The Clarion Volume 1 Number1 (2012) PP 84-88

Toxic impact of sodium arsenite in *Channa punctatus*

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Abstract

Arsenic is a potent environmental toxin. Arsenic is ubiquitous in the environment and chronic or acute exposure through food and water as well as occupational sources can contribute to a well-defined spectrum of disease. Present study has been designed to evaluate the toxic effects of sodium arsenite (Arsenic) on *Channa punctatus*. Fishes were treated with different concentrations of sodium arsenite within the range 0.5mM-2mM.On exposure to 2mM of NaAsO2, the survival time of the fishes were 2 and half hours. Survival time was inversely proportional to the concentration of NaAsO2.Survival time was found to be above 3hr, 4hr and 16hr for 1.5mM, 1mM and 0.5mM of NaAsO2.Behavioural and morphological manifestations were recorded at a definite interval after being exposed to NaAsO2.Micronuclei test assay and chromosomal studies confirmed significant alterations.

Keywords: Arsenic, *Channa punctatus*, micronuclei test assay, morohological manifestations.

1. Introduction

Arsenic is a metalloid and its toxicity is due to the interaction of arsenic ions with protein thiols. The primary exposure pathway of arsenic is ingestion (water and food).Inhalation is regarded as a minor pathway and dermal absorption is negligible. Arsenic is rapidly absorbed from the gastrointestinal tract and lungs and is widely distributed in most tissues of the organisms. Large amounts are deposited in the liver, kidney, lungs and skin. Arsenate (As5+) is rapidly reduced to arsenite by the enzyme arsenate reductase. Arsenite is then methylated by the enzyme methyl transferase or arsenite methyl using S-adenosyl methionine as a methyl group donor to form Monomethylarsonate(MMA5+) and dimethylarsinic acid(DMA5+),with the formation of intermediate metabolites(MMA3+ and DMA3+).These trivalent forms of arsenic are now found to be more toxic than the inorganic species(Klassen,2008)

Arsenic reacts with thiol groups (-SH) especially the enzymes or cofactors which possess two thiols, resulted in the alteration of various enzymes related to tissue respiration. Arsenic causes mitochondrial damage (Liu, 2005), alters DNA repair mechanism, alters DNA methylation, oxidative stress, cell proliferation, co-carcinogenesis and tumour promotion (Hughes, 2002).The genotoxic effect of arsenic has not yet been fully understood. However it is suggested that arsenic inhibit DNA replication or repair mechanisms and it acts as a phosphate

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analog(Li,1989).Chemical fingerprints of water samples collected at different locations confirmed that water with highest arsenic content originated from human built ponds while water coming from irrigated rice fields had the lowest arsenic content (Potera,2010). Total arsenic accumulation was greatest in organisms exposed to inorganic arsenic, particularly at 100micrograms per litre(Spehar,1980).

2.Materials and Methods

2.1 Collection of fishes

Live fishes collected from different markets of Guwahati, were brought to the laboratory of the Department of Zoology, Cotton College.

2.2.Accimatization of the fishes

Fishes were allowed to acclimatize in normal temperature and normal water for a week. Scoop net was used for handling the fishes. Proper aeration was done. Water was changed once after every three days. Fishes weighing 50-100g and having the length between 15-21cm were selected. Fishes were kept in two aquariums in 25 litre of water. Each aquarium contains 16 numbers of fishes (Ahmed, 2008)

2.3. Sodium arsenite treatment

Four glass jars were taken for sodium arsenite treatment. Each glass jar contains 8 litres of water. Sodium arsenite were added with the concentration of 0.5mM, 1mM, 1.5mM and 2mM in the respective jars to determine the LC50 value. Acclimatized fishes from the aquarium were selected and transferred to the glass jars(4 fishes in each jar)by the scoop net. The survival time of the fishes were recorded in each jar. Behavioural and morphological changes were also observed.

2.4. Micronuclei assay

Micronuclei assay was carried out to find genotoxicity.The first work with micronuclei assay was done by Manna *et at.*,(Manna,1984),who reported micronucleus in Oreochromis mossambicus.The utility of micronuclei test in *Channa punctatus* for detecting genotoxicity was again reported by Barat *et at.*,(Barat,1997).Micronuclei are cytoplasmic chromatin masses formed by fragmentation of chromosomes induced by mutagenic agents. Micronuclei test was carried out in treated as well as control fishes. Blood was collected from caudal vein and smeared on clean slides and stained with 6-10%Giemsa as well as May Grunnwald's solution (Basdeo, 2000).

2.5. Chromosome preparation

Chromosome studies are carried out by preparing chromosomes from the tissues of gill, kidney, liver and intestine. Metaphase chromosomes are studied by subjecting the cells to hypotonic treatment followed by fixation and staining. Chromosomes are prepared directly from the cells with a large proportion of dividing stages such as cells of kidney,gills,liver and intestine in fishes which can be arrested at metaphase stage by injecting colchicines,a spindle inhibitor.

3. Results and discussion

Channa punctatus were exposed to different concentrations of sodium arsenite ($NaAsO_2$) and the survival times were recorded. The survival period was found to be inversely proportional to the concentration of $NaAsO_2$. 2mM of $NaAsO_2$ was found to be lethal for the exposed fishes. Lethality was found to occur within 2 and half hours of exposure which is recorded in table1.Survival period was above 3hour in 1.5mM, above 4hour in 1mM and above 16hour in 0.5mM.

Table 1: Survival period of fish after exposure to NaAsO2 dissolved in distilled water.

$NaAsO_2$ Concentration	Sample No.	Length & weight of fish	Survival period
0.5mM	1	L:19cm W:57gm	19hr
	2	L:21cm W:64.5gm	17hr
1mM	1	L:20cm W:58gm	4hr and 15min
	2	L:17cm W:40.2gm	5hr
1.5mM	1	L:21cm W:64.8gm	3hr and 45 min
	2	L:20cm W:64gm	3hr and 15 min
2mM	1	L:20cm W:58.2gm	2 and half hr
	2	L:19cm W:57.9gm	2 hr and 40min

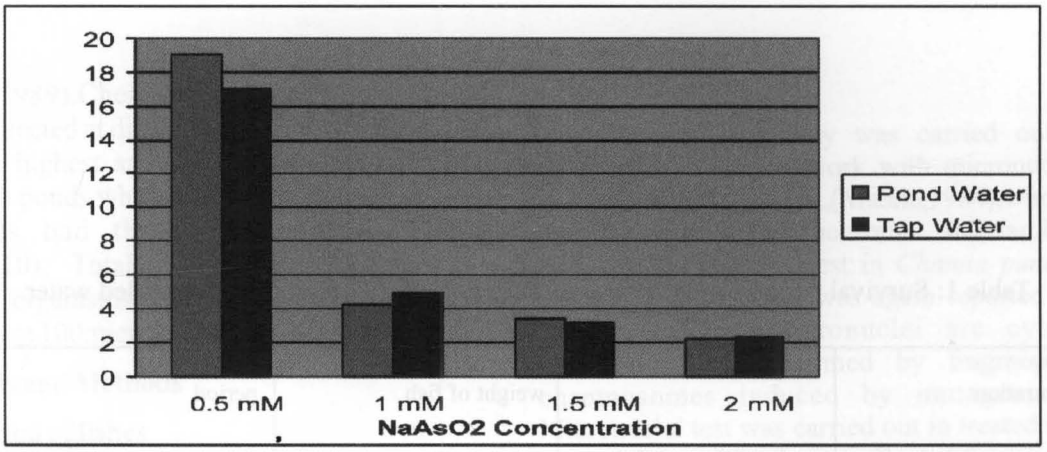

Fig.1 showing the survivality period in different concentration of sodium arsenite.

After the fishes were exposed to NaAsO2, morphological and behavioural manifestations were observed and recorded in a definite interval. The fishes were seen to produce slime due to arsenic toxicity. Fishes were found to show erratic behaviour. Ulcers and tail rot were seen in the fishes. Fishes were also found to suffer from respiratory problem, observed the higher number of operculum movement.

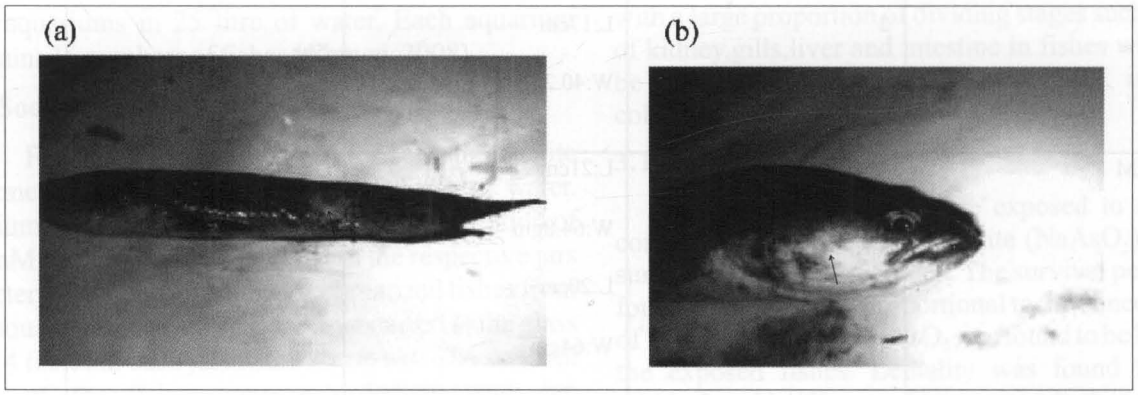

Fig.2 Showing (a) Skin ulceration and tail rot (b) rupture of blood vessels in jaws

Micronuclei test was carried to detect genotoxicity.In control groups, the frequency of micronuclei was low, while in treated groups, the frequency of micronuclei was found to be higher. Micronuclei were found to increase with an increase in arsenic concentration. Increase in micronuclei is a marker for arsenic toxicity. Remarkable chromosomal abnormality in all the tissue studied were also observed.

4. Conclusion

Arsenic in many forms are toxic to all animals, including human being. There are natural sources of arsenic in many areas of Assam. Therefore present investigation was carried to determine arsenic toxicity in Channa punctatus and established that arsenic toxicity causes morphological, behavioural changes as well as changes in chromosome and micronuclei.

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