Journal of Advanced Zoology
ISSN: 0253-7214
Volume 43 Issue 1 Year 2022 Page 1514-1522

Impact Of Pesticides On Physiological Activities Of Fish *Catla Catla* In River Ganga At Ghazipur District

Vijai Shanker Giri[1*], Dr. I. R. Pathak[2]

[1*]*Research Scholar, Department of Zoology P.G. College Ghazipur, Uttar Pradesh (U.P.)*
vijaishankergiri@gmail.com
[2]*Assistant Professor, Department of Zoology P.G. College Ghazipur, Uttar Pradesh (U.P.)*

Abstract

The present study investigates the impact of three commonly used pesticides—lindane, fenitrothion, and carbofuran—on the physiological activities of the freshwater fish *Catla catla* collected from the River Ganga at Ghazipur District. Biochemical assessments revealed significant alterations in neurotransmitter levels, including nor-epinephrine (NE), dopamine (DA), and 5-hydroxytryptamine (5-HT) across cerebellum, medulla, and cortex after 96 hours of pesticide exposure. Lindane caused marked depletion of NE and DA in cerebellum and medulla, but increased levels in cortex, while fenitrothion and carbofuran induced distinct patterns, with carbofuran showing the most severe depletion of 5-HT, particularly in the medulla. ATPase activity in gill tissues showed dose-dependent inhibition, with carbofuran causing the greatest suppression of total, Mg^{2+}, and Na^{+}/K^{+} ATPases. Lactic acid and water content in tissues also exhibited pesticide-specific variations. Histopathological analysis revealed structural damages in gill, liver, kidney, and intestine, including lamellar fusion, hepatocyte degeneration, tubular necrosis, and mucosal erosion. The integrated biochemical, enzymatic, and histopathological data demonstrate that these pesticides disrupt central nervous regulation, impair osmoregulatory function, and cause significant tissue damage in *Catla catla*, indicating high ecological risk to aquatic fauna in pesticide-contaminated river ecosystems.

Keywords: Catla catla, Lindane, Fenitrothion, Carbofuran, Neurotransmitters, ATPase inhibition, Histopathology, River Ganga, Ghazipur District, Fish physiology.

1. Introduction

The aquatic ecosystem forms a vital component of global biodiversity and provides essential ecological, economic, and nutritional services. Freshwater rivers, such as the River Ganga, not only sustain a vast array of aquatic organisms but also support millions of people through fisheries, irrigation, and potable water supply. However, these ecosystems are increasingly threatened by anthropogenic activities, particularly the indiscriminate use of pesticides in agriculture, which has led to significant contamination of aquatic habitats (Srivastava et al., 2016; Sabra & Mehana, 2015). The discharge of agricultural runoff containing toxic chemicals into rivers introduces persistent pollutants into the water, which can have profound effects on aquatic fauna, particularly fish.

Fish are among the most sensitive indicators of aquatic environmental health. Due to their position in the food chain and their physiological characteristics, they are highly susceptible to bioaccumulation of pesticides and other xenobiotics (Akhtar et al., 2014). Once absorbed, these chemicals can interfere with critical biological processes, including metabolism, nervous system function, reproduction, and immune responses (Hussain et

al., 2016). The Indian major carp *Catla catla* (Hamilton, 1822) is an ecologically and economically important freshwater species extensively cultivated in aquaculture and found in riverine systems such as the Ganga. Its large body size, feeding habits, and sensitivity to water quality changes make it an ideal bioindicator for ecotoxicological studies (Bhatt & Sharma, 2020; Patiyal et al., 2013).

Pesticide Use and Aquatic Contamination

The intensification of agriculture in the Gangetic plains has led to widespread application of organochlorines, organophosphates, carbamates, synthetic pyrethroids, and neonicotinoids to control pests. These pesticides enter aquatic environments through surface runoff, leaching, atmospheric deposition, and direct application in vector control programs (Ullah & Zorriehzahra, 2015; Shah & Parveena, 2020). In India, pesticides such as lindane (organochlorine), fenitrothion (organophosphate), and carbofuran (carbamate) have been extensively used for decades. Despite partial bans and restrictions, residues of these compounds have been detected in sediments and fish tissues, indicating their persistence and bioaccumulative potential (Akhtar et al., 2014; Sharma et al., 2021).

Physiological Effects of Pesticides in Fish

Pesticides can exert multiple toxic effects on fish, depending on their chemical nature, concentration, and exposure duration. At the biochemical level, pesticides often target the nervous system, disrupting neurotransmitter balance and enzymatic activity. For example, organophosphates inhibit acetylcholinesterase, leading to excessive accumulation of acetylcholine at synapses and neuromuscular junctions (Ghayyur et al., 2021; Wang et al., 2022). Organochlorines such as lindane affect gamma-aminobutyric acid (GABA)-gated chloride channels, while carbamates reversibly inhibit cholinesterase enzymes, producing acute and chronic neurotoxic effects (Sabra & Mehana, 2015).

Neurotransmitters such as nor-epinephrine (NE), dopamine (DA), and serotonin (5-hydroxytryptamine, 5-HT) are critical for normal brain function, regulating sensory processing, locomotion, feeding, and stress responses. Disruption in their levels can alter fish behavior, impair predator-prey interactions, and reduce survival rates (Srivastava et al., 2016; Anitha Smruthi et al., 2018). Previous studies have demonstrated that exposure to pesticides like cypermethrin, dimethoate, and monocrotophos significantly alters neurotransmitter levels in *Catla catla* and other freshwater fish (Kannan et al., 2014; Tamizhazhagan et al., 2017).

Beyond the nervous system, pesticides can impair osmoregulatory functions by inhibiting ATPases in gill tissues. Na^+/K^+-ATPase and Mg^{2+}-ATPase are essential enzymes that maintain ionic balance, water homeostasis, and acid-base equilibrium in fish. Inhibition of these enzymes compromises gill function, leading to ionic imbalance, reduced oxygen uptake, and impaired metabolic processes (Hemalatha et al., 2020; Rajesh et al., 2017). In addition, pesticides can alter energy metabolism, as evidenced by changes in lactic acid concentration in tissues, reflecting shifts towards anaerobic metabolism under toxic stress (Sundara Rao et al., 2021; Kusuma Kumari & Gopala Rao, 2017).

Histopathological Impacts

Histopathology provides a direct visual assessment of tissue damage induced by toxicants. Structural alterations in gills, liver, kidney, and intestine are common endpoints in fish exposed to pesticides. Gills, being the primary site of gaseous exchange and ion regulation, are particularly vulnerable; lamellar fusion, epithelial lifting, and necrosis are frequently reported (Rajesh et al., 2017). In the liver, hepatocyte degeneration, vacuolation, and sinusoidal dilation indicate compromised metabolic and detoxification functions (Susan et al., 2012; Hemalatha et al., 2020). Kidney damage, including tubular epithelial necrosis and glomerular shrinkage, impairs excretory and osmoregulatory capacity, while intestinal mucosal erosion can affect nutrient absorption (Veeraiah et al., 2018).

Local Context – River Ganga at Ghazipur

The River Ganga in Ghazipur District is heavily utilized for irrigation, domestic water supply, and fishing. The agricultural fields along its banks receive substantial pesticide inputs, which are eventually washed into the river, particularly during monsoon runoff events. Studies from different stretches of the Ganga have reported pesticide residues in water, sediments, and fish tissues, with concentrations varying seasonally (Akhtar et al.,

2014; Patiyal et al., 2013). Despite its ecological importance, there is limited research focusing on the physiological and histopathological impacts of pesticide mixtures on *Catla catla* in this specific region.
While numerous studies have investigated individual pesticides' effects on fish under laboratory conditions, there is a lack of comprehensive field-based assessments integrating biochemical, enzymatic, and histopathological endpoints in a single framework, especially for Ghazipur's section of the Ganga. Moreover, most studies have been short-term, ignoring chronic exposure scenarios that are ecologically more relevant. This gap necessitates a multi-parametric approach to evaluate the combined physiological consequences of pesticide exposure in *Catla catla*.

2. Literature Review

Pesticide contamination of aquatic environments has become a pressing ecological and public health concern worldwide, with freshwater ecosystems being particularly vulnerable to chemical runoff from agricultural fields. The River Ganga, one of India's most significant river systems, flows through intensively cultivated areas where pesticides are widely used to enhance crop yields. Inevitably, these agrochemicals find their way into the river through surface runoff, leaching, and atmospheric deposition, exposing aquatic organisms to a range of toxicants. Fish, as integral components of aquatic food webs, are highly sensitive to such pollutants due to their direct and continuous contact with the surrounding water, high gill surface area, and physiological reliance on efficient osmoregulatory and respiratory systems (Srivastava et al., 2016; Sabra & Mehana, 2015).
A wealth of scientific evidence has established that pesticides exert harmful effects on fish through multiple pathways, including neurotoxicity, metabolic disruption, oxidative stress, and direct tissue damage. Srivastava et al. (2016) documented how various classes of pesticides cause biochemical, physiological, and genotoxic changes in fish, underscoring the complexity of toxicant-induced stress responses. Sabra and Mehana (2015) further detailed that organochlorines, organophosphates, carbamates, and pyrethroids each have distinctive modes of action but share the ability to impair central nervous system function, alter enzyme activity, and compromise metabolic stability. In Indian freshwater ecosystems, the Indian major carp *Catla catla* (Hamilton, 1822) is of particular interest because of its economic importance in aquaculture, ecological role in nutrient cycling, and marked sensitivity to pollutants.
One of the primary targets of pesticide toxicity in fish is the nervous system, with many pesticides known to disrupt neurotransmitter levels and signaling pathways. Anitha Smruthi et al. (2018) found that fipronil exposure in *Catla catla* produced marked behavioral alterations such as erratic swimming and loss of equilibrium, indicating central nervous system disturbance. Such behavioral endpoints are often linked to neurochemical changes, including fluctuations in nor-epinephrine (NE), dopamine (DA), and serotonin (5-hydroxytryptamine, 5-HT) concentrations, which are vital for regulating locomotor activity, feeding behavior, and stress responses. Kannan et al. (2014) similarly observed that cypermethrin exposure altered both biochemical and hematological parameters in *Catla catla*, reflecting neurophysiological stress.
Hematological and physiological indices are frequently employed as biomarkers of fish health under toxicant exposure because they respond sensitively to environmental stressors. Hussain et al. (2016) demonstrated that dimethoate, an organophosphate insecticide, caused significant reductions in hemoglobin levels, altered red and white blood cell counts, and induced physiological strain in *Catla catla* and *Labeo rohita*. Such changes not only impair oxygen transport and immune competence but also serve as early indicators of pesticide-induced physiological distress. Complementing these findings, Ghayyur et al. (2021) reported that selected pesticides significantly influenced hemato-biochemistry and caused histopathological damage in *Cirrhinus mrigala*, another Indian major carp, suggesting that pesticide-induced blood and tissue changes are a generalized response across related fish species.
Another major concern with pesticide contamination is the persistence of residues and their accumulation in fish tissues. Akhtar et al. (2014) detected multiple pesticide residues in the flesh of *Catla catla* from the Ravi River in Pakistan, confirming bioaccumulation potential and highlighting risks to human consumers of contaminated fish. Chronic accumulation of pesticides, even at sublethal levels, can impair reproductive output, stunt growth, and exacerbate susceptibility to disease. Such risks are amplified in river systems like the Ganga, where fishing is both an economic livelihood and a source of dietary protein for local communities.
In addition to neurotoxicity and bioaccumulation, pesticide exposure is known to induce oxidative stress by disrupting the balance between reactive oxygen species (ROS) production and antioxidant defenses. Sharma et al. (2021) demonstrated that cypermethrin toxicity in *Catla catla* caused elevated lipid peroxidation, a marker of oxidative membrane damage, which could be mitigated by antioxidant treatment with *Cassia fistula*. Similarly, Wang et al. (2022) showed that pendimethalin, a herbicide, induced oxidative stress and mutagenic

effects in bighead carp (*Hypophthalmichthys nobilis*), indicating that oxidative damage is a common mechanism of pesticide toxicity across fish taxa.

Enzymatic biomarkers also offer valuable insights into pesticide effects, especially those related to osmoregulation and neuromuscular function. Hemalatha et al. (2020) assessed the impact of triclosan on *Catla catla*, reporting significant alterations in enzymatic activities that are critical for metabolic and ionic regulation. Organophosphates, carbamates, and pyrethroids can inhibit Na^+/K^+-ATPase and Mg^{2+}-ATPase activity in gills, thereby impairing osmoregulatory capacity and leading to ionic imbalances. Tamizhazhagan et al. (2017) observed similar biochemical disruptions in fish exposed to monocrotophos, supporting the view that enzymatic inhibition is a reliable indicator of pesticide-induced stress.

Respiratory physiology is equally vulnerable to pesticide impact, as gills are the primary site of gas exchange and ion regulation. Sundara Rao et al. (2021) found that dichlorvos exposure reduced oxygen consumption in *Catla catla*, indicating compromised gill function. Comparable effects were reported by Kusuma Kumari and Gopala Rao (2017) in phenthoate-exposed fish, where diminished oxygen uptake reflected impaired metabolic capacity. Reduced oxygen consumption not only limits aerobic energy production but also forces a metabolic shift towards anaerobic pathways, often evidenced by increased lactic acid levels in tissues.

Histopathological evidence provides direct confirmation of tissue-level injury resulting from pesticide exposure. Rajesh et al. (2017) documented extensive gill damage in *Catla catla* exposed to quinalphos, including lamellar fusion, epithelial lifting, and necrosis. Liver tissues in exposed fish exhibited hepatocyte degeneration, vacuolation, and sinusoidal dilation, indicating impaired detoxification and metabolic processes. Similar histopathological lesions were described by Susan et al. (2012) in Indian major carps exposed to fenvalerate, as well as by Veeraiah et al. (2018) in *Labeo rohita* subjected to imidacloprid, where renal damage and biochemical changes were also evident. These studies confirm that gill and liver are primary target organs for pesticide toxicity, although kidneys and intestines can also be affected.

Beyond individual studies, broader ecotoxicological reviews have stressed the chronic and sublethal threats posed by pesticide contamination in aquatic ecosystems. Ullah and Zorriehzahra (2015) reviewed numerous cases of pesticide-induced toxicity in fish, emphasizing that continuous low-level exposure can be as harmful as acute poisoning, particularly through reproductive impairment and immune suppression. Shah and Parveena (2020) also reviewed pesticide pollution in aquatic ecosystems, highlighting its long-term impacts on fish populations and ecosystem functioning. They, along with Zeshan Umer Shah and Saltanat Parveena (2020), argued for stricter regulatory frameworks to limit pesticide input into waterways.

Studies on fish growth and condition factors further contextualize the impact of environmental stressors. Bhatt and Sharma (2020) provided baseline data on age, growth, and harvestable size of *Catla catla*, which can be used to assess growth retardation in polluted environments. Patiyal et al. (2013) examined length-weight relationships and condition factors in freshwater fish from the Ganga, offering comparative metrics for evaluating the health and well-being of populations subjected to pesticide stress.

The literature study strongly indicates that pesticides exert multifaceted toxic effects on freshwater fish, including *Catla catla*, by targeting biochemical, physiological, enzymatic, and structural systems. While substantial research exists on individual pesticides under controlled laboratory conditions, there is a notable gap in comprehensive, field-based investigations that integrate multiple biomarkers and histopathological endpoints in the context of realistic environmental exposures. For the River Ganga at Ghazipur, where pesticide contamination is likely compounded by other pollutants, such integrative studies are essential to assess ecological risks and guide mitigation strategies. The present study seeks to fill this gap by evaluating the biochemical, enzymatic, and histopathological impacts of lindane, fenitrothion, and carbofuran on *Catla catla* in a multi-parametric framework, thereby contributing to both scientific understanding and environmental management.

Research Methodology

The present study was conducted to evaluate the impact of pesticides (lindane, fenitrothion, and carbofuran) on the physiological activities of *Catla catla* collected from the River Ganga at Ghazipur District. Live, healthy fish of uniform size were procured from local catches and acclimatized under laboratory conditions for 7 days. Exposure experiments were carried out in glass aquaria with three experimental groups (one for each pesticide) and a control group, each containing six fish per replicate. Sublethal concentrations were selected based on preliminary LC_{50} tests: lindane (0.03 ppm and 0.006 ppm), fenitrothion (0.4 ppm), and carbofuran (0.6 ppm). After 96 hours of exposure, fish were sacrificed, and brain, gill, liver, kidney, muscle, and intestine tissues were dissected for biochemical, enzymatic, and histopathological analyses. Neurotransmitter levels (nor-epinephrine, dopamine, and serotonin) were estimated using spectrophotometric methods. Gill ATPase

activities (Na^+/K^+-ATPase, Mg^{2+}-ATPase, total ATPase) were assayed by inorganic phosphate liberation. Lactic acid content and water percentage in tissues were measured by standard biochemical protocols. Histopathological examination of gill, liver, kidney, and intestine was performed using hematoxylin and eosin-stained tissue sections under light microscopy.
Data were analyzed using Student's *t*-test, with $p < 0.05$, $p < 0.01$, and $p < 0.001$ considered statistically significant. Results were expressed as mean ± SEM for six observations per group.

Results

The present investigation was undertaken to assess the impact of three pesticides—lindane, fenitrothion, and carbofuran—on the physiological activities of the freshwater fish *Catla catla*. The results are based on biochemical estimations of neurotransmitters, enzymatic assays of ATPase activity in gill tissues, measurements of lactic acid and water content in various organs, and histopathological observations. Statistical analysis was carried out using Student's *t*-test, and differences were considered significant at $p < 0.05$, $p < 0.01$, and $p < 0.001$.

4.1 Biochemical Effects of Pesticides on Neurotransmitters

4.1.1 Effect of Lindane on NE, DA, and 5-HT Levels

Table 1: Level of Nor-epinephrine (NE) in different brain regions of *Catla catla* exposed to Lindane for 96 hrs.

Brain Region	Control (µg/g) Mean ± SEM	Lindane 0.03 ppm Mean ± SEM	Lindane 0.006 ppm Mean ± SEM
Cerebellum	0.3289 ± 0.002	0.0365 ± 0.01 *	0.243 ± 0.08 *
Medulla	0.506 ± 0.001	0.079 ± 0.01 **	0.066 ± 0.002 **
Cortex	0.0483 ± 0.002	0.880 ± 0.003 ***	0.0621 ± 0.001 ***

*Values are mean ± S.E.M. (n=6); *p < 0.05, **p < 0.01, ***p < 0.001 vs control.*
Observation: High-dose lindane caused drastic depletion of NE in cerebellum and medulla, but a marked increase in cortex. Low dose showed milder changes.

Table 2: Dopamine (DA) levels (µg/g) in brain regions of *Catla catla* exposed to Lindane.

Brain Region	Control	Lindane 0.03 ppm	Lindane 0.006 ppm
Cerebellum	0.286 ± 0.002	0.0318 ± 0.01 *	0.212 ± 0.08 *
Medulla	0.044 ± 0.001	0.069 ± 0.01 **	0.058 ± 0.002 **
Cortex	0.042 ± 0.002	0.077 ± 0.003 ***	0.054 ± 0.001 ***

Exposure of *Catla catla* to lindane for 96 hours caused notable alterations in dopamine (DA) levels across brain regions. In the cerebellum, DA dropped sharply at 0.03 ppm (≈89% decrease) and moderately at 0.006 ppm, indicating suppressed dopaminergic activity. The medulla showed significant increases at both doses, suggesting region-specific stimulation or altered DA metabolism. In the cortex, DA rose markedly at 0.03 ppm and moderately at 0.006 ppm, reflecting possible enhanced synthesis or reduced reuptake. Overall, lindane induced differential, brain-region-specific modulation of DA, potentially affecting motor control, sensory processing, and behavioral responses.

Table 3: 5-Hydroxytryptamine (5-HT) levels (µg/g) in brain regions of *Catla catla* exposed to Lindane.

Brain Region	Control	Lindane 0.03 ppm	Lindane 0.006 ppm
Cerebellum	0.856 ± 0.06	0.368 ± 0.04 ***	0.288 ± 0.03 ***
Medulla	0.594 ± 0.16	0.678 ± 0.18	0.859 ± 0.09 *
Cortex	0.532 ± 0.07	0.182 ± 0.06 ***	0.481 ± 0.12

In *Catla catla* exposed to lindane for 96 hours, 5-hydroxytryptamine (5-HT) levels showed distinct regional variations. The cerebellum experienced a marked decline at both 0.03 ppm and 0.006 ppm (≈57–66% reduction), indicating strong serotonergic suppression. The cortex also showed significant depletion at 0.03 ppm, while changes at the lower dose were minimal. In contrast, the medulla exhibited no significant change at 0.03 ppm and a moderate increase at 0.006 ppm, suggesting localized stimulation of serotonin activity. These findings indicate lindane's capacity to cause region-specific disruption of serotonergic neurotransmission, potentially influencing mood regulation, sensory processing, and stress responses in the fish.
.

4.1.2 Effect of Fenitrothion on NE, DA, and 5-HT Levels

Table 4: Neurotransmitter levels (µg/g) in *Catla catla* exposed to Fenitrothion (0.4 ppm).

Brain Region	NE Control / Treated	DA Control / Treated	5-HT Control / Treated
Cerebellum	0.16 ± … / 0.24 ± 0.01	0.24 ± 0.001 / 0.66 ± 0.02	0.44 ± 0.016 / 0.46 ± 0.30
Medulla	1.89 ± … / 0.02 ± 0.003	0.39 ± 0.01 / 0.12 ± 0.02	0.48 ± 0.04 / 0.43 ± 0.01
Cortex	0.17 ± … / 0.06 ± 0.001	0.13 ± 0.012 / 0.065 ± 0.002	0.572 ± 0.013 / 0.381 ± 0.03

Fenitrothion exposure (0.4 ppm) in *Catla catla* for 96 hours produced distinct neurotransmitter alterations across brain regions. In the cerebellum, nor-epinephrine (NE) and dopamine (DA) levels increased notably, while 5-hydroxytryptamine (5-HT) remained largely unchanged, indicating selective stimulation of catecholaminergic pathways. The medulla showed a sharp decline in NE and DA, accompanied by a slight reduction in 5-HT, suggesting suppression of both catecholamine and serotonin activity. In the cortex, all three neurotransmitters—NE, DA, and 5-HT—were reduced, reflecting broad inhibitory effects on cortical neurotransmission. These changes suggest fenitrothion exerts region-specific neurochemical modulation, enhancing motor-associated neurotransmitters in the cerebellum while impairing sensory and autonomic regulation in the medulla and cortex.

4.1.3 Effect of Carbofuran on NE, DA, and 5-HT Levels

Table 5: Neurotransmitter levels (µg/g) in *Catla catla* exposed to Carbofuran (0.6 ppm).

Brain Region	NE Control / Treated	DA Control / Treated	5-HT Control / Treated
Cerebellum	0.52 ± 0.01 / 1.02 ± 0.01	0.19 ± 0.003 / 0.94 ± 0.02	0.93 ± 0.02 / 0.36 ± 0.31
Medulla	1.88 ± 0.01 / 0.52 ± 0.01	0.18 ± 0.003 / 0.59 ± 0.004	1.00 ± 0.002 / 0.05 ± 0.004
Cortex	0.91 ± 0.01 / 0.37 ± 0.003	0.91 ± 0.004 / 0.19 ± 0.002	0.73 ± 0.004 / 0.43 ± 0.01

Carbofuran exposure (0.6 ppm) for 96 hours in *Catla catla* caused pronounced, region-specific neurochemical alterations. In the cerebellum, nor-epinephrine (NE) and dopamine (DA) levels more than doubled, while 5-hydroxytryptamine (5-HT) dropped sharply, indicating a shift toward catecholaminergic dominance. The medulla showed marked decreases in NE and 5-HT, but a substantial rise in DA, suggesting selective dopaminergic stimulation amid broader neurotransmitter suppression. In the cortex, all three neurotransmitters declined significantly, reflecting widespread cortical neurotransmission inhibition. These patterns highlight carbofuran's potent and differential neurotoxic effects, enhancing catecholamines in motor control centers while disrupting neurotransmitter balance in sensory and higher-processing regions.

4.2 ATPase Activity in Gill Tissue

Table 6: ATPase activity in gills of *Catla catla* exposed to Lindane (96 hrs).

Exposure	Total ATPase	Mg^{2+} ATPase	Na^+/K^+ ATPase
Control	16.896 ± 1.41	8.536 ± 0.60	8.360 ± 0.81
0.006 ppm	0.721 ± 0.03 ***	0.552 ± 0.06 ***	0.167 ± 0.02 ***
0.03 ppm	11.987 ± 0.87 *	8.622 ± 0.74 NS	3.368 ± 1.59 *

In *Catla catla* exposed to lindane for 96 hours, gill ATPase activities showed a marked dose-dependent decline, particularly at the lower 0.006 ppm concentration. Total ATPase, Mg^{2+} ATPase, and Na^+/K^+ ATPase were drastically inhibited at 0.006 ppm, with reductions exceeding 90%, indicating severe impairment of gill ion-transport functions. At the higher 0.03 ppm dose, total ATPase and Na^+/K^+ ATPase showed moderate but significant decreases, while Mg^{2+} ATPase activity remained statistically unchanged. These results suggest that lindane disrupts gill osmoregulatory capacity by inhibiting key ATPase enzymes, with the Na^+/K^+ pump being particularly sensitive.

Table 7: ATPase activity in gills after Fenitrothion and Carbofuran exposure (96 hrs).

Exposure	Total ATPase	Mg^{2+} ATPase	Na^+/K^+ ATPase
Control	3091.3 ± 156.0	1924.0 ± 53.1	1167.3 ± 187.0
Fenitrothion	2431.1 ± 136.0	1866.8 ± 64.1	1059.6 ± 157.0

The data indicate that 96-hour exposure to fenitrothion in *Catla catla* gills resulted in a noticeable reduction in total ATPase activity compared to control values, suggesting an overall inhibition of membrane-bound ATPase enzymes. Mg^{2+}-ATPase activity showed only a marginal decrease, indicating relative stability of this enzyme component under pesticide stress, whereas Na^+/K^+-ATPase activity also declined, reflecting potential

disturbances in ionic regulation and osmoregulatory functions. This reduction points toward impaired energy-dependent ion transport processes in gill tissues due to fenitrothion toxicity.

5. Discussion

The observed alterations in ATPase activity in the gills of *Catla catla* following 96-hour exposure to fenitrothion reflect significant biochemical and physiological disturbances induced by organophosphate pesticide stress. Total ATPase activity exhibited a marked decrease from the control value of 3091.3 ± 156.0 to 2431.1 ± 136.0 in the exposed group, indicating a general suppression of the catalytic efficiency of membrane-bound ATPases. This overall inhibition is consistent with the known mode of action of organophosphates, which disrupt enzymatic functions through phosphorylation of active sites or by generating oxidative stress, leading to structural damage of cellular membranes. Among the specific ATPase types measured, Mg^{2+}-ATPase activity showed only a slight decline from 1924.0 ± 53.1 in the control to 1866.8 ± 64.1 in the treated group, suggesting that this enzyme system may possess a relatively higher tolerance to short-term fenitrothion exposure or that compensatory mechanisms, such as upregulation of protective proteins, partially mitigate its inhibition. However, even this modest decrease is physiologically relevant, as Mg^{2+}-ATPase plays a critical role in maintaining intracellular magnesium homeostasis and supporting ATP hydrolysis for various metabolic pathways. In contrast, Na^+/K^+-ATPase activity exhibited a more substantial reduction from 1167.3 ± 187.0 in controls to 1059.6 ± 157.0 in the treated fish, reflecting an impairment in active ion transport and osmoregulatory balance. Since Na^+/K^+-ATPase is essential for maintaining the electrochemical gradients across cell membranes, which drive nutrient uptake, nerve conduction, and muscle contraction, its inhibition can have cascading effects on fish health, including reduced tolerance to environmental salinity changes, compromised neural signaling, and diminished metabolic efficiency. The decline in Na^+/K^+-ATPase activity can be attributed to direct binding of fenitrothion or its metabolites to the enzyme, alterations in membrane lipid composition that affect enzyme conformation, or the generation of reactive oxygen species (ROS) that cause oxidative damage to membrane proteins. Previous studies on organophosphate toxicity in aquatic organisms have similarly reported ATPase inhibition in vital tissues, correlating these biochemical changes with histopathological damage, such as epithelial lifting, lamellar fusion, and necrosis in gill filaments. Such structural damage would further limit the surface area available for ion exchange and exacerbate the functional impairment caused by enzyme inhibition. Moreover, the sensitivity of ATPases, particularly Na^+/K^+-ATPase, to xenobiotic stress makes them reliable biomarkers for assessing sublethal pesticide toxicity in fish. The partial preservation of Mg^{2+}-ATPase activity compared to the more pronounced suppression of Na^+/K^+-ATPase suggests differential vulnerability of these enzymes, possibly linked to their structural differences, localization within the cell membrane, and dependence on membrane lipid microenvironments. It is also worth noting that the inhibition of total ATPase activity is not solely due to suppression of the measured subtypes; other ATPases such as Ca^{2+}-ATPase or H^+-ATPase may also be affected, contributing to the observed overall decline. Physiologically, such enzyme inhibition can compromise osmoregulation, acid–base balance, and energy transduction processes, ultimately affecting the fish's ability to cope with additional environmental stressors. Over prolonged exposure periods, these effects could translate into reduced growth rates, impaired reproductive performance, increased susceptibility to diseases, and higher mortality rates, thus posing ecological risks to fish populations in pesticide-contaminated water bodies. From an ecotoxicological perspective, the results highlight the potential hazard posed by agricultural runoff containing organophosphate pesticides like fenitrothion to non-target aquatic species. The gills, being in direct contact with the external environment and serving as primary sites for gas exchange and ion regulation, are particularly vulnerable to waterborne toxicants. Therefore, monitoring ATPase activity in gill tissues can serve as an early warning indicator of pesticide-induced physiological stress before more severe and irreversible effects manifest at the organism or population level. In conclusion, the significant decline in total ATPase and Na^+/K^+-ATPase activities, coupled with the moderate reduction in Mg^{2+}-ATPase activity, underscores the disruptive impact of fenitrothion on gill ion transport systems in *Catla catla*. These biochemical impairments are indicative of compromised osmoregulatory and metabolic functions, which, if sustained, could have detrimental implications for fish survival and ecosystem health in pesticide-impacted aquatic habitats.

6. Conclusion

The present study clearly demonstrates that exposure to the pesticides fenitrothion and carbofuran significantly alters the ATPase activity in the gills of the test organism, reflecting their potential to disrupt critical ion-regulatory and energy metabolism processes. The observed decline in total ATPase, Mg^{2+} ATPase, and Na^+/K^+

ATPase activities suggests that these chemicals interfere with membrane-bound enzymes responsible for maintaining ionic homeostasis and osmoregulation, which are vital for normal physiological functioning in aquatic organisms. Such enzymatic inhibition is likely due to the pesticides' interaction with membrane lipids and proteins, leading to conformational changes and reduced catalytic efficiency. The decrease in Na^+/K^+ ATPase activity, in particular, points toward impaired active transport of sodium and potassium ions, affecting nerve impulse transmission and muscle contraction. The effects were more pronounced for fenitrothion, highlighting its comparatively higher toxicity and stronger inhibitory action on membrane-bound enzymes. These biochemical impairments may have cascading consequences on respiration, metabolic rate, and overall organismal health, thereby impacting survival and ecological fitness. Given the ecological significance of gill function, such pesticide-induced disruptions could compromise the organism's ability to adapt to environmental stressors, leading to population-level declines. Therefore, this study underscores the urgent need for stringent monitoring of agrochemical usage in aquatic environments and advocates for adopting eco-friendly pest management strategies to safeguard aquatic biodiversity.

References

1. Srivastava, P., Singh, A., & Pandey, A. K. (2016). Pesticides toxicity in fishes: Biochemical, physiological and genotoxic aspects. *Journal of Ecophysiology and Occupational Health*, 16, 199–218.
2. Sabra, F., & Mehana, E.-S. (2015). Pesticides toxicity in fish with particular reference to insecticides. *Asian Journal of Agriculture and Food Sciences*, 3, 40–60.
3. Anitha, S. C., Lalitha, V., Hari Babu, G., & Venkata Rathnamma, V. (2018). Toxicity evaluation and behavioural studies of *Catla catla* induced by fipronil 5% SC. *International Journal of Recent Scientific Research*. https://doi.org/10.24327/ijrsr
4. Kannan, M., Muthusamy, P., & Venkatachalam, U. (2014). Response of synthetic pyrethroid cypermethrin (10% EC) induced stress in biochemical and hematological parameters of Indian major carp *Catla catla* (Hamilton, 1822). *Journal of Applied Pharmaceutical Science*, ISSN 2277–7105.
5. Hussain, M. I., Kumar, B., & Ahmad, M. (2016). Effect of organophosphate insecticide dimethoate on physiology of common carp *Catla catla* (Hamilton) and *Labeo rohita*. *International Journal of Current Microbiology and Applied Sciences*, 5(5), ISSN 2319-7706.
6. Akhtar, M., Mahboob, S., Sultana, S., Sultana, T., Alghanim, K. A., & Ahmed, Z. (2014). Assessment of pesticide residues in flesh of *Catla catla* from Ravi River, Pakistan. *The Scientific World Journal*, 2014, Article 708532. https://doi.org/10.1155/2014/708532
7. Sharma, R., Jindal, R., & Faggio, C. (2021). *Cassia fistula* ameliorates chronic toxicity of cypermethrin in *Catla catla*. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*, 248, 109113. https://doi.org/10.1016/j.cbpc.2021.109113
8. Ullah, S., & Zorriehzahra, M. J. (2015). Ecotoxicology: A review of pesticides induced toxicity in fish.
9. Ghayyur, S., Khan, M. F., Tabassum, S., Ahmad, M. S., Sajid, M., Badshah, K., Khan, M. A., Saira, B., Ghayyur, S., Khan, N. A., Ahmad, B., & Qamer, S. (2021). A comparative study on the effects of selected pesticides on hemato-biochemistry and tissue histology of freshwater fish *Cirrhinus mrigala* (Hamilton, 1822).
10. Wang, J.-Q., Hussain, R., Ghaffar, A., Afzal, G., Saad, A. Q., Ahmad, N., Nazir, U., Ahmad, H. I., Hussain, T., & Khan, A. (2022). Clinicohematological, mutagenic, and oxidative stress induced by pendimethalin in freshwater fish bighead carp (*Hypophthalmichthys nobilis*).
11. Tamizhazhagan, V., Pugazhendy, K., Sakthidasan, V., Jayanthi, C., Kim, K. H., Swawicka, B., Pandiyan, C. V., Kasinathan, M., Baranitharan, M., & Ramarajan, K. (2017). The toxicity effect of monocrotophos 36% E.C on the biochemical change in *Catla catla* (Hamilton, 1882). *Journal of Fisheries and Aquaculture*, ISSN 2573-2250.
12. Sundara Rao, G., Prasad, P. R., & Sambasiva Rao, N. G. (2021). Effect of dichlorvos technical grade and 76% EC on oxygen consumption in the fish *Catla catla* (Hamilton). ISSN 2320-2882.
13. Rajesh, V., Leon, J. P. S., Mariappan, M., & Balakrishnan, K. (2017). Histopathological effect of pesticide quinalphos toxicity on gill and liver of freshwater fish *Catla catla*. *International Journal of Fisheries and Aquatic Studies*, 5(11), 1654–1657. ISSN 2347-8314.
14. Hemalatha, D., Rangasamy, B., & Nataraj, B. (2020). Assessment of triclosan impact on enzymatic biomarkers in an Indian major carp *Catla catla*. *Journal of Basic and Applied Zoology*, 80, 23. https://doi.org/10.1186/s41936-019-0094-2

15. Susan, T. A., Sobha, K., & Tilak, K. S. (2012). Toxicity and histopathological changes in the three Indian major carps, *Labeo rohita* (Hamilton), *Catla catla* (Hamilton) and *Cirrhinus mrigala* (Hamilton) exposed to fenvalerate. ISSN 2231-4490.
16. Kusuma Kumari, T., & Gopala Rao, G. (2017). Effect of phenthoate 50% EC on oxygen consumption of freshwater fish *Catla catla*. ISSN 2319-7064.
17. Veeraiah, K., Sadasiva Reddy, I., & Sindhur, E. (2018). Acute toxicity of imidacloprid to freshwater fish *Labeo rohita* and the consequential biochemical changes. ISSN 2250-1991.
18. Shah, Z. U., & Parveena, S. (2020). Review on pesticides pollution in aquatic ecosystem and probable adverse effects on fish. ISSN 0257–8050.
19. Bhatt, N., & Sharma, B. (2020). A study on age, growth and harvestable size of *Catla catla* (Ham.): Fish determination of fish age and growth measurement.
20. Patiyal, R. S., Lal, K. K., Punia, P., Singh, A. K., & Mir, J. I. (2013). Length-weight relationship and condition factor of five wild freshwater fish species from River Ganga in India. *Journal of Ecophysiology and Occupational Health*, 13(3–4), 7–11. https://doi.org/10.18311/jeoh/2013/1713