Review

A Comprehensive Assessment of the Impacts of Metal and Metal Oxide Nanoparticles on Fish

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ABSTRACT

Metal or metal oxide nanoparticles (M/MONPs) have recently transformed into a wide range of sectors, including drug delivery, diagnostics, medicine, cosmetics, sensing, paints, textiles, and energy production, owing to their unique properties such as nanoscale size, large surface area, exceptional mobility, high reaction rates, and quantum effects. Silver, titanium dioxide, copper and copper oxides, iron oxides, aluminum, gold, zinc oxide, and silicon nanoparticles are currently employed across diverse applications. However, improper or unintentional disposal of M/MONPs can adversely affect aquatic organisms. Because these organisms are critical components of the food chain, the potential risks associated with M/MONPs exposure must be carefully evaluated and mitigated. Bioaccumulation, biotransformation, and biomagnification of nanoparticles in aquatic species have raised significant concerns regarding toxicity. This review compiles recent research on nanoparticle release, toxicity, stability, cellular interactions, genotoxicity, and comparative impacts in aquatic environments, with particular emphasis on fish species. It also outlines mechanisms of existing nanomaterial risk management for hazardous nanomaterials and explores future possibilities for safe and sustainable handling. Finally, the review underscores the urgent need for policies and further research aimed at minimizing and controlling nanoparticle toxicity in aquatic ecosystems.

KEYWORDS: metal and metal oxide nanoparticles; toxicity; aquatic environments; biotransformation; fish

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INTRODUCTION

Particles having sizes roughly between 1 and 100 nm in any dimension are classified as nanomaterial [1]. Over the past three decades, researchers have increasingly focused on commercializing nanomaterial-based products due to the exceptional physicochemical properties of various metal and metal oxide nanoparticles (M/MONPs) compared to their bulk counterparts. These properties have the potential to benefit a wide range of sectors, including energy, electronics, construction, food preservation, agriculture, cosmetics, biomedicine, and pharmaceuticals, thereby driving industrial growth and fostering start-up opportunities in the modern era [2].

At the same time, the indiscriminate use of such products may increase environmental concentrations of metal nanoparticles (MNPs), posing potential toxicity risks to living organisms. Toxic MNPs include titanium dioxide, iron oxides, aluminum oxide, copper oxide, silicon dioxide, cerium dioxide, silver, gold, zinc oxide, and carbon nanotubes [3]. Several MNPs, particularly gold, silver, copper, iron, and TiO_2, have attracted significant attention within the scientific community due to their remarkable antibacterial, antiviral, antifungal, antioxidant, anticancer, biosensing, photocatalytic, thermal, and electrical conductivity properties, which enable wide-ranging applications across diverse fields [4,5]. Consequently, global production of MNPs is estimated to reach approximately 2 million metric tons per year [6].

Nanoparticle (NP) accumulation in aquatic ecosystems is higher than in terrestrial ones. It is estimated that approximately 0.4–7% of global NP production is released into water bodies [7]. Other reports indicate that millions of tons of Si, Ti, and Zn-containing nanomaterials ultimately end up in waterways [8]. As a result, aquatic organisms are more affected than terrestrial species due to their greater sensitivity to NP exposure[9]. Ions released from copper oxide, gold, and zinc oxide MNPs disperse readily in aquatic environments, leading to toxicity in biological systems. Numerous studies have highlighted the role of varying MNP concentrations in inducing severe toxic effects in aquatic organisms.

Despite ongoing research into the fate of MNPs in aquatic ecosystems, a substantial gap remains in understanding their effects and behavior in aquatic organisms, making it challenging to predict and mitigate potential risks [10]. The toxicity of MNPs has been shown to cause serious problems in aquatic environments [1,11]. For example, exposure to MNPs was shown to negatively impact early embryonic development, leading to organ malformations. In fish, NPs are known not only to impair development but also to induce genotoxic effects, leading to mutations, cancer, and other life-threatening conditions [12,13]. Water bodies serve as major reservoirs of MNPs, facilitating their transfer through different trophic levels within the ecosystem [14]. As a result, both flora and fauna in aquatic systems are affected, with reported impacts on embryonic growth, metabolic rates, and abnormal behavior and development. For

instance, exposure to MNPs can increase mucus production in fish, leading to fin nipping and aggressive behavior, which may signal gill irritation and potential brain damage. Single-walled carbon nanotubes (SWCNTs) are known to elevate oxidative stress, thereby disrupting fish osmoregulatory and respiratory systems [15,16]. Furthermore, models suggest that the concentration of MNPs in sediments is several orders of magnitude higher than in the overlying water, resulting in greater exposure and risk to benthic organisms [17,18].

Moreover, the unusual and undesirable surface properties of MNPs, including their high surface area, reactivity, and distinctive sizes and shapes, can exert hazardous effects on benthic organisms. Studies have shown that MNPs impact various developmental stages of fish, with embryos being particularly vulnerable to severe and often fatal malfunctions upon exposure. The diverse morphologies and orientations of MNPs confer unique capabilities to interact with physical, biological, and chemical processes within organisms [19]. Additionally, MNPs can penetrate cellular organelles, alter metabolic activity, and trigger apoptosis. They disrupt ion signaling and transport by compromising cell membrane integrity. Cationic MNPs, in particular, destabilize the lipid bilayer, leading to structural changes within the cell [20]. Since aquatic organisms are integral to the food chain, their disruption inevitably affects the broader ecological balance [21,22]. Research has shown that exposure to ZnO NPs increases reactive oxygen species (ROS) production [21,22] and impairs developmental stages in zebrafish by penetrating the chorion layer [23,24].

The effects of various MNPs on animal models have been extensively studied. For example, experiments on zebrafish have shown that MNPs can severely disrupt embryonic development and reproductive processes [25,26]. Additional studies using both in vivo and in vitro cultures of animal tissues demonstrated that exposure to silver MNPs induces oxidative stress and triggers apoptosis [27]. Certain NPs, such as silver, zinc oxide, and copper oxide, also possess antibacterial properties that can accelerate wound healing [28]. However, despite their widespread use in industry, research, and consumer products, global regulatory frameworks governing MNP release remain limited. This regulatory gap has raised growing concern over the need for robust risk assessment strategies [29].

In light of the above, this review presents a critical examination of the formation methods, types, classification, properties, applications, and sources of metal and metal oxide NPs. It further explores the behavior and kinetics of such MNPs, their environmental release, and their fate within aquatic ecosystems. The review also synthesizes existing studies on the toxicity of various MNPs, including copper, silver, iron, selenium, zinc, gold, platinum, nickel, and aluminum, along with their oxides, across multiple developmental stages of fish, from embryos to adults.

CLASSIFICATION OF METAL AND METAL OXIDE NPs

To better understand their unique attributes, specific applications, interaction patterns, and risk assessments based on composition, nanomaterials are commonly categorized into four groups. The first group consists of pure metal-based NPs, such as Ag, Au, and Pt. The second group includes metal oxide NPs, which may exhibit magnetic properties (e.g., Fe_3O_4, Fe_2O_3) or semiconductor characteristics (e.g., TiO_2, ZnO). The third group comprises chalcogenide NPs, including selenides, sulfides, and tellurides (e.g., ZnSe, ZnS, CdS, PbS, CdTe). Finally, the fourth group encompasses metal NPs, metal oxides, and doped metals, such as Pt–Ni and Zn–Ag alloys [30,31]. This classification of NPs is illustrated in Figure 1.

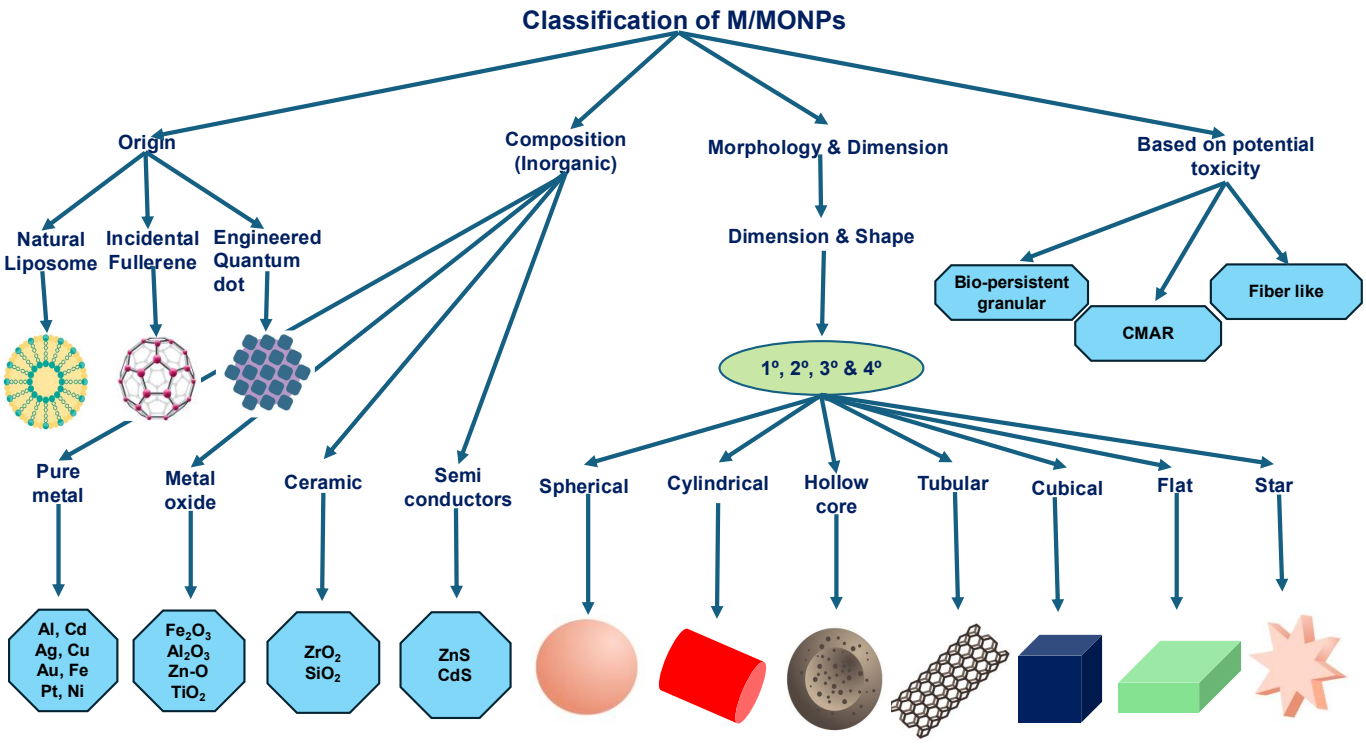

Figure 1. Classification of NPs based on origin, composition, morphology and dimension and potential toxicity.

Pure Metal-Based NPs

Gold, silver, platinum, and other metals have been widely employed in the synthesis of NPs due to their broad availability and non-specific catalytic, antipathogenic, and antibacterial properties [32].

Metal Oxide NPs

These exhibit both magnetic and semiconductor properties. Their semiconductor characteristics have led to rapid growth in gas-sensor, hydrogen generation and photocatalysis applications, thanks to their efficiency and low cost [33–35].

Chalcogenide NPs

Such chalcogenide NPs include ZnSe, ZnS, CdS, PbS, CdTe etc. [11,36]. The unique properties of chalcogenide nanostructures, such as visible photon-capturing ability, defined energy gaps, and their distinct applications in solar energy, photocatalysis, gas conversion, solar-driven fuel generation, sensor technology, and hazardous pollutant removal, have attracted considerable research interest [37,38].

Alloyed Metallic and Doped Metal Oxide NPs

Doped NPs, such as Pt–Ni and Zn–Ag, can be synthesized using methods including flame spray pyrolysis, modified sol–gel techniques, and homogeneous precipitation. Various doped metal oxide NPs are also widely used as doping has been shown to enhance their catalytic, photocatalytic activity, as well as sensitivity towards gases [39].

SOURCES OF MNPS IN AQUATIC BODIES

The precise number of MNPs present in the environment remains unknown, with only limited estimates available for specific nanoparticle concentrations. Current research is particularly focused on NPs used in fertilizers, pesticides, and soil and water remediation products, as these represent major sources of environmental contamination. Carbon-based NPs, metallic NPs, and metal oxides are among the most extensively studied [40,41]. Aquatic systems become contaminated through multiple pathways, including direct MNP entry during remediation activities, accidental leaks or product use, atmospheric deposition, effluents from wastewater treatment plants, and run off from polluted soils [16,42,43] (see Figure 2).

Landfills are known as significant sources of ecosystem contamination [16]. Wastewater treatment processes remove a substantial portion of NPs from polluted water; for instance, about 97% of Ag NPs were reported to be transferred from the water column into sewage sludge [44]. This sludge is sometimes applied to soil due to its high organic matter content and the presence of metal oxides such as ZnO. While certain MNPs in sewage sludge compost can enhance soil fertility, their broader environmental implications require careful evaluation [45], they also pose hazards because of their high predicted concentrations. These concentrations can range from tens of mg/kg/L for ZnO NPs, hundreds to thousands of mg/kg/L for TiO_2 NPs, and dozens of μg/kg/L for Ag NPs [16,25,46]. Predictive models estimate that European surface waters contain tens to hundreds of ng/L of ZnO NPs, tens to thousands of ng/L of TiO_2 NPs, and units to tens of ng/L of Ag NPs [14,23,44].

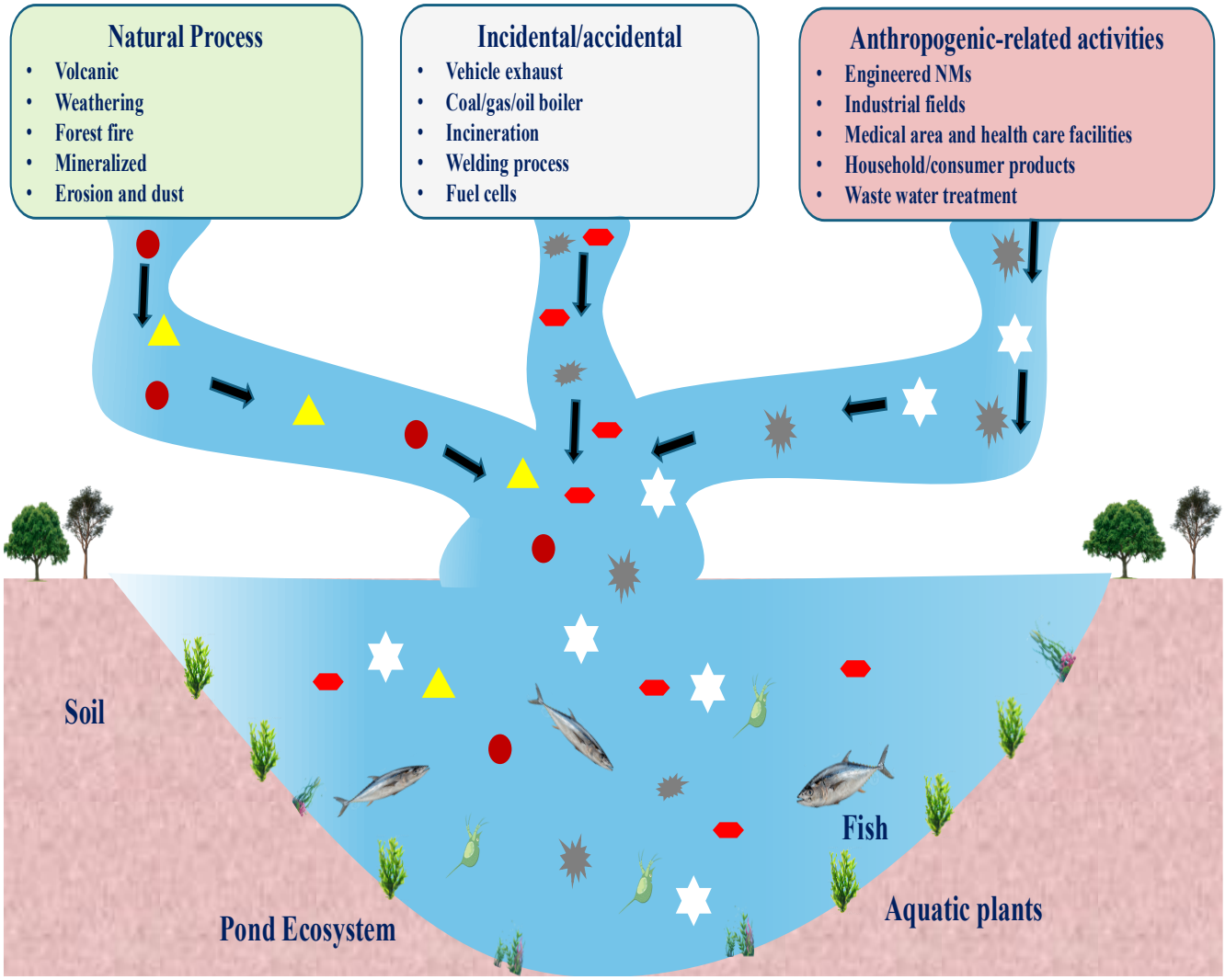

Figure 2. Various sources of nanoparticle release and contamination in aquatic ecosystems.

NATURE AND STABILITY OF METAL-ION-CONTAINING NPS IN AQUATIC BODIES

Metal-containing NPs are generally hydrophilic but demonstrate low solubility in water, which partly explains why metal ions like Ag^+ are less toxic than silver NPs [47]. In natural waters, soluble Ag^+ ions rapidly react to form insoluble, less toxic compounds such as chlorides and sulfides, which readily settle out of the water column [48]. Once NPs enter aquatic environments, they undergo a series of interactions, including the ones described below (see Figure 3).

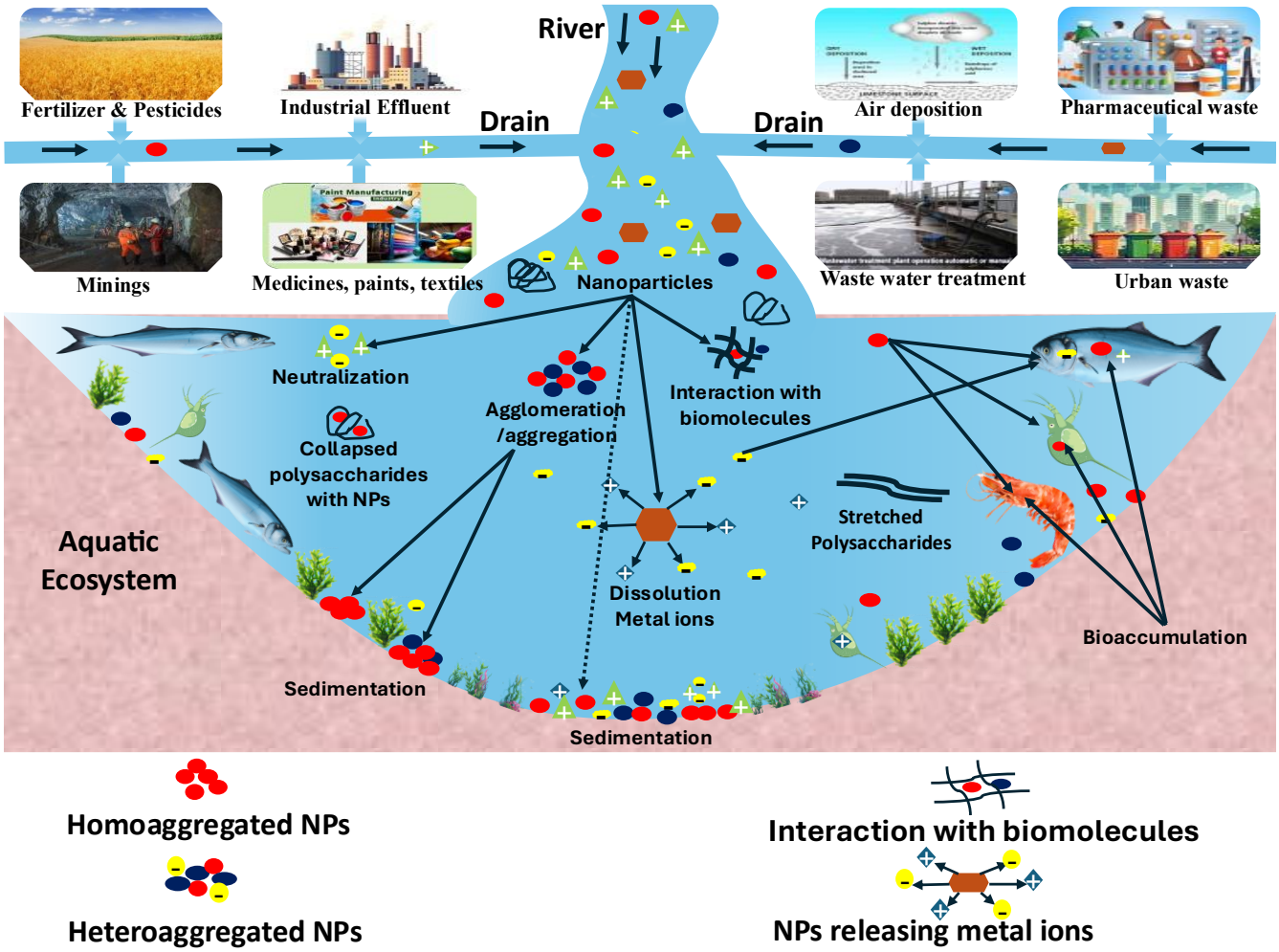

Figure 3. Sources of NP release and their interactions in aquatic ecosystem.

Aggregation

Aggregation and agglomeration are terms that are often used interchangeably in the NP field, but they refer to different processes. Aggregation involves permanent structural changes caused by irreversible chemical interactions, such as the formation of covalent bonds. In contrast, agglomeration refers to the reversible association of NPs through weak physical interactions, including electrostatic attraction, hydrogen bonding, or van der Waals forces [49]. Sedimentation is typically preceded by particle aggregation, which depends on factors such as pH, ionic strength, the presence of cations, and the size, shape, and charge of the particles [50–52] (see Figure 3). In marine and brackish water environments, the high ionic strength promotes rapid NP aggregation, resulting in lower NP concentrations in the water column [53,54]. Aggregation may occur as homoaggregation (among NPs) or heteroaggregation (between NPs and naturally occurring colloids). These colloids include inorganic substances (e.g., metal oxides, sulfides, and amorphous silica), organic compounds (e.g., polysaccharides, humic substances, and microbial proteins), and biological agents (e.g., bacteria

and viruses). Fibrous materials tend to enhance NP aggregation, whereas humic substances can coat NP surfaces and neutralize their charges, thereby promoting dispersion [48,52].

Effects of NP shape and size

Irregularly-shaped NPs, such as nanowires, nanotubes, and nanorods [55], along with their small size, which modifies surface reactivity, electronic structure and surface charge [56], are known to significantly influence their aggregation behavior in aqueous environments.

Impact of solution chemistry on NP aggregation

Several studies have demonstrated that variations in aqueous media chemistry, such as ionic strength and pH, significantly influence the nature of NP aggregation [57,58]. These factors regulate the surface charge and charge density of metal-containing NPs, thereby affecting their stability. When the surface charge approaches zero, electrostatic repulsion diminishes, leading to enhanced Van der Waals attraction and increased aggregation. For instance, for TiO_2 NPs, Guzman and coworkers observed an increase in the hydraulic diameter of aggregates as the solution pH reached the zero-point charge pHzpc [59,60].

Impact of surface coating and hydrophobicity

Surface-coated MNPs can be stabilized through enhanced steric, electrostatic, or electrosteric repulsion between particles, which collectively influence their aggregation behavior [61,62]. Typically, three types of surface coating are employed: surfactants, polymers, and polyelectrolytes [63,64]. Surfactants, whether covalently bound or adsorbed, contribute to aggregation stability by increasing electrostatic repulsion and surface charge, or by lowering the interfacial energy between the solvent and particles [65,66]. Polymers such as polyvinylpyrrolidone (PVP) stabilize MNPs via steric repulsion. For instance, when compared to bare Ag NPs, PVP-coated Ag NPs were reported to exhibit a critical coagulation concentration value that was four times higher, indicating improved colloidal stability [67,68]. Cationic polyelectrolytes, such as polydiallyldimethylammonium chloride (PDDA), protect NPs from oxidation and agglomeration, thereby promoting aggregation under certain conditions [69].

Nanoparticle aggregation is also influenced by surface hydrophobicity, which alters the Hamaker constant (AH), a parameter that reflects the strength of mutual attraction between interacting particles [70].

Effect of natural organic matter

Natural organic matter (NOM), primarily composed of humic and fulvic substances, can modify the physicochemical properties and interfacial interactions of MNPs by adsorbing onto their surfaces, thereby influencing

aggregation behavior [71]. NOM has been shown to slow NP aggregation even under conditions of elevated ionic strength [6]. The impact of humic acid (HA) on NP aggregation depends on both ionic strength and the nature of the electrolytes present. For gold NPs, HA promotes aggregation in the presence of monovalent cations at low ionic strength; however, high concentrations of divalent ions significantly enhance aggregation [72].

Impact of solution dissolved oxygen and temperature

Temperature modulates the kinetics of metal-containing NP aggregation by influencing the Brownian motion of particles [73]. As temperature increases, the kinetic energy of such NPs rises, leading to higher collision frequencies and enhanced aggregation rates [74]. In aquatic systems, dissolved oxygen (DO) contributes to the oxidation of metallic NPs. For silver NPs, DO has been shown not only to promote aggregation but also to facilitate the formation of silver ions (Ag^+) [75,76]. Under these conditions, hydrodynamic diameters exhibit random distributions and periodic fluctuations. The aggregation rate of Ag NPs has been reported to be three to eight times faster in the presence of dissolved oxygen [77].

Agglomeration

Primary particles form loosely packed clusters through readily reversible weak van der Waals interactions. Strongly fused clusters lead to the formation of undesirable aggregates, as they reduce surface area and alter reactivity, catalytic behavior, optical properties, and biological interactions (e.g., antimicrobial activity). Aggregation can be prevented through strategies such as surface coating, electrostatic and steric stabilization, optimized synthesis conditions, sonication, dispersing agents, solvent selection, increasing zeta potential, mechanical dispersion, and proper storage conditions [78].

Neutralization

Neutralization of NPs occurs through changes in pH, the addition of electrolytes, and interactions with oppositely charged molecules. These processes influence NP stability, leading to agglomeration or aggregation, sedimentation, reduced mobility, and altered reactivity. However, neutralization plays a significant role in applications such as wastewater treatment, material synthesis, drug delivery, sensor development, and catalyst fabrication [79]

TOXICODYNAMICS OF METALLIC NPS IN FISH

Nanoparticles affect aquatic organisms through multiple mechanisms that often result in similar biological outcomes. The most prominent mechanism is the elevation of ROS production, which induces oxidative stress. This stress destabilizes cellular membranes through lipid

peroxidation, ultimately leading to genotoxic effects and cell death [80,81]. In addition, metal-containing NPs significantly increase cytotoxicity by releasing metal cations that form complex with thiol groups of enzymes and proteins, thereby inhibiting their functions [82,83]. Thiol containing enzymes, such as lactate dehydrogenase and glutathione, play critical roles in mitigating oxidative stress [84].

Silver ions inhibit Na^+/K^+ ATPase activity, blocking the active uptake of Na^+ and Cl^- ions in the basolateral cells of fish gills, thereby disrupting osmoregulation and leading to mortality [85–87]. Yue and colleagues demonstrated that this inhibition was caused by both $AgNO_3$ and Ag NPs, with a 2.3% dissolution rate of Ag^+ accounting for approximately 16% of the observed effect [88]. Mechanical effects are closely linked to the aggregation rates of metal-containing NPs. Adsorption of such NPs onto fish gills can obstruct the exchange of respiratory gases, as reported in previous studies [89,90]. In fish embryos, NP aggregation on the chorion surface can block pore canals, leading to hypoxia and a subsequent reduction in hatching rates [91,92].

NPs can also act as 'Trojan horses' due to their small size, large surface area, and strong sorption capacity, enabling them to bind environmental contaminants and facilitate their transport into organisms [93]. This phenomenon was observed by Zhu et al. [94] in danio sp. embryos co exposed to TiO_2 NPs and tributyltin. While TiO_2 NPs alone at 2 mg/L exhibited no developmental toxicity, their presence increased tributyltin toxicity 20-fold, significantly reducing hatching rates. For metal-based NPs with antimicrobial properties, such as ZnO, TiO_2, and Ag NPs, their effects on the diversity and composition of fish microbiomes warrant careful consideration [95,96].

KINETICS OF METALLIC NPS

Aquatic organisms can internalize NPs, which can adversely impact biological systems across diverse taxa, including algae, invertebrates, and fish [97,98]. In fish, NPs may be absorbed via multiple epithelial pathways: the gastrointestinal tract (through feeding and drinking), the skin, and the gills [99,100] (see Figure 4). Among these pathways, transdermal uptake is relatively limited due to protective mucus secretion, which chelates NPs, and the absence of metal transporters, unlike the epithelium of fish gills [100,101]. Nevertheless, endocytic uptake of silver NPs has been documented, with subsequent localization in endosomes and lysosomes of RTgill-W1 gill cells [88] (see Figure 5). Importantly, the intracellular distribution of Ag NPs differs from that of silver ions (Ag^+), which are predominantly found in the cytosol and associated with metallothionein-like protein fractions.

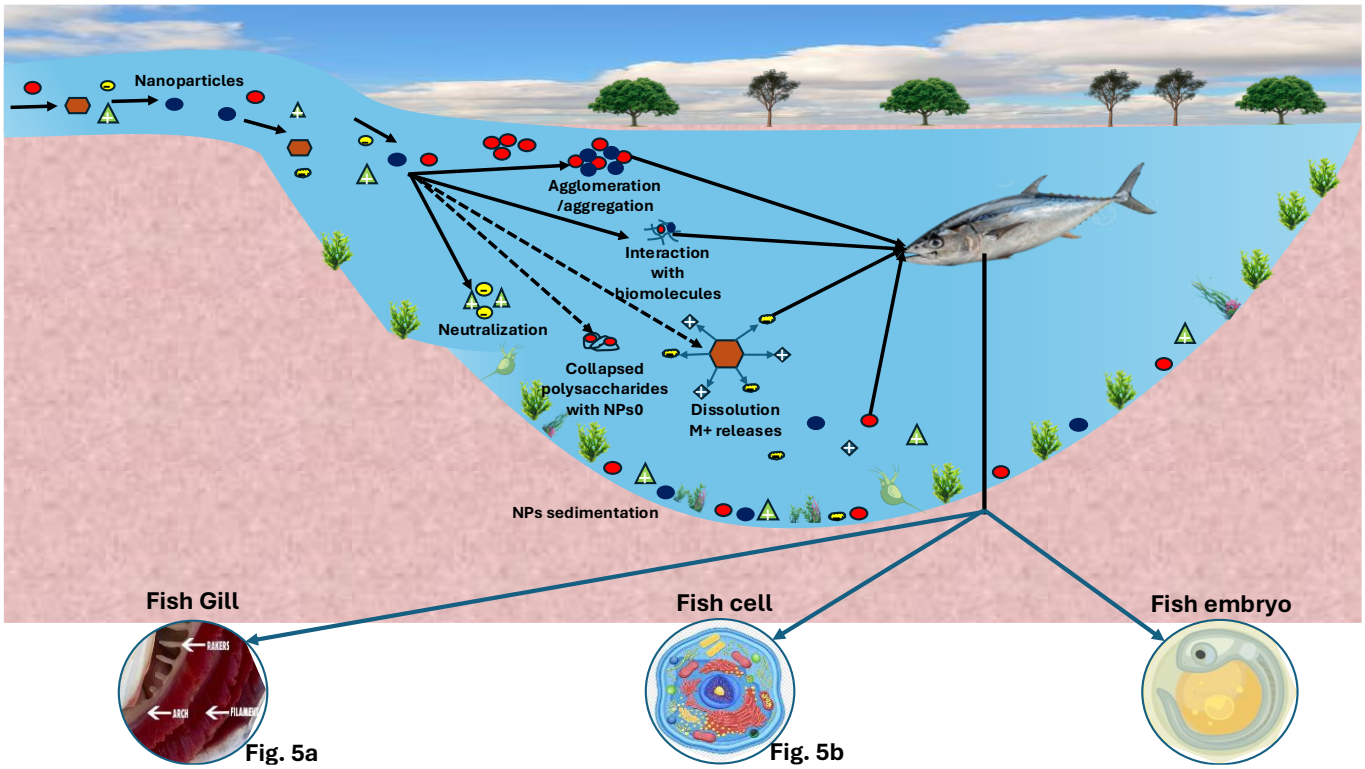

Figure 4. Toxicodynamics of MNPs in aquatic ecosystem.

Gastrointestinal uptake of metal-containing MNPs is supported by findings from Gaiser et al. [98], who exposed *Cyprinus carpio* (common carp) to nano- and micro-sized Ag particles for 21 days. They observed silver accumulation in several vital organs, including the intestine, gills, blood, kidney, liver, gallbladder, and brain [98,102]. Chronic dietary exposure to ZnO NPs in *Cyprinus carpio* was shown to adversely affect immune function and homeostasis, inducing nephrotoxicity and hepatotoxicity despite limited tissue accumulation [103]. Ag NPs have also been found to partially compromise cellular and lysosomal membrane integrity and metabolic activity [104]. Structural damage such as epithelial inflammation or erosion may weaken natural barriers, thereby facilitating NP translocation to internal organs via the circulatory system (see Figure 5a,b). Notably, Chupani et al. [105] documented increased apoptosis in the intestinal epithelium and elevated expression of proteins linked to cancer cell survival of the intestinal mucosal layer following ZnO NP exposure in *Cyprinus carpio*.

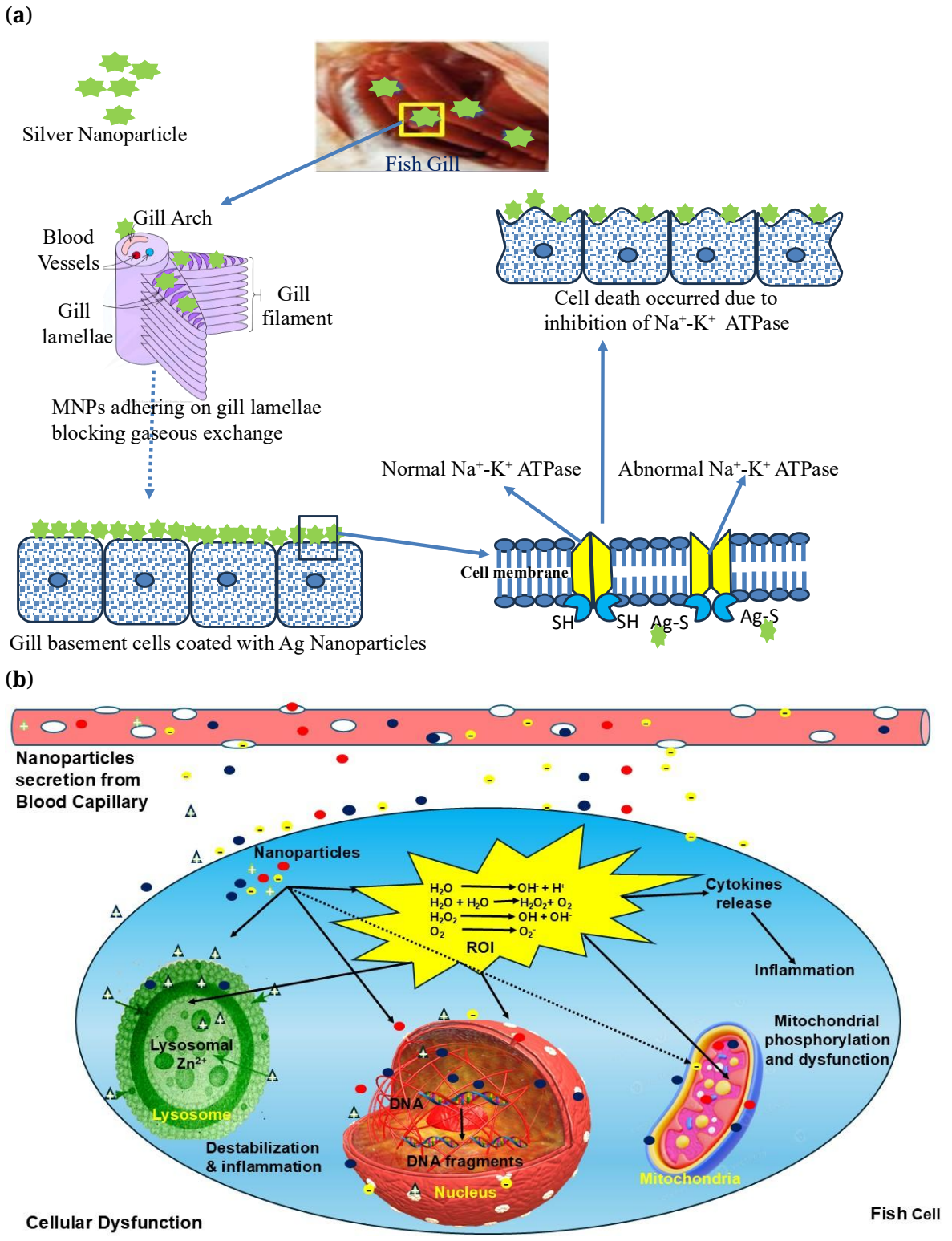

Figure 5. Toxicodynamics of MNPs at organ, cellular and embryonic levels. (**a**) Cellular necrosis of fish gills caused by AgNPs. (**b**) Toxicity of metal/metal oxide NPs in fish cells.

TRANSPORT OF METAL AND METAL OXIDE NPS IN VARIOUS TROPHIC LEVELS

Bioaccumulation and transfer of metal-containing NPs through the food chain across different trophic levels can lead to biomagnification

within aquatic ecosystems (see Figure 6) [99,106]. Ates et al. [107] demonstrated the accumulation of CuO and ZnO NPs in the gills, intestine, and liver of both *goldfish (Carassius auratus*) and *Crustaceans* (*Artemia salina*). Similarly, Ates et al. and Chen et al. [107,108] reported the trophic transfer of TiO_2 NPs from algae (*Scenedesmus obliquus*) to aquatic fleas (*Daphnia magna*), resulting in biomagnification. A comparative study by Yoo et al. [109] evaluated the bioaccumulation and biomagnification of Ag NPs across multiple trophic levels, including green algae (*Chlorella* spp.), bloodworms (*Chironomus* spp.), aquatic fleas (*Moina macrocopa*), and silver barb (*Barbonymus gonionotus*), and found substantial accumulation in algae but relatively low levels in fish. The trophic transfer of M/MONPs from aquatic ecosystems to terrestrial food webs has serious implications for human health, contributing to disorders such as neurodegenerative diseases and cancer [110]. It has been reported that prolonged consumption of mercury-contaminated fish can lead to neurological disorders in humans, characterized by symptoms including muscle weakness, ataxia, limb numbness, and impairments in speech and swallowing [110].

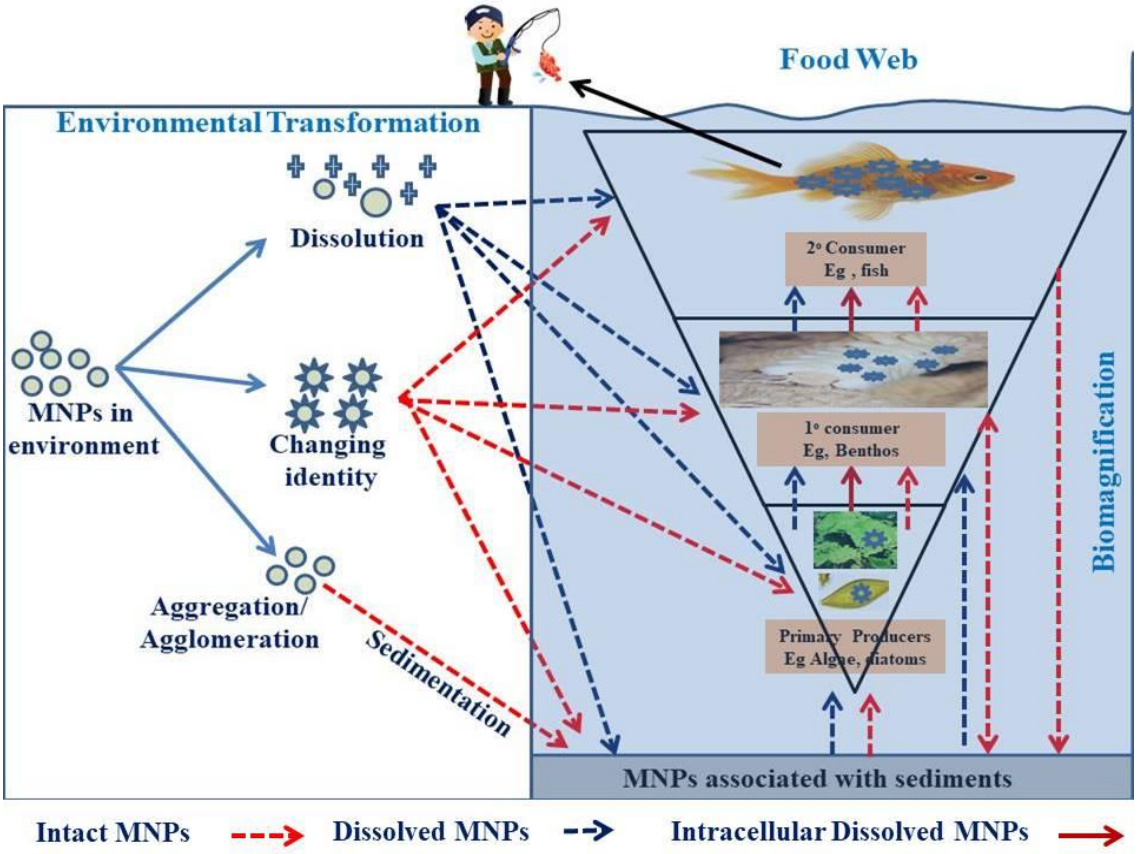

Figure 6. Effects and dynamics of metal-containing NPs across different trophic levels.

IMPACT OF NANOPARTICLES

Large NP accumulations in aquatic environments have harmful effects on multiple stages of fish development. For example, increased

concentrations of silver NPs were found to elevate stress-related molecule levels in young salmon, which inhibited the activity of Na^+/K^+-ATPase enzymes, leading to osmoregulation failure [111]. Exposure to approximately 100 μg/L of silver nanoparticles induced necrosis in gill lamellae and resulted in a 73% mortality rate among the fish [111]. Similarly, studies on juvenile *zebrafish* reported a lethal concentration (LC_{50}) of about 1.78 mg/L for dissolved copper and 0.71 mg/L for copper NPs, demonstrating that MNPs are more toxic than their dissolved counterparts.

Extensive evidence indicates that MNPs can induce adverse effects, including inhibition of Na^+/K^+-ATPase activity, increased oxidative stress, and tissue degradation [112]. In fish, NPs are rarely excreted through the kidneys but can be eliminated via bile secretion [24].

Silver NPs

These days, silver NPs are widely utilized across various industries worldwide, including in textiles, medical imaging, and as antimicrobial agents. Despite their beneficial applications, numerous studies have reported undesirable impacts on aquatic lives (see Table 1). For example, in *Salvelinus alpinus* and *S. fontinalis*, a 24-h intravenous exposure to 5 nm polyvinylpyrrolidone-coated silver NPs (nAg-PVP) resulted in severe pacemaker dysfunction and disrupted cardiomyocyte iono-regulation [113]. Similarly, exposure to 5.54 ± 2.2 nm spheroidal Ag NPs coated with polyvinyl alcohol (Ag NP–PVA) caused elevated hematological parameters, including hematocrit, hemoglobin, glucose, total plasma protein, red blood cell count (RBC), mean hemoglobin concentration (MHC), mean corpuscular volume (MCV), and white blood cell count (WBC), as well as respiratory disorders in adult *Colossoma macropomum* [114].

In *Nile tilapia*, silver NPs were reported to produce oxidative stress, hepatotoxicity, genotoxicity, and epithelial cell hyperplasia [115]. In embryos of *Danio rerio* (*zebrafish*), silver NPs stabilized by polyvinylpyrrolidone, maltose, and gelatin decreased hatching and heart rates, while causing yolk sac edema and spine deformities [92]. Additional studies found sterility and growth rate reduction linked to Ag NP exposure [116]. At the molecular level, Ag NPs were shown to upregulate pro-inflammatory and pro-apoptotic genes such as IL-1β, TNF-α, and caspases while downregulating key embryonic development genes (sox17, gsc, ntl, otx2) [117].

Moreover, 22–26 nm spherical polyvinylpyrrolidone-coated Ag NPs were found to elevate liver oxidative stress, altered detoxifying enzymes, and reduced acetylcholinesterase (AChE) activity in the brains of *zebrafish* [118]. Neural development-related genes were negatively regulated, whereas metallothionein genes were positively upregulated following exposure [119] In adult *Pimephales promelas*, mucous secretion triggered by citrate- and polyvinylpyrrolidone-coated Ag NPs exposure led to renal and cardiac necrosis [120].

Table 1. Recent studies on Ag NP toxicity across various fish species.

Nanoparticles	Size (nm)	Model	Stage	Duration	Effect	Ref.
Dietary Ag NPs	61.9	*Denio rerio*	Adult	28 days	Ag NPs accumulated in body organs like liver and spleen led to weight loss in male fish at high-dose exposure (10–50 mg/kg)	[121]
Ag NPs	3.76 ± 1.00	*Denio rerio*	Adult	96 h	Exposure to 0.0331, 0.250, and 5.00 µg/L caused breakdown of the integumentary barrier, manifested as epidermal lesions and altered mucus, with increasing severity at higher concentrations	[122]
Ag NPs	45 ± 5 spherical	*Oreochromis niloticus*	Adult	2 weeks	Dose (0, 1, 3, 6, 9, 12 µg/L) dependent impact on blood parameters and liver enzyme activities were observed.	[123]
$AgNO_3$ NPs	20 and 40	*Cyprinus carpio*	Fingerlings	28 days	Exposure to high concentrations of $AgNO_3$ NPs (150 mg/L) altered fish growth rates and affected liver function	[124]
Thyme Ag NPs	5	*Oreochromis niloticus, E. faecalis*	Adult	96 h	Exposure to Ag NPs reduced immune function, increased oxidative damage, and caused histopathological alterations along with changes in gene expression	[125]
Ag NPs with Polystyrene nanoplastics(PS NPs)	20	*Denio rerio*	Larvae	120 hpf (hour post fertilization)	Lipid metabolism disorder, hepatomegaly, oxidative stress and liver dysfunction were observed.	[126]
nAg PVP	5	*Salvelinus alpinus, S. fontinalis*	Adult	24 h	An IV dose of nAg-PVP (>500 µ/L) to fish disrupted the regulation of pacemakers and cardiomyocyte ion regulation, leading to life-threatening symptoms.	[113]
Ag NPs	80 ± 5	*Denio rerio*	Adult	96 h	No alterations were observed at low concentrations, while sequential increases in exposure levels led to elevated mortality and pronounced respiratory stress	[127]
Ag NPs	95.32 ± 1.47, nearly spherical	*Corbicula fluminea*	Adult	48 h	Higher concentration of Ag NPs (125 mg/L) caused severe tissue damage in multiple organs, induced oxidative damage by unbalancing oxidative enzymes, increased inflammation and reproductive toxicity	[5]
Ag NPs	NA	*Ctenopharyngodon Idella*	Adult	14 and 28 days	Exposure to 30 mg/L Ag NPs caused DNA damage in RBC and alteration in RBCs	[128]
Fabricated Ag NPs	NA	*Cyprinus carpio*	Adult		The highest concentration of fabricated Ag NPs (0.08 mg/L) accumulated in the gills and intestine and caused notable histological alterations	[129]
Ag NPs	Spherical, nanowires, nanoflakes	*Danio rerio*	Adult	48 h	Spherical Ag NPs caused more severe toxicity in multiple organs (brain, liver, and gills) than Ag nanowires and nanoflakes	[130]
Ag NPs		*Ruditapes philippinarum*	Adult	7 days	Increased oxidative stress caused by exposure to 5 µg/L Ag NPs exposure led to DNA damage, mainly in gills	[131]
Ag NPs-PVA	5.54 ± 2.2, spheroidal	*Colossoma Macropomum*	Adult	10 days	Dose of 187.5 µg/L caused elevation in hematocrit, total plasma protein, glucose, hemoglobin, RBC, MCV, MHC, & WBC, as well as respiratory disorder	[114]
Ag NPs	30–60	*Nile Tilapia*	Adult	60 days	1.98 mg/L Ag NPs increased oxidative stress and led to hepatotoxicity and genotoxicity	[115]

Ag NPs	30	*Capoeta capoeta*	Adult	96h	Ag NPs influenced negatively hematological, biochemical parameters as well as anatomy of liver and gills	[132]
Ag NPs	NA	*Oreochromis mossambicus*	Adult	7 days	Ag accumulated in gill's tissue increased oxidative stress, damaged gills, and caused epithelial cell hyperplasia	[133]
Ag-β-cyclo -dextrin	130	*Danio rerio*	Adult	10 days	With no morphological changes, gills, kidneys, and liver of *danio rerio* get damaged after exposure to 30µl/L Ag NPs	[134]
Ag NPs	11.11 ± 3.40	*Cyprinus carpio*	Fingerling	24 h and 7-day recovery	After exposure to 0.1 mg/L Ag NPs, Ag accumulated in gills, causing epithelial hyperplasia and lamellar fusion.	[135]
Ag NPs	20	*Pangasianodon hypothalamus*	Juvenile	14 days	Increased ALP, LDH, AST, ALT, and decreased T3 were observed after exposure to 0, 3.37, 7.46, 18.66 mg/L Ag NPs	[136]
Ag NP (PVP stabilized, and maltose and gelatin stabilized)	58.4 ± 8.9 and 30.7 ± 0.6	*Danio rerio*	embryo	96 h	The Hatching rate decreased on PVP stabilized Ag NPs exposure. Oedemas in yolk sac, heart and spine deformations on maltose and gelatin-stabilized Ag NPs exposure were found. LC 50 for Ag NPs exceeded 100mg/L	[92]
Ag NPs	<100	*Oncorhynchus mykiss*	Juvenile	28 days	Mortality increased and body weight decreased	[137]
Ag NPs	NA	*Cyprinus carpio*	Adult	24 h & 96 h	Exposure to 0.1 ppm and 0.5 ppm Ag NPs led to LC_{50} of 0.1 ppm. Fish exhibited several physiological and cellular abnormalities, including increased mucus secretion, DNA damage, elevated heart rate, and damage to stem cells, liver cells, and the swim bladder	[116]
Ag NPs and $AgNO_3$	0.1–5 and 0.01–0.1	*Carassius auratus*	Adult	14 days	Edema, hyperplasia, lifting of gill epithelium, fusion of gill lamellae, hemorrhage and hemosiderosis. $AgNO_3$ (0.1 ppm) proved more potent than Ag NPs (5 ppm).	[138]
Ag NPs (PVP coated)	10–20	*Danio rerio*	Embryo	48 h	As dose increased, mortality also increased, accompanied by hatching delays, pericardial edema, and notochord deformities. Expression levels of marker genes involved in early embryonic development were significantly reduced	[117]
Ag NPs	50	*R. labio*	Adult	7 days	Hematological parameters decrease, antioxidant enzymes increase, and histological changes in muscle, gill, and liver	[139]
Ag NPs (citrate and PVP coated)	20 and 20	*Pimephales promelas*	Adult	24 h	On exposure to 50.3 µg/L and 56.0 µg/L, production of mucus increased by 4 h, but decreased beyond 28 h. Hypertrophy and necrosis in heart, renal necrosis, and positive acute-phase proteins were observed	[120]
Ag NPs	4 and 10	*Danio rerio*	embryo	96 h	Exposure to Ag NPs in embryos resulted in the development of small eyes and heads, cardiac defects, and a hypoplastic hindbrain. Significant Ag accumulation in the head region was observed.	[119]
Ag NPs (citrate and PVP coated)	20	*Pimephales promelas*	Adult	24 h	Gill's goblet cells and Na^+ /K^+-ATPase activity decreased. Ionic Ag was found	[140]

					more potent while citrate-coated silver NPs showed higher toxicity (10 ± 0.32 vs. 2.4 ± 0.6)	
Ag NPs	1-13	*Onchorhynchus. Mykiss*	Eleuthero embryos, larvae, and juveniles	96 h	When treated in water containing 100, 32, 10, 3.2, 1, 0.32, 0.1, and 0.032 mg/L, LD 50 values were 0.25, 0.71, and 2.16 mg/L. respectively found.	[141]
Ag NPs	11.3	*C. carpio*	Adult	7 days	Ag accumulates in the gill, liver, gastrointestinal tract, brain, and skeletal muscle.	[142]
Ag NPs (Polyacrylate sodium stabilized)	8.39 ± 0.98	*Danio rerio*	Embryo	96 h	Mortality elevated, and morphological deformities, bradycardia, and hatching delay occurred. ROS production increased, but glutathione level decreased.	[143]
Ag NPs (Spherical)	120	*Danio rerio*	Embryo	120 h	Hatching rate was suppressed, and morphological defects occurred at high concentrations.	[144]
Ag NPs (PVP coated)	81	*Danio rerio*	Adult	48 h	The operculum movement and breathing rate increased, with LC_{50} being of 84 µg/L	[145]
Ag NPs (Stabilized with bovine serum albumin and Starch)	5–20	*Danio rerio*	Embryo	96 h	Ag NPs stabilized with bovine serum albumin and starch caused dose-dependent increases in mortality and non-hatched embryos, along with pericardial edema, notochord deformities, bradycardia, and cardiac arrhythmia	[146]
Ag NPs	26.6 ± 8.8	*Danio rerio*	Adult	48 h	Ag NPs accumulated in gills and the whole body, with no morphological changes observed. Exposure of Ag NPs and ionic Ag affected gene expression differently	[147]
Ag NPs	3, 10, 50, and 100, spherical	*Danio rerio*	Embryo	120h	Size-dependent elevation in death rate and sublethal effects, with higher toxicity for smaller NPs. Ag NP demonstrated higher toxicity than Au NPs	[148]
Ag NPs	10–20	*Danio rerio*	Embryo	72 h	Decrease in hatching rate, abnormal notochord, damaged eyes, weak heartbeat, curved tail, and elevated catalase activity were recorded over dose	[149]

Gold NPs

Gold NPs have gained widespread popularity due to their relatively simple preparation, catalytic activity, stability, optoelectronic properties, high biocompatibility, and low toxicity. These characteristics enable their application across diverse technical fields, including photovoltaics, probes, electronics, catalysis, and sensing. In medicine, Au NPs play a pivotal role in both diagnostics, such as tumor detection and imaging, and therapeutic approaches, including drug delivery and photothermal therapy [150–152]. Their extensive global use has resulted in an annual production volume estimated at 1–3 tons [3,153].

Despite their benefits, Au NPs have demonstrated various adverse effects on aquatic organisms (see Table 2). In *Danio rerio* (*zebrafish*), citrate- and polyvinylpyrrolidone-coated Au NPs were found to cause cardiac edema and behavioral changes [92]. In juvenile *Sparus aurata*, exposure to Au NPs was reported to lead to red blood cell destruction and

DNA damage [154]. Citrate-capped Au NPs also elevated the genetic expression of catalase, superoxide dismutase, and metallothionein in embryos, and altered brain acetylcholinesterase (AChE) activity along with oxidative stress and mitochondrial metabolism gene expression in adult zebrafish [155]. Furthermore, such NPs were reported to influence the expression of pro-apoptotic and DNA repair genes, contributing to mitochondrial dysfunction and DNA mutations [156] (see Table 2).

Table 2. Recent studies on Au NP toxicity across various fish species.

Nanoparticles	Size (nm)	Model	Stage	Duration	Effect	Ref.
Au NPs	4 and 82	*Denio rerio*	Embryo	4 hpf (hour post fertilization)	Au NPs exposure inhibited hatching of embryo and induced HE expression. Pxr (pregnane X receptor) also affected locomotor activity.	[157]
Au NPs	NA	*Oreochromis niloticus*	Adult	50 days	Increase in dose concentration showed negative impact on liver and gill histology as well as function	[158]
Glutathione conjugated gold nanoclusters (GSH-Au NCs)	380	*Denio rerio*	Embryos	14 days	The exposure of GSH-Au NCs at 6-20 μg/mL decreased survival and hatching rates.	[159]
BRP-Au NPs (Brazilian red propolis)	523	*Denio rerio*	Embryo	96 h	BRP-GNPs induced developmental toxicity in dose-dependent manner.	[160]
Pristine Au NPs	13-84	*Denio rerio*	Embryo	48 hpf	Most of eggs coagulated, heartbeat of embryo stopped and development retarded on exposure of 0.25 mg/mL of Au NPs	[161]
Au NPs	NA	*Ctenopharyngodon Idella*	Adult	14 and 28 days	Au NPs caused DNA damage in RBC and alteration in RBC	[128]
Au NPs	10–100	*Danio rerio*	Larva	96 h	Persistence of some Au NPs for long term exposure impacted on renal tissues.	[162]
Au nanorods	20–50 987654236	*Danio rerio*	Adult	168 h	Significant elevation in activity of superoxide dismutase, lipid peroxidation and catalase was observed after exposure to 0.03 mg/L Au NPs.	[163]
Au NPs (Citrate capped and PVP capped)	1.16 and 11.16	*Danio rerio*	Embryo	96 h	Lower toxicity was observed overall, although cardiac edema occurred, and movement ceased at higher concentrations	[164]
Au NPs (Citrate and PVP coated)	7 and 40	*Sparus aurata*	Juvenile	96 h	Au NPs accumulated in liver and spleen, altering the expression of around 26 proteins	[165]
Au NPs-Citrate capped	14 ± 2	*Danio rerio*	Embryo	96 h	Exposure to 20 mg/L resulted in increased expression of catalase, superoxide dismutase, and metallothionein genes. In addition, swimming speed decreased, and overall swimming patterns were altered	[155]
Au NPs (Citrate and PVP coated)	45	*Sparus aurata*	Juvenile.	96 h	Exposure to 80μg/L of AuNPs caused RBC nuclear abnormalities	[154]
Au NPs citrate coated	40	*Sparus aurata*	Adult	24 h	Short term exposure to Au NPs (50 μg/L) modulated expression of genes related to liver and many biochemical parameters of fish	[166]
Au NPs PEG-coated	10 nm	*Hypostomus plecostomus*	Adult	48 h	Subchronic exposure to 0.48 and 4.8 mg/L caused inflammatory reponses in liver, spleen, and muscle tissues	[167]

Au NPs citrate reduced	50	*Sparus aurata*	Juvenile	96 h	Concentration of ~1600 µg/L of Au NPs induced alteration in gene expression of gills.	[168]
Au NPs	38.1 ± 2.8, nanospheres	*Danio rerio*	Embryo	72h	Nanorod exposure increased mortality and upregulated oxidative-stress-related genes. Hatching rate decreased, and bradycardia was observed. Surface coating and double-coating of the nanorods substantially reduced their toxicity	[151]
Au NPs (coated with citrate and PVP)	40	No specific fish	Adult	96 h	Exposure to 1600 µg/L and 80 µg/L of Au NPs coated with citrate and PVP lead to modulation of gene expression related to cell-tissue repair, oxidative stress, immune function and apoptosis	[169]
Au NPs	14	*Danio rerio*	Adult	20 days	Brain AChE activity increased, and the expression of genes related to oxidative stress, mitochondrial metabolism, and DNA repair was modulated following exposure to Au NPs at concentrations of 0.25 ± 0.05 and 0.8 ± 0.1 µg/L	[170]
AuNPs	12 and 50	*Danio rerio*	Adult	36 and 60 days	Au NPs exposure altered the expression of genes enrolled in DNA repair, detoxification, apoptosis, mitochondrial and brain dysfunction.	[171]
Au NPs	1.5	*Danio rerio*	Embryos	122 days post fertilization	Acute exposure (50 µg/mL) impacted on larval behavior persisting even in adulthood	[172]

Titanium Dioxide NPs

Titanium dioxide (TiO_2) NPs are widely used as photocatalysts in solar panels, paints, plastics, pharmaceuticals, and even as food coloring agents. Owing to their transparency and UV protective properties, they are also commonly incorporated into cosmetics, particularly sunscreens [173,174]. Global production of TiO_2 NPs is estimated at approximately 3000 tons per year [175]. Titanium dioxide is valued for its catalytic activity, electrical conductivity, high light reflectance, and high refractive index, and it is both chemically stable and insoluble in water [176].

Although generally considered safe and non-toxic, TiO_2 NPs, especially those smaller than 25 nm, have demonstrated adverse biological effects at different level (see Table 3). In juvenile *Acipenser schrenkii*, 25-nm-sized TiO_2 NPs were reported to disrupt lipid metabolism and altered the KEGG pathway related to immune response [177]. Exposure in adult *Oreochromis niloticus* resulted in liver and intestinal damage, erythrocytic DNA impairment, and downregulation of antioxidant and apoptosis-related genes. In juvenile hybrid groupers, it also triggered the upregulation of inflammatory genes such as TNF-α [15]. Chronic exposure in adult *Oncorhynchus mykiss* resulted in a 24% increase in brain acetylcholinesterase (AChE) activity [178]. Histopathological alterations have also been reported, including epithelial cell separation, fusion and thickening of the secondary gill lamellae, edema, and hemorrhage in fingerlings of fish [179]. Accumulation of TiO_2 NPs increased the risk of

pathomorphological alterations in the liver and spleen and reduced bacterial resistance in *Pimephales promelas* [180].

Titanium dioxide exists mainly in three crystalline forms, anatase, brookite, and rutile, with anatase being the most chemically reactive form [181]. TiO_2 particles smaller than 25 nm have been shown to cause early hatching and titanium accumulation in the liver, heart and brain of *Danio rerio* embryos [182]. Chronic and sub-chronic exposure to anatase TiO_2 also led to reproductive toxicity in *zebrafish*, including a 29.5% reduction in egg production [183].

Table 3. Recent studies on TiO_2 NP toxicity across various fish species.

Nanoparticles	Size (nm)	Model	Stage	Duration	Effect	Ref.
TBPH-TiO_2 NPs (Tetrabromophthalate)	NA	*Denio rerio*	Adult	28 days	Co-exposure increased NP bioaccumulation, leading to enterohepatic toxicity and enhanced LPS circulation along the gut–liver axis.	[184]
TiO_2 NPs	22.2 ± 3	*Onchorhynchus mykiss*	Adult	28 days	Exposure to 210 µg/L NPs altered the gill microbiota composition and modulated the expression of innate immune–related genes in the fish	[185]
TiO_2 NPs	NA	*Ctenopharyngdon idella*	fingerlings	15 days	Exposure to TiO_2 at increasing doses (0.5, 1.5, and 2 mg/kg) caused a range of histopathological alterations, including hyperplasia, necrosis, reduced gill filament length, mild acute filamentation, and hydropic degeneration.	[186]
TiO_2 NPs	NA	*Cyprinus carpio*	Adult	21 days	TiO_2 exposure (between 200 and 300 mg/L) induced potent oxidative stress causes decrease in antioxidant enzyme activity and increase in lipid peroxidation with RBC DNA damage.	[187]
TiO_2 NPs	30	*Danio rerio*	Embryo	6 hpf and 150 days	Parental exposure to TiO_2 (100 µg/L) induced transgenerational thyroid disruption and developmental neurotoxicity.	[188]
TiO_2 NPs citrate coated	25	*Acipenser schrenckii*	Juvenile	14 days	Exposure to TiO_2 NPs disrupted lipid metabolism in liver and altered KEGC pathway of immune response.	[177]
TiO_2 NPs	45	*Sparus aurata*	Adult	90 days	Alteration in hepatic tissue structure, expression of genes related to proteins enrolled in lipid and fatty acid metabolism, transport and homeostasis	[189]
Sn-TiO_2 NPs	40, 28, 21	*Danio rerio*	Adult	21days	Doping of TiO_2 (103.47 mg/L) with Sn enhanced its toxicity causing severe conditions ins several organs	[190]
TiO_2 NPs	< 25	*Oreochromis niloticus*	Adult	7, 14, 21 days	Exposure to TiO_2 NPs caused histological liver and intestinal alteration and erythrocytic DNA damage.	[15]
TiO_2 NPs	25± 5	Hybrid groupers fish	Juveniles	14 days	Alteration in liver and intestinal histology occurred. Antioxidant and apoptosis-related transcriptions were downregulated while inflammatory TNF- α was upregulated in liver.	[191]
TiO_2 NPs	15	*Oncorhynchus mykiss*	Adult	28 days	Exposure to 50.1 mg/L TiO_2 induced neurotoxic effects, including a 24% increase in AChE activity	[178]

TiO_2 NPs (100% anatase)	4.9±1.0, 30± 7, and 5.1 ± 1.2	*Danio rerio*	Embryo	56 h	No harmful impact observed up to 100 mg.	[192]
TiO_2 NPs	21	*Prochilodus lineatus*	juvenile	5 days/ 30 days	Liver pathologically changed and AChE activity in the muscle decreased.	[193]
TiO_2 NPs (100% anatase)	585	*Pimephales promelas*	Adult	28 days	Accumulation of Ti caused pathomorphological changes in kidney and spleen. Immune power decreased.	[180]
TiO_2 NPs (80% anatase and 20% rutile)	30	*Carassius auratus* and *Danio rerio*	Adult	21 days and 48 days	Hepatocytes changed morphologically and levels of malondialdehyde increased.	[194]
TiO_2 NPs	20	*Danio rerio*	Embryo	120 h	Velocity and activity level of fish decreased, with malformed organs formation up to 10 mg/L.	[195]
TiO_2 NPs (100% anatase)	240–360	*Danio rerio*	Embryo	13 weeks	Ti accumulation in ovary decreased the numbers of eggs, IV-stage follicles, altering oxidative stress regulation, metabolism, proteolysis, apoptosis, insulin signaling, and oocyte maturation.	[183]
TiO_2 NPs (75% anatase and 25% rutile)	21	*Oncorhynchus mykiss*	Juvenile	14 days	Gills and enterocytes were damaged due to decline in activity of Na^+-K^+ ATPase. Thiobarbituric acid level increased in intestine, gills and brain.	[196]

Iron based NPs

Iron-containing NPs are broadly utilized in medical field like drug delivery, magnetic detection, hyperthermia treatment, and magnetic resonance imaging (MRI), owing to their unique magnetic and physicochemical properties [107]. Several iron-based oxides are commonly encountered, such as magnetite (Fe_3O_4), hematite (Fe_2O_3), wüstite (FeO), and iron oxyhydroxides like goethite (FeOOH). Although their biomedical advantages are well documented, the potential toxicological impacts of these materials on aquatic organisms should not be overlooked. Several studies reporting toxicity of above-mentioned respective NPs to aquatic ecosystem are listed in Table 4.

In adult *Oncorhynchus mykiss*, exposure to iron oxide NPs reduced swimming speed and decreased the activities of key antioxidant enzymes, including superoxide dismutase, glutathione, and malondialdehyde [197]. Similarly, *Gonoproktopterus kolus* fingerlings exposed to ferric chloride NPs for 96 h exhibited remarkable reductions in tissue protein, glycogen, and lipid content [198]. In *Labeo rohita*, exposure to Fe_3O_4 NPs at concentrations of 100, 1500, and 3000 ppm affected behavioral parameters such as bottom resting, respiration, and jerk movements, with mortality rates increasing significantly at 3000 ppm [199]. A 25-day exposure in adult *Labeo rohita* further resulted in elevated levels of hemoglobin (Hb), hematocrit, erythrocytes, mean corpuscular volume (MCV), and mean corpuscular hemoglobin concentration (MCHC), accompanied by a decrease in white blood cell (WBC) counts [200]. Iron oxide NPs have also been reported to bioaccumulate in fish tissues [201]. In *Danio rerio*, exposure to iron-containing NPs was reported to cause delayed hatching, increased mortality, and organ deformities [202,203].

Table 4. Recent studies on Fe-containing NPs and their toxicity across various fish species.

Nanoparticles	Size (nm)	Model	Stage	Duration	Effect	Ref.
Iron oxide NPs	15	*Cardina fossarum*	Adult	14 days	Remarkable variations in oxidative biomarkers, metabolic profile and biochemical parameters	[204]
Iron oxide NPs	30	*Not specific fish*	Adult	Variable duration	Exposure to Fe NPs caused increase in bioaccumulation, DNA damage, genotoxicity and oxidation stress.	[205]
Iron oxide NPs	20	*Cyprinus carpio*	Juveniles	7 days	Bioaccumulation of NPs via erythrocytes of vessel lumen led to increase in liver pathology and structural damage.	[206]
Iron oxide NPs	8–9	*Oreochromis niloticus*	Adult	24 or 96 h	FeO NPs accumulated in liver, pancreases, gills, kidney and muscles, leading to their malfunctioning.	[207]
Iron oxide NPs	30	Not specific fish	Adult	120 h	10 mg/L iron oxide solution was toxic to embryonic development, resulting in hatching delays, increased mortality, and various malformations	[202]
Iron oxide NPs	30	*Oncorhynchus mykiss*	Adult	24 h	Spermatozoa velocity was reduced, and the activities of catalase and superoxide dismutase decreased. Total glutathione and malondialdehyde levels were markedly elevated following exposure to 50, 100, 200, 400, and 800 mg/L	[197]
Fe_3O_4 NPs	<50	*Labeo rohita*	Adult	7 days	Exposure to 100, 1500, and 3000 ppm iron oxide reduced bottom-resting behavior, surface respiration, and jerk movements. Mortality increased markedly at concentrations above 1500 ppm	[199]
Iron oxide NPs	100	*Labeo rohita*	Adult	25 days	Erythrocytes, hemoglobin, hematocrit, MCHC and MCV increased, and WBC levels decreased on exposure to 500 mg/L iron oxide NPs in water	[200]
Fe^{2+} and Fe^{3+}	NA	*Heteropneustes fossilis*	Adult	24, 48, and 72 h	Iron accumulated in fish tissues following exposure to 109, 68, and 45 mg/L iron oxide solutions	[201]
Iron oxide NPs	22.37	*Capoeta fusca*	Adult	96h	Iron aggregation was observed in the gill tissues of fish exposed to 105, 111, 117, 123, 129, and 135 mg/L iron oxide solutions	[208]
Iron oxide NPs	29–40	*Oreochromis mossambicus*	Adult	96 h	Exposure to 0.5, 5, and 50 mg/L iron oxide caused significant alterations in RBC, WBC, HCT, and Ht levels, along with changes in SGPT and SGOT activities	[209]

Al-Containing NPs

Aluminum NPs and aluminum oxide (Al_2O_3) NPs are widely used in optoelectronics, electronics, medical applications, and drug-delivery systems. Despite their technological advantages, numerous studies have reported their toxicological impacts on aquatic organisms (See Table 5). For example, Al NPs were shown to promote lipogenesis and induce steatohepatitis shortly after the larval-to-juvenile transition in *Danio rerio* [210]. In adult *Oreochromis niloticus*, exposure to Al NPs significantly impacted liver function by altering antioxidant enzyme activity [211]. Long-term exposure in *Oreochromis mossambicus* resulted irreversible damage in the liver, gills, and brain [212]. Similarly, in *Carassius auratus*, Al NPs induced gill hyperplasia and liver degeneration, accompanied by increased activities of glutathione S-transferase, catalase, and superoxide dismutase [213].

Table 5. Recent studies on Al-containing NPs and their toxicity across various fish species.

Type of NPs	Size (nm)	Model	Stage	Duration	Effect	Ref.
Aluminium nitrate non-hydrate Al $(NO_3)_3 9H_2O$	90	*Oreochromis niloticus*	Adult	28 days	Al NPs (2.15 mg/kg) induced hepatocellular vacuolization, dilation of central veins and hepatic sinusoids, vascular congestion, and gill hyperplasia.	[214]
Al NPs	55–80	*Danio rerio*	Larvae	14 dpf	Dose-dependent mortality was observed at 40, 80, and 160 mg/L, with corresponding mortality rates of 10%, 50%, and 75%. In addition, morphological and gastrointestinal alterations were recorded.	[215]
Aluminum NPs	13 and 50	*Danio rerio*	Larvae	14 dpf	Exposure to 160 mg/L Al NPs resulted in external morphological abnormalities, Neurodevelopmental dysfunction and gastrointestinal malformations during the larval–juvenile transition	[216]
γAl_2O_3 and αAl_2O_3 NPs	5–400 and 50–3500	*Gammarus pulex*	Adult	96 h	Both NPs increased oxidative stress and antioxidant parameters alteration.	[217]
Aluminum NPs	40–76	*Danio rerio*	Larvae	1st to 14th dpf (days post fertilization)	Al NPs (160 mg/L) induced lipogenesis and steatohepatitis during transition from larval to juvenile phase.	[210]
Al_2O_3	55–80	*Oreochromis niloticus*	Adult	96 h	Al NP exposure altered the levels of GABA, serum amino-acid neurotransmitters, and monoamines in brain tissue	[218]
Al_2O_3	~18	*Cyprinus carpio*	Adult	5 to 96 h	Significant damage to the gills and liver was observed in fish exposed to 50 µg/L Al NPs. The bioconcentration factor was higher in muscle and gill tissues compared with the brain and liver.	[219]
Al_2O_3	37.4	*Oreochromis niloticus*	Adult	96 h	Exposure to 52.4 ppm Al NPs caused decrease in antioxidant enzymes activity like superoxide dismutase, catalase and glutathione peroxidase, while increase in glutathione-S-transferase enzyme activity, which overall severely damaged liver tissues.	[211]
Al_2O_3	15	*Oreochromis niloticus*	Adult	20 days	Al_2O_3 NPs (1 mg/L) accumulated extensively in gill tissues under both acute and chronic exposure conditions in the presence of salt. A marked decrease in ATPase activity was observed.	[220]
Al_2O_3 NPs	10	*Oreochromis niloticus*	Adult	7 days	Less accumulation in fish gills, liver and muscle tissues and also influenced less Liver SOD, CAT and GPx enzyme activities at the 10 mg/L concentration.	[221]
Al_2O_3	31.4 ± 4.8	*Oreochromis niloticus*	Adult	7 days	Dose-dependent increase in superoxide dismutase, catalase activity, and thiobarbituric acid reactive substance levels.	[222]
Al_2O_3	40	*Oreochromis niloticus*	Adult	28 days	Oxidative stress was increased due accumulation of Al NPs in liver of fish when exposed to 0, 1, 5, 25 mg/L Al NPs.	[223]
Al_2O_3	<50	*Oreochromis niloticus*	Adult	7 days	Gills and liver tissues showed elevation in thiobarbituric acid reactive substances, catalase, glutathione peroxidase and superoxide dismutase	[224]
Al_2O_3	<50	*Danio rerio*	larvae	69 h	Dose-dependent increase in genotoxicity of larval cells were observed.	[225]
Al_2O_3	16.7	*Oreochromis mossambicus*	Adult	96 h and 60 days	Chronic exposure to 4 mg/L NPs induced irreversible alterations in the gill, liver, and brain tissues of fish.	[212]
Al_2O_3	27.65 ± 10.32	*Clarias gariepinus*	Embryo	48 h	Studied dose dependent effect on bioaccumulation, survival, hatchability, morphological abnormalities, swimming speed, enzymatic antioxidants at the concentration of 0, 0.5, 1, 5, and 10 mg/L.	226
Al_2O_3	40	*Oreochromis mossambicus*	Adult	96 h	Acute exposure to NPs severely disrupted the histoarchitecture of multiple fish tissues,	[227]

					including brain, kidney, gills, muscles, and intestine	
Al_2O_3	16.7	*Oreochromis mossambicus*	Adult	15, 30, 60 days	Long-term exposure caused alternation in antioxidant defense system of fish, leading to persistent toxic effect in liver, brain and gills	[212]
Al_2O_3	16.7	*Oreochromis mossambicus*	Adult	96 h	Median lethal dose observed was 40 mg/L of Al_2O_3.	[228]
Al_2O_3	30	*Carassius auratus*	Adult	7, 14, 21 days	Gill hyperplasia and liver degeneration occurred alongside amplified activities of glutathione S-transferase, catalase, and superoxide dismutase.	[213]
Al NPs	51	*Danio rerio*	Adult	48 h	Al NPs exhibited minimal acute toxicity but caused a reduction in Na^+-K^+ ATPase activity.	[229]

Platinum NPs

Platinum NPs, which are widely used in automobile catalytic converters, can enter aquatic ecosystems through runoff from rainwater. In a study on adult *Cirrhinus mrigala*, exposure to Pt NPs resulted in behavioral abnormalities such as loss of balance, erratic swimming, and restlessness [230]. Cellular studies have also demonstrated platinum uptake, disaggregation, and stability in fish cell lines, including Epithelioma, Papulosum, Cyprini (EPC) and Bluegill Fry (BF) cells [231].

In *Danio rerio* (*zebrafish*) embryos, Pt NP exposure was observed to lead to a dose-dependent reduction in heart rate, axial curvature deformities, and delayed hatching. Interestingly, Pt NP accumulation in embryos was lower compared to silver and gold NPs, most likely due to their smaller size, which limited retention within embryonic tissues [232]. Like above there are several reports regarding Pt NPs toxicity to aquatic organism which are listed as below in Table 6.

Table 6. Recent studies on Pt NPs and their toxicity across various fish species.

Nanoparticles	Size (nm)	Model	Stage	Duration	Effect	Ref.
Pt NPs	4–9	Fish	Adult	48 h	Pt NP uptake, disaggregation, and stability were evaluated in Epithelioma Papulosum Cyprini(EPC), and Bluegill fry cell lines.	[231]
Pt NPs	NA	*Cirrhinus mrigala*	Adult	96 h	Marked behavioral alterations following acute NP exposure, including loss of balance, abnormal swimming patterns, and restlessness.	[230]
Pt NPs	3–10	*Danio rerio*	Embryo	72 hpf (hours post fertilization)	Pt NPs caused minimal embryotoxicity, likely due to their smaller particle size compared with other metal NPs.	[232]

Nickel-Containing NPs

The widespread extraction and industrial use of nickel and its alloys, found in jewelry, medical implants, cadmium batteries, stainless steel production, and nickel plating, have increased the risk of human exposure through environmental contamination and occupational contact. Nickel oxide (NiO) NPs accumulate in fish tissues and induce marked pathological genotoxicol response [233]. In *Heteropneustes fossilis*, exposure to NiO NPs significantly altered hematological and biochemical parameters, as well as key enzyme activities [234]. Similarly, in *Labeo rohita*, depletion of

antioxidant enzyme activity following exposure resulted in pronounced pathological lesions in the kidney and liver [235].

In *zebrafish* embryos, exposure to nickel NPs of varying sizes (30, 60, and 100 nm) caused mortality and developmental abnormalities, including reduced intestinal thickness and skeletal muscle damage. Notably, the toxic effects of these NPs were comparable to those of soluble nickel, indicating that NP form does not substantially reduce toxicity [236]. For additional details, see Table 7.

Table 7. Recent studies on Ni-containing NPs and their toxicity across various fish species.

Name of NPs	Size (nm)	Model	Stage	Duration	Effect	Ref.
Ni NPs	≤ 100	*Sparus aurata*	Adult	28 days	Lipid peroxidation levels in the gills increased significantly, accompanied by a marked elevation in glutathione S-transferases activity.	[237]
Nickel Oxide NPs	NA	*Cirrhinus mrigala*	adult	60 days	Adversely affected hematological parameters including WBCs, RBCs, hemoglobin, MCHC, hematocrit, platelets and increased % of tail DNA.	[238]
Nickel oxide NPs	52.41	*Nile Tilapia*	Finger lings	14 days	Superoxide dismutase and CAT activity increased in gills, kidneys, liver and muscles.	[239]
Ni NPs	NA	*Carassius auratus*	Adult	7 days	Antioxidant level decreased while level of MDA increased.	[240]
Nickel oxide NPs	<50	*Heteropneustis fossilis*	Adult	14 days	Significant fluctuations noticed in hematological parameters (erythrocyte, leucocyte count, Hb content, Ht. %), enzymatic activities (AST, ALP, ALT, and LDH) and biochemical parameters on exposure to 48 g/L of NPs.	[234]
NiO NPs	<50	*Heteropneustes fossilis*	Adult	14 days	Hematological and biochemical parameters were highly influenced by exposure to Ni NPs	[241]
NiO NPs	53.4	*Labeo rohita*	Adult	90 days	Time-dependent variations in catalase and superoxide dismutase activities were observed, accompanied by cellular toxicity associated with elevated oxidative stress	[242]
Ni nanowire	35	*Danio rerio*	Adult	24 h	Acute toxicity was found below threshold level.	[243]
Ni NPs	43 ± 6	*Labeo rohita*	Adult	21 days	Depletion of antioxidant enzyme activities was associated with an increased incidence of pathological lesions in the kidney and liver	[235]
Nickel nanocomposite	17	*Danio rerio*	Embryos	7 days	Exposure to nanocomposite only induced behavioral changes and decreased swimming distance	[244]
Ni NPs	40	*Danio rerio*	Adult	72 h/7 days	Ni NPs with high adsorptive capacity caused severe damage to gills, liver, and digestive tract, including epithelial degeneration and necrosis.	[245]
Ni NPs	45	Bluegill Sunfish Cells	Cells	24 h	Dose-dependent cytotoxic response was observed after 24 h, reflected by alterations in lysosomal and mitochondrial activities, and in increased lactate dehydrogenase release.	[246]
Ni NPs	<100	*Danio rerio*	Larvae	96 h	Ni NPs caused acute toxicity in larvae by releasing Ni ion	[247]
NiO NPs	10–20	*Onchorhynchus mykiss*	Adult	30, 60 days	Dose-dependent increase in pancreatic lesions was observed, including necrosis of acinar cells, edema in the connective tissue, and pronounced cell shrinkage	[248]
Ni NPs	5	*Danio rerio*	Adult	48 h	While acute exposure produced measurable effects in fish, higher concentrations posed a greater risk and were associated with more severe adverse impacts, including increased mortality.	[249]
Ni NPs	56	*Oreochromis mossambicus*	Adult	14 days	Antioxidant enzymes were depleted in gills and liver while significant accumulation of NPs was found in liver, gills and skin	[250]
NiO NPs	<50	*Danio rerio*	Adult	30 days	Chronic exposure resulted in increased toxicity, with an LC_{100} value of 100 mg/L.	[251]
NiO NPs	30, 60, 100 and 60 clusters	*Ctenopharyngodon idella*	Fingerlings	7, 14, 21 days	Dose dependent DNA deterioration and RBC damage were observed the concentration 6.75 mg/L, 4.50 mg/L, 2.25 mg/L,	[252]

Copper and Copper Oxide NPs

Copper oxide (CuO) NPs possess unique physicochemical properties, including small size, high surface area, and pronounced reactivity, that support their widespread use in biomedical, industrial, electrical, and environmental applications. These same attributes, however, increase the likelihood of human and ecological exposure. In aquatic organisms, chronic CuO NP exposure has been linked to cytotoxicity, oxidative stress, genotoxicity, neurotoxicity, immunotoxicity, and inflammation [253]. In *Danio rerio* (*zebrafish*), engineered CuO NPs delayed embryo hatching by coating the chorion and inducing foaming within the perivitelline fluid [254]. Sub-lethal, long-term exposure to rod-shaped CuO NPs (32.84 nm) for 45 days in *Labeo rohita* (*rohu*) triggered oxidative stress, metal accumulation, and genotoxic effects [96]. Comparative studies further indicate that CuO and ZnO NPs exert greater toxicity in embryos and juvenile fish than their corresponding metal salts, with concentration-dependent reactive oxygen species (ROS) generation emerging as a key mechanism underlying CuO NP-induced toxicity [255].

Exposure to CuO NPs has also produced abnormal phenotypes in *zebrafish* embryos, including delayed epiboly and reduced eye and head size [256]. Acute exposure to high concentrations caused hepatotoxicity and neurotoxicity in zebrafish larvae and embryos. A comparative toxicity assessment across rainbow trout, fathead minnow, and *zebrafish* reported lowest-observed-effect concentrations (LOECs) of 0.17, 0.023, and <0.023 mg/L, respectively, values below the predicted environmental concentrations of copper NPs [257]. In human cell studies, Karlsson et al. demonstrated that CuO NPs caused substantial damage to human lung epithelial cell lines, whereas iron oxides (Fe_2O_3 and Fe_3O_4) exhibited comparatively lower toxicity [258].

Zinc Oxide NPs

Zinc oxide NPs, which are widely used across industrial and biomedical sectors, pose significant risks to aquatic organisms under chronic exposure. In C*larias gariepinus*, prolonged exposure to ZnO NPs inhibited growth, increased oxidative stress, and induced hematotoxicity through disruption of gene expression [259]. In *Takifugu obscurus*, exposure to 50-nm ZnO NPs across multiple developmental stages, including fertilized eggs, hatched embryos, and two-month-old juveniles, resulted in reduced hatching rates, organ malformations, and decreased survival [260].

Zn^{2+} ions released from ZnO NPs have been associated with genotoxic effects and impaired locomotor activity [261]. In *Danio rerio* embryos, exposure to 100-nm ZnO NPs increased oxidative stress, reduced antioxidant levels, and inhibited key enzymes such as Na^+/K^+-ATPase and acetylcholinesterase (AChE) [262]. Sub-lethal exposure in *Scarus coeruleus* (Blue Parrotfish) over 15 days triggered oxidative stress, antioxidant

responses, decrease hepatoenzymes activity, disrupt tissue level of cations (Na^+,K^+, Ca^+)and histological alterations [263].

In rare minnows, a 60-day exposure to ZnO NPs (30 ± 10 nm) induced hepatotoxicity, characterized by vacuolization, irregular or absent nuclei, reduced body weight, and a decreased hepato-somatic index (HSI) [264]. In *Danio rerio*, ZnO NPs were shown to interact with *zebrafish* hatching enzymes (ZHE1) and superoxide dismutase (SOD1), further implicating their involvement in developmental toxicity [265]. In *goldfish*, Zn^{2+} ions accumulated in the liver, gills, and kidneys after 14 days of exposure, leading to significant alterations in serum biochemical markers, hepatic enzyme activity, immune responses, and antioxidant levels [266].

ZnO NPs are widely used in cosmetics, sunscreens, photonics, electrical appliances, and ceramics due to their antimicrobial properties, strong photocatalytic activity, transparency, and biocompatibility associated with their high isoelectric point [267]. Although Zn^{2+} ions released from ZnO NPs have been implicated in toxicity, some researchers argue that the nanoparticles themselves are primarily responsible for the observed adverse effects [268].

Surface modifications of ZnO NPs can significantly influence their biological interactions. For example, chitosan-coated (ZnO-CTS) and polyethylene glycol-coated (ZnO-PEG) ZnO NPs exhibit reduced deposition on *zebrafish* embryo surfaces, with ZnO-CTS in particular improving embryo survival rates [269]. Nevertheless, exposure to ZnO NPs has been shown to exert toxic effects on fish embryos and larvae, including delayed hatching, tail deformities, tissue damage, reduced larval body size at lower concentrations, and increased embryo mortality at higher doses [269].

Additionally, Zn^{2+} ions may contribute to overall toxicity, as evidenced by delayed hatching, skin ulceration, and elevated mortality in *zebrafish* exposed to ZnO NPs [270]. These findings underscore the complex interplay between NP composition, surface chemistry, and biological impact.

NANOMATERIAL RISKS, ENVIRONMENTAL POLICIES, AND FUTURE RECOMMENDATIONS

Nanomaterials have contributed significantly to advancements across multiple sectors worldwide because of their unique properties described above. However, our findings indicate that once released into aquatic environments, they can pose substantial ecological and health risks. These risks depend largely on nanomaterial characteristics, such as shape, size, surface chemistry, and concentration, as well as environmental factors including pH, organic matter content, and salinity. Major categories of risk in aquatic systems include bioaccumulation, toxicity to aquatic organisms, sediment contamination, alterations in water chemistry, synergistic interactions with other pollutants, trophic transfer, and genetic or molecular effects [271]. Nevertheless, the adverse impacts of metal and metal oxide NPs can be mitigated through polymer-based delivery systems,

metal–organic frameworks, responsive surface modifications, and artificial-intelligence-based computational approaches [272].

Here we critically evaluate existing regulatory frameworks governing NP risks in aquatic environments, identifying region-specific regulatory gaps, particularly in developing regions such as South Asia and Sub-Saharan Africa, and propose policy measures to strengthen the protection of freshwater, estuarine, and marine ecosystems.

Due to the relatively new and rapidly evolving nature of NPs, global regulatory standards remain fragmented, although most developed countries incorporate NP-related disposal requirements into existing hazardous waste, chemical, and environmental regulations. In the United States, for example, two key regulatory bodies, the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), oversee NP management through frameworks such as the Toxic Substances Control Act (TSCA) and the Resource Conservation and Recovery Act (RCRA). Under these regulations, manufactured NPs are evaluated for their environmental fate and toxicity before disposal guidelines are approved [273,274].

The European Union currently implements some of the most advanced and specific regulations for nanomaterial waste management, primarily through the REACH Regulation (Registration, Evaluation, Authorization and Restriction of Chemicals) and the Waste Framework Directive (2008/98/EC). Manufacturers are required to register nanomaterials and provide comprehensive safety data, including information on disposal pathways. In industrial sectors, the labeling and tracking of nanomaterials within waste streams are mandatory. The European Chemicals Agency (ECHA) has also issued detailed technical guidance for the treatment and management of nano-waste [275].

The United Kingdom, which has adopted many REACH-aligned restrictions, regulates ecotoxic nanomaterial waste as hazardous substances. Its regulatory framework emphasizes risk-based disposal, controlled-condition incineration, and preventing NP entry into landfills and sewage systems [276]. Several other countries have also established regulatory mechanisms to ensure proper nano-waste management and environmental protection. These include China's Measures on the Environmental Management of New Chemical Substances, Japan's Chemical Substances Control Law (CSCL) and Industrial Safety and Health Law, Australia's National Industrial Chemicals Notification and Assessment Scheme (NICNAS), and Canada's Canadian Environmental Protection Act (CEPA) [271].

However, despite the measures implemented by many developed countries, several challenges persist. These include limited data on long-term toxicity, difficulties in detecting NPs within waste streams, the absence of standardized nano-specific testing methods and globally harmonized disposal protocols, insufficient environmental monitoring

systems, and the rapid pace of technological innovation that continues to outpace policy development [277].

In this chapter, we highlight the regulatory gaps in developing regions such as South Asia and Sub-Saharan Africa, where formal risk-assessment protocols for nanomaterials are largely absent. Accordingly, we recommend that both developed and developing countries adopt the following actions [278]:

(i) develop harmonized, nano-specific aquatic toxicity testing protocols, including mandated OECD nano-specific guidelines and standardized characterization of MNPs;
(ii) develop and implement advanced, effective, and environmentally friendly water-treatment technologies such as adsorption, membrane filtration, and photocatalytic degradation;
(iii) ensure full registration of all industrial-scale NP manufacturers;
(iv) achieve ≥ 70% engineered NP removal efficiency in municipal wastewater treatment systems;
(v) conduct annual biomonitoring using sentinel fish species;
(vi) establish strict legal regulations governing the discharge, transport, and accumulation of nanoparticles in aquatic environments;
(vii) promote research on the ecotoxicity of NPs to strengthen risk-assessment frameworks and improve understanding of their impacts on aquatic ecosystems and human health;
(viii) expand public education and awareness initiatives targeting communities, stakeholders, and policymakers regarding the potential risks associated with nanoparticles; and
(ix) adopt a comprehensive, multidisciplinary approach, integrating engineering, toxicology, environmental science, and policy development, to guide future nanotechnology research. International cooperation remains essential for achieving sustainable nanomaterial management.

Several studies have developed various matrices, such as functionalized polymeric materials, activated carbon, graphene oxide, chitosan composites, and biochar, for the immobilization of hazardous nanomaterials [277,278]. Therefore, it is recommended to design advanced, compact, and functionalized matrices optimized for key conditions such as pH and temperature to effectively immobilize nanomaterials released from industrial effluents and laboratory discharges.

Future research aimed at supporting clean and sustainable aquatic environments should focus on developing advanced matrices, including AI- and ML-based models, capable of effectively immobilizing nanomaterial-based pollutants. Such innovations are essential for achieving zero discharge of NPs into aquatic ecosystems.

CONCLUSIONS

The rapid advancement of global technology has driven increasing demand for nanoparticle-based materials across a wide range of sectors, including electronics and optoelectronics, sunscreens, paints, cosmetics, textiles, and medicine. However, the extensive use of nanomaterials also introduces ecological risks, particularly when they enter aquatic environments, where they can disrupt food webs and destabilize ecosystem functioning. The toxicity of metal ion–containing nanoparticles (NPs) largely stems from their unique physicochemical properties, including their chemical composition, small size, large surface area, high mobility, and surface modifications. This review highlights that common industrial nanomaterials, including NPs of TiO_2, CuO, ZnO, Al_2O_3, FeO, Pt, SiO_2, CeO_2, gold, and silver, adversely affect tissue development, sperm function, embryonic growth, hematological parameters, physiological processes, and metabolic functions in various fish species. Notably, these nanomaterials often exhibit greater toxicity than their soluble ionic forms, with titanium dioxide and silver NPs showing particularly pronounced effects in aquatic organisms. Given these findings, there is an urgent need to develop technologies capable of trapping or adsorbing engineered and hazardous nanomaterials within polymeric matrices before effluents are discharged into aquatic ecosystems. There is also a pressing need for globally standardized protocols for the safe handling and disposal of nanomaterials, regardless of a country's economic status. Implementing scientifically updated standard operating procedures, along with the responsible and ethical use of nanomaterials, is essential for mitigating environmental risks and protecting aquatic ecosystems.

DATA AVAILABILITY

No data were generated from the study.

CONFLICTS OF INTEREST

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