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Interactive Toxicity of Agrochemicals and Heavy Metals in Edible Fish

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Abstract

Aquatic ecosystems are increasingly burdened by multiple pollutants, particularly pesticides and heavy metals, entering water bodies through agricultural runoff, industrial effluents, and urban discharge. Edible fish species inhabit these contaminated environments, exposing them to complex chemical mixtures rather than single agents. While the toxic effects of individual pesticides and metals are well documented, interactions between these contaminants can produce synergistic effects—where combined exposures result in exacerbated toxicity greater than the sum of individual effects. Such interactions elevate oxidative stress, impair physiological processes, disrupt neuroendocrine function, and lead to cumulative bioaccumulation, posing serious threats to fish health and food safety. Synergism arises due to alterations in metabolic detoxification pathways, modulation of antioxidant defenses, and enhanced cellular uptake. These combined effects have significant ecological implications, including reduced growth, reproductive impairment, immune suppression, and increased mortality in fish populations. Furthermore, humans consuming contaminated fish face elevated risks due to enhanced residue loads and metabolite interactions. This review synthesizes recent research on pesticide–metal synergistic toxicity in edible fish, focusing on mechanisms, physiological and biochemical endpoints, species-specific responses, and contributing environmental factors. The review also addresses current challenges in mixture toxicity assessment, identifies knowledge gaps, and discusses future directions such as omics-based analyses, predictive modeling, and improved risk assessment frameworks. Comprehensive understanding of synergistic toxicity is vital for shaping regulatory strategies, improving aquatic health monitoring, and safeguarding human nutrition in polluted environments.

Keywords: *Pesticide toxicity, Heavy metals, Synergistic effects, Edible fish, Oxidative stress, Bioaccumulation, Neurotoxicity, Immune dysfunction, Aquatic pollution, Ecological risk assessment*

Introduction

1. Pollution in Aquatic Ecosystems

Freshwater and marine ecosystems worldwide are subject to increasing chemical pollution from diverse anthropogenic activities. Agriculture contributes significantly to the introduction of pesticides—organophosphates, carbamates, pyrethroids, and herbicides—into surface and groundwater through runoff and leaching. Concurrently, industrial processes, mining, and urban waste discharge heavy metals such as cadmium (Cd), lead (Pb), mercury (Hg), chromium (Cr), and arsenic (As), which persist in the environment due to their non-degradable nature. Fish represent integral components of aquatic food webs and serve as vital protein sources for human populations. Because they are exposed to contaminated water throughout their life cycles, fish serve as sentinel organisms in ecotoxicological studies. Traditionally, toxicological research has focused on the effects of individual contaminants. However, environmental reality involves *mixtures* of chemicals, leading to interactions that can alter toxicity profiles.

2. Importance of Synergistic Interactions

When organisms are simultaneously exposed to pesticides and metal contaminants, the combined effects may not simply be additive. Instead, chemicals can interact to produce *synergistic* toxicity, where the observed effect is significantly greater than predicted by summing individual effects. Synergistic interactions complicate risk assessment and management because they can lead to unexpected biological responses at lower concentrations than regulatory thresholds. Recognizing mixture interactions is essential for accurate ecological risk evaluations and establishing effective environmental regulations. Edible fish exposed to pollutant mixtures may undergo physiological disruptions that compromise survival, growth, and reproduction, with cascading impacts on ecosystem health and fisheries productivity.

3. Mechanisms Underlying Synergy

Synergistic toxicity often results from shared or interacting mechanisms at molecular and cellular levels. Pesticides may impair detoxification pathways shared with metals, leading to increased bioavailability of toxic species.

Both classes of contaminants can generate reactive oxygen species (ROS), overwhelm antioxidant defenses, and induce oxidative damage to lipids, proteins, and DNA. Synergistic effects can also disrupt ion regulation, mitochondrial function, and neurotransmission, resulting in metabolic dysfunction and neurotoxic outcomes. Moreover, metals may modulate the metabolism of pesticides, altering their breakdown and persistence within biological tissues. Conversely, pesticides may affect metal uptake by influencing membrane transporters. The resulting interactions can amplify toxic responses beyond expectations based on single exposures.

4. Impact on Fish Physiology and Health

In addition to oxidative stress and enzyme impairment, synergistic exposure to pesticides and heavy metals can disrupt energy metabolism, mitochondrial function, and ion regulation in fish. Liver and kidney tissues often show histopathological alterations, including necrosis, vacuolation, and inflammation. Chronic exposure can alter hematological parameters, reduce hemoglobin levels, and impair oxygen transport. Neurotoxic effects may include altered swimming behavior, predator avoidance, and learning deficits, while endocrine disruption can disturb hormone balance, leading to delayed sexual maturation and lower spawning success. Overall, combined pesticide–metal toxicity compromises fish health, survival, and population sustainability, with implications for aquatic ecosystems and human consumption.

5. Challenges in Risk Assessment

Current regulatory frameworks largely rely on single-chemical toxicity data. Assessing mixture toxicity requires more sophisticated approaches, including whole-organism bioassays, biochemical biomarkers, and advanced modeling techniques. Spatial and temporal variations in contaminant profiles further complicate assessments. Thus, understanding the influence of environmental factors such as pH, temperature, and water hardness on synergistic interactions is crucial.

6. Scope and Objectives

This review aims to consolidate existing research on pesticide–metal synergistic toxicity in edible fish, focusing on mechanisms, biological impacts, contributing factors, and risk implications. It also highlights gaps in knowledge and emerging methodologies that can enhance risk prediction and guide environmental policy.

Sources and Co-Occurrence of Pesticides and Metals in Aquatic Systems

Agricultural Runoff and Pesticide Entry

Agricultural activities are major contributors to pesticide contamination in aquatic ecosystems. Pesticides, including organophosphates, carbamates, pyrethroids, and herbicides, are applied extensively to control pests and enhance crop productivity. Rainfall and irrigation often wash these chemicals from fields into nearby rivers, streams, and ponds, a process known as surface runoff. Additionally, pesticides can infiltrate soil and percolate into groundwater, contaminating aquifers used for drinking water and aquaculture. Residues from repeated pesticide application can persist in sediments and bioaccumulate in aquatic organisms. Seasonal variations in rainfall and farming practices can influence the magnitude of pesticide influx into water bodies, creating fluctuating exposure scenarios for fish.

Industrial and Urban Metal Pollution

Industrial effluents are a significant source of heavy metals in aquatic environments. Mining operations, battery production, electroplating, and metal fabrication discharge metals such as lead (Pb), cadmium (Cd), mercury (Hg), chromium (Cr), and arsenic (As) into water bodies. Urban areas contribute through stormwater runoff, sewage discharge, and untreated wastewater, which often carry a mixture of metals from infrastructure corrosion, domestic products, and industrial activities. Unlike organic pesticides, heavy metals are non-degradable and can persist in sediments and the aquatic food chain, resulting in long-term exposure to aquatic organisms, including edible fish species.

Co-Occurrence Patterns

Recent monitoring studies indicate that pesticides and heavy metals frequently co-occur in freshwater and estuarine systems. Agricultural runoff often carries both agrochemicals and metal residues from fertilizers or nearby industrial areas, leading to multi-contaminant scenarios. Fish inhabiting these waters are simultaneously exposed to both chemical classes, resulting in complex toxic interactions. Such co-exposure can exacerbate physiological stress, enhance bioaccumulation, and amplify ecological and human health risks. Understanding these co-occurrence patterns is crucial for risk assessment and developing effective pollution mitigation strategies.

Mechanisms of Synergistic Toxicity

Oxidative Stress Pathways

A central mechanism underlying the synergistic toxicity of pesticides and heavy metals in fish is the induction of oxidative stress. Both classes of contaminants promote the excessive generation of reactive oxygen species (ROS), including superoxide radicals, hydrogen peroxide, and hydroxyl radicals. Individually, fish possess enzymatic and non-enzymatic antioxidant defenses, such as superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH), to neutralize ROS. However, simultaneous exposure to pesticide–metal mixtures can overwhelm these protective mechanisms. This imbalance leads to lipid peroxidation, protein oxidation, DNA damage, and mitochondrial dysfunction, resulting in impaired cellular function, apoptosis, and tissue degeneration. Oxidative stress is particularly pronounced in metabolically active organs like the liver, gills, and kidneys, which are central to detoxification and nutrient assimilation.

Impairment of Detoxification and Metabolic Enzymes

Pesticides and metals interfere with enzymatic pathways responsible for detoxification and metabolic homeostasis. Key detoxification enzymes, including glutathione S-transferase (GST), catalase (CAT), and cytochrome P450 isoforms, can be inhibited or downregulated under co-exposure conditions. Such inhibition reduces the biotransformation and clearance of contaminants, increasing their bioaccumulation in tissues. Furthermore, metabolic enzymes involved in energy production, lipid metabolism, and protein synthesis may be disrupted, compromising growth, immune response, and overall physiological resilience. Chronic interference in these pathways exacerbates tissue damage and mortality in exposed fish populations.

Neurotoxicity and Behavioral Effects

Neurotoxic effects constitute another critical outcome of synergistic pesticide–metal exposure. Organophosphate and carbamate pesticides inhibit acetylcholinesterase (AChE), causing acetylcholine accumulation at synapses, while metals like mercury (Hg) and lead (Pb) disrupt neurotransmission, impair ion channel function, and alter synaptic plasticity. Co-exposure can amplify these effects, leading to pronounced behavioral abnormalities, such as reduced swimming activity, impaired predator avoidance, erratic schooling, and compromised foraging efficiency. These neurobehavioral impairments reduce survival fitness, reproduction, and population stability, with cascading ecological consequences.

Physiological and Biochemical Effects in Fish

Growth and Development

Synergistic exposure to pesticide–metal mixtures significantly impairs growth and development in fish. Reduced feed assimilation and altered nutrient metabolism are common outcomes, as contaminants interfere with digestive enzyme activity and energy allocation. Juvenile fish are particularly vulnerable, showing stunted growth, delayed development, and lower body mass compared to unexposed controls. In adults, prolonged exposure can lead to decreased weight gain, reduced condition factor, and impaired organ development. Chronic stress from these contaminants also diverts energy from growth toward detoxification and repair mechanisms, further limiting normal physiological development.

Immune Dysfunction

Exposure to combined pesticides and metals compromises the fish immune system. Studies report reduced leukocyte counts, suppressed phagocytic activity, and decreased production of antibodies and immunoglobulins. This immunosuppression increases susceptibility to bacterial, viral, and parasitic infections, reducing survival rates. Stress from oxidative damage and metabolic disruption can exacerbate immune dysfunction, impairing the fish's ability to mount effective immune responses under natural environmental pressures.

Reproductive Toxicity

Reproductive performance is highly sensitive to co-exposure. Gamete quality is often reduced, with lower sperm motility, decreased egg viability, and abnormal gonadal histology. Hormonal disturbances, including altered levels of testosterone, estradiol, and thyroid hormones, disrupt reproductive cycles and spawning behavior. These reproductive impairments can lead to population-level consequences, reducing recruitment and threatening the sustainability of fish stocks in contaminated water bodies.

Bioaccumulation and Tissue Distribution

Combined pesticide and metal exposure frequently enhances bioaccumulation in fish tissues. Metals and lipophilic pesticides tend to accumulate in metabolically active organs such as the liver and kidney, while muscle tissue accumulates residues relevant to human consumption. Increased bioconcentration factors (BCFs) in co-exposed fish highlight the potential for amplified toxic effects and elevated food safety risks. Chronic accumulation can also exacerbate organ damage and metabolic disturbances, reinforcing the ecological and health implications of multi-contaminant exposure.

Biomarkers of Synergistic Toxicity

Biochemical biomarkers (AChE inhibition, antioxidant enzyme activity, lipid peroxidation) are used to detect early toxic effects. Multi-omics approaches (transcriptomics, proteomics) offer deeper insights into mechanistic responses.

Factors Influencing Synergistic Effects

Species Variability

Different fish species vary in sensitivity due to differences in metabolic rates, gill morphology, and detoxification capacity.

Life Stage Sensitivity

Larvae and juveniles are more vulnerable to chemical stress than adults.

Environmental Modulators

Temperature, pH, dissolved organic matter, and water hardness affect contaminant bioavailability.

6. Human Health and Food Safety Implications

Eating fish with enhanced contaminant loads poses risks such as neurological disorders, carcinogenic effects, and endocrine disruption. Dietary exposure models must consider synergistic residues.

7. Current Research Gaps

Significant gaps remain, including limited mixture toxicity data, lack of long-term exposure studies, and inadequate models for predicting synergistic outcomes. Data standardization is needed.

8. Future Directions

Emerging approaches include predictive quantitative mixture toxicity models, biomarker panels, integrative omics, environmental monitoring networks, and regulatory frameworks that incorporate interaction effects.

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Conflicts of interest

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