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Research Article

Consequences of Arsenic Contamination on the Blood Performance and Physiological Status of *Anabus testudineus*

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ABSTRACT

Background: Arsenic (As) is a naturally occurring element found in several water sources, in the form of Arsenate (As (V)) and Arsenite (As (III)). The widespread use of arsenic-based pesticides coupled with industrial activities and mining operations, has increased the concentration of arsenic in aquatic systems. Chronic exposure of freshwater organisms, such as fish, to low levels of arsenic can result in bioaccumulation which may disrupt the metabolic processes and impair the organ system. **Aim:** The present work studies the influence of sub-lethal toxicity of sodium arsenite on haematological and histological changes in the liver of a small indigenous fish of Assam, *Anabas testudineus.* **Materials and Methods:** Live fishes collected from the fish market after being acclimatized in laboratory conditions, were divided into three groups I, II, and III. Group I was taken as control and Group II and III were exposed to sublethal concentration (15 mg/L) of sodium arsenite for 14 and 28 days respectively. RBC and WBC count was done by Neubauer's improved haemocytometer using Heyem's and Turk's solution as a diluting fluid respectively. Haemoglobin was estimated by Sahli's method. Blood cell morphology was studied by making blood smear and observed under light microscope. Liver histopathology was studied by Hematoxylin and Eosin (H&E) staining and light microscopy. **Results:** Arsenic detrimental effects in the blood tissue of the fish increased progressively with increased exposure period. The values of total Erythrocyte Count (RBCs) and haemoglobin concentration decreased with increased duration of exposure but the value of total Leucocyte Count (WBCs), increased contrastingly. The study of the blood cell morphology shows abnormally shaped cells with broken plasma membranes and cytoplasmic blebbing. The liver histology shows degeneration of hepatocytes and sinusoid shrinkage. **Conclusion:** Arsenic's toxic effects in aquatic ecosystems are a growing concern, as it impacts the health of individual organisms and can have broader ecological consequences, potentially affecting food webs and biodiversity. Monitoring and managing arsenic levels in water sources and efforts to reduce anthropogenic sources of contamination are critical for safeguarding aquatic life and public health.

Keywords: Arsenic, Fish, Haematology, Histology, Toxic.

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INTRODUCTION

Arsenic exposure is one of the major menaces to public health targeting India at the top third threatened position. Approximately 140 million people across no less than 70 countries consume water with arsenic levels surpassing the WHO provisional guideline of 10 µg/L.[1]

In 1983, West Bengal was the first influenced state of India to suffer from groundwater contamination of arsenic and since then several other states namely, Jharkhand, Bihar, Uttar Pradesh, the flood plain of the Ganga River, Manipur Assam (flood plain of the Brahmaputra) and Imphal rivers are in trap of its pollution. Besides, the situation is also alarming in Tripura, Nagaland and Arunachal Pradesh (Figure 1).[2] The districts of Assam where arsenic-contaminated water is found include: Nalbari, Jorhat, Barpeta, Dhemaji, Golaghat, Darrang, Nagaon, Sonitpur, Dhubri, Lakhimpur, Cachar, Hailakandi, Karimganj, Goalpara, Kamrup, Sibsagar, Dibrugarh, Bongaigaon, Kokrajhar and Tinsukia. Out of all maximum arsenic was observed in Jorhat, Lakhimpur, Nalbari and Nagaon districts.[3]

Arsenic exists in the environment in various inorganic and organic chemical forms, including Arsenite [As (III)], arsenate [As (V)], Monomethyl Arsenic Acid (MMA), Dimethylarsinic Acid (DMA), Trimethylarsine Oxide (TMAO) and Reobtain (AsB).[4] The toxicity, mobility and solubility of these forms differ.

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Inorganic Arsenic (As (III)) is more toxic than arsenate (As (V)), while organic arsenic compounds are generally less harmful.[5]

The source of arsenic exposure to humans can be natural and anthropogenic sources. In nature, the earth's crust is a copious source of arsenic providing approx. 5 mg/Kg and Arsenopyrite is the most profuse form of it.[6]

Mining, metal smelting and fossil fuel combustion are major sources of arsenic contamination in air, water and soil.[7] Besides burning vegetation and volcanic activity, too imparts arsenic contamination.[8]

Out of all heavy metal arsenic is one of the most persistent in the environment and poses detrimental effects on aquatic organisms, especially to fishes. Arsenic concentration in the environment is of great concern as it is recognised as a cumulative poison to humans and animals. Most organs affected by arsenic are the ones that are involved in absorption, accumulation and excretion processes that occur within various biological systems, including the gastrointestinal tract, circulatory system, skin, liver and kidneys. These mechanisms significantly influence the haematological, biochemical and ion-regulatory parameters of organisms, with particular emphasis on fish in aquatic environments. Alterations in these parameters can serve as valuable indicators for environmental biomonitoring of arsenic contamination.[9]

Arsenic concentration in the environment is of great concern as it is recognised as a cumulative poison to humans and animals. Its exposure to humans is linked to multitudes of disorders which in turn threaten the health, economic and social status of society. Diabetes, hyperkeratosis, cancer, hypertension, neurodegeneration etc., are evident manifestations of its exposure mostly through drinking water in humans.[10]

Usually, fish are contemplated as organisms of choice when evaluating the effects of environmental pollution on aquatic ecosystems.[11] They are continuously exposed to it through their gills and skin and by consuming contaminated food. Fish have been used as sentinels for biomonitoring of aquatic environmental pollutants and are good indicators of As toxicity.[12]

The response of fish to heavy metals is manifested through physiological, biochemical, cellular and molecular changes within the body which may differ among the fishes.[13] Such changes can be used as potential biomarkers to monitor the existence of harmful chemicals in the aquatic environment.[14] These metals alter the structure and function of proteins, enzymes and hormones by binding with their molecular components such as nitrogen, Sulphur, oxygen etc., and interfere with the normal metabolic activities which ultimately damage different organs of fish.[15] The blood profile of an individual serves as a crucial tool for clinically assessing various metabolites and constituents present in the body. This analysis is essential for evaluating the physiological, nutritional and pathological status of an individual. Detection of haematological parameters (RBC, WBC, Hb, Glu etc.,) enzyme and hormone levels is a comparatively easy and quick way to detect any changes/alterations within the fish body due to heavy metals exposure.[16]

Non-essential heavy metals are usually detoxified but due to failure in detoxification accumulation of heavy metals in various organs of fish in various degrees causes pathological changes in the tissues such as gills, liver, kidneys etc.[16,17]

Therefore, by keeping in view all the above-mentioned points a study was planned to monitor the impact of Arsenic trioxide contamination on haematological and histopathological status of *Anabas testudines*.

MATERIALS AND METHODS

Materials

Sample collection and acclimatization

Adult specimens of *Anabas testudines* (weight 25.02±0.4 g) were obtained from the local fish market, Six Mile, Guwahati and were stocked in the laboratory in 40 L glass aquaria with 20 L of water. The fish were acclimatized in the laboratory condition for 7 days. During the period of acclimatization, fish were fed twice daily with floating carp feed and water in the culture tank was exchanged daily to keep the water free of fish excreta and other wastes. Tanks were oxygenised continuously. After acclimatization, only healthy fish were chosen for the experiment.

Preparation of stock solution and determination of LC_{50} value

A stock solution of research-grade Sodium Arsenite ($NaAsO_2$) was prepared by adding 10 g of sodium arsenite to 1000 mL of distilled water. Then, an experiment was performed with different concentrations of metals ($NaAsO_2$:0, 15, 30, 40, 50, 70 and 150 mg/L) to identify the LC_{50} of 96 hr. The stocking density of fish was 7-8 fish individuals per 15 L of water in aquaria. Fish mortality for each concentration was documented at logarithmic time intervals, namely at 6, 12, 24, 48, 72 and 96 hr of fish exposure. The LC_{50} value was determined by following the earlier work done.[18] These aquaria were assorted into three groups.

Group I: control, exposed to 0 mg/L for 28 days,

Group II: exposed to sublethal concentration of Arsenic trioxide (15 mg/L) for 14 days,

Group III: was exposed to sublethal concentration of Arsenic trioxide (15mg/L) for 28 days.

Methods

RBC Count

Red Blood Cells (RBC) were counted by Neubauer's improved haemocytometer using Hyem's and Turk's solution as diluting fluid respectively.[19]

Firstly, fill up the RBC pipette to 0.5 marks with a blood specimen, then fill the same pipette with RBC diluting fluid up to 101 marks and mix the blood and diluting fluid. Placed the mixture in a clean Neubauer chamber and viewed it under a microscope. The RBC count was done using the following formula:

$$\text{RBC} = \text{Number of cells counted (N)} \times \text{Dilution factor} / \text{Area} \times \text{Depth}$$

WBC Count

White Blood Cells (WBC) were counted by Neubauer's improved haemocytometer using Heyem's and Turk's solution as a diluting fluid respectively.[19] described by Darmady and Davenport.

Firstly, fill up the WBC pipette to 0.5 marks with blood specimen, then fill the same pipette with WBC diluting fluid (preferably Turk's fluid) up to 11 marks, mixed the blood and diluting fluid. Placed the mixture in a clean Neubauer chamber and viewed it under a microscope. The WBC count was done using the formula:

$$\text{WBC} = \text{Number of cells counted (N)} \times \text{Dilution factor} / \text{Area} \times \text{Depth}$$

Haemoglobin Count

For this Sahli's method was adopted. The graduated tubes were first cleaned with distilled water and dried thoroughly before use. With the help of a dropper placed N/10 HCl in a diluting tube up to the mark 20. The blood was taken in Hb pipette up to 20 cubic mm mark and blew it into a diluting tube. After 10 min add distilled water in drops and mix the tube until it has exactly the colour as comparison standards and reading were taken.

Histological preparations

The fixed liver tissue was dehydrated in a graded alcohol series, cleaned with xylene and embedded in paraffin. Embedded tissues were sectioned at a thickness of 5 µm with a microtome machine (HM 430; Thermo-Scientific). The tissue slices were arranged on slides like ribbons and stained with standard Haematoxylin-Eosin (H-E) protocol before being mounted with DPX and a coverslip. Finally, stained slides were examined under a microscope to identify histological abnormalities in liver tissue.

Study of blood cell morphology

Blood samples from the three groups were taken from the caudal veins of the fishes with the help of a heparin-rinsed micro-syringe. A thin blood film was drawn on a clean grease-free slide, fixed in methanol and was stained in Leishman's stain.[20]

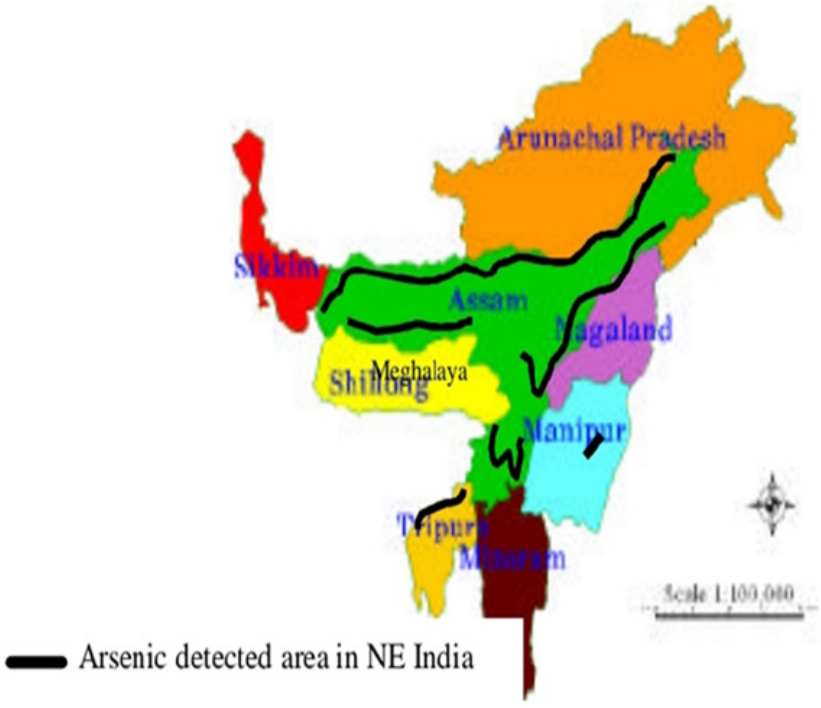

Figure 1: Distribution of Arsenic in North Eastern Region of India.

RESULTS

RBC and WBC count

The treatment of fishes with the sublethal concentration of 15 mg/L of arsenic for 28 days showed impactful results in haematology (Table 1). The RBC count decreased significantly (p=0.001, at a significance level of $p \leq 0.05$) at 14 days and (p=8.14E-074, at a significance level of $p \leq 0.05$) at 28 days respectively.

Conversely, the WBC count increased significantly both on the 14 and 28 days. (p=0.008 at a significance level of $p \leq 0.05$). The haemoglobin % was found to decline significantly with increased time of exposure. (p=3.31E-07 at a significance level of $p \leq 0.05$).

BLOOD CELL MORPHOLOGY

Control: Normal nucleated erythrocytes, eosinophils, neutrophils, small lymphocytes and monocytes are seen. Cells are regular and are even (Figure 2a).

Treated (14 Days)

Abnormal-shaped cells with broken plasma membrane are seen.

Cell sticking, fused erythrocytes with cytoplasmic blebbing are seen (Figure 2b).

Treated (28 Days)

The numbers of abnormal cells increase, erythrocytes become slender with plasma membrane invagination. The nucleus of the RBC has become enlarged. Echinocytes or spiculated RBC can be seen (Figure 2c).

HISTOPATHOLGY

Control

In a controlled fish's liver, histoarchitecture was seen as normal, with distinct normal hepatocytes, central vein, normal sinusoids and hepatocyte nuclei (Figure 3 a).

Treated (14 Days)

When treated in as for 14 days the histoarchitecture of the liver showed dilation in the central vein. Some areas showed necrosis of liver cells. Additionally, the normal architecture of hepatocytes was lost and the liver section showed cell degeneration and cellular debris. Shrinkage of sinusoids is observed (Figure 3b).

Treated (28 Days)

Upon further treatment for 28 days, the liver histoarchitecture showed increased degeneration of hepatocytes. Central vein dilation and congestion in blood vessels were seen. Hepatocytes underwent furthermore degeneration and sinusoid shrinkage is observed (Figure 3c).

DISCUSSION

Aquatic animals accumulate the metallic materials present in the water bodies as an input of pollution from industrial, mining or agricultural waste and other contaminants in their tissue, which upon metabolism gets released from the body, but failure of metabolism causes bioaccumulation.[21] Consolidation of heavy metals affects a variety of physiological systems, counting fish growths, reproduction, immune function, hemato-biochemical changes, hormonal dysfunctions, histopathological anomalies, embryonic and larval development retardation and enzyme activity.

Results derived from the present study reveal significant alterations in the haematological parameters of the treated fishes upon exposure to sub-lethal concentrations of arsenic when compared to the control group (Figure 4). Haematological parameters such as RBC, WBC, Hb and Ht of fish indicate the health status of an organism.[22]

There is a significant decrease (p<0.05) in RBC count and Hb content. A striking difference of 4.98±0.05 is observed between the RBC count of the control group and treated 28-day fishes. Hb content also reduced gradually with time from 16.11±0.13 to 6.66±1.76 with a gradual time of exposure (Figure 4). Reduction in Hb content is an excellent cause of the anaemic condition of the fish.[23] Inhibition of erythropoiesis due to damage to viable cells can be the leading cause of decreased Hb as has been documented.[24] The results converge with the findings of another worker[25] who reported changes in haematological, biochemical and enzymological biomarkers due to fluoride toxicity in the same fish, *Anabas testudines*. The observation of blood cell morphology shows devastating effects in the Group II and III fishes including cell swelling, cell blebbing, cell sticking, broken plasma membrane and cell invaginations which are all signs of cell injury, generally caused by xenobiotics, toxins and heavy metal exposure.[26]

The correlation scatter plot (Figure 5) clearly shows a strong negative correlation of WBC with RBC count (R^2=0.8386). This increase may be induced as a result of the body's increased

Table 1: Effect of Arsenic trioxide in RBC and WBC count of *Anabas testudines*.

Fish	Total RBCs (×106/mm3)	Total WBCs (×106/mm³)	Haemoglobin (g/dL)
Control	7.07±0.07	7.08±0.48	16.11±0.13
Treated (14 days)	4.72±0.34*	10.32±1.08*	9.10±0.10*
Treated (28 days)	2.09 ±0.02*	14.07±1.21*	6.66±1.76 *

The asterisk on the values (mean±SE) in the row represents a significant statistical ($p \leq 0.05$) difference among treatment groups compared to control.

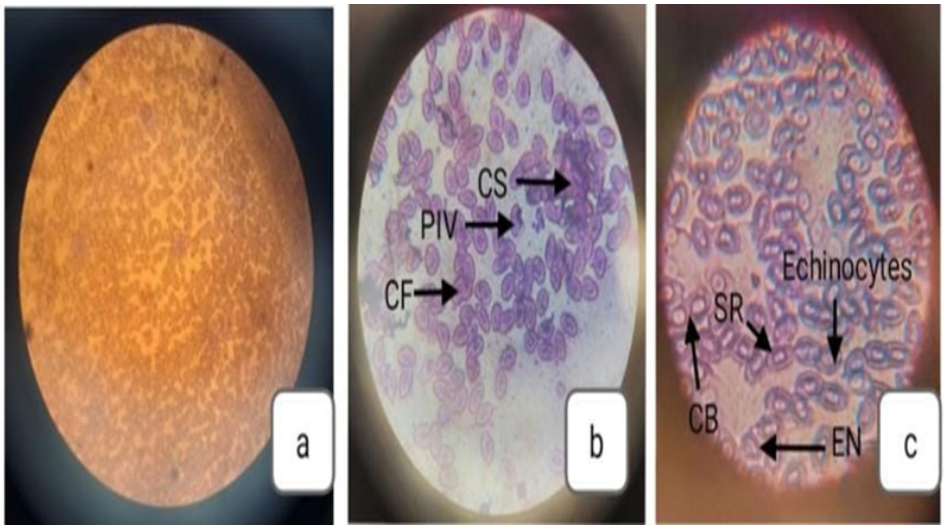

Figure 2: (a) Blood cells of control fish (4X), (b) Blood cells of 14 days treated (10X); CF: Cell fusion (due to breakage of PM); PIV: Plasma membrane invagination; CS: Cell sticking, (c) Blood cells of 28 days treated (10X); CB: Cell blebbing; Echinocytes; EN: Enlarged nucleus; SR: Slender RBC.

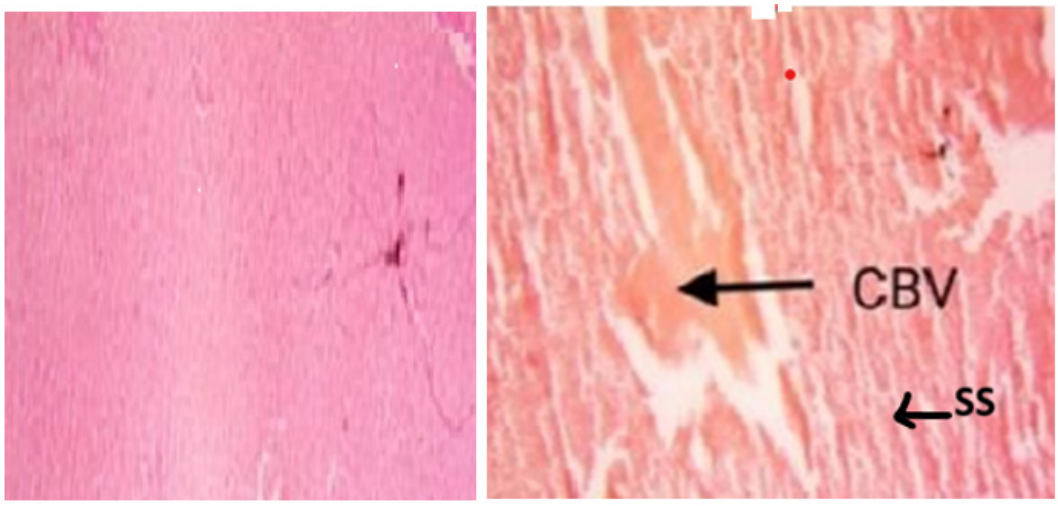

Figure 3: (a) Liver of control fish (10X); (b) Liver of 28 days treated (10X); CBV: Congestion of Blood Vessels, SS: Shrinkage of Sinusoids.

immune response to protect the fish from further damage due to arsenic stress.[27] A similar decline in RBC count and Hb concentration along with a hike in leucocyte count were noticed when *Clarius batrachus* was exposed to different concentrations of $HgCl_2$.[28] Various other toxic elements also lead to induction in leucocyte count in fishes.[29] However, the present results disagree with a previous report where a decrease in leucocyte count was observed in *Channa striata* when exposed to ammonium and lead salts.[30]

The liver, recognized as the largest organ in the human body, functions as the primary site for detoxification and serves as a major centre for metabolism. Due to its role in processing various substances, the liver is particularly vulnerable to a range of disorders resulting from exposure to both intrinsic and extrinsic toxins. Moreover, the liver is vital for regulating energy levels and ensuring the structural integrity of the body.

Though the liver plays an important role in the metabolic process and detoxification, acute exposure to arsenic may lead to its accumulation in the liver and cause pathological alteration.[31]

The findings of the present study show normal histoarchitecture of the liver in control fishes, while 14 days As (Arsenic trioxide) treated shows changes in histoarchitecture such as dilation in the central vein, necrosis of liver cells and shrinkage of sinusoids (Figure 3).

Upon continued exposure for 28 days, further deterioration of histoarchitecture took place such as more dilation in the central vein, blood vessel congestion, further degeneration of hepatocytes and sinusoid shrinkage increased (Figure 3). Cellular degeneration and the subsequent necrosis would be the result of oxygen deficiency, as a result, there is dilation in hepatic blood vessels leading to the formation of hemosiderosis areas.[32]

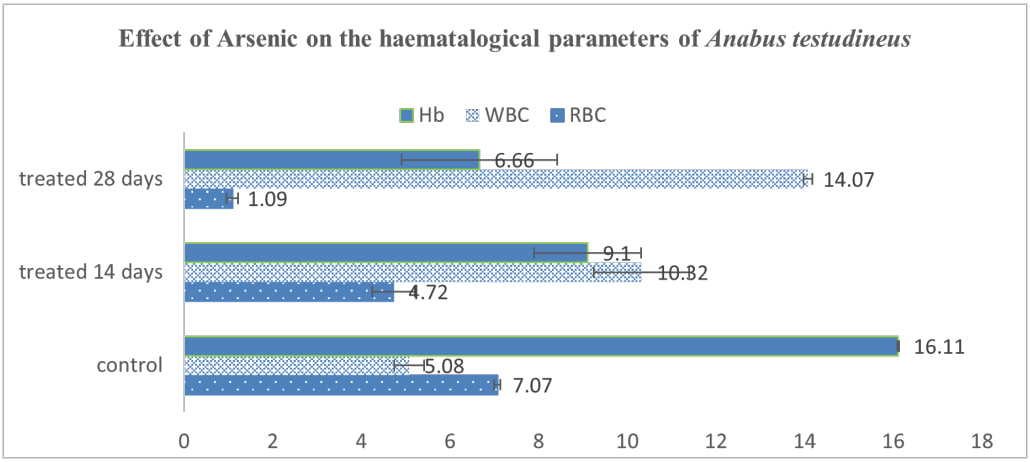

Figure 4: Bar graph with error bars showing significant variation of RBC, WBC and Haemoglobin content in Arsenic treated *Anabas testudines*.

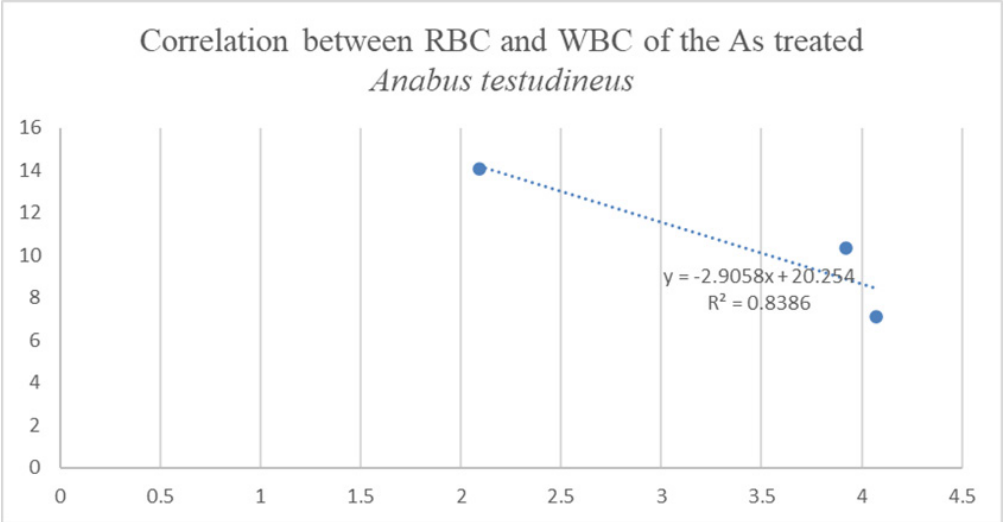

Figure 5: Scatter plot showing the correlation between RBC and WBC count of the Arsenic treated *Anabas testudines*.

Fish liver is integral to the processes of absorption, bioaccumulation, biotransformation and elimination of arsenic. In the hepatic tissues of *Clarias batrachus* and *Catla catla*, various pathologies were identified, including congestion, cloudy hepatocyte swelling, karyolysis, vacuolar degeneration, nuclear hypertrophy and dilation of the sinusoids.[33] Furthermore, acute exposure to the organophosphate Dichlorvos in the liver tissues of *Cirrhinus mrigala* exhibited comparable injuries, which were characterized by hepatocyte shrinkage, sinusoidal dilation, cloudy swelling, vacuolar degeneration, focal necrosis and nuclear hypertrophy.[34]

CONCLUSION

From the present study, it can be confirmed that acute exposure to arsenic has a mighty potential to transmute the biochemical and haematological factors in various tissues of *Anabas testudines*. Arsenic primarily affects the blood of fish by inducing hemolysis (destruction of red blood cells), leading to anaemia. The accumulation of arsenic in the blood also disrupts enzyme activity, contributing to cellular damage and changes in blood parameters, including a reduction in haemoglobin concentration and red blood cell count, all of which impair the fish's ability to survive in oxygen-poor environments. The haematological criterion is a very keen criterion for supervision/monitoring of the toxic responses of the fish. Arsenic also targets various organs in fish, including the liver.

Comprehending the noxious consequence of As in the aquatic terrain it is important to mitigate its detrimental effects on aquatic health, particularly in fish and the organisms including humans who consume fish. The accumulated metals that travel through the food chain have a range of adverse effects, including physiological, metabolic, histopathological, biochemical and behavioural changes.

Regular monitoring of arsenic levels and their health effects in aquatic organisms can provide insights into overall aquatic health and may also serve as a warning for potential impacts on the food chain.

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CONFLICT OF INTEREST

The authors declare that they have no financial or non-financial interests that are directly or indirectly related to this research work.

ABBREVIATIONS

RBC: Red Blood Cell; **WBC:** White Blood Cell; **WHO:** World Health Organisation; **As:** Arsenic; **Hb:** Haemoglobin; **Glu:** Glucose; **Ht:** Haematocrit; **$HgCl_2$:** Mercuric Chloride; **CBV:** Congestion of Blood Vessels; **SS:** Shrinkage of Sinusoids; **NE:** North East; **CS:** Cell Sticking; **CB:** Cell Blebbing; **EN:** Enlarged nucleus; **SR:** Slender; **CF:** Cell Fusion; **PIV:** Plasma membrane invagination; **HCl:** Hydrochloric acid; **DPX:** Dibutylphthalate Polystyrene Xylene.

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