Scientific Papers. Series D. Animal Science. Vol. LXIX, No. 1, 2026
ISSN 2285-5750; ISSN CD-ROM 2285-5769; ISSN Online 2393-2260; ISSN-L 2285-5750

FISH WELFARE RESPONSES TO HEAVY METAL EXPOSURE: A REVIEW

Andrei ARHIP, George-Cătălin MUNTEAN, Tudor PĂPUC, Daniel COCAN, Radu CONSTANTINESCU, Paul UIUIU, Raul SAVIN, Călin LAȚIU, Aurelia COROIAN

University of Agricultural Sciences and Veterinary Medicine of Cluj-Napoca,
3-5 Calea Mănăștur Street, 400372, Cluj-Napoca, Romania

Corresponding author email: calin.latiu@usamvcluj.ro

Abstract

Heavy metals are persistent contaminants with a high potential for bioaccumulation and biomagnification in aquatic ecosystems, exerting toxic effects that impair physiological functions and compromise fish welfare. Although numerous studies have investigated oxidative stress, endocrine disruption, histopathological alterations, and sublethal effects, the integration of these processes within a comprehensive fish welfare framework remains limited.This review synthesizes current knowledge on the toxic effects of major heavy metals (Cd, Pb, Hg, As), focusing on their bioaccumulation patterns, tissue distribution, physiological and behavioral alterations, and their implications for fish welfare. By integrating ecotoxicological evidence with contemporary welfare concepts, this study highlights key mechanisms underlying welfare impairment and identifies major gaps in the current literature. The findings emphasize that sublethal and chronic exposure to heavy metals can compromise fish welfare even in the absence of immediate mortality. Overall, this review provides a functional perspective on heavy metal toxicity and outlines priority directions for future research and the development of monitoring and management strategies aimed at mitigating the impact of heavy metal contamination in aquatic ecosystems.

Key words*: aquatic ecotoxicology, bioaccumulation, biomagnification, oxidative stress, physiological responses.*

INTRODUCTION

Globally, the pollution of aquatic ecosystems with heavy metals represents a major concern due to the toxicity of these elements and their ability to accumulate within aquatic ecosystems (Ahmad et al., 2010; Aski et al., 2023). Heavy metals enter aquatic ecosystems through natural sources and, in particular, through anthropogenic activities, with anthropogenic inputs often exceeding natural fluxes and thereby increasing the risk of contamination in water bodies and aquatic organisms (Masindi & Muedi, 2018).

Unlike other pollutants, heavy metals are non-biodegradable and can undergo bioaccumulation and biomagnification throughout the food web, generating toxic effects even at locations distant from the original pollution source (Ahmad et al., 2010).

Sediment serves as a reservoir for heavy metals in aquatic ecosystems, gradually releasing these contaminants into the water and contributing to the chronic exposure of fish (Pelić et al., 2024). Fish can absorb heavy metals through multiple mechanisms, including ingestion of suspended particles, consumption of contaminated food, ionic exchange at the gill surface, and direct adsorption onto tissues (Islam et al., 2015). Fish may accumulate substantial quantities of heavy metals in their tissues (Łuczyńska et al., 2022). However, muscle tissue generally tends to accumulate the lowest concentrations of metals (Zeitoun & Mehana, 2014).

In contrast, accumulation occurs more frequently in organs such as the liver, kidneys, gills, and reproductive tissues, where metals can induce cellular alterations such as vacuolization, necrosis, or endocrine disruption, thereby affecting health and physiological function (Sharma et al., 2024).

Furthermore, heavy metals can directly impair fish welfare through complex toxicological mechanisms. Exposure to elevated concentrations can induce oxidative stress, genotoxicity, and endocrine disturbances, altering essential physiological functions, growth, and reproduction. At the morphological

and functional level, heavy metals are associated with vertebral deformities, fin abnormalities, and developmental impairments during embryonic and larval stages, which reduce swimming performance, feeding capacity, and survival. Fish exhibit heightened sensitivity to these effects, especially during early life stages, which represent critical periods for normal development and the maintaining of adequate welfare (Sfakianakis et al., 2015).

Exposure to heavy metals affects not only tissue structures but also behavioral patterns essential for survival and welfare. Several studies have reported alterations in vital functions such as swimming performance, avoidance responses, feeding behavior, and cognitive processes, even at sublethal metal concentrations. Such behavioral modifications represent sensitive indicators of toxic stress and can serve as early warning signs of declining welfare in aquatic organisms (Morales, 2022).

Additionally, fish are considered sensitive bioindicators of heavy metal pollution because they respond rapidly to changes in the aquatic environment and reflect the level of ecosystem contamination (Habib et al., 2023). In the European Union, Council Directive 98/58/EC establishes general welfare standards for animals kept for production purposes, explicitly including fish, which must be protected from suffering and inadequate environmental conditions (European Council, 1998).

However, the scientific literature still lacks an integrated synthesis connecting the toxicological mechanisms of heavy metals with their physiological, behavioral, and welfare-related impacts on fish. Most studies address these aspects in a fragmented manner, without integrating their consequences in accordance with modern welfare evaluation criteria.

In this context, the present review aims to provide a critical and comprehensive analysis of the impact of heavy metals on fish, bringing together evidence on bioaccumulation, tissue-level effects, physiological responses, behavioral alterations, and welfare implications. Such an integrative approach is essential for understanding the real risks at both individual and population levels and for informing future directions in monitoring and protecting fish within aquatic ecosystems.

MATERIALS AND METHODS

A comprehensive literature search was conducted to identify studies on heavy metal exposure and its implications for fish welfare, using Web of Science, Google Scholar, ResearchGate, and ScienceDirect.

The search strategy employed combinations of keywords such as “heavy metals”, “fish”, “fish welfare”, “bioaccumulation”, “oxidative stress”, “toxicity”, and “behavioral alterations”, applied individually and in combination to ensure broad coverage.

Studies published between 2015 and 2026 were primarily considered to reflect current knowledge, while earlier highly cited or relevant studies were also included.

Selection criteria focused on studies addressing aquatic organisms, particularly fish, with emphasis on peer-reviewed articles investigating physiological, biochemical, behavioral, or toxicological responses to heavy metal exposure. Studies lacking direct relevance or scientific robustness were excluded as far as possible.

The selected literature was analyzed and organized into key thematic areas, including toxicity mechanisms, bioaccumulation and tissue distribution, environmental behavior of heavy metals, and their implications for fish welfare, enabling an integrated synthesis of the available evidence.

RESULTS AND DISCUSSIONS

Linking Heavy Metal Toxicity to Fish Welfare: A Functional Perspective

Fish welfare is recognised as an integral component of aquatic animal health and is closely linked to the maintenance of normal biological functioning and adaptive capacity, with chemical agents acknowledged as potential hazards capable of adversely affecting aquatic animal health (OIE, 2019).

Physiological stress responses and associated biomarkers are widely used to assess fish welfare (EFSA, 2009). Prolonged or chronic stress has been shown to impair immune function, growth and reproduction in fish, providing strong indications of compromised welfare, reflecting disruptions of physiological homeostasis and tertiary stress responses

(Huntingford et al., 2006; Jerez-Cepa & Ruiz-Jarabo, 2021).

Fish welfare is increasingly recognised as an attribute of the individual, with individuals showing consistent behavioural and physiological differences in their responses to the same environmental stimulus (Torgerson-White & Sanchez-Suarez, 2022), and can therefore be functionally defined as the capacity of an individual to maintain health and appropriate biological functioning when exposed to environmental challenges (Huntingford et al., 2006).

Given their intimate contact with the aquatic environment through the gills, fish are particularly vulnerable to water-borne pollutants, including heavy metals, which may compromise welfare through chronic physiological stress and tissue damage (Huntingford et al., 2006).

Traditional fish welfare assessments have primarily focused on mitigating negative states such as chronic stress and pain; however, increasing evidence suggests that the absence of suffering does not equate to good welfare, highlighting the importance of evaluating both physiological and behavioural indicators within a broader welfare framework (Fife-Cook & Franks, 2019).

Within this context, exposure to both sublethal and lethal concentrations of heavy metals has been associated with the induction of physiological stress responses in fish, following accumulation in various tissues. Several metals promote redox reactions that contribute to the formation of reactive oxygen species (ROS), which play essential roles under controlled conditions. However, excessive ROS production may disrupt redox homeostasis and induce oxidative stress and subsequent impairment of cellular components such as lipids, proteins, and DNA (Shahjahan et al., 2022). Such cellular damage is particularly relevant for organs directly involved in essential physiological functions, as it can impair processes such as gas exchange, ion regulation, and energy metabolism.

To counteract these effects, oxidative balance in fish is maintained through antioxidant defence systems that include enzymatic antioxidants such as superoxide dismutase, catalase and glutathione peroxidase, together with non-enzymatic antioxidants like glutathione. In addition, metal-binding proteins such as metallothioneins play an important role in the detoxification of toxic metals and the regulation of essential metal homeostasis (Sevcikova et al., 2011).

Accordingly, physiological biomarkers are widely employed in fish welfare assessment. They provide measurable indicators of endocrine, oxidative and immune alterations associated with environmental stress. Cortisol is commonly used to evaluate stress responses and associated shifts in energy allocation. Likewise, changes in antioxidant defences and immune parameters reflect disturbances in homeostatic regulation, supporting their relevance as indicators of compromised welfare (Oliveira et al., 2024). Importantly, these biomarker responses reflect underlying physiological adjustments that may reduce the energy available for growth, reproduction, and immune defence.

Beyond oxidative imbalance, heavy metal exposure has been associated with alterations in antioxidant enzyme activities, including catalase, superoxide dismutase and glutathione-dependent systems. Such disturbances may contribute to oxidative damage at the cellular level, including genotoxic effects and histopathological alterations in target organs. Moreover, heavy metal accumulation has been linked to disruptions in reproductive organs, altered hormone production and impaired gamete development (Bhat et al., 2025).

Heavy metal exposure has also been associated with developmental abnormalities and morphological deformities in fish, which may impair survival, growth and overall fitness, thereby representing additional indicators of compromised welfare (Ali et al., 2019). Collectively, these physiological impairments may interfere with normal biological functioning by disrupting key processes such as respiration and osmoregulation (via gill impairment), as well as metabolism and reproduction. These functional alterations reduce the capacity of fish to maintain homeostasis and cope with environmental challenges, ultimately resulting in compromised welfare even in the absence of mortality. Thus, heavy metal exposure should not be interpreted solely as a toxicological stressor, but also as a direct welfare challenge that affects the functional integrity, adaptive capacity, and biological performance of fish.

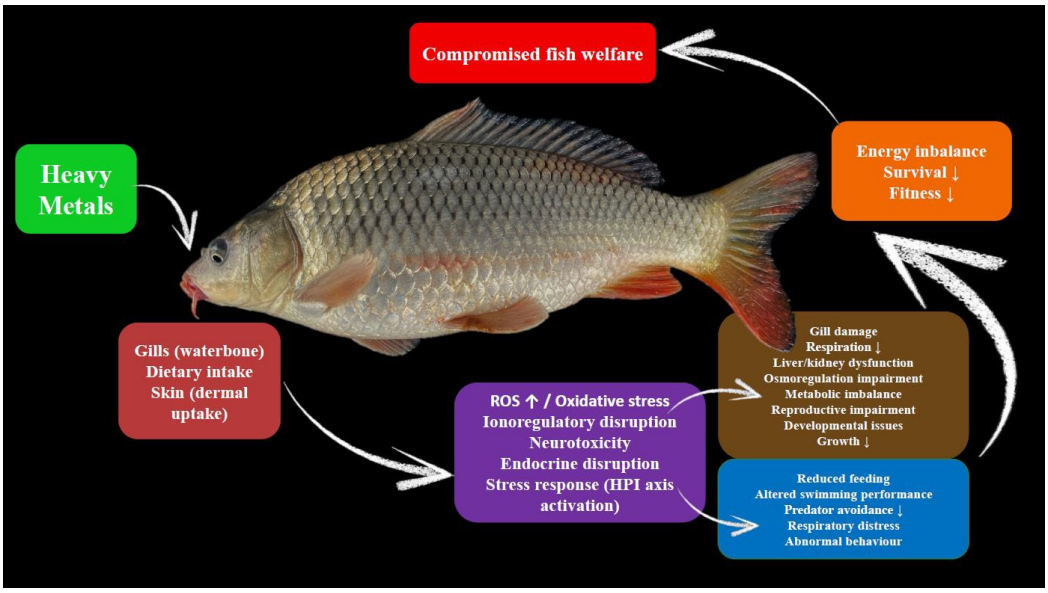

Figure 1. Conceptual framework linking heavy metal exposure pathways, physiological mechanisms and resulting impacts on fish welfare (generated using artificial intelligence tools)

Bioaccumulation and Tissue Distribution of Heavy Metals in Fish

The distribution and behavior of heavy metals in aquatic environments, including speciation, bioaccumulation, and toxicity, represent major concerns (Oziegbe et al., 2025).

Bioaccumulation represents the progressive increase in the concentration of a contaminant within an organism as a result of environmental exposure and trophic intake, whereas biomagnification describes the progressive increase in contaminant concentration across successive trophic levels of the food chain (Ali et al., 2025; Ali & Khan, 2018). Bioaccumulation and biomagnification reflect the limited capacity of organisms to detoxify heavy metals, which contributes to their persistence and toxicity in aquatic systems (Ray & Vashishth, 2024).

Heavy metals enter fish via waterborne and dietary exposure, and their accumulation is influenced by both biological and environmental factors. Age and lifespan represent important determinants of metal accumulation, as metal concentrations generally increase with prolonged exposure of fish to contaminated aquatic environments. Once introduced into aquatic systems, metals are distributed between water, sediments, and food webs, where their accumulation in benthivorous and predatory species largely depends on metal concentrations in water and habitat conditions. Environmental parameters such as water hardness, pH, and organic matter can further influence metal bioaccumulation in fish (Moiseenko & Gashkina, 2020).

Habitat type also plays a significant role in determining metal accumulation patterns. Differences in metal concentrations among fish species may reflect variations in ecological requirements, metabolism, and feeding behaviour. Several studies have reported that metal concentrations in fish largely depend on habitat characteristics. In aquatic systems, sediments are considered a major pathway for metal uptake and play an important role in the accumulation of heavy metals in fish. Fish inhabiting areas close to the sediments and feeding on benthic invertebrates or organic material may accumulate metals transferred from the sediment compartment into aquatic food webs. Consequently, benthic and benthopelagic fish generally exhibit higher metal concentrations than demersal species. Trophic level may further influence the accumulation of heavy metals. Previous studies have reported that piscivorous fish occupying higher trophic levels tend to accumulate greater concentrations of metals compared with omnivorous and herbivorous species, reflecting

trophic transfer processes within aquatic food chains (Hossain et al., 2022). However, this relationship is not always consistent, as some studies have reported higher metal accumulation in omnivorous species compared to carnivorous fish (Ali & Khan, 2019). These inconsistencies suggest that trophic transfer alone cannot fully explain metal accumulation patterns, highlighting the importance of species-specific ecology and exposure pathways.

In addition, uptake pathways may differ depending on metal form, with dissolved fractions being primarily absorbed through the gills, while particulate forms are mainly taken up via the alimentary tract (Ali & Khan, 2019). Contrasting patterns have also been reported. For example, Bawuro et al. (2018) observed higher metal concentrations in pelagic fish (omnivorous or herbivorous) compared with benthic carnivorous species, suggesting that accumulation patterns may vary depending on ecological conditions and species-specific feeding strategies.

Furthermore, ontogenetic changes may influence contaminant dynamics. Older fish may possess more developed enzymatic systems and enhanced elimination pathways, such as excretion or reproduction, compared with younger individuals. Changes in diet during growth may also influence contaminant accumulation, as dietary composition can affect the intake of heavy metals (Salaah et al., 2025).

Fish accumulate heavy metals through multiple pathways, including branchial absorption from the aquatic environment, dermal contact with contaminated sediments, and dietary ingestion via the gastrointestinal tract through ingestion of food or sediment particles, as well as across permeable body surfaces (Ali & Khan, 2018; Sonone et al., 2020; Azar & Vajargah, 2023; Oros, 2025). Following uptake, heavy metals are distributed through the bloodstream to different organs, where they interact with metal-binding proteins, leading to their accumulation in tissues (Pargi et al., 2024). The degree of accumulation is determined by the physicochemical properties of the metals as well as by the physiological characteristics of the species. Gills represent the primary site of absorption due to their extensive contact surface and ion-exchange functions, while direct interaction with sediments also promotes accumulation, as reflected by the higher concentrations observed in benthic species compared with pelagic ones. Trophic exposure also constitutes a major pathway for metal incorporation, particularly at higher trophic levels (Oros, 2025). Zhou & Wong (2000) demonstrated that mercury undergoes clear biomagnification along the aquatic food chain, with higher concentrations in predatory species, whereas other metals such as Pb and Cd did not show biomagnification within the same system, indicating lower trophic transfer potential.

Exposure of fish to sublethal or lethal concentrations induces stress responses and leads to the accumulation of metals in various tissues and organs, including gills, kidneys, liver, skin, and muscle (Naz et al., 2023).

Most metals accumulate preferentially in the liver, kidneys, and gills, while muscle tissue generally exhibits lower concentrations compared with the other organs (Sonone et al., 2020). Given the essential roles of these organs in respiration, detoxification, and osmoregulation, their preferential accumulation of heavy metals may directly impair key physiological functions, thereby contributing to reduced biological performance and compromised welfare.

Sediments act as accumulation compartments for heavy metals (Ali et al., 2019; El-Sharkawy et al., 2025), as well as for a wide range of contaminants originating from both natural processes and anthropogenic activities (El-Sharkawy et al., 2025). They are recognized as long-term sinks for heavy metals, capable of retaining contaminants and acting as secondary sources under certain conditions (Ali et al., 2019; El-Sharkawy et al., 2025).

Due to their high density, heavy metals tend to accumulate in bottom sediments, where they can be taken up by benthic organisms and reintroduced into aquatic food webs (Almashhadany et al., 2024).

Fish, being positioned at higher trophic levels in aquatic ecosystems, are considered suitable model organisms for toxicological studies. Their high sensitivity to environmental changes, as well as their capacity to metabolize, detoxify, and accumulate heavy metals, make them effective bioindicators for monitoring the condition of aquatic ecosystems (Ray & Vashishth, 2024).

Overall, these findings indicate that heavy metal bioaccumulation in fish is a highly context-dependent process, influenced by species-specific traits, habitat characteristics, and exposure pathways. In particular, benthic and higher-trophic-level species are more prone to chronic exposure due to their feeding strategies and environmental interactions. Consequently, such patterns may lead to sustained accumulation and increase the risk of sublethal physiological disturbances, even in the absence of acute toxicity.

Heavy metals in aquatic environments: sources, behavior, and relevance for fish welfare

The term heavy metals refers to metals and metalloids characterized by an atomic density greater than 4 g/cm³ or at least five times greater than the density of water (Masindi & Muedi, 2018; Piwowarska et al., 2024).

Depending on their toxic properties, heavy metals are classified into essential and non-essential metals. Essential metals, such as zinc (Zn), copper (Cu), iron (Fe), nickel (Ni), and molybdenum (Mo), are required in small amounts for fundamental biological processes in animal life, exhibiting low toxicity at low concentrations and playing an important role in maintaining physiological functions, including enzymatic activity and metabolic regulation. In contrast, non-essential metals are characterized by a high toxic potential; cadmium (Cd), lead (Pb), mercury (Hg), and arsenic (As) are frequently identified as the most hazardous elements in this category (Ali & Khan, 2018; Piwowarska et al., 2024; Mustapha et al., 2025). The behavior of these metals in the environment is controlled by specific physicochemical properties that determine their mobility, persistence, and bioavailability (Mustapha et al., 2025).

Heavy metals are commonly present in natural waters at low concentrations; however, many of them exhibit toxicity even at very low levels (Masindi & Muedi, 2018). The fate of heavy metals in aquatic systems is controlled by physicochemical factors such as pH, redox potential, solubility, and metal speciation, which determine their mobility, persistence, and toxicity (Mustapha et al., 2025). These physicochemical characteristics directly influence metal bioavailability, thereby determining the extent of uptake and potential toxic effects in aquatic organisms, including fish.

In general, decreasing pH increases the solubility and mobility of metals, promoting their release into the water column, while oxidation state and complexation processes influence the chemical form and toxic potential of metals (Mustapha et al., 2025).

Environmental contamination with heavy metals is primarily driven by anthropogenic pressure associated with population growth, industrialization, and intensive agriculture (Aziz et al., 2023), although both natural and anthropogenic activities may contribute to environmental contamination, often exceeding the assimilative capacity of ecosystems (Mustapha et al., 2025).

The introduction of heavy metals into aquatic environments occurs through both anthropogenic and natural sources, with anthropogenic inputs being dominant and largely associated with industrial and agricultural activities such as mining operations, metallurgical processes, irrigation with contaminated water, and various industrial discharges. In particular, modern industrial processes such as electroplating, electronic equipment manufacturing, mining, metal smelting, fertilizer production, and activities within the chemical and energy sectors generate significant volumes of wastewater containing heavy metals, which enter aquatic environments through direct discharge or surface runoff. Natural contributions, although generally secondary in magnitude, include processes such as rock weathering, volcanic activity, fires, and biogenic mechanisms, which also supply heavy metals to aquatic ecosystems (Sonone et al., 2020; Aziz et al., 2023; Singh & Sharma, 2024). Once introduced into the environment, heavy metals may occur in various chemical forms, including hydroxides, oxides, sulfides, sulfates, phosphates, silicates, and organic compounds (Sonone et al., 2020). These properties result in their non-biodegradability, capacity for bioaccumulation, environmental stability, long-term persistence, and biotoxicity (Aziz et al., 2023; Edo et al., 2024). As a result, fish inhabiting contaminated environments may be continuously exposed to elevated metal

concentrations, increasing the likelihood of chronic accumulation and sublethal physiological stress.

Among the highly toxic heavy metals and metalloids of environmental relevance are chromium (Cr), nickel (Ni), copper (Cu), zinc (Zn), cadmium (Cd), lead (Pb), mercury (Hg), and arsenic (As) (Edo et al., 2024). Unlike organic compounds, most metals cannot be readily metabolized into less toxic forms, reflecting their lack of biodegradability. Consequently, once introduced into aquatic environments, metals are redistributed within the water column, accumulate in sediments, or are incorporated into biomass through trophic processes (Sonone et al., 2020). As a result, the persistence of heavy metals in aquatic systems is largely determined by their chemical stability, low degradation rates, and high affinity for sediment particles (El-Sharkawy et al., 2025).

Overall, the persistence and bioavailability of heavy metals in aquatic environments highlight their potential to act as chronic stressors for fish. Continuous exposure, even at low concentrations, may facilitate bioaccumulation and increase the risk of sublethal physiological disturbances, with potential implications for fish welfare.

A synthesis of the main findings on heavy metal exposure, including exposure routes, target organs, and observed effects in fish, is presented in Table 1.

Table 1. Summary of heavy metal exposure routes, target organs and observed effects in fish

Heavy Metal	Species	Concentration	Exposure Route	Target Organ	Observed Effect	Reference
Cd^{2+}	*Cyprinus carpio*	60-100 mg/L	Waterborne	Gills, liver, kidney	Gill damage (hyperplasia, necrosis); liver degeneration; kidney lesions; behavioral alterations (erratic swimming, loss of equilibrium, increased breathing).	Ambiya et al., 2024
Cd^{2+}	*Paralichthys olivaceus*	2-8 mg/L	Waterborne	Liver, kidney, gills, muscle	Oxidative stress (altered antioxidant enzymes, increased lipid peroxidation); growth reduction; behavioral alterations (reduced feeding, sluggish swimming, decreased activity), (liver > kidney > gills > muscle).	Cao et al., 2012
Cd^{2+}	*Lates calcarifer*	10 mg/L	Waterborne	Gills, liver, kidney	Behavioral alterations (erratic swimming, excessive mucus production, hyperventilation); gill lesions (edema, aneurism, hyperplasia, lamellar fusion); liver swelling/vacuolation; kidney tubular damage and necrosis.	Thophon et al., 2003
Cd^{2+}	*Lates calcarifer*	0.8 mg/L	Waterborne	Gills, liver, kidney	Reduced food intake; gill epithelial edema, hypertrophy/hyperplasia and lamellar fusion; liver lipid droplet and dark granule accumulation; kidney tubular degeneration, vacuolation and necrosis.	Thophon et al., 2003
Cd^{2+}	*Terapon jarbua*	0.08-0.86 μg g^{-1} day^{-1}	Dietary	Digestive tract, liver, gills	Metallothionein induction; limited physiological effects (no significant growth reduction).	Dang & Wang, 2009
Pb^{2+}	*Oreochromis mossambicus*	0.5-5 mg/L	Waterborne	Gills, liver, kidney, intestine, brain	Oxidative stress (GSH alteration, lipid peroxidation); ionoregulatory disruption (Na^+/K^+-ATPase inhibition); reduced feeding; lethargy; abnormal swimming behavior; respiratory distress (excessive mucus production).	Kaya & Akbulut, 2015
Pb^{2+}	*Squalus acanthias*	4.14 mg/L	Waterborne	Skin, gills, kidney, gut	Pb accumulation mainly in skin and gills; moderate accumulation in kidney and gut; decreased plasma K+; transient increase in blood pH; no mortality.	Eyckmans et al., 2013
Pb^{2+}	*Squalus acanthias*	20.72 mg/L	Waterborne	Skin, gills, kidney, gut, liver, muscle	Pb accumulation in all tissues (highest in skin and gills); decreased plasma K+, Na+ and Cl−; reduced gill Na+/K+-ATPase activity (ionoregulatory disruption); increased urea and ammonia excretion; decreased liver protein and muscle glycogen; no mortality.	Eyckmans et al., 2013
Pb^{2+}	*Cyprinus carpio*	0.21 mg/L	Waterborne	Gills, liver, kidney	Histopathological alterations in gills (hyperplasia, epithelial lifting); liver degeneration; renal histopathological alterations (tubular degeneration, glomerular alterations).	Mustafa et al., 2017

Pb^{2+}	*Cyprinus carpio*	0.42 mg/L	Waterborne	Gills, liver, kidney	Severe histopathological alterations in gills (edema, lamellar fusion, necrosis); liver degeneration (vacuolation) and necrosis; renal histopathological alterations (tubular degeneration, vacuolation, necrosis).	Mustafa et al., 2017
Pb^{2+}	*Clarias gariepinus*	6.1 mg/L	Waterborne	Gills, liver, kidney, muscle	Mild histopathological alterations in gills (epithelial lifting, blood congestion); liver vacuolar degeneration; kidney glomerular alterations; muscle structural gaps.	Al-Balawi et al., 2013
Pb^{2+}	*Clarias gariepinus*	24.4 mg/L	Waterborne	Gills, liver, kidney, muscle	Severe histopathological alterations in gills (hypertrophy, lamellar degeneration, vacuolar degeneration); liver vacuolation and necrosis; kidney tubular necrosis and glomerular alterations; muscle fiber separation.	Al-Balawi et al., 2013
Pb^{2+}	*Oncorhynchus mykiss*	0.57-1.2 mg/L	Waterborne	Kidney	Renal Pb accumulation; decreased glomerular filtration rate (GFR); altered urine pH; increased ammonia and glucose excretion; disrupted Ca^{2+} and Mg^{2+} reabsorption; disruption of ion homeostasis.	Patel et al., 2006
Pb^{2+}	*Danio rerio*	0.0025-0.005 mg/L	Waterborne	Liver	Dose-dependent histopathological alterations, ranging from mild to severe, including bile duct degeneration and epithelial detachment, vessel/sinusoid congestion, cytoplasmic vacuolization, increased lipid droplets, macrophage proliferation, apoptotic and necrotic hepatocytes, and lysed areas; progressive increase in severity and earlier onset at higher concentration; upregulation of mtf1, cat and sod1.	Macirella et al., 2023
Pb^{2+}	*Oreochromis niloticus*	100-800 mg/kg diet	Dietary	Kidney, liver, gills, spleen (lower in muscle, brain)	Dose-dependent Pb accumulation (highest in kidney); oxidative stress (↑ MDA, ↓ GSH, SOD, GSH-Px, T-AOC); DNA damage in blood cells. Although dietary exposure implies gastrointestinal uptake, no direct measurements of Pb accumulation in intestinal tissues were reported.	Dai et al., 2012
Pb^{2+}	*Sebastes schlegelii*	30-240 mg/kg diet	Dietary	Liver, gills, brain, muscle	Increased SOD and GST activity, decreased GSH levels (oxidative stress); AChE inhibition in brain and muscle (neurotoxicity); increased MT expression in liver; effects varying with exposure concentration and duration.	Kim et al., 2017
iHg	*Brycon amazonicus*	0.15 mg/L	Waterborne	Gills, liver, white muscle, heart	Behavioral alterations (hyperactivity, aggressiveness, loss of equilibrium, abnormal swimming, dark skin pigmentation, loss of scales); Hg bioaccumulation (gills > liver > heart ≈ white muscle); antioxidant response activation; lipid peroxidation and protein oxidation; MT increase in liver, gills and heart.	Monteiro et al., 2010
iHg	*Gambusia affinis*	0.02 mg/L	Waterborne	Gills, viscera, head, body	Behavioral alterations (erratic swimming, loss of equilibrium, lethargy) and reduced swimming performance; severe gill damage (hyperplasia, hypertrophy, lamellar fusion, necrosis); Hg bioaccumulation (viscera > head > body).	Jakka et al., 2007
iHg	*Diplodus sargus*	0.002 mg/L	Waterborne	Brain (hypothalamus, optic tectum, cerebellum)	Brain cell loss (neuronal and glial), reduced cerebellum volume; impaired swimming performance (reduced distance and velocity); altered anxiety-like and mood-related behavior; persistent post-exposure effects.	Pereira et al., 2016
MeHg	*Perca flavescens (larvae)*	0.007-0.075 mg/L	Waterborne (embryonic exposure)	Not specified	Impaired locomotor activity, altered visual motor response, reduced foraging efficiency, and decreased survival at highest exposure concentration.	Mora-Zamorano et al., 2017

MeHg	*Danio rerio*	0.001-0.015 mg/L	Waterborne	Not specified	Significant behavioral alterations including reduced swimming performance, speed, and acceleration, impaired orientation, increased inactivity, decreased aggression, and memory deficits; increased oxidative stress biomarkers (SOD, MDA), particularly at higher concentration.	Strungaru et al., 2018
iHg	*Hoplias malabaricus*	Exposure via contaminated prey (0.15 mg L^{-1} $HgCl_2$; 8.11 mg kg^{-1} Hg in prey; ~0.45 mg Hg/fish/96 h)	Dietary	Liver, gills, white muscle, heart	Increased aggressiveness and altered feeding behavior; increased liver mass and HSI; decreased condition factor; induction of oxidative stress (↑ SOD, CAT, GPx, GST, GR); lipid and protein oxidation; glutathione imbalance (↓ GSH/GSSG ratio); increased metallothioneins; tissue-specific mercury bioaccumulation (gills > liver > muscle = heart).	Monteiro et al., 2013
As V	*Danio rerio*	0.001-0.1 mg/L	Waterborne	Brain (functional)	Impaired long-term memory (LTM); decreased avoidance latency; increased protein oxidation.	de Castro et al., 2009
As III	*Oreochromis mossambicus*	(3-56 mg/L; LC50 = 28.22 mg/L)	Waterborne	Gills, liver	Dose-dependent behavioral alterations (hyperactivity followed by lethargy and loss of equilibrium); respiratory impairment; mucus hypersecretion; dose-dependent mortality; gill damage (necrosis, epithelial lifting, hyperplasia, lamellar fusion, aneurism); liver damage (vacuolation, necrosis, macrophage infiltration, sinusoidal dilation).	Ahmed et al., 2013
As III	*Betta splendens*	0.01-0.1 mg/L	Waterborne	Not specified	Loss of mate preference at 0.1 mg/L (no effect at 0.01 mg/L); increased anxiety-like behavior (increased time in dark); no change in total association time; no change in cortisol levels.	Tudor et al., 2019
As III	*Sebastes schlegelii*	0.05-0.4 mg/L	Waterborne	Liver, kidney, spleen, gill, intestine, muscle	Dose- and time-dependent bioaccumulation (liver > kidney > spleen > gill > intestine > muscle); induction of antioxidant defense system (↑ SOD, GST, GSH in liver and gills); strong positive correlation between tissue accumulation and antioxidant enzyme activity.	Kim & Kang, 2015
As III & As V	*Carassius auratus*	50 mg/kg diet	Dietary	Liver	Time-dependent bioaccumulation in liver (peak at 30 d followed by decline); species-dependent accumulation (higher in AsV vs AsIII at specific time points); biotransformation to less toxic forms (increased AsB, MMA, DMA); time-dependent modulation of antioxidant responses (SOD, CAT, GSH); transient oxidative damage (elevated MDA at 15 d in AsIII group); subsequent recovery indicating adaptive physiological response.	Cui et al., 2020

Table 1 summarizes the main patterns of heavy metal toxicity in fish, showing consistent tissue-specific accumulation and multi-level biological effects. Oxidative stress, histopathological damage, and behavioral alterations are among the most commonly reported responses across different metals and exposure routes. These findings support the relevance of these endpoints in assessing fish welfare under metal exposure.

Cadmium

Cadmium is a highly toxic non-essential metal that can induce both acute and chronic effects in aquatic organisms even at low concentrations (Elbeshti et al., 2018). In aquatic environments, cadmium originates from both natural geological processes and anthropogenic sources, the latter being the dominant contributors, particularly through agricultural inputs (e.g., fertilizers), mining, metal smelting, and the use of pesticides and herbicides (Yang et al., 2025). Fish absorb dissolved cadmium mainly primarily through the gills, but also via the gastrointestinal tract and skin, while trophic transfer through a contaminated diet represents an additional route of exposure (Garai et al., 2021). The skin and, especially, the gills constitute the primary entry routes for waterborne cadmium, as indicated by the absence of digestive tract lesions commonly associated with dietary exposure (Annabi et al., 2013).

Following uptake, cadmium accumulates in fish tissues through binding to metallothioneins, which facilitate its intracellular sequestration. In addition, renal excretion of cadmium involves multidrug and toxic extrusion (MATE) transporters; however, increased intracellular Cd^{2+} levels can inhibit this pathway, potentially enhancing metal retention within the organism (Lee et al., 2024).

At the cellular level, cadmium toxicity is strongly associated with oxidative stress. It can impair mitochondrial electron transport and indirectly stimulate the formation of reactive oxygen species (ROS) by increasing the availability of redox-active iron, thereby promoting Fenton-related reactions (Garai et al., 2021; Zahran et al., 2025).

Verbost et al. (1987) demonstrated that cadmium inhibits Ca^{2+} uptake at the gill level in rainbow trout by impairing branchial calcium transport mechanisms, resulting in disturbances in calcium homeostasis and hypocalcemia. However, Franklin et al. (2005) reported no significant alterations in Ca^{2+} uptake or plasma Ca^{2+} levels under chronic exposure to lower cadmium concentrations, suggesting that these effects may be concentration-dependent. Such disruptions in calcium homeostasis may impair ion regulation and gill function, with direct consequences for respiratory efficiency and overall physiological stability.

Once absorbed, cadmium is unevenly distributed among fish tissues, with the highest accumulation typically observed in the liver and kidney, while gills, intestine, and muscle may also show elevated concentrations depending on exposure conditions and duration (Perera et al., 2015). Similarly, cadmium exposure has been associated with structural and functional impairment of major organs, particularly the gills, liver, and kidneys (Elbeshti et al., 2018; Garai et al., 2021; Emon et al., 2023). Cadmium exposure leads to physiological disturbances and reduced growth performance (Perera et al., 2015; Emon et al., 2023), and has also been associated with renal dysfunction and impaired reproductive capacity in fish, reflecting chronic toxic effects on key physiological systems (Afshan et al., 2014). These effects may reflect increased energetic costs associated with detoxification and stress responses, reducing the resources available for growth and reproduction. Collectively, these alterations impair key physiological functions, particularly those associated with gill and kidney function, including respiration, osmoregulation, and detoxification, thereby compromising fish welfare by reducing their capacity to cope with environmental stressors.

Lead

Lead is a non-biodegradable and bioaccumulative metal that readily binds to oxygen- and sulfur-containing groups in proteins, forming stable complexes that contribute to its persistence and toxicity in biological systems (Sarkar et al., 2022).

Lead occurs naturally in the Earth's crust; however, elevated concentrations in aquatic environments are primarily associated with anthropogenic activities such as mining, smelting, industrial discharges, and the use of lead-containing products, as well as atmospheric deposition and surface runoff (Ishaque et al., 2020; Simatupang et al., 2024).

In aquatic environments, lead bioavailability is strongly influenced by its speciation, as it rapidly binds to suspended particles and sediments, reducing its dissolved ionic fraction. However, sediment-associated lead and interstitial water can still represent important exposure routes, particularly for benthic organisms and fish interacting with bottom substrates (Prosi, 1989).

The meta-analysis conducted by Demir et al. (2025) showed that Pb accumulation in fish varies among tissues, with higher concentrations generally observed in the liver and gills, followed by muscle tissue. Mean Pb concentrations were reported as 0.41 μg/g in liver, 0.33 μg/g in gills, and 0.27 μg/g in muscle. Lead accumulation and its physiological effects in fish show considerable variability depending on exposure route and duration. For instance, Crespo et al. (1986) reported pronounced intestinal and systemic alterations following chronic dietary exposure, highlighting the importance of gastrointestinal uptake in mediating toxicity. Ciardullo et al. (2008) observed relatively low muscle accumulation under long-term exposure conditions, but distinct tissue-specific distribution patterns, with lead preferentially accumulating in skin, kidney, and gills. Similarly, Javed (2012)

demonstrated that sublethal exposure resulted in age-dependent differences in both accumulation and growth performance, suggesting that physiological responses to lead are strongly influenced by developmental stage. Together, these findings indicate that lead toxicity cannot be generalized across exposure scenarios, but rather reflects a complex interaction between exposure pathway, duration, and organismal characteristics.

Histopathological alterations such as lamellar shortening, epithelial disruption, and structural disorganization have also been reported in gill tissues of lead-exposed fish (Jan et al., 2022). Acute lead toxicity damages the gill epithelium, impairing respiration and potentially leading to suffocation, while also promoting reactive oxygen species (ROS) production, which induces oxidative stress and results in damage to lipids, proteins, and DNA (Authman et al., 2015). Given the central role of gills in gas exchange and ion regulation, such structural damage may directly impair respiratory efficiency and osmoregulatory balance, thereby affecting overall physiological stability in fish.

Lead exposure in fish induces neurotoxicity by disrupting cellular signaling and neurotransmission, leading to behavioral impairments, while also altering immune function and overall physiological performance, ultimately compromising long-term fitness (Lee et al., 2019; Das et al., 2022). These alterations may reduce the ability of fish to respond to environmental challenges, affecting feeding behavior, predator avoidance, and overall performance.

Lead can also impair early life stages in fish and is associated with delayed hatching, developmental retardation, and morphological abnormalities in embryos and larvae (Sneha et al., 2026). In addition, lead exposure alters hematological and biochemical parameters, including changes in blood profiles and enzyme activities in plasma and liver, indicating physiological stress and tissue damage (Saket et al., 2025). Fish acutely exposed to Pb may exhibit haemoglobin alterations, black tail disease, and spinal deformities (Das et al., 2022).

Collectively, these physiological and behavioral alterations indicate that lead exposure disrupts key functions such as respiration, ion regulation, and neurological control, linking tissue-level damage to reduced organismal performance and compromised fish welfare.

Mercury

Mercury occurs in various chemical forms in aquatic environments, and its toxicity largely depends on its speciation. In particular, microbial processes can transform inorganic mercury into methylmercury, a highly bioavailable form responsible for most toxic effects in aquatic organisms (Ullrich et al., 2001). This biomethylation process occurs predominantly under anaerobic conditions in sediments, where microbial activity facilitates the conversion of inorganic mercury into methylmercury (Gworek et al., 2016).

Methylmercury is efficiently absorbed in fish, with approximately 95% of ingested MeHg taken up through the gastrointestinal tract. Following absorption, it readily binds to thiol-containing molecules such as cysteine and glutathione, facilitating its distribution into red blood cells and critical target tissues, including the brain. Due to its limited excretion and long biological half-life, methylmercury is retained within the organism, contributing to its progressive accumulation and persistence in fish tissues (Rice et al., 2014). Methylmercury undergoes efficient trophic transfer in aquatic food webs, resulting in biomagnification and elevated mercury concentrations in predatory fish, while bioconcentration from the water column further contributes to its overall bioaccumulation in aquatic organisms (Lavoie et al., 2013; Wu et al., 2019). Wang et al. (2010) showed that trophic transfer plays a more important role than waterborne exposure in mercury accumulation in tilapia species. This highlights the importance of dietary exposure in higher trophic levels, where biomagnification may lead to sustained internal mercury burdens and increased risk of chronic toxicity.

Mercury contamination in aquatic systems arises from both natural and anthropogenic sources. Natural emissions are associated with geological and geochemical processes, including volcanic eruptions, geothermal activity, soil degassing, and forest fires, with their intensity depending on regional characteristics. However, anthropogenic inputs represent a major source of mercury release,

mainly driven by industrial activities such as mining and smelting, artisanal gold mining, coal combustion, oil refining, chlor-alkali processes, and cement production (Al-Sulaiti et al., 2022).

In fish tissues, mercury is predominantly present as methylmercury (≈70-95%), with inorganic forms representing only a minor fraction (≈5-15%) (Suedel et al., 1994). Mercury in fish tissues is typically highest in the liver, followed by the kidney, muscle, gonads, and brain, with the liver being a primary target organ for toxicity and histopathological assessment (Zulkipli et al., 2021). Mieiro et al. (2009) reported that in *Liza aurata*, mercury accumulation followed the hierarchy kidney > liver > muscle > brain > gills > blood, with the highest concentrations observed in kidney and liver. The authors also noted that tissue distribution patterns may vary among species, although most studies consistently identify liver and kidney as the main organs of mercury accumulation.

Vieira et al. (2009) showed that acute exposure (96 h) to environmentally relevant mercury concentrations (3.1-200 μg L^{-1}) in *Pomatoschistus microps* impaired swimming behavior and induced oxidative stress and neurotoxicity (AChE inhibition), leading to reduced functional performance and fitness. Such neurotoxic effects may impair essential behaviors such as feeding, predator avoidance, and spatial orientation, thereby reducing survival and ecological performance.

Jasim et al. (2016) reported that acute mercury exposure in *Oreochromis* sp. induced behavioral disturbances, including hyperactivity, aggressiveness, and respiratory distress, while also resulting in preferential accumulation in the liver and gills, accompanied by marked branchial and hepatic lesions. Kania & O'Hara (1974) reported that mercury exposure in mosquitofish (*Gambusia affinis*) impairs escape responses, increasing vulnerability to predation and reducing survival, with behavioral alterations observed at body burdens as low as 0.67 ppm (wet weight).

Mercury exposure has been associated with reproductive impairment in fish, including reduced gametogenesis, altered steroidogenesis, and decreased spawning success, likely mediated by disruption of the hypothalamic-pituitary-gonadal axis (Crump & Trudeanu, 2009). These effects are further exacerbated during early life stages, where exposure reduces hatching success, delays development, increases mortality, and induces morphological abnormalities, including skeletal deformities. Such alterations impair functional performance and survival, while additional effects on feeding behavior may further compromise growth and overall fitness (Zheng et al., 2019).

Collectively, these alterations indicate that mercury exposure disrupts key functions such as neurological control, reproduction, and behavior, linking tissue-level accumulation to reduced performance and compromised fish welfare.

Arsenic

Inorganic arsenic forms generally exhibit higher toxicity than organic species. In aquatic environments, arsenic occurs mainly as arsenite As(III) and arsenate As(V), with arsenite being the more toxic form. The relative distribution of these species is controlled by environmental redox conditions, which in turn influence arsenic bioavailability and toxicity in aquatic organisms (Sharma & Sohn, 2009; Kumari et al., 2017). In addition to redox conditions, arsenic speciation is influenced by environmental factors such as pH and interactions with other elements, contributing to its dynamic behavior in aquatic systems (Campbell & Nordstrom, 2014).

Arsenic enters aquatic environments from both natural and anthropogenic sources. Natural inputs mainly result from rock weathering and geothermal activity, while major anthropogenic sources include mining, smelting, industrial discharges, and agricultural practices. Atmospheric transport and deposition also contribute to arsenic input in aquatic systems. Once released, arsenic accumulates in water and sediments, which can act as both sinks and secondary sources of contamination (Wang et al., 2022; Patel et al., 2023).

In fish, dietary arsenic is absorbed through the gastrointestinal tract and subsequently distributed to various organs via the circulatory system (Zhang et al., 2022). In addition to dietary exposure, waterborne arsenic can be absorbed through the gills, skin, and digestive tract, with the skin representing a potentially important uptake route in smaller fish due to their higher surface area-to-volume ratio (Ghosh

et al., 2022). In higher trophic level organisms, food intake represents a major exposure pathway and can account for the majority of arsenic bioaccumulation (Zhang et al., 2022). Due to its structural similarity to phosphate, arsenate can also enter cells via phosphate transport pathways, representing a key mechanism of cellular uptake (Yang et al., 2012).

Following uptake, arsenic is unevenly distributed among fish tissues, with higher concentrations typically found in the liver, gills, and gastrointestinal tract, and lower levels in muscle. Both inorganic (As(III), As(V)) and organic species, such as arsenobetaine and DMA, are present across tissues, with their relative proportions varying depending on tissue type and species (Hoy et al., 2023). Arsenic does not consistently biomagnify across aquatic food webs and behaves differently from elements such as mercury, with trophic transfer often being variable or even characterized by biodilution (Córdoba-Tovar et al., 2022). The liver is consistently reported as the primary site of arsenic accumulation, reflecting its central role in metabolism and detoxification processes. In general, arsenic distribution follows a decreasing pattern from metabolically active organs such as the liver and kidney to less active tissues, including muscle (Jabeen & Javed, 2011).

Arsenic exposure has been shown to alter fish behavior, including increased aggression and reduced opercular movement, indicating potential respiratory impairment. Additionally, arsenic exposure can negatively affect growth and increase mortality, with both waterborne and dietary pathways contributing to these effects (Kanwal et al., 2019). Tang et al. (2024) further demonstrated that acute arsenic exposure induces pronounced behavioral alterations, respiratory distress, and visible gill damage, including loss of equilibrium, increased ventilation, and structural deterioration of gill tissues. At the tissue level, Ahmed et al. (2013) demonstrated that arsenic exposure induces histopathological alterations in fish, including gill lesions such as epithelial lifting, hyperplasia, and lamellar fusion, as well as degenerative changes in the liver. Such impairments in gill structure and function may directly reduce respiratory efficiency and limit the ability of fish to maintain adequate oxygen supply under environmental stress.

Consistent with these findings, arsenic exposure is associated with increased aminotransferase activity, indicating hepatotoxic effects in fish (Kanwal et al., 2019). In addition, arsenic toxicity is associated with the induction of oxidative stress through the generation of reactive oxygen species (ROS), which can lead to lipid peroxidation and protein damage. Antioxidant defenses, particularly glutathione (GSH), play a key role in mitigating these effects, while depletion of GSH is linked to increased susceptibility to arsenic-induced damage (Ventura-Lima et al., 2011). This imbalance between pro-oxidant and antioxidant processes may compromise cellular integrity and reduce the overall physiological resilience of fish.

Overall, the reviewed evidence indicates that the relative toxicity of heavy metals in fish depends on metal speciation, exposure route, tissue distribution, and the physiological system affected. Cadmium primarily targets the gills, liver, and kidneys, with toxicity strongly linked to oxidative stress, calcium homeostasis disruption, impaired ion regulation, and reduced growth or reproductive performance. Lead also affects gill and kidney function, but its toxicity is particularly relevant through ionoregulatory disruption, neurotoxicity, hematological alterations, and behavioral impairment. Mercury, especially in its methylated form, shows the highest concern for trophic transfer and biomagnification, with strong neurotoxic, behavioral, reproductive, and developmental effects, particularly in predatory fish. Arsenic toxicity is strongly influenced by speciation, with inorganic forms, especially As(III), being more toxic than organic forms; its effects are mainly associated with gill and liver damage, oxidative stress, impaired respiration, altered behavior, and variable trophic transfer. Therefore, mercury appears most critical in terms of biomagnification and neurobehavioral risk, cadmium and lead are highly relevant for organ-level physiological disruption, while arsenic toxicity is strongly dependent on chemical form and environmental conditions.

CONCLUSIONS

Heavy metals represent persistent aquatic contaminants of major concern due to their non-biodegradability, bioaccumulation potential, and capacity to impair fish health and welfare.

The reviewed literature indicates that heavy metal exposure affects fish through multiple toxicological pathways, including oxidative stress, endocrine disruption, tissue damage, and developmental impairment, with consequences for physiological functioning.

Bioaccumulation patterns are influenced by environmental conditions, trophic position, exposure route, and species-specific traits, while metal accumulation occurs preferentially in organs such as the liver, kidneys, and gills rather than in muscle tissue.

Beyond tissue-level and physiological alterations, heavy metals also induce behavioral disturbances that serve as sensitive indicators of toxic stress and declining welfare.

Evidence synthesized across cadmium, lead, mercury, and arsenic highlights that both acute and chronic exposure can compromise respiration, osmoregulation, growth, reproduction, behavior, and overall functional performance in fish.

Overall, this review supports the view that heavy metal exposure represents a significant welfare risk in aquatic environments, including under sublethal conditions where impaired biological functioning occurs in the absence of immediate mortality. Importantly, these findings demonstrate that traditional mortality-based assessments underestimate the real impact of heavy metals, as substantial welfare impairment can arise even at sublethal exposure levels.

REFERENCES

Afshan, S., Ali, S., Ameen, U. S., Farid, M., Bharwana, S. A., Hannan, F., & Ahmad, R. (2014). Effect of different heavy metal pollution on fish. *Research Journal of Chemical and Environmental Sciences*, 2(1), 74-79.

Ahmad, M. K., Islam, S., Rahman, C. S. O., Haque, M., & Islam, M. (2010). Heavy metals in water, sediment and some fishes of Buriganga River, Bangladesh. *International Journal of Environmental Research*, 4, 321-332.

Ahmed, M. K., Habibullah-Al-Mamun, M., Parvin, E., Akter, M. S., & Khan, M. S. (2013). Arsenic induced toxicity and histopathological changes in gill and liver tissue of freshwater fish, tilapia (*Oreochromis mossambicus*). *Experimental and Toxicologic Pathology*, 65(6), 903-909.

Ali, H., & Khan, E. (2018). Bioaccumulation of non-essential hazardous heavy metals and metalloids in freshwater fish: Risk to human health. *Environmental Chemistry Letters,* 16, 1-15. https://doi.org/10.1007/s10311-018-0734-7

Ali, H., & Khan, E. (2019). Trophic transfer, bioaccumulation, and biomagnification of non-essential hazardous heavy metals and metalloids in food chains/webs - Concepts and implications for wildlife and human health. *Human and Ecological Risk Assessment: An International Journal*, 25(6), 1353-1376.

Ali, H., Khan, E., & Ilahi, I. (2019). Environmental chemistry and ecotoxicology of hazardous heavy metals: environmental persistence, toxicity, and bioaccumulation. *Journal of Chemistry*, 2019, 6730305.

Al-Balawi, H. F. A., Al-Akel, A. S., Al-Misned, F., Suliman, E. A. M., Al-Ghanim, K. A., Mahboob, S., & Ahmad, Z. (2013). Effects of sub-lethal exposure of lead acetate on histopathology of gills, liver, kidney and muscle and its accumulation in these organs of Clarias gariepinus. *Brazilian Archives of Biology and Technology*, 56, 293-302.

Almashhadany, D. A., Rashid, R. F., Altaif, K. I., Mohammed, S. H., Mohammed, H. I., & Al-Bader, S. M. (2024). Heavy metal(loid) bioaccumulation in fish and its implications for human health. *Italian Journal of Food Safety*, 14(1). https://doi.org/10.4081/ijfs.2024.12782

Al-Sulaiti, M. M., Soubra, L., & Al-Ghouti, M. A. (2022). The causes and effects of mercury and methylmercury contamination in the marine environment: a review. *Current Pollution Reports*, 8(3), 249-272

Ambiya, M. G., Nath, S., Haque, S., & Mondal, K. (2024). Studies on the effect of cadmium chloride on the behavioral and histopathological changes in Cyprinus carpio: a short-term bioassay. *Environ Ecol*, 42(2B), 790-800.

Annabi, A., Said, K., & Messaoudi, I. (2013). Cadmium: bioaccumulation, histopathology and detoxifying mechanisms in fish. *American Journal of Research Communication*, 1(4), 62.

Aski, M. A. H., Ghobadi, S., Sari, A. A., Ardeshir, R. A., Arabi, M. H. G., & Manouchehri, H. (2023). Health risk assessment of heavy metals (Zn, Pb, Cd, and Hg) in water and muscle tissue of farmed carp species in North Iran. *Environmental Science and Pollution Research*, 30, 32464–32472. https://doi.org/10.1007/s11356-022-24043-z

Authman, M. M., Zaki, M. S., Khallaf, E. A., & Abbas, H. H. (2015). Use of fish as bio-indicator of the effects of heavy metals pollution. *Journal of Aquaculture Research & Development*, 6(4), 1-13.

Azar, H., & Vajargah, M. F. (2023). Investigating the effects of accumulation of lead and cadmium metals in fish and its impact on human health. *J Aquac Mar Biol*, 12(2), 209-213.

Aziz, K. H. H., Mustafa, F. S., Omer, K. M., Hama, S., Hamarawf, R. F., Rahman, K. O. (2023). Heavy metal pollution in the aquatic environment: Efficient and low-cost removal approaches to eliminate their

toxicity: a review. *RSC Advances*, 13(26), 17595-17610. https://doi.org/10.1039/D3RA00723E

Bawuro, A. A., Voegborlo, R. B., & Adimado, A. A. (2018). Bioaccumulation of heavy metals in some tissues of fish in Lake Geriyo, Adamawa State, Nigeria. *Journal of Environmental and Public Health*, 2018(1), 1854892.

Bhat, R. A., Alam, A., Jha, D. N., Kumar, V., Kumar, J., Thakur, V. R., & Das, B. K. (2025). Fate and effects of heavy metals in fishes: Antioxidant defense system, miRNA/gene expression response, and histopathological reproductive manifestations. *Biological Trace Element Research*, 203(8), 4326-4346.

Campbell, K. M., & Nordstrom, D. K. (2014). Arsenic speciation and sorption in natural environments. *Reviews in mineralogy and geochemistry*, 79(1), 185-216.

Cao, L., Huang, W., Shan, X., Ye, Z., & Dou, S. (2012). Tissue-specific accumulation of cadmium and its effects on antioxidative responses in Japanese flounder juveniles. *Environmental Toxicology and Pharmacology*, 33(1), 16-25.

Ciardullo, S., Aureli, F., Coni, E., Guandalini, E., Iosi, F., Raggi, A., Rufo, G., Cubadda, F. (2008). Bioaccumulation potential of dietary arsenic, cadmium, lead, mercury, and selenium in organs and tissues of rainbow trout (*Oncorhyncus mykiss*) as a function of fish growth. *Journal of Agricultural and Food Chemistry*, 56(7), 2442-2451.

Córdoba-Tovar, L., Marrugo-Negrete, J., Barón, P. R., & Díez, S. (2022). Drivers of biomagnification of Hg, As and Se in aquatic food webs: a review. *Environmental Research*, 204, 112226.

Crespo, S., Nonnotte, G., Colin, D. A., Leray, C., Nonnotte, L., & Aubree, A. (1986). Morphological and functional alterations induced in trout intestine by dietary cadmium and lead. *Journal of Fish Biology*, 28(1), 69-80.

Crump, K. L., & Trudeau, V. L. (2009). Mercury-induced reproductive impairment in fish. *Environmental Toxicology and Chemistry*, 28(5), 895-907.

Cui, D., Zhang, P., Li, H., Zhang, Z., Song, Y., & Yang, Z. (2020). The dynamic effects of different inorganic arsenic species in crucian carp (Carassius auratus) liver during chronic dietborne exposure: bioaccumulation, biotransformation and oxidative stress. *Science of the Total Environment, 727, 138737.*

Das, S., Mandal, S., Dhara, H., & Chatterjee, P. N. (2022). Dietary amelioration of lead toxicity in fish-A review. *Indian J Anim Health*, 61(2), 173-189.

Dang, F., & Wang, W. X. (2009). Assessment of tissue-specific accumulation and effects of cadmium in a marine fish fed contaminated commercially produced diet. *Aquatic Toxicology*, 95(3), 248-255.

Dai, W., Liu, S., Fu, L., Du, H., & Xu, Z. (2012). Lead (Pb) accumulation, oxidative stress and DNA damage induced by dietary Pb in tilapia (*Oreochromis niloticus*). *Aquaculture Research*, 43(2), 208-214.

Demir, T., Tutun, S., & Karapınar, B. (2025). A Systematic Review and Meta-Analysis of Lead Concentrations in Fish: Implications for Dietary Exposure and Risk Assessment. *MEMBA Su Bilimleri Dergisi*, 11(3), 375-387.

de Castro, M. R., Ventura Lima, J., Salomão de Freitas, D. P., de Souza Valente, R., Seus Dummer, N., Borba de Aguiar, R., Cougo dos Santos, L., Marins, L. F., Geracitano, L. A., Monserrat, J. M., & Martí Barros, D. (2009). Behavioral and neurotoxic effects of arsenic exposure in zebrafish (*Danio rerio*, Teleostei: Cyprinidae). *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*, 150(3), 337-342.

Edo, G. I., Samuel, P. O., Oloni, G. O., Ezekiel, G. O., Ikpekoro, V. O., Obasohan, P., ... & Agbo, J. J. (2024). Environmental persistence, bioaccumulation, and ecotoxicology of heavy metals. *Chemistry and Ecology*, 40(3), 322-349.

Elbeshti, R. T. A., Elderwish, N. M., Abdelali, K. M. K., & Tastan, Y. (2018). Effects of heavy metals on fish. *Menba Journal of Fisheries Faculty*, 4(1), 36-47.

El-Sharkawy, M., Alotaibi, M. O., Li, J., Du, D., & Mahmoud, E. (2025). Heavy metal pollution in coastal environments: Ecological implications and management strategies: A review. *Sustainability*, 17(2), 701. https://doi.org/10.3390/su17020701

Eyckmans, M., Lardon, I., Wood, C. M., & De Boeck, G. (2013). Physiological effects of waterborne lead exposure in spiny dogfish (Squalus acanthias). *Aquatic Toxicology*, 126, 373-381.

Emon, J. F., Rohani, M. F., Sumaiya, N., Tuj Jannat, M. F., Akter, Y., Shahjahan, M., ... & Goh, K. W. (2023). Bioaccumulation and bioremediation of heavy metals in fishes - a review. *Toxics*, 11(6), 510.

European Council. (1998). Council Directive 98/58/EC of 20 July 1998 concerning the protection of animals kept for farming purposes. Available online: https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:31998L0058

European Food Safety Authority (EFSA). (2009). Species-specific welfare aspects of the main systems of stunning and killing of farmed Atlantic salmon. *EFSA Journal*, 1012, 1-77.

Fife-Cook, I., & Franks, B. (2019). Positive welfare for fishes: Rationale and areas for future study. *Fishes*, 4(2), 31.

Franklin, N. M., Glover, C. N., Nicol, J. A., & Wood, C. M. (2005). Calcium/cadmium interactions at uptake surfaces in rainbow trout: waterborne versus dietary routes of exposure. *Environmental Toxicology and Chemistry*, 24(11), 2954-2964.

Garai, P., Banerjee, P., Mondal, P., & Saha, N. C. (2021). Effect of heavy metals on fishes: *Toxicity and bioaccumulation. J Clin Toxicol*. S, 18(001).

Ghosh, D., Ghosh, A., & Bhadury, P. (2022). Arsenic through aquatic trophic levels: effects, transformations and biomagnification - a concise review. *Geoscience Letters*, 9(1), 20.

Gworek, B., Bemowska-Kałabun, O., Kijeńska, M., & Wrzosek-Jakubowska, J. (2016). Mercury in marine and oceanic waters - a review. *Water, Air, & Soil Pollution*, 227(10), 371.

Habib, S., Naz, S., Fazio, F., Cravana, C., Ullah, M., Rind, K., Attaullah, S., Filiciotto, F., & Khayyam, K. (2023). Assessment and bioaccumulation of heavy metals in

water, fish (wild and farmed) and associated human health risk. *Biological Trace Element Research*, 202. https://doi.org/10.1007/s12011-023-03703-2

Hossain, M. B., Tanjin, F., Rahman, M. S., Yu, J., Akhter, S., Noman, M. A., & Sun, J. (2022). Metals bioaccumulation in 15 commonly consumed fishes from the lower Meghna river and adjacent areas of Bangladesh and associated human health hazards. *Toxics*, 10(3), 139.

Hoy, K. S., Davydiuk, T., Chen, X., Lau, C., Schofield, J. R. M., Lu, X., ... & Reichert, M. (2023). Arsenic speciation in freshwater fish: challenges and research needs. *Food Qual. Saf*., 7, fyad032.

Huntingford, F. A., Adams, C., Braithwaite, V. A., Kadri, S., Pottinger, T. G., Sandøe, P., & Turnbull, J. F. (2006). Current issues in fish welfare. *Journal of Fish Biology*, 68, 332-372.

Islam, M. S., Ahmed, M. K., Habibullah-Al-Mamun, M., & Masunaga, S. (2015). Assessment of trace metals in fish species of urban rivers in Bangladesh and health implications. *Environmental Toxicology and Pharmacology*, 39, 347-357. https://doi.org/10.1016/j.etap.2014.12.009

Ishaque, A., Ishaque, S., Arif, A., & Abbas, H. G. (2020). Toxic effects of lead on fish and human. *Biological and Clinical Sciences Research Journal*, 2020(1), e045.

Jabeen, G., & Javed, M. (2011). Evaluation of arsenic toxicity to biota in river Ravi (Pakistan) aquatic ecosystem. *International Journal of Agriculture and Biology*, 13(6).

Jakka, N. M., Gnaneshwar Rao, T., & Venkateswara Rao, J. (2007). Locomotor behavioral response of mosquitofish (*Gambusia affinis*) to subacute mercury stress monitored by video tracking system. *Drug and Chemical Toxicology*, 30(4), 383-397.

Jasim, M. A., Sofian-Azirun, M., Yusoff, I., & Rahman, M. M. (2016). Bioaccumulation and histopathological changes induced by toxicity of mercury (HgCl2) to tilapia fish *Oreochromis niloticus*. *Sains Malaysiana*, 45(1), 119-127.

Jan, A., Banerjee, S., & Chouhan, R. (2022). Heavy metal toxicity, bioaccumulation and oxidative stress in freshwater fishes: A systematic review. Uttar Pradesh *Journal of Zoology*, 43(24), 333–349.

Javed, M. (2012). Effects of zinc and lead toxicity on the growth and their bioaccumulation in fish. *Pak. Vet. J,* 32(3), 357-62.

Jerez-Cepa, I., & Ruiz-Jarabo, I. (2021). Physiology: An important tool to assess the welfare of aquatic animals. *Frontiers in Physiology*, 12, 755706. https://doi.org/10.3389/fphys.2021.755706

Kanwal, M., Hussain, M., & Younus, W. (2019). Hazardous effects of arsenic contaminated water on the biological characteristics of fishes: A review. *BioScientific Review*, 1(3), 01-08.

Kania, H. J., & O'Hara, J. (1974). Behavioral alterations in a simple predator-prey system due to sublethal exposure to mercury. *Transactions of the American Fisheries Society*, 103(1), 134-136.

Kaya, H., & Akbulut, M. (2015). Effects of waterborne lead exposure in Mozambique tilapia: oxidative stress, osmoregulatory responses, and tissue accumulation. *Journal of Aquatic Animal Health*, 27(2), 77-87.

Kim, J. H., & Kang, J. C. (2015). The arsenic accumulation and its effect on oxidative stress responses in juvenile rockfish, Sebastes schlegelii, exposed to waterborne arsenic (As3+). *Environmental Toxicology and Pharmacology*, 39(2), 668-676.

Kim, J. H., Oh, C. W., & Kang, J. C. (2017). Antioxidant responses, neurotoxicity, and metallothionein gene expression in juvenile Korean rockfish Sebastes schlegelii under dietary lead exposure. *Journal of Aquatic Animal Health*, 29(2), 112-119.

Kumari, B., Kumar, V., Sinha, A. K., Ahsan, J., Ghosh, A. K., Wang, H., & DeBoeck, G. (2017). Toxicology of arsenic in fish and aquatic systems. *Environmental Chemistry Letters*, 15(1), 43-64.

Lavoie, R. A., Jardine, T. D., Chumchal, M. M., Kidd, K. A., & Campbell, L. M. (2013). Biomagnification of mercury in aquatic food webs: a worldwide meta-analysis. *Environmental Science & Technology*, 47(23), 13385-13394.

Lee, J. W., Choi, H., Hwang, U. K., Kang, J. C., Kang, Y. J., Kim, K. I., & Kim, J. H. (2019). Toxic effects of lead exposure on bioaccumulation, oxidative stress, neurotoxicity, and immune responses in fish: A review. *Environmental Toxicology and Pharmacology*, 68, 101-108.

Lee, J. W., Jo, A. H., Kang, Y. J., Lee, D., Choi, C. Y., Kang, J. C., & Kim, J. H. (2024). Review of cadmium bioaccumulation in fish exposed to cadmium. *Toxics*, 13(1), 7.

Łuczyńska, J., Pietrzak-Fiećko, R., Purkiewicz, A., & Łuczyński, M. J. (2022). Assessment of fish quality based on the content of heavy metals. *International Journal of Environmental Research and Public Health*, 19, 2307. https://doi.org/10.3390/ijerph19042307

Macirella, R., Curcio, V., Ahmed, A. I., Talarico, F., Sesti, S., Paravani, E., ... & Brunelli, E. (2023). Morphological and functional alterations in zebrafish (*Danio rerio*) liver after exposure to two ecologically relevant concentrations of lead. *Fishes*, 8(7), 342.

Masindi, V., & Muedi, K. L. (2018). Environmental contamination by heavy metals. In Heavy Metals. *London, UK: IntechOpen*. https://doi.org/10.5772/intechopen.76082

Mieiro, C. L., Pacheco, M., Pereira, M. E., & Duarte, A. C. (2009). Mercury distribution in key tissues of fish (Liza aurata) inhabiting a contaminated estuary - implications for human and ecosystem health risk assessment. *Journal of Environmental Monitoring*, 11(5), 1004-1012.

Moiseenko, T. I., & Gashkina, N. A. (2020). Distribution and bioaccumulation of heavy metals (Hg, Cd and Pb) in fish: Influence of the aquatic environment and climate. *Environmental Research Letters*, 15(11), 115013.

Monteiro, D. A., Rantin, F. T., & Kalinin, A. L. (2010). Inorganic mercury exposure: toxicological effects, oxidative stress biomarkers and bioaccumulation in the tropical freshwater fish matrinxã, Brycon amazonicus (Spix and Agassiz, 1829). *Ecotoxicology*, 19(1), 105-123.

Monteiro, D. A., Rantin, F. T., & Kalinin, A. L. (2013). Dietary intake of inorganic mercury: bioaccumulation and oxidative stress parameters in the neotropical fish Hoplias malabaricus. *Ecotoxicology*, 22(3), 446-456.

Morales, D. V. (2022). Behavioral and physiological effects of heavy metals on fish: A review and preliminary results (Master's thesis). Hunter College, City University of New York, New York, NY, USA.

Mora-Zamorano, F. X., Klingler, R., Basu, N., Head, J., Murphy, C. A., Binkowski, F. P., ... & Carvan III, M. J. (2017). Developmental methylmercury exposure affects swimming behavior and foraging efficiency of yellow perch (Perca flavescens) larvae. *ACS Omega*, 2(8), 4870-4877.

Mustapha, L. S., Obayomi, O. V., & Obayomi, K. S. (2025). A comprehensive review on potential heavy metals in the environment: Persistence, bioaccumulation, ecotoxicology, and agricultural impacts. *Ecological Frontiers*. https://doi.org/10.1016/j.ecofro.2025.10.009

Mustafa, S. A., Al-Faragi, J. K., Salman, N. M., & Al-Rudainy, A. J. (2017). Histopathological alterations in gills, liver and kidney of common carp, Cyprinus carpio exposed to lead Acetate. *Adv. Anim. Vet. Sci,* 5(9), 371-376.

Naz, S., Chatha, A. M. M., Téllez-Isaías, G., Ullah, S., Ullah, Q., Khan, M. Z., Shah, M. K., Abbas, G., Kiran, A., Mushtaq, R., Ahmad, B., & Kari, Z. A. (2023). A comprehensive review on metallic trace elements toxicity in fishes and potential remedial measures. *Water*, 15(16), 3017. https://doi.org/10.3390/w15163017

Oliveira, J., Oliva-Teles, A., & Couto, A. (2024). Tracking biomarkers for the health and welfare of aquaculture fish. *Fishes*, 9(7), 289.

Oros, A. (2025). Bioaccumulation and trophic transfer of heavy metals in marine fish: Ecological and ecosystem-level impacts. *Journal of Xenobiotics*, 15(2), 59. https://doi.org/10.3390/jox15020059

Oziegbe, O., Oziegbe, E. J., & Ojo-Omoniyi, O. (2025). Bioremediation of heavy metals in aquatic environment: A review. *Cleaner Chemical Engineering*, 11, 100193. https://doi.org/10.1016/j.clce.2025.100193

Patel, K. S., Pandey, P. K., Martín-Ramos, P., Corns, W. T., Varol, S., Bhattacharya, P., & Zhu, Y. (2023). A review on arsenic in the environment: contamination, mobility, sources, and exposure. *RSC Advances*, 13(13), 8803-8821.

Patel, M., Rogers, J. T., Pane, E. F., & Wood, C. M. (2006). Renal responses to acute lead waterborne exposure in the freshwater rainbow trout (Oncorhynchus mykiss). *Aquatic Toxicology*, 80(4), 362-371.

Pargi, N. A., Vasava, R. J., Kalaria, K. K., Tandel, R., & Hariprasad, M. K. (2024). Assessment of heavy metal toxicity in fish and its impact on human health: A review. *International Journal of Advanced Biochemistry Research*, 10(26174693.2024), v8.

Pereira, P., Puga, S., Cardoso, V., Pinto-Ribeiro, F., Raimundo, J., Barata, M., Pousao-Ferreira, P., Pacheco, M., & Almeida, A. (2016). Inorganic mercury accumulation in brain following waterborne exposure elicits a deficit on the number of brain cells and impairs swimming behavior in fish (white seabream - *Diplodus sargus*). *Aquatic Toxicology,* 170, 400-412.

Perera, P. A. C. T., Kodithuwakku, S. P., Sundarabarathy, T. V., & Edirisinghe, U. (2015). Bioaccumulation of cadmium in freshwater fish: An environmental perspective. *Insight Ecology* 4: 1-12.

Pelić, M., Mihaljev, Ž., Živkov Baloš, M., Popov, N., Gavrilović, A., Jug-Dujaković, J., & Ljubojević Pelić, D. (2024). Health risks associated with the concentration of heavy metals in sediment, water, and carp reared in treated wastewater from a slaughterhouse. *Water*, 16, 94. https://doi.org/10.3390/w16010094

Piwowarska, D., Kiedrzyńska, E., Jaszczyszyn, K. (2024). A global perspective on the nature and fate of heavy metals polluting water ecosystems, and their impact and remediation. *Critical Reviews in Environmental Science and Technology*, 54(19), 1436-1458. https://doi.org/10.1080/10643389.2024.2317112

Prosi, F. (1989). Factors controlling biological availability and toxic effects of lead in aquatic organisms. *Science of the Total Environment*, 79(2), 157-169.

Ray, S., & Vashishth, R. (2024). From water to plate: Reviewing the bioaccumulation of heavy metals in fish and unraveling human health risks in the food chain. *Emerging Contaminants*, 10(4), 100358. https://doi.org/10.1016/j.emcon.2024.100358

Rice, K. M., Walker Jr, E. M., Wu, M., Gillette, C., & Blough, E. R. (2014). Environmental mercury and its toxic effects. *Journal of preventive medicine and public health*, 47(2), 74.

Salaah, S. M., El-Khayat, H. M., & Gaber, H. S. (2025). Size-dependent heavy metal and parasite bioaccumulation in Mugil cephalus from lake manzala: human health risks and fish histopathological lesions. *Scientific Reports*, 15(1), 32659.

Saket, P. K., Chaurasiya, A., Saket, R. D., & Datta, R. (2025). Bioaccumulation of Heavy Metals in Fishes. *Asian Journal of Fisheries and Aquatic Research*, 27(12), 143-151.

Sarkar, O., Dey, K. K., Islam, S., & Chattopadhyay, A. (2022). Lead and aquatic ecosystems, biomarkers, and implications for humankind. In Biomarkers in Toxicology (pp. 1-28). *Cham: Springer International Publishing*.

Sevcikova, M., Modrá, H., Slaninova, A., & Svobodova, Z. (2011). Metals as a cause of oxidative stress in fish: A review. *Veterinarni Medicina*, 56(11), 537–546.

Sfakianakis, D. G., Renieri, E., Kentouri, M., & Tsatsakis, A. M. (2015). Effect of heavy metals on fish larvae deformities: A review. *Environmental Research*, 137, 246–255. https://doi.org/10.1016/j.envres.2014.12.014

Shahjahan, M., Taslima, K., Rahman, M. S., Al-Emran, M. D., Alam, S. I., & Faggio, C. (2022). Effects of heavy metals on fish physiology – a review. *Chemosphere*, 300, 134519.

Sharma, A. K., Sharma, M., Sharma, S., Malik, D. S., Sharma, M., Sharma, M., & Sharma, A. K. (2024). A systematic review on assessment of heavy metals

toxicity in freshwater fish species: Current scenario and remedial approaches. *Journal of Geochemical Exploration*, 262, 107472. https://doi.org/10.1016/j.gexplo.2024.107472

Sharma, V. K., & Sohn, M. (2009). Aquatic arsenic: toxicity, speciation, transformations, and remediation. *Environment International*, 35(4), 743-759.

Simatupang, M. M., Veronika, E., Irfandi, A., & Azteria, V. (2024). Potential impacts of lead on health: a review of environmental exposure, population at risk, and toxic effects. *Journal of Environmental Health*, 16(3), 277-288.

Singh, G., Sharma, S. (2024). Heavy metal contamination in fish: Sources, mechanisms and consequences. *Aquatic Sciences*, 86. https://doi.org/10.1007/s00027-024-01121-7.

Sneha, L., Badal, M., Sharma, V., Sharma, A., Kumar, R., & Singh, S. M. (2026). Impact of Pathotoxicity and Bioaccumulation of Lead on Fish Health and Ecosystem Dynamics. *Annals of Animal Science*, 26(1), 15-25.

Sonone, S., Jadhav, S., Singh Sankhla, M., & Kumar, R. (2020). Water contamination by heavy metals and their toxic effect on aquaculture and human health through food chain. *Letters in Applied NanoBioScience*, 10, 2148-2166. https://doi.org/10.33263/LIANBS102.21482166

Strungaru, S. A., Robea, M. A., Plavan, G., Todirascu-Ciornea, E., Ciobica, A., & Nicoara, M. (2018). Acute exposure to methylmercury chloride induces fast changes in swimming performance, cognitive processes and oxidative stress of zebrafish (Danio rerio) as reference model for fish community. *Journal of Trace Elements in Medicine and Biology*, 47, 115-123.

Suedel, B. C., Boraczek, J. A., Peddicord, R. K., Clifford, P. A., & Dillon, T. M. (1994). Trophic transfer and biomagnification potential of contaminants in aquatic ecosystems. *Reviews of Environmental Contamination and Toxicology*, 21-89.

Tang, S., Gao, L., Qin, D., Wang, H., Huang, L., Wu, S., ... & Chen, Z. (2024). Toxic effects of arsenic on four freshwater aquatic species and its transformation metabolism in Crucian carp (Carassius auratus). *Toxics*, 12(3), 221.

Thophon, S., Kruatrachue, M., Upatham, E. S., Pokethitiyook, P., Sahaphong, S., & Jaritkhuan, S. (2003). Histopathological alterations of white seabass, Lates calcarifer, in acute and subchronic cadmium exposure. *Environmental Pollution*, 121(3), 307-320.

Torgerson-White, L., & Sanchez-Suarez, W. (2022). Looking beyond the shoal: Fish welfare as an individual attribute. *Animals*, 12(19), 2592. https://doi.org/10.3390/ani12192592

Tudor, M. S., Lopez-Anido, R. N., Yocius, C. A., Conlin, S. M., & Hamlin, H. J. (2019). Ecologically relevant arsenic exposure alters female mate preference and anxiety-like behavior in Betta splendens. *Heliyon*, 5(10).

Ullrich, S. M., Tanton, T. W., & Abdrashitova, S. A. (2001). Mercury in the aquatic environment: a review of factors affecting methylation. *Critical reviews in environmental science and technology*, 31(3), 241-293.

Ventura-Lima, J., Bogo, M. R., & Monserrat, J. M. (2011). Arsenic toxicity in mammals and aquatic animals: a comparative biochemical approach. *Ecotoxicology and Environmental Safety*, 74(3), 211-218.

Verbost, P. M., Flik, G. E. R. T., Lock, R. A., & Wendelaar Bonga, S. E. (1987). Cadmium inhibition of Ca2+ uptake in rainbow trout gills. American *Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 253(2), R216-R221.

Vieira, L. R., Gravato, C., Soares, A. M. V. M., Morgado, F., & Guilhermino, L. (2009). Acute effects of copper and mercury on the estuarine fish Pomatoschistus microps: linking biomarkers to behaviour. *Chemosphere*, 76(10), 1416-1427.

Wang, N., Ye, Z., Huang, L., Zhang, C., Guo, Y., & Zhang, W. (2022). Arsenic occurrence and cycling in the aquatic environment: a comparison between freshwater and seawater. *Water*, 15(1), 147.

Wang, R., Wong, M. H., & Wang, W. X. (2010). Mercury exposure in the freshwater tilapia Oreochromis niloticus. *Environmental Pollution*, 158(8), 2694-2701.

World Organisation for Animal Health (OIE). (2019). Aquatic Animal Health Code (22nd ed.). Paris, France.

Wu, P., Kainz, M. J., Bravo, A. G., Åkerblom, S., Sonesten, L., & Bishop, K. (2019). The importance of bioconcentration into the pelagic food web base for methylmercury biomagnification: A meta-analysis. *Science of the Total Environment*, 646, 357-367.

Yang, H. C., Fu, H. L., Lin, Y. F., & Rosen, B. P. (2012). Pathways of arsenic uptake and efflux. *In Current topics in membranes* (Vol. 69, pp. 325-358). Academic Press.

Yang, X., Huang, C., Sanjeewa, K. A., Gao, X., Mao, X., & Wang, L. (2025). A comprehensive review of heavy metals in aquatic products and their removal techniques. *Food Control*, 178, 111520.

Zahran, E., Mamdouh, A. Z., Elbahnaswy, S., El-Son, M. M., Risha, E., ElSayed, A., ... & Sebaei, M. G. E. (2025). The impact of heavy metal pollution: bioaccumulation, oxidative stress, and histopathological alterations in fish across diverse habitats. *Aquaculture International*, 33(5), 371.

Zeitoun, M. M., & Mehana, E. S. E. (2014). Impact of water pollution with heavy metals on fish health: Overview and updates. *Global Veterinaria*, 12, 219-231. https://doi.org/10.5829/idosi.gv.2014.12.02.82219

Zhang, W., Miao, A. J., Wang, N. X., Li, C., Sha, J., Jia, J., ... & Ok, Y. S. (2022). Arsenic bioaccumulation and biotransformation in aquatic organisms. *Environment International*, 163, 107221.

Zheng, N. A., Wang, S., Dong, W. U., Hua, X., Li, Y., Song, X., ... & Li, Y. (2019). The toxicological effects of mercury exposure in marine fish. *Bulletin of Environmental Contamination and Toxicology*, 102(5), 714-720.

Zhou, H. Y., & Wong, M. H. (2000). Mercury accumulation in freshwater fish with emphasis on the dietary influence. *Water Research*, 34(17), 4234-4242.

Zulkipli, S. Z., Liew, H. J., Ando, M., Lim, L. S., Wang, M., Sung, Y. Y., & Mok, W. J. (2021). A review of mercury pathological effects on organs specific of fishes. *Environmental Pollutants and Bioavailability*, 33(1), 76-87.