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A Study of Arsenic Effect on Blood and Tissue Glucose Concentrations in A Fish Model

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ABSTRACT

Glucose concentrations of blood, liver, kidney and muscle were determined in the live-fish *Anabas testudineu*s (Bloch) exposed to sodium arsenite in water for one month. The values were found to be significantly lower in the arsenic treated fishes, as compared to the controls maintained in arsenic-free water. The changes were moderate in blood and liver, most pronounced in kidney and least in muscle. The overall findings indicate an adverse influence of chronic arsenic toxicity on glucose metabolism.

Keywords: Glucose, Blood, Liver, Kidney, Muscle.

INTRODUCTION

Chronic arsenic toxicity has variously been reported cause different physiological abnormalities like anemia, kerotosis trial and pigmentation of skin, hepatic malfunction, renal tissue damage degenerative changes of gonad and cancers of different organs in human subjects laboratory mammals, farm animals and fish models (Guha and Sarkar 2000, Bates1992). However the relationship between arsenic toxicity and blood or tissue glucose levels remains a controversial issue as yet. Acute arsenic treatment has been reported to adversely affect gluconeogenesis and blood level in laboratory mammals. Another report indicates a marked decrease of glucose concentrations in liver, muscle, gill and brain of the freshwater fish (Sarkar and Sarkar 2001).

In the present study, we have estimated glucose concentration of blood liver, kidney and muscle of the freshwater liver- fish, *Anabas testudineus* (Bloch) following its exposure to sodium arsenite in water for one month (Kumar 2008). The objective of the study is to add to the existing knowledge of arsenic effect on blood and tissue glucose concentrations. We have chosen a fish model for the study because a possibility of exposure of fishes to arsenic in nature cannot be nullified in ponds which are filled up in the summer with arsenic contaminated ground water by using pumps.

MATERIALS AND METHODS

Adult male specimens of *Anabas testudineus* (08-10 cm in length and 60-75 in weight) were maintained in some aquaria for one month with 10 mg of sodium arsenate ($NaAsO_2$) per liter of water (total volume of water was 10 liter per aquarium ($NaAsO_2$) was chosen for the study because a major portion of arsenic detected in groundwater as well as surface water-bodies in nature occurs in a soluble arsenite from (Thornton and Farago 1997). The water of the aquaria were periodically changed and with each change, $NaAsO_2$ was added afresh. The concentration of $NaAsO_2$ was kept

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Table 1. Blood and tissue glucose concentrations (mg/100 ml of blood and mg/g of tissue) in the fish model during the first study (November-December 2019) (values represent means + SD of five fisher in each group). Significantly different from control value at 1% level.

Tissue	Glucose concentration Control group	Arsenic-treated group
Blood	127.4+8.98	94.8 +7.17*
Liver	17.49+1.01	13.51+0.95*
Kidney	61.2 +0.41	3.96 +0.40*
Muscle	16.54+0.87	14.12+0.75*

Table 2. Blood and tissue glucose. Concentrations (mg/100ml of blood and mg/g of tissue) in the fish model during the second study (January – February 2020) (values represent means + SD of five fishes in each group. Significantly different from control value at 1% level.

Tissue	Glucose concentration Control group	Arsenic treated
Blood	123.8+6.33	89.0 +572*
Liver	17.11+0.92	12.98+0.94*
Kidney	06.16+0.42	63.74+0.38*
Muscle	16.24+0.82	13.59+0.81*

same as that used by and earlier group of workers in case of *Tilapia mossambica* (Shobha Rani *et al.* 2000). Control fisher were maintained in absence of $NaAsO_2$ in separate aquaria containing chlorine free tap water. The water of all aquaria used in either the experimental or the control study had a pH of 7.1 to 7.2 a dissolved oxygen concentration of 6.5 to ppm and a free carbon dioxide concentration of 1.4 to 1.5 ppm All fithes were provided with commercial fish Pellet and Tubifex as food adlibitum. Moreover, all fishes were allowed to acclimatize to the laboratory conditions for one week prior to the beginning of the study.

Both experimental and control fishes (five specimens per group) were sacrificed by striking their heads on a laboratory table and subjected to a puncture at the caudal penducle. Blood samples were allowed to drip into clean watch glasses from the caudal vein of the fishes. The samples were prevented from clotting by allowing a thin film of sequestrene (0.1 ml of a 1.0% solution) to dry on each watch glass one hour before collecting the samples. Tissue samples (liver, kidney and muscle) were homogenized in presence of chilled phosphate buffer (pH 7.0) (2.0 ml of the buffer was added to 1.0 g of each tissue). Glucose concentration of blood and tissue samples were then determined colorimetrically following a modified version (Nath and Nath 1990) of the well known arsenomolybdate method (Somegyi 1952). In the adopted method, 5.0% zinc sulfate and 0.5 N sodium hydroxide solutions have been used for deprotenization of blood and tissue samples while 5.0% zinc sulfate and 0.3 N barium hydroxide solutions were used in the original method.

All studies were carried out twice for confirmation of results, initially in October-November 2019 and then in November- December, 2019. The temperature of water at the mid-day varied between 26.5 to 28.5ºC during the first study and between 24.5 to 27.2ºC during the second study. The difference between the result obtained from the experimental and the control the result obtained from the groups of fishes were analyzed statistically by means of Fisher's two-tailed–test (Goon *et al.* 1981).

RESULTS AND DISCUSSION

In either the first study (November-December 2019) or the second study (January-February 2020) glucose concentrations of blood liver kidney as well as muscle were found to be significantly decreased in the experimental group of fishes exposed to sodium arsenate in water for one month as compared to the corresponding values observed in the control group of fishes. The most remarkable reduction of glucose concentration was noted in case of kidney (35.03–39.3% reduction) while the least reduction was noted in case of muscle (14.6–16.3% reduction). Moderate reductions of glucose concentrations were noted in blood and liver (25.6-06-27.9% and 22.7–24.1% reduction, respectively) of the treated fishes, as compared to the controls. Glucose concentrations recorded in blood and different tissues of the experimental as well as the control groups of fishes in two of our successive studies are presented in Tables 1 and 2 respectively.

Our findings on tissue glucose concentrations in arsenic-treated and control groups of the fish model,

Anabas testudineus are in conformity with that reported in another fish model (*Tilapia mossambica*) by an earlier group of workers (Shobha Rani *et al.* 2000) who, however, did not determine blood glucose concentration. In so far as blood glucose concentration is concerned our finding in the fish model is comparable to that in laboratory mammals by some other earlier workers (Reichl *et al.* 1988, Szinicz and Forth 1988).

Considering our finding in conjunction with that of all the aforesaid workers. We conclude that chronic arsenic toxicity may considerably lower blood glucose concentration, probably by adversely affecting the rate of gluconeogeneses (Reichl *et al.* 1988, Szinicz and Forth 1988) in liver and kidney, which are the main sites of gluconeogenesis in the vertebrate body (Martin *et al.* 1981, Kotsanis and Iliopoulos- Georgudaki 1999). Muscle is not an active site of gluconeogenesis in the vertebrate body (Martin *et al.* 1981). Therefore a decreased blood glucose concentration might have been responsible for a decreased supply of glucose to the tissue resulting in a fall of muscle glucose concentration. It may be added here that the arsenic treated fishes are likely to be weaker than the control fishes owing to decreased blood and tissue glucose concentrations.

Finally, it seems necessary to account for the apparent discrepancy between our finding on blood glucose concentration in and arsenic treated fish model and that of others in arsenic affected human subjects. On account of their gill breathing habit fishers are continually exposed to arsenic in water. Moreover arsenic concentration of water was 10 mg/liter in our study in *Anabas testudineus* well as another study in *Tilapia mossambica* by an earlier group of workers (Shobha Rani *et al.* 2000). On the other hand human subject studied by some earlier workers (Guha Mazumder *et al.* 1997, Buchancova *et al.* 1998) were drinking water with an arsenic concentration ranging between 0.5 to 3.2 mg/liter. Therefore it is likely that a relatively larger quantity of arsenic entered everyday into the body of a fish than into the body of a human subject. This is why the blood glucose concentration remained unaffected in the aforesaid human subjects. However, it is difficult to understand the relationship between ingestion of arsenic with artesian well water and a high incidence of diabetes mellitus (hyperglycemia), as reported in case of some people in a locality of Taiwan by another group of workers (Lai *et al.* 1994).

In view of the facts that (i) A decrease of blood glucose concentration has repeatedly been reported in arsenic treated mammalian models (Reichl *et al.* 1988, Szinicz and Forth 1988) and (ii) Hyperglycemia has not been found in any of the many studied human subjects drinking arsenic and contaminated water in our country (Guha Mazumder *et al.* 1997). We express that whether arsenic or any other substance present in ground water was responsible for diabetes mellitus in some human subject of a particular locality requires to be re-examined by means of as many sensitive methods as possible.

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