International Journal of Forest, Animal and Fisheries Research (IJFAF)
ISSN: 2456-8791
[Vol-10, Issue-4, Oct-Dec, 2026]
Issue DOI: https://dx.doi.org/10.22161/ijfaf.10.4
Article DOI: https://dx.doi.org/10.22161/ijfaf.10.4.3

Metabolic Enzymes and Glycogen disruptions in Freshwater Fish *Cyprinus carpio* L. Induced by Sub-lethal Exposure to Halosulfuron-methyl Herbicide

Darshan Kumar T[1] and Parimala B[2*]

[1]Research Scholar, DOSR in Zoology, University College of Science, Tumkur University, Tumakuru.
[2*] Professor, Department of Studies and Research in Zoology, Tumkur University, Tumakuru.

Received: 10 Sep 2026; Received in revised form: 28 Oct 2026; Accepted: 12 Nov 2026; Available online: 18 Nov 2026
©2026 The Author(s). Published by AI Publications. This is an open-access article under the CC BY license (https://creativecommons.org/licenses/by/4.0/)

Abstract *— The present research investigation was designed to conduct the sub-lethal toxicological effects of Halosulfuron-methyl on several metabolic enzymes activities in freshwater fish Cyprinus carpio L. Succeeding with the identification of 96-hour LC50, healthy acclimatized fishes was exposed to 1/8th of LC50 under static renewal conditions for 96 hours. Several metabolic enzymes activities of Succinate Dehydrogenase (SDH), Malate Dehydrogenase (MDH) and Lactate Dehydrogenase (LDH), along with glycogen contents was estimated in five major tissues. Tissues are as follows gills, liver, brain, intestine and muscles. Exposure to Halosulfuron-methyl, produced a significant and tissue-specific alterations in all parameters. SDH and MDH activities was markebly reduced across in all tissues, this clearly indicating an impairment of aerobic mitochondrial metabolisms and disruptions of the tricarboxylic acid (TCA) cycle. LDH activity was significantly found elevated in all tissues, this clearly indicating a shift toward anaerobic glycolysis from aerobic glycolysis. Glycogen content was degenerated considerably in gills, liver, brain, intestine and muscle, this clearly providing a direct evidence of carbohydrate mobilizations under herbicide induced metabolic stress. The liver showed most biochemical disruptions, consistent with its primary role in detoxification. These findings confirm the potentiality of SDH, MDH, LDH and tissue glycogen as a dependable and sensitive biomarkers for evaluating herbicide induced toxicity in aquatic organisms and this evidenced and highlighting the ecological hazards caused by Halosulfuron-methyl to freshwater ecosystems.*

Keywords *— **Halosulfuron-methyl, Cyprinus carpio, Succinate Dehydrogenase, Malate Dehydrogenase, Lactate Dehydrogenase, Glycogen, Sub-lethal toxicity.***

I. INTRODUCTION

The development of agricultural activities from recent years has directed to extensive use of herbicidal applications in different crop managements, resulting in progressive accumulations in freshwater ecosystems. Accumulations of herbicide occurs through surface runoffs, percolation and leaching activities (Aktar *et al.*, 2009; Igbedioh, 1991). Once herbicidal chemical compounds enters to aquatic environments, they clearly exerts toxicological pressures on aquatic organisms, among which freshwater fishes are particularly seviourly vulnerable to herbicides and fishes survive with intimate contact with the surrounding water environment. Halosulfuron-methyl ($C_{13}H_{15}ClN_6O_7S$), was a broad-spectrum sulfonylurea herbicide, which is extensively used in the control of sedges and broadleaf weeds in crops, main crops it is used as herbicides such as rice, sugarcane, maize and bottle gourd. Its primary role of biochemical mode of action involves the selectively inhibition of acetolactate synthase

enzyme (ALS), this enzyme plays a major role in the biosynthesis of branched chain amino acids in plants (Zhang and Zhao, 2017). Although this clear mechanism specifically targets plant biochemistry, this accumulating evidence clearly indicates that sulfonylurea herbicides can induce measurable toxicological effects in aquatic organisms if come in contact with aquatic environment, Halosulfuron-methyl also causes disruption of several critical metabolic pathways (Kavitha and Rao, 2026; Sharma *et al*., 2026).

Metabolic enzymes are very sensitive indicators of sub-lethal herbicidal toxicity in freshwater fish. Several enzymes plays a very crucial roles in maintaining physiological activities, such as Succinate dehydrogenase, malate dehydrogenase and Lactate dehydrogenase. Succinate dehydrogenase is a mitochondria bound enzymes that plays major role in electron transport chain at Complex II and as a main and important component of the Tricarboxylic acid cycle, it plays fundamental role in aerobic energy generation. Any critical disturbances of Succinate dehydrogenase activities directly compromises with oxidative phosphorylation and cellular Adenosine triphosphate productions (Bhattacharya and Bhattacharya, 1997). Malate dehydrogenase catalyzes the reversible interconversion of malate to oxaloacetate and oxaloacetate to malate in multiple ways, it acts as central enzymes to both Tricarboxylic acid cycle and gluconeogenesis. Its inhibition signals causes alteration and disturbance of normal biological functions, pathways, of aerobic energy metabolisms. Lactate dehydrogenase is a cytoplasmic enzyme plays a major role in pyruvate to lactate conversions under anaerobic conditions. It is a recognized indicator of cellular injury (Bojarski and Witeska, 2018). Tissue glycogen contents serves as a direct index of carbohydrate energy reserves, its reductions under toxicological chemical stress shows there is a increased metabolic demands, associated with detoxification and physiological stress responses (Bhattacharya and Bhattacharya, 1997).

Common carp (*Cyprinus carpio* L.) is one of the most widely culturable freshwater aquatic fish species around the world, it holds a considerable ecological and economic significances in Indian aquaculture reservoirs. Its having several visualized characters like broad environmental tolerances, physiological responsiveness and sensitivity to chemical pollutants establishes *C. carpio* as an internationally accepted model organism for ecotoxicological studies and investigations (Abedi *et al*., 2013). The present research study, therefore, aimed to evaluate the effects of sub lethal concentrations of Halosulfuron-methyl on the activities of SDH, MDH and LDH, as well as on tissue glycogen contents, in five major tissues of *C. carpio*. Clearly understanding herbicidal alarms to *C. carpio* at a microscopic levels.

II. MATERIALS AND METHODS

2.1 Experimental Fish and Acclimatization

Healthy species of *Cyprinus carpio L*. having mean body weight 6 ± 0.3 g, mean total length 7 ± 0.5 cm was procured from a state fisheries farm in Turvekere, Karnataka India. This was maintained by state government fisheries department as per standard recommended protocols. Procured fishes *C.carpio* was maintained in glass aquaria having 60 L capacity, supplied with dechlorinated tap water at an ambient temperature of 26 ± 1 °C, under a natural photoperiods. Fish were acclimatized for 25 days prior to experimentation and fed daily with commercial fish pellets (Optium, Aquatic P. feed). Feeding was withdrawn before 24 hours, before the commencement of bioassays. Water quality parameters was monitored in accordance with standard methods (APHA, 2025).

2.2 Toxicant and LC50 Determination

Halosulfuron-methyl was obtained from a certified agrochemical supplier with in a local market. The 96 hour median lethal concentration (LC50) was determined in a previous phase of this research work, using the probit analysis method following OECD (1992) guidelines and was experimentally recognized at 557 mg/L with a strong dose response relationships (Darshan Kumar and Parimala, 2026).

2.3 Sub-lethal Toxicity Bioassay

For sub-lethal toxicity studies, fishes was exposed to 1/8th of the 96-hour LC50, that is at 69.6 mg/L of test chemical Halosulfuron-methyl, under static renewal bioassay conditions for 96 hours. Control groups was separately maintained

simultaneously under normal water conditions, without a test chemical. The bioassay water was renewed every 24 hours to maintain nominal concentrations of test chemical. Each experimental group comprised with ten fishes and experiment was conducted in triplicate to get accuracy (OECD,1992).

2.4 Tissue Collection and Homogenate Preparation

At the end of the exposure period, fish from both control and exposed groups was anaesthetized and sacrificed. Carefully dissected five major tissues like gills, liver, brain, intestine and muscles on ice, rinsed them in ice cold phosphate buffered saline 0.1 M, pH 7.4 was maintained and immediately processed them for biochemical estimation. Tissue homogenates (10% w/v) was prepared in the appropriate buffers for each assay and centrifuged at 3,000 rpm for 10 minutes at 4 °C. All five major tissues supernatant was used for enzyme assays (Carroll *et al*., 1956).

2.5 Biochemical Estimations

2.5.1 Succinate Dehydrogenase (SDH)

Succinate dehydrogenase activity was estimated by the method of Slater and Bonner (1952). Tissue homogenate was prepared in 0.1 M phosphate buffer at pH 7.4. The reaction mixture contained sodium succinate as substrate and 2, 6 - dichlorophenolindophenol as an electron acceptor. The reduction of 2, 6 - dichlorophenolindophenol was observed spectrophotometrically at 600 nm and final unit was recorded. Succinate dehydrogenase activity was expressed in international units per milligram of protein ie., IU/mg.

2.5.2 Malate Dehydrogenase (MDH)

Malate dehydrogenase activity was measured according to the standard method of Ochoa (1955). The reaction mixture contained L-malate as a substrate and Nicotinamide adenine dinucleotide as a cofactor, with in 0.1 M phosphate buffer at pH 7.4 was maintained. The rate of Nicotinamide adenine dinucleotide hydrogen formation was measured spectrophotometrically at 340 nm and final unit was recorded. Malate dehydrogenase activity was expressed as IU/mg protein as per international standard.

2.5.3 Lactate Dehydrogenase (LDH)

Lactate dehydrogenase activity was estimated following the method of Wroblewski and Ladue (1955). The reaction mixture contained sodium pyruvate as a substrate and Nicotinamide adenine dinucleotide hydrogen as a cofactor in 0.1 M saline phosphate buffer at pH 7.4 was maintained. The oxidation of Nicotinamide adenine dinucleotide hydrogen was monitored spectrophotometrically at 340 nm and final unit was recorded. Lactate dehydrogenase activity was expressed as IU/mg protein as per international standard.

2.5.4 Glycogen Content

In all five tissues glycogen contents was determined by using anthrone reagent method of Carroll *et al*., (1956). All five tissue samples was digested in 30% Potassium hydroxide at 100°C and glycogen was precipitated with absolute ethanol by dissolving in distilled water. The anthrone reagent was added finally, resultant green coloured complex was monitored spectrophotometrically at 620 nm against a glucose standards and final results was recorded. Glycogen was expressed as mg per gram wet tissue (mg/g).

2.5.5 Protein Estimation

Total protein concentrations in all five tissue homogenates was determined by the method of Lowry *et al*., (1951) using Bovine serum albumin as a standard. The blue coloured complex was formed with Folin Ciocalteu reagent was spectrophotometrically measured at 660 nm and final results was recorded.

III. RESULTS

2.6 SDH, MDH, LDH and Glycogen in Gill Tissue

Sub lethal exposure of *C. carpio* to Halosulfuron-methyl resulted in a noticeable reduction in SDH activities in gill tissue, declining from 6.00 IU/mg in controls to 2.00 IU/mg in a test chemical exposed fish. Similarly, MDH activity was decreased from 83.00 IU/mg in controls to 47.00 IU/mg in a test chemical exposed fish. In contrast, LDH activity was increased significantly from 312.00 IU/mg in controls to 426.00 IU/mg in test chemical exposed fish. Finally, gill glycogen contents declined from

2.50 mg/g in controls to 1.20 mg/g in a test chemical exposed fish (Table 1; fig 1).

Table 1. Biochemical parameters in gill tissue of control and Halosulfuron-methyl exposed Cyprinus carpio L.

Parameter	Control	Exposed	Unit
SDH	6.00	2.00	IU/mg protein
MDH	83.00	47.00	IU/mg protein
LDH	312.00	426.00	IU/mg protein
Glycogen	2.50	1.20	mg/g wet tissue

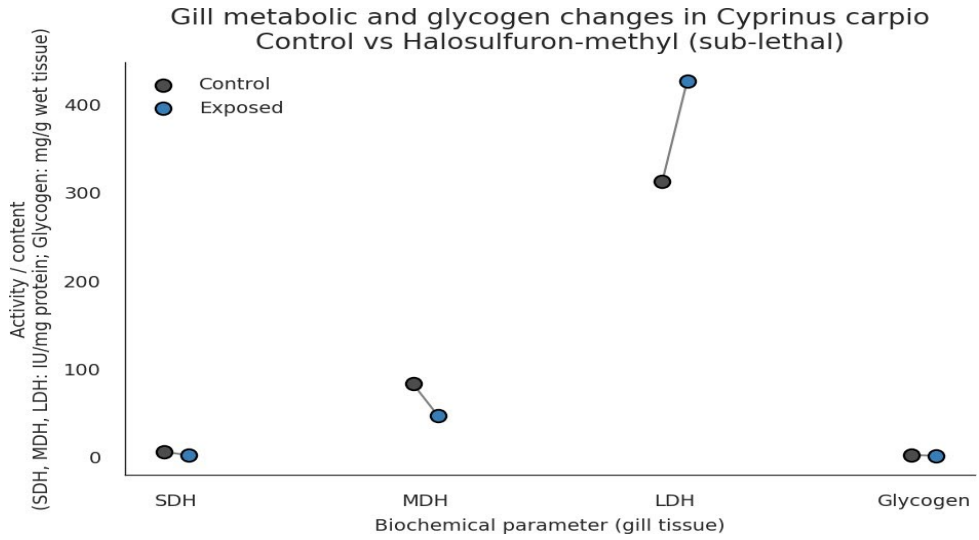


Fig.1: Biochemical parameters of gill tissue control vs exposed

2.7 SDH, MDH, LDH and Glycogen in Liver Tissue

The liver showed most noticeable alterations among all five tissues examined. SDH activity declined from 22.00 IU/mg in controls to 7.00 IU/mg in test chemical exposed fish, clearly representing a reduction was approximately 68%. MDH activity declined substantially from 226.00 IU/mg in controls to

38.00 IU/mg in test chemical exposed fish. LDH activity was evidently higher, rising from 320.00 IU/mg in controls to 562.00 IU/mg in test chemical exposed fish. Hepatic glycogen content was reduced by 50% from 32.00 mg/g in controls to 16.00 mg/g in the test chemical exposed fish (Table 2).

Table 2. Biochemical parameters in liver tissue of control and Halosulfuron-methyl exposed Cyprinus carpio L.

Parameter	Control	Exposed	Unit
SDH	22.00	7.00	IU/mg protein
MDH	226.00	38.00	IU/mg protein
LDH	320.00	562.00	IU/mg protein
Glycogen	32.00	16.00	mg/g wet tissue

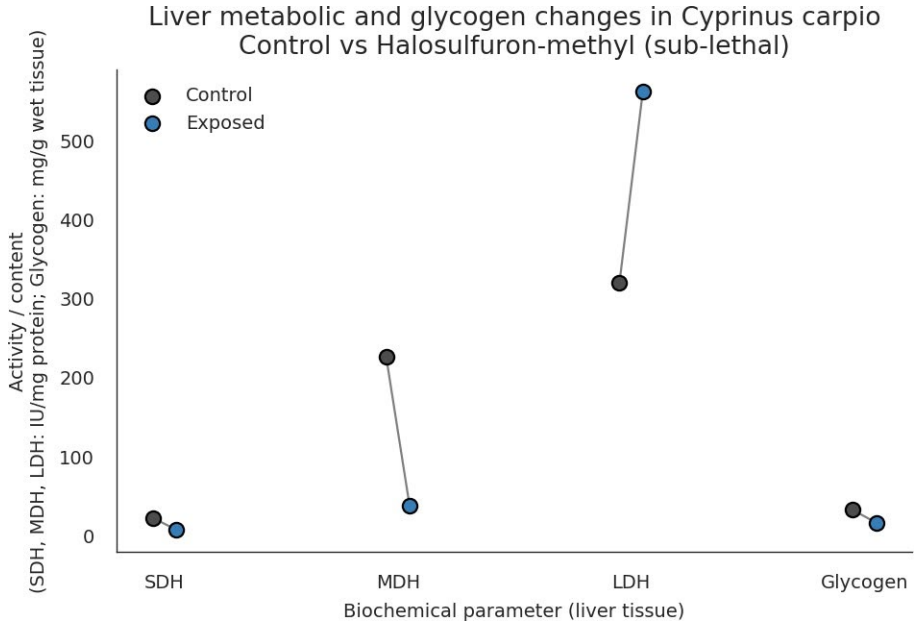


Fig.2: Biochemical parameters of Liver tissue control vs exposed

2.8 SDH, MDH, LDH and Glycogen in Brain Tissue

Brain tissue also showed a significant biochemical alteration. SDH activity was reduced from 22.00 IU/mg in controls to 7.00 IU/mg in test chemical exposed fish. MDH activity declined from 202.00 IU/mg in controls to 122.00 IU/mg in test chemical exposed fish. LDH activity raised from 325.00 IU/mg in controls to 533.00 IU/mg in test chemical exposed fish. Brain glycogen content was reduced by 50% declining from 3.00 mg/g in controls to 1.50 mg/g in test chemical exposed fish, clearly suggesting there is a significant damage to neural energy reserves (Table 3).

Table 3. Biochemical parameters in brain tissue of control and Halosulfuron-methyl exposed Cyprinus carpio L.

Parameter	Control	Exposed	Unit
SDH	22.00	7.00	IU/mg protein
MDH	202.00	122.00	IU/mg protein
LDH	325.00	533.00	IU/mg protein
Glycogen	3.00	1.50	mg/g wet tissue

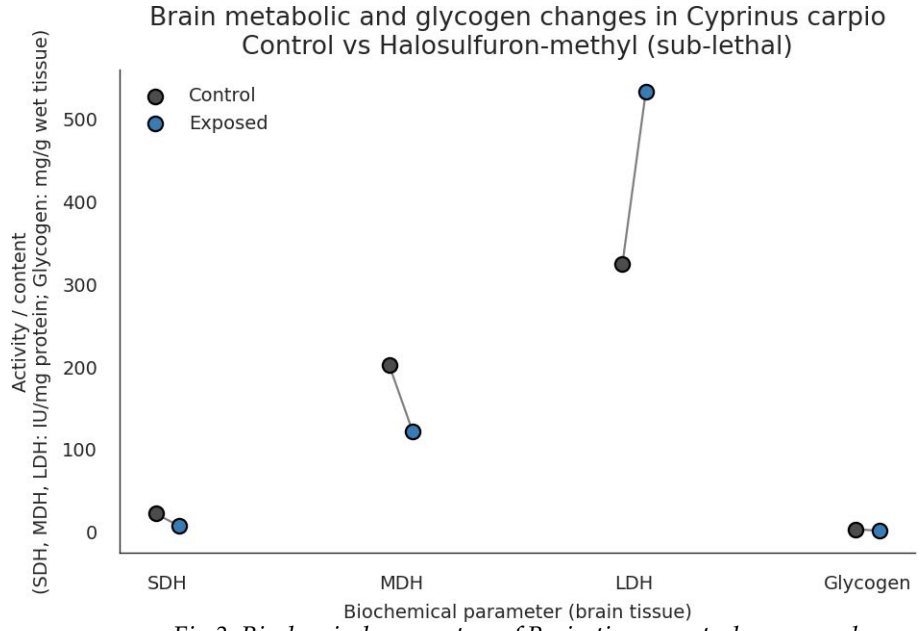


Fig.3: Biochemical parameters of Brain tissue control vs exposed

2.9 SDH, MDH, LDH and Glycogen in Intestinal Tissue

Intestinal tissue showed a reduction in SDH activity from 16.00 IU/mg in controls to 6.00 IU/mg in test chemical exposed fish. There is a clear decline in MDH activity from 185.00 IU/mg in controls to 87.00 IU/mg in test chemical exposed fish. LDH activity increased considerably from 226.00 IU/mg in controls to 428.00 IU/mg in test chemical exposed fish. Glycogen content was significantly depleted, declining from 16.00 mg/g in controls to 7.00 mg/g in test chemical exposed fish (Table 4).

Table 4. Biochemical parameters in intestinal tissue of control and Halosulfuron-methyl exposed Cyprinus carpio L.

Parameter	Control	Exposed	Unit
SDH	16.00	6.00	IU/mg protein
MDH	185.00	87.00	IU/mg protein
LDH	226.00	428.00	IU/mg protein
Glycogen	16.00	7.00	mg/g wet tissue

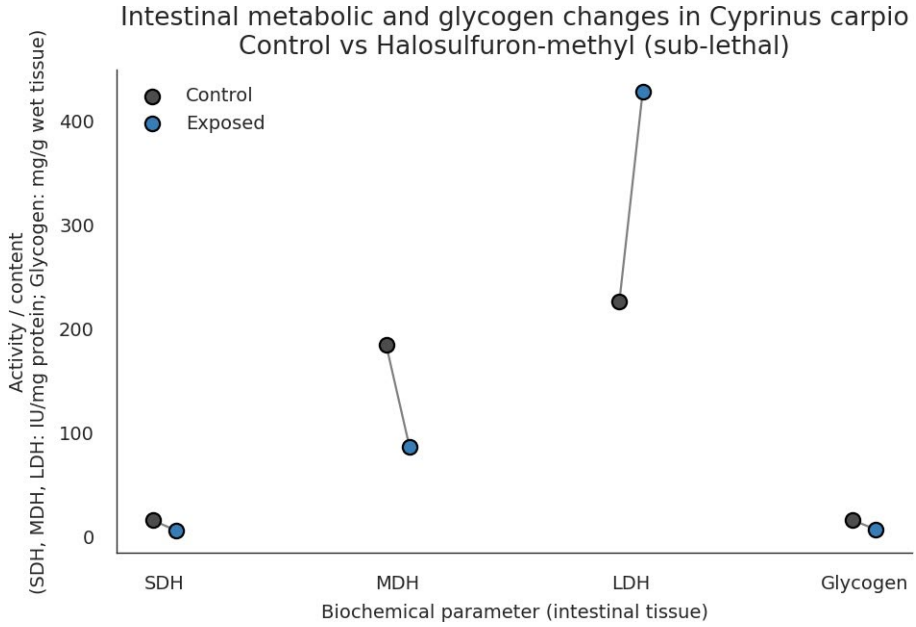


Fig.4: Biochemical parameters of Intestinal tissue control vs exposed

2.10 SDH, MDH, LDH and Glycogen in Muscle Tissue

Muscle tissue showed a most severe reduction in SDH activity, declining from 14.00 IU/mg in controls to 2.00 IU/mg in test chemical exposed fish, this clearly indicating serious impairments of locomotor energy metabolisms. MDH activity decreased from 132.00 IU/mg in controls to 62.00 IU/mg in test chemical exposed fish. LDH activity was markedly elevated from 489.00 IU/mg in controls to

732.00 IU/mg in test chemical exposed fish. Muscle glycogen content was drastically depleted, declining from 14.00 mg/g in controls to 3.00 mg/g in test chemical exposed fish, this clearly representing a loss of approximately 79% of glycogen reserves (Table 5).

Table 5. Biochemical parameters in muscle tissue of control and Halosulfuron-methyl exposed Cyprinus carpio L.

Parameter	Control	Exposed	Unit
SDH	14.00	2.00	IU/mg protein
MDH	132.00	62.00	IU/mg protein
LDH	489.00	732.00	IU/mg protein
Glycogen	14.00	3.00	mg/g wet tissue

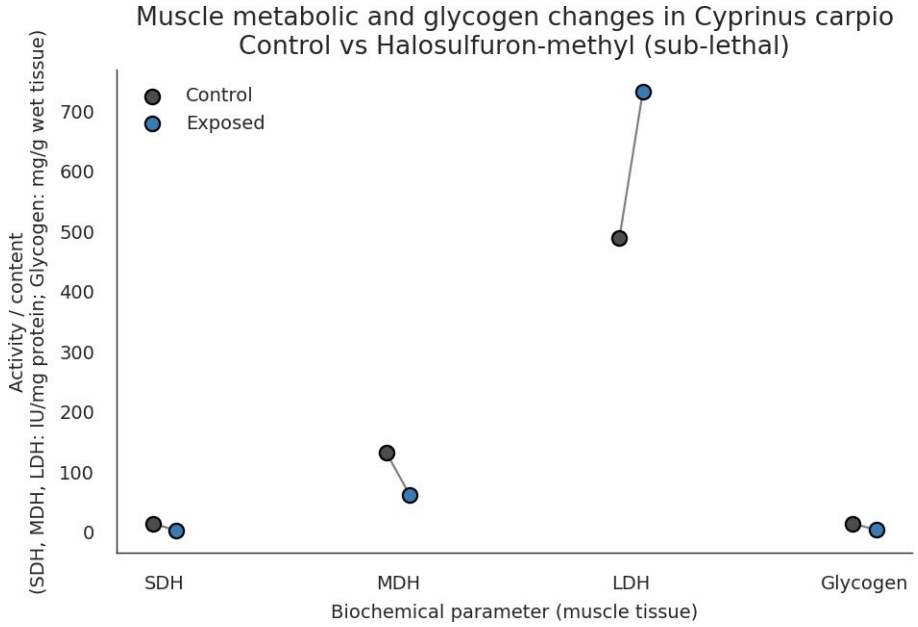


Fig.5: Biochemical parameters of muscle tissue control vs exposed

IV. DISCUSSION

The results of present research study provides clear biochemical evidences that sub lethal exposure to Halosulfuron-methyl induces significant tissue specific disruptions in metabolic enzyme activities and also in carbohydrate energy reserves in *Cyprinus carpio* L. The consistent patterns of reduced SDH and MDH activities and elevated LDH activities across all five tissues of *C. carpio* is a clear symptomatic indicative of a progressive shift from aerobic to anaerobic energy metabolisms, This mechanism explains clearly that there is a clear hallmark of induced chemical stress in freshwater fishes. Significant declining in SDH activities was observed in all five tissues of herbicide exposed fish shows particular mechanisms, since SDH is exclusively situated in the intersection of the Tricarboxylic acid cycle and mitochondrial electron transport chain. Its inhibition clearly impairs oxidative phosphorylation and directly reduces cellular Adenosine triphosphate generations. Similar reductions in SDH activities have documented in *Cyprinus carpio* L. exposed to cadmium (Bhattacharya and Bhattacharya, 1997).

Following sub lethal exposure to metsulfuron-methyl in *Labeo rohita* (Kavitha and Rao, 2026) and chlorimuron ethyl treated with *C. carpio* (Sharma *et al*., 2026) all these are previous evidences align with present investigations. In present research study, muscle tissue recorded the most severe SDH reductions from 14.00 in control to 2.00 IU/mg in herbicide exposed *C. carpio* fish, pointing to extensive impairment of locomotor capacity in fish. Additionally, these observations are reliable with the behavioral disturbances of previously documented results (Darshan and Parimala, 2026).

The associated decline in MDH activities across all five examined tissues explains that there is a disruption of aerobic energy metabolisms. MDH participates in Tricarboxylic acid cycle, where it catalyzes the conversion of malate to oxaloacetate in gluconeogenesis mechanism. Its inhibition results in reduced delivery of oxaloacetate to the Tricarboxylic acid cycle, thereby intensifying the energy deficits initiated by SDH impairment. The liver showed the most affected MDH reductions from 226.00 in control to 38.00 IU/mg in herbicide exposed fish, Liver as the primary organ responsible for herbicide biotransformation, liver undergoes significant metabolic stress during the detoxification. This research study was consistency giving additional evidences pointing to a studies conducted by Zhang and Zhao (2017), they employing a GC-MS metabolomics approaches in zebrafish. Fishes are exposed to acetamiprid insecticide, identified a significant disruption in Tricarboxylic acid cycle

intermediates, additionally causing disruptions at malic and fumaric acid levels this confirmed critically that liver is the primary target organ for sub-lethal herbicide toxicity.

The marked and consistent elevation of LDH activities across all five tissues reflects a compensatory activation of anaerobic glycolysis in response to the energy deficits caused by mitochondrial dysfunctions. Anaerobic glycolysis is a less efficient adenosine triphosphate generating pathway and is associated by lactate accumulations, which further disturbs intracellular pH and enzyme functions. Elevated LDH activities is a recognized biomarker of cellular damage and the activation of anaerobic metabolism (Abedi *et al*., 2013; Bhattacharya and Bhattacharya, 1997), a physiological response similarly triggered by exposure to herbicides such as Paraquat (Moron *et al*., 2025) and 2, 4 - D dimethylamine salt (Hamisu *et al*., 2024). The highest LDH elevation in the present research study was observed in muscle tissue from 489.00 in control to 732.00 IU/mg in herbicide exposed fish, indicating a compensatory shift towards anaerobic glycolysis to meet the urgent Adenosine triphosphate demands of locomotor tissues under chemical stress.

Glycogen depletion was also observed in all five tissues in herbicide exposed fish. The most noticeable depletion was found in muscle tissue from 14.00 in control to 3.00 mg/g in herbicide exposed fish, while the most significant proportional decline was noted in gill tissue from 2.50 in control to 1.20 mg/g in herbicide exposed fish. Glycogen depletion under chemical stress provides direct clear evidence in increased carbohydrates level to meet higher demands of cellular detoxification and stress response mechanisms. Consistent with the findings of the present study, prior research has documented comparable glycogen depletion in *Colossoma macropomum* exposed to Paraquat (Moron *et al*., 2025), *Clarias gariepinus* following glyphosate based herbicide exposure (Fagbenro *et al*., 2025) and *C. carpio* exposed to Isoproturon (Devikarani and Parimala, 2025).

Among all five tissues, liver exhibited the most severe and multifactorial biochemical disruptions, characterized by significant SDH and MDH inhibitions, hepatic glycogen depletions and elevated LDH, liver displayed the most severe biochemical disruptions, confirming its high susceptibility to herbicide-induced metabolic stress (Bojarski and Witeska, 2018; Zhang and Zhao, 2017). Additionally, Brain tissue also showed significant alterations in all biochemical parameters, documenting the neurotoxic potential of Halosulfuron-methyl and supporting behavioral disturbances reported in species *C. carpio* following herbicide exposures (Devikarani and Parimala, 2025; Darshan Kumar and Parimala, 2026). Enzymatic and glycogen modifications in the gills indicates there is a direct impairment of respiratory functions and branchial metabolisms.

Collectively, all biochemical profiles documented in the present research study reveals there is tissue specific reductions in SDH and MDH alongwith elevation of LDH and depletion of glycogen reserves. Aligning with previous finding of sulfonylurea herbicides in freshwater fish (Kavitha and Rao, 2026; Sharma *et al*., 2026). These results confirms the appropriateness of SDH, MDH, LDH and tissue glycogen as tissue specific biomarker for the assessment of sub lethal herbicide toxicity in aquatic organisms. This highlights risks caused by Halosulfuron-methyl to freshwater aquatic life. The research findings highlights critical need for monitoring sulfonylurea herbicide residues in aquatic areas, as controlled by Central insecticides board and registration committee (CIBRC) and the Food safety and standards authority of India (FSSAI) (Mishra *et al*., 2006; Ullah *et al*., 2014).

V. CONCLUSION

Sub lethal exposure of Halosulfuron-methyl to *Cyprinus carpio* L. at 69.6 mg/L of 1/8th of LC50 for 96 hours incorporated a significant tissue specific disruptions in all metabolic enzyme activities and carbohydrate energy reserves. SDH and MDH activities was significantly reduced in all five examined tissues. Tissues like gills, liver, brain, intestine and muscle clearly shows there is damage to cellular organells it affects mitochondrial function and disruption of the tricarboxylic acid cycle. LDH activities was shown to be significantly raised in all five tissues, clearly demonstrating there is a activation of anaerobic metabolic pathway. Tissue glycogen content was markedly depleted, this

providing an direct evidence of increased energy demands and carbohydrate mobilization under herbicide induced stress. Liver tissue shows major disruptions compared to other tissues. These all critical findings in research study explains clearly that SDH, MDH, LDH and glycogen acts as sensitive biomarkers to Halosulfuron-methyl toxicity in freshwater fish *C. carpio*, and this highlights the warnings of sulfonylurea herbicide to aquatic ecosystem.

ACKNOWLEDGEMENTS

The authors are grateful to the University College of Science, Department of Studies and Research in Zoology, Tumkur University, Tumakuru, for providing the necessary laboratory facilities to carry out this research work.

REFERENCES

[1] Abedi, Z., Hasantabar, F., Khalesi, M.K. and Babaei, S. (2013). Enzymatic activities in common carp; *Cyprinus carpio* influenced by sublethal concentrations of cadmium, lead and chromium. *Journal of Survey in Fisheries Sciences*, 377–422.

[2] Aktar, M.W., Sengupta, D. and Chowdhury, A. (2009). Impact of herbicides use in agriculture: their benefits and hazards. *Interdisciplinary Toxicology*, 2(1), 1–12.

[3] APHA (2025). Standard Methods for the Examination of Water and Wastewater, 24th edition. APHA Press, Washington DC.

[4] Bhattacharya, T. and Bhattacharya, S. (1997). Cadmium-induced changes in metabolic enzyme activities in *Cyprinus carpio*. *Environmental Pollution*, 95(2), 215–220.

[5] Bojarski, M. and Witeska, M. (2018). Physiological and histological effects of herbicides in fish. *Annals of Warsaw University of Life Sciences*, 57.

[6] Carroll, N.V., Longley, R.W. and Roe, J.H. (1956). The determination of glycogen in liver and muscle by use of anthrone reagent. *Journal of Biological Chemistry*, 220(2), 583–593.

[7] Darshan Kumar, T. and Parimala, B. (2026). Determination of LC_{50} and behavioral modifications in *Cyprinus carpio* L. exposed to Halosulfuron-methyl herbicide. *International Journal of Fisheries and Aquatic Research*, 11(2), 55–59.

[8] Devikarani, H.K. and Parimala, B. (2025). Estimation of LC_{50} and behavioral modifications of *Cyprinus carpio* (L.) in response to Isoproturon Herbicide. *International Journal of Environmental Sciences*, 11(4s), 83–92.

[9] Fagbenro, O.S., Akinyanju, T.F. and Ajisebiola, B.S. (2025). Glyphosate-based herbicide induced cytogenetic, haematological and histopathological responses in *Clarias gariepinus* (Burchell, 1882). The *Nucleus*, 1–12.

[10] Hamisu, Y., Auta, J. and Bawa, B.S. (2024). Toxicity of the herbicide 2,4-D dimethylamine salt on behavioral, growth and hematology of *Clarias gariepinus* (Burchell, 1822) fingerlings. *Nigerian Journal of Fisheries and Aquaculture*, 12(2), 19–29.

[11] Igbedioh, S.O. (1991). Effects of agricultural herbicides on humans, animals and higher plants in developing countries. *Archives of Environmental Health: An International Journal*, 46(4), 218–224.

[12] Ivantsova, E. and Martyniuk, C.J. (2024). Environmental presence and toxicological outcomes of the herbicide pendimethalin in teleost fish. *Ecotoxicology*, 33(6), 531–545.

[13] Kavitha, S. and Rao, P.R. (2026). Metabolic enzyme alterations in *Labeo rohita* exposed to sub-lethal concentrations of metsulfuron-methyl herbicide. *Journal of Aquatic Biology and Fisheries*, 14(1), 55–68.

[14] Lowry, O.H., Rosebrough, N.J., Farr, A.L. and Randall, R.J. (1951). Protein measurement with the Folin phenol reagent. *Journal of Biological Chemistry*, 193(1), 265–275.

[15] Mishra, D.K., Bohidar, K. and Pandey, A.K. (2006). Responses of interrenal cells of freshwater teleost, *Channa punctatus* (Bloch), exposed to sublethal concentrations of carbaryl and cartap. *Journal of Ecophysiology and Occupational Health*, 6(3–4), 137–141.

[16] Moron, S.E., Pereira Lacerda Vieira, L. and Libanio Reis Santos, E. (2025). Acute toxicity and sublethal effects of Paraquat in *Colossoma macropomum*. *Chemistry and Ecology*, 1–16.

[17] Ochoa, S. (1955). Malic dehydrogenase from pig heart. *Methods in Enzymology*, 1, 735–739.

[18] OECD (1992). OECD Guidelines for the Testing of Chemicals, Section 2, Test No. 203: Fish, Acute Toxicity Test. *Organisation for Economic Cooperation and Development*, Paris.

[19] Qayoom, I. (2025). Toxicological evaluation and effects of organophosphate compounds on hematological profile of juvenile common carps (*Cyprinus carpio*). *Indian Journal of Animal Research*, 25(4), 28–36.

[20] Sharma, R., Verma, A. and Negi, S. (2026). Biochemical and metabolic responses of *Cyprinus carpio* to chlorimuron-ethyl herbicide: enzyme activity and glycogen dynamics. *Ecotoxicology and Environmental Safety*, 289, 117045.

[21] Slater, E.C. and Bonner, W.D. Jr. (1952). The effect of fluoride on the succinic oxidase system. *Biochemical Journal*, 52(2), 185–196.

[22] Ullah, R., Zuberi, A., Ullah, S., Ullah, I. and Dawar, F.U. (2014). Cypermethrin induced behavioral and biochemical changes in mahseer, *Tor putitora*. *The Journal of Toxicological Sciences*, 39(6), 829–836.

[23] Wroblewski, F. and Ladue, J.S. (1955). Lactic

dehydrogenase activity in blood. Proceedings of the Society for *Experimental Biology and Medicine*, 90(1), 210–213.

[24] Zhang, H. and Zhao, L. (2017). Influence of sublethal doses of acetamiprid and halosulfuron-methyl on metabolites of zebra fish (*Brachydanio rerio*). *Aquatic Toxicology*, 191, 85–94.