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ACUTE AND SUBACUTE TOXIC EFFECTS OF MERCURY ON THE FRESHWATER FISH, CLARIAS LAZERA: INFLUENCE ON SOME PHYSIOLOGICAL PARAMETERS.

A.M. HILMY, N.A. EL-DOMIATY, A.Y. DAABEES AND F.I. MOUSSA.

Department of Zoology, Faculty of Science, Alexandria University, Alexandria, Egypt.

ABSTRACT

1- This work evaluated the changes that occurred in the levels of seven noncellular blood constituents under the stress of mercury exposure.
2- The intoxication was characterized by elevations in serum total proteins, blood urea nitrogen, uric acid, serum total lipids and cholesterol during both the acute and subacute studies.
3- While serum glucose level was increased during the acute test, it was reduced during the subacute experiments. On the other hand, serum creatinine levels were not changed.

INTRODUCTION

Concerning water pollution, the industrial waste products have been discharged into water ways for many years ago. Mercury pollution in the aquatic environment has been recognized in many areas of the world, and high concentrations have been found in many species of fish. Various mechanisms for the biological toxicity of mercury have been proposed (e.g. Vallee and Ulmer, 1972; Yonaha et al., 1982; Fukino et al., 1984; Working et al., 1985). In fact, there is no precise outline about the physiological basis of mercury toxicity reported in the literature for fish. So, the main objective of this study was to document the changes in the concentrations of some blood parameters in the freshwater fish **Clarias lazera** as a consequence of acute and subacute exposure to mercury.

MATERIALS AND METHODS

Treatment procedure: Adult specimens of **Clarias lazera** were subdivided in five tanks,each containing 80 liters of dechlorinated tap water and maintained in a 12 hr photoperiodic condition. One tank was used for controls, one for the acute study and three for the subacute one. During the acute test, animals were exposed to 0.72 mg Hg^{+2}/l (the 96 hr TLm value) in a static system, while in the subacute experiments, fish were subjected to 0.10, 0.22 and 0.40 mg Hg^{+2}/l in renewal systems. Experimental controls were run simultaneously under identical conditions, except that no toxic cation was added to the water.

Serum preparation: After 1, 2, 3 and 4 days and 1, 2, 3 and 4 weeks of exposure, for the acute and subacute tests respectively, four animals were removed from each tank and the blood was withdrawn from the caudal vessel after carefully bleeding the sectioned caudal peduncle. The serum was obtained by centrifugation of the blood and stored in tightly capped vials at 20°C until time of analysis.

Clinical chemistry: During the physiological analyses, Barnett and Youden (1970) procedure was used for the determination of total proteins. Blood urea nitrogen and uric acid were estimated according to the methods described previously by Kaplan (1965) and Hoffman (1966) respectively. Serum creatinine was determined by the alkaline picrate method as described by Harold Varley (1963). Seurm total lipids were measured by the method of Frings et al. (1972). For the estimation of serum cholesterol and glucose, the methods of Clerch and Miale (1970 and 1971) were applied, respectively.

All the results obtained were statistically analysed using the formulae of Arkin and Colton (1963).

RESULTS

Mercury treatment appeared to have significant effects on almost all the studied physiological parameters (Figs. 1-10). Its exposure caused increases in serum total proteins, blood urea nitrogen, uric acid, total lipids and cholesterol during both the acute and subacute experiments.

Mercury exposure had no significant effect on the creatinine content allovet the experimental periods. On the other hand, considerable changes were observed in serum glucose content, there was a significant increase than controls during the acute test, while in the subacute tests there was a steady increase at the first week followed by a decrease in glucose levels during the remainder of the experimental period.

DISCUSSION

Serum proteins in vertebrates are a diveres group of proteins, the functions of which are manifold. In the present study, hyperprot einemia was observed during both acute and subacute studies. Similar results were reported by Christensen (1975) in alevine of brook trout, **Salvelinus fontinalis:** after mercury exposure. An elevation in serum proteins has also been observed to exist in the killifish **Aphanius dispar** following exposure to mercury (Hilmy et al., 1980) and in **Anguilla vulgaris** treated with the same pollutant (Yacou*, 1986). This increase may possibly be due to change in the molecular dynamics of the animals, such as those involving induction effects on protein forming enzymes, thus causing a general increase in protein metabolism. Another possible explanation, is the affinity of mercury to thiol groups of proteins which is far in excess of its affinity for other biologically occuring ligands (Perkins, 1961; Cember, 1962; Rothstein and Hayes, 1960 and Katz and Samitz, 1973).

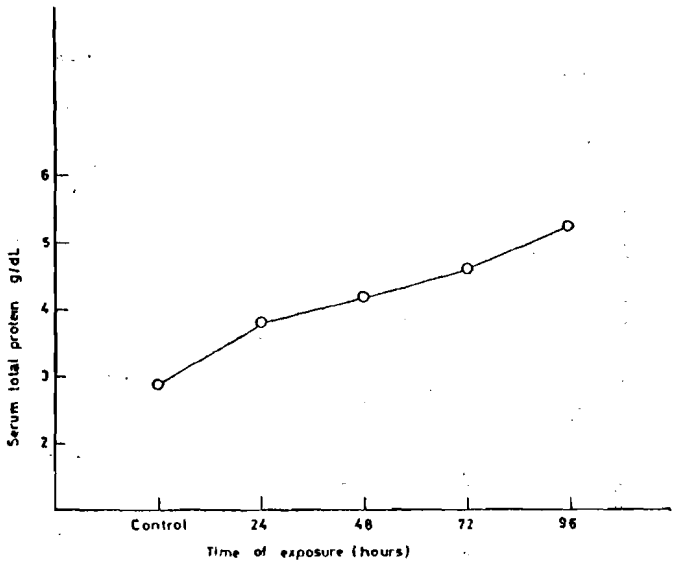

Fig. (1)
Graphic representation of serum total protein (g/dl), of **Clarias lazera** after acute mercury exposure.

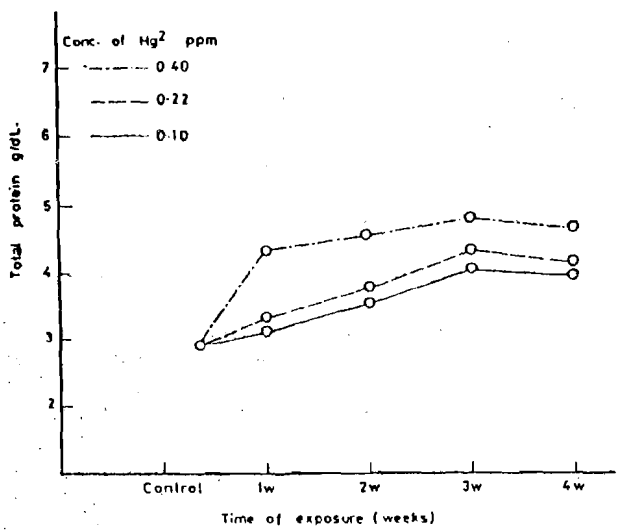

Fig. (2)
Graphic representation of serum total protein (g/dL), of **Clarias lazera** after subacute exposure to graded concentration of mercury.

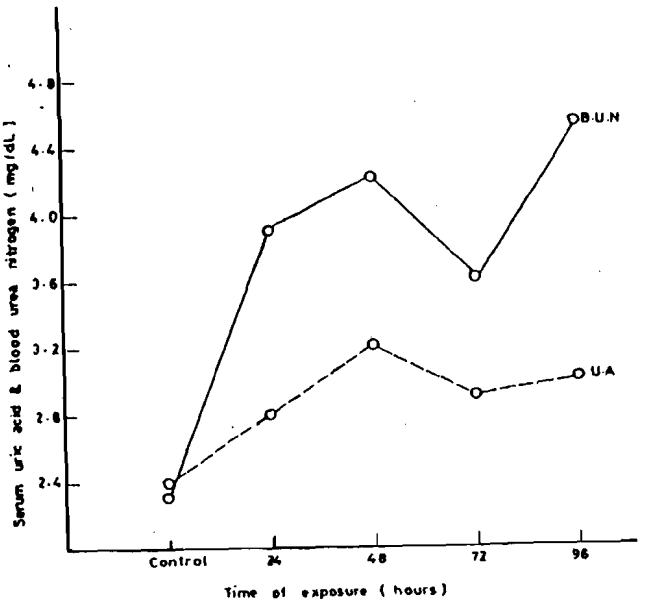

Fig. (3)
Graphic representation of serum uric acid (mg/dL), and serum blood urea nitrogen (mg/dL), of **Clarias lezara** after acute mercury exposure.

Creatinine content did not show any trend to change during this study. Hawk et al. (1965) stated that creatinine is the least variable nitrogenous constituent of the blood of animals and it is normally excreted in constant amounts. Although Paller (1985) noticed an increase in the plasma creatinine of rats dosed with 4 mg Hg/Kg, he found an improvement in its level between the thrid and fourth days after mercury administration, with normal creatinine concentration one week after mercury dosing.

In the investigation designed here, significant elevations in the blood urea nitrogen and uric acid were detected after mercury exposure. This may be due to the fact that the kidney is the target organ of Hg accumulation and this accumlation induces renal failure (Zalme et al., 1976; Yonaha et al., 1982 and Fukino et al., 1984). In this study, total lipids and cholesterol in serum were increased singificantly following acute and subacute exposure to mercury. Yoshikawa et al. (1974) and Mathur and Tandon (1979), reported that the absorption of metals in excess amounts disturbs the metabolosm of lipids. The high levels of these constituents may therefore in part be due to disturbance in their metabolism as a result of mercury poisoning, causing their leakage from the liver. This effect might be related to either the inhibition of the activity of enzymes of lipid metabolism, such as lipoprotein lipase (De Bruin, 1976), or to hormonal effect as it is known that an inverse relationship exists between the serum level of thyroxine and the above mentioned lipid constituent.

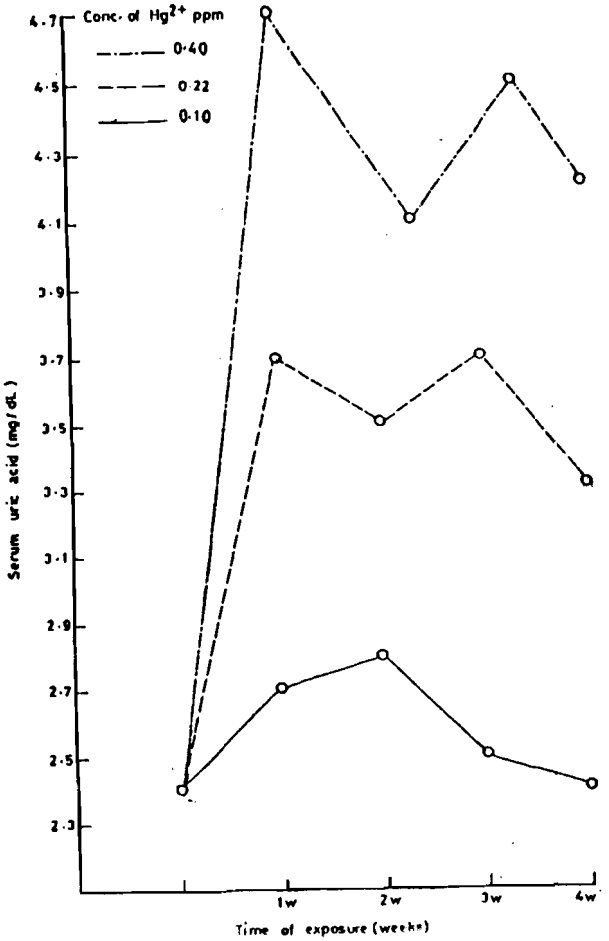

Fig. (4)
Graphic representation of serum uric acid (mg/dL) of **Clarias lazera** after subacute exposure to graded concentrations of mercury.

The most dramatic changes were recorded in glucose level after mercury treatment. It was increased during the acute exposure and decreased at the end of the subacute experiments. This is in agreement with the results obtained by Hilmy et al. (1980) for **Aphanius dispar** after acute and chronic exposure to $HgCl_2$.

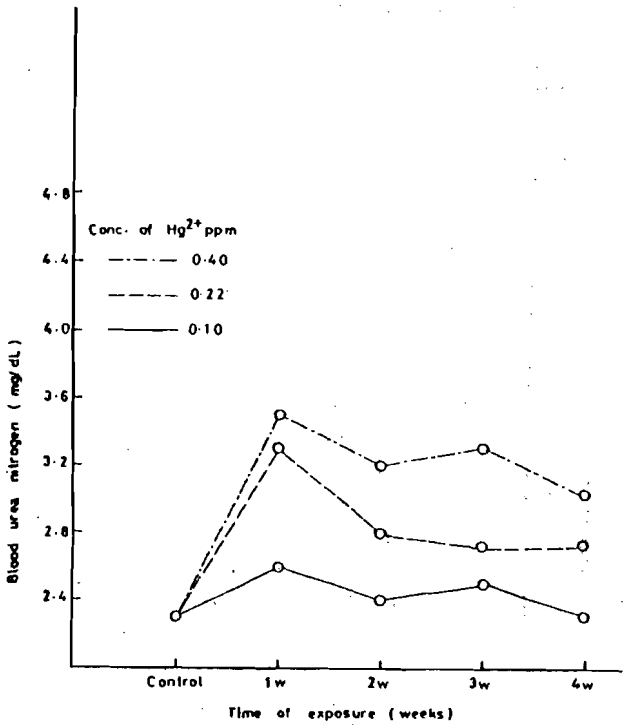

Fig. (5)
Graphic representation of serum blood urea nitrogen (mg/dL) of **Clarias lazera** after subacute exposure to graded concentrations of mercury.

It is well established that exposure of fishes to chemical or physical stress prompts adrenal responses. Increases in catecholamine and glucocorticoids have been reported under conditions of handling, forced activity, thermal shock and contact with chemical pollutants (e. g; Chavin and Kovacevic, 1961; Nakano and Tomlinson, 1967; Chavin and Young, 1970), and this has been correlated with the mobilization of tissue glycogen and the establishment of a state of hyperglycemia. A decrease in liver and muscle glycogen and an increase in serum glucose level were reported by Yacout (1936) for the fish **Anguilla vulgaris** subsequent to acute exposure to mercury. Thus the marked elevation in serum glucose found in this study during the acute test, may be due to impaired carbohydrate metabolism, mediated perhaps by a pituitary adrenal response. On the other hand, the decreased glucose values reported at the end of the subacute tests could be due to increased tissue uptake and clearance rates in conjunction with increased adrenal-hormones secretion.

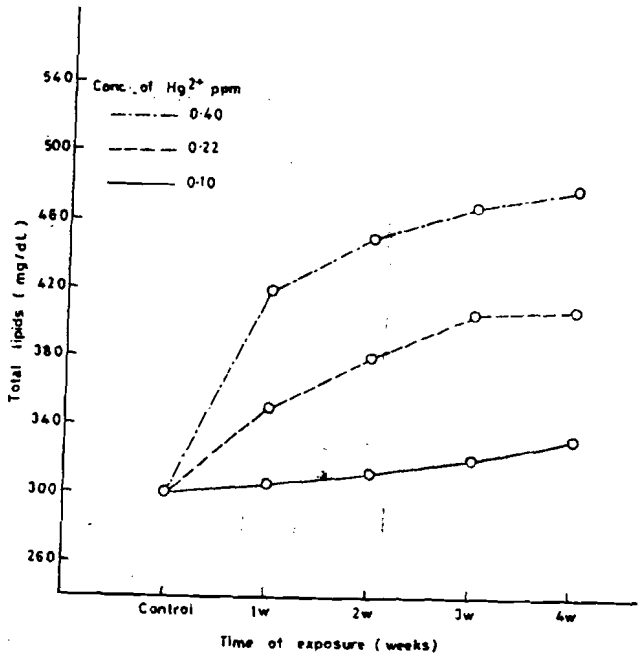

Fig. (7)
Graphic representation of serum total lipids (mg/dL) of **Clarias lazera** after subacute exposure to graded concentrations of mercury.

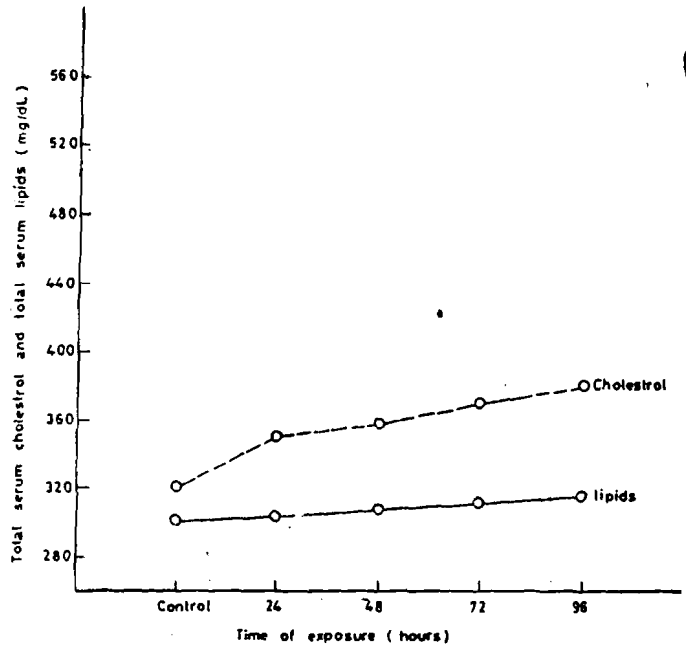

Fig. (6)
Graphic representation of serum total lipids (mg/dL), and serum total cholestrol (mg/dL) of **Clarias lazera** after acute mercury exposure.

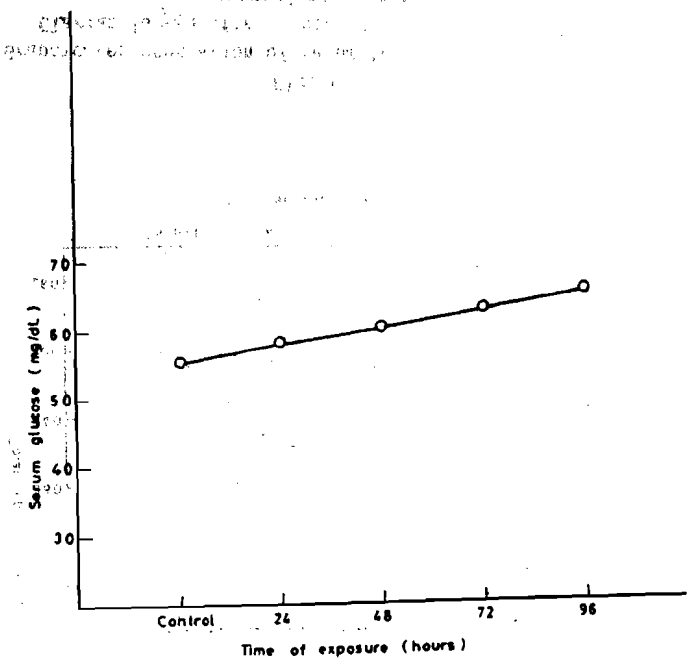

Fig. (9)
Graphic representation of serum glucose (mg/dL) of **Clarias lazera** after acute mercury exposure.

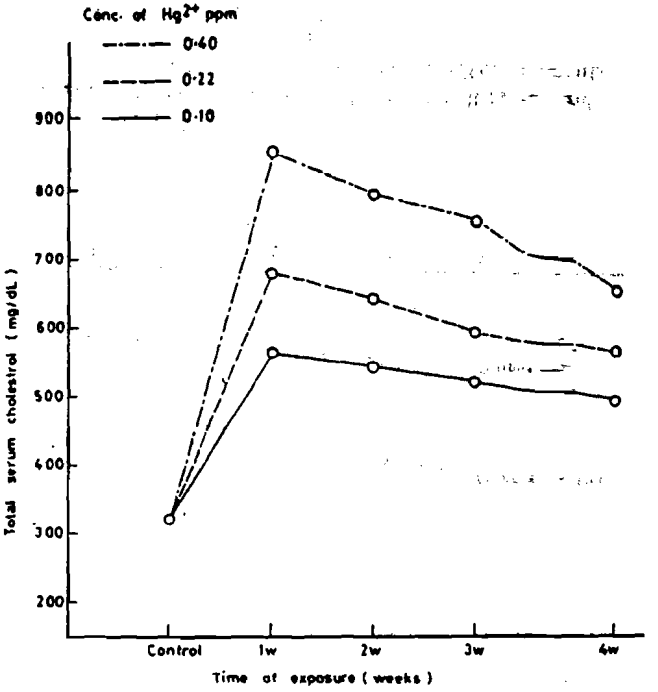

Fig. (8)
Graphic representation of serum total cholestrol [illegible] mg/
of **Clarias lazera** after subacute exposure to gr[illegible]ded
concentration of [illegible].

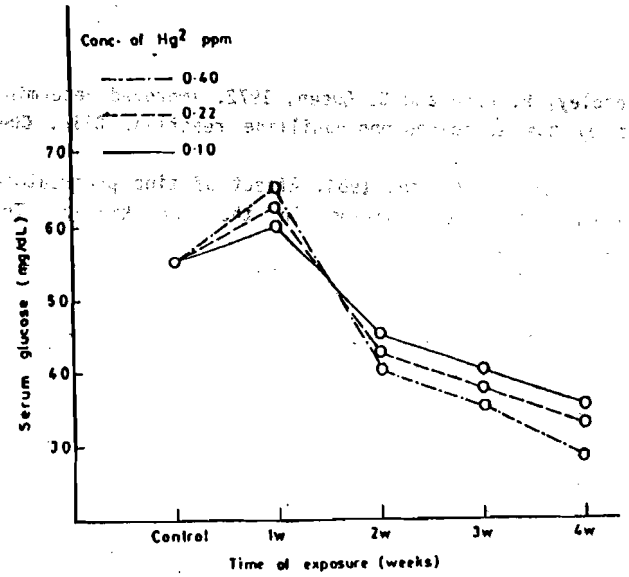

Fig. (10)
Graphic representation of serum glucose (mg/dL) of **Clarias lezara** after subacute exposure to graded concentrations of mercury.

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