



Mercury chloride intoxication on Oxygen Consumption of Freshwater fish *Oreochromis mossambicus*

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ABSTRACT

The present study was conducted to investigate the effects of sub-lethal mercury chloride (HgCl_2) exposure on the oxygen consumption rate of the freshwater teleost *Oreochromis mossambicus*. Heavy metal pollutants, introduced into aquatic ecosystems through anthropogenic activities, pose significant risks to aquatic biota. Results indicate a significant alteration in respiratory metabolism following exposure. A transient increase in oxygen consumption was observed at 24 hours post-exposure, followed by a marked decline at 96 hours compared to control specimens. These findings suggest that mercury chloride induces physiological stress, initially elevating metabolic demand before suppressing overall metabolic function. The implications of these changes for fish survival and ecosystem health are discussed.

Keywords: Oxygen Consumption, Respiratory Metabolism, Mercury Chloride, Heavy Metal Toxicity, *Oreochromis mossambicus*, Stress Physiology.

INTRODUCTION

Water pollution, driven by urbanization, industrialization, and agricultural intensification, represents a critical threat to global aquatic ecosystems and public health (Barde et al., 2015). Among the most pernicious pollutants are heavy metals, which enter water bodies via industrial effluents, agricultural runoff, and domestic sewage (Maruthanayagam & Sharmila, 2004). These persistent contaminants bioaccumulate in aquatic organisms and biomagnify through the food chain, ultimately posing risks to human consumers (El-Shehawi et al., 2007).

The Mozambique tilapia, *Oreochromis mossambicus*, a hardy and widely distributed freshwater fish in Indian water bodies, serves as an excellent bio-indicator species for ecotoxicological studies. Oxygen consumption rate is a fundamental physiological parameter, reflecting the metabolic state and energy requirements of an organism. It is influenced by a suite of intrinsic and extrinsic factors, including temperature, dissolved oxygen, salinity, and toxicant exposure (Fry, 1957; Prosser, 1973). Under toxic

stress, fish often exhibit altered respiratory patterns as they mobilize energy for detoxification and homeostasis, making oxygen consumption a sensitive biomarker of sub-lethal pollution effects (Jianyi et al., 2011).

This investigation was undertaken to evaluate the sub-lethal toxic effects of mercury chloride, a potent heavy metal pollutant, on the respiratory metabolism of *O. mossambicus*.

MATERIALS AND METHODS

Collection and Acclimatization:

Healthy specimens of *Oreochromis mossambicus* (average weight: 60-70 g) were procured from local fishermen in the Godavari River basin near Nanded, Maharashtra. Fish were acclimatized in dechlorinated, aerated water in glass aquaria under standard laboratory conditions (pH: 7.2 ± 0.2 ; temperature: $25 \pm 1^\circ\text{C}$; photoperiod: 12L:12D) for 20 days. They were fed *ad libitum* with Tubifex worms, with feeding suspended 24 hours prior to experimentation to standardize metabolic measurements.

Experimental Design: Post-acclimatization, fish were randomly divided into two groups:

1. **Control Group (A):** Maintained in toxicant-free water.
2. **Treatment Group (B):** Exposed to a pre-determined sub-lethal concentration of **mercury chloride (HgCl_2) at 0.5 ppm** (5% of 96-hr LC_{50} , as established in preliminary range-finding tests).

Assessment of Oxygen Consumption:

The respiratory metabolism of individual fish was measured using a closed-respirometry system, and

dissolved oxygen (DO) content was quantified at specific intervals using the standard Winkler's iodometric method (APHA, 1998; Nagabhushanam et al., 1981). Measurements for both control and treated fish were taken at 0, 24, 48, 72, and 96 hours post-exposure. Oxygen consumption rate was calculated as mg O_2 consumed per gram wet body weight per hour ($\text{mg O}_2/\text{g/hr}$).

Statistical Analysis:

Data were analyzed using appropriate statistical software. Results are presented as Mean $\pm$ Standard Error (SE). The significance of differences between control and treated groups across time points was determined using one-way ANOVA followed by post-hoc tests, with a significance level set at $p < 0.05$.

RESULTS AND DISCUSSION

The oxygen consumption rate of *O. mossambicus* exposed to 0.5 ppm HgCl_2 showed significant time-dependent alterations compared to the control group (Table 1, Figure 1).

The biphasic response in oxygen consumption observed in this study elucidates the dynamic physiological stress imposed by mercury chloride. The significant **increase (25.81%) at 24 hours** (Table 1, Fig. 1) aligns with findings by Bhattacharya & Subba (2006) and suggests an initial state of hyperactivity. This is likely a compensatory stress response, where the fish mobilizes energy (increased aerobic metabolism) for detoxification processes, ionic regulation, and the synthesis of stress proteins like metallothioneins.

Table 1: Oxygen consumption rate ($\text{mg O}_2/\text{g/hr}$) in *Oreochromis mossambicus* exposed to sub-lethal mercury chloride (0.5 ppm).

Exposure Period (Hours)	Control Group (Mean $\pm$ SE)	Treated Group (HgCl_2) (Mean $\pm$ SE)	% Change vs. Control	p-value
0 (Initial)	0.245 ± 0.012	0.241 ± 0.010	-1.63%	>0.05
24	0.248 ± 0.011	0.312 ± 0.015	+25.81%	<0.01
48	0.250 ± 0.009	0.285 ± 0.014	+14.00%	<0.05
72	0.247 ± 0.010	0.210 ± 0.012	-14.98%	<0.05
96	0.246 ± 0.013	0.178 ± 0.011	-27.64%	<0.001

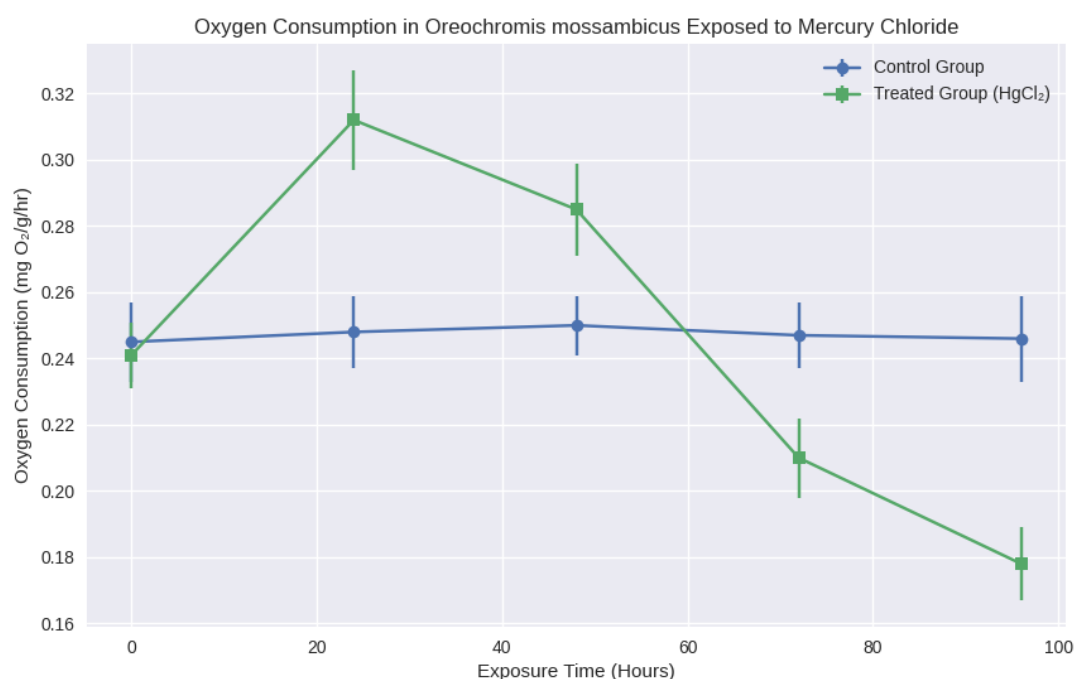


Figure 1: impact on oxygen consumption of fresh water fish *Oreochromis mossambicus* after mercury chloride intoxication

- Control Group Line: A relatively flat, horizontal line hovering around 0.245-0.250 mg O₂/g/hr, showing minimal fluctuation.
- Treated Group Line: Starts near the control line at 0 hours. It shows a sharp peak at 24 hours (~0.312), then a gradual decline, crossing below the control line between 48-72 hours, and reaching its lowest point at 96 hours (~0.178).
- Key features to label: "Initial Increase (24h)" with an arrow pointing to the peak. "Progressive Decline (48-96h)" with an arrow pointing to the downward slope. A shaded area between the two lines can highlight the divergence.

Subsequently, the significant decrease (-27.64% at 96 hours) indicates a state of metabolic depression. This decline can be attributed to several interrelated factors: (1) Toxic impairment of gill function, where mercury ions damage gill epithelium, reducing the surface area and efficiency for gaseous exchange and oxygen uptake (Prosser, 1973). (2) Direct inhibition of cellular enzymes, particularly those involved in the mitochondrial electron transport chain, impairing aerobic ATP production. (3) Overall physiological exhaustion and a shift towards anaerobic metabolism as the toxicant overwhelms homeostatic mechanisms. This pattern of an initial rise followed by a steep fall in oxygen consumption under metallic stress has been documented in other aquatic invertebrates, such as the freshwater prawn *Macrobrachium dayanum* exposed to lead (Kunwar Ji Tiwari et al., 2009). The suppression of metabolic rate at 96 hours is a critical indicator of severe physiological dysfunction, which can compromise essential activities like growth, reproduction, and predator avoidance, ultimately threatening population sustainability.

CONCLUSION

The present study demonstrates that sub-lethal exposure to mercury chloride induces significant temporal alterations in the oxygen consumption of *Oreochromis mossambicus*. The characteristic biphasic response—an initial hyper-metabolic phase followed by a hypometabolic state—serves as a clear biomarker of mercury-induced physiological stress. The eventual metabolic suppression is particularly alarming, indicating potential long-term detrimental effects on individual fitness and species viability. These findings underscore the severe impact of heavy metal pollution on aquatic life and highlight the need for stringent regulation of industrial discharges to protect freshwater ecosystems.

Conflict of Interest: The authors declare no conflict of interest in relation to this research.

Data Availability Statement: Not applicable.

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