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EFFECT OF ARSENIC ON AQUATIC ANIMALS: A REVIEW

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Abstract: The origin and distribution of arsenic is mainly geogenic but anthropogenic activities can also lead to arsenic contamination across various environments. Although arsenic contamination in natural systems is often too low to cause mortality but it is sufficient to interfere with normal functioning of the body. Continuous exposure of freshwater organisms including fish to low concentrations of arsenic may result in bioaccumulation in which liver and kidney having high accumulation in most of the cases, altering growth, haematological and biochemical parameters as well as many physiological and biochemical activities in aquatic animals. The toxicological aspects of arsenic have mainly been discussed in connection with their environmental persistence and the ability of arsenic to induce a variety of adverse effects in aquatic animals, particularly in fish. The arsenic even at very low concentration is haemato-immunotoxic to fish and the changes observed haematological, immunological and biochemical parameters may provide a useful early biomarker of low-level xenobiotic exposure. The high solubility and mobility of arsenic in aquatic environments affects its global cycling.

Keywords: Arsenic, Aquatic ecosystem, Environment, Fish, Pollution, Tissue, Toxicity.

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INTRODUCTION

In most of the natural aquatic ecosystems, metal concentrations are increased over natural levels due to a continuous release of metals from industrial and agricultural sources into the water bodies (Kumar and Singh, 2010). Aquatic environments are flooded with contaminants released by human beings (Arya, 2019; Prakash and Verma, 2022). These contaminants disturb the balance of aquatic ecosystem (Singh *et al.*, 2023; Rani *et al.*, 2024).

Arsenic (As) is a ubiquitous element present naturally in the earth's crust and one among the 20 most common elements. It is a metalloid element abundant in the aquatic environment as a result of both natural and anthropogenic processes. It is added to the water either by natural sources including weathering of rocks, sediments, volcanic eruptions and aquifers, or by anthropogenic sources including herbicides, wood preservatives, metal smelting, drugs,

pesticides, burning of coal, agriculture runoff and petroleum refining processes among others. The untreated and uncontrolled discharge of arsenic by industries into the natural water bodies poses serious threat to aquatic fauna by deteriorating water quality and making it unsuitable for fishes (Bissen and Frimmel, 2003). Arsenic can enter into fish body through buccal cavity with contaminated food and water and also absorbed through skin and gills (Amsath *et al.*, 2017). Such fishes have different mechanisms to bio-transform it to less toxic states for excretion (Bears *et al.,* 2006). It has a tendency to accumulate in fish tissues and organs and cause serious damages to gills, gastrointestinal tract, kidneys, heart, brain and other organ.

Arsenic is one of WHO's 10 chemicals of major public health concern. Its contamination has become a major environmental concern at global scale with widespread effects on health of living organisms including humans. In the aquatic environment, arsenic exists either as arsenite or arsenate forms, which are inter converted through redox and methylation reactions and its trivalent salt (sodium arsenite) is more toxic than other forms. In aquatic environments, it occurs as a mixture of arsenate and arsenite but former is usually predominating (Prakash and Verma, 2019). The unrestricted application of arsenic pesticides, industrial activities and mining operations has led to the global occurrence of soluble arsenic above permissible levels of 0.010 mg/L. The current recommendation of arsenic in drinking water is 10μg/ L(ppb). High dose of arsenic in drinking water causes skin disease, vascular disease, ischemic heart disease, renal disease, cardiovascular disease, lung disease, cerebro-vascular disease, neurological disorder, reproductive effects and cancer of skin, lung, liver, kidney and bladder in humans (Prakash and Verma, 2021).

Continuous exposure of aquatic organisms to low concentrations of arsenic results in its bioaccumulation in organs especially in liver, kidney and muscles. This deposited arsenic in the fish imposes serious damage to physiology, biochemical disorders such as poisoning of gills, livers, decrease fertility, tissue damage, lesions, and cell death. The accumulation of arsenic affects a variety of physiological systems, including fish growth, reproduction, immune function, and enzyme activity (Datta *et al.,* 2009). It also enters in the cell and produces reactive oxygen species which increases the level of stress which further concentrates the oxidative enzymes and cortisol levels in fish. Such damages alter fish behaviour, homoeostasis, haematology and biochemical mechanisms. Arsenic accumulation variability is observed in fish species. Contaminated fish consumption led to variety of complications in body organ system along epigenetic changes and genetic mutations leading to cancer (Dwivedi and Singh, 2021).

Since fishes are one of the important food sources of man, the man made pollution will return to him through the food they eat. The uncontrolled discharge of arsenic and its devastating impact on fish diversity is a major concern for aquaculture progress and economic stability.

ACUTE TOXICITY OF ARSENIC

Literature review shows that acute toxicity of arsenic is varying from one species to another species. In general, toxicity of arsenic depends on species, sex, age, dose, exposure period, their valence, nature, concentration and organic and inorganic form (Luh *et al.,* 1973). Gupta and Sastry (1981) have reported that difference in acute toxicity may be due to changes in water quality and test species. Sensitivity of fish to arsenic is variable in terms of 96 hr of LC50 with range of10.8 to 105 mg/L (USEPA, 1985). Among the different forms, inorganic arsenicals are more toxic than the organic compounds (Allen *et al.,* 2004). The fish exposed to arsenic were erratic, speedy with exodus movement, hanging vertically with mouth pointed towards the surface and have a slow response to food. A phenomenon known as *nudge and nip* (aggressive response i.e. fishes are biting to other fishes) has been observed (Sahu and Kumar, 2021).

Impact of Arsenic on Histomorphology:

Arsenic causes altered histomorphology of the epidermis. Excessive mucous secretion, increased body discolouration, loosening of skin

and complete loss of skin from the head region and fins were also observed in arsenic exposed fishes. The mucous cells of arsenic exposed aquatic animals show great hyperplasia and hypertrophy. A thick layer of slime very often protects the surface of the skin (Singh and Banerjee, 2008). Increased mucous secretion also reduces respiratory surface and area for gaseous exchange thereby leading to suffocation of fish which consequently, meets its oxygen demand by increasing air gulping (Aktar *et al*., 2017).

Impact of Arsenic on Histopathology:

Arsenic exposure reduced total hepatocyte protein content and suppressed the proliferation of hepatocytes in fishes. Arsenic-induced changes in liver were characterized by dilated sinusoids, formation of intracellular edema, megalocytosis, vacuolation and appearance of hepatic cells with distorted nuclei (Dutta *et al.,* 2007).

Impact of Arsenic on Haematological and Immunological Parameters:

Haematological parameters of fish can be helpful to identify the target organ toxic effects and also the general health condition of harmful changes in stressed organisms and arsenic can leads to changes in haematological parameters of fish (Kavitha *et al*., 2010; Han *et al*., 2019). The level of erythrocytes and haemoglobin were significantly decreased whereas the leucocytes and clotting time increased significantly in arsenic exposed fishes. Further the duration of exposure determines the rate the impact on blood parameters (Kavitha *et al*., 2010). Less production of RBC or an increased loss of these cells in arsenic exposed *fishes* might be the main reason for haemoglobin depletion. Another reason of the decreased haemoglobin content in arsenic exposed aquatic animals was the suppression of haemopoietic activity of the kidney. The decrease in RBCs density and haemoglobin content resulted in diminished oxygen supply (Kumar and Banerjee, 2016). Packed cell volume (PCV) of arsenic exposed fishes was also decreased progressively; the progressive decrease in PCV values is due to the decreased number of RBCs. Haematological indices like MCV, MCH and MCHC are important indicators in diagnosis of anaemia in most animals. The MCV and MCH were also altered in arsenic exposed fishes (Kumar and Banerjee, 2016). The MCV is an indicator of status or size of the red blood cell and reflects the normal or abnormal cell division during erythropoiesis. In arsenic exposed fishes, abnormal shape of RBC was also noted (Vanitha *et al*., 2017).

Leucocytes play key role in the regulation of immunological functions and their numbers increase as a protective response in fish to stress. It has immunosuppressive effect on aquatic animals (Datta *et al*., 2009). High leucocytes count indicate damage due to infection of body tissues, severe physical stress, and as well as leukaemia (Kumar and Banerjee, 2016). Enzyme linked immunosorbent assay indicated that chronic exposure to arsenic suppressed the production of pro-inflammatory 'IL-1 beta like' factors from HKM (Datta *et al*., 2009). These arsenic (As) induced alterations in various haematological parameters may be due to haemolysis or haemorrhage under the action of arsenic induced toxins to the fish (Singh and Banerjee, 2008).

Arsenic exposure affects RBC and WBC because toxicity of arsenic is related to bone marrow damage (Ferrario *et al*., 2008). Such hemato-poietic tissue damage may result in faulty erythropoiesis and low concentration of haematocrit and haemoglobin. In addition, arsenic-induced anaemia due to haemolysis of intravascular erythrocytes may also occur (Cockell *et al.,* 1991).

Impact of Arsenic on Serum Biochemical Parameters:

The presence of arsenic in aquatic media exerts its effect at the cellular or molecular level, which results in significant changes in biochemical parameters along with cytological changes, was observed (Datta *et al.,* 2007). Among these, the blood glucose level was used as an indicator of environmental stress and reflected the changes in carbohydrate metabolism under hypoxia and stress conditions.

The serum glucose level was increased whereas serum protein and lipid contents decreased

significantly in arsenic exposed fish. The increased level of glucose may be due to release of carbohydrates after the breakdown of essential macromolecules such as proteins and lipids from different organ systems, leading to decreasing the concentrations of lipid and protein. Arsenic also affects the glucose metabolism by uncoupling of oxidation and phosphorylation causing excessive availability of unutilized glucose molecules in the tissue. The lowering of protein concentration was perhaps accompanied by the glucose increase, to meet the high energy demand necessary to struggle with the arsenic stress (Kumar and Banerjee, 2016). Decreased plasma protein can be a cause of protein synthesis disorder and appears to be the result of arsenic accumulation in the liver (Lavanya *et al.*, 2011). Serum calcium and magnesium level also decreases in arsenic exposed fishes (Han *et al.*, 2019).

Impact of Arsenic on Metabolism:

Arsenic affected the metabolic process of fish and reduces the nutritive value of fish because even the low concentrations of arsenic are toxic to fishes and alters the biochemical components of fish tissues. Available literature shows a significantly decreased in lipid and increased cholesterol content of liver and muscles tissues of arsenic induced fishes suggested that lipid might have undergone lypolysis, and increased in cholesterol could be due to alteration of steroid biosynthesis or the mitochondrial injury, which impaired the function of Tricarboxylic Acid (TCA) cycle and the fatty acid oxidation mechanism during the stressful situation in the intoxicated fishes (Prakash and Verma, 2019).

Carbohydrates are the primary and immediate source of energy. In stress condition, carbohydrate reserve is depleted to meet energy demand. During stress conditions fishes require more energy to detoxify the toxicants and overcome stress (Das *et al.*, 2012). The decreased in carbohydrate and glycogen content of liver and muscles tissues of arsenic induced fishes suggested that the tissue carbohydrate and glycogen might have undergone glucogenolysis, during the stressful situation in the intoxicated fishes (Verma and Prakash, 2019). Anoxia or hypoxia increases carbohydrate consumption and thereby induces a sort of respiratory stress on organisms even at a sublethal level resulting in additional expenditure of energy. The increased glycogenolysis indicated a general disturbance in carbohydrate metabolism, which might have an adverse effect on the life of exposed animals (Aruljothi *et al.*, 2013; Verma and Prakash, 2019). The decrease in the levels of glycogen and increase in glucose level in different tissues of arsenic induced fish (Aruljothi *et al.*, 2013; Prakash and Verma, 2020a). A significant reduction in the quantity of protein may be due to its conversion to ammoniated residues in order to increase amino acids pool (Pazhanisamy and Indra, 2007; Aruljothi *et al.*, 2013; Kumari and Ramashankar, 2016; Prakash and Verma, 2020b). Verma and Prakash (2020) reported that decrease in alkaline phosphatase activity in liver and muscles facilitate the activity of phosphorylase enzyme and subsequent breakdown of glycogen for energy release during arsenic stress condition.

The tissue protein might have undergone proteolysis, during the stressful situation in the intoxicated fishes to accomplish the impending energy demands (Prakash and Verma, 2020c). The decrease in protein level may be due to their degradation and also to their utilization in metabolic (Das *et al.*, 2012). Increased protease activity and decreased protein levels caused a significant increase in the concentration of free amino acids in fish tissues. Free amino acids increased in fish tissues during arsenic exposure, while elevated fee amino acids levels were used for energy production by delivering them as keto acids to the TCA cycle via amino transferases to increase energy requirements during toxic stress (Prakash and Verma, 2020a).

High concentration of arsenic in the environment causes severe oxidative stress in living organisms thereby the cellular metabolic impairment and tissue damage was observed in fishes (Haque *et al.*, 2016). Thus, the nutritive value of the fish could be altered during the arsenic exposure.

Impact of Arsenic on Nucleic acids:

Nucleic acids contents in epidermis were disturbed in arsenic exposed fish. (Singh and Banerjee, 2008). They reported that RNA and

DNA contents of epidermis were decreased significantly after long term exposure to arsenic in fishes. The decrease in RNA content indicates that arsenic greatly disturbs protein synthesis. Another possible reason for the decrease in protein synthesis was the sloughing of RNA along with the slime.DNA contents was decreased due to the degenerative of cells. Arsenic was found to cause liver chromosomal DNA fragmentation and development of DNA ladder. These are the symptoms of apoptosis. Arsenic causes disturbances in the physiological state of the animal, affecting the activity of enzymes (Das *et al.*, 1012). Degeneration of mitochondrial cristae and condensation of chromatin was also evident in arsenic-exposed hepatocytes (Dutta *et al.*, 2007).

Impact of Arsenic on Enzyme activity:

Enzyme activity changes due to cell death, increased or decreased enzyme production, blockage of a normal secretory pathway, increased cell membrane permeability, or decreased blood flow (Wang *et al.*, 2004). Enzymes, acid and alkaline phosphatases are membrane-bound lysosomal enzymes and the sensitive biomarkers in toxicological study as they provide early information regarding potentially hazardous changes in aquatic organisms inhibited in contaminated water (Verma and Prakash, 2020). These enzymes synthesized in the liver catalyse the hydrolysis of monophosphate esters and their activities usually find a relation to cellular damage. Both enzymes are concerned with the biosynthesis of fibrous proteins. Enzyme SGOT and SGPT activities increase in arsenic exposed fishes (Abdel-Hameid, 2009). The increased levels of these enzymes reflect liver damage due to arsenic toxicity. This means exposure to metal toxicity, such as arsenic, can lead to elevated plasma enzymes as a whole, and significant increases in high concentrations of arsenic suggest that liver regeneration may proceed to restore GOT and GPT levels when exposed to low concentrations of arsenic (Roy and Bhattacharya, 2006).

CONCLUSION

Arsenic is a widespread environmental pollutant that enters the aquatic ecosystem both from nature and from human activities. This review showed that arsenic caused significant changes in behaviour, morphology, haematological and immunological parameters, serum biomolecules as well as in carbohydrate, protein, lipid and nucleic acid metabolism by changing the enzymatic activity of aquatic animals, and these changes may be due to the toxic stress caused by this heavy metal in modal fish. Thus, fish not only provide information about the general health of water bodies, but can also act as a monitor of possible effects in the food chain. Fish with low protein content are not used for nutritional purposes.

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