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Ecotoxicological Effects of Nanoplastics on Aquatic Organisms

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Abstract. The health of aquatic ecosystems is a cornerstone of global environmental security and human well-being. As an emerging contaminant, nanoplastics (NPs) have attracted widespread attention due to their pervasive presence in production processes and the environment, high bioavailability, and potential to induce adverse biological effects. However, significant knowledge gaps persist in current research, which includes an unclear understanding of the ecological risks from long-term, low-dose exposure, insufficient investigation into the combined and synergistic toxic mechanisms of nanoplastics with various traditional and emerging pollutants, and a lack of systematic integration of toxic effects from the molecular to the ecosystem level. To address these gaps and advance the development of toxicology, this review systematically screened and summarized relevant research, ultimately providing a comprehensive evaluation of the ecotoxicological effects of NPs on aquatic organisms.

Keywords: nanoplastics, aquatic organisms, ecotoxicology, toxicity mechanism, aquatic risk assessment.

1. Introduction

Nanoplastics (NPs), typically defined as plastic particles with a size of less than 100 nm, have attracted critical attention in recent years due to widespread distribution, potential for bioaccumulation, and ecotoxicity. Global plastic production, which stood at 464 million metric tons in 2020, is projected to increase to 884 million metric tons by 2025[1]. Against the backdrop of continuously rising global plastic production, the environmental prevalence of NPs and their ecological risks have become a frontier research topic in environmental science and toxicology.

NPs primarily originate from two distinct pathways including secondary fragmentation of larger plastic debris and direct anthropogenic sources. For secondary sources, the further degradation of existing microplastics (MPs) in the environment, where larger plastic particles fragment into smaller nano-sized particles through processes like weathering, photodegradation, and biological fragmentation[2]. A striking example is rotifers, which mechanically break down ingested microplastics. Each individual potentially generating about 3.5×10^5 nanoplastic particles, and this process likely widespread in global water systems[3]. Primary sources involve direct release from industrial wastewater, personal care and cleaning products, and medical waste[4]. Particularly following the COVID-19 pandemic, the surge in usage of medical plastic product has contributed to an increasing in NPs input to the environment[5].

Due to their minuscule size, high specific surface area, and strong mobility, NPs are difficult to remove from the water column through natural sedimentation. They can diffuse more widely in water systems[6], penetrate biological barriers, and enter cells, the circulatory system, and various organs, inducing mechanical damage, oxidative stress, and diverse toxic impacts. Furthermore, NPs also act as carriers for pollutants, adsorbing heavy metals, pesticides, pharmaceuticals and personal care products, per- and polyfluoroalkyl substances (PFAS), and other traditional and emerging contaminants. This alters the environmental fate and bioavailability of the adsorbed substances, exacerbating the risks of combined pollution.

NPs are widely distributed in various water bodies, with their presence even detected in polar regions[7]. Globally, the ocean receives the annual flux of plastic debris from riverine transport, atmospheric deposition, and direct discharge. Notably, NPs are continuously replenished through the degradation of larger particles, posing a long-term and cumulative threat to aquatic ecosystems[8].

Additionally, NPs can migrate from soil and pore media into groundwater systems[9], raising potential risks in drinking water safety.

Although existing studies have preliminarily revealed individual-level toxicity of NPs, such as growth inhibition and increased mortality, their toxic mechanisms at the molecular and cellular levels remain unclear. The ecological effects of long-term, low-dose exposure, and the synergistic toxic mechanisms under combined pollution scenarios remain unclear. Moreover, most current research is confined to single-exposure conditions in the laboratory, which struggles to realistically reflect the complex exposure scenarios involving multiple co-existing pollutants in natural environments. There is also a lack of integrated toxicity assessment spanning from individuals to populations and even ecosystem levels.

This review aims to systematically summarize the sources, distribution, transport, and transformation behaviors of NPs in aquatic environments, with a focus on analyzing their individual and combined toxic effects and mechanisms. By discussing the existing data and mechanistic gaps in current research, providing a scientific basis for future ecological risk assessment and management strategies about NPs.

2. Combined Pollution of Nanoplastics and Other Major Water Pollutants

Unlike studies on single pollutants, aquatic organisms are rarely exposed to isolated contaminants. They face complex water bodies with multiple coexisting substances. Traditional risk assessment methods for single pollutants may significantly underestimate or overestimate the actual risk. This reality necessitates a shift in focus from single-substance risk assessment to the study of combined effects, emphasizing the investigation of antagonistic or synergistic interactions between pollutants. These interactions can drastically modify toxicological outcomes and is crucial for simulating their risks in real-world environments, such as industrial wastewater discharges.

When assessing the toxicity of combined pollutants, the joint effects are primarily categorized into three types based on the interactions between components: additive, synergistic, and antagonistic. An additive effect occurs when the combined toxicity equals the sum of the individual toxicities of each substance. A synergistic effect refers to a combined effect greater than the sum of the individual effects of the two chemicals. An antagonistic effect means the combined effect is lower than the sum of the effects observed during individual exposure tests.

2.1 Traditional Heavy Metals

Among the environmental factors threatening aquatic ecosystems, heavy metals are ubiquitous, persistent, and highly toxic pollutants[10,11]. Unlike many organic pollutants, heavy metals are failed to be biodegraded, causing a clearer tendency to accumulate in aquatic organisms and exhibit significant toxicity even at low concentrations[12,13]. For example, fish exposed to heavy metal-contaminated water in the Yamuna River showed significant accumulation of heavy metals in their liver and kidneys, accompanied by abnormal enzyme activity, immune suppression, and DNA damage[14].

The combined effect of NPs and heavy metals is key to their environmental risk assessment[15]. Owing to their large specific surface area and hydrophobic properties, NPs efficiently adsorb heavy metals from the environment and facilitate their uptake into organisms[16,17]. Studies on Prussian carp confirmed that the coexistence of cadmium (Cd) significantly enhances the accumulation of polystyrene nanoparticles (PS-NPs) in liver tissue[18]. A previous study further indicated that zebrafish co-exposed to NPs and Cu^{2+} accumulate substantially higher copper levels than those exposed to Cu^{2+} alone[19]. Higher bioaccumulation is correlated with stronger toxic effects, subsequent translocation of these heavy metals to the intestine enables dissociation from NP surfaces, and the released Cu^{2+} triggers a severe oxidative stress response. Research has established that NPs act as effective carriers for Cu^{2+}, enhancing its intestinal accumulation in aquatic organisms[20], laying the groundwork for subsequent synergistic toxicity.

Of particular concern is the impact of synergistic toxicity on the cellular energy metabolism system. In co-exposure groups, mitochondria showed obvious vacuolization and disappearance of cristae structures[20]. Transcriptomic profiling via RNA sequencing (RNA-seq) further revealed that co-exposure significantly downregulated the expression of genes related to energy metabolism pathways such as oxidative phosphorylation (OXPHOS) and the respiratory electron transport chain (ETC)[20]. *In vitro* validation also confirmed that combined exposure led to a marked decrease in cellular ATP compared to single exposure, along with the collapse of the mitochondrial membrane potential ($\Delta\Psi_m$), corroborating the failure of the energy metabolism system[20].

Furthermore, co-exposure to NPs and heavy metals produce synergistic reproductive toxicity[21]. Zebrafish embryo experiments demonstrated that co-exposed groups exhibited significantly higher hatching inhibition rates and elevated morphological rates[20,22]. Histopathological analysis via hematoxylin-eosin (HE) staining revealed that profound intestinal structure damage, manifested as thinning of the intestinal wall, incomplete villi structure, and a reduction in epithelial cell number[20].

The interaction between NPs and heavy metals is not always synergistic[23]. Research in a nematode model found that the number of offspring decreased after feeding on *E. coli* contaminated with Cd, but indirect co-exposure with PS-NPs turn out to alleviate the reproductive toxicity of Cd[24], suggesting that PS-NPs might produce an antagonistic effect by influencing the distribution of Cd within the body, although the specific mechanisms require further investigation.

2.2 Organic Pollutants

The advent of synthetic organic chemistry introduced large quantities of persistent organic pollutants (POPs) into the aquatic environment, including polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and various pesticides[25]. These characteristic lipophilicity facilitates them to accumulate in organisms' fatty tissues and biomagnify to concerning concentrations in top predators of the food chain[26].

The combined effects of NPs and organic pollutants are a current hotspot in environmental toxicology research. As both are widely present in aquatic ecosystems, multiple studies have demonstrated that NPs not only act as carriers for pollutants but also significantly change their bioavailability, toxicity, and ecological risk through processes including physical adsorption, biological uptake, and intracellular release[8,27]. These interactions are not solely synergistic or antagonistic.

Under certain environmental conditions, NPs can reduce the bioavailability of organic pollutants through adsorption. A study practiced by Trevisan[28] et al showed that PS-NPs could adsorb PAHs from Elizabeth River sediment extracts (ERSE), forming larger aggregates, reducing the freely dissolved concentration of PAHs in water, and thereby significantly alleviating typical PAH toxicity symptoms in zebrafish embryos, such as pericardial edema and heart malformations. Research operated by Sayed[29] on Nile tilapia also indicated that while co-exposure to NPs and EO caused severe inflammation and oxidative stress, but no more data show other severe effects than single EO exposure on some histopathological indicators, such as gill and kidney damage, suggesting that NPs might attenuate the acute toxicity of EO to some extent.

Additionally, NPs can also enhance the toxicity of organic pollutants through other mechanisms, exhibiting synergistic effects[30]. NPs can be mistakenly ingested by organisms or enter through the body surface, acting as pollutant carriers, and then release high concentrations of pollutants within tissues and cells, leading to enhanced toxicity[19]. For example, NPs penetrate the mitochondrial membrane. After entering mitochondria, they can disrupt the electron transport chain, inhibit ATP synthesis, and increase ROS production. If they carry organic pollutants at this time, mitochondrial damage might be exacerbated. 2-Ethylhexyl diphenyl phosphate (EHDPP) is an organic compound widely found in organophosphorus flame retardants[31]. For zebrafish, the addition of NPs lowered the threshold for EHDPP to induce various toxic effects. The co-exposure group significantly enhanced oxidative stress and neurotoxic effects and promoted the gene expression of pro-inflammatory cytokines Tnf-α, IL-6, and IL-1β, activating apoptosis-related genes[31]. Several studies clearly indicate

that NPs ,promote their accumulation in aquatic organisms and producing significant synergistic toxic effects as carriers for organic pollutants[30,32].

2.3 Emerging Contaminants

Emerging contaminants are a class of pollutants previously unrecognized or unregulated, and their impact on aquatic toxicology is becoming increasingly evident[33]. This category includes MPs/NPs, pharmaceuticals, endocrine-disrupting chemicals (EDCs), among others. Their pathways into water bodies and toxic effects are diverse[34,35].

The toxic interactions between NPs and emerging contaminants are not simple additions of each component but are complexly influenced by factors such as pollutant type, number, concentration ratio, and carrier effects. An analysis by Yang Jianyuan[36] of 156 nanoplastic-sulfonamide antibiotic mixture systems indicated that the toxicity of the mixtures increased with the number of components, suggesting that a greater variety of pollutants in the environment may lead to a higher risk of combined toxicity. Furthermore, the concentration ratio plays a key role in determining the direction of the combined toxic effect. Study by Yang Jianyuan also found that when the proportion of NPs in the mixture increased, the overall toxicity is weakened, implying a possible antagonistic effect, perhaps stemming from the adsorption of organic compounds by NPs reducing their bioavailability.

A study conducted by Wang et al[30] on co-exposure to triclosan (TCS) and polystyrene NPs (PS-NPs) provides deeper mechanistic insights. Although TCS adsorbed onto the surface of PS-NPs, its distribution in zebrafish did not change. Instead, co-exposure significantly exacerbated neurodevelopmental toxicity, intestinal microbiota dysbiosis, and histopathological damage through oxidative stress pathways, manifesting as a typical synergistic effect. Another study found that a combination of high-concentration NPs and ciprofloxacin could trigger severe disturbances in key pathways in soybean leaves[37], such as D-amino acid metabolism and isoflavone synthesis. These research indicate that NPs, acting as pollutant carriers, may promote the accumulation and transport of toxic substances within organisms, thereby amplifying their physiological disruptive effects.

The interactions between NPs and emerging contaminants constitute **intricate, dynamic processes** governed by the precise combination of pollutants, exposure concentrations, and biological pathways[38,39]. When evaluating **real-world environmental risks**, the **synergistic toxicity potential** of multiple co-occurring pollutants must be explicitly accounted for—particularly when **NPs** act as carriers that enhance the bioavailability of co-pollutants. In such cases, their combined toxicity can **substantially exceed** the sum of effects from individual pollutants[36,38] .

3. Ecotoxicological Mechanisms at Molecular and Cellular Levels

3.1 Molecular Mechanisms:

NPs induce the overproduction of reactive oxygen species (ROS) in aquatic organisms[40]. The generation of ROS disrupts the homeostasis of the antioxidant defense system, leading to oxidative stress. Cells respond by secreting antioxidant enzymes (SOD, CAT) to reduce ROS levels. If ROS are not effectively cleared, the intracellular malondialdehyde (MDA) content increases, causing cell membrane damage[41]. For instance, under PS-NPs stress, the marine alga Prorocentrum donghaiense exhibited increased activities of SOD and CAT, alongside intracellular lipid peroxidation, resulting in oxidative damage[42]. Furthermore, NPs can inhibit the production of lysozyme, also leading to oxidative damage[42]. For example, in mud crabs, exposure to NPs reduced lysozyme production, inhibiting the hydrolysis of bacterial cell walls and the normal synthesis and secretion of other immune factors, ultimately causing oxidative damage[43].

The aging of NPs also influences their toxicity[44]. Exposure of aquatic organisms to NPs may induce oxidative stress in nerve cells, which could be one of the mechanisms behind NP-induced neurotoxicity[45]. PET—one of the most common NPs in the ocean—after photo-aging, can not only disrupt the balance of cellular antioxidant enzymes but also significantly induce lipid peroxidation. Studies show that cells exposed to aged PET experienced a 6.73–8.24-fold increase in SOD

concentration and a 4.55–5.26-fold decrease in GSH-Px concentration, leading to an imbalance in antioxidant enzymes and consequent toxicity. Exposure to 20 μg/mL of PET-24h and PET-48h increased MDA content in PC12 cells by 2.03 and 5.12 times, respectively, verifying that the degree of aging significantly influences PET toxicity. The accumulation of ROS due to reduced antioxidant enzyme activity can lead to severe toxic effects[44].

NPs can cause DNA damage. In zebrafish liver tissue exposed to high concentrations of NPs, the content of single-stranded DNA significantly increased, which is a direct molecular marker of DNA damage and the initiation of apoptosis.

3.2 Cellular and Tissue Level

Nanoplastic-induced stress perturbs the energy metabolic homeostasis of aquatic organisms, compromising their capacity to meet the elevated energy demands imposed by stress. This functional impairment ultimately culminates in metabolic dysfunction, eroding their ability to survive or reproduce under polluted condition. Multiple studies indicate that this disruption primarily manifests as the inhibition of glucose metabolism pathways and the tricarboxylic acid (TCA) cycle, Under chronic stress, the body's high energy demand leads to massive consumption of glucose, causing blood glucose levels to decrease instead of rise[46]. Metabolomic analysis confirmed that zebrafish larvae exposed to NPs showed widespread reduction in the levels of intermediate metabolites in the TCA cycle, ultimately leading to decreased ATP levels. This could be due to reduced metabolic substrates or NPs reducing mitochondrial coupling efficiency. The collapse of this energy metabolism system renders organisms vulnerable to additional stresses, posing a serious threat to their survival and growth[28].

Ingested NPs have no substantial nutritional value[47]. The accumulation of plastic particles in the digestive tract can hinder the absorption of food, affect the feeding behavior of aquatic animals[48], and an imbalanced intake of food can also lead to energy and metabolic disorders. For example, NPs can reduce nutrient intake in mud crabs, affecting their normal development, lowering their energy absorption efficiency, and resulting in malnutrition[43].

NPs exhibit significant apoptotic toxicity in aquatic animals, primarily through the modulation of multiple key genes and signaling pathways. Studies have found that although exposure to PS-MPs did not cause obvious morphological changes or lethal effects, acridine orange staining revealed a dose-dependent increase in apoptotic cells in treated embryos[49]. At the molecular level, the expression of pro-apoptotic genes p53, caspase-3, and caspase-9 was significantly upregulated, while the expression of the anti-apoptotic gene bcl-2 was significantly downregulated. This indicates that PS-MPs activate the mitochondria-dependent apoptotic pathway, inducing programmed cell death through a p53-mediated caspase cascade.

Furthermore, PS-MPs induced oxidative stress, manifested as increased lipid peroxidation (LPO) levels and decreased activities of superoxide dismutase (SOD) and catalase (CAT), which further exacerbated cellular damage and promoted apoptosis. Simultaneously, the downregulation of brain-derived neurotrophic factor (bdnf) expression impaired neuronal survival and function[49]. This, combined with elevated nitric oxide (NO) levels, further enhanced the activation of the p53 and caspase pathways, ultimately leading to neuronal apoptosis and behavioral abnormalities.

3.3 Individual and Population Level

NPs exposure can induce pronounced behavioral dysfunctions through multifaceted pathways. Aberrant behaviors in aquatic organisms are frequently rooted in impaired nervous system function, as NPs accumulate in neural tissues and exert direct neurotoxicity[50]. For instance, in nematode models, NPs exposure has been shown to accelerate head thrashing and increase body bending frequencies—key behavioral markers of motor neuron dysfunction[51].

The neurotoxicity of NPs, which leads to motor behavioral disorders, is often mediated through the inhibition of acetylcholinesterase (AChE)—an important biomarker for environmental pollutants. For instance, zebrafish treated with NPs showed a general 27%-40% reduction in AChE activity[52],

which can cause neurotoxicity, impairing their motor ability and leading to behavioral disorders. In predator avoidance tests, zebrafish in the high-concentration (1.5 ppm) NP exposure group showed a significantly reduced average distance to predators, indicating sluggish escape response and impaired predator avoidance behavior[52]. Such deficits would directly lead to a sharp increase in predation risk in natural environments.

Notably, the mechanisms of behavioral toxicity are not limited to direct neural effects but are also closely related to systemic energy metabolism disorders. Recent studies show that offspring of maternal zebrafish exposed to environmentally relevant concentrations of polystyrene NPs exhibited significantly reduced locomotor capacity, even without direct exposure[53]. The underlying mechanism involves maternal transfer of PS-NPs, causing mitochondrial dysfunction in the offspring, particularly significant inhibition of the oxidative phosphorylation pathway. Impaired mitochondrial function leads to insufficient ATP synthesis, unable to provide adequate energy for muscle contraction and neural activities, directly resulting in motor behavioral impairments.

NPs exposure can cause stress and damage to the central nervous system (CNS), leading to abnormal CNS development and subsequent behavioral abnormalities. gfap and α1-tubulin are key markers for CNS development[54]. Research shows that NPs can significantly upregulate the expression of these genes[52]. gafp is the main cytoskeletal protein of astrocytes, and its significant upregulation is a classic marker of reactive astrogliosis and an indicator of neurotoxicity in mammalian brains. α1-tubulin is a major protein component of neuronal microtubules. The upregulation of these nervous system-related genes indicates the neurotoxic effects of NPs on zebrafish, which can significantly impact their behavior.

NPs pose a serious threat to the reproductive systems of aquatic organisms. Their toxic effects can be achieved through direct accumulation in and damage to gonads. NPs can preferentially accumulate in the gonadal tissues of fish, impairing reproductive function[55]. Studies show that 70nm PS-NPs can exhibit gonadotropism in zebrafish. This accumulation is believed to occur through the gonadal blood barrier, potentially related to the blood supply to the gonads[56]; NPs absorbed through the gills can circulate via arterial blood to organs like the gonads and liver[55]. This direct accumulation can exert toxic effects on germ cells. More importantly, this toxicity has transgenerational effects. Studies indicate that after long-term exposure of maternal zebrafish to PS-NPs, their offspring, even without direct exposure, showed increased embryonic malformation rates, and significant reductions in head size and body length. This reveals that maternally accumulated NPs can cause persistent harm to the germ cells and early development of offspring[53]. In marine copepods, exposure to micron-sized PS for two months resulted in a reduction in egg size, suggesting decreased energy reserves per egg, which may negatively affect larval survival rates. Additionally, both hatching success and ingested carbon biomass significantly declined. In the later stages of the experiment, the number of deceased individuals in the microplastic-exposed group increased[57].

NPs also affect endocrine function, disrupting the normal synthesis and regulation of sex hormones, leading to impaired reproduction. In zebrafish experiments, male fish exposed to NPs showed significantly elevated levels of vitellogenin (VTG) in the liver, confirming that NPs have estrogen-mimicking effects and can disrupt normal endocrine signaling pathways[56]. Furthermore, mechanistic studies indicate that NPs can downregulate the expression of cyp19a1a, thereby inhibiting the synthesis of estradiol (E2) and halting follicular development[21,58]. Recent research further found that maternal PS-NPs exposure significantly suppressed the expression of genes related to energy metabolism and DNA repair in offspring embryos[21]. These molecular-level disturbances are among the root causes of developmental malformations and reduced reproductive potential in the offspring.

Sensitive species in water bodies long-term exposed to NPs show declining population trends[59]. This is not due to a single factor but results from the combined effects of increased predation risk due to behavioral abnormalities and decreased birth rates and offspring survival due to reproductive impairments. Studies have proven that long-term direct exposure significantly reduces the survival rate of nematodes, showing a clear dose-response relationship [51]. Transgenerational toxicity studies indicate that even if offspring live in clean environments, their adaptability and survival

competitiveness are compromised, posing a deeper threat to the long-term persistence of populations. Although indirect exposure or low-dose exposure might have a smaller impact on the lifespan of some nematode species, this underscores the species-specificity and exposure pathway dependency of toxic effects[21,58]. Overall, the risk of nanoplastic pollution at the population level in aquatic organisms cannot be ignored.

4. Toxicity Response Mechanisms in Different Aquatic Organisms

NPs exhibit pronounced toxicological disparities across distinct groups of aquatic organisms. The evolutionary divergence and physiological traits of these organisms fundamentally govern their susceptibility and adaptive response mechanisms to NPs.

4.1 Aquatic Plants

Aquatic plants play a crucial role in maintaining water cleanliness and ecological balance, but their response to NPs has not been fully studied. Preliminary research indicates that NPs can adhere to plant roots or leaf surfaces, affecting nutrient uptake and photosynthetic efficiency[60]. For instance, water hyacinth (Eichhornia crassipes) under nanoplastic stress shows symptoms like poor root development and activation of the antioxidant enzyme system[61]. High concentrations of NPs may inhibit their growth, weakening their potential application in ecological remediation[62,63].

NPs can affect the morphological development of aquatic plants, altering their shoot-to-root ratio (S:R). NPs carry a negative charge and can physically adsorb onto the surfaces of plant roots like dust[39]. The root surface is a key site for water and nutrient absorption, as well as substance secretion. The adsorption of NPs can hinder nutrient uptake and block the secretion of enzymes and other substances by the roots[64]. Furthermore, NPs can induce the production of reactive oxygen species (ROS) in plant cells, causing cellular damage[63]. This can lead to an abnormal increase in root biomass, as the plant diverts limited energy to root maintenance, resulting in a decreased S:R[39,64].

4.2 Algae and Plankton

As primary producers, the growth status of algae can objectively reflect the condition of the aquatic ecosystem. The sensitivity of algae and plankton to NPs directly affects the stability of the entire aquatic food web. Studies show that NPs can adhere to algal cell surfaces, interfering with their photosynthetic mechanisms, leading to growth inhibition or even cell death[65]. For example, A study on *Alexandrium* has demonstrated that higher concentrations of NPs significantly inhibit the growth rate and photosynthetic efficiency of algae[66]. Studies show Chlorophyll-a content, malondialdehyde (MDA) accumulation, antioxidant enzyme activity, and STX levels initially exhibit adaptive increases under short-term exposure to low concentrations but decline with increasing NP concentrations. Such abnormal fluctuations in physiological indicators can disrupt the homeostatic regulation of algal communities, potentially triggering algal blooms.

Plankton ingest NPs, leading to their accumulation in the gut and causing damage. However, excessively high concentrations of NPs can have the opposite effect, possibly because high concentrations can attach to the body surface of plankton, inconveniencing their feeding activities. For instance, the inhibition rates of Daphnia magna activity by PVDF and PTFE MPs/NPs showed an "inverted U-shaped" curve with changing concentration and time[67].

Regarding reproduction, NPs show significant toxic potential to plankton. They may interfere with the endocrine system, affect the molting process, and lead to growth retardation. For example, in chronic exposure experiments, the average number of molts in Daphnia magna increased[68], reflecting disruption in its development process. More importantly, recent full life-cycle exposure experiments indicate that even low concentrations of NPs considered "safe" in short-term tests may lead to a significant shortening of plankton lifespan under long-term exposure. Kelpsiene et al. (2020) found that Daphnia magna exposed to 0.32 mg/L of 53 nm aminated polystyrene (PS-NH_2) nanoparticles had their lifespan shortened by nearly threefold. Furthermore, carboxylated NPs (PS-COOH), which

were non-toxic in short-term experiments, also exhibited significant lethality in long-term exposure[67]. This suggests that short-term toxicity assessments may severely underestimate the ecological risk of NPs.

The reproductive toxicity of NPs to plankton mainly manifests as inhibitory effects, reducing offspring numbers through oxidative stress and delayed sexual maturity, among other ways[69]. Additionally, NPs can induce abnormal embryonic development, thereby reducing offspring survival rates. However, there is still controversy regarding the reproductive toxicity to plankton. Some studies have observed an increase in offspring numbers Under food-sufficient conditions, NPs stress can stimulate reproduction of Daphnia magna[70]. This might be an adaptive strategy of organisms coping with environmental stress, but its long-term consequences could be counterproductive, leading to decreased population stability[71], suggesting their ecological risk might be underestimated.

Most current research focuses on acute or subacute exposure, whereas organisms in real water bodies are often exposed to low concentrations of NPs for their entire lives. To more accurately assess ecological risk, mesocosm studies provide crucial evidence. Ekvall found in a semi-natural wetland ecosystem that the impact of NPs on aquatic organisms shows significant species specificity and threshold effects[72]. The study revealed that Daphnia magna, as an efficient filter feeder, is extremely sensitive to NPs; when exposure concentrations reached 214–2141 μg/L, the Daphnia magna population completely collapsed. However, copepods, another type of zooplankton, were not significantly affected. This selective toxicity can restructure the aquatic ecosystem food web. The disappearance of Daphnia magna reduces predation pressure on phytoplankton, leading to the proliferation of tolerant algae such as cyanobacteria or cryptophytes, while sensitive algae like diatoms may decrease due to the direct toxicity of NPs. This imbalance in phytoplankton community structure increases the risk of cyanobacterial blooms. Meanwhile, the benthic ecosystem was not significantly affected at any concentration.

Furthermore, the accumulation of NPs in organisms can be transferred through the food chain, affecting higher trophic levels. For example, fish that consume plankton containing NPs may exhibit behavioral abnormalities, metabolic disorders, and other neurotoxic responses[73]. Future research needs to strengthen multi-generational experiments and multi-species food chain models to more accurately predict the long-term impacts of NPs on aquatic ecosystems.

4.3 Fish

Fish, as higher consumers in aquatic ecosystems, are particularly sensitive to NPs. The main intake routes include gill adsorption and ingestion. After intake, NPs can affect their growth, development, behavior, and physiological functions. Zebrafish, with their good optical transparency and short generation cycle, which facilitate real-time observation using fluorescent markers, are an ideal model for transgenerational toxicity and neurotoxicity testing[74]. Zebrafish embryos exposed to PS-NPs exhibited hatching delays, heart rate abnormalities, and somite development malformations; adult fish commonly show liver oxidative stress, inflammatory responses, and metabolic disorders[50,53,56,58,75].

The particle size of NPs significantly influences their toxic effects on zebrafish. 80nm PS-NPs were mainly distributed on the body surface of zebrafish larvae, while 25nm PS-NPs showed lower accumulation signals on the body surface and primarily accumulated in the eyes, intestine, and forebrain/ hindbrain tissues[46]. Smaller-sized PS-NPs exhibited stronger tissue penetration and biomembrane permeability, allowing them to enter deeper tissues and cells[76]. Another study in tilapia [29] exposed to NPs also found primary accumulation in the digestive tract and brain, suggesting this might be a common phenomenon in fish. Furthermore, the influence of particle size on toxic effects can also be reflected in neurotoxicity. 25 nm NPs, due to their ability to cross the blood-brain barrier and accumulate in brain tissue, significantly inhibited the immediate escape response of zebrafish larvae upon tactile stimulation, manifested as a significant shortening of the escape swimming distance. In contrast, 80 nm NPs, due to limited deposition in the brain, did not significantly affect the stress escape behavior of larvae[46]. This result indicates that the neurobehavioral toxicity of NPs

in fish has obvious size dependence, with smaller particles exhibiting stronger neurodisruptive effects due to their unique biodistribution characteristics.

Nanoplastic exposure induces stress in fish. Most studies prove that stress usually causes an increase in glucose levels. High stocking density can significantly increase glucose concentration in tilapia serum[29]. However, after 48-hour exposure to 25nm PS-NPs, the levels of glucose and key glycolytic intermediates in zebrafish larvae decreased significantly, and the end product of anaerobic respiration, lactate, dropped to 51.75% compared to the control group[46]. This suggests that NPs can cause an excessive consumption of glucose due to the sustained stress state in fish, leading to the inhibition of overall glycolytic flux. However, stress-induced changes in blood glucose may be phasic: in the initial stage of stress, glucose levels typically rise, but with homeostatic mechanisms and feedback regulation of the hypothalamic-pituitary-adrenal axis, its levels may gradually recover or even decrease. For example, in rainbow trout, glucose levels rose sharply immediately after stress but decreased back to original levels after 24 hours[77].

For fish, ingestion is an important exposure route for NPs. Larvae typically prefer prey smaller than 1mm. Larvae feeding on plankton may have difficulty distinguishing and discerning NPs, leading to accidental ingestion[73]. Ingested NPs can accumulate in the digestive tract, affecting the growth and development of larvae. Small plastic particles can accumulate to form larger plastic fragments[78], which can then aggregate and block the gastrointestinal tract, interfering with its function and affecting absorption and digestion, potentially leading to death in severe cases[79]. The growth rate of larvae that ingested plastic particles was lower compared to the control group, and a significant portion of larvae died shortly after ingesting plastic particles[80]. NPs remaining in the digestive tract may interfere with feeding; ingested NPs might create a feeling of satiety, thereby reducing feeding rates. However, there is currently limited research on how NPs affect the hunting ability of larvae. Some studies also suggest that plastic particles in water can stimulate the feeding response in fish[73].

5. Advances in Toxicological Research Technologies and Methods

5.1 Traditional Toxicity Tests

Traditional toxicological testing represents the classical approach in aquatic toxicology, encompassing acute toxicity tests, chronic toxicity tests, and biomarker analysis. These methods have played a vital role in early environmental standard setting and chemical risk ranking[81]. Their primary advantage lies in the direct observation of ultimate adverse effects in organisms, providing the most intuitive and reliable phenotypic anchors for toxicity mechanism research. However, these methods often struggle to reveal the underlying toxic mechanisms and face challenges due to high costs and long experimental durations.

Traditional toxicity tests provide internationally recognized, reproducible testing standards for determining core indicators such as the median lethal concentration (LC50) and sublethal effects[82]. These classic toxicological tests have furnished substantial and conclusive evidence for contemporary aquatic toxicology research. Studies employing these methods provide clear clues for subsequent mechanistic exploration.

5.2 Omics Technologies

The advent of omics technologies has significantly enhanced the breadth and depth of toxicological research. Transcriptomics, for instance, can analyze differentially expressed genes under pollutant exposure, revealing the molecular mechanisms of toxic action. This shifts the research perspective from organs and individuals down to the level of genes, proteins, and metabolites. For instance, scRNA-seq demonstrated that nanoplastic exposure markedly downregulated genes associated with oxidative phosphorylation and the electron transport chain in zebrafish liver cells, leading to disrupted ATP synthesis[83]. A key advantage of these technologies is their ability to identify biomarkers and aid in the deep understanding of molecular-level mechanisms. However, the vast amount of data generated place high demands on subsequent analytical efforts.

5.3 Exposure Science and Ecological Simulation

To bridge the gap between controlled laboratory conditions and real ecological environments, exposure science utilizes actual environmental concentration data, making the doses used in laboratory studies more environmentally relevant. Multi-species microcosm/mesocosm tests can simulate the transport, transformation, and transfer of pollutants along food webs within ecosystems, greatly enhancing the ecological relevance of studies—a feat unattainable by single-species tests. Physiologically Based Pharmacokinetic (PBPK) modeling goes a step further, enabling the quantitative prediction of actual exposure doses in target organs[84]. Despite their power, these methods are often complex and require high costs, which limits their widespread application.

6. Application and Management in Aquatic Ecotoxicology

6.1 Development and Update of Environmental Standards

As an emerging contaminant, NPs currently lack specific water quality criteria in the environmental quality standards of most countries and regions. Toxicological research provides the critical scientific basis for filling this regulatory gap.

Existing water quality standards primarily target traditional pollutants. The widespread toxic effects of NPs and their synergistic effects as pollutant carriers, as revealed in this review, indicate an urgent need for dedicated risk assessment of NPs. This is to derive key thresholds such as the Predicted No-Effect Concentration (PNEC), laying the groundwork for establishing water quality criteria. This involves using toxicity data from organisms across different trophic levels to construct Species Sensitivity Distribution curves, thereby determining a safe concentration of NPs that protects the vast majority of aquatic life.

Current standard systems struggle to address pollutants with complex environmental behaviors like NPs. Therefore, management strategies must consider their combined pollution effects. Research on NPs, particularly the mechanisms of their synergistic toxicity enhancement with heavy metals and organic pollutants, suggests that future standard revisions need to incorporate considerations of this "carrier effect" or implement stricter joint controls for specific pollutant combinations.

The progressive incorporation of emerging contaminants like Per- and polyfluoroalkyl substances (PFAS) into regulatory frameworks provides a template for NP management. Evidence on the Persistence, Bioaccumulation potential, and Toxicity (PBT) of NPs provided by toxicological research is the core driver for pushing their inclusion into priority pollutant lists[85].

6.2 Ecological Risk Assessment and Early Warning

Ecological risk assessment and early warning serve as the bridge translating laboratory data into practice[86]. Given that environmental concentrations of NPs are typically low but cumulative, risk assessment must integrate environmental monitoring data and focus on their cumulative risks[87]. Specifically, assessing the risk in an estuary receiving various wastewaters requires integrating data on NPs, PAHs, heavy metals, etc., to evaluate the combined risk to key species. However, current assessments face significant uncertainties, including a lack of long-term, low-dose exposure data and vast differences in sensitivity among biological groups. To address these challenges, integrating distribution data of toxicologically sensitive species with NP pollution source data can help create ecological risk maps[88]. This enables the identification of high-risk areas, facilitating zoned and tiered management strategies and the application of the precautionary principle, taking prudent preventive measures in the face of scientific uncertainty.

6.3 Pollution Control and Remediation

Advancements in toxicological research technologies are driving environmental management towards intelligent and predictive approaches[89,90]. The deep molecular mechanisms uncovered by omics technologies offer the potential for discovering early, sensitive biomarkers, enabling earlier

risk warnings. Furthermore, using machine learning models to integrate the physicochemical properties of NPs with multi-omics toxicity data holds promise for the rapid virtual screening and prioritization of numerous new NPs[91]. Simultaneously, Physiologically Based Pharmacokinetic (PBPK) modeling and microcosm/mesocosm experiments can better simulate the fate of NPs in real environments, enhancing the reliability of extrapolating laboratory data to field ecosystems and providing solid support for developing more ecologically relevant management strategies.

In summary, research on NPs in aquatic ecotoxicology directly serves environmental management practices by being translated into environmental standards, risk assessment tools, and pollution control strategies. Facing the remaining scientific uncertainties, future efforts need to strengthen research under long-term, low-dose, and combined pollution conditions, and promote the deep integration of toxicology and management science. The ultimate goal is to build a more scientific, precise, and efficient risk management system for the aquatic environment.

7. Summary

This review provides a systematic elucidation of how NPs exert ecotoxicological impacts on aquatic organisms that extend far beyond simple physical stress. Through core mechanistic pathways—including oxidative stress induction, energy metabolism disruption, and apoptosis activation—NPs trigger cascading adverse effects spanning molecular, cellular, organismal, and population levels. Crucially, NPs act as potent carriers for co-occurring pollutants including heavy metals and organic contaminants, often amplifying synergistic toxicity when present in mixtures. This phenomenon significantly exacerbates combined ecological risks, posing a fundamental challenge to conventional risk assessment frameworks predicated solely on single-pollutant evaluations. To address these gaps, future research must transition from acute, single-stressor toxicity assays toward studies that mimic real-world environmental conditions—prioritizing long-term, low-dose exposure paradigms and unraveling toxicity transmission patterns across an organism's lifespan and even transgenerationally. Technologically, leveraging high-resolution tools such as single-cell RNA sequencing and in-situ mass spectrometry will be critical to tracking NP in vivo behavior with spatial and temporal precision. Concurrently, integrating multi-omics data such as genomics, transcriptomics, metabolomics and deploying artificial intelligence (AI)-driven models will enable deep decoding of molecular initiation events in toxic pathways and their network-based cascading amplification mechanisms. Ultimately, translating these advanced scientific insights into actionable management strategiesis imperative. By developing predictive risk assessment models that incorporate combined pollution effects and climate dynamics, this work will establish an indispensable scientific foundation for formulating forward-looking environmental quality standards and precision ecological early warning systems.

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