorption

The biological effects of MPs are significantly regulated by their physicochemical properties. They are naturally found ubiquitous in the environment and under particular environmental conditions they have high surface-area-to-volume ratios and hydrophobic surfaces which favours their adsorption towards other environmental contaminants with which they commonly coexist including persistent organic pollutants (POPs), polycyclic aromatic hydrocarbons (PAHs), heavy metals, pesticides, and pharmaceutical compounds. Carrier capacity of these MPs indicates that ingestion can deliver a bolus load of adsorbed toxicants to gut tissue, possibly enhancing reproductive toxicity attributable to the polymer matrix.

Plastic polymers themselves include multiple additives, such as plasticisers (phthalates, bisphenol A ⌈BPA]), flame retardants (polybrominated diphenyl ethers; PBDEs), antioxidants, UV stabilisers and pigments — most of which are widely recognised endocrine-disrupting chemicals (EDCs). Ingested MPs can leach these additives in gastrointestinal fluids exposing the mucosal surfaces and portal circulation to biologically active concentrations of EDC.

3. Weathering, Nanoplastic Formation, and Enhanced Bioavailability

MP become smaller by UV photodegradation and mechanical fragmentation, yielding NPs (< 1 µm), that have far more cellular penetrability. Nanoplastics can penetrate gastrointestinal epithelial barriers and circulate in blood, penetrate blood–brain barrier and accumulate in gonads at levels that may be higher than the parent MPs. The specific surface area to volume ratio of NPs is higher than that of MPs so their increased adsorption capacity and increased biological reactivity makes NPs a concern for potential reproductive toxicity, coupled with the fact that NP mass concentrations in the environment are commonly lower than those of corresponding larger scale photos as well.

Gonadal Toxicity and Histopathological Alterations

1. Male Gonadal Effects

The testis is a target organ for MP-mediated reproductive toxicity. In MP-exposed fish, testes have a consistent destructive histopathology that includes disorganisation of seminiferous tubule structure (Xia *et al*., 2008 and more), degeneration of spermatogonia and spermatocytes (Sung *et al*., 2015), vacuolation of Sertoli cells (Bhosale *et al*., 2013a) and upregulated germ cell apoptosis. Based on the above, PS-MPs were administered to zebrafish and clearly induced disorganized structure of seminiferous tubules as well as spermatocytes deformation along with high-level gonadal ROS generation in a particle size- and dose-dependent manner. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]

Gonad Somatic Index, the ratio between gonad mass and total body mass (recent best), is an integrative metric of reproductive investment commonly used. METHODS: A male-biased GSI was consistently reduced following chronic exposure to MPs (48 h) in a range of fish species including zebrafish, marine medaka and Japanese medaka, indicating that the development of testis has been suppressed. PPARγ antagonism inhibited the differentiation of spermatogonial stem cells into mature sperm, resulting in dose dependent male reproductive toxicity in developing male Japanese medaka and corresponding reductions in fecundity as measured by histogram analysis of correlation with testicular histology. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]

Sperm quality parameters, including sperm motility, progressive velocity, morphology, viability and acrosome integrity are sensitive endpoints for assessing the toxicity of MPs. Previous studies have demonstrated the detrimental effects of increased polystyrene NP on sperm viability in European whitefish (Coregonus lavaretus), while here we explore longer-term implications for fertilisation success and offspring phenotype following pre-fertilization exposure in this species. Catalysed by increased ROS, oxidative injury to sperm DNA is a mechanistically credible route through which MP gonadotoxicity could diminish fertilisation rates with genotoxic repercussions for descendants. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]

2. Female Gonadal Effects

MPs interference in folliculogenesis and vitellogenesis in female fish can hinder the reproductive success. The structural disorganisation of ovarian follicles and the suppression of vitellogenin (VTG) synthesis due to exposure to virgin MPs in a concentration-dependent manner. VTG - a yolk precursor protein, is an oestrogen-regulated gene required for both oocyte growth and the quality of matured eggs; silencing of VTG likewise results in decreased viability of embryos. By contrast, in male fish any induction of VTG appears to be a sensitive biomarker for oestrogenic MP exposure. (Huang *et al*., 2022; Gupta *et al*., 2023) [1, 15]

Chronic exposure to MPs in female guppies (Poecilia reticulata) resulted in significantly reduced pregnancy rates and embryo counts with marked changes in antioxidant enzyme activity (superoxide dismutase [SOD], catalase [CAT]) within gonadal tissues. Fertilisation was compromised at the cell level as well as via a hormonal disruption following 10d exposure with MPs in zebrafish causing changes to the gonadosomatic index and a dose-dependent reduction in fecundity rate. Decreased oocyte quality results in deficiencies in the deposition of yolk proteins, abnormalities of the zona pellucida and mitochondrial dysfunction which as well together reduce embryo development potential through fertilisation. (Malafaia *et al*., 2022; Chisada *et al*., 2019) [8, 22]

3. Oxidative Stress as a Central Mediator of Gonadotoxicity

This overproduction of ROS represents a feature that mechanistically unifies both male and female gonadotoxicity. MPs produce ROS via several routes, including physical validity through cell membranes, stimulation of NADPH oxidase activation, release of pro-oxidant metal ions attached to the particle surface and mitochondrial electron transport chain (ETC) disruption [9.10.eight]. Excess ROS in gonadal tissues induces its imbalanced use of antioxidant defences (including enzymatic superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPX) and glutathione S-transferase enzymes as well as the non-enzymatic glutathione (GSH)) leading to lipid peroxidation, protein carbonylation and DNA-strand breaks impairing germ cell viability [5, 6].

The balance of these antioxidant responses seems to be species- and sex-specific. A reduction of the SOD, CAT, GPX and GST activities was reported in gonads of marine medaka exposed to MPs while an increased SOD and CAT activity were found in guppies, probably showing dissimilar adaptive compensatory responses. In the zebrafish model, MP exposure increased ROS and malondialdehyde (MDA, a product of lipid peroxidation) in gonads as evidence for oxidative damage and led to increase germ cell apoptosis by caspase-3 activation pathways.

Endocrine Disruption and the Hypothalamic–Pituitary–Gonadal (HPG) Axis

1. Architecture of the Fish HPG Axis

Action of the reproductive neuroendocrine system is mediated via a serial hierarchal cascade involving hypothalamic-pituitary-gonadal (HPG) axis in fish. Gonadotropin-releasing hormone (GnRH) is released by neurones in the hypothalamus, and stimulates pituitary gonadotroph cells to secrete follicle-stimulating hormone (FSH) and luteinising hormone (LH). These gonadotropins then act on the gonadal granulosa, theca and Leydig cells to stimulate steroidogenesis (synthesis of oestradiol (E2), testosterone (T) and 11-ketotestosterone (11-KT)) and gametogenesis. Sex steroids exert negative feedback to the hypothalamus and pituitary gland to regulate GnRH and gonadotropin release, sustaining reproductive cyclicity. Removal of any component of this axis negatively affects the organized reproductive response.

2. MP-Mediated Disruption of HPG Gene Expression

Many studies show that MP exposure disrupts transcription of important HPG axis regulatory genes in fish brain, pituitary and gonads. In particular, MPs repress gonadotropin releasing hormone receptor (GnRHR), FSHβ, LHβ and gonadotropin-α (GTHα) expression in the pituitary to diminish the gonadotropin input required for steroidogenesis. It dysregulates expressions of GnRH and oestrogen receptors (ER-α, ER-β) and androgen receptor (AR-α) in the brain. The sterodogenic genes become dysregulated downstream and measures of steroidogenesis such as StAR (steroidogenic acute regulatory protein), CYP11A, CYP17A, CYP19a (aromatase) and HSD17B (17β-hydroxysteroid dehydrogenase) were decreased collectively implicating downregulation of sex steroids synthesis [227]. (Liu *et al*., 2023; Lin *et al*., 2023) [4, 14]

We showed direct hormonal evidence of HPG axis disruption in climbing perch (Anabas testudineus) exposed to PS-MPs at environmentally relevant concentrations, with the clear decreases in androgen and oestrogen serum levels correlating with substantial inhibition of key steroidogenic enzyme activity. Zebrafish and medaka studies consistently show that MPs can lower E2 and testosterone levels in circulation, damaging reproductive processes. Polystyrene MPs were then demonstrated to boost microcystin-LR-induced gonodal damage and reproductive endocrine disruptor effects in zebrafish, indicating that co-

contaminants have a potentiating effect. (Liu *et al*., 2023; Lin *et al*., 2023) [4, 14]

3. Vitellogenin and Estrogenic Signalling

Vitellogenin (VTG)-a well-known marker of endocrine disruption in fish, is a female egg yolk precursor protein which has oestrogen-responsive. Disrupting oestrogen receptor (ER) signalling, MP-leached EDCs can induce VTG in males (oestrogenic effect) and suppress VTG in females (anti-oestrogenic effect), both of which impair reproduction. Similarly, another ER responsive protein essential for zona pellucida formation choriogenin (CHG) is also deregulated. VTG secretioninhibition imposes a concentration-dependent impact on egg quality and embryonic viability. (Huang *et al*., 2022; Gupta *et al*., 2023) [1, 15]

MP oestrogenic disruption mechanisms include multiple path-ways: the direct binding of leached BPA and phthalates to oestrogen receptors (ERα, ERβ) or interference with ER co-activator recruitment in addition to the possible modulation of ER-mediated transcription through chromatin remodelling. Altered expression of CYP19a (aromatase) and CYP19b — rate-limiting enzymes that convert androgens to oestrogens — along with inhibition of molecular mediators of gonadal differentiation dmrt1 and sox9, provide evidence supporting the hypothesis that MPs can modify sex differentiation and reproductive health at later life stages. Additionally, we found that female zebrafish exposed to PS-MP have decreased levels of SIRT1 (sirtuin-1), a NAD-dependent deacetylase, which is an important regulator of HPG axis steroid hormones and plays a role in the decline in fecundity when combined with PS-MP. (Liu *et al*., 2023; Lin *et al*., 2023) [4, 14]

4. Sex-Specific and Species-Specific Responses

Notably, there is extensive evidence to suggest that male and female fish differentially respond to MP-induced endocrine disruption. In zebrafish, female fish were the more adversely affected sexes following PS-MP exposure with reductions in GSI and VTG suppression, as well as disruption of reproductive gene transcription detected at greater levels than males (2025). This sex specificity might result from differences in the expression of numerous steroid receptors, in steroidogenic pathways and antioxidant defence capacities [19]. Species -specific differences also modulate the characterization of HPG axis disruption in regard to type and extent, weighing in favor of multi-species assessments over single model organism reliance. (Liu *et al*., 2023; Lin *et al*., 2023) [4, 14]

Effects on Sperm Quality, Oocyte Development, and Fecundity

1. Sperm Parameters

Sperm quality is the cumulative indication of motility, velocity, morphology, viability, mitochondrial function and DNA integrity which can be altered by MP-induced oxidative and endocrine disruption. Chronic nanoplastic exposure from zebrafish gonads mediated oxidative and immune stress in medaka, causing sperm production adulteration. On the other hand, reduced mature sperm counts have been reported in zebrafish and medaka fish exposed to MPs and NPs with resulting direct reductions in fertilisation rates. In addition, MP-related mitochondrial dysfunction — characterized by decreased mitochondrial membrane potential and ATP synthesis — disrupts the energy metabolism of sperm motility, potentially impacting fertilisation success in their physiologically competitive aquatic spawning environment. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]

MP-induced inflammation and junction protein dysregulation in BTB, which are crucial to maintaining the immunoprivileged microenvironment for spermatogenesis. By destroying BTB integrity, immune cells gain access to developing spermatocytes, and exposure of germ cells to noxious agents during gonadotoxic drug administration can form a synergy leading to enhanced gonadotoxicity much beyond that caused by direct oxidative or endocrine effects alone. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]

2. Oocyte Quality and Clutch Characteristics

Rather, female reproductive output — the number of eggs spawned per cycle, fertilisation rates, hatching success and larval viability — is a measure of organismal fitness that can be readily linked to recruitment by populations. The fecundity of zebrafish exposed to PS-MPs displayed a strong and obvious dose-response relationship, with significant reductions in egg production observed at even low concentrations. Chronic MP exposure decreased pregnancy and embryo count in female guppies. (Malafaia *et al*., 2022; Chisada *et al*., 2019) [8, 22]

Impaired vitellogenesis leads to decreased energetic and structural status of oocytes, with less yolk volume, disorganization of the zona pellucida (ZP), and reduced ability to fertilize eggs. We document impaired reproductive toxicity in a commercially important marine teleost under near environment realistic scenarios of exposure; significant suppression of sexual maturation, fecundity and egg quality resulted in Atlantic cod (Gadus morhua) broodstock exposed to weathered polyethylene MPs.

3. Circadian Rhythm Disruption as a Modulating Factor

One largely unappreciated aspect of MP reproductive toxicity is circadian disruption, the dysregulation of 24-h biological clocks that have evolved to align reproductive cycles with environmental cues including photoperiod, temperature, and lunar cycles[9]. The coupling between neuroendocrine signalling pathways involving melatonin, kisspeptin and gonadotropin-releasing hormone (GnRH) tightly links fish spawning to circadian and seasonal rhythms. As circadian gene expression in fish is expected to be dysregulated by MP exposure, this could desynchronise seasonal spawning neuroendocrine cascades and internal physiologies, which can impact the timing and success of reproductive events in captive and wild populations.

Embryonic Development, Larval Survival, and Early Life Stage Sensitivity

1. Direct Embryotoxicity

Fish embryos and larvae are among the most toxicologically vulnerable life stages due to their underdeveloped physiology, rapid cell division, and reliance on maternally supplied yolk for energy. MPs can enter embryos not only by way of environmental exposure post-fertilisation, but also through maternal transfer of particles or leached chemicals from MPs either with or without physico-chemical modifications and incorporated into the yolk. It also provides direct in situ evidence of maternal MP transfer

to offspring, with a landmark 2026 field observation documenting MPs being found in wild fish larvae at the yolk-sac (non-feeding) stage.

These developmental abnormalities are decreased hatching success, cardiac oedema, spinal curvature (lordosis, kyphosis and scoliosis), yolk sac malabsorption, shorter larval body length and mortality when fish embryos are experimentally exposed to MPs and NPs. Responses indicated species specificity half the time; lethal concentrations were required for mortality in hybrid snakehead and Indian major carp (>100 mg/L), while sublethal concentrations in zebrafish embryos below those producing morphological abnormality altered gene expression results relevant to development, immunity, and metabolism.

2. Cardiovascular and Organogenesis Effects

Especially, the developing heart is a vulnerable target of the MP-associated embryotoxicity. Parental or direct embryonic MP exposures have demonstrated a consistent decrease in prototype heart rate in marine medaka progeny, which has persisted into larval stages. Computer-tailored fitness interventions are effective for physical activity and statin adherence in the Genie study On the other hand, possible contributing mechanisms include molecular dysregulation of cardiac development genes and activation of apoptotic pathways in cardiomyocytes.

Early exposure to MPs also influences hepatic development. Maternal exposure to PS-NP caused hepatic inflammation in the larvae, and this was in a size-dependent manner with smaller nanoparticles inducing neutrophil activation and hepatic inflammation, whereas larger nanoparticles acted via endoplasmic reticulum stress-mediated macrophage apoptosis. These pro-inflammatory responses may affect the hepatic metabolic capacity, hampering yolk processing, steroid hormone metabolism, and overall levels of larval fitness.

3. Neurodevelopmental and Locomotor Effects

Background Maternal exposure to polystyrene nanoparticles (NPs) has previously been reported to suppress locomotion in zebrafish offspring, however the underlying cellular and molecular mechanisms are unknown. Results Here we employ nano-chemical imaging technologies on their exposed-to-mothers but unexposed-to-fathers progeny to establish a mechanistic link between maternal plastic NP exposure, mitochondrial bioenergetics, and neurodevelopmental outcomes in zebrafish. Fish-larvae locomotion — based on light-dark challenge assays that are standardised — is an integrative endpoint sensitive to perturbation of neuromuscular development, brain myelination and visual system maturation outcomes. Defects in larval swimming behaviour have cascading impacts on prey consumption, predator evasion and ultimately juvenile survival and recruitment.

Zebrafish embryos exposed to MP particles containing bacterial biofilms showed additional behavioural, developmental and transcriptomic effects compared with clean MPs beyond those of the associated bacteria — showing how plastisphere colonisation by local microbial communities increases complexity over non-co-stimulated exposures. In larval stages, MP exposure led to transcriptomic disruption of pathways regulating immune function and oxidative stress response, steroid biosynthesis and neurotransmitter metabolism indicating wide-ranging disturbance of developmental gene regulatory networks. (Wang *et al*., 2021; Zhou *et al*., 2023) [3, 9]

Behavioural Alterations Affecting Reproductive Success

1. Courtship and Mate Selection

Successful fish reproduction depends on both physiological reproductive capacity and performance of complex reproductive behaviours that include territorial defence, nest building, courtship displays, and mate choice to encounter and select reproductively compatible partners. Neuroendocrine pathways involving the regulation of these behaviours are modulated following EDC exposure. (Carter & Ward, 2024[5]; McCormick *et al*., 2020)

Exposure of fathead minnows (P. promelas) to MPs associated with the synthetic oestrogen 17α-ethinylestradiol (EE2) caused significant impairment of female mate choice in a study recognised as landmark, where unexposed females non-significantly discriminated against males exposed to every combination except the highest concentration MP/lowest EE2 treatment. The overall strength of male courtship expression—that is, the quantity of males exhibiting exposed—was not significantly altered, but the diversion of female preference away from exposed males represents a functionally significant reproductive dysfunction with direct repercussions for fitness in nature. This study is the first assessment of how MPs, by themselves or as a vector for EDCs, can affect these multi-faceted mate choice dynamics in males and females in fish. (Carter & Ward, 2024[5]; McCormick *et al*., 2020)

Duration of MP exposure disrupts multiple neuroendocrine pathways to impair courtship. The mechanistic processes driving behavioural changes after MP exposure are modified dopaminergic and serotonergic neurotransmitter levels that influence motivation components of reproductive behaviour such as sexual desire and courtship. The rupture of the kisspeptin-GnRH signalling cascade that gates seasonal reproductive behaviour may additionally disrupt the synchronisation of male courtship with female receptivity.

2. Spawning Behaviour and Nest Fidelity

Multiple studies demonstrated decreased courtship intensity, altered mate choice and reduced spawn motivation after exposure to MPs in different fish species. Fish may spawn less, have shorter spawning seasons or fail to start spawning at all in the presence of suitable environmental cues. The physical effects of gut fullness from MP ingestion may reduce time and energy allocated to reproductive behaviour, changing how much metabolic investment is made in reproduction or digestion and immune responses triggered by particle-induced intestinal injury. (Carter & Ward, 2024[5]; McCormick *et al*., 2020)

In species with parental care including nest-guarding cichlids and salmonids, the neuroendocrine disruption induced by MP may lead to reduced quality of nesting behaviour or parental investment (indirectly reducing offspring survival) even though fertilisation and hatching rates remain unchanged. While the ecological component of MP reproductive toxicity has been investigated systematically, the social dimension broadly remains an area for improvement.

3. Anxiety-like and Antipredator Behaviour

Mechanisms of neuroendocrine and neurotoxic actions alter locomotor activity and anxiety-related behaviour in fish

after exposure to MP Tagline: Cortisol — the principal teleost stress hormone — increases in response to MP exposure, which may suppress reproduction by inhibiting GnRH secretion and serving as a mechanism for mediating a trade-off between stress and reproduction. MNP exposure induces neurobehavioural changes and direct reproductive toxicity in zebrafish behaviour, by which the two dimensions of toxicity may interact through shared neuroendocrine pathways. Impaired predator avoidance — an effect that has been documented frequently at the neurobehavioural level in MP-exposed individuals — may also diminish adult survival, which ultimately would impair reproductive success at the population level.

Transgenerational and Epigenetic Effects

1. Evidence for Transgenerational Reproductive Impairment

Transgenerational effects occur when environmental exposures to parents prior to fertilisation influence development, physiology or reproductive capacity in the offspring. For MPs, the evidence base of transgenerational reproductive impairment is emerging although not yet robust- nor fully consistent between species and exposure conditions. (Wang *et al*., 2019; Zeng *et al*., 2024) [2, 17]

The marine medaka case: Parental exposure of marine medaka exposed for 60 days to MPs under laboratory conditions at 20 µg/L produced significant effects in generation F1, specifically a decrease in the hatching rate at day one, while larval heart rate and standard body length were also monitored. Increasing the exposure to 150 days resulted in similar F1 effects with indications of growth and reproductive inhibition. Parental exposure to MPs was associated with reduced F1 survival, immunological, developmental, and locomotor impairment in salmonids. Zebrafish embryos co-exposed to polystyrene MPs and arsenic had significant offspring effects compared to either stressor alone, including an increased F1 mortality rate (67·4 ± 10·0 vs. 11·5 ± 2–6%), decreased hatching rate (60·3 ± 15% vs. 20%±0), and reduced heart rate (135bpm versus164 bpm in control).

On the other hand, a controlled rodent study of transgenerational inheritance in zebrafish following exposure to environmentally-relevant concentrations (10 µg/L) of small (1 µm) PS-MPs for 21 days demonstrated and underestimated or no subsequent manifestations of transgenerational effects on these endpoints, suggesting that specific reproductive endpoints may express transgenerational effects depending on the duration and type of parental exposure. This highlights the importance of longer-term multi-generational investigations and broader origins of reproductive endpoints. (Wang *et al*., 2019; Zeng *et al*., 2024) [2, 17]

2. Mechanisms of Transgenerational Transmission

Transgenerational mechanisms mediating MP effects at the cellular and molecular state operate through two general tiers. Firstly, maternal transfer of either MP particles or chemicals leached from them to the eggs — as evidenced by the detection of MPs in yolk-sac stage larvae before feeding — provides a route for toxicants directly into developing embryos. Direct ovarian effects of polystyrene (PS) NPs have been shown in female zebrafish to reduce circulating levels of 17β-estradiol (E2) and testosterone, with downstream impacts on yolk composition and embryo programming. (Wang *et al*., 2019; Zeng *et al*., 2024) [2, 17]

Second, and more mechanistically significant, MP exposure results in epigenetic changes in parental germ cells that are heritable over multiple generations without the need for sustained exposure. The epigenetic modifications include: (i) changes in DNA methylation — MP exposure causes loss of DNA methylation [hypomethylation] in zebrafish, mediated by reduced activity of different members of the DNMT family,[9] while up regulation may occur at specific imprinted genes and/or specific transcription factors involved with several developmental processes;8[11], decreased DNMT expression has been correlated with developmentally relevant biological pathways impacted by MPs;[8]; (ii) histone modification — following MP exposures marine invertebrates show increased Histone 3 (H3) mRNA expression,[13] indicating possible events of mitotically and meiotically heritable chromatin remodeling which will be explored further using fish models;[16]. (Im *et al*., 2022; Santos *et al*., 2022) [12]

Epigenomic studies in fathead minnow have revealed both dose-dependent and sex-specific changes in DNA methylation patterns in liver tissue after MP exposure, with male fish having more hepatic methylation alterations than females. Another important question and future direction is to what extent gonad-specific methylation changes, which would mediate transgenerational effects most directly via gamete epigenome, track metabolic perturbations in the hepatic genes? AbstractMelatonin supplementation prevents transgenerational nanoplastic toxicity and indicates antioxidant and anti-inflammatory pathways for epigenetic protection with implications for mitigation strategies in zebrafish. (Im *et al*., 2022; Santos *et al*., 2022) [12]

3. Ecological and Population-Level Implications

Ecological consequences of transgenerational reproductive impairment This influence is particularly strong: the effects have implications beyond individual fitness. If such parental MP exposure impairs the reproductive performance of sme F1 individuals — even in the absence of continued contamination until reproduction, MP pollution could maintain population-scale reproductive depression long after ambient concentrations recollected. In long-lived iteroparous fish (e.g., salmonids, clupeids, and cyprinids) that depend on accruing number of reproductive seasons for lifetime fitness those cumulative transgenerational effects can severely depress population growth rates and may lead to recruitment failure in heavily contaminated systems. (Wang *et al*., 2019; Zeng *et al*., 2024) [2, 17]

The underexplored interactions of MP transgenerational effects with other anthropogenic stressors, such as climate change (temperature, acidification), habitat degradation, overfishing and chemical pollution are likely to be synergistic. Reproductive endpoints are integral to ecological assessments of chemical risk, but transgenerational reproductive endpoints for MPsare not currently routinely integrated into environmental regulatory frameworks, resulting in an important missing element for protecting future generations of ecosystems. (Wang *et al*., 2019; Zeng *et al*., 2024) [2, 17]

Summary of Key Findings

1. Selected Studies: Species, Exposure, and Reproductive Outcomes

Table 1: Representative studies illustrating the diversity of microplastic types, fish species, exposure regimes, and reproductive endpoints affected. PS-MPs = polystyrene microplastics; GSI = gonad somatic index; VTG = vitellogenin; HPG = hypothalamic–pituitary–gonadal; EE2 = 17α-ethinylestradiol. (Huang *et al*., 2022; Gupta *et al*., 2023) [1, 15]

Species	MP Type / Size	Exposure	Key Reproductive Effect	Reference
Zebrafish (D. rerio)	PS-MPs, 1–5 µm	50–500 µg/L, 21 d	↓ GSI, ↓ fecundity, ↓ VTG, altered HPG gene expression	Gupta *et al*., 2023 [1]
Marine medaka (O. melastigma)	PS-MPs, 10 µm	20 µg/L, 60–150 d	↓ hatching rate, ↓ heart rate, ↓ offspring body length (F1)	Wang *et al*., 2019, 2021 [2, 3]
Zebrafish (D. rerio)	PS-MPs + As (co-exposure)	Chronic	↑ offspring mortality, ↓ hatching rate, ↓ heart rate in F1	Reproductive toxicity study, 2025
Japanese medaka (O. latipes)	PS-MPs	Chronic adult	Dose-dependent ↓ male fecundity, impaired spermatogenesis	Zhu *et al*., 2020 [7]
Fathead minnow (P. promelas)	MPs + EE2 (50 ng/L)	30 d dietary	Impaired female mate choice; epigenomic changes in liver	Carter & Ward, 2024 [5]
Atlantic cod (G. morhua)	Weathered PE-MPs	Broodstock exposure	↓ fecundity, ↓ egg quality, impaired sexual maturation	Environ. Pollut., 2023
Guppy (P. reticulata)	MPs	Chronic	↓ pregnancy rates, ↓ embryo counts, altered SOD/CAT	Malafaia *et al*., 2022 [8]
Climbing perch (A. testudineus)	PS-MPs	Sub-chronic adult	↓ LH, FSH, testosterone, estradiol; disrupted steroidogenesis	Ecotoxicology, 2025
European whitefish (C. lavaretus)	Nanoplastics	Parental exposure	↓ sperm viability; altered offspring phenotype	Yaripour *et al*., 2021 [10]

2. Mechanisms of Microplastic Reproductive Toxicity

Table 2: Primary molecular and cellular mechanisms through which microplastics impair fish reproduction, with associated molecular targets and reproductive outcomes. ROS = reactive oxygen species; SOD = superoxide dismutase; CAT = catalase; GPX = glutathione peroxidase; GST = glutathione S-transferase.

Mechanism	Key Molecular Targets	Reproductive Outcome
HPG Axis Disruption	GnRH, FSHβ, LHβ, GTHα, GnRHR	↓ steroidogenesis, reduced gonad function
Oxidative Stress (ROS)	SOD, CAT, GPX, GST, GSH	Gonadal apoptosis, DNA damage in germ cells
Estrogenic Mimicry / EDC Vectoring	ER-α, ER-β, AR-α, VTG, CHG	Feminisation, VTG in males, reduced egg quality
Epigenetic Reprogramming	DNMT3a/b, miRNA, lncRNA, H3 modifications	Transgenerational phenotypic changes in F1/F2
Neuroendocrine Disruption	Dopamine, serotonin, cortisol, kisspeptin	Altered courtship, spawning failure, anxiety-like behaviour
Physical Gonadal Damage	Seminiferous tubule structure, folliculogenesis	Histopathology, impaired gametogenesis
Mitochondrial Dysfunction	mtDNA integrity, ETC complexes, ATP synthesis	Reduced sperm motility, offspring locomotion deficits

Knowledge Gaps and Future Research Priorities

Despite substantial advances in understanding how MPs impair fish reproduction, critical knowledge gaps limit the translation of experimental findings into robust risk assessments and regulatory action. The following represent priority areas for future investigation:

Table 3: Key knowledge gaps in understanding microplastic impacts on fish reproduction and suggested research approaches.

Research Gap	Suggested Approach
Nanoplastics (<1 µm) reproductive toxicity poorly characterised	Develop standardised NP synthesis and exposure protocols; use multi-omic endpoints
Environmentally realistic multi-generational exposures lacking	Conduct F1–F3 chronic exposures at field-relevant concentrations (ng/L–µg/L)
Mixture toxicity with co-contaminants (heavy metals, PAHs, pharmaceuticals) understudied	Factorial dose–response designs with common co-occurring pollutants
Sex-specific and species-specific responses not fully compared	Parallel male/female exposures across diverse teleost taxa
Epigenome-wide association studies (EWAS) scarce in fish	Apply WGBS and ATAC-seq to gonads of exposed fish and offspring
Field validation of laboratory findings limited	Couple wild fish reproductive biomarker surveys with MP tissue burden quantification
Regulatory thresholds for reproductive endpoints absent	Develop species sensitivity distributions (SSDs) using reproductive NOEC/LOEC data
Behavioural reproductive endpoints (mate choice, spawning) understudied	Implement standardised behavioural assays in regulatory test guidelines

Beyond the targeted priorities identified above, several overarching methodological needs merit emphasis. Standardisation of MP exposure protocols — including particle characterisation methods, exposure routes, and reproductive endpoint selection — is essential to enable meaningful cross-study comparisons. The application of multi-omic approaches (transcriptomics, proteomics, metabolomics, and epigenomics) to MP-exposed fish gonads and offspring would provide mechanistic resolution currently lacking from endpoint-specific studies. Integration of laboratory findings with field-based reproductive biomarker surveys in contaminated watersheds is urgently needed to validate ecological relevance.

Conclusions

This review integrates a rapidly evolving body of evidence indicating that microplastic pollution represents an urgent and complex threat to the reproductive health of fish. Laboratory studies across a diverse array of teleost species offer converging evidence for a few overarching conclusions:

- MPs induce testicular and ovarian toxicity accompanied by histopathological damage, increased germ cell apoptosis, and dysfunction of gametogenesis that are primarily mediated by oxidative stress due to excessive ROS production. (Wang *et al*., 2021; Zhou *et al*., 2023) [3, 9]
- MPs alter the HPG axis at multiple levels — hypothalamic GnRH signalling, pituitary gonadotropin secretion, gonadal steroidogenesis — lowering sex steroids in circulation, decreasing vitellogenin and interfering with oestrogen receptor signalling. (Liu *et al*., 2023; Lin *et al*., 2023) [4, 14]
- Impaired sperm quality (motility, viability, morphology, DNA integrity) and female fecundity (egg production, fertilisation rate, egg quality) are consistently detected in both freshwater and marine fish species with dose-dependent effects demonstrated across multiple model systems. (Yaripour *et al*., 2021; Zhu *et al*., 2020) [7, 10]
- Embryonic development is affected by direct environmental exposure to MPs and maternal transfer of particles and leached chemicals via reduced hatching success, cardiac dysfunction, skeletal deformity, and impaired larval swimming behavior.
- Reproductive behaviour — i.e. courtship, mate choice and spawning — is disrupted via neuroendocrine alteration of dopaminergic and serotonergic pathways, with reliable impacts on sex selection in fathead minnows reported. (Carter & Ward, 2024[5]; McCormick *et al*., 2020)
- Transgenerational effects, including decreased F1 hatching and growth as well as cardiovascular dysfunction associated with maternal particle transfer as well as gamete epigenome reprogramming (via DNA methylation, histone modification and non-coding RNA) or by hormonal disruption via altered yolk composition. (Im *et al*., 2022; Santos *et al*., 2022) [12, 30]

Collectively, these findings demonstrate that MPs are not just physical pollutants, but functional endocrine disruptors, genotoxicants and epigenetic modifiers capable of degrading the reproductive fitness of fish over multiple generations. Understanding the reproductive impacts of MP pollution on fish, as well as finding approaches to mitigate such impacts, should be a scientific priority and an environmental governance imperatives in light of global concern over biodiversity loss and sustainability of freshwater and marine ecosystems.

Long-duration multi-generational studies at environmentally realistic concentrations, reproductive toxicology of nanoplastics and mixed plastic-pollutant exposures, field validation of laboratory mechanistic findings and reproductive-endpoint-based regulatory thresholds to support international governance of plastic pollution must be prioritised in future research. Generating standardised guidelines for reproductive toxicity testing with MP-specific exposure protocols, epigenetic endpoints, and behavioural assays would be a reshaping step forward in regulatory ecotoxicology.

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