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Ecotoxicological Implications of Microplastics on Aquatic Vertebrates: Disruption of Homeostasis and Regulatory Pathways

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Abstract: *Microplastics (MPs) and nanoplastics (NPs) have become ubiquitous contaminants across global aquatic environments, posing severe risks to aquatic vertebrates which serve as critical sentinel species for ecosystem health and human food security. This review synthesizes current literature (2015–2025) to move beyond simple ingestion documentation and analyze the sophisticated mechanisms by which MPs induce systemic homeostatic failure. The analysis highlights that toxicity is size-dependent, with nanoplastics (<100 nm) presenting enhanced risks due to superior mobility and sorption capacities. Key mechanisms identified include the "vector effect," where MPs transfer co-contaminants like endocrine-disrupting chemicals (EDCs) and heavy metals to biological tissues, significantly altering their bioavailability. This exposure triggers a cascade of physiological disruptions, primarily targeting the neuroendocrine system (HPG, HPT, and HPA axes), leading to reproductive toxicity and metabolic deregulation. Furthermore, MPs induce oxidative stress, immunotoxicity, and gut dysbiosis, which collectively compromise organismal fitness and survival. The review concludes that*

MP exposure represents a chronic, multi-modal assault on aquatic life, necessitating standardized risk assessment protocols and urgent policy interventions to mitigate these profound ecological impacts.

Keywords: *Microplastics (MPs), Nanoplastics (NPs), Aquatic Vertebrates, Ecotoxicology, Vector Effect, Endocrine Disruption, Homeostasis, Oxidative Stress*

I. Introduction

Microplastics (MPs), standardized as synthetic polymer particles measuring less than 5 mm in diameter, have been established as ubiquitous contaminants across all aquatic environments globally, including marine gyres, freshwater systems, tropical wetlands, and polar regions (Hossen et al., 2025). These particles originate from two major pathways: the breakdown and fragmentation of larger plastic debris, and the direct release of manufactured items such as microbeads and synthetic fibers (Pal et al., 2025). The magnitude of this contamination is underscored by industrial production trends. Global plastics output reached over 359 million tons (Mt) in 2018 (PlasticsEurope, 2019), continuing a significant expansion since the 1950s Projections indicate that plastic emissions into the aquatic environment are anticipated to range between 20 and 53 Mt/year by 2030 (Borrelle et al., 2020).

Within this broad class of contaminants, nanoplastics (NPs), defined as particles 100 nm or smaller, pose a unique and severe environmental risk due to their enhanced mobility and superior sorption capacities compared to larger MPs (Athey et al., 2020; Alimba & Faggio, 2019). While global concern has grown significantly, current knowledge remains fragmented and often restricted to specific geographic regions or ecological compartments. Therefore, a critical synthesis is required to systematically analyze MPs' dispersion patterns, ecological consequences, and, crucially, their intricate interactions with co-pollutants such as heavy metals, pharmaceuticals, and endocrine-disrupting chemicals (EDCs) (Alimi, n et al., 2018).

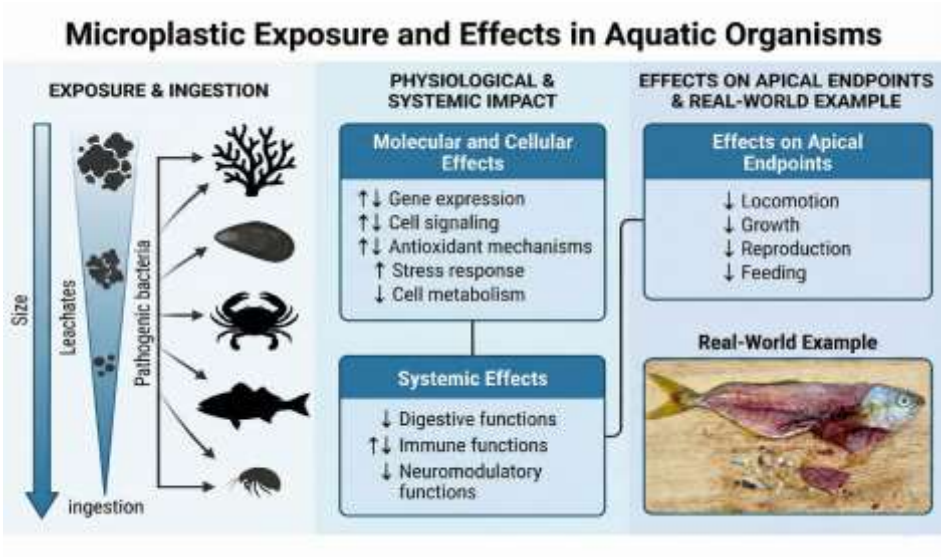
Aquatic vertebrates, particularly fish, serve as critical sentinel organisms in ecotoxicology, as they are among the first organisms to encounter waterborne emerging contaminants like MPs and associated EDCs their exposure pathway is highly relevant, given their ecological importance and their role in the human food supply. The contamination of fish by MPs constitutes a major hazard to human food security (Ghosh, 2025), establishing a direct and urgent link between environmental ecotoxicology and public health concerns (Jayasundara et al., 2020).

Over the past decade, the focus of microplastic research has undergone a significant evolution. Early reviews (pre-2018) were primarily dedicated to documenting distribution and prevalence (Yua et al., 2020). However, the scientific community has since matured its approach, shifting towards

investigating the sophisticated mechanistic consequences of exposure. Recent literature (2019–2025) emphasizes pathway-based risk assessment, focusing on cellular and physiological damage, including oxidative stress, inflammation, neurotoxicity, and hormone disruption (Pyambri et al., 2025). This shift reflects a recognition that the true hazard is systemic, driven by subtle molecular interference rather than just physical ingestion. Furthermore, given the density and environmental fluctuations in managed systems, the findings have immediate and critical implications for sustainable aquaculture practices, necessitating targeted mitigation strategies (Sun et al., 2021).

This review article systematically examines the ecotoxicological mechanisms by which microplastics induce the failure of systemic homeostatic controls and deregulate critical physiological pathways in aquatic vertebrates. (Multisanti et al., 2025). Moving beyond documentation of presence, this synthesis explores the molecular and cellular disruptions including oxidative stress, inflammation, and neurotoxicity that characterize MP exposure. Detailed attention is given to the disruption of key regulatory axes, notably the neuroendocrine, immune, and metabolic pathways, thereby providing a comprehensive overview of how MPs compromise internal balance in fish and other aquatic vertebrates. (Fioretto et al., al 2025)

Figure 1.1 Conceptual Framework of Microplastic-Induced Homeostatic Disruption and Systemic Toxicity in Aquatic Organisms.



2. Physicochemical Drivers of Microplastic Ecotoxicity: Size, Composition, and the Vector Effect

The hazard profile of microplastics is highly heterogeneous and contingent upon their intrinsic physical and chemical properties. These properties govern bioavailability, tissue translocation, and the efficacy

of chemical transfer, making them crucial determinants in predicting toxicological outcomes (Khaki et al 2025).

2.1. Bioavailability and Tissue Translocation

Microplastics are readily assimilated and ingested by a wide range of aquatic organisms across various trophic levels. While the gastrointestinal tract serves as the initial and often largest reservoir of MPs (Meena et al., 2023), the smallest particles are capable of crossing biological barriers and translocating to deep tissues. Studies on nanoplastics (NAPs) indicate that accumulation is not confined to the gut; NAPs are detected in the gills, liver, heart, gonads, and brain of fish. This accumulation is documented across all developmental stages, from larvae to adults, suggesting chronic and systemic exposure risks throughout the entire life cycle of aquatic vertebrates (Athey et al., 2020)

2.2. The Critical Role of Particle Heterogeneity

2.2.1. Size-Dependent Toxicity

Toxicity is fundamentally size-dependent, with particle dimensions strongly influencing the resulting biological impact. Nanoplastics (100 nm or smaller) exert the most complex and potentially harmful effects compared to larger microplastics (MIPs). (Zhang et al., 2020). This enhanced toxicity is mechanistically attributed to the superior physical attributes of NAPs they possess an extremely large surface area relative to their volume and exhibit greater absorption capacity. This allows NAPs to efficiently adsorb and subsequently release higher concentrations of co-contaminants into biological tissues, enabling them to cross critical barriers and affect central regulatory systems (Suarez, et al., 2020).

2.2.2. Polymer and Shape Influence

The chemical composition and morphology of microplastic particles introduce further variability in toxicity profiles Commonly studied polymers include Polystyrene (PS), Polyethylene (PE), and Polyvinyl Chloride (PVC) Polystyrene, for instance, is the most frequently encountered nanoplastic polymer in aquatic environments (89% of studied NAPs) and has been specifically linked to reproductive disruption and metabolic disorders (Lu et al., 2019).

Furthermore, particle shape influences localized ecological impact. In many environmental surveys of fish and shellfish, microplastics found are predominantly in fiber form, often accounting for the vast majority of contamination (up to 99.23%) (Asadi et al., 2022). These high-aspect-ratio particles may induce mechanical irritation and physical damage that differs fundamentally from the chemical toxicity

or physical obstruction caused by spherical MPs. The overall non-uniformity of the pollutant necessitates comprehensive risk models that account for these interacting physical and chemical parameters. (Osman et al., 2020).

Based on the document provided, the following tables would effectively synthesize the complex information regarding particle toxicity, physiological disruption, and chemical interactions. These tables will improve readability by organizing dense text into scanable formats.

Table 2.1: Comparative Toxicity by Particle Type and Shape

Particle Type / Shape	**Key Characteristics**	**Documented Biological Mechanism & Impact**
Nanoplastics (NPs)	<100 nm; extremely high surface area; superior mobility	Capable of crossing blood-brain and gut barriers to enter deep tissues (brain, gonads). High capacity for releasing co-contaminants
Polystyrene (PS)	Most frequent NP (89%); commonly used in lab studies.	Specifically linked to reproductive disruption, metabolic disorders, and transgenerational effects
Fibers	High-aspect-ratio; constitute up to 99.23% of contamination in some surveys.	Induce mechanical irritation and physical damage distinct from chemical toxicity
Polyethylene (PE)	Common polymer type	Significant alteration of gut microbial composition (dysbiosis)

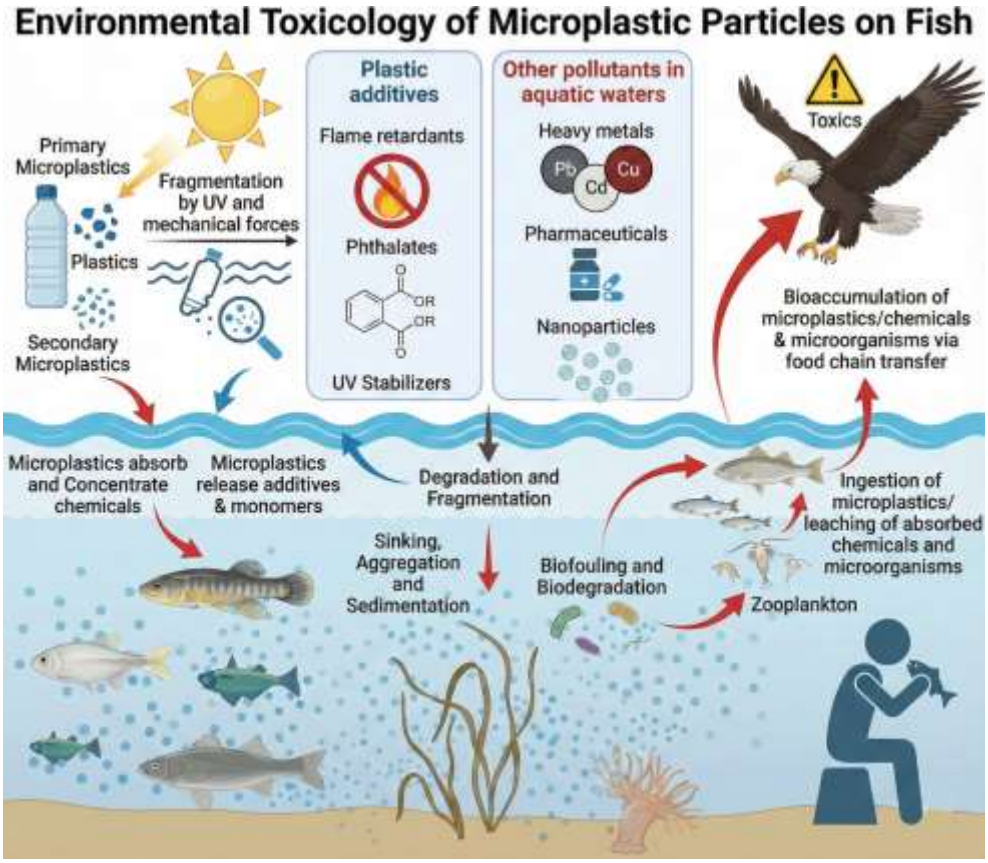
2.3. The Vector Effect: Transfer of Co-Contaminants and Altered Bioavailability

One of the most consequential mechanistic pathways for MP toxicity is the 'vector effect,' where MPs act as efficient carriers for environmental contaminants Due to their chemical properties and large surface area, microplastics adsorb a wide array of harmful chemicals, including Hydrophobic Organic Chemicals (HOCs), Polycyclic Aromatic Hydrocarbons (PAHs), heavy metals, pharmaceuticals, and endocrine disruptors (EDCs). These EDCs may be either environmental contaminants or additives used in plastic manufacturing, such as bisphenols and phthalates (Barboza et al., 2016).

The hypothesis that MPs transfer hazardous HOCs to marine animals has been strongly validated. Studies confirm that MPs binding to PAHs, such as pyrene, demonstrate a significant ability to transfer these chemicals to exposed organisms. This transfer capability influences the bioavailability and subsequent toxicity of the sorbed chemicals (Cole et al., 2019).

Crucially, exposure via contaminant-spiked MPs (e.g., microplastics carrying chlorpyrifos, MP-CPF) results in chemical accumulation in fish and fundamentally alters the spatial distribution of the toxicant within the organism compared to conventional exposure routes. This alteration can shift the internal distribution toward organs most exposed to the particle, often increasing the contaminant load and risk in the intestine. This validated vector function confirms that MPs serve as a major pathway for systemic toxicity, imposing a high chemical burden on aquatic vertebrates (de Souza et al., 2020).

Figure 2.1 The Microplastic Vector Effect: Interactions with Co-Contaminants, Additives, and Trophic Transfer Pathways.



3. Disruption of the Neuroendocrine Regulatory Axis

The neuroendocrine system, responsible for coordinating metabolism, reproduction, and stress response, is profoundly vulnerable to microplastic exposure, largely through the delivery of endocrine-disrupting substances. (Bossio et al., 2025)

3.1. Molecular Mechanisms of Endocrine Disruption

The endocrine hazard originates from two sources: EDCs absorbed from the aquatic environment and EDCs used as plastic additives (e.g., bisphenols, phthalates) that easily leach from the particles because they are not covalently bonded to the polymer matrix (Flaws et al., 2020).

The primary molecular mechanism involves the structural similarity between EDCs and specific hormone receptors. This mimicry allows EDCs to interfere with the normal hormonal action of endocrine glands (By binding to or blocking hormone receptors particularly those for sex steroid hormones these chemicals deregulate hormonal signaling, leading to physiological abnormalities (Jayasundara et al., 2020).

3.2. Impact on the Hypothalamic-Pituitary-Gonadal (HPG) Axis

Microplastics and nanoplastics are documented to disrupt the entire hypothalamic-pituitary axis, including the master reproductive control pathway, the HPG axis This failure results in severe reproductive toxicity outcomes.Documented consequences include observable tissue damage in the testes and ovaries, sex-specific reproductive disruption, and measurable decreases in sperm quality (Lu et al., 2019). For instance, polystyrene microplastics were shown to cause tissue damage, sex-specific reproductive disruption, and observable transgenerational effects in marine medaka (*Oryzias melastigma*) The documentation of developmental abnormalities and transgenerational effects highlights that MP exposure can permanently compromise the recruitment and reproductive potential of wild and farmed aquatic vertebrate populations (Athey et al., 2020).

3.3. Disruption of the HPT and HPA Axes and Associated Stress

The toxic effects of MPs/NPs extend beyond reproduction, targeting the hypothalamic-pituitary-thyroid (HPT) and hypothalamic-pituitary-adrenal (HPA) axes. Because all endocrine systems are intimately interconnected, interference with one primary regulatory axis (e.g., sex steroid signaling) inevitably leads to the deregulation of others governing stress response, growth, and metabolism. (Fioretto et al., 2025).

This endocrine deregulation is not an endpoint but a primary trigger. The disruption of regulatory pathways by MPs/NPs is explicitly documented to precede and contribute to the subsequent induction

of systemic cellular stress, including oxidative stress, neurotoxicity, cytotoxicity, and immunotoxicity. Therefore, the instability of the endocrine system serves as a key precursor to widespread homeostatic failure. (Jahedi et al., 2025).

A crucial limitation in the current understanding of endocrine disruption involves the upstream control centers. While significant evidence details the adverse effects of MPs/NPs on peripheral glands (thyroid, testes, ovaries), research still needs to definitively identify the direct effects of these particles on the hypothalamus. As the coordinating center of the entire axis, confirmation of hypothalamic disruption is paramount to establishing the comprehensive molecular basis for the observed cascading failures across HPG, HPT, and HPA systems. (Ullah et al., 2023).

4. Alteration of Innate Immunity and Cellular Homeostasis

The physical and chemical presence of microplastics necessitates heightened cellular defense mechanisms, leading to chronic inflammation, oxidative damage, and systemic metabolic overload that threaten organismal homeostasis. (Subaramaniyam et al., 2023).

Table 3.1 The "Vector Effect": Contaminant Transfer and Outcomes

Contaminant Class	Specific Example	Interaction with Microplastics	Biological Outcome
PAHs	Pyrene	Strong binding to MP surface; efficient transfer to organisms	Increased bioavailability and toxicity of the sorbed chemical
Pesticides	Chlorpyrifos (CPF)	Spiked MPs (MP-CPF) alter internal distribution	High accumulation in the intestine; increased chemical burden in specific organs
Plastic Additives	Bisphenols, Phthalates	Leach from particles (not covalently bonded)	Endocrine disruption due to structural mimicry of hormones
Dioxin-like Chems	TCDD	Adsorption to MPs creates high xenobiotic load	Significant upregulation of CYP1A mRNA in kidney tissues

4.1. The Mechanism of Oxidative Stress Induction

Microplastic exposure causes fundamental cellular and physiological damage largely mediated by the induction of oxidative stress. This toxicological endpoint reflects a failure in cellular homeostasis, specifically the inability of the organism to manage the balance of reactive oxygen species (ROS). This chronic stress is measurable through changes in antioxidant status and immune-related gene expression (Savuca et al., 2023)

4.2. Immunotoxicity, Inflammation, and Co-Stressor Interaction

MPs induce inflammation and disrupt the innate immune system in aquatic vertebrates this immunotoxicity is evidenced by changes in immune-related gene expression and observable tissue damage the evaluation of MP effects must also consider environmental co-stressors, which often exacerbate intrinsic toxicity. For instance, studies on fish have shown that MPs interact complexly with conditions such as hypoxia (low oxygen), influencing the severity of the resulting immune response and impacting vital functions like specific growth rate in species such as the yellow catfish (*Pelteobagrus fulvidraco*) (Sun et al., 2021).

The fact that MPs induce immune responses and histopathological damage coupled with the challenging environmental conditions typical of high-density production systems, suggests that the combined stressor effect could significantly compromise the health and commercial viability of aquaculture stocks, justifying specific policy and management attention to this sector (Hirt et al., 2020).

4.3. Consequences for Detoxification and Xenobiotic Metabolism

The chemical load delivered by the MP vector effect necessitates a massive mobilization of detoxification pathways. The organism attempts to maintain homeostasis by upregulating xenobiotic metabolism, clearly evidenced by the significant induction of the Cytochrome P450 (CYP) family genes (Kim et al., 2024). CYP1 family genes, including CYP1A, CYP1B, CYP1C1, and CYP1C2, are known to be integral to this detoxification process.

Exposure to high xenobiotic loads, such as dioxin-like chemicals often adsorbed by MPs, results in a significant upregulation of these CYP1 family genes in key metabolizing organs, including the gills, liver, and kidney (Choi et al., 2023). For example, TCDD exposure resulted in a highly significant increase in CYP1A mRNA expression in the kidney. This measurable upregulation of CYP1 genes serves as a strong biological marker, indicating that the organism is actively processing a substantial chemical burden. This validates the finding that the vector function of MPs is potent enough to induce systemic toxicological responses comparable to exposure to highly concentrated traditional chemical pollutants (Barboza et al., 2016).

This metabolic overload directly correlates with severe histopathological damage in major organs. The sustained effort to detoxify results in observable pathological changes, including liver vacuolation and kidney necrosis, underscoring the systemic burden and failure to maintain tissue homeostasis (Wallig et al., 2022).

5. Impact on Digestive and Neurobehavioral Regulatory Systems

The gastrointestinal tract is the primary entry point for microplastics, leading to localized dysregulation that extends rapidly into systemic metabolic and neurological dysfunction via interconnected regulatory axes. (Sofield et al., 2024)

5.1. Gastrointestinal Tract Accumulation and Tissue Damage

MPs accumulate extensively within the gastrointestinal tract, hindering both feeding and digestive processes. This accumulation results in localized physical presence and chemical irritation, contributing to observable tissue damage the gut thus becomes the initial focus of homeostatic failure, which subsequently propagates through internal systems. (Meena et al., 2023)

5.2. Disruption of the Gut Microbiome and Dysbiosis

Accumulation in the gastrointestinal tract is a direct cause of gut microbiome disruption, resulting in dysbiosis a pathogenic imbalance in the microbiota. Specific microplastic types, such as 5 micrometer polystyrene and polyethylene microplastics, have been demonstrated to significantly alter microbial composition, often leading to the proliferation of pathogenic bacteria (Lu et al., 2019).

This disruption of the microbiota balance is profoundly significant because it is linked directly to systemic inflammation and is an identified mechanism underlying chronic diseases in exposed organisms this confirms that intestinal health and the microbial balance are primary regulatory mechanisms targeted by MP exposure. (Yuan et al., 2024).

5.3. Consequences for Energy Metabolism and Growth

The disruption of the gut microbiome, coupled with chronic inflammation and tissue damage, leads directly to metabolic disorders, particularly those affecting energy metabolism. This profound metabolic inefficiency, evidenced in species like *Oryzias javanicus* exposed to polystyrene microplastics, accumulates into macro-level physiological consequences, most notably inhibited development and growth retardation (Borrelle et al., 2020).

5.4. Neurotoxicity and Behavioral Disturbances

Exposure to microplastics is linked to neurotoxicity, manifesting as clear behavioral abnormalities in fish. The mechanism involves both the translocation of neurotoxicants carried by MPs/NPs across the blood-brain barrier Specific research confirms that contaminants delivered via MP carriers, such as chlorpyrifos (CPF), cause significant inhibition of acetylcholinesterase (AChE), a neurotransmitter enzyme critical for nervous system function (de Souza et al., 2020). This neurochemical disruption results in hyperactivity, a behavioral anomaly that has significant ecological consequences. Increased hyperactivity in affected fish results in heightened vulnerability to predation, transforming a cellular disruption into an ecologically relevant factor that can influence population mortality rates and trophic energy transfer dynamics (Couillard et al., 2008).

Furthermore, the inflammation and dysbiosis originating in the gut suggest a potential perturbation of the gut-brain axis, potentially linking intestinal health failure to systemic neurological compromise (Meena et al., 2023).

6. Conclusion and Future Research Directions

6.1. Comprehensive Synthesis: Integrating Multi-Pathway Disruption

The ecotoxicology of microplastics in aquatic vertebrates is defined by a multi-modal assault on systemic homeostasis. The evidence from the 2015–2025 period confirms that physical exposure is inseparable from the chemical vector function, leading to chronic instability across neuroendocrine, immune, and metabolic regulatory pathways (Hossen et al., 2025)

The size of the particle is the critical differentiator, with nanoplastics (NPs) driving the shift toward greater chemical delivery, increased oxidative stress, reproductive compromise, and neurotoxicity. The consistent findings of significant reproductive failure, growth retardation, and ecologically compromising behavioral changes. collectively indicate that MP exposure represents a significant, chronic threat that compromises the overall ecological fitness and viability of aquatic vertebrate populations. The initial disruption of regulatory pathways, such as the HPG axis, ultimately leads to a cascade failure across multiple physiological systems. (Kumari et al., 2025)

6.2. Addressing Critical Knowledge Gaps (2015–2025)

To advance risk assessment frameworks, future research must address several key knowledge gaps identified within the past decade. First, research must increase its focus on the smallest fraction of plastic debris, nanoplastics (NAPs, smaller than 1 micrometer), as current knowledge on their fate, translocation, and long-term consequences remains limited (Horn et al., 2020). Second, while systemic

effects are clear, greater mechanistic clarity is required. Future studies should focus on elucidating the precise cellular pathways involved in the immune response and must undertake specific investigations to document the direct toxicological effects of MPs/NPs on the hypothalamus (Sun et al., 2021)

Third, given the substantial experimental variability observed across various studies—particularly concerning polymer type, concentration, and particle size there is an urgent need to standardize protocols for mechanistic endpoint determination. Standardization of biomarkers (e.g., for oxidative stress or CYP gene expression) is essential to enable reliable comparison and effective global risk assessment (Hossen et al., 2025). Finally, researchers must work to improve the validity of extrapolating controlled laboratory findings on vector effects to complex, variable field environments, where contaminant mixtures and environmental kinetics are far more complex (Horn et al., 2020).

6.3. Strategies for Mitigation and Policy Development

Effective mitigation of microplastic ecotoxicology requires an interdisciplinary approach that integrates ecological health surveillance with public health initiatives Strategies must target source reduction through improved waste management and recycling systems, the adoption of biodegradable plastic alternatives, and the enforcement of evidence-based policy interventions (Yuan et al., 2024). Implementing standardized, collaborative monitoring efforts is necessary to accurately track microplastic loads and corresponding biological effects across diverse aquatic ecosystems, thereby enabling tailored, ecosystem-specific mitigation techniques (Sigmund et al., 2023).

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