Effect of Sodium Arsenite on Carbohydrate Metabolism in Male Albino Rats

Kshama Dwivedi
Department of Zoology, Bareilly College, Bareilly. (UP), INDIA

ABSTRACT

The present experiment has been designed to find out the effect of arsenic on carbohydrate metabolism of albino rats. The effect was observed on serum glucose and liver glycogen after 30 and 60 days’ oral exposure to sodium arsenite (NaAsO₂). The size of liver was also compared with that of control. The animals were exposed for two different doses i.e. High and Low.

Study shows increase in serum glucose and depletion of liver glycogen. The high doses of arsenic showed more adverse effect as compared to low dose. The increase in serum glucose under low dose for 30 and 60 days was 11.74% and 12.15%. The increase in serum glucose under high dose for 30 and 60 days was 15.15% and 26.67%. The decrease in liver glycogen under low dose for 30 and 60 days were found to be 21.90% and 29.94% respectively. The decrease in liver glycogen under highest dose of arsenic for 30, and 60 days was found to be 31.44%, and 37.84% respectively. Exposure to 60 days’ duration, showed maximum increase of serum glucose while fall in glycogen percentage. Hepatomegaly was also observed showing that the rats were under stress due to arsenic toxicity. The arsenic toxicity was proportional to dose and time.

Keywords--- Carbohydrate metabolism, arsenic, albino rats.

I. INTRODUCTION

Arsenic (As) is a common element ubiquitously present in rocks, soil and vegetation. There are several chances in which people are exposed to As in daily life. Occupational As exposures are observed frequently in smelter workers from inhalation of As fumes. As is found as sulphide compounds in various ores as contaminant and has become an essential component in the production of semiconductor chips. Inorganic As has been used pharmacologically for treatment of malaria, syphilis, leukaemia or psoriasis under the name of Fowler’s solution etc. Skin lesion, including dermal malignancies, was observed in the patients who prescribed arsenical medicines. The prevalence of malignancy was correlated with arsenic intake (Cuzick et al., 1992).

Chronic As poisoning in the general population has been widely reported in many areas of the world today. In these situations arsenic exposure occurs by consumption of drinking water that naturally contains high amount of inorganic forms of arsenic (Smith et al., 2000b). Ground water with elevated concentrations of arsenic has been recognised as a problem of global concern. Out of them the most effected countries are India, Bangladesh, China and Taiwan (Brinkelet et al., 2009). Arsenic contamination in ground water in the Ganga-Brahmaputra fluvial plains in India and Padma-Meghna fluvial plains in Bangladesh and its consequences to the human health have been regarded as one of the world’s biggest natural ground water calamities to the mankind (Singh and Rana, 2007).

II. MATERIALS AND METHODS

Experimental animals

Albino rats of 6 to 10 weeks old weighing approx. 150-160 grams were purchased from the Laboratory Animal Resource Section, Indian Veterinary Research Institute (IVRI) Izatnagar Bareilly, U. P. and maintained in experimental animal shed of the division. Animals were kept for a week to be conditioned to the
new environment prior to the start of experiments. Animals were kept under conventional condition (6 rats per steel cage, 12 hr. light to dark cycle). The animals were made available to standard rat food and tap water ad libitum. All the chemicals used were from Sigma Chemicals Co., Merk and Qualigens.

**Experimental design**

The experimental albino rats were divided into three groups A, B and C each comprising of 6 animals. Group A (control) received plain tap water while group B with low dose of sodium arsenite (4.3 mg/kg.b.wt.) and group C with high dose of sodium arsenite(8.6 mg/ kg.b.wt.). Albino rats were exposed to two test doses of sodium arsenite (NaOAs₂) for exposure periods of 30 and 60 days. The compound was given in tap water per os by gavage. Mortality rate, food consumption, clinical signs and symptoms were recorded daily. Bodyweight gain was calculated weekly.

After termination of the experimental period, animals were sacrificed under chloroform anaesthesia, dissected and the organs along with liver were removed and washed in ice-cold saline solution. Serum was separated from anticoagulant free blood. The samples were refrigerated at 4°C until biochemical estimations were carried out. Serum glucose estimation was done by O-Toludine method. Single step method described by Cooper and McDaniel (1970). Liver tissue was weighted and homogenised in potassium phosphate buffer, pH 7.4 in mortar and pastel. The glycogen levels were determined by Anthrone Method of Vander (1954).

### III. OBSERVATION

The results of the effect of sodium arsenite on serum glucose and liver glycogen reserve in albino rats are given in table 1 and 3 while the percentage change in table 2 and 4.

**Table 1:** Effect of sodium arsenite (NaAsO₂) on the Serum Glucose (mg/dl) of albino rats

<table>
<thead>
<tr>
<th>Groups</th>
<th>Duration of exposure</th>
<th>30 days</th>
<th>60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A (Control)</td>
<td>117.15±15.54</td>
<td>120.50±0.86</td>
<td></td>
</tr>
<tr>
<td>Group B (L)</td>
<td>130.91±16.82</td>
<td>135.15±4.40</td>
<td></td>
</tr>
<tr>
<td>Group C (H)</td>
<td>134.89±18.21</td>
<td>152.50±4.40</td>
<td></td>
</tr>
</tbody>
</table>

*As (L) = Sodium Arsenite Low Dose
*As (H) = Sodium Arsenite High Dose*

**Table 2:** Percentage change in the Serum Glucose (mg/dl) of albino rats exposed to sodium arsenite (NaAsO₂)

<table>
<thead>
<tr>
<th>Groups</th>
<th>Duration of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group B (L)</td>
<td>11.74</td>
</tr>
<tr>
<td>Group C (H)</td>
<td>15.15</td>
</tr>
</tbody>
</table>

*As (L) = Sodium Arsenite Low Dose
*As (H) = Sodium Arsenite High Dose*

The mean value of serum glucose after 30 days’ treatment was 117.15±15.54 mg/dl, 130.91±16.82 mg/dl and 134.89±18.21 mg/dl for group A, B and C respectively. Increase of serum glucose is more in high dose. After 60 days, post treatment mean value of serum glucose was 120.50±0.86 mg/dl, 135.15±4.40 mg/dl and 152.5±4.40 mg/dl for group A, B and C respectively which shows increase of 26.67% of serum glucose in high arsenic dose. Increase is more significant in rats exposed for 60 days in comparison to that of 30days.

**Table 3:** Effect of sodium arsenite (NaAsO₂) on the liver glycogen reserve of albino rats (mg/g of wet tissue)

<table>
<thead>
<tr>
<th>Groups</th>
<th>Duration of exposure</th>
<th>30 days</th>
<th>60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A (Control)</td>
<td>0.954±0.042</td>
<td>0.949±0.02</td>
<td></td>
</tr>
<tr>
<td>Group B (L)</td>
<td>0.745±0.022</td>
<td>0.665±0.03</td>
<td></td>
</tr>
<tr>
<td>Group C (H)</td>
<td>0.654±0.032</td>
<td>0.590±0.03</td>
<td></td>
</tr>
</tbody>
</table>

*As (L) = Sodium Arsenite Low Dose
*As (H) = Sodium Arsenite High Dose*

**Table 4:** Percentage change in the liver glycogen reserve of albino rats exposed to sodium arsenite (NaAsO₂)

<table>
<thead>
<tr>
<th>Groups</th>
<th>Duration of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 days</td>
</tr>
</tbody>
</table>

68
In animals treated for the duration of 30 days, Group C with high dose of sodium arsenite shows more depletion in liver glycogen in comparison to group B when compared to control (Group A). Group B and C shows percentage change of 29.94% and 37.8% respectively.

The trends in 60 days’ exposure period is not much different from 30 days’ exposure period. Group B and C showed significant depletion of liver glycogen reserve. The fall of 21.45% and 31.43% was observed in group B and C respectively when compared to control. Percentage fall of sodium arsenite in 60 days treated rats is high compared to 30 days treated rats.

### IV. DISCUSSION

Data presented in this paper indicate that inorganic arsenic alters the carbohydrate metabolism. This was reflected with increase of serum glucose level (hyperglycaemic condition) and depletion of liver glycogen with increase of liver size (hepatomegaly) in arsenic treated rats for duration of 30 and 60 days.

The acute and subacute administration of trivalent arsenic to rats results in marked disturbances of carbohydrate metabolism (Ghafghaziet al., 1980) which involves both direct target tissue of carbohydrate metabolism and homeostatic organ system which regulate overall in vivo glucose metabolism. Santruet al. (2000) reported that the incidence of hepatomegaly was greatly increased in hospitalised arsenicosis patients of West Bengal India and a positive correlation between level of arsenic in drinking water and hepatic arsenic content. Recently various experimental and epidemiological studies have shown an association between chronic arsenic exposure and development of diabetes mellitus (Navas-Acien et al., 2006). Khan et al. (2007) and Virmaniet al. (2007) while working on green mussels and fish liver also observed fall in glycogen of these tissues.

Disturbances in carbohydrate metabolism, due to arsenic toxicity causing hyperglycaemia are probably mediated by effects on the adrenal glands. According to Ghafghaziet al., 1980, it appears to involve altered adrenal gland release of catecholamines, decreased tissue utilization or disturbed pancreatic function might be the reason of hyperglycemic condition. Elevated concentration of glycyslated haemoglobin, an indicator of high blood glucose level, was reported in several Danish workers exposed to arsenic (Jensen and Hansen, 1998). Lim et al. (1998a) and Mitchell et al. (2000) reported that sodium arsenate increases serum glucose level when given to rats. Arsenic induces diabetes possibly by inhibition of insulin dependent glucose uptake in adipocytes by trivalent arsenicals (Walton et al., 2004).

Carbohydrate depletion (glucose and glycogen) is reported to be a major problem in acute arsenic poisoning (Berry and Smythe, 1959; Reichlet et al., 1998; Szinicz and Forth 1988). Trivalent arsenicals cause complete carbohydrate depletion in animals which died due to arsenic exposure during the experiment (Berry and Smythe, 1959; Szinicz and Forth, 1988). Fall in the glycogen content of liver was reported by Lowatinde and Niimi (1984) in rainbow trout. Reichlet et al. (1990) reported that after acute poisoning with arsenic trioxide all the animals intoxicated showed a significant decrease in the liver glucose and glycogen content as compared to control animals. The present findings fall on similar lines arsenic and cadmium intoxication caused decline in liver glycogen. The decline was dose and time dependent.

Metal exposure stimulates hormones that accelerate glycogen breakdown or inhibition of those chemicals involved in glycogen synthesis. Metal toxicity stresses induced by hyperactivity with simultaneous secretion of adrenalin, resulted in increased rate of glycolysis together with glycogenolysis in liver and muscles vis-a-vis impaired metabolism and poor body growth.

According to Pimpar and Bhave (2010) exposure to high levels of arsenic causes diabetes and increased levels of glucose in blood. Decrease in glycogen level in fish due to pesticide effect has been reported (Vijayavelet et al., 2006; Crestaniet al., 2005). Chandra Mouli (2008) also reported a fall in glycogen levels in fish Heteropneustes fossilis exposed to cypermethrin. Arsenic accumulation was more pronounced in liver than pancreatic tissue (Patel and Kalia, 2010).

Thus, more catabolic rate of glycogen could be another possible reason for decrease of glycogen and increase of blood glucose in the present investigation. Hepatomegaly may be due to arsenic accumulation and inability of liver to handle bile production. The effect becomes more significant when the dose and exposure duration is increased. Thus, effect of arsenic in albino rats was found to be dependent on the period of exposure and concentration of metal.

### ACKNOWLEDGEMENT

The author is thankful to Dr. Prabhaker Dwivedi, Principal Scientist, IVRI, Bareilly for providing the facilities to carry out this project and also for their help and cooperation.

### REFERENCES


