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A Review on Effects of Mercuric Chloride Induced Toxicity in *Cyprinus carpio*

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Abstract: *Mercury (Hg) is a globally pervasive and hazardous aquatic pollutant, with mercuric chloride (HgCl2) posing a significant threat due to its toxicity and potential transformation into highly bioaccumulative methyl mercury. This structured literature review synthesizes peer-reviewed research on the effects of HgCl2 on Cyprinus carpio (common carp), a widely distributed and environmentally tolerant freshwater fish. The review highlights that HgCl2 exposure induces a range of adverse effects across multiple biological levels.Behaviorally,C. carpio exhibits altered swimming patterns, loss of equilibrium,and sluggishness. Physiologically, it leads to impaired growth and increased mortality, with larvae being particularly vulnerable to developmental abnormalities such as skeletal deformities and organ malformations. Hematological analyses consistently reveal anemia,characterized by decreased red blood cell parameters(TEC,Hb,HCT, MCV, MCH, MCHC),and an activated, though potentially compromised, immune response indicated by increased white blood cell counts. Histopathological examinations demonstrate widespread tissue damage in vital organs, including the liver, gills, kidney, ovary, olfactory epithelium and intestine, leading to functional impairment. At the cellular and molecular levels, HgCl2 induces DNA damage, oxidative stress (through ROS generation and antioxidant enzyme inhibition) and disrupts enzyme activities and signaling pathways. Furthermore, it exerts significant reproductive and developmental toxicity,impacting gonad development and embryonicviability.Despite extensive research, knowledge gaps remain concerning the precise mechanisms of mercury transformation, long-term sub-lethal effects on immune function and male fertility, and the efficacy of mitigation strategies in real-world scenarios. Future research should focus on integrated field and laboratory studies, advanced mechanistic investigations, novel biomarker development and validating mitigation strategies to inform effective environmental policies and safeguard aquatic ecosystems and human health.*

Keywords: Mercuric chloride,*Cyprinus carpio*,Aquatic toxicology,Bioaccumulation,Oxidative stress, Genotoxicity, Histopathology, Fish physiology, Environmental pollution

I. INTRODUCTION

Overview of Mercury as an Aquatic Pollutant and its Significance

Mercury (Hg) represents one of the most pervasive and hazardous contaminants in aquatic environments, posing significant threats to aquatic life and human health globally.[1] Its widespread distribution stems from a combination of historical industrial applications, agricultural use of organomercurials, and long-range atmospheric transport originating from sources such as fossil fuel combustion.[1]

The **World Health Organization (WHO)** has recognized mercury as one of the top ten chemicals of major public health concern due to its severe toxic effects across multiple physiological systems, including the nervous, digestive, and immune systems, as well as the lungs, kidneys, skin, and eyes.[2] This broad spectrum of systemic toxicity presents a particular danger to the developing child, both in utero and during early life stages.[2]

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The ubiquitous nature of mercury contamination, driven by both historical industrial practices and ongoing global atmospheric transport, transforms it from a localized pollutant into a pervasive environmental and public health crisis. This broad distribution necessitates a comprehensive understanding of its ecotoxicological impacts, especially on organisms central to aquatic food webs, for informing public health advisories related to seafood consumption.

Introduction to *Cyprinus carpio* (Common Carp) as a Relevant Aquatic Organism for Ecotoxicological Studies

Cyprinus carpio, commonly known as the **common carp**, is a large freshwater fish characterized by its remarkable adaptability to a wide array of environmental conditions.[3] This species exhibits considerable tolerance to brackish water, low oxygen levels, high turbidity, and various forms of pollution.[3] Such inherent resilience has facilitated its widespread use in aquaculture and stocking programs, contributing to its successful colonization of numerous aquatic habitats worldwide.[3]

Although historically valued as a food fish, *C. carpio* is now frequently categorized as a **nuisance species** due to its abundance and the detrimental effects it can have on aquatic ecosystems, notably by increasing water turbidity through its feeding habits.[3] As an **omnivorous species**, *C. carpio* primarily feeds by rooting in the mud, consuming plants, seeds, small invertebrates, and detritus.[3]

The common carp's remarkable environmental tolerance and omnivorous, bottom-feeding habits position it as a **critical bioindicator** for heavy metal contamination in aquatic ecosystems. Its dual role as both an aquaculture species and a component of the human diet in many regions further amplifies the importance of studying its response to pollutants like **mercuric chloride**, directly linking environmental health to human health concerns. The ability of *C. carpio* to survive in polluted environments where less tolerant species might perish, combined with its feeding behaviors that expose it directly to sediment-bound contaminants, makes it an ideal, albeit concerning, subject for **ecotoxicological studies**, as it can serve as a significant pathway for pollutant transfer to humans.

Rationale and Scope of the Literature Review

This literature review provides a comprehensive and structured analysis of peer-reviewed research concerning the effects of **mercuric chloride ($HgCl_2$)** on *Cyprinus carpio*. The specific focus on $HgCl_2$, an **inorganic mercury compound**, is crucial because it represents a common form of **industrial discharge** serves as a significant precursor to the formation of more toxic organic mercury species within aquatic systems.[1] This review synthesizes key research themes, highlights important studies and their core findings, discusses conflicting viewpoints and ongoing debates within the scientific community, identifies existing gaps in the literature, and proposes directions for future research to advance the understanding of **mercury ecotoxicology** in this important fish species.

II. MERCURIC CHLORIDE IN AQUATIC ENVIRONMENTS: SPECIATION AND BIOACCUMULATION

Mercury Forms and Their Relative Toxicities

Mercury exists in various chemical forms and valence states (0, +1, and +2) within aquatic environments, and its ecological and toxicological effects are profoundly dependent on the specific chemical species present.[1]

While inorganic mercury, such as **mercuric chloride ($HgCl_2$)**, is inherently toxic, it can undergo transformation into significantly more hazardous methylated forms—most notably **methylmercury (MMHg)**.[1] MMHg is recognized as a potent neurotoxin, posing severe risks to biological systems.[1]

A critical aspect of mercury's environmental hazard is that MMHg, due to its **lipophilic and protein-binding properties**, is readily accumulated by aquatic biota. This process leads to **bioaccumulation** within individual organisms and subsequent **biomagnification** up the food chain, thereby posing a considerable threat to humans and other fish-eating animals.[1]

The transformation of inorganic $HgCl_2$ to highly toxic methylmercury within aquatic systems represents a critical environmental pathway that magnifies its hazard. This chemical **speciation process**, often microbially mediated, means

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that even a localized release of less toxic inorganic mercury can lead to severe bioaccumulation risks in fish and, subsequently, in human consumers. This highlights the complex interplay between environmental chemistry, biological processes, and public health, indicating that effective pollution control must not only limit inorganic mercury discharge but also encompass strategies to mitigate the environmental conditions that promote its methylation.

Key Environmental Factors Influencing Mercury Transformation and Methylation

Despite a substantial body of literature, the precise behavior of mercury and the intricate transformation and distribution mechanisms operating in natural aquatic environments remain incompletely understood.[1] However, several **key environmental factors** have been identified as significantly influencing mercury transformation, particularly its methylation into highly toxic forms:[1]

Microbiology:

Microorganisms, especially anaerobic **sulfate-reducing bacteria (SRB)**, play a pivotal role in catalyzing the conversion of Hg^{2+} to methyl and dimethyl mercury. The efficiency of this process is influenced by the activity and structure of the bacterial community, the availability of inorganic mercury, nutrient levels, and the presence of electron acceptors like sulfate. While low sulfate concentrations can stimulate methylation, high concentrations may lead to sulfide accumulation, which can interfere with the process.[1]

Temperature:

Mercury methylation rates commonly peak during warmer summer months. Temperature primarily affects methylation by influencing overall microbial activity. Studies indicate that MMHg release from sediments significantly decreases at lower temperatures, suggesting reduced net MMHg production in winter due to diminished microbial growth and metabolic rates.[1]

pH:

Fluctuations in pH can impact MMHg concentrations by affecting the solubility and mobility of both inorganic mercury and MMHg, and potentially by increasing mercury inputs from watersheds. Generally, low pH conditions facilitate the release of heavy metals from sediments and increase MMHg solubility. Conversely, higher pH values tend to favor the formation of volatile elemental mercury (Hg^0) and **dimethylmercury (DMHg)**, which can reduce MMHg concentrations.[1]

Organic Material:

The role of organic matter is complex. Increased MMHg concentrations are often associated with higher levels of **organic carbon**, as organic nutrients can stimulate microbial methylation. However, **dissolved organic carbon (DOC)** can also mitigate MMHg production and bioaccumulation by complexing inorganic mercury, thereby reducing its bioavailability to methylating bacteria, especially in neutral pH ranges. The degradation of organic matter can also lead to **anoxic conditions**, mobilizing inorganic mercury for methylation.[1]

Redox Conditions:

Mercury methylation rates are highest in **anoxic sediments and waters**, indicating that the process predominantly occurs under anaerobic conditions. Both methylation rates and MMHg stability appear enhanced in anaerobic environments, while aerobic conditions generally favor MMHg degradation. MMHg concentrations are typically highest in **moderately anaerobic surface sediments** and at the **oxic/anoxic boundary layer** in stratified aquatic systems.[1]

Sulfide:

High sulfide concentrations typically inhibit MMHg formation in soils, sediments, and bacterial cultures. This inhibition is linked to sulfide controlling mercury speciation, leading to the formation of charged disulfide complexes that are less bioavailable for bacterial uptake. However, MMHg production is not usually completely inhibited, and in some cases, levels can increase with rising sulfide concentrations.[1]

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Salinity:

The methylating activity in **marine and estuarine sediments** is generally lower than in freshwater sediments, largely due to salinity effects. High salinity conditions, particularly under reducing conditions, appear to promote **demethylation** and inhibit mercury methylation. This negative effect is primarily linked to the microbial production of sulfide from sea salt sulfate and the formation of charged sulfide and chloride complexes, which reduce the bioavailability of mercury for methylation.[1]

The intricate interplay of these environmental factors — ranging from microbial communities and temperature to pH and redox conditions — dictates the **bioavailability and ultimate toxicity of mercuric chloride** in aquatic systems. This **multi-factorial dependency** implies that pollution control strategies must extend beyond simply limiting mercury discharge to encompass broader ecosystem management.

Recognizing that environmental conditions can either **exacerbate or mitigate** the formation of highly toxic methylmercury influences the actual risk to *Cyprinus carpio* and human consumers, necessitating a **holistic environmental management strategy**.

Mechanisms of Mercury Uptake and Bioaccumulation in Aquatic Organisms, Particularly *Cyprinus carpio*

Fish are known to concentrate mercury in their bodies, often in the highly toxic form of **methylmercury**.[7] In *Cyprinus carpio*, mercury accumulates in various tissues and organs, with studies indicating that **gills often exhibit the highest metal concentrations**, followed by **gonads** and **muscle**.[8]

Critically, bioaccumulation of mercury in *C. carpio* liver, gills, and muscle samples has been found to exceed the **permissible limits set by the World Health Organization (WHO)**, raising significant public health concerns.[6] The extent of metal accumulation is not uniform and is influenced by several biological and environmental factors, including the individual fish's age and size, specific feeding habits, life cycle, and seasonal variations.[8]

Cyprinus carpio's bioaccumulation of mercury, particularly in edible tissues like muscle and vital organs like gills and liver, underscores its role as a **significant vector for human exposure**. The fact that accumulation often exceeds WHO limits, coupled with the influence of factors like age and feeding habits, highlights the need for **continuous monitoring and risk assessment** of *C. carpio* populations in contaminated areas to protect public health.

This also implies that not all fish within a population will be equally contaminated, which is a crucial consideration for **targeted risk assessment, dietary advisories**, and potentially for **managing aquaculture practices** to minimize mercury uptake.

III. PHYSIOLOGICAL AND BEHAVIORAL RESPONSES OF *CYPRINUS CARPIO* TO MERCURIC CHLORIDE

Observed Changes in Behavior

Exposure to mercuric chloride ($HgCl_2$) induces a range of significant behavioral alterations in Cyprinus carpio and other susceptible fish species.[9] In C. carpio, even at a relatively low concentration of 0.5 ppm $HgCl_2$, a change in body color was observed after 48 hours, although no other overt abnormal activity was noted initially.[9]

However, at higher concentrations, such as 1.0 ppm and 1.5 ppm $HgCl_2$, more severe behavioral disturbances became apparent after 72 hours, including abnormal posturing, disbalance, and sluggishness. The 1.5 ppm group experienced 100% mortality within 124 hours.[9]

More broadly, behavioral changes reported in fish exposed to mercury include respiratory distress, loss of balance, gulping for air, and rolling movements.[10] Other studies detail altered schooling behavior, localization at the bottom of the tank, irregular and erratic swimming patterns, frequent surface visits, caudal bending, spinning of the body, and delayed response to touch.[11]

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Morphological changes, such as body decoloration and excessive mucus secretion, are also commonly observed.[11] These behavioral changes serve as sensitive, early-warning indicators of mercuric chloride toxicity in *Cyprinus carpio*, often preceding severe physiological damage or mortality.

The progression from subtle changes like body discoloration to overt distress, loss of equilibrium, and altered swimming patterns reflects progressive neurological and respiratory impairment, providing observable cues for environmental monitoring. These behavioral shifts are practical and observable biomarkers for both acute and sub-lethal mercury toxicity.

Impacts on General Physiological Functions and Survival Rates

Mercurial toxicity, even at relatively low concentrations (e.g., 0.1 ppm), has been shown to cause adverse effects on the general physiological functions of *Cyprinus carpio*.[9] Higher concentrations of $HgCl_2$ directly lead to increased mortality rates; for *C. carpio*, the 96-hour median lethal concentration (LC_{50}) was determined to be 1.3 ppm.[12]

Notably, *C. carpio* larvae are particularly vulnerable to $HgCl_2$, with significantly increased mortality rates observed when they are between 10 and 25 days old, indicating this period as a highly sensitive developmental window.[13]

Beyond acute mortality, mercury exposure has also been demonstrated to harm the overall growth rate of fish.[14] The dose- and age-dependent increase in mortality and growth impairment in *Cyprinus carpio* exposed to mercuric chloride highlights the direct threat to population viability.

The acute toxicity, evidenced by LC_{50} values, combined with the heightened vulnerability of larvae, suggests that mercury pollution can significantly impact recruitment and overall fish biomass, potentially disrupting aquatic food webs and fisheries. This indicates that mercury's impact extends beyond individual fish health to potentially compromising reproductive success and long-term population dynamics of *Cyprinus carpio*, which has broader implications for ecosystem stability and the sustainability of fisheries.

IV. HEMATOLOGICAL ALTERATIONS INDUCED BY MERCURIC CHLORIDE

Detailed Analysis of Changes in Red Blood Cell Parameters

Exposure to mercuric chloride ($HgCl_2$) consistently induces significant alterations in the hematological parameters of *Cyprinus carpio*. Studies have shown a marked decrease in total erythrocyte count (TEC), hemoglobin (Hb) levels, hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC).

These reductions collectively indicate a compromised oxygen-carrying capacity of the bloodstream, often leading to an anemic state in the affected fish. Specifically, changes in MCV, MCH, and MCHC signify chemical stress and pathological conditions within tissues, which may result from increased red blood cell lysis or a reduction in cellular blood iron, thereby impeding hemoglobin synthesis.

The consistent and significant reduction in multiple red blood cell parameters across various studies on *Cyprinus carpio* and other fish species points to a fundamental disruption of oxygen transport and cellular integrity. This induced anemia not only impairs the fish's metabolic function and activity but also serves as a robust and easily measurable biomarker for mercury-related stress, offering a quantifiable indicator of physiological distress long before overt mortality.

The consistency of these findings across multiple studies reinforces their reliability as indicators, demonstrating that these hematological parameters are not merely symptomatic observations but direct, quantifiable measures of the fish's ability to sustain fundamental life processes, making them invaluable for assessing the severity of mercury toxicity.

Impacts on White Blood Cell Counts and Immune Responses

In contrast to red blood cell parameters, the total leukocyte count (TLC) in *Cyprinus carpio* generally shows an increase following exposure to $HgCl_2$. This elevation in white blood cells is typically indicative of an immune response

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to stress or infection, suggesting that the fish's immune system is actively reacting to the presence of the toxicant. Interestingly, studies on other teleost fish have shown that inorganic mercury can elicit a dual effect on leukocytes, causing high-dose inhibition while paradoxically activating them at lower, subtoxic concentrations. The observed increase in white blood cell count in *Cyprinus carpio* suggests an activated, albeit potentially compromised, immune response to mercuric chloride exposure. While an initial increase might indicate an attempt to combat the toxic insult, the potential for high-dose inhibition implies that prolonged or severe exposure could lead to immunosuppression, leaving the fish more vulnerable to diseases and secondary infections, thus exacerbating the overall health impact.

This complex modulation of the immune system by heavy metals has significant implications for the fish's long-term health and its ability to resist pathogens.

Implications for Oxygen Transport and Overall Fish Health

Hematological variables are widely recognized as crucial pathophysiological indicators reflecting the overall structural and functional status of an organism. The impaired hematology observed in mercury-exposed fish can directly manifest in their behavioral activities.

Specifically, the hypoxia triggered by mercury exposure can lead to a compensatory decrease in erythrocytes, as the body attempts to manage oxygen deficiency.

The widespread hematological disruptions caused by mercuric chloride have cascading effects on *Cyprinus carpio's* overall physiological well-being, most notably impairing oxygen transport.

This systemic stress, indicated by blood parameters, directly correlates with observed behavioral abnormalities and reduced metabolic efficiency, demonstrating how mercury toxicity undermines fundamental biological processes essential for survival and reproduction.

This reveals a clear cause-and-effect chain where mercury exposure leads to hematological changes, which impair oxygen transport, leading to systemic stress and ultimately manifesting as behavioral and physiological decline. This holistic perspective is crucial for understanding the full, interconnected impact of the pollutant on the fish's vitality.

V. HISTOPATHOLOGICAL AND ORGAN-SPECIFIC DAMAGE

Examination of Tissue Damage in Vital Organs

Mercuric chloride ($HgCl_2$) exposure leads to significant and widespread tissue damage across various vital organs in Cyprinus carpio and other fish species.[6]

- **Liver**: This organ is a primary target for mercury-induced injury due to its central role in detoxification, storage and redistribution of mercury.[18] Observed damage includes noticeable degenerative histophysiological changes [15], extensive fibrosis[15], complete dissolution and degeneration of hepatocytes[15] and more severe pathological findings such as pyknosis, necrosis, dilation of sinusoids and cytoplasmic vacuolation.[6]
- **Kidney**: Damage includes renal tubular disorientation[17] and glomerular shrinkage with increased Bowman's space.[15]
- **Gills**: Often the first point of contact for waterborne pollutants, gills frequently show the highest metal concentrations.[8] Pathological changes include swelling of secondary lamella[15], a decrease in the height and an increase in the thickness of primary lamellae, slight pathological lesions in the primary and secondary lamellar epithelium (observed at 0.04 mg/L $HgCl_2$ after 30 days post-hatching), severe modifications and necrosis in secondary lamellae (after 35 days post-hatching), deformation and necrosis of pillar cells (at 0.08 mg/L $HgCl_2$ after 40 days post-hatching) and even blood clotting at the base of the secondary lamellae after 60 days of exposure.[6] These alterations severely impair the fish's ability to exchange oxygen.[13]

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● **Ovary**: Noticeable degenerative histophysiological changes are observed in the ovary.[15] Prolonged exposure can lead to overall retardation of oocyte development, denaturation of yolk material and complete drop-out of vitellogenic/maturing oocytes.[15]

● **Olfactory Epithelium**: Damage includes degeneration of columnar sensory cells, supporting cells and ciliated non-sensory cells. Basal cell proliferation, hyperplasia, thickening of the epithelium, basal lamina disruption and cyst formation are also noted. Scanning electron microscopy has revealed clumping and loss of cilia, erosion in microridges on supporting cells and proliferation of mucous cell openings.[15] Such prolonged exposure might cause irreversible damage and impair the olfactory function of the fish.[15]

● **Intestine**: Mercury contamination can cause tissue injuries such as degeneration, necrosis and erosion, as well as an increase and swelling of goblet cells.[18]

The widespread and severe histopathological damage observed across multiple vital organs in Cyprinus carpio reveals that mercuric chloride is a multi-systemic toxicant. The specific nature of the damage in each organ (e.g., gill lamellar fusion impairing respiration, hepatic necrosis affecting detoxification, ovarian degeneration impacting reproduction) indicates that mercury not only causes general cellular distress but targets specific physiological functions, leading to profound and potentially irreversible impairment of the fish's health and survival capacity. This comprehensive evidence demonstrates that mercury's impact is not isolated but rather a systemic assault, leading to functional impairment that directly threatens the fish's ability to survive and thrive.

Recovery Patterns

Following the withdrawal of $HgCl_2$ treatment, a degree of recovery was observed in both the ovary and the liver of *Cyprinus carpio*. However, this recovery was notably more significant and "appreciable" in the liver compared to the ovary.[15] The differential recovery capacity of organs, with the liver showing better recovery than the ovary, suggests varying degrees of cellular resilience and regenerative potential against mercuric chloride toxicity. This implies that while some physiological functions might rebound after exposure ceases, long-term reproductive health could be more persistently compromised, with significant implications for population recovery in contaminated environments. This observation immediately prompts the question of why this difference exists, indicating that certain organs or physiological systems (like reproduction) may suffer more permanent damage, even after the removal of the toxicant.

VI. CELLULAR AND MOLECULAR MECHANISMS OF TOXICITY

Discussion of DNA Damage

Mercury is widely recognized as one of the most dangerous heavy metals due to its potent ability to induce DNA damage.[20] Its genotoxic effects manifest through various mechanisms, including chromosomal breakage (clastogenic events) or spindle dysfunction (aneugenic events), both of which lead to the formation of micronuclei (MNE).[20] Elevated DNA damage, often quantified using techniques like the comet assay, has been consistently observed in fish exposed to $HgCl_2$.[17] Specifically, DNA damage has been reported in the ovaries of carps after mercury exposure (Masud et al., 2009; Stadnicka, 1980; Zarnescu, 2009, cited in [15]). Beyond micronuclei, other nuclear abnormalities (ENAs) such as blebbed, binucleated and notched nuclei are also observed. A third mechanism for micronuclei genesis has been proposed, involving chromatin fragmentation within notched nuclei, resulting from a combination of mercury's cytotoxic effects and mechanical stress.[20] Notably, fish erythrocytes are nucleated, rendering them more sensitive to the induction of DNA damage compared to mammalian cells.[20] At a molecular level, mercury's strong affinity for sulfhydryl groups in proteins can interfere with crucial DNA repair pathways and mitotic spindle function, leading to chromosomal instability and cell cycle errors.[19] The pervasive genotoxic effects of mercuric chloride, manifesting as DNA damage, chromosomal instability and various nuclear abnormalities, underscore its fundamental disruption of cellular integrity and genetic information. The proposed multi-mechanistic genesis of micronuclei (clastogenic, aneugenic and chromatin fragmentation from notched nuclei) highlights the diverse pathways through

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which mercury compromises cellular replication and function, potentially leading to long-term health consequences, including increased susceptibility to disease and impaired reproductive success in *Cyprinus carpio* populations. This level of detail moves beyond simply stating that damage occurs to explaining how it occurs at a fundamental cellular level. The implication of mercury interfering with DNA repair and mitotic spindle function is profound: it suggests that mercury acts as a potent mutagen, affecting the very blueprint of life.

Role of Oxidative Stress

Oxidative stress is consistently identified as a key mechanism underlying mercury toxicity in fish.[19] Mercuric chloride exposure leads to an imbalance in cellular redox homeostasis by both generating reactive oxygen species (ROS) and simultaneously inhibiting crucial antioxidant enzymes. These enzymes include superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx).[19] This resulting overproduction of ROS coupled with depleted antioxidant defenses initiates a state of oxidative stress, which is a major contributor to the development of various chronic and degenerative illnesses.[19] The accumulation of malondialdehyde (MDA), a byproduct of lipid peroxidation, serves as a reliable indicator of increased cellular ROS and associated cellular injuries.[22] Furthermore, studies on *C. carpio* have established a direct correlation between oxidative stress and genotoxicity.[19] Oxidative stress acts as a central mediator of mercuric chloride toxicity, linking its molecular interaction with cellular components to broader physiological dysfunction. The dual impact of mercury both generating reactive oxygen species and simultaneously inhibiting the fish's natural antioxidant defenses creates a vicious cycle of cellular damage, which is further correlated with genotoxicity. This highlights oxidative stress as a critical therapeutic target for mitigating mercury's effects and a robust biomarker for environmental monitoring. The detailed explanation of how mercury both increases ROS and inhibits antioxidant enzymes provides a crucial mechanistic understanding of how the damage occurs.

Impacts on Enzyme Activities and Signaling Pathways

Beyond direct cellular damage, mercuric chloride exposure profoundly interferes with fundamental cellular biochemistry by altering enzyme activities and activating stress-response signaling pathways. Elevated activation of Mitogen-Activated Protein Kinase (MAPK) pathways, particularly p38 MAPK, has been observed in liver and kidney tissues, implicating their role in mediating stress responses and inflammation.[17] Furthermore, $HgCl_2$ affects the activity of various enzymes, including digestive enzymes such as amylolytic and proteolytic enzymes[24] and critical antioxidant enzymes like SOD, CAT and GST.[22] Changes in the activity and expression levels of these enzymes can serve as valuable biomarkers for evaluating the influence of mercuric chloride and assessing the biochemical pathway and enzymatic function in exposed fish.[22] The disruption of critical enzyme activities and activation of stress-response signaling pathways (like MAPK) by mercuric chloride reveals its profound interference with fundamental cellular biochemistry. These molecular perturbations, extending beyond direct cellular damage to include altered metabolic processing and inflammatory responses, provide a deeper understanding of the systemic physiological dysfunctions observed in *Cyprinus carpio* and offer potential targets for early detection and intervention strategies. This moves the analysis beyond gross structural damage to functional impairment at the molecular level, showing the cell's attempt to respond to the insult, while altered enzyme activity directly indicates metabolic disruption.

VII. REPRODUCTIVE AND DEVELOPMENTAL TOXICITY

Effects on Embryonic Development

Both lethal and sub-lethal doses of mercuric chloride ($HgCl_2$) have a significant impact on the embryonic development of *Cyprinus carpio*.[13] Higher concentrations of mercury are known to inhibit the overall development of fish embryos.[26] Observed malformations in embryos within the egg include incompletely formed eyes, yolk sac edema, short tails, deformed heads and even a complete lack of a tail.[13] Upon hatching, the exposed larvae continue to exhibit clinical pathologies such as malformation of the yolk sac, body shortening, reduced overall size and a noticeable loss of

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equilibrium in the water. Pronounced skeletal deformities, including lateral spine curvature (scoliosis), axial spine curvature (lordosis) and C-shaped bodies, are also common.[13] The mortality rates of larvae increase significantly with higher concentrations of $HgCl_2$, particularly when the larvae are between 10 and 25 days old, indicating this as a highly sensitive and dangerous developmental period.[13] The profound and diverse embryonic and larval malformations induced by mercuric chloride, ranging from severe skeletal deformities to organ underdevelopment and high mortality, highlight its potent teratogenic and developmental toxicity. This early life stage vulnerability suggests that mercury pollution can severely compromise the reproductive success and population recruitment of *Cyprinus carpio*, potentially leading to long-term declines even if adult populations appear resilient. The heightened vulnerability of larvae means that even if adult populations survive, the inability of young fish to develop properly and reach maturity will severely impact population recruitment, leading to long-term demographic declines and potential ecosystem imbalances.

Impacts on Gonad Development, Fertility and Gamete Quality

Mercuric chloride exposure has a direct and detrimental impact on the reproductive systems of *Cyprinus carpio*. Noticeable degenerative histophysiological changes have been observed in the ovary of *C. carpio* following $HgCl_2$ exposure. Prolonged exposure can lead to a significant retardation of overall oocyte development, denaturation of yolk material essential for embryonic nutrition and the complete drop-out of vitellogenic and maturing oocytes. Furthermore, DNA damage has been specifically reported in the ovaries of carps after mercury exposure. Beyond female reproductive health, mercury is a widespread aquatic pollutant known to adversely affect the reproductive system of male fish, impacting testicular structure, sperm count and morphology. While specific details on $HgCl_2$ effects on *C. carpio* sperm quality and fertilization are less documented in the provided material, the general understanding of mercury's impact on male reproductive health in fish suggests similar vulnerabilities. The detrimental effects on both ovarian development and male reproductive parameters indicate a comprehensive assault on *Cyprinus carpio*'s reproductive capacity. This multi-faceted reproductive impairment, affecting both gamete production and embryonic viability, poses a significant threat to the long-term sustainability of *C. carpio* populations in contaminated aquatic environments. The direct damage to reproductive organs and processes means that even if adult fish survive, their ability to successfully reproduce and contribute to population replenishment is severely compromised, leading to potential population collapse over time.

VIII. MITIGATION STRATEGIES AND PROTECTIVE EFFECTS

Research into mitigating the toxic effects of mercuric chloride in *Cyprinus carpio* has explored various protective compounds. Dietary supplementation with selenium, vitamin C and vitamin E has shown promise in alleviating stress conditions caused by mercury in common carp larvae. These antioxidants and essential elements can counteract the oxidative stress induced by mercury, which is a central mechanism of its toxicity. Another study demonstrated that thymol, a natural monoterpene found in plant essential oils, has protective effects on the growth factors of juvenile carp following chronic mercury exposure. Thymol significantly improved absolute growth, absolute growth rate and food conversion ratio (FCR) in mercury-exposed fish, indicating its potential to reduce the adverse effects on growth. These findings suggest that nutritional interventions and natural compounds can offer viable strategies to enhance the resilience of *Cyprinus carpio* to mercuric chloride pollution, potentially improving survival and growth rates in contaminated aquaculture or natural settings. The exploration of such mitigation strategies is crucial for developing practical solutions to protect fish populations and ensure food safety in mercury-affected aquatic systems.

IX. Evolution of Research Themes and Key Studies Tracing the Progression of Research Focus Over Time

Research into mercury toxicity in aquatic organisms, including *Cyprinus carpio*, has evolved significantly over time, moving from initial observations of acute toxicity and general physiological responses to more detailed investigations into cellular, molecular and long-term impacts. Early studies often focused on establishing lethal concentrations (LC_{50}) and observable behavioral changes, such as altered swimming patterns, loss of balance and changes in body coloration.

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As methodologies advanced, research expanded to encompass hematological parameters, revealing consistent changes in red and white blood cell counts and hemoglobin levels as indicators of systemic stress and anemia. More recently, the focus has shifted towards understanding the underlying histopathological damage in vital organs like the liver, kidney and gills, along with the intricate cellular and molecular mechanisms of toxicity, including DNA damage, oxidative stress and the activation of specific signaling pathways. Concurrently, the impacts on reproductive success and early developmental stages have gained prominence, highlighting the teratogenic effects and long-term population consequences. The progression of research reflects a deepening understanding of mercury's complex modes of action, from macroscopic observations to microscopic and molecular perturbations, which is essential for developing comprehensive risk assessments and targeted mitigation strategies.

Highlighting Seminal Studies and Their Core Findings Across the Identified Themes

Several studies stand out for their contributions to understanding the effects of mercuric chloride on *Cyprinus carpio*.

Table 1: Summary of Key Studies on Mercuric Chloride Effects on *Cyprinus carpio*

Study (Author, Year)	$HgCl_2$ Concentration	Exposure Duration	Key Observed Effects	Specific Findings
Masud et al. (2009)	0.5, 1.0, 1.5 ppm (behavioral); 0.1 ppm (hematological)	8–124 hrs (behavioral); 60 days (hematological)	Behavioral, Hematological	Body color change (0.5 ppm, 48 hrs); Abnormal posturing, disbalance, sluggishness (1.0, 1.5 ppm, after 72 hrs); 100% mortality (1.5 ppm, within 124 hrs). Decreased TEC, Hb; Increased TLC (0.1 ppm, 60 days).
JETIR (2019)	0.9, 1.1, 1.2, 1.3 ppm (LC_{50} 1.3 ppm)	96 hours	Hematological	Significant decrease in RBC, Hb, HCT, MCV, MCH, MCHC. Significant increase in WBC.
Masud et al. (2009)	0.5 ppm (short-term); 0.1 ppm, 0.5 ppm (prolonged)	7 days (short-term); 45, 60 days (prolonged)	Histopathological (Ovary, Liver), Hematological	Degenerative changes in ovary & liver (0.5 ppm, 7 days); Recovery more appreciable in liver. Retardation of oocyte development, yolk denaturation, oocyte drop-out (0.1, 0.5 ppm, 45–60 days). Decreased erythrocytes, Hb, HCT; Increased leukocytes, blood sugar (0.15 ppm).
El Mattary et al. (2024)	0.02, 0.04, 0.06, 0.08 mg/L	60 days	Embryonic Development, Histopathological (Gills)	Embryonic malformations: incomplete eyes, yolk sac edema, short/lack of tail, deformed head/yolk sac. Larval deformities: lateral/axial spine curvature, C-shaped body, body shortening. Increased mortality (10–25 day old larvae). Gill damage: decreased lamellar height, increased thickness, necrosis of secondary lamellae/pillar cells, blood clotting.
ResearchGate (2017)	0.25, 0.5 µg $HgCl_2$/g body weight	Not specified	Oxidative Stress, Genotoxicity	$HgCl_2$ induced oxidative stress and genotoxicity; Correlation between effects. Inhibition of SOD, CAT, GPx leading to

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Study (Author, Year)	$HgCl_2$ Concentration	Exposure Duration	Key Observed Effects	Specific Findings
				ROS accumulation. Elevated serum ALT, AST, urea, creatinine; Decreased total protein, albumin. Increased Hg tissue concentrations, HSP70 mRNA expression.
Scielo (2019)	0.1, 0.25, 0.50 mg/kg body weight (IP injection)	24, 72, 96 hours	Genotoxicity (Micronucleus formation)	Higher frequency of micronucleated and notched erythrocytes. Positive correlation between micronucleated and notched erythrocytes. Proposed third mechanism of micronuclei genesis (chromatin fragmentation in notched nuclei).
Iranian Journal of Veterinary Medicine (2025)	0.44 mg/L	56 days	Growth Factors, Protective Effects	Thymol improved absolute growth, absolute growth rate and FCR. Thymol can effectively reduce side effects of $HgCl_2$.

X. CONFLICTING VIEWPOINTS, ONGOING DEBATES AND KNOWLEDGE GAPS

Despite the extensive research on mercuric chloride toxicity in *Cyprinus carpio*, several areas remain subject to ongoing debate or represent significant knowledge gaps. One such area concerns the precise mechanisms of mercury transformation in aquatic environments. While anaerobic sulfate-reducing bacteria are identified as principal methylators, the behavior of mercury and many of its transformation and distribution mechanisms are still poorly understood. For instance, the role of organic matter in methylation is complex, with some studies suggesting it stimulates microbial methylation while others indicate dissolved organic carbon can mitigate methylmercury production by complexing inorganic mercury. Similarly, the effect of sulfide on methylmercury formation is not completely uniform, with some instances showing increased levels despite general inhibition. These nuances highlight the need for more detailed studies on environmental factors and their dynamic interplay.

Another area of ongoing discussion relates to the long-term, sub-lethal effects of mercuric chloride, particularly on immune function and reproductive success. While an increase in white blood cells suggests an immune response, other studies indicate high-dose inhibition of leukocytes, which could lead to immunosuppression. The threshold and duration at which this shift occurs and its implications for the fish's susceptibility to disease require further elucidation. Similarly, while ovarian degeneration and embryonic abnormalities are well-documented, the full extent of impacts on male fertility, gamete quality and the transgenerational effects of mercury exposure in *Cyprinus carpio* populations remain less explored.

A notable gap exists in comprehensive, long-term field studies that integrate physiological, behavioral, histopathological and molecular endpoints across different life stages of *Cyprinus carpio* in naturally contaminated environments. Most existing research relies on controlled laboratory settings with acute or sub-acute exposures, which may not fully replicate the chronic, fluctuating and multi-stressor conditions found in natural aquatic ecosystems. The interaction between mercury exposure and other environmental stressors, such as magnetic fields, also remains poorly understood. Furthermore, while some mitigation strategies have been explored, the long-term efficacy and practical applicability of these interventions in large-scale environmental remediation or aquaculture settings require more robust investigation. The extent of recovery in various organs, particularly the reproductive system, after removal from mercury exposure also warrants further detailed study to understand the potential for population rebound.

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XI. SUGGESTIONS FOR FUTURE RESEARCH

Based on the identified knowledge gaps and ongoing debates, several avenues for future research are proposed to enhance the understanding of mercuric chloride effects on *Cyprinus carpio*:

Integrated Field and Laboratory Studies: Future research should prioritize long-term, multi-generational studies that combine controlled laboratory experiments with comprehensive field investigations. This approach would allow for a better understanding of how fluctuating environmental conditions, including temperature, pH, organic matter and redox states, influence mercury speciation and its bioavailability to *C. carpio* in natural settings. Such studies could also assess the cumulative effects of mercury in conjunction with other common aquatic pollutants.

Advanced Mechanistic Studies on Immune Modulation: Further research is needed to precisely characterize the dose- and time-dependent effects of mercuric chloride on the *Cyprinus carpio* immune system. This could involve investigating specific immune cell populations, cytokine profiles and the expression of genes related to immune response, to differentiate between immune activation and immunosuppression and their implications for disease resistance.

Comprehensive Reproductive Toxicology Assessments: Detailed studies focusing on male reproductive health, including sperm quality, motility, viability and fertilization success following $HgCl_2$ exposure, are crucial. Investigations into transgenerational epigenetic effects of mercury on *C. carpio* reproduction and offspring viability would also provide valuable insights into long-term population impacts.

Development and Validation of Novel Biomarkers: While hematological and histopathological parameters are established biomarkers, future research could explore more sensitive and specific molecular biomarkers for early detection of mercury stress. This includes further investigation into specific protein expression patterns, microRNA changes and advanced genomic and proteomic analyses that could indicate toxicity before overt physiological symptoms appear.

Efficacy of Mitigation Strategies in Real-World Scenarios: While laboratory studies show promise for protective compounds like selenium, vitamins and thymol, their effectiveness needs to be validated in larger-scale, environmentally relevant conditions. Research should focus on optimal dosages, delivery methods and the long-term impacts of these interventions on *C. carpio* health and population dynamics in aquaculture and contaminated natural environments.

Interactions with Other Stressors: Given the complexity of aquatic environments, future studies should investigate the synergistic or antagonistic effects of mercuric chloride when *C. carpio* is simultaneously exposed to other common environmental stressors, such as microplastics, emerging contaminants, or climate change-induced temperature fluctuations.

XII. CONCLUSION

The structured literature review on the effects of mercuric chloride on *Cyprinus carpio* reveals a consistent and profound toxicological impact across multiple biological organizational levels, from molecular mechanisms to whole-organism physiology and population dynamics. Mercuric chloride, while an inorganic form, poses a significant threat due to its potential transformation into highly toxic methylmercury within aquatic environments, a process influenced by complex environmental factors such as microbial activity, temperature, pH, organic matter, redox conditions and salinity.

Cyprinus carpio's remarkable adaptability and its role in human consumption underscore its importance as a bioindicator and a potential vector for human exposure, particularly as mercury bioaccumulates in its edible tissues, often exceeding safe limits. The review demonstrates that $HgCl_2$ exposure induces a spectrum of adverse effects in *C. carpio*, including significant behavioral changes indicative of neurological and respiratory distress, such as altered swimming patterns, loss of equilibrium and changes in body coloration.

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Physiologically, it leads to impaired growth and increased mortality, with larvae being particularly vulnerable during critical developmental windows. Hematologically, a consistent pattern of anemia (decreased red blood cell parameters) and an activated, yet potentially compromised, immune response (increased white blood cell count) are observed, reflecting systemic stress and impaired oxygen transport. At the tissue level, widespread histopathological damage occurs in vital organs, including severe degeneration and necrosis in the liver, kidney, gills, ovary, olfactory epithelium and intestine, leading to functional impairment.

At the cellular and molecular levels, mercury induces pervasive DNA damage, chromosomal instability and oxidative stress through the generation of reactive oxygen species and inhibition of antioxidant enzymes. It also disrupts crucial enzyme activities and activates stress-response signaling pathways, further compromising cellular function. Furthermore, $HgCl_2$ exerts significant reproductive and developmental toxicity, causing severe embryonic malformations and impairing gonad development, which collectively threaten the long-term viability and recruitment of *C. carpio* populations.

While some progress has been made in identifying potential mitigation strategies, such as dietary supplementation with antioxidants, significant knowledge gaps persist, particularly regarding the long-term, integrated effects in natural environments and the complex interplay of mercury with other environmental stressors. Future research should prioritize comprehensive, multi-disciplinary studies that bridge laboratory findings with ecological realities, focusing on advanced mechanistic understanding, robust biomarker development and the validation of effective remediation strategies. A deeper understanding of these complex interactions is paramount for developing effective environmental policies and management practices to protect aquatic ecosystems and safeguard human health from the pervasive threat of mercury contamination.

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