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Toxic Effects of Phenthoate (50% EC) on Respiratory Metabolism and Oxygen Utilization in Ctenopharyngodon idella under Lethal and Sublethal Conditions

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ABSTRACT

The present study was undertaken to examine the effect of phenthoate (50% EC), an organophosphate pesticide, on the respiratory metabolism of the freshwater fish Ctenopharyngodon idella. Oxygen consumption was used as an indicator to evaluate the physiological stress caused by sublethal and lethal exposure over a period of 24 hours. At the beginning of the experiment (0 h), no noticeable variation in oxygen consumption was observed between the control and treated groups. In the sublethal exposure group, oxygen consumption showed an initial increase and reached a peak at 4 h (17.92%), followed by a gradual decline during the later stages of exposure. In the lethal exposure group, oxygen consumption decreased continuously throughout the experimental period, with the maximum reduction observed at 22 h (37.98%). The decline in oxygen consumption suggests respiratory stress and disturbance in normal metabolic activity due to phenthoate exposure. The findings of the present study indicate that phenthoate can significantly affect the respiratory physiology of Ctenopharyngodon idella, particularly under lethal exposure conditions. The study also suggests that changes in oxygen consumption may serve as a useful biomarker for assessing pesticide-induced stress in aquatic organisms.

Keywords: Phenthoate (50% EC), Ctenopharyngodon idella, oxygen consumption, respiratory metabolism, organophosphate pesticide, aquatic toxicology

INTRODUCTION

Oxygen consumption is one of the important physiological parameters used to understand metabolic activity in fish. It reflects the energy status and overall physiological condition of the organism. The rate of oxygen uptake is influenced by several factors such as body size, temperature, feeding condition, level of activity, and environmental quality (Job, 1955; Golterman et al., 1978). Any disturbance in oxygen consumption may indicate alterations in normal metabolic processes and is therefore considered a useful indicator of physiological stress. Several studies have reported that oxygen consumption can serve as a sensitive biomarker for evaluating sublethal toxicity and metabolic disturbances in fish exposed to environmental pollutants (Saha et al., 2022; Majumder et al., 2024).

Fish obtain oxygen directly from water through their gills, which function as the primary respiratory organ. Due to their continuous contact with the aquatic environment, fish are highly sensitive to waterborne pollutants. Toxic substances entering freshwater ecosystems can damage gill tissues, interfere with gas exchange, and affect normal respiratory efficiency (David et al., 2003; Tilak et al., 2007). Previous investigations have shown that pesticide exposure can alter gill ventilation and respiratory activity, resulting in physiological stress and impaired metabolism (Qayoom et al., 2024; Cabrera et al., 2024). The increasing presence of pesticide residues in aquatic environments has also become a matter of ecological concern because of their harmful effects on aquatic organisms (Abdallah et al., 2024; Krishnan et al., 2025).

Oxygen consumption has therefore been widely used to assess the physiological stress caused by toxicants in fish (Magare and Patil, 2000; Patil and David, 2008). Exposure to pesticides often produces noticeable changes in respiratory activity, indicating disturbances in metabolic and respiratory functions (Mukherjee et al., 2025). In many cases, fish initially show an increase in oxygen consumption as an adaptive response to stress, followed by a gradual decline as exposure continues and metabolic exhaustion develops. Similar observations have been reported in Ctenopharyngodon idella and other freshwater fishes exposed to pesticides (Nabi et al., 2022; Shekhar, 2025).

Several studies have demonstrated that pesticides belonging to different chemical groups, including organophosphates, organochlorines, carbamates, and pyrethroids, can significantly affect respiratory metabolism in fish. These compounds are known to induce oxidative stress, neurotoxicity, and biochemical disturbances that impair normal metabolic functions (Amaeze et al., 2020; Sharma et al., 2019; Ghayyur et al., 2021). Prolonged exposure to pesticides may reduce oxygen consumption due to gill damage, reduced efficiency of gas exchange, and disruption of cellular respiration (Banaee et al., 2023; Qayoom et al., 2024). Similar responses have been observed in Labeo rohita exposed to ethion (Prasanna et al., 2020) and in Channa punctatus exposed to dimethoate (Chandra Shekhar et al., 2025).

Phenthoate (50% EC) is an organophosphate insecticide commonly used in agriculture. Due to its extensive use, residues of the pesticide may enter aquatic ecosystems through agricultural runoff and leaching, posing a potential threat to non-target aquatic organisms such as fish. Earlier studies on organophosphate pesticides have shown that these compounds can alter enzymatic activity, induce oxidative stress, and produce histopathological changes in fish tissues (Rahman et al., 2020; Banaee et al., 2023). They may also interfere with energy metabolism and respiratory functions, leading to physiological stress in exposed organisms (Mukherjee et al., 2025; Mustafa et al., 2024).

Although considerable work has been carried out on pesticide toxicity in fish, information regarding the effects of phenthoate on respiratory metabolism in Ctenopharyngodon idella is still limited. Therefore, the present study was undertaken to evaluate the effect of sublethal and lethal concentrations of phenthoate (50% EC) on oxygen consumption in Ctenopharyngodon idella. The study was aimed at understanding the respiratory stress responses produced by pesticide exposure and their possible impact on aquatic organisms.

MATERIALS AND METHOD

Experimental Fish

Healthy fingerlings of Ctenopharyngodon idella were collected from a nearby fish farm and transported to the laboratory in aerated containers. The fish were acclimatized for seven days in well-aerated dechlorinated water under laboratory conditions. During acclimatization, the fish were fed ad libitum to maintain normal physiological condition. Feeding was stopped 48 hours before the experiment in order to avoid the influence of feeding on metabolic activity (Rao and Mane, 1978).

Test Chemical

Phenthoate (50% EC), a commercial-grade organophosphate pesticide, was used as the test toxicant for the present study. A stock solution was prepared using distilled water, and the required concentrations

were obtained by suitable dilution. The lethal concentration was selected based on the experimentally determined 96 h LC_{50} value, while the sublethal concentration was taken as one-tenth of the 96 h LC_{50} value.

Experimental Design

The experimental fish were divided into three groups: control, sublethal exposure, and lethal exposure. Each group was maintained separately under identical laboratory conditions. The exposure period was carried out for 24 hours, and observations were recorded at intervals of 2 hours.

Oxygen consumption was estimated by adopting the standard closed respirometric method described by Job (1955) and Golterman et al. (1978). Individual fish were introduced into airtight containers containing a known volume of water, and dissolved oxygen levels were measured before and after each exposure interval. Oxygen consumption was calculated and expressed as mg O_2/g body weight/hour. Similar methods have also been employed in recent respiratory toxicity studies on Ctenopharyngodon idella exposed to pesticide stress (Bollu et al., 2026). (IJNRD)

Determination of 96 h LC_{50}

Acute toxicity studies were conducted using a static renewal bioassay method. Fish were exposed to different concentrations of phenthoate, and mortality was recorded at 24, 48, 72, and 96 hours. The LC_{50} values obtained were 4.4 ppm, 4.0 ppm, 3.6 ppm, and 3.2 ppm, respectively. The median lethal concentration for 96 hours was calculated using Finney's probit analysis method (Finney, 1971).

Statistical Analysis

The experimental data were expressed as mean ± standard deviation (SD). Percentage variation in oxygen consumption was calculated relative to the control group. Statistical significance between control and treated groups was analyzed using Student's t-test, and differences were considered significant at $p < 0.05$.

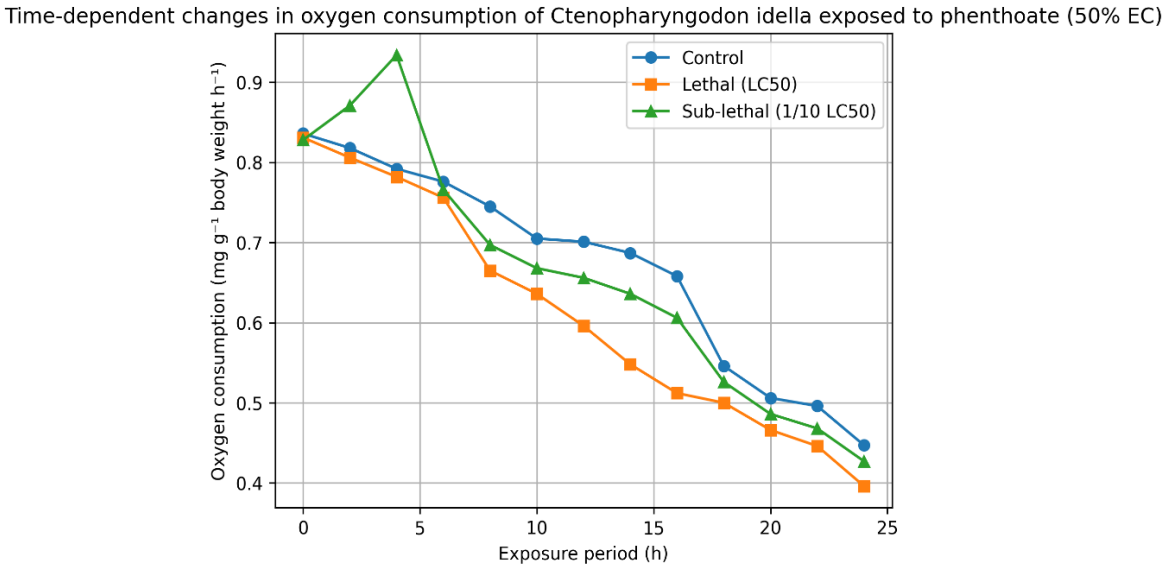
Table 1

Oxygen consumption (mg O_2/g body weight/hour) of *Ctenopharyngodon idella* exposed to lethal and sublethal concentrations of phenthoate (50% EC)

Time (h)	Control (Mean ± SD)	Sublethal (Mean ± SD)	% Change	Lethal (Mean ± SD)	% Change
0	0.836 ± 0.003	0.828 ± 0.004	↓0.95	0.831 ± 0.003	↓0.59
2	0.842 ± 0.002	0.879 ± 0.003	↑4.39	0.802 ± 0.004	↓4.75
4	0.792 ± 0.003	0.934 ± 0.003	↑17.92	0.781 ± 0.004	↓1.39
6	0.825 ± 0.004	0.812 ± 0.003	↓1.57	0.745 ± 0.005	↓9.69
8	0.745 ± 0.003	0.703 ± 0.004	↓5.63	0.665 ± 0.003	↓10.73
10	0.786 ± 0.002	0.654 ± 0.003	↓16.79	0.602 ± 0.004	↓23.41
12	0.812 ± 0.003	0.598 ± 0.004	↓26.35	0.564 ± 0.005	↓30.54
14	0.798 ± 0.002	0.562 ± 0.003	↓29.57	0.534 ± 0.004	↓33.08
16	0.658 ± 0.004	0.521 ± 0.003	↓20.82	0.512 ± 0.006	↓22.18
18	0.702 ± 0.003	0.498 ± 0.004	↓29.06	0.476 ± 0.003	↓32.19
20	0.689 ± 0.002	0.472 ± 0.003	↓31.49	0.441 ± 0.004	↓36.00
22	0.674 ± 0.003	0.451 ± 0.004	↓33.08	0.418 ± 0.003	↓37.98

24	0.447 ± 0.003	0.427 ± 0.003	↓4.47	0.396 ± 0.004	↓11.40

Figure 1: Time-dependent changes in oxygen consumption of *Ctenopharyngodon idella* exposed to phenthoate (50% EC)



RESULTS AND DISCUSSION

The pattern of oxygen consumption in Ctenopharyngodon idella exposed to lethal and sublethal concentrations of phenthoate (50% EC) is presented in Table 1 and Figure 1. The results showed that exposure to phenthoate produced clear time-dependent changes in respiratory metabolism when compared with the control group.

In the control group, oxygen consumption gradually decreased during the 24-hour experimental period. This reduction may be due to fasting conditions, reduced activity, and stabilization of metabolic processes under laboratory conditions. Similar reductions in oxygen consumption in unfed fish have been reported earlier and are considered a normal physiological adjustment under experimental conditions (Job, 1955; Rohankar and Kulkarni, 2005).

Fish exposed to the sublethal concentration of phenthoate showed an initial increase in oxygen consumption during the early hours of exposure. The increase reached a maximum during the initial exposure period, indicating elevated metabolic activity and respiratory stress. This response may be associated with increased opercular movements, enhanced gill ventilation, and activation of detoxification mechanisms to counteract the toxic effects of the pesticide. Similar increases in oxygen consumption have been reported in fishes exposed to organophosphate pesticides such as ethion, dimethoate, and chlorpyrifos (Kalavathy et al., 2001; Prasanna et al., 2020; Sharma and Singh, 2019).

As exposure continued, oxygen consumption in the sublethal group gradually declined. The reduction indicates metabolic depression caused by prolonged pesticide stress. The decline may be due to structural and functional damage to gill tissues, reduced oxygen diffusion, and disturbance in cellular respiration. Prolonged exposure to toxicants may also interfere with mitochondrial activity and energy production, leading to reduced oxygen utilization.

A similar but more pronounced response was observed in fish exposed to the lethal concentration of phenthoate. During the early phase of exposure, oxygen consumption increased sharply, suggesting a higher level of physiological stress under lethal conditions. This increase may represent an adaptive response of the fish to overcome toxic stress through increased respiratory activity.

However, prolonged exposure resulted in a rapid decline in oxygen consumption in the lethal group, with markedly low values recorded during the later stages of exposure. This decline indicates severe respiratory stress and metabolic exhaustion. The reduction in oxygen consumption may be associated with damage to the gill epithelium, mucus accumulation, lamellar degeneration, and reduced surface area available for gaseous exchange. Organophosphate pesticides such as phenthoate are also known to

inhibit acetylcholinesterase activity, which may impair neuromuscular coordination and reduce opercular movement, thereby affecting respiration.

The decline in oxygen consumption may further be related to oxidative stress and disruption of enzymatic systems involved in energy metabolism. Prolonged exposure to pesticides can lead to depletion of energy reserves, inhibition of respiratory enzymes, and reduced ATP production, resulting in metabolic suppression. Similar reductions in oxygen consumption have been reported in Labeo rohita, Channa punctatus, and Cyprinus carpio exposed to pesticides (Veeraiah and Durga Prasad, 2001; David et al., 2002; Khan et al., 2021).

The present study therefore demonstrates a biphasic response in oxygen consumption, characterized by an initial stimulatory phase followed by a phase of inhibition. The initial increase appears to be an adaptive response to acute toxic stress, whereas the later decline reflects failure of compensatory mechanisms during prolonged exposure. Similar patterns have been reported in fish exposed to various pesticides (Tilak et al., 2007; Patil and David, 2008). Comparable alterations in respiratory metabolism were also observed in Ctenopharyngodon idella exposed to tebuconazole (Bollu et al., 2026).

The more pronounced effects observed under lethal exposure conditions indicate a concentration-dependent effect of phenthoate on respiratory metabolism. Such alterations in oxygen consumption may affect growth, survival, and normal physiological functions of fish, thereby influencing the overall health of aquatic ecosystems.

Ethical Approval

All experimental procedures involving fish were carried out in accordance with standard laboratory guidelines for the care and handling of experimental animals.

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