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Effects of aldrin on serum and liver constituents of freshwater catfish *Clarias batrachus* L.

YAGANA BANO
Department of Zoology, Aligarh Muslim University, Aligarh 202 001, India

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Abstract. The changes in total protein, total phosphorus, calcium and cholesterol contents were observed in serum and liver of treated and control fish *Clarias batrachus* L. Aldrin caused more pronounced effect in liver than in serum. Values remained significantly low in liver of experimental fish than in control, while the serum constituents fluctuated widely. The variations observed in different values are explained as transfer or development of tolerance. A constant increase of cholesterol in serum corresponding to a regular decrease in liver is interpreted as deportation of cholesterol from liver to serum as a result of liver damage by aldrin poisoning.

Keywords. Aldrin effects ; serum ; liver constituents ; *C. batrachus.*

1. Introduction

Investigations have proved that chlorinated hydrocarbons are highly toxic to fish. Acute toxicity causes damage to the central nervous system, resulting in instability, respiratory difficulties and sluggishness. Other chronic effects are residue accumulation in fats, damage to liver and kidneys, reduced reproduction and restricted growth (Donald 1968). Besides, many alterations have also been reported to occur in blood and tissue chemistry as a result of toxicants. A haemoglobin decrease without a change in erythrocyte count was observed due to the effect of DDT (Rudd and Genelly 1956). Exposure to endrin resulted in increased concentration of sodium, potassium, calcium, and cholesterol in serum with a lower values of sodium, potassium, calcium and zinc in the liver of northern puffer *Sphaeroides maculatus* than control (Eisler and Edmunds 1966). Changes in serum proteins and free amino acids were reported in *Channa punctatus* after exposure to malathion, endrin and dieldrin (Shakoori *et al* 1976). In the same fish Lone and Javaid (1976) observed the variation in blood caused by the effect of DDT and dieldrin.

But so far no such investigation has been reported on aldrin. We report here the changes in chemical constituents of serum and liver tissue of a catfish *Clarias batrachus* which was exposed to various concentrations of aldrin.

2. Material and methods

C. batrachus (23–25 cm in length and weighing 80–100 g) were collected from a local pond and acclimatized for two weeks in a large size aquarium. Fish meal was provided daily up to 24 hr before the aldrin administration.

15 fish were maintained in each of the 5 buckets, containing 20 litres of tap water (pH 6·7, water temperature 25–30° C). Appropriate quantity of technical grade aldrin was dissolved in acetone and the final concentrations of 0·1, 0·2, 0·5 and 1 ppm were added to each of four buckets. The control fish (5th bucket) received only 1 ml of acetone. No fish meal was provided during the experiment. 5 fish from each treatment were sacrificed after 12, 60 and 132 hr of aldrin exposure.

Blood from each fish was collected in a clean test tube after serving the caudal peduncle and was placed in a refrigerator at 10° C for 24 hr. After centrifugation at 3500 r.p.m. for 15 min, serum was drained out and returned to the refrigerator for storage at 4° C. The liver was removed, washed with physiological saline solution and kept in refrigerator till use. The total cholesterol in serum and liver was estimated using the method of Zaltikis *et al* (1953). Other chemical constituents were measured using methods as described by Oser (1965).

3. Results

The 24 hr lethal concentration (LC_{100}) for *C. batrachus* was observed to be 1 ppm aldrin. At lower concentrations, no mortality occurred in experimental fish except for two additional counts at 0·5 ppm.

3.1. *Observations on serum*

As indicated in figure 1, the serum constituents varied markedly with doses and exposure time. At 12 hr exposure the serum protein showed a gradual fall ($P < 0·01$) in all concentrations. Longer exposure (60 and 132 hr) exhibited a rapid fall ($P > 0·01$) in total protein for 0·1 ppm, the constituent rose higher for 0·2 ppm and declined again for 0·5 ppm ($P > 0·01$).

The value of total phosphorus was found increasing with increasing concentration at different exposure times. Total phosphorus, which showed a continuous increase with concentration of 12 hr exposure, declines slightly for 60 hr exposure at 0·2 ppm and more markedly at 0·5 ppm. At 132 hr exposure a small but constant decline of trend was observed for concentration higher than 0·1 ppm.

The cholesterol level showed a continuous increase with aldrin concentration and exposure time, being maximum in fish exposed for 132 hr and minimum in 12 hr exposed fish ($P > 0·01$).

Calcium content declined steadily in fish treated with 0·1 ppm after 12 hr and increased later with rising concentration. At 60 hr exposure the level rose for 0·1 and 0·2 ppm and thereafter declined a little, remaining all the time higher than the control value as well as the corresponding values for 12 hr exposure. For 132 hr exposure the slight increase observed at 0·1 ppm (higher than 60 hr values) was followed by a continuous declining trend for higher concentrations.

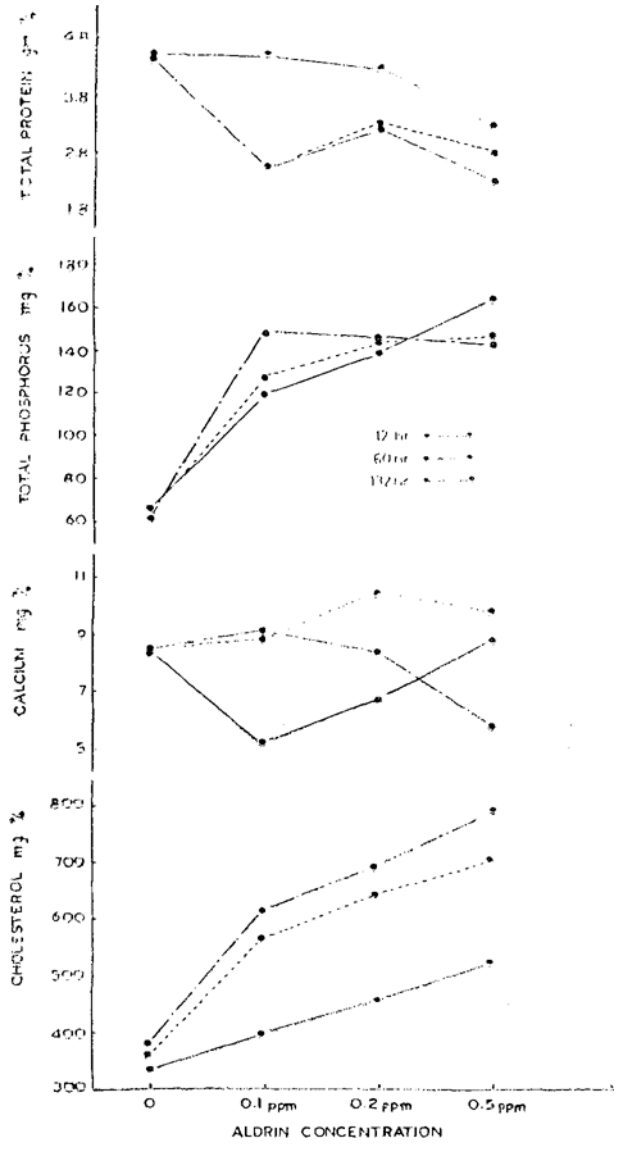

Figure 1. Serum constituents of *Clarias batrachus* for different exposure time.

3.2. *Observations on liver*

The liver constituents exhibited a more regular pattern of variation (figure 2). In all experimental fish the values of protein, calcium and cholesterol content decreased gradually with increasing aldrin toxicity at different exposure times. The value of total protein and calcium was significantly higher for 60 hr exposure ($P > 0{\cdot}05$) than 12 and 132 hr. The decline of protein content for 12 hr exposure was steep and steady up to 0·2 ppm. Thereafter values remained low at higher

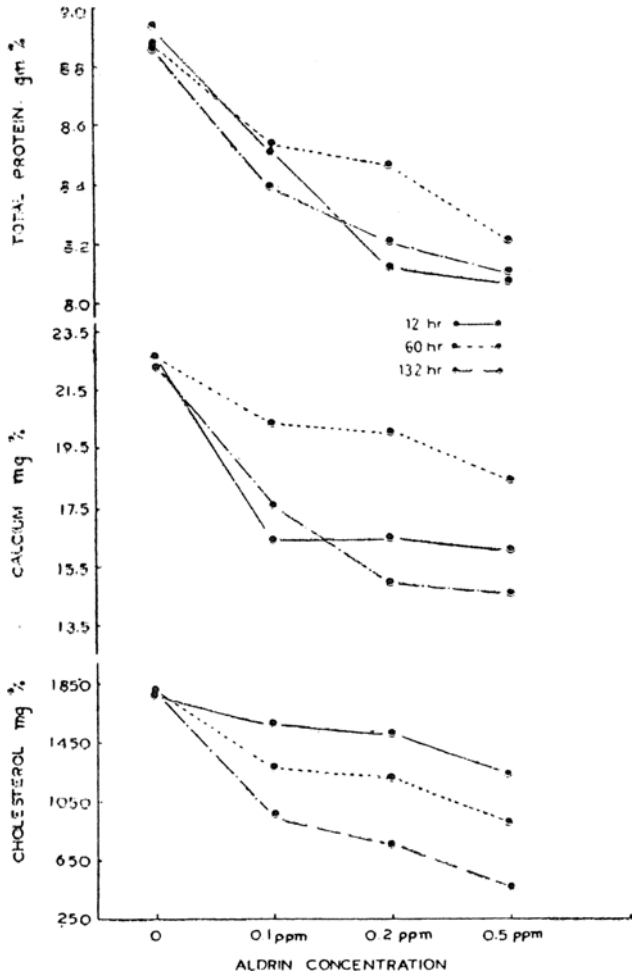

Figure 2. Liver constituents of *Clarias batrachus* for different exposure time.

concentrations. Whereas at this exposure calcium content decreased only up to 0·1 ppm and after that the values were found uniform with rising concentrations.

A slow and steady decrease in cholesterol level was observed both with exposure time and concentration, values for 12 hr exposure being highest and those for 132 hr the lowest ($P > 0{\cdot}001$).

4. Discussion

Boyle *et al* (1966) reported that in a fish tissue aldrin starts converting into dialdrin 8 hr after the fish is exposed to it, and the conversion reaches 94% in about 32 days. In our observation also, this conversion must have taken place a little in 12 hr and considerably more for 60 and 132 hr in increasing proportion.

The median tolerance limit of four species of fish has been reported 24 hr at aldrin concentrations ranging from 0·089 to 0·018 ppm and for dialdrin 0·062

to 0·014 ppm (Gakstatter 1968). The present paper describes time dependence of tolerance towards aldrin and dialdrin as shown by liver and serum and other tissues by inference.

The level of protein decreased 25 to 50% in serum and 8 to 10% in liver correspondingly (figures 1 and 2). Low levels in serum protein have also been reported in goldfish (Grant and Mehrle 1970). The protein graphs for serum and liver show quite a different pattern of variation with concentration and time. In serum for a 12 hr exposure, the effect of aldrin is rather small at 0·1 and 0·2 ppm and becomes more marked at 0·5 ppm in a systematic and expected manner. The effect is found to be markedly enhanced at longer exposure thereby showing an important time-dependence of the aldrin affect. At 0·1 ppm, the protein values are the same after 60 and 132 hr. From this it is inferred that at this dose aldrin and its by-product cease to effect protein after 60 hr exposure. The upward bend of graphs for higher concentrations indicates fish tolerance to the chemical which is time dependent as well as concentration dependent. For a longer exposure time and higher concentration, the tolerance starts declining.

In liver the protein graph showed no evidence of tolerance developed at 0·1 and 0·2 ppm doses for 12 hr exposure, therefore steep constant decline. At a higher dose, sufficient amount of antibodies appear to be formed to check a further decline of protein to ar appreciable extent. For 60 hr exposure, the time is long enough to permit the interference by produced antibodies even at 0·1 ppm level. This raises the observed protein values at this concentration as compared to 12 hr. This tendency continues up to 0·2 ppm indicating a maximum tolerance near this point after which the tolerance declines resulting in low protein values. For 132 hr exposure the tolerance cycle appears to have come down to zero again at 0·1 ppm drug level to permit a little more decline of protein value than for 12 hr. At 0·2 and 0·5 ppm, the tolerance appears to have decreased appreciably below its maximum value, but it remains effective enough to place the 0·2 and 0·5 ppm points for 132 hr above those for 12 hr.

The higher level of total phosphorus in the serum of aldrin-treated fish appears to be related to the changes in liver produced under the effect of toxicant. Induction of serum aminotransferases (SGOT and SGPT) lactic dehydrogenase and alkaline phosphatase are reported to be as a result of hepatic changes caused by pesticides (Matsumura 1975).

The continuous increase in total phosphorus for 12 hr exposure means a large concentration is almost proportionately more effective in causing the damage that is releasing phosphorus. Regarding the time factor it is noticed that at 0·1 ppm the released phosphorus increases with period of exposure though not in proportion to the time but at a reduced rate. At 0·2 ppm, an increase in the released quantity of phosphorus is observed with time, but slightly less in quantity from 12 to 60 hr exposure, and very little in going from 60 to 132 hr. At 0·5 ppm the trend is reverse and the released phosphorus decreases from 12 to 60 and then from 60 to 132 hr.

Figures 1 and 2 reveal that for a 12 hr exposure the calcium value declines to about 36% in serum and 29% in liver at 0·1 ppm dose. The level remains constant in liver for higher concentrations up to 0·5 ppm but rises steadily in serum correspondingly. This precludes a direct transfer from liver to blood or *vice versa* for this exposure due to the abrupt liver damage. The constant level

in liver can be interpreted as the stoppage to further liver damage due to the production of antibodies under higher doses called tolerance. The rise of calcium content in serum may be at least partly also due to a similar reason but a sizeable part of the excess calcium should result from various tissues, other than liver because no loss of calcium at the corresponding point is seen (figure 2). For 60 hr exposure the values of liver calcium are related to about maximum tolerance and 132 hr values associated to tolerance that comes down again to almost zero.

In serum the calcium values were observed to be higher than the control value at certain concentrations and exposure times. This peculiarity is hard to explain on tolerance basis and the transfer of calcium appears evident in these conditions from other tissues to serum. The decline for longer period will then indicate a greater excretion.

On comparing figure 1 with figure 2 it is seen that the trends of cholesterol variation in liver and serum are opposite to each other. Almost a regular increase observed in serum at different concentrations and exposures, contrasts to a regular decrease in liver. This is a clear case of transfer of cholesterol from liver to serum and from other organs and confirms the observations of Eisler and Edmunds (1966) on puffers with endrin.

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