



Ameliorative Effects of Quercetin on HgCl₂-Induced Liver and Kidney Dysfunction in *Labeo rohita*

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ABSTRACT

*Mercuric chloride (HgCl₂) is a prevalent environmental pollutant that can induce severe hepatorenal toxicity in aquatic organisms. This study aimed to evaluate the impact of HgCl₂ exposure on liver and kidney function in *Labeo rohita* and also assessed the protective role of Quercetin (Que). Fish were subjected to sub-lethal concentration of HgCl₂ (0.17 mg/L; 1/5th of LC50) for 15 days. Concurrently the fish were administered quercetin-supplemented diets (400 mg/kg) for same duration. SGOT, SGPT, ALP, ACP, urea, and creatinine, the serum biochemical indicators related to liver and kidney function, were analysed. HgCl₂ exposure resulted in significant rise in all assessed liver and kidney parameters, indicating pronounced hepatotoxicity and nephrotoxicity. The co-administration of Que effectively attenuated these effects, restoring the biochemical indices toward normal levels, highlighting its protective efficacy. These results emphasize the susceptibility of fish to HgCl₂-induced organ damage and suggest Que as a promising natural therapeutic agent in aquaculture practices.*

Keywords: Mercuric chloride, *Labeo rohita*, Hepatotoxicity, Nephrotoxicity, Quercetin, Liver and kidney function

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INTRODUCTION

Industrial development has led to release of various hazardous substances, including heavy metals, into local water bodies, often in untreated forms. This results in the deterioration of the physicochemical quality of aquatic environments [1]. Heavy metals can exert a wide range of physiological effects on aquatic organisms, with bioaccumulation patterns varying among different species [2]. Mercury (Hg) is recognized as one of the most toxic heavy metals in aquatic environments. It enters freshwater systems mainly through atmospheric deposition and agricultural waste containing pesticides [3]. Among its various forms including elemental (Hg²⁺), organic (methylmercury, MeHg), and inorganic (mercuric chloride, HgCl₂), HgCl₂ is the most prevalent in industrial effluents and aquatic habitats [4]. While MeHg is highly neurotoxic, HgCl₂ is capable of crossing biological membranes, forming organo-mercury complexes, and disrupting critical biochemical and physiological processes in fish [5].

Fish are particularly vulnerable to Hg accumulation due to their constant interaction with the aquatic environment. Hg is absorbed through gill membranes, skin, and gastrointestinal tract, bioaccumulates in tissues, and biomagnifies along the trophic levels [6]. Among the various forms, methylmercury is especially toxic due to its high lipid solubility, which facilitates rapid uptake and penetration into vital organs, including the brain, liver, and kidneys [7].

The toxic mechanism of Hg in fish involves multiple biochemical and physiological disruptions [8]. Furthermore, Hg binds to thiol groups in proteins and enzymes, inhibiting their normal function and disrupting metabolic processes. Such interference impairs critical physiological activities, including osmoregulation, respiration, reproduction, and immune response [9,10].

In addition to systemic toxicity, Hg exerts pronounced histopathological effects on vital organs, particularly the liver and kidneys. The liver, as the principal site of detoxification and metabolism, often exhibits hepatocellular vacuolation, necrosis, and inflammatory infiltration following mercury exposure [11]. Similarly, the kidneys display tubular degeneration, glomerular shrinkage, and compromised excretory function [12]. These pathological alterations are typically associated with elevated levels of serum biochemical markers such as glutamic oxaloacetic transaminase (SGOT), glutamic pyruvic transaminase

(SGPT), alkaline phosphatase (ALP), acid phosphatase (ACP), urea and creatinine, reflecting hepatic and renal functional impairment [13].

Given these toxicological consequences, there is growing interest in identifying natural compounds capable of alleviating mercury-induced damage. Quercetin, a polyphenolic flavonoid abundantly found in fruits, vegetables, and medicinal plants, has gained attention due to its potent antioxidant, anti-inflammatory, and metal-chelating properties [14]. It is known to scavenge reactive oxygen species (ROS), stabilize cellular membranes, and modulate signaling pathways associated with oxidative stress. Several studies have demonstrated the protective role of quercetin in mammals and certain aquatic organisms under toxicant-induced stress [15,16] though its efficacy against mercuric chloride (HgCl₂)-induced toxicity in freshwater fish remains inadequately explored [12]

Labeo rohita (rohu), a commercially valuable freshwater fish widely cultured across South Asia, serves as an ideal model for evaluating heavy-metal toxicity and antioxidant-mediated protection [17]. Understanding its physiological and histological responses to Hg exposure, and potential recovery through dietary quercetin supplementation, holds significant implications for sustainable aquaculture and food safety [18]. The inclusion of quercetin as a natural dietary antioxidant may represent an eco-friendly and cost-effective strategy to mitigate the sublethal impacts of mercury contamination in aquaculture systems [19].

Despite the established toxicological profile of Hg in aquatic organisms, there is a paucity of comprehensive studies that simultaneously examine the behavioural, biochemical, and histopathological effects of HgCl₂ exposure in *Labeo rohita*, particularly over short-term periods. Moreover, the role of quercetin as a dietary intervention against such toxicity has not been well-characterized in this species. Most available studies have focused on chronic exposures or single-parameter assessments, leaving a gap in our understanding of how early-stage toxicity can be alleviated through natural antioxidants.

This study aims to evaluate the short-term effects of HgCl₂ exposure on *Labeo rohita*, focusing on liver and kidney function markers. It further investigates the ameliorative potential of dietary quercetin in mitigating these toxic effects, thereby contributing to the development of preventive strategies in aquaculture health management.

MATERIAL AND METHODS

Ethics statement

The care and management of fish followed the guiding principles approved by the Animal Ethics Committee of Maharaja Agrasen University (IEC/MAU/2023/08).

Experimental Fish and Acclimatization

Healthy fingerlings of *Labeo rohita*, with an average weight of 29.43 ± 0.4 g and length of 15.95 ± 0.9 cm, were obtained from Government Fish Seed Farm in Nalagarh, Himachal Pradesh, India. Prior to initiation of experimental procedures, fish were acclimatized for 15 days in dechlorinated water under laboratory conditions. The physicochemical parameters of test water during acclimatization were maintained as follows: temperature at 25 ± 0.5 °C, dissolved oxygen concentration at 9.0 ± 0.30 mg/L, pH ranging from 7.0 to 7.2, and water hardness measured at 93.06 ± 3.0 mg/L as CaCO₃. During acclimation period, fish were fed a basal diet twice daily, with feed quantity adjusted to 3% of their body weight.

Mercuric chloride (HgCl₂) and Quercetin dietary supplement

Technical grade, anhydrous mercuric chloride (HgCl₂; HiMedia Laboratories Pvt. Ltd., India), characterized as an odorless white crystalline solid with a molecular weight of 271.52 g/mol, was employed to induce mercury exposure. A sublethal concentration of HgCl₂ (0.17 mg/L, corresponding to one-fifth of the LC₅₀) was selected based on the LC₅₀ value previously determined in our earlier research [12].

Quercetin (purity ≥ 98%, HiMedia Laboratories Pvt. Ltd., India) was incorporated into the basal diet at a concentration of 400 mg/kg, following the procedure outlined in our prior study [12]

Experimental Design

For experimental trial, four groups of fishes were maintained in glass aquaria: group I (Control, basal diet), group II (0.17 mg/L HgCl₂-exposed), group III (Quercetin-supplemented, 400 mg/kg diet), and group IV (HgCl₂ 0.17 mg/L + Quercetin 400 mg/kg diet). The experiment was conducted for 15 days following Twenty-four hours renewal bio assay method, and each experiment was repeated three times to ensure the reproducibility. After the completion of exposure, liver and kidney tissues were collected from each group (n = 6).

Biochemical parameters

Blood serum obtained from the fish was allowed to clot for 1 hr at room temperature and centrifuged at 3,000 rpm for 10 min further analysed for hepatic and renal function markers. Liver function was evaluated by estimating serum glutamate pyruvate transaminase (SGPT) [20], serum glutamate oxaloacetate transaminase (SGOT) [21], alkaline phosphatase (ALP) [22], and acid phosphatase (ACP) [23] levels. Kidney

function was assessed by measuring serum urea [24] and creatinine [25]. All analyses were performed spectrophotometrically using a UV-VIS spectrophotometer (LABTRONICS LT- 2700), and results were expressed in IU/L for enzymes and mg/dL for metabolites.

Statistical analysis

The analysis of data was done with SPSS software (version 21.0) where the Kolmogorov-Smirnov test was used for establishing normal distribution for biochemical analysis. The data was then tested for significant differences with ANOVA (one-way analysis of variance) followed by Tukey's post hoc between different groups. Results are expressed in the form of mean \pm SD showing significance at $p < 0.05$.

RESULTS

The activities of liver enzymes SGOT, SGPT, ACP and ALP varied significantly across the experimental groups. Fish exposed to HgCl₂ led to a significant increase ($p < 0.05$) in SGOT, SGPT, ALP and ACP activities compared to control group, indicating substantial liver damage caused by HgCl₂ exposure. Following 15 days of HgCl₂ exposure, fish exhibited marked elevations in hepatic enzyme markers - SGOT levels increased by 47% ($p < 0.05$), while SGPT levels rose by 34% ($p < 0.05$). Similarly, ACP and ALP activities showed significant increases of 43% and 31%, respectively ($p < 0.05$). The Que-only group displayed no significant differences compared to the control ($p > 0.05$). In the combined HgCl₂ + Que group, SGOT, SGPT, ACP, and ALP levels showed moderate increases of 16.2%, 8.8%, 7.7%, and 14.2%, respectively, relative to the control ($p < 0.05$) (Fig.1a-d). Therefore, it is evident that co-treatment with Que markedly ameliorated HgCl₂-induced alterations, restoring SGOT by 65.8%, SGPT by 74.9%, ACP by 82.2%, and ALP by 54.4% when compared with the HgCl₂-exposed group.

HgCl₂ exposure caused a significant elevation in renal markers, with both urea and creatinine showing marked increases compared to the control group. Fish exposed to HgCl₂ exhibited a 51.5% rise in serum urea and a 53.1% rise in creatinine levels ($p < 0.05$). The Que-only group displayed no significant changes. In the HgCl₂ + Que group, urea and creatinine values remained slightly higher than the control—by 15.1% and 13.7%, respectively ($p < 0.05$)—but were substantially lower than those observed under HgCl₂ exposure alone (Fig. 2a–b). When compared directly with the HgCl₂ group, co-administration of Que reduced urea levels by 70.7% and creatinine by 74.3%, demonstrating strong protective efficacy.

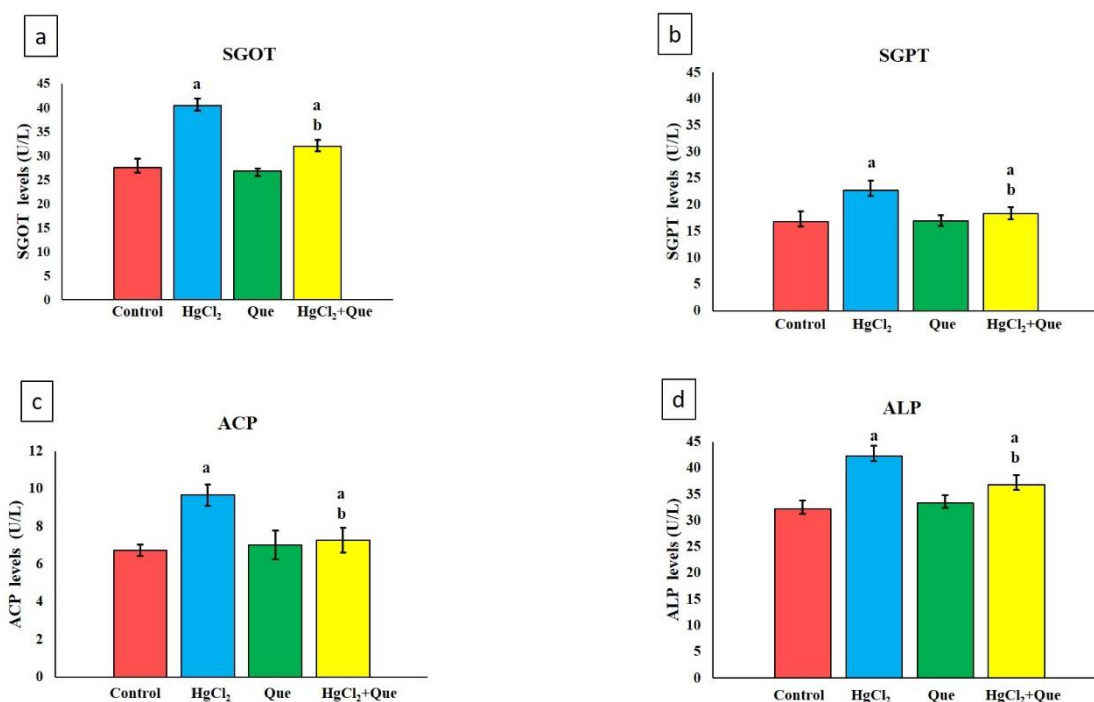


Figure:1 Effects of HgCl₂ and Que supplementation on hepatic enzyme activities in *Labeo rohita* after 15 days of exposure. (a) Serum glutamate oxaloacetate transaminase (SGOT), (b) serum glutamate pyruvate transaminase (SGPT), (c) acid phosphatase (ACP), and (d) alkaline phosphatase (ALP). Values are expressed as mean \pm SD (n=6). Bars sharing different letters indicate significant differences ($p < 0.05$) among groups, determined by one way ANOVA followed by Tukey's post hoc test. 'a' denotes significant difference compared to the control group, while 'b' indicates a significant difference between HgCl₂ and HgCl₂ + Que groups.

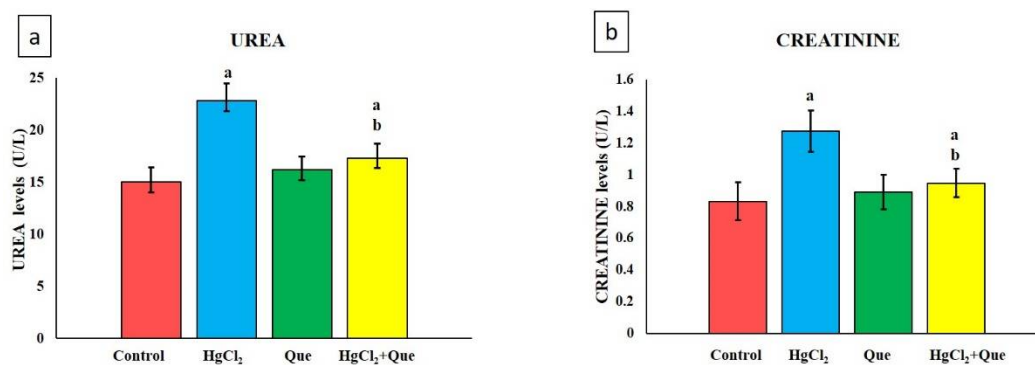


Figure:2 Effects of HgCl₂ and Que supplementation on renal function biomarkers in *Labeo rohita* after 15 days of exposure. (a) Urea and (b) creatinine levels in serum. Values are expressed as mean ± SD (n = 6). Bars with different letters indicate significant differences ($p < 0.05$) among treatment groups, determined by one way ANOVA followed by Tukey's post hoc test. 'a' denotes a significant difference compared to the control group, while 'b' indicates a significant difference between HgCl₂ and HgCl₂ + Que groups.

DISCUSSION

The liver and kidney are the primary detoxification and regulatory organs in fish, liver metabolizes nutrients and neutralizes toxicants, whereas kidney filters waste, regulates ions, and maintains internal homeostasis. Due to their direct involvement in process of circulating contaminants, both organs are highly vulnerable to heavy-metal exposure and demonstrate early biochemical alteration under toxic stress condition [26]. The present study demonstrates that sub-chronic exposure to HgCl₂ induces pronounced hepato-renal dysfunction in *Labeo rohita*, as evidenced by significant alterations in serum biochemical markers. As Hg is a well-established toxicant known to impair cellular integrity through oxidative stress, enzyme inhibition and disruption of metabolic pathways [27] the marked elevation in SGOT, SGPT, ALP, and ACP activities observed in HgCl₂-treated fish reflects substantial hepatocellular damage and compromised membrane stability. The increase in SGOT and SGPT by 47% and 34%, respectively, suggest leakage of intracellular enzymes into the circulation, a common indicator of hepatotoxicity [28]. Similarly, the elevated ACP and ALP levels imply impaired hepatic function, lysosomal destabilization, and possible biliary obstruction. These biochemical changes were consistent with heavy-metal-driven oxidative stress, mitochondrial dysfunction, and activation of apoptotic pathways reported in recent fish studies on mercury toxicity [29]. Our results align with Alam et al (2021) who reported increased serum AST, ALT, and ALP levels in *O. niloticus* exposed to methyl mercury chloride [30]. In *Channa punctata*, a recent study demonstrated that sublethal exposure to HgCl₂ (0.039 and 0.078 mg/L) over 15–45 days significantly increased serum SGOT and SGPT [31]. Similarly, in *Heteropneustes fossilis*, HgCl₂ exposure (0.15 mg/L) produced elevated ALP and transaminases (AST/ALT) in the liver [32]. In contrast, dietary supplementation with quercetin significantly ameliorated the biochemical disruptions induced by HgCl₂. The Que-only group remained comparable to the control, confirming its non-toxic nature at the administered dose. Co-administration of Que markedly improved hepatic and renal markers, restoring enzyme activities toward normal levels. In *Nile tilapia*, quercetin supplementation reduced SGPT, SGOT, ALP indicating strong hepato-renal protection [14].

Creatinine is an important indicator of kidney health and reflects efficacy of removal of nitrogenous wastes from body [33]. In present study, fish exposed to HgCl₂ showed a clear rise in creatinine levels, suggesting that mercury stress impaired renal function and reduced the ability to eliminate metabolic waste [34]. However, fish that received Que in their diet-maintained creatinine values similar to the control group. Notably, the HgCl₂ + Que group showed a marked improvement, indicating that Que helped counteract harmful effects of HgCl₂ and supported normal kidney function. Urea, another major nitrogenous waste product formed during protein metabolism, followed a similar pattern [35]. Urea levels increased significantly in the HgCl₂-exposed fish, reflecting disrupted nitrogen metabolism and further evidence of nephrotoxicity [36]. In contrast, fish fed with the quercetin-supplemented diet showed no significant change in urea concentration. The reduction in urea levels in the HgCl₂ + Que group compared with the HgCl₂ group further demonstrates the protective influence of quercetin in maintaining metabolic balance under mercury stress. These results are in line with the work of Shin et al. (2015), who reported that quercetin significantly reduced HgCl₂-induced elevations in serum creatinine and urea and prevented renal tubular damage in experimental models [37]. Furthermore, recent evidence shows that quercetin also suppresses inflammation and autophagy pathways activated by mercury, thereby preserving kidney

function and metabolic homeostasis [38]. Similar improvements in liver and kidney biochemical parameters have been observed in other fish species exposed to heavy metals when Que was included in the diet [39]. These consistent findings across models highlight Que ability to ameliorate HgCl₂-induced alterations in liver and kidney enzymes.

Conclusion

HgCl₂ exposure induces clear hepato-renal toxicity in *Labeo rohita*, evidenced by significant increases in serum transaminases (SGOT, SGPT), phosphatases (ALP, ACP), and renal markers (urea, creatinine). Que supplementation effectively mitigates these toxic effects, demonstrating its hepatoprotective and nephroprotective potential. This study underscores the risk posed by mercury contamination to fish health and highlights quercetin as a promising natural intervention to preserve liver and kidney function in aquaculture species.

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Conflict of Interest

There is no conflict of Interest

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