

Endrin Toxicosis on Few Enzymes in Liver and Kidney of *Channa punctatus* (BLOCH)

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The various toxic effects by endrin include several pathological changes in gills, liver, brain, gonad and pancreas of *Salmo clarki* (ELLER 1971), retardation of growth, gonad development, thyroid activity, alterations in serum characteristic behaviour and mortality in *Carassius auratus* (GRANT and MEHRLE 1970), damage of central nervous system and reproductive system of *Pimephalus notatus* and *Lebistes reticulatus* (MOUNT 1962) and altered liver function (EISLER and EDMUNDS 1966). FERGUSON et al. (1965), SAANIN (1960) and TARZWELL (1965) have reported that endrin in water is highly toxic to fishes. JOHNSON (1968) and KATZ et al. (1969) reviewed the literature on the effects of various pesticides on fishes.

In the present study, we have examined the effect of endrin on few phosphatases in the liver and kidneys of a fresh water teleost fish, *Channa punctatus*, *in vivo*.

MATERIALS AND METHODS

Animals and treatment:

Living specimens of *Channa punctatus*, 11 ± 3 cm in length and 50 ± 7 g in weight, were collected from local fresh water sources. They were allowed to acclimatize to the laboratory aquaria for three days. In order to observe the effect of a sublethal concentration, the fishes were treated with 0.03 mg/L of endrin in tap water. In each case, twenty-four fishes were examined.

Preparation of tissue homogenates and enzyme assay:

After 15 and 30 days of exposure, both groups of control and experimental fishes were dissected. Liver and kidneys were collected and 10% (W/V) homogenates were prepared in ice cold 0.25 M sucrose solution using a chilled Potter-Elvehjem homogenizer. The homogenates were centrifuged for 20 min at 1000 G and the clear supernatant fluids were used as the source of enzymes. 0.016 M sodium β-glycerophosphate was used as the sub-

trate in pH medium of 5.0 and 9.3 for acid and alkaline phosphatase respectively. The incubation time was one hour. The enzyme activity was estimated according to the method of BODANSKY (1933). SWANSON's method (1965) was followed for the estimation of glucose-6-phosphatase activity. 0.01 M glucose-6-phosphate solution was incubated for 15 min at a pH of 6.5. All the incubations were made at 37°C. The activity is expressed as mg of inorganic phosphate liberated per mg of protein/hr.

Protein determination:

Total protein content of the homogenate was estimated according to the method of LOWRY et al. (1951) with bovine serum albumin as standard.

Statistical methods:

The t-test described by FISHER (1950) was employed to calculate the statistical significance between control and experimental values.

RESULTS AND DISCUSSION

The results of the experiments conducted are given in Table 1. Very little work has been done on the effects of endrin on enzyme activities so an attempt has been made to examine the effect of this pesticide on the activities of acid, alkaline and glucose-6-phosphatases. Exposure of fishes for 15 days to endrin in vivo resulted in the inhibition of activities of acid phosphatase and glucose-6-phosphatase in the liver. This is followed by an elevation above normal level in fishes treated for 30 days. In contrast to liver, kidney revealed an increased activity of all the three enzymes at both the experimental periods. At 15 days of exposure, there is no significant change in acid phosphatase activity but kidney showed an elevation in activity. In our earlier studies (SASTRY and SHARMA 1977a,b) also an elevation was noted by this pesticide in acid phosphatase activity. Cellular damage is usually accompanied by an increase in acid phosphatase activity but according to NOVIKOFF (1961) and DE DUVE (1963), the elevated acid phosphatase activity may be associated with the pre-necrotic changes. Alkaline phosphatase is a brush border enzyme. The elevation in alkaline phosphatase activity in the two tissues at both experimental periods may be due to an increase in transphosphorylation activity. NELSON (1955) found increased levels of alkaline phosphatase in the serum of rats which had been exposed to endrin and this, according to HARPER (1963), is associated with pathological changes. The hydrolysis of glucose-6-phosphate is

TABLE 1
Phosphatase activities in control and experimental fishes^a

Enzyme	Tissue	No. of experiments conducted	Control	Experimentals	
				15 days exposure	30 days exposure
Acid phosphatase	Liver	3	0.027 \pm ^b 0.012	0.026 \pm 0.000(-)	0.096 \pm 0.024(-)
	Kidney	3	0.040 \pm 0.027	0.067 \pm 0.037(-)	0.075 \pm 0.015(-)
Alkaline phosphatase	Liver	4	0.015 \pm 0.013	0.072 \pm 0.025(+) ^c	0.129 \pm 0.015(+)
	Kidney	4	0.013 \pm 0.005	0.017 \pm 0.003(-)	0.217 \pm 0.015(-)
Glucose-6-phosphatase	Liver	4	0.126 \pm 0.092	0.112 \pm 0.068(-)	0.351 \pm 0.013(+)
	Kidney	4	0.068 \pm 0.032	0.092 \pm 0.022(+)	0.193 \pm 0.038(+)

a. The activities are expressed in mg of inorganic phosphate liberated per mg of tissue protein per hour.

b. Values are Mean \pm SE.

c. '+' indicates statistically significant from controls. The 95% confidence interval was used in all t-tests.

a keystone in gluconeogenesis and in the conversion of liver glycogen to blood glucose (HOCHACHAKA 1969). The decrease in glucose-6-phosphatase activity may be attributed due to disturbances in the general metabolism of the cell and due to mitochondrial damage. GRANT and MEHRLE (1973) have reported inhibition of mobilization of liver glycogen by low doses of endrin and blockage by high doses. No possible explanation can be given at this primary stage as further experiments are in progress.

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