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CHAPTER 3

Microplastic Pollution and Fish: Occurrence, Toxicological Mechanisms and Implications for Aquaculture and Human Health

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Abstract: Microplastics, plastic particles smaller than 5 mm, are now among the most widespread contaminants of aquatic ecosystems, and fish positioned across all trophic levels, serve both as sentinels of this contamination and as a route through which it reaches human consumers. This chapter synthesises recent literature, with emphasis on work published between 2024 and 2026, on the occurrence of microplastics in fish across freshwater, marine and South Asian systems, their exposure pathways and tissue-specific accumulation, and their toxicological consequences at the organism, cellular and molecular level. Field surveys document widespread ingestion, with the gastrointestinal tract as the principal reservoir and secondary accumulation in gills, liver, muscle,

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heart and brain, and emerging evidence of trophic magnification in oceanic food webs. Toxicological evidence links exposure to intestinal and hepatic damage, gill hyperplasia, haematological and immune alterations, neurotoxicity, oxidative stress, gut dysbiosis and impaired growth and reproduction, with severity governed by particle size, polymer type, concentration and exposure duration rather than by exposure route alone. Early life stages are especially vulnerable, and the sub-100 nm fraction, nanoplastics, shows greater bioavailability and more systemic toxicity than microplastics. Microplastics further act as vectors for heavy metals, persistent organic pollutants, antibiotic resistance genes and pathogens, extending the hazard into food safety for the growing share of aquatic protein supplied by aquaculture, whose own infrastructure and feeds contribute to contamination. The chapter closes with detection and standardisation challenges, mitigation strategies spanning policy, technology and nutrition, and the research gaps that must be closed to protect fish populations, aquaculture productivity and consumer health.

Keywords: Nanoplastics; oxidative stress; bioaccumulation; trophic transfer; food safety

1. Introduction

Microplastics, commonly defined as plastic particles smaller than 5 mm, have become a persistent environmental pollutant in aquatic ecosystems worldwide. They are either the result of fragmentation of larger plastic debris or from manufactured consumer products such as exfoliants and cleaning agents[1]. Because of their biochemical stability, small size, highly reactive nature, they are readily ingested by many aquatic organisms, and their resemblance to zooplankton and other food particles makes fish particularly susceptible to ingestion[1,2]. Fishes occupy a central position in transfer of these particles through aquatic food chains towards humans[3,4].

The scientific interest in this topic has grown rapidly. A systematic global review of microplastic contamination in fish documented a drastic increase in the corresponding scientific publications. Inter-disciplinary studies from all over the world have been published by scientists from countries such as China, India and Italy. The focus of corresponding studies has shifted from pure toxicology to molecular biology and the impact on ecosystems as well as to human health[5]. Aquaculture is simultaneously a victim and a contributor: the industry increasingly relies on plastic-based infrastructure and materials, creating multiple routes of contamination within culture systems, while farmed fish themselves are documented to contain microplastics, forming an intake pathway for consumers[6,7].

This chapter is a review of the current state of knowledge on the occurrence of microplastics in fish, with emphasis on literature published between 2024 and 2026. It presents no original experimental data and is intended as a synthesis for students and researchers in aquatic toxicology, fisheries science and aquaculture.

2. Sources, types and environmental occurrence of microplastics

2.1 Sources and polymer types

There are two main pathways in which microplastics become introduced to aquatic environments. Primary microplastics are manufactured at a small scale and then released to the environment through various products and industries. Secondary microplastics are the result of degradation of larger items of plastic debris through processes such as ultraviolet radiation and mechanical abrasion[1]. Aquatic ecosystems additionally receive primary and secondary particles from urban runoff, industrial waste and degraded plastics, with surface waters and sediments acting as the main accumulating compartments[2].

In terms of polymer identification, most studies consistently point to a limited set of materials. For example, in syntheses of studies on wild freshwater fish, the most frequently identified particles were polyethylene and polypropylene, typically as blue fibres of 0.1 to 0.5 mm[8]. Moreover, studies carried out on farmed Nile tilapia, found that fibres and fragments found in fish were made of polyethylene, polypropylene and polyethylene terephthalate. The sources of these particles were partly external such as wastewater, urban activities and textiles, along with internal sources such as wear of netting, pipes and equipment and contaminated feed[9]. In a case study carried out in aquaculture ponds in Bangladesh, fibres made of 60.86% of water-borne particles were found in the water, with identified polymers including polyethylene, polypropylene, nylon, polyvinyl chloride (PVC) and acrylic[10].

2.2 Occurrence in freshwater and marine fish

Field surveys demonstrate that microplastic ingestion by wild fish is widespread across continents and habitats. A review of 144 articles published since 2016 documented contamination of 450 wild freshwater fish species across 45 countries, with studies focussed in China, Bangladesh, India and Indonesia, and the most studied order were the Cypriniformes. The predominant sampling sites were the rivers and majority of species studied were classified as omnivores. Notably, 35 of the contaminated species are listed on the IUCN Red List, including 2 critically endangered, 8 endangered and 12 vulnerable species.

In the Amazon basin, more than 60% of analysed fish from Lake Janauacá and the Anavilhanas Archipelago contained microplastics in their gastrointestinal (GI) tracts, with 91.5% of particles located in intestine and the remainder in stomach, with no significant differences among feeding habits or sampling locations[11]. In a tropical bay in Brazil, 20.01% of 1,339 individuals across 19 species were contaminated, with particles present in all trophic guilds with highest ingestion in piscivorous species, indicating contamination even in less populated areas[12]. Along an urbanisation gradient in the Suzhou River, Shanghai, 71.29% of 310 fish across 10 species contained suspected microplastics with significantly higher abundance in core urban and intermediate urban reaches than in suburban reaches, and with small, transparent fibres of 1 mm or less predominating[13]. In Lake Tana, Ethiopia, microplastics were detected in 69.5% of samples from 3 commercially important species, with ingestion rates varying by feeding ecology. The omnivorous African catfish showed the highest average contamination of 4.92 particles per fish, the phytoplanktivorous Nile tilapia 2.17, and the herbivorous *Labeo barbus* species only with 0.17, with polyethylene, polypropylene and polyethylene terephthalate as the primary polymers[14].

Marine and oceanic systems show the same pattern, and evidence of biomagnification is emerging. In the Laccadive Sea of the Western Indian Ocean, 95% of samples spanning trophic levels from zooplankton to top predators contained microplastics. A significant positive correlation between trophic level and gut particle abundance and a trophic magnification factor of 4.4 indicated biomagnification within the oceanic food web. Top predators such as swordfish carried the greatest loads, at 832.25 particles per individual on average, and polypropylene and polyethylene dominated pelagic species while high-density polymers such as polyurethane and polyethylene terephthalate dominated bathypelagic taxa[15]. A study of commercially important fish from coastal and offshore waters likewise documented significant interspecies differences in accumulation, attributable to feeding strategy, habitat depth and trophic position, with filter feeders and omnivorous species carrying higher burdens[16].

2.3 Microplastics in South Asian waters

South Asian freshwater systems, which support enormous fishing and aquaculture activity, are increasingly documented as contamination hotspots. A synthesis of Indian freshwater environments reports microplastic contamination in the Ganga, Yamuna, Gomti, Saryu, Brahmaputra, Mahanadi, Cauvery, Netravathi, Periyar rivers and Vembanad Lake, with fibres and fragments as the dominant particle types and polyethylene, polypropylene, polyethylene terephthalate and polystyrene as the predominant polymers, and with contamination concentrated in highly urbanised and industrialised river stretches[17]. Another review of Indian rivers and lakes describes negative ecotoxicological

impacts on aquatic organisms, including bioaccumulation, oxidative stress, histopathological alterations, decreased growth rates and altered behaviour[18].

Species-level data confirm exposure of Indian food fish. In the river Yamuna, microplastics were detected in all 220 fish examined across 12 species spanning multiple trophic guilds, with 1,678 particles recovered and significantly higher abundance in the highly urbanised Delhi stretch than upstream. The GI tract held the greatest load (751 particles), followed by gills (605) and muscle (322), fibres dominated at 77.8%, and polymer hazard index analysis classified the recovered particle mix as a high ecological hazard category[19]. In the river Ganga at Varanasi, microplastics were detected in the GI tracts of approximately two-thirds of 62 fish from four commonly consumed species and in the muscle of 15%, with the highest GI concentration reaching 19.04 items per gram, and polymer hazard index assessment categorised roughly 37% of the detected polymers as hazardous, highlighting a potential pathway of human exposure through edible tissues[20].

Occurrence is equally documented in farmed fish elsewhere in the region. In the aquaculture ponds of western Bangladesh, fish showed an average microplastic concentration of 1.19 items per gram, equivalent to 23.37 items per individual, with gill concentrations of 1.05 to 5.04 items per gram exceeding GI concentrations of 0.40 to 2.26 items per gram across 11 species, and with fibres detected in 100% of fish samples; strong correlations between fish size and microplastic abundance indicated that environmental contamination of the ponds is a significant driver of ingestion[10].

3. Exposure Pathways and Tissue-Specific Accumulation

3.1. Routes of exposure

Fish are exposed to microplastics via their environments and their diet. Ingestion is the principal exposure route, with gill uptake representing an additional pathway, particularly for juvenile fish during foraging[2,6]. The characteristics of particles matter because microplastics resemble zooplankton and other prey items, thereby facilitating ingestion[2]. Larvae are non-selective feeders and mistake size-matched particles for their natural food which promotes accumulation of these particles[2]. Aquaculture adds internal sources, since plastics are used in pipelines, filters, tanks and netting components that gradually degrade during operation, and since feed ingredients, including fishmeal derived from microplastic-exposed fish, can themselves carry particles[6,21].

3.2. Tissue-specific accumulation

Once inside the body, microplastics do not remain confined to the digestive tract. After ingestion, particles have been detected in gills, muscles, liver, heart, swim bladders, ovaries, spinal cords and even brains, indicating infiltration of the circulatory and lymphatic systems and distribution to

multiple organs[1]. A synthesis of 78 field and laboratory studies on freshwater fish established the GI tract as the organ of highest accumulation, with a real-world mean abundance of 6.57 items per gram and an artificial-exposure bioconcentration factor of 37.7, confirming oral ingestion as the dominant source of the microplastic burden; the same framework identified particle characteristics such as colour, shape and size, fish biology such as feeding habits, gill structure and reproductive strategies, and environmental conditions such as water flow velocity and turbidity as the key factors influencing pathway-specific accumulation[22].

Field data reflect this tissue hierarchy. In wild freshwater fish, the GI tract accounts for 51.10% of records, gills for 19.38% and muscle for 10.57%[8]. The Yamuna study reproduced the same ordering at the level of absolute particle counts, with the GI tract, gills and muscle carrying 751, 605 and 322 particles respectively[19]. In farmed tilapia, most studies document GI accumulation with additional detection in gills, liver and muscle, the latter raising potential human-health concerns[9]. Table 1 summarises the principal tissues of accumulation and associated effects.

Table 1. Principal tissues of microplastic accumulation in fish and their associated effects.

Tissue	Reported accumulation/effect
Gastrointestinal tract	Dominant site of accumulation (51.10% of wild freshwater records; mean 6.57 items/g); mucosal damage, blockage, dysbiosis
Gills	Hyperplasia, excess mucus, impaired oxygen exchange; concentrations up to 5.04 items/g in farmed fish
Liver	Hepatic damage, altered enzyme activity
Muscle	Detection raising consumer-safety concerns
Heart, blood and circulatory system	Cardiotoxicity, altered haematological indices
Brain and nervous system	Neuroinflammation, altered acetylcholinesterase activity
Gonads	Steroidogenesis disruption, reproductive dysfunction

3.3. Modulating factors

The severity of accumulation and toxicity is governed less by the route of exposure than by particle size, dose, exposure duration, food consumption and species[23]. Toxicity scales with particle size, type, concentration and duration of exposure, with smaller particles showing greater tissue penetration and bioavailability[2,24]. Interactions with co-occurring contaminants may further modify biological effects[6], and exposure is amplified in polluted, highly urbanised waters[13].

4. Toxicological Effects at the Organism and Cellular Level

4.1. Gastrointestinal tract and liver

The digestive system bears the first impact of ingested particles. Exposure damages hepatic and gut tissue, impairs intestinal barrier function and induces dysbiosis of microbial composition, altering host metabolism and the activity of digestive enzymes and ultimately affecting growth performance[23]. At the organ-system level, intestinal and hepatic damage, disrupted digestive enzyme activity, altered gut microbiota and digestive tract dysfunction are consistently reported, with microplastic accumulation in the gut and gills followed by bloodstream penetration to internal organs[24]. In aquaculture-relevant species, altered digestive enzyme activity and impaired feeding and feed utilisation translate contamination into production-relevant outcomes[6].

4.2. Gills and the respiratory system

Accumulation of plastics in the gills induces hyperplasia, excessive mucus secretion and impaired oxygen exchange, compromising the primary respiratory surface and creating a direct pathway to the bloodstream[24]. Gill tissue damage and altered ion regulation have been documented, and gill concentrations in farmed fish frequently exceed those of the GI tract, indicating substantial branchial uptake from contaminated water[10].

4.3. Haematology, immunity and oxidative stress

The circulatory system is affected by microplastic bioaccumulation in tissues, influencing haematological indices connected with immunity, osmotic pressure, blood clotting, molecular transport and fat metabolism[23]. Immune responses are altered through physicochemical damage, and exposure activates pro-inflammatory reactions with increased expression of immune proteins[23,24]. Oxidative stress emerges as a central, though not exclusive, mechanistic pathway: microplastics create an imbalance between reactive oxygen species generation and antioxidant defence, producing oxidative injury that links to inflammation, immunotoxicity and further downstream effects[6,23]. When defence capacity is exceeded, lipid peroxidation, protein carbonylation and DNA damage follow, alongside neurotoxicity expressed as altered acetylcholinesterase activity[23].

4.4. Neurotoxicity and behaviour

Upon reaching the nervous system, microplastics cause neurotoxicity, oxidative stress, cellular damage and neuroinflammation[24]. Altered acetylcholinesterase activity is a recurring biomarker of neurotoxic effect[23]. At the whole-animal level, waterborne microplastics belong to the chemical

pollutants that modify swimming, schooling, feeding, anti-predator and anxiety-like behaviours, providing sensitive readouts of sub-lethal stress with consequences for welfare and production[25].

4.5. Growth, endocrine disruption and reproduction

Microplastic exposure acts on the hypothalamic-pituitary-gonadal axis, disturbing steroidogenic pathways and promoting apoptotic processes in gonadal tissue, with impared reproductive function as the outcome[23]. Reproductive, neurological, hormonal and immune disruptions are documented across organ systems[1], and in aquaculture settings reduced growth and impaired feed utilisation are recurring outcomes[6]. The consequences can extend across generations, with transgenerational effects, metabolic disruption and behavioural alteration reported as longer-term hazards of exposure[26].

5. Effects on Early Life Stages

Fish embryos, larvae and juveniles are more susceptible to microplastics than adults because their physiological and detoxification systems are immature[2]. Exposure pathways are stage-specific. Embryos are exposed primarily through maternal transfer and adhesion of particles to the permeable chorion, leading to aggregation on the egg surface that can impair hatching and development, whereas larvae face heightened ingestion risks as particles mimic zooplankton prey and cause gut accumulation, physical blockage and reduced feeding, and juveniles exhibit ingestion and gill uptake with tissue distribution to gut, liver and muscle[2].

The consequences are measurable. Physically, ingested particles can obstruct the digestive tract, lowering hatching success and disrupting organ development; chemically, plasticisers, leached, leached heavy metals and pollutants adsorbed onto particle surfaces generate oxidative stress, endocrine disruption and metabolic dysfunction. Exposed fish also show poorer swimming performance and reduced predator avoidance, lowering survival[2]. Higher microplastic concentrations correlate with reduced hatching rates, increased mortality, stunted growth and amplified developmental issues[2]. Surface adhesion on embryos can block oxygen diffusion, inducing hypoxia, delayed hatching and malformations such as spinal and tail defects[2]. Because early life stages concentrate in shallow nursery habitats and spawning grounds, these habitats are priority zones for contamination monitoring[2].

A further ecological consideration is habitat type. Freshwater systems generally exhibit higher microplastic concentrations than marine environments, with reported surface-water levels of 1.8 to 2.4 pieces per litre in freshwaters against 0.9 pieces per litre in marine settings, implying that freshwater nurseries and estuarine hotspots deserve particular attention[2].

6. Nanoplastics: A distinct and more mobile hazard

Nanoplastics are particles below 100 nm which arise from the same fragmentation processes as microplastics but behave differently in biological systems[1]. A systematic review of 114 studies concluded that, relative to larger particles, nanoplastics adsorb contaminants more efficiently, present a far greater surface area for interaction with biological tissues and release associated co-contaminants more readily; accumulation has been documented in the skin, muscle, gill, gut, liver, heart, gonads and brain at every developmental stage, from embryos to adults[27]. Reported effects include increased oxidative stress, decreased locomotor and foraging activity, altered growth, immunity and lipid metabolism, induced neurotoxicity, modulation of estrogen-androgen-thyroid-steroidogenesis pathways and potential intergenerational effects[27]. Comparative assessments indicate that, while both particle classes are harmful, nanoplastics generally exert stronger and more systemic effects owing to higher bioavailability, cellular uptake and molecular reactivity. Microplastics primarily impose mechanical stress, whereas nanoplastics disrupt cellular homeostasis at lower exposure levels, often at the subcellular level, with size-, surface-chemistry and concentration-dependent toxicity[28].

Mechanistic reviews describe nanoplastic transport across biological barriers and intracellular cytotoxicity involving membrane disruption, reactive oxygen species production. DNA damage, lysosomal instability and mitochondrial depolarization, with implications extending through the food chain to human health[29]. Reviews of nanoplastic toxicity in aquatic organisms nonetheless caution that long-term effects of low-dose exposure and standardised testing methodologies remain critical gaps, and that human toxicokinetics and cumulative toxicity in combination with other pollutants are poorly understood[30]. In the context of aquaculture, micro- and nanoplastic toxicity mechanisms in farmed fish, including reduced feed utilisation, physical abrasion, oxidative stress, inflammation and gut microbiota disruption, are increasingly recognised as targets for mitigation[31].

7. Microplastics as Vectors of Chemical Contaminants and Pathogens

Beyond their intrinsic physical and chemical toxicity, microplastics act as carriers of other hazardous substances. Because particles adsorb and concentrate pollutants, microplastic ingestion can transfer persistent organic pollutants, heavy metals and pathogenic bacteria to fish tissue, and bioaccumulation of these co-contaminants has been linked to toxicological impacts in humans consuming affected seafood[5]. The capacity of particles to disseminate antibiotic resistance genes through aquatic food webs adds an antimicrobial-resistance dimension to microplastic risk[5]. The small dimensions and high chemical reactivity of microplastics increase their bioavailability, promoting tissue penetration and accumulation at multiple levels of food chain[2].

Interactions with heavy metals are now a substantial literature in their own right. A comprehensive review of 154 publications on microplastic-metal co-exposure in fish between 2016 and 2025 concluded that the two pollutant classes interact to form toxic mixtures that amplify harm across aquatic food webs. It was noted that research is heavily skewed towards polystyrene combined with cadmium or copper, that haematological, behavioural and genotoxic endpoints remain largely overlooked, and that advanced molecular techniques are rarely employed[32]. Field evidence supports co-occurrence. In 2 fish species from a polluted river in Iran, 79% of samples were microplastic-contaminated, and regression analysis revealed a significant positive correlation between the metal pollution index of muscle tissue and microplastic abundance in both species, with polymer hazard index classification placing polymers in the very high-risk category[33].

Experimental studies clarify the direction of these interactions. Polystyrene microplastics acted as a significant vector enhancing the bioaccumulation of methylmercury, but not inorganic mercury. In zebrafish embryos, the result of aggravating oxidative stress and disturbing lipid, amino acid and energy metabolism, demonstrated that the chemical form of the co-contaminant matters for combined toxicity[34]. Aging of particles amplifies the hazard. Aged polystyrene adsorbed more cadmium than virgin material and produced more severe liver damage through gut barrier disruption, altered gut microbial composition and gut-liver axis metabolic dysregulation[35]. In Nile tilapia fingerlings, 42-day co-exposure to polyamide microplastics and mercury increased mortality, suppressed growth and blood physiology, downregulated growth hormone and insulin-like growth factor gene expression, and produced severe structural deformities in gills, intestine, liver and kidney relative to single-exposure treatments[36]. Trophic transfer and biomagnification have been documented as broader ecological risks[3], and field evidence of trophic magnification in oceanic food webs is now available[15], although reviews caution that consistent biomagnification has not yet been universally established[6].

8. Microplastics in Aquaculture Systems

Aquaculture presents a distinctive contamination environment because plastic materials are embedded in production infrastructure. In recirculating and industrial systems, pipelines, filters, tanks and netting components gradually degrade during long-term operation, generating microplastic particles that enter cultured organisms through both water and feed, creating conditions favourable to accumulation within closed systems[21]. Reviews of aquaculture identify degraded equipment, contaminated water, atmospheric deposition and feed ingredients, including fishmeal derived from microplastic-exposed fish, as the primary entry routes[6].

A systematic review of 90 peer-reviewed publications concluded that both anthropogenic influences near aquaculture facilities and internal factors such as infrastructure, feed and animal behaviour determine contamination[7]. Microplastic abundance varies significantly by taxon but not by natural feeding preference or habitat, and that the widespread presence of microplastics in aquaculture animals constitutes a potential intake pathway for humans[7]. Species-specific syntheses confirm the pattern. In Nile tilapia, one of the most widely farmed freshwater species, 34 studies from 2016 to 2025 document global occurrence in natural, aquaculture and experimental systems, with fibres and fragments of polyethylene, polypropylene and polyethylene terephthalate predominating and physiological effects including digestive disturbances, oxidative stress and reduced growth[9]. Field data from Bangladeshi pond aquaculture corroborate the production-scale concern, recording high contamination levels in both water and fish and tracing contributions to agricultural runoff, vehicle tyres, fishing nets, fish feed, household waste and plastic feeding equipment[10].

These findings position microplastic exposure as a multidimensional stressor connecting environmental contamination with physiological dysfunction and impaired aquaculture performance, with implications for feed and farm management, fish health protection and food safety[6].

9. Human Health Implications through Seafood Consumption

Fish is a vital protein source consumed regularly worldwide, and contaminated fish therefore represents a direct route of microplastic exposure for consumers[4]. Following ingestion by humans, the small fragments can cause GI blockage, decreased appetite and transfer of harmful adsorbed materials into human tissue, alongside physical and chemical harms comparable to those documented in fish[4]. Evidence indicates that consumption of contaminated fish may be a significant route of microplastic ingestion for the general population, raising serious public health concerns[3].

The risk is compounded by the vector role of particles. Microplastics in seafood can carry persistent organic pollutants, heavy metals and pathogenic bacteria, and their role as carriers of antibiotic resistance genes adds a further hazard dimension[5]. Human bioaccumulation of ingested microplastics might contribute to decreased immune function or local particle toxicity[7], and consumer-facing comparisons of aquaculture-derived versus wild-caught sources highlight that contamination risk differs by production origin[5]. Regional data sharpen the concern for edible tissues. In Ganga fish, hazardous polymers were identified in edible muscle and GI samples[20], and in Yamuna fish the high polymer hazard index classification underlines the risk from consumed

species[19]. These considerations translate microplastic research directly into food-safety and consumer awareness[5,26].

10. Detection, Standardisation and Methodological Challenges

Reliable risk assessment depends on accurate detection and quantification, and this remains a methodological weak point. Reviews of tools and methodologies used to detect and quantify microplastics in fish highlight persistent challenges in achieving standardised and reliable results[3]. A systematic review of characterisation methods in fish concluded that Raman and Fourier-transform infrared spectroscopy are the most frequently employed techniques, with Raman offering greater sensitivity for particles smaller than 20 μm and FTIR providing reliable identification of larger particles, while stereomicroscopy and scanning electron microscopy offer limited chemical specificity. The same review recommended multi-analytical approaches and integrated biomarker assays for ecological risk assessment[37]. Broader methodological reviews catalogue the full analytical toolbox, including pyrolysis gas chromatography-mass spectrometry, fluorescence-based approaches, biosensors, electroanalytical techniques, microfluidic systems and emerging laser direct infrared spectroscopy, and stress that sample preparation, matrix interference, nanoplastic detection, spectral databases and method standardisation remain unsolved challenges[38,39].

In wild-fish research, significant gaps include standardised methods, broader inclusion of Neotropical fish species, investigation of biological responses and studies of trophic transfer and biomagnification[8]. Standardised frameworks from international marine pollution bodies are recommended to promote comparable metrics combining particle number and mass, and polymer confirmation by techniques such as Fourier-transform infrared spectroscopy is increasingly treated as the verification standard in field studies[2,10].

11. Mitigation Strategies

Mitigation operates at several levels. At the source level, reductions in plastic use, improved wastewater treatment and development of biodegradable plastic alternatives address the generation of microplastics at its origin[26]. At the policy level, stringent regulatory policies, public awareness campaigns and strong institutional frameworks are advocated to limit contamination and protect aquatic biodiversity[3,26]. At the technology level, emerging remediation techniques and bioremediation research are highlighted as priorities for future development[3,26]. Within aquaculture, effective responses include improved feed safety, plastic waste management and system design that limits plastic-water contact, integrated into biosecurity and food-safety programmes[6,7].

A promising addition to this portfolio is nutritional mitigation. A recent synthesis of micro- and nanoplastic toxicity in farmed fish identified targeted nutritional interventions, including macronutrient optimisation to balance metabolism, fortification with micronutrients such as vitamins, selenium and zinc to enhance antioxidant defences, and functional additives such as probiotics, astaxanthin and microalgae that reduce particle absorption, oxidative damage and inflammation, while directing future research towards precision nutrition and microecology modulation[31]. Coordinated action among scientists, policymakers and industry stakeholders is repeatedly identified as necessary to address this escalating environmental and health challenge[3].

12. Research Gaps and Future Directions

Several gaps limit current understanding. Long-term health effects of microplastic exposure remain insufficiently characterised, and more effective remediation techniques are needed[3]. Evidence for trophic transfer and biomagnification across food webs is incomplete: oceanic evidence of trophic magnification exists[15], but reviews caution that consistent biomagnification has not yet been universally established and that long-term, multigenerational and environmentally derived nanoplastic assessments are lacking[6,28]. Standardised toxicity assessment and testing protocols are needed to make results comparable across studies[26,30].

Geographic and biological coverage is also uneven. Wild freshwater fish are less extensively studied than marine counterparts[1], Neotropical species remain underrepresented despite high contamination records[8], and dose-response relationships under realistic farm conditions are uncertain[7]. In co-exposure research, most studies examine polystyrene with cadmium or copper under artificial conditions, leaving other particle-metal combinations, haematological, behavioural and genotoxic endpoints, and molecular mechanisms largely unexplored[32]. Nanoplastic research faces parallel gaps in environmental detection, chronic low-dose exposure, species coverage and human toxicokinetics[27,30]. Finally, the interactions of microplastics with co-occurring contaminants, including heavy metals and pharmaceuticals, and their consequences across multiple generations, require systematic investigation before ecological and human-health risks can be fully quantified[6,26].

13. Conclusions

Microplastic pollution is now documented in fish from tropical rivers to aquaculture ponds, with ingestion the dominant exposure route, the GI tract the principal reservoir, and the secondary accumulation in gills, liver, muscle and nervous tissue. The toxicological evidence, spanning intestinal and hepatic damage, gill hyperplasia, haematological and immune alterations, neurotoxicity, oxidative stress, gut dysbiosis and reproductive dysfunction, establishes microplastics

as a multidimensional stressor whose severity is governed by particle characteristics, dose and duration. Nanoplastics extend this hazard to the subcellular scale, and the field evidence of trophic magnification in oceanic food webs shows that the particles, and the contaminants they carry, move up the chain to top predators and consumers.

The role of microplastics as vectors for heavy metals, persistent organic pollutants, antibiotic resistance genes and the pathogens extend the hazard beyond the particles themselves and into food safety for the growing share of aquatic protein supplied by aquaculture. Closing the identified gaps, through standardised detection protocols, long-term and multigenerational exposure research, realistic co-exposure designs, broader geographic and taxonomic coverage and nutritional and technological mitigation, is essential if fish populations, aquaculture productivity and consumer health are to be protected. Until then, source reduction, improved wastewater treatment, biodegradable alternatives, stringent policy and coordinated international action remain the most credible lines of defence against this escalating environmental and public-health challenge[3,26].

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