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Effect of Lethal and Sublethal Exposure of Tebuconazole (25.9% EC) on Oxygen Consumption in the Freshwater Fish *Ctenopharyngodon idella* as a Respiratory Biomarker

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Abstract

Pesticide contamination of freshwater ecosystems has a negative impact on non-target aquatic animals by disrupting physiological and metabolic processes. Oxygen consumption is a sensitive physiological metric that may be used to detect metabolic stress in fish exposed to toxicants. The current study assessed the impact of deadly (96 h LC_{50}) and sub-lethal (1/10th LC_{50}) tebuconazole 25.9% EC concentrations on oxygen consumption in the freshwater fish Ctenopharyngodon idella (Valenciennes). The fish were exposed for 24 hours, with oxygen consumption monitored every 2 hours. Both deadly and sub-lethal exposure groups saw an initial rise in oxygen demand, followed by a steady drop. The amount of the change was concentration-dependent, with more significant effects reported at fatal levels.

The results clearly indicates that tebuconazole induces the respiratory stress in *C. idella*, and oxygen consumption can be effectively used as a biomarker of pesticide toxicity in freshwater fishes.

Keywords: Tebuconazole 25.9% EC; *Ctenopharyngodon idella*; oxygen consumption; LC_{50}; pesticide toxicity

Introduction

Oxygen consumption plays a significant role in fish physiology and is commonly used to measure metabolic rate. It indicates fish's total physiological and energetic status and is affected by elements such as body size, temperature, activity, nutritional status, and environmental quality (Job, 1955; Golterman et al., 1978). Any variation in oxygen uptake often implies a metabolic imbalance and physiological stress.

Fish get oxygen directly from the surrounding water via their gills, making them especially sensitive to aquatic pollution. Toxicants that enter freshwater systems can directly damage respiratory surfaces and affect gas exchange, creating changes in metabolic and respiratory functioning (David et al., 2003; Tilak et al., 2007). As a result, oxygen consumption is often

considered as a sensitive physiological stress indicator in pesticide-exposed fish (Magare and Patil, 2000; Patil and David, 2008).

Several studies have shown that pesticides classified as organophosphates, organochlorines, carbamates, or pyrethroids dramatically modify oxygen consumption in freshwater fish. Pesticide exposure often results in an initial increase in oxygen consumption followed by a reduction, indicating adaptive stress responses followed by metabolic depletion. Similar trends were seen in Labeo rohita exposed to Ethion (Prasanna et al., 2020) and in Channa punctatus exposed to dimethoate (Chandra Shekhar et al., 2025).

Tebuconazole is a systemic triazole fungicide widely used in agriculture and commonly found in aquatic ecosystems owing to surface runoff. Recent studies have found physiological, biochemical, and metabolic abnormalities in fish exposed to tebuconazole. However, there is minimal evidence on its influence on respiratory metabolism, particularly oxygen consumption, in Ctenopharyngodon idella. Therefore, the present investigation was carried out to investigate the influence of fatal and sub-lethal doses of tebuconazole 25.9% EC on oxygen consumption in C. idella.

Materials and Methods

Experimental Fish

Healthy fingerlings of Ctenopharyngodon idella were obtained from a nearby fish farm and acclimatized to laboratory conditions in well-aerated, dechlorinated water for seven days. Fish were fed freely throughout acclimation. To eliminate metabolic fluctuation caused by digestion, feeding was halted 48 hours before the experiment (Rao and Mane, 1978).

Test Chemical

Commercial-grade tebuconazole 25.9% EC was used as the test toxicant. The lethal concentration was based on the experimentally determined 96 h LC_{50} value, while the sub-lethal concentration was taken as one-tenth of the 96 h LC_{50}.

Experimental Design

Fish were divided into three groups: control, lethal concentration, and sub-lethal concentration. Each group was maintained under identical laboratory conditions. Oxygen consumption was measured for 24 h at 2 h intervals using a standard respirometric method described by Job (1955) and Golterman et al. (1978). Oxygen consumption was expressed as mg O_2/g body weight/hour.

Determination of 96 h LC_{50}

Acute toxicity studies were performed after a static renewal bioassay. Fish were subjected to various doses of tebuconazole, and mortality was measured at 24, 48, 72, and 96 hours. The 96-hour LC_{50} value was computed using Finney's probit analysis approach.Acute toxicity studies were performed after a static renewal bioassay. Fish were subjected to various doses of tebuconazole, and mortality was measured at 24, 48, 72, and 96 hours. The 96-hour LC_{50} value was computed using Finney's probit analysis approach.

Statistical Analysis

Data were expressed as mean ± standard deviation (SD). Percentage change in oxygen consumption was calculated with respect to control values. Differences were considered statistically significant at $p < 0.05$.

Table 1

The amount of oxygen consumed (mg/g body weight/hour) of the fish *Ctenopharyngodon idella* exposed to lethal and sub-lethal concentrations of tebuconazole 25.9% EC

Exposed period (h)	Control (Mean ± SD)	Lethal – LC_{50} (Mean ± SD)	% Change	Sub-lethal (Mean ± SD)	% Change
0	0.823 ± 0.004	0.816 ± 0.003	0.85	0.820 ± 0.004	0.36
2	0.782 ± 0.003	0.836 ± 0.004	−6.90	0.812 ± 0.003	−3.83
4	0.781 ± 0.002	0.899 ± 0.004	−15.10	0.862 ± 0.003	−10.37
6	0.788± 0.003	0.939 ± 0.005	−19.16	0.894 ± 0.004	−13.45
8	0.843 ± 0.004	0.881 ± 0.004	−4.50	0.852 ± 0.003	−1.06
10	0.732 ± 0.003	0.750 ± 0.004	−2.45	0.723 ± 0.003	1.22
12	0.713± 0.003	0.671 ± 0.004	5.89	0.642 ± 0.004	9.95
14	0.657± 0.004	0.621 ± 0.004	5.47	0.632± 0.005	3.80
16	0.644 ± 0.004	0.559 ± 0.005	13.19	0.584 ± 0.004	9.31
18	0.617 ± 0.005	0.489 ± 0.004	20.74	0.519 ± 0.004	15.88
20	0.578± 0.004	0.448 ± 0.005	22.49	0.473 ± 0.004	18.16
22	0.557 ± 0.005	0.412 ± 0.006	26.03	0.446 ± 0.005	19.92
24	0.514 ± 0.004	0.369 ± 0.005	28.21	0.403 ± 0.004	21.59

Table 2: Summary of exposure concentrations used for oxygen consumption studies

Treatment	Concentration (mg L^{-1})	Basis
Control	0.00	Untreated groundwater
Sub-lethal	2.1	1/10 of 96 h LC_{50}
Lethal	21.0	96 h LC_{50}

O2 consumed by fish / gram body weight / hour =

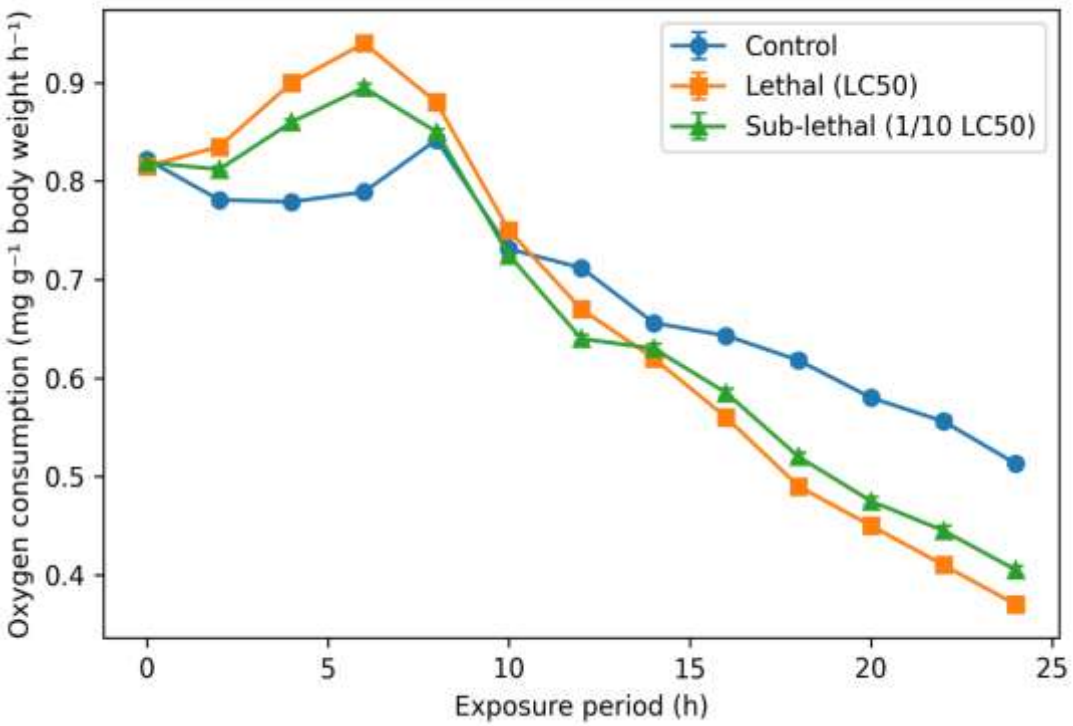
α - β x N of hypo x 8 x 1000

Vol. of the sample x Correction factor x Wt. of the fish x Time interval for sample

α = hypo rundown before exposure

β = hypo rundown after exposure

Student,s t-test was employed to calculate the significance of the differences between control and experimental means. P values of 0.05 or less were considered statistically significant (Fisher, 1950).



Results and Discussion

The oxygen consumption pattern of *Ctenopharyngodon idella* exposed to lethal and sub-lethal concentrations of tebuconazole 25.9% EC is presented in **Table 1** and illustrated in **Figure 1**.

In the control group, oxygen consumption gradually declined throughout the experimental period. This reduction can be attributed to fasting, reduced locomotor activity, and metabolic adjustment under laboratory conditions, as commonly reported in fish respiration studies (Job, 1955; Rohankar and Kulkarni, 2005).

Fish exposed to the sub-lethal concentration exhibited a distinct stimulatory response during the early exposure period. Oxygen consumption increased significantly during the first 6 h of exposure, indicating enhanced metabolic demand and respiratory activity. This early elevation may be associated with stress-induced hyperactivity and increased opercular movements as an immediate physiological response to toxic stress. Similar early-phase increases in oxygen consumption have been reported in fishes exposed to pesticides such as ethion, dimethoate, and chlorpyrifos (Kalavathy et al., 2001; Prasanna et al., 2020; Sharma and Singh, 2019).

Following the initial stimulatory phase, oxygen consumption in the sub-lethal group declined progressively with increasing exposure duration. The marked reduction observed during later hours suggests the onset of metabolic depression and reduced respiratory efficiency due to prolonged toxic stress.

A comparable but more pronounced response was observed in fish exposed to the lethal concentration of tebuconazole. Oxygen consumption increased sharply during the initial exposure period, reaching a maximum at 6 h. The magnitude of this increase was significantly higher than that observed in the sub-lethal group, indicating greater physiological stress at higher toxicant concentrations.

With prolonged exposure, oxygen consumption in the lethal group declined rapidly, showing a substantial reduction by 24 h. This sharp decline suggests severe respiratory impairment and metabolic exhaustion, possibly due to toxicant-induced damage to respiratory surfaces and disruption of oxidative metabolism. Similar reductions in oxygen consumption under lethal pesticide exposure have been reported in *Labeo rohita*, *Channa punctatus*, and *Cyprinus carpio* (Veeraiah and Durga Prasad, 2001; David et al., 2002; Khan et al., 2021).

Overall, the observed biphasic response—initial stimulation followed by inhibition of oxygen consumption—is characteristic of pesticide-induced stress in fishes. The early increase reflects an adaptive response to acute stress, whereas the subsequent decline indicates failure of compensatory mechanisms during prolonged exposure (Tilak et al., 2007; Patil and David, 2008). The greater reduction observed at lethal concentration confirms the concentration-dependent effect of tebuconazole on respiratory metabolism.

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