

Toxicity Of Malathion On Some Biochemical Changes On Kidney Of Fish *Channa Punctatus* (Bloch.)

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Abstract: Malathion is an organophosphorous insecticide widely used in agricultural and non agricultural purposes in India, creates a serious threat to the environment as well as target and non target organisms. the objective of the study was to determine the toxicity of malathion at sublethal concentration for 28 days on certain biochemical parameter including blood urea, creatinine and blood urea nitrogen of fresh water fish *channa punctatus* (Bloch.). The sub lethal concentration was 0.09ml (1/5 of LC50) of malathion for which the fish was exposed at different time intervals 7, 14, 21 and 28 days. The present study showed statistically increase value in blood urea, creatinine and blood urea nitrogen.

I. INTRODUCTION

Pollution due to pesticides needs considerable attention because of the toxicants, which lack the capacity to dramatically increase the rate of mortality in exposed population, may still cause their ecological death after a long time of exposure, probably as a result of the cumulative effect of impaired metabolic functions (Ghosh and Shrotri, 1992). Such disrupted activities may also occur in non-target organisms. These activities lead to changes in the composition of different tissues, which may serve as sensitive criteria on the effect of chronic toxicity of the pesticides (Colianese and Neff, 1982). Aquatic toxicologists assess the physiological aspects of fishes living in polluted waters (Gingerich and Weber, 1979). The biochemical changes occurring in the body of the organisms give first indication of stress. Several investigators have reported a number of changes in biochemical parameters of aquatic organisms due to pesticidal exposure Remia *et al.*, 2008; Patil and David, 2007 and Vijakumar *et al.*, 2009). Among these pesticides, organophosphorous compounds are commonly used insecticides. Malathion is commonly use organophosphorous pesticide. Once malathion is introduced into the environment,

it may cause serious intimidation to aquatic organisms and is notorious to causes evere metabolic disturbances in non target species like fish and freshwater mussels.

II. MATERIALS AND METHODS

Fresh-water catfish, *Channa punctatus* has been selected as a model specimen for the present investigation. The active and healthy specimens of fish were collected from the local fish market in the month of December. The weight and length of the experimental animals varied between 55– 65g and 15 – 18 cm respectively. . The fishes were carefully analyzed and treated before stocking with 0. 2% KMnO₄ solution to get rid off any dermal infection of water. They were supplied daily with commercial fish food at a rate of 2.5% body weight. They were acclimatized to the laboratory conditions for 15 days before exposure to experimental chemical from unfavorable gases. The dechlorinated water used was changed every alternative day. Various physiochemical characteristics of test water such as temperature, pH and hardness were regularly reported. The feeding was stopped 24 hours prior to the exposure period. One set of fishes was maintained as control

besides experimental group in tap water. The experiment was conducted in five aquariums one was used for control and the used for the pollution study. The experimental fishes were exposed to sub lethal concentration of 0.9 ml of malathion at different times interval 7, 14, 21, 28 days. The LC₅₀ of malathion was calculated by probit analysis of Finney. Blood from the experiment and control groups was collected from the cut caudal vein into the plain sterilized glass centrifuge tubes. The blood was used for the biochemical estimation of urea by Columbe and Favreau method (1965), Creatinine by Bones and Tausky Method (1945) and Blood Urea nitrogen by Talke and Schubert (1979). *Channa punctatus* exposed to sub lethal concentration of 0.09 ml of Malathion at different intervals of time exhibit many biochemical alterations have been summarized in tables.

III. RESULTS AND DISCUSSION

Channa punctatus exposed to sub lethal concentration of 0.09 ml of Malathion at different intervals of time exhibit many biochemical alterations have been summarized in tables.

EFFECT OF MALATHION ON BLOOD UREA

Higher levels of blood urea are abnormal and indicate a severe disorder of kidney. Urea revealed significant increment in blood urea of *channa punctatus* at the different time intervals (7, 14, 21, 28 days) under malathion toxic stress. Higher level of urea might be due to kidney impairment and possible enhancing protein catabolism together with accelerated amino acid deamination for gluconeogenesis is properly acceptable postulate to interpret the rise level of urea in blood serum of *channa punctatus* under malathion stress. The present findings gain support with the findings of Malhotra and Sharma (2003) observed significant increase in blood urea of *channa stratus* under monocil stress. Zaki et al (2007) supported kidney dysfunction may also increase in serum urea in *clarias lazera* under toxic stress. Misaila et al (2008) suggested the presence of urea in blood product resulted from the catabolisation of proteins and amino acids. Zaki et al. (2008) supported serum urea level elevation due to potassium permanganate stress in *Tilapia nilotica* it suggested that kidney impairment.

IV. BLOOD UREA

Biochemical parameter	Control group Range Mean ±S.E.m	Exposure in days			
		7 days range Mean ±S.E.m	14 days range Mean ±S.E.m	21 days range Mean ±S.E.m	28 days range Mean ±S.E.m
Urea (mg/dl)	31.45-37.11 33.09 ± 0.718	50.55-58.42 55.48 ± 1.33***	59.64-68.34 63.80 ± 1.13***	69.50-81.25 75.48 ± 1.28***	92.23-99.55 95.72 ± 1.68***

Results are expressed as ± S.E.m. NS = Non-Significant $p > 0.05$; * = Significant at $p < 0.05$; ** highly significant at $p < 0.01$; *** = very highly significant at $p < 0.01$.

Table 1: Urea content of *channa punctatus* exposed to sub lethal concentration of malathion

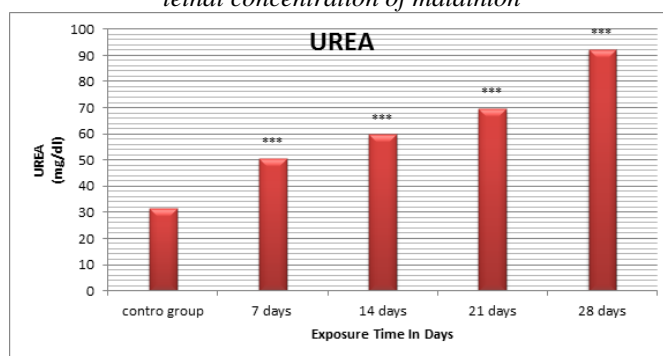


Figure 1: Biochemical estimation of blood urea after malathion intoxication in experimental fish *channa punctatus*.

EFFECT OF MALATHION ON SERUM CREATININE

Creatinine is filtered freely through the glomerulus and is not absorbed in the tubules therefore creatinine is more reliable to measure of SGR. So creatinine is a more reliable measure to GFR and renal insufficiency. It is more specific indicator to renal disease. Creatinine level showed significant in blood serum of *channa punctatus* under the stress of Malathion. It may be possible of glomerular insufficiency due to renal injury causing tubular cell necrosis by the direct action of malathion and increase muscle tissue catabolism. The present findings gain support with Jyothi and Narayan (2000) observed increase serum creatinine in *clarias batrachus* under pesticidal stress. Abdelmeguid et al. (2002) indicated a significant rise in serum creatinine concentration in fish *tilapia zilli* due to water pollution. Hassan et al. (2007) suggested higher level of creatinine level in *tetodon patoca* under toxic profile main effect on kidney function. Sastry K.V. et al. (2010). Acute and toxic effects of the fresh water teleost, *channa punctatus*. Zhang et al. (2011) supported rise level of creatinine due to stress in Crucian carp suggested kidney impairment.

SERUM CREATININE

Biochemical parameter	Control group Range Mean ±S.E.m	Exposure in days			
		7 days range Mean ±S.E.m	14 days range Mean ±S.E.m	21 days range Mean ±S.E.m	28 days range Mean ±S.E.m
Creatinine (mg/dl)	1.29-1.65 1.4 ± 1.0	2.20-2.54 2.29 ± 0.349 ^{NS}	3.18-3.32 3.23 ± 0.068***	5.18-5.38 5.24 ± 0.065***	7.17-7.38 7.24 ± 0.86***

Results are expressed as ± S.E.m. NS = Non-Significant $p > 0.05$; * = Significant at $p < 0.05$; ** highly significant at $p < 0.01$; *** = very highly significant at $p < 0.01$.

Table 2: Creatinine content of *channa punctatus* exposed to sub lethal concentration of malathion

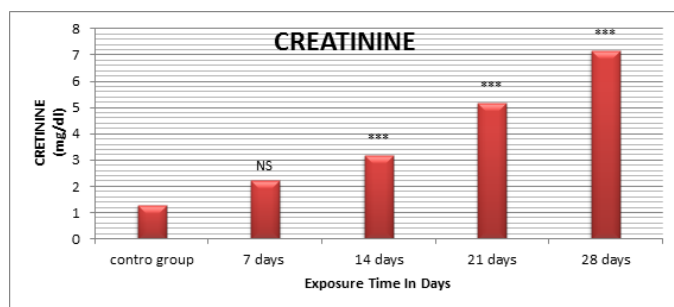


Figure 1: Biochemical estimation of serum creatinine after malathion intoxication in experimental fish *channa punctatus*

V. EFFECT OF MALATHION ON BLOOD UREA NITROGEN

Blood Urea Nitrogen (BUN); Blood urea nitrogen measures the amount of nitrogen, a waste product of protein metabolism in the blood. Urea is formed by the liver and carried by blood to the kidneys for excretion. So this test can be used as a test of renal function. The blood urea nitrogen in the fresh water fish *channa punctatus* shows increasing trend after malathion toxicity at different time intervals (7,14,21,28). The elevated blood urea nitrogen (BUN) indicates low renal perfusion or renal dysfunction due to malathion toxicity which effects harmful effects on kidney tissues. The present findings support by Begum et al.(2004)reported abnormal increase in blood urea nitrogen as a result of liver and renal failure. Kerem et al.(2007)resulted elevated BUN serum after high dose fenthion exposure. It may be renal failure, so kidney function was affected. Zhang *et al.* (2013) resulted BUN increase level in crucian carp under stress suggested the occurrence of kidney impairment.

BLOOD UREA NITROGEN (BUN)

Biochemical parameter	Control group Range Mean ±S.E.m	Exposure in days			
		7 days range Mean ±S.E.m	14 days range Mean ±S.E.m	21 days range Mean ±S.E.m	28 days range Mean ±S.E.m
BUN (mg/dl)	42.20-47.52 44.72 ± 0.70	51.94-58.87 55.26 ± 0.90**	71.60-76.70 73.27 ± 0.764**	80.85-87.72 84.93 ± 0.830**	96.87-99.96 98.16 ± 0.60**

Results are expressed as \pm S.E.m. NS = Non-Significant $p > 0.05$; * = Significant at $p < 0.05$; ** highly significant at $p < 0.01$; *** = very highly significant at $p < 0.01$.

Table 3: Blood urea nitrogen content of *channa punctatus* exposed to sub lethal concentration of malathion

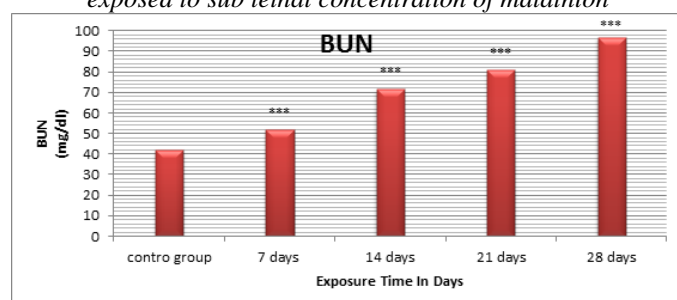


Figure 2: Biochemical estimation of blood urea nitrogen after malathion intoxication in experimental fish *channa punctatus*

VI. CONCLUSION

In the present study Urea, Creatinine and blood urea nitrogen showed increasing trend to sub lethal concentration of malathion on fresh water fish *channa punctatus* because of kidney impairment, glomerular insufficiency and renal injury causing tubular cell necrosis and increase muscle tissue catabolism by the direct action of malathion on fish kidney.

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