PESTICIDAL IMPACT ON FRESHWATER TELEOST, *TILAPIA MOSSAMBICA* RETINA

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ABSTRACT

The fish, *Tilapia mossambica* showed greater susceptibility in heptachlor. Sublethal chronic exposure resulted increased blood sugar levels in heptachlor. Retinal tissue showed decrease in the total carbohydrate content and proteins and increase in the amino acids and lipids.

INTRODUCTION

It is estimated that on a world-wide basis, about 20 million tons of organic chemicals enter the environment annually[1]. Of the large number of pesticide chemicals developed for use in agriculture, horticulture and veterinary and medical fields, a high proportion has been recognized as highly toxic to many non-target organisms, although in normal use, most are unlikely to enter fresh waters in biologically significant amounts[2]. This indiscriminate use of pesticides to boost agricultural productivity etc has affected the ichtyofauna either directly or indirectly. Any change in the natural ecosystem leads to conspicuous architectural changes in the aquatic organisms, especially fish[3,4]. Pesticidal stress induces hyperglycemic condition in non-target organisms[5–8]. The diabetic condition is known to cause certain changes in retina ultimately leading to defective vision in higher animals, but nothing is known about lower vertebrates. An attempt has therefore been made to study the fate of retina in freshwater fish, *Tilapia mossambica* (Peters) under chronic heptachlor intoxication.

MATERIALS AND METHODS

The fish, *T. mossambica* weighing 15 ± 2 g were collected from the local ponds around Tirupati, transferred to the laboratory aquaria and maintained at 27 ± 2° C for two weeks. They were fed daily *ad libitum* on groundnut cake and were then exposed to different pesticides according to their biomass ratio[9] and LC_{50} was determined. Since heptachlor showed greater toxic effect than other pesticides on the life of freshwater teleost[10,11], the fish were exposed for 30 days under sublethal concentration (0.03 mg/l) of heptachlor. The control fish were maintained under identical conditions of tapwater. The pesticide medium was also changed daily by mixing the required amount of stock pesticide solution to remove feaces, food residues and to maintain the correct concentration of the medium. On successive completion of the exposure period, the fish were stunned to death and the retina was collected both from control and from experimental fish. The levels of carbohydrates[12], proteins[13], lipids[14] and aminoacids[15] were estimated in the retina of the control and the experimental fish. The blood was drawn directly from the heart and the glucose level was estimated by the method of Mendal *et al*[16].

Disc electrophoresis using cyanogum-41 (Sigma make) as a medium for separation of gel, was carried out with tris glycine buffer (0.1 M) pH 8.3 both as tank buffer and gel buffer for separation of protein bands. The 4% solution (0.25 ml) was directly added to the separation gel[17] and the gels were removed and stained with 1% amido black solution and destained with methanol : water : acetic acid (5:5:1) until the background of the gel was clear. Following the complete band development, the gels were scanned in Kratos (Schoeffel instrument) SD 3000 spectrodensitometer at a wavelength of 550 nm.

RESULTS AND DISCUSSION

Table 1 indicates that the fish showed hyperglycemic condition under pesticidal stress. The increase in blood glucose level may be due to the stimulation of adrenocortico trophic hormone (ACTH) with the existence of asphyxiated condition[7] noticed in heptachlor-exposed fish. Secondly, the hyperglycemic condition could be due to the stimulation of pancreatic hormonal secretion, glucogen[18] by the pesticide.

The increase in lipid content (figure 1) indicates lipogenesis under heptachlor intoxicated fish retina.

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Table 1 *Changes in blood glucose (mg/100 ml) levels of control and pesticidal-exposed fish. Each value is the mean ± S D. of 6 individual observations, p = t* test

Pesticide	Gluclose levels: Control	30 days exposed	% change
Heptachlor	71.60 ±1.286	97.31 ±1.85 $P < 0.001$	35.90
Phosphamidon	71.60 ±1.286	93.99 ±2.265 $P < 0.001$	31.27
Dichlorovos	71.60 ±1.286	95.30 ±2.398 $P < 0.001$	33.10

The depleted levels in carbohydrate (figure 1) possibly suggest that the reserve animal starch in the tissue is also utilized to supplement the rapid loss of some total carbohydrate. The depleted retinal protein content (figure 1) under heptachlor is attributed to the impairment of protein synthesis. This decrease in protein may also be due to their degradation to amino acids, which in turn is fed into TCA cycle through aminotransferase pathway, probably to cope with the high energy demands during the state of depleted glycogen reserves, induced by heptachlor. The electrophoretic analysis showed nearly 16 protein bands (figure 2) in control fish retina, whereas in experimental fish retina a few of them disappeared and the remaining were with low peaks

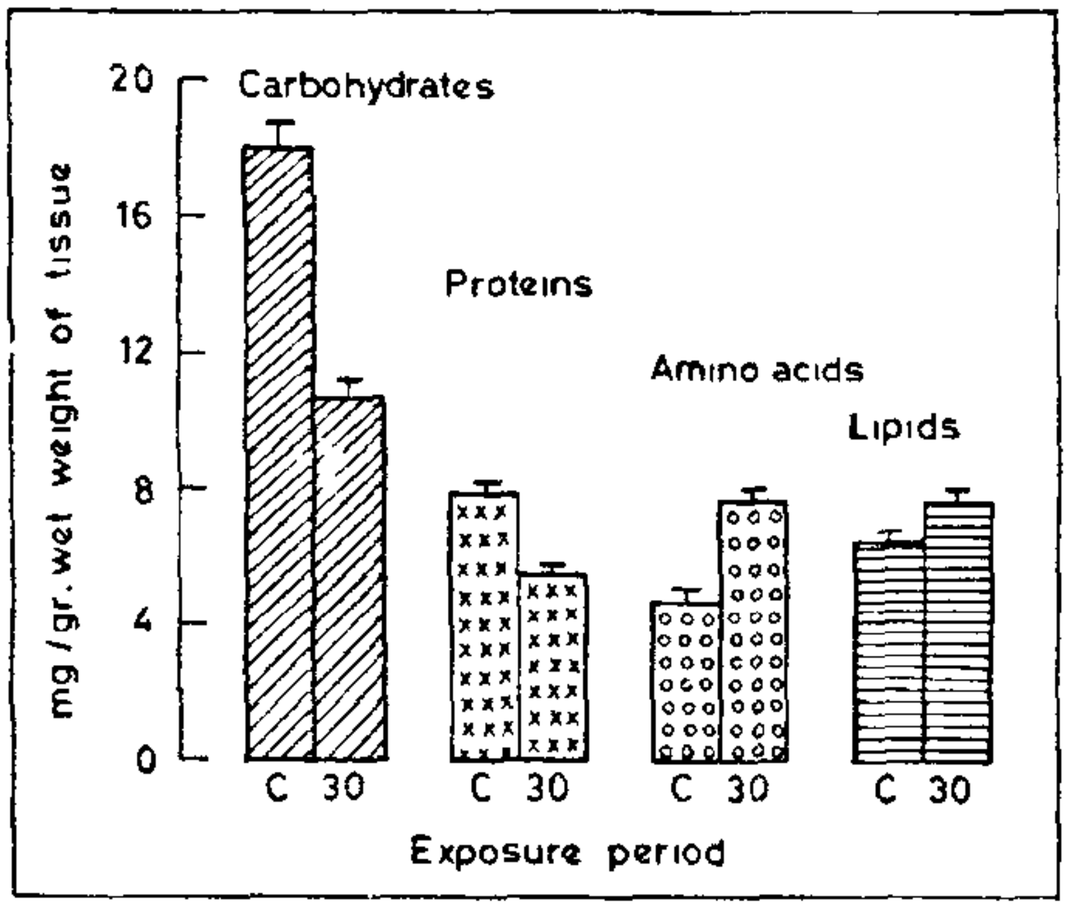

Figure 1. Levels of carbohydrates, proteins, amino acids and lipids in control (C) and 30 days heptachlor-exposed fish retina.

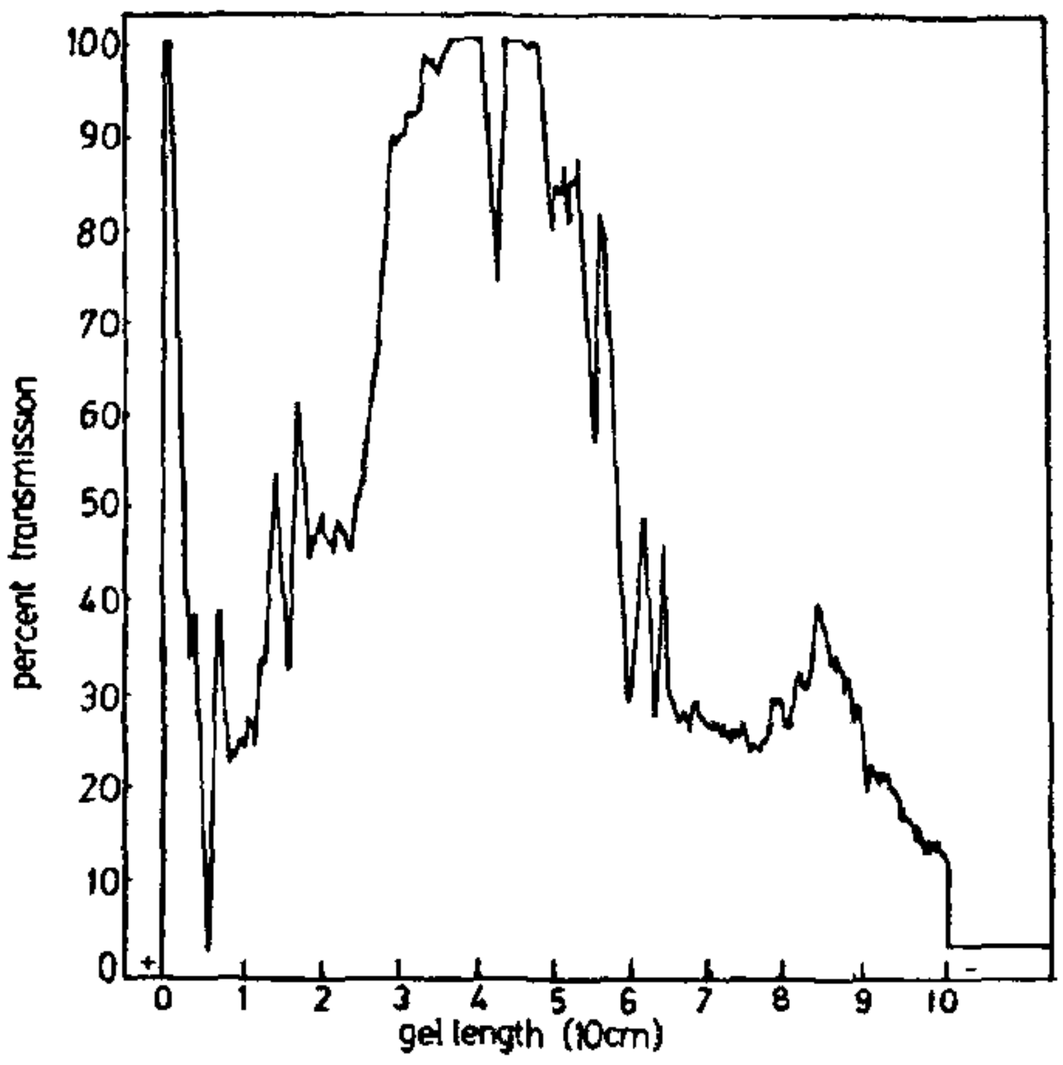

Figure 2. Electrophoretic profiles of control fish retina protein mixture.

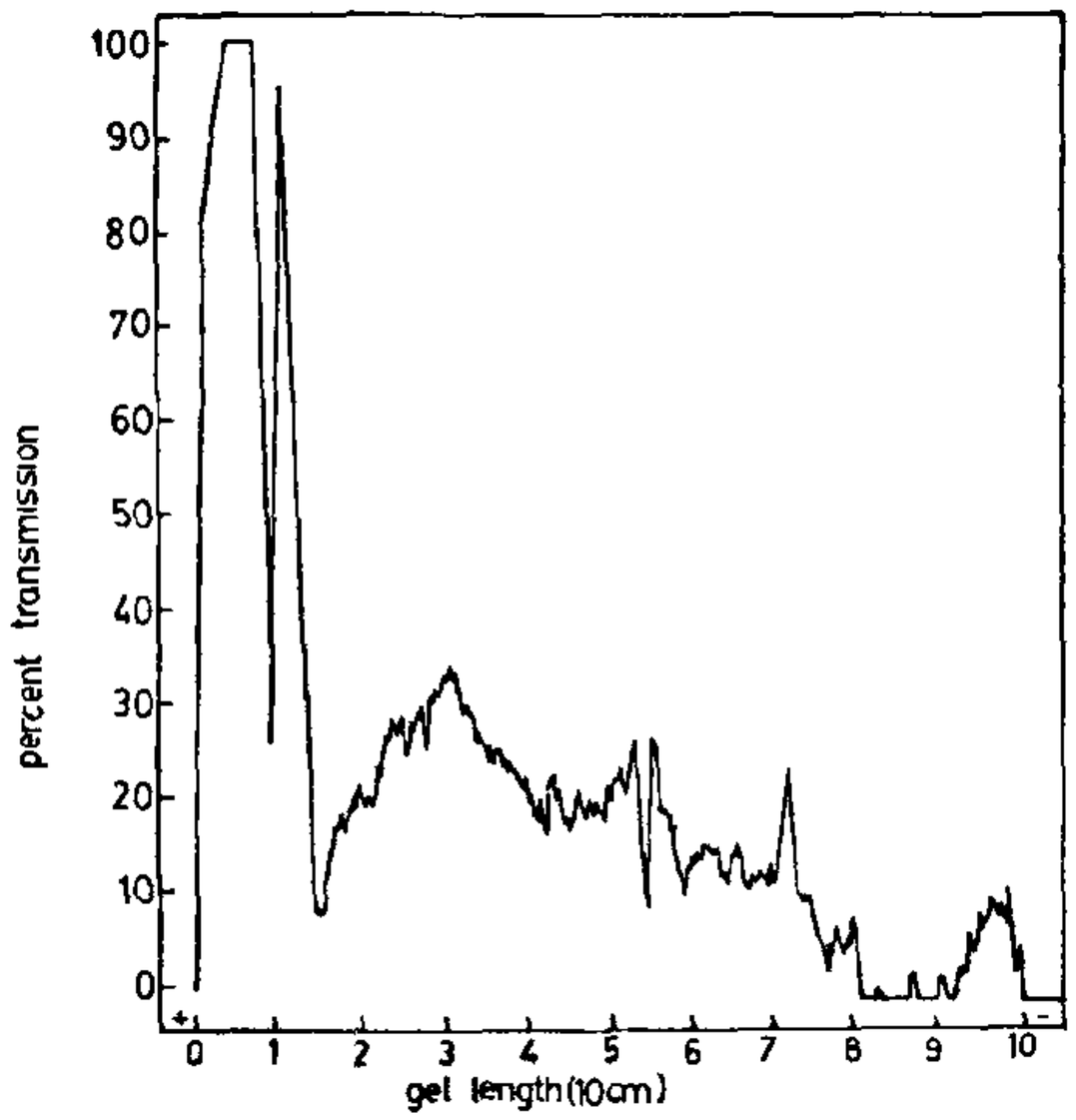

Figure 3. Electrophoretic profiles of heptachlor exposed fish retina protein mixture.

(figure 3) indicating the degradation ot protein content. As a result, the actively metabolizing cells of the retina are unable to synthesize visual pigment from the protein and vitamin A[19] finally leading to retinal diseases. Biochemical and histological studies on these are in progress.

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NEWS

KIDNEYS OF THE NEWBORN SPLITS PROTEINS INTO AMINO ACIDS

A group of Uzbek scientists headed by Academician Kamildzhan Zufarov, member of the Uzbek Academy of Sciences, established that in the newlyborns kidneys perform the functions of a digestive tract, splitting proteins into amino acids.

Before this discovery was made scientists wondered how a newlyborn gets everything it needs from the only product available—the mother's milk. At some stage of the digestive process its proteins have to be split, but the baby's digestive organs are too underdeveloped to perform this operation: So where and how this happens? The answer is provided by the discovery of Uzbek scientists.

Their discovery also explains why artificially-fed babies sometimes suffer from kidney diseases. Their kidneys are unable to process proteins so different from proteins contained in their mothers' milk. The first practical application of the new discovery will be in the field of baby food. (*Soviet Features—Science and Technology*, Vol. XXVI, No. 84, Monday, July 20, '87. Published by the Information Department, USSR Embassy in India, P.B. 241, 25 Barakhamba Road, New Delhi 110 001.)