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Review Article

BIOACCUMULATION AND ECOTOXICOLOGICAL EFFECTS OF HEAVY METALS IN EDIBLE FISH SPECIES: IMPLICATIONS FOR FOOD SAFETY AND PUBLIC HEALTH: A REVIEW

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ABSTRACT

Aquatic habitats contaminated by heavy metals have become a serious environmental and public health issue. Because edible fish species can bioaccumulate hazardous elements like lead, cadmium, mercury, and arsenic through trophic transmission, they serve as important bioindicators. The origins, routes, and bioaccumulation patterns of heavy metals in fish are summarized in this review, which focuses on the ecotoxicological effects of these metals on physiological, biochemical, and histological parameters. Long-term exposure to these metals damages fish's brain and reproductive systems, interferes with metabolic processes, and causes oxidative stress. Additionally, eating tainted fish puts people at serious risk for both carcinogenic and non-carcinogenic health impacts. The evaluation also emphasizes risk assessment techniques, such as the bioaccumulation factor and hazard quotient, to identify possible dangers. To guarantee food safety and safeguard public health, it is crucial to comprehend these relationships in order to create efficient monitoring plans, legal regulations, and sustainable practices.

Keywords: Ecotoxicology, Food safety, Public health, Bioaccumulation, Edible fish, Heavy metals.

INTRODUCTION

Heavy metal pollution in aquatic habitats has become a global environmental concern due to heavy metals' persistence, non-biodegradability, and adverse effects on living organisms. Anthropogenic factors such as industrialization, urbanization, agricultural runoff, and residential waste discharge are the main causes of elevated levels of heavy metals in aquatic environments (Emon *et al.,* 2023; Zahran *et al.,* 2025). Heavy metals can infiltrate the aquatic food chain and pose major risks to human health and the environment when they accumulate in sediments and biota after being added to water bodies. Fish's capacity to bioaccumulate heavy metals from water, sediments, and food sources makes them significant bioindicators of aquatic pollution. Toxic metals like lead (Pb), cadmium (Cd), mercury (Hg), and arsenic (As) gradually build up in fish tissues as a result of the bioaccumulation process (Korkmaz *et al.,* 2017; Ahmad *et al.,* 2022). Both necessary and non-essential roles are played by these metals, but excessive accumulation interferes with physiological and metabolic processes and can have serious toxicological effects (Varol & Sünbül, 2019; Emon *et al.,* 2023). Heavy metals have ecotoxicological consequences on fish that include cellular damage, oxidative stress, enzyme inhibition, and histopathological changes in critical organs such the kidney, liver, and gills.

One important mechanism behind metal-induced toxicity is the production of reactive oxygen species (ROS), which causes DNA damage, protein degradation, and lipid peroxidation (Zahran *et al.,* 2025). Fish population growth, reproduction, and survival are eventually hampered by these changes, which has an impact on aquatic biodiversity and environmental stability. Additionally, eating tainted fish is a significant way that people are exposed to heavy metals. Serious health problems, such as neurotoxicity, carcinogenicity, nephrotoxicity, and developmental abnormalities, can arise from long-term exposure through diet. Heavy metals are more dangerous for top consumers, including humans, because of their capacity to biomagnify

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along trophic levels (Javed & Usmani, 2016; Jiang *et al.,* 2016). In order to guarantee food safety and safeguard public health, it is essential to evaluate heavy metal contamination and bioaccumulation in edible fish. In this regard, the current review seeks to give a thorough summary of the bioaccumulation of heavy metals in edible fish species, their ecotoxicological effects, and the threats they pose to human health. In order to lessen the negative consequences of heavy metal contamination in aquatic environments, it also highlights the significance of monitoring plans and risk assessment techniques.

MATERIALS AND METHODS

Study Design and Reporting Framework

To guarantee transparency, repeatability, and methodological rigor, this evaluation was carried out in accordance with PRISMA principles. The review methodology was created to methodically find, filter, and compile pertinent material on the ecotoxicological effects and bioaccumulation of heavy metals in edible fish species.

Literature Search Strategy

Major electronic databases, such as PubMed Central, SpringerLink, and MDPI, were searched thoroughly for relevant material. To guarantee more thorough coverage, additional resources like Scopus and Google Scholar were also utilized. Combinations of "heavy metals," "bioaccumulation," "edible fish," "ecotoxicology," "risk assessment," "food safety," and "public health" were among the search terms. The search was narrowed down using boolean operators (AND, OR). Only English-language papers published between 2010 and 2025 were included in the search.

Study Selection Process

Duplicate articles were eliminated once all retrieved articles were integrated into a reference management system. Three steps comprised the selection process: (1) title screening, (2) abstract screening, and (3) full-text evaluation. The studies were assessed by two separate reviewers, and disagreements were settled by discussion. A PRISMA flow diagram was used to record the selecting procedure.

Data Extraction and Synthesis

A systematic data collection form was used to capture pertinent information, such as the author or authors, year of publication, study location, fish species, types of heavy metals examined (such as Pb, Cd, Hg, and As), concentration levels, bioaccumulation factors, and reported toxicological effects. To find trends, patterns, and research gaps, data were qualitatively synthesized. Results were compared between species and geographical areas when appropriate.

Quality Assessment

Sampling strategy, analytical methods, statistical analysis, and reporting clarity were among the criteria used to assess the methodological quality of the included research. Higher weight was given in the synthesis to studies with reliable experimental designs and validated analytical techniques (such as AAS and ICP-MS).

Risk Assessment Approach

Commonly reported metrics, such as Estimated Daily Intake (EDI), Target Hazard Quotient (THQ), Hazard Index (HI), and Carcinogenic danger (CR), were used to evaluate the danger to human health linked with heavy metal exposure from fish eating. To assess possible non-carcinogenic and carcinogenic risks, these indicators were interpreted in accordance with international safety norms.

Table 1. Data Extraction on Heavy Metal Bioaccumulation in Edible Fish.

S. No.	Author(s), Year	Study Location	Fish Species	Heavy Metals Analyzed	Sample Type	Analytical Method	Bioaccumulation Factor (BAF)	Key Ecotoxicological Findings	Human Health Risk Indicators (THQ/HI/CR)	Remarks
1	Emon *et al*., 2023	Bangladesh	*Oreochromis niloticus*	Pb, Cd, Hg, As	Muscle, Liver	ICP-MS	High (Hg > Cd > Pb)	Oxidative stress, liver damage	THQ > 1 (Hg)	High consumer risk
2	Varol & Sünbül, 2019	Turkey	*Cyprinus carpio*	Pb, Cd, Cr, Zn	Muscle	AAS	Moderate	Enzyme inhibition, tissue damage	HI > 1	Chronic exposure risk
3	Zahran *et al*., 2025	Egypt	*Tilapia zillii*	Pb, Cd, Cu	Liver, Gills	ICP-OES	High	Histopathological alterations, ROS generation	THQ > 1	Environmental pollution indicator
4	Ahmad *et al*., 2022	India	*Labeo rohita*	Pb, Cd, Hg	Muscle	AAS	Moderate-High	Neurotoxicity, reduced growth	HI > 1	Food safety concern

5	Javed & Usmani, 2016	India	*Channa punctatus*	Fe, Zn, Pb, Cd	Whole body	AAS	Variable	Biochemical alterations	THQ < 1 (except Pb)	Localized contamination
6	Jiang *et al*., 2016	China	*Ctenopharyngodon idella*	Hg, As	Muscle	ICP-MS	High (Hg dominant)	DNA damage, oxidative stress	CR > acceptable limit	Carcinogenic risk
7	Korkmaz *et al*., 2017	Turkey	Multiple species	Pb, Cd, Ni	Muscle	AAS	Moderate	Gill and liver damage	THQ > 1	Multi-metal exposure
8	MDPI Foods, 2025	Danube River	Mixed species	Pb, Cd, Hg, As	Muscle	ICP-MS	High	Metabolic disruption	HI > 1	Public health concern

Heavy Metals Influencing Toxicity in Edible Fishes

Fish are poisoned by heavy metals, which are persistent environmental pollutants that cause oxidative stress, bioaccumulation, and physiological system disturbance. Fish species, exposure length, metal type, and concentration all affect how hazardous they are (Emon *et al.,* 2023; Zahran *et al.,* 2025). Metals can accumulate over time in tissues and cause toxicological reactions in aquatic systems when they are absorbed through gills, consumed through contaminated food, or absorbed through epithelial surfaces.

Lead (Pb) Toxicity

Lead is a very hazardous and non-essential metal that disrupts fish neurological and enzymatic processes. It mostly builds up in the tissues of the gills, liver, and kidneys, where it causes hematological changes, behavioral abnormalities, and stunted growth. Exposure to lead promotes oxidative stress by increasing the formation of reactive oxygen species (ROS) and interfering with ion control (Tolkou *et al.,* 2023; Zahran *et al.,* 2025). Lead impedes heme production and oxygen transport at the biochemical level by inhibiting important enzymes including δ-aminolevulinic acid dehydratase (ALAD). Additionally, it causes ionic imbalance and osmoregulatory failure by interfering with Na+/K+-ATPase function in gill membranes. Hepatic necrosis, epithelial lifting, and gill lamellar fusion are examples of histopathological findings. In fish populations, long-term exposure causes neurotoxicity, decreased eating behavior, and poor reproductive outcomes.

Cadmium (Cd) Toxicity

Due to its extreme toxicity even at low concentrations, cadmium is one of the most dangerous heavy metals. It accumulates in tissues like the liver and kidneys after entering fish through food and water. According to Lee *et al.,* (2025), exposure to cadmium results in severe oxidative stress, circulatory malfunction, and enzyme inhibition, which eventually induce tissue deterioration and death. Antioxidant enzymes like superoxide dismutase (SOD) and catalase (CAT) are rendered inactive by cadmium's strong affinity for sulfhydryl (-SH) groups. Energy depletion is also caused by lipid peroxidation and mitochondrial malfunction. By competing with Ca2+ ions, Cd interferes with calcium metabolism, leading to skeletal abnormalities and decreased muscular contraction. Fish survival is greatly impacted because it causes vacuolar degeneration in hepatocytes and tubular degeneration in kidneys.

Mercury (Hg) Toxicity

Mercury is extremely bioaccumulative and biomagnifies up the food chain, especially in its organic form (methylmercury). It mainly affects the neurological system, resulting in neurotoxicity, changed swimming patterns, and decreased fish reproductive success. Fish survival and ecosystem health are greatly impacted by mercury exposure, which causes oxidative stress and DNA damage (Naz *et al.,* 2023; Zahran *et al.,* 2025). Methylmercury easily penetrates biological membranes and attaches itself to protein thiol groups to interfere with neural signaling. It impairs coordination and causes sensory dysfunction by interfering with neurotransmitter systems, especially the acetylcholine and dopamine pathways. Mercury causes cellular death by damaging mitochondria and activating caspase pathways. Long-term exposure also has an impact on the development of the embryo, resulting in abnormalities and decreased hatchability.

Arsenic (As) Toxicity

Fish metabolism and cellular function are severely harmed by the metalloid arsenic. Long-term exposure causes oxidative damage, energy metabolism disturbance, and carcinogenic consequences. It builds up in tissues like muscle and liver, which puts fish and human consumers at grave risk (Tolkou *et al.,* 2023). By preventing oxidative phosphorylation, arsenic hinders the synthesis of ATP and lowers the amount of energy available to cells. Additionally, it causes chromosomal abnormalities and breaks in DNA strands, which results in genotoxicity. Fish exposed to arsenic have altered endocrine processes, poor glucose metabolism, and aberrant enzyme activity. Hepatocellular degeneration, damage to the gill epithelium, and increased mucus secretion are examples of histopathological consequences that hinder breathing.

Chromium (Cr) Toxicity

There are two types of chromium: poisonous (Cr VI) and essential (Cr III), with hexavalent chromium being extremely toxic. In fish, it results in oxidative stress, enzyme inhibition, and DNA damage. Exposure affects respiration and metabolic activities by causing histological abnormalities in the liver and gills (Naz *et al.,* 2023). Via

sulfate transport channels, Cr (VI) readily crosses cell membranes and is converted to Cr (III), producing ROS and reactive intermediates. Lipids, proteins, and nucleic acids are severely oxidatively damaged by this redox cycle. Exposure to chromium also lowers erythrocyte counts and hemoglobin levels, among other hematological parameters. Reduced development rates and anatomical abnormalities are the outcomes of chronic poisoning.

Copper (Cu) Toxicity

Although copper is a necessary trace element, high amounts of it can be hazardous. Overexposure to Cu causes oxidative stress, ion balance disruption, and gill tissue destruction. It eventually hinders fish development and survival by interfering with osmoregulation and enzymatic activity (Emon *et al.,* 2023). Gill function is mainly impacted by copper poisoning because it damages epithelial cells and lowers the efficiency of oxygen uptake. Additionally, it causes oxidative stress by catalyzing the production of hydroxyl radicals through Fenton-like processes. Increased Cu levels interfere with metabolic enzymes like cytochrome oxidase and cause problems with protein synthesis. It is typical to see behavioral changes including irregular swimming and decreased feeding.

Zinc (Zn) Toxicity

Another necessary element for enzymatic processes is zinc, but excessive amounts might be hazardous. Zn poisoning causes histological damage in fish tissues, metabolic problems, and decreased reproduction. Protein synthesis and antioxidant defense systems are also impacted (Naz *et al.,* 2023). Ionic imbalance results from excess zinc interfering with the absorption of other vital ions like calcium and iron. Additionally, it causes oxidative stress by changing the activity of antioxidant enzymes. Histological alterations include kidney destruction, liver vacuolation, and gill hyperplasia. Extended exposure impairs gonadal development, which lowers fertility and the success of reproduction.

Nickel (Ni) Toxicity

Fish exposed to nickel experience decreased growth, oxidative stress, and metabolic system disruption. Enzyme systems are impacted, and important organs including the liver and kidneys may sustain histological damage. Immune responses may also be compromised by prolonged exposure (Emon *et al.,* 2023). Changes in metabolic activity result from nickel's interference with metal-binding proteins and enzymes. By raising lipid peroxidation and lowering antioxidant defenses, it causes oxidative stress. Hematological characteristics are also impacted by Ni exposure, which can result in anemia and decreased oxygen transport capacity. Fish are more vulnerable to illnesses when their immune systems are weakened by prolonged exposure.

General Mechanisms of Heavy Metal Toxicity

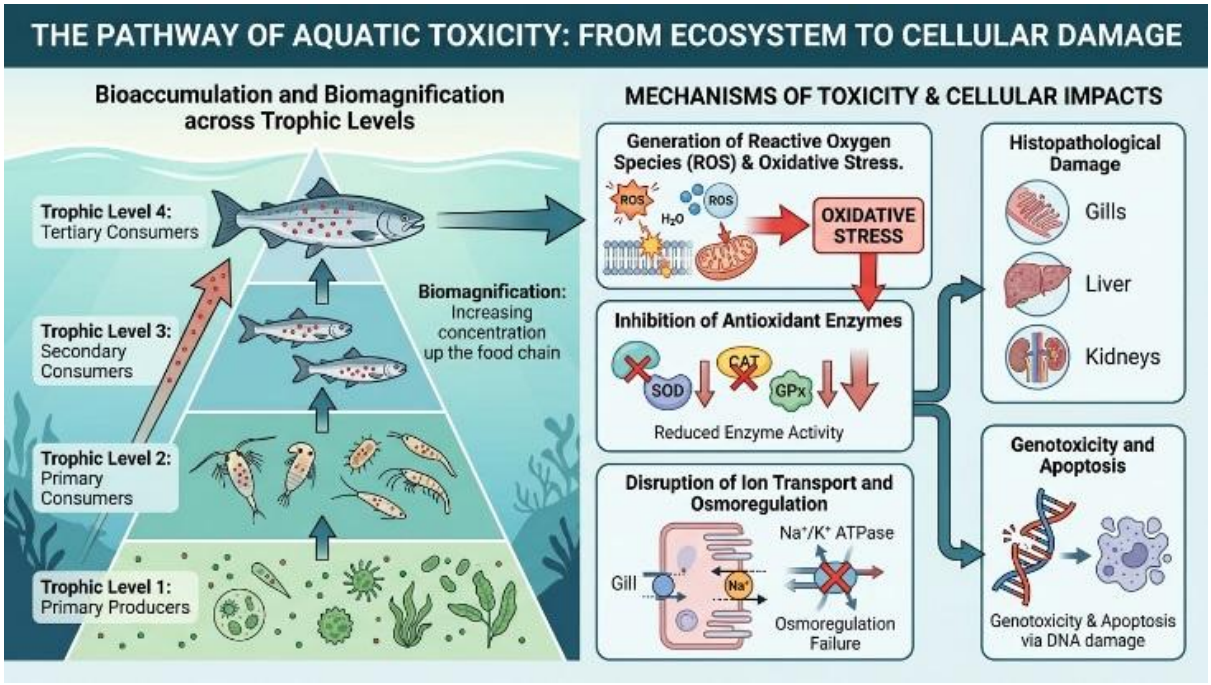

Figure 2. Aquatic toxicity due to cellular damage of fishes.

Bioaccumulation and Biomagnification across Trophic Levels

Aquatic ecosystems are exposed to heavy metals like cadmium (Cd), lead (Pb), mercury (Hg), and chromium (Cr) through air deposition, agricultural runoff, and industrial discharge. These metals tend to build up in fish tissues including muscle, gills, and liver because they are not biodegradable. Bioaccumulation happens within individual species over time, whereas biomagnification raises concentrations at higher trophic levels, endangering people and predators. Aquatic creatures experience long-term toxicity and physiological disruptions as a result of chronic exposure (Zhang *et al.,* 2024; Galli *et al.,* 2005). Research has demonstrated that prolonged exposure raises tissue concentrations, which affects fish metabolism and reproduction (Fatima *et al.,* 2015).

Generation of Reactive Oxygen Species (ROS) and Oxidative Stress

Reactive oxygen species (ROS) such superoxide radicals, hydrogen peroxide, and hydroxyl radicals are produced in excess when heavy metals are present. Redox cycling, electron transport chain disruption, and mitochondrial malfunction all produce these ROS. Oxidative stress, which results in lipid peroxidation, protein oxidation, and DNA damage, happens when ROS generation surpasses antioxidant defense capacity. After being exposed to pollutants, fish tissues such as the liver, brain, kidney, and gills have been shown to produce ROS (Gomes *et al.,* 2022; Zhang *et al.,* 2024). Heavy metal poisoning and cellular dysfunction are mostly caused by this oxidative imbalance (Galli *et al*., 2005).

Inhibition of Antioxidant Enzymes (SOD, CAT, GPx)

Fish have an antioxidant defense system that comprises ROS-neutralizing enzymes such glutathione peroxidase (GPx), catalase (CAT), and superoxide dismutase (SOD). Depending on the dosage and duration, exposure to heavy metals can either first stimulate or eventually inhibit these enzymes. Long-term exposure frequently weakens cellular defense and increases oxidative damage by suppressing the activity of antioxidant enzymes. Cadmium exposure, for example, has been demonstrated to disrupt the antioxidant system by downregulating the expression of the genes SOD, CAT, and GPx (Liu *et al.,* 2023; Shaw *et al.,* 2022). Similar to this, extended exposure to pollutants lowers enzymatic efficiency, which causes tissue damage and oxidative stress (Massarsky *et al.,* 2023).

Disruption of Ion Transport and Osmoregulation

Fish ion transport systems, especially those across gill membranes, which are essential for osmoregulation, are disrupted by heavy metals. Ionic imbalance and compromised osmoregulatory function result from metals like Cd and Pb interfering with calcium channels and Na+/K+-ATPase action. Physiological stress and decreased survival result from this disturbance of the water and electrolyte balance. Hypoxia brought on by toxicant-induced gill injury exacerbates metabolic problems and the generation of ROS (Gomes *et al.,* 2022). Fish respiration, acid-base balance, and general homeostasis are ultimately impacted by impaired ion regulation.

Histopathological Damage in Gills, Liver, and Kidneys

Heavy metal toxicity in fish is mostly shown by histopathological changes. The gills show structural damage, including necrosis, lamellar fusion, and epithelial lifting, as the main site of exposure. Hepatocellular degeneration, vacuolation, and necrosis are shown in the liver, a key organ involved in detoxification; tubular degeneration and glomerular damage are seen in the kidneys. The physiological processes of breathing, detoxification, and excretion are all hampered by these tissue changes. According to Zhang *et al.,* (2024) and Fatima *et al.,* (2015), chronic exposure to heavy metals has been strongly linked to severe histopathological abnormalities in several organs, which show cumulative toxic effects.

Genotoxicity and Apoptosis via DNA Damage

Through direct or indirect interactions with DNA through the production of ROS, heavy metals have genotoxic consequences. Chromosome abnormalities, mutations, and breaks in DNA strands are all caused by oxidative stress. Fish exposed to metals like chromium and cadmium undergo programmed cell death due to the activation of apoptotic pathways including p53, Bax, and caspases. Following metal exposure, experimental research has shown elevated DNA damage indices and upregulated genes linked to apoptosis (Zhang *et al.,* 2024). In aquatic environments, these molecular changes have an impact on population stability, growth, and reproduction in addition to cell survival. Together, these processes limit fish population growth, reproduction, immunological suppression, and survival, which eventually impacts the stability of aquatic ecosystems and food safety (Zahran *et al.,* 2025).

Ecotoxicity and Its Implications for Food Safety and Public Health

The term "ecotoxicity" describes how chemical pollutants, especially heavy metals and persistent organic contaminants, negatively impact ecosystems and living things, which directly affects human health and food safety. Toxic elements including cadmium (Cd), lead (Pb), mercury (Hg), and arsenic (As) build up in water bodies and sediments in aquatic habitats and eventually find their way into the food chain through aquatic creatures like fish and shellfish. These pollutants go through bioaccumulation and biomagnification, which raises their concentrations in edible tissues and puts human consumers at serious risk (Zahran *et al.,* 2025). Chronic toxicity from ongoing exposure to tainted food can damage important organs and raise the risk of neurological, renal, and cardiovascular conditions (Galli *et al.,* 2005).

Inducing oxidative stress through excessive production of reactive oxygen species (ROS) is one of the main mechanisms behind ecotoxicity. In aquatic organisms, heavy metals cause DNA damage, protein oxidation, and lipid peroxidation by upsetting the cellular redox balance. This oxidative damage lowers the nutritional value and safety of seafood meant for human consumption in addition to having an impact on fish health and survival (Gomes *et al.,* 2022; Zhang *et al.,* 2024). Furthermore, oxidative stress has been connected to the production of carcinogenic chemicals and hazardous metabolites, which increase the hazards to the public's health when such contaminated fish are eaten for extended periods of time (Massarsky *et al.,* 2023). Additionally, aquatic animals' physiological and biochemical functions, such as enzyme activity, metabolism, and immunological responses, are disrupted by ecotoxic contaminants. Key antioxidant enzymes like glutathione peroxidase (GPx), catalase (CAT) and superoxide dismutase (SOD) are inhibited, weakening the organism's defenses and leaving it more vulnerable to illnesses and environmental stressors (Liu *et al.,* 2023). Furthermore, detoxifying and excretory processes are hampered by histopathological changes in critical organs including the liver, gills, and kidneys, which increases the buildup of harmful compounds in edible tissues (Zahran *et al.,* 2025). In addition to endangering fish health, these pathological alterations have significance for food safety monitoring since they are biomarkers of environmental contamination.

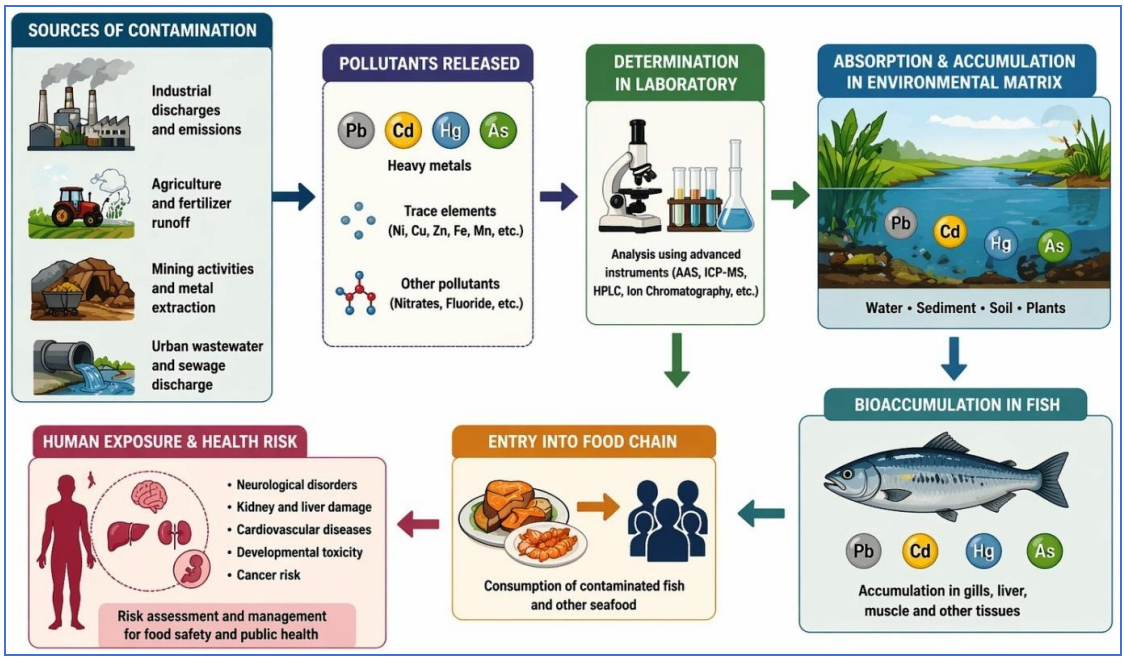

Figure 3. Implications for Food Safety and Public Health.

From the standpoint of public health, eating tainted fish and aquatic products is one of the main ways that people are exposed to pollutants in the environment. Long-term exposure to heavy metals has been linked to developmental defects, endocrine disruption, genotoxicity, and an increased risk of cancer. Because these pollutants are bioaccumulative, vulnerable groups such as children and pregnant women—are especially at danger (Shaw *et al.,* 2022). Additionally, by lowering fish population health, biodiversity, and aquatic ecosystem productivity, ecotoxicological contamination might compromise food security by impacting the availability of wholesome and safe food supplies. To sum up, ecotoxicity is a key factor in the relationship between environmental contamination, food safety and public health outcomes. In addition to having an impact on the health of organisms, the presence of hazardous pollutants in aquatic ecosystems presents long-term dangers to humans through dietary exposure. To reduce ecotoxicological risks and guarantee the security of food systems, it is crucial to create stringent regulatory frameworks, adopt sustainable practices, and continuously monitor environmental pollutants (Zahran *et al.,* 2025).

Histopathological Examinations of Ecotoxicity-Impacted Fishes

An essential method in ecotoxicology for assessing the biological effects of environmental pollutants on fish is histopathological evaluation. It offers concrete proof of tissue-level harm brought on by contaminants including pesticides, heavy metals, and industrial effluents. Fish exposed to contaminated aquatic environments show a variety of cellular and structural changes that are indicative of both acute and long-term toxic effects. These alterations are commonly acknowledged as sensitive biomarkers for

ecological risk assessment and environmental monitoring (Bernet *et al.,* 1999; Hinton & Laurén, 1990). Because of their constant contact with the surrounding water, the gills are among the most vulnerable organs to ecotoxic stress. Epithelial hyperplasia, lamellar fusion, edema, aneurysm, and necrosis are among the histopathological changes frequently seen in gills. Fish survival is ultimately impacted by these modifications, which lower respiratory efficiency and compromise ionic control. It has been demonstrated that exposure to toxicants including organic pollutants and heavy metals can seriously alter the morphology of gill tissues, indicating impaired physiological function (Mallatt, 1985; Camargo & Martinez, 2007). Reduced oxygen intake and elevated metabolic stress in afflicted fish are frequently linked to such structural damage.

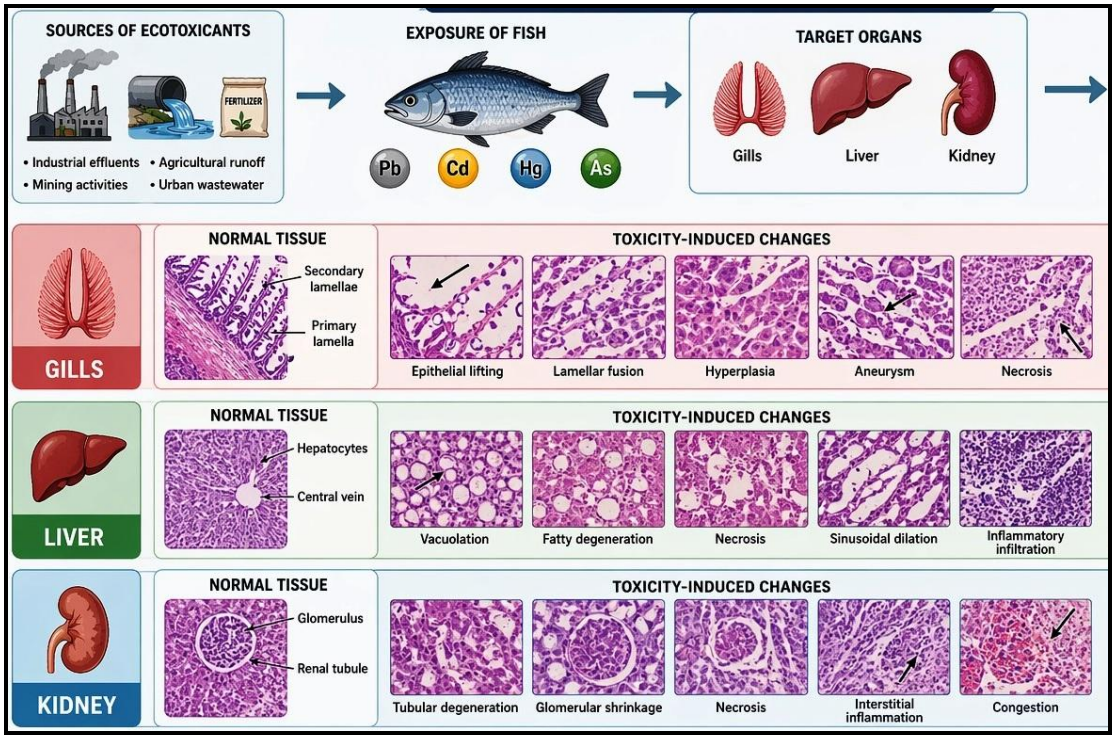

Figure 4. Histopathological Changes in Fish Exposed to Environmental Pollutants.

Because it is vital for detoxification, metabolism, and storage, the liver is a prime target for toxic attacks. Histopathological changes in the liver include nuclear abnormalities, necrosis, fibrosis, fatty degeneration, and hepatocyte vacuolation. These alterations point to disturbances in metabolic and detoxifying processes. Long-term pollution exposure can cause growing liver damage, which can affect enzymatic activity and overall physiological balance. According to research, liver histopathology is a useful biomarker of stress brought on by pollutants and can reveal sublethal effects of environmental contaminants (Wolf & Wolfe, 2005; Figueiredo-Fernandes *et al.,* 2007).

Because kidney tissues are involved in excretion and osmoregulation, they are also extremely vulnerable to ecotoxic injury. Renal tubule degeneration, glomerular shrinkage, interstitial inflammation, and necrosis are common histopathological findings in kidneys. These changes cause systemic toxicity by reducing filtering efficiency and upsetting fluid and electrolyte balance. Environmental contaminants, particularly heavy metals, have been demonstrated to cause significant kidney damage in fish (Takashima & Hibiya, 1995; Thophon *et al*., 2003). Fish survival and health are jeopardized as a result. Histopathological alterations at the cellular level are intimately related to molecular and metabolic processes including apoptosis and oxidative stress.

Reactive oxygen species (ROS) are produced by pollutants and cause DNA damage, protein denaturation, and lipid peroxidation. Cell death and tissue deterioration are the ultimate outcomes of these processes. The degree of pollutant exposure and length are frequently correlated with the severity of histological changes, underscoring the significance of combining histopathology with biochemical indicators for thorough ecotoxicological evaluation (Van der Oost *et al.,* 2003). To sum up, histopathology analyses offer vital information about the detrimental impacts of ecotoxic chemicals on fish health. Changes in the structure of the kidneys, liver, and gills are trustworthy markers of biological stress and environmental pollution. These results highlight the significance of histology investigations in environmental monitoring programs and its applicability in guaranteeing the health of aquatic ecosystems and food safety.

CONCLUSION

Heavy metals bioaccumulate in edible fish, increasing their concentration down the food chain and endangering consumers' health. These metals cause oxidative stress, organ damage, and physiological dysfunction in fish, among other ecotoxicological effects. A significant route of human exposure to contaminated seafood can raise the risk of toxicity and chronic illnesses. To guarantee seafood safety, stringent environmental restrictions and routine heavy metal level monitoring are crucial. In general, maintaining ecosystem health, food security, and public health all depend on reducing water pollution.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

Not applicable

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AI TOOL DECLARATION

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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