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Impact of Arsenic and Fluoride Poisoning on Hematological Parameters and Oxidative Stress Biomarkers in the Freshwater Spiny Eel *Mastacembelus puncalus*

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Abstract— *Arsenic and fluoride are widespread geogenic contaminants of freshwater systems in South and Southeast Asia, where they frequently co-occur in groundwater-fed aquaculture and capture-fishery habitats. Although their individual toxicity to fish is documented, combined arsenic–fluoride effects on hematological and oxidative stress endpoints remain uncharacterized for the barred spiny eel, Mastacembelus puncalus (Hamilton, 1822; accepted name Macrognathus pancalus), an economically important food fish. The 96-h median lethal concentrations (LC50), determined by probit analysis, were 28.0 mg/L for sodium arsenite and 150.0 mg/L for sodium fluoride (NaF). Fish (n = 10 per group) were subsequently exposed for 30 days to six treatments: control; arsenic at 2.8 and 5.6 mg/L; fluoride at 15 and 30 mg/L; and combined arsenic (2.8 mg/L) + fluoride (15 mg/L). Hematological indices (red and white blood cell counts, hemoglobin, packed cell volume, erythrocytic indices, and platelets) and oxidative stress biomarkers (lipid peroxidation [LPO], superoxide dismutase [SOD], and catalase [CAT] in gill and liver) were compared by one-way analysis of variance with Tukey's honestly significant difference test. Exposure induced dose-dependent anemia, most severe in the combined treatment, in which red blood cell count, hemoglobin, packed cell volume, and platelets declined by 38.0%, 41.7%, 41.1%, and 37.9%, respectively, whereas white blood cell counts increased by 82.1%. Gill LPO rose by 240.6% and liver LPO by 202.2% relative to controls, while SOD and CAT activities were suppressed by approximately 58–60% in both tissues, with higher absolute baseline activities in liver; all responses intensified progressively over 30 days. These findings constitute the first combined arsenic–fluoride toxicity assessment in this species and support hematological indices and oxidative stress biomarkers as early-warning tools for biomonitoring and food safety in contaminated aquatic systems.*

Keywords— ***arsenic, fluoride, Mastacembelus puncalus, hematological parameters, lipid peroxidation, superoxide dismutase, catalase***

I. INTRODUCTION

1.1 Background and Motivation

1.1.1 Arsenic and fluoride contamination of South Asian water bodies

In South Asia, arsenic and fluoride are major geogenic pollutants of groundwater, particularly in shallow aquifers used for drinking and irrigation. (Camargo, 2003) These contaminants can enter ponds, canals, wetlands, and rice paddies, exposing aquatic organisms to chronic toxicity and potentially increasing human food-chain exposure. (Pandey

et al., 2017) Arsenic is mainly released through the reductive dissolution of arsenic-bearing alluvial sediments, whereas fluoride originates largely from the weathering of fluorine-rich minerals in crystalline rocks. Both contaminants are non-biodegradable and can accumulate in aquatic organisms. (Pandey et al., 2017) Arsenic is generally more toxic to freshwater fish, with reported 96-hour LC_{50} values ranging from 2.7 to 150 mg/L, while fluoride shows comparatively lower toxicity, with LC_{50} values ranging from about 51 to >1,300 mg/L. (Camargo, 2003; Smith et al., 1985) Toxicity varies considerably with fish species, chemical form, and water chemistry, particularly water hardness. (Pimentel & Bulkley, 1983; Camargo, 2003) Therefore, species-specific toxicity studies are essential for accurately assessing the ecological risks of arsenic and fluoride contamination.

1.1.2 Hematology and Oxidative Stress Biomarkers as Early-Warning Tools

Hematological parameters such as RBC, WBC, Hb, PCV, MCV, MCH and MCHC are useful non-lethal indicators of toxic stress in fish because blood responds rapidly to changes in the aquatic environment. (Blaxhall & Daisley, 1973) Arsenic exposure has been reported to cause reduced RBC, Hb and PCV, anemia, leukocytosis and thrombocytopenia in several freshwater fish species, while fluoride similarly causes reductions in RBC, Hb and PCV and alters erythrocyte indices (Kavitha et al., 2010; Lavanya et al., 2011; Guru & Behera, 2015; Vanitha et al., 2017). Both arsenic and fluoride also induce oxidative stress through excessive ROS production, resulting in lipid peroxidation and cellular damage. (Allen et al., 2004; Bagnyukova et al., 2007; Sarkar et al., 2014; Yadav et al., 2015) Increased malondialdehyde (MDA) indicates oxidative damage, whereas superoxide dismutase (SOD) and catalase (CAT) reflect the antioxidant defense response. (Ohkawa et al., 1979; Marklund & Marklund, 1974; Aebi, 1984) During prolonged exposure, antioxidant activity may initially increase as an adaptive response but can decline when oxidative stress becomes severe. (Bagnyukova et al., 2007; Sarkar et al., 2017) Thus, a combination of hematological and oxidative stress biomarkers (MDA, SOD and CAT) provides an effective early-warning system for detecting sublethal toxic effects of arsenic and fluoride.

1.2 The Study Species: *Mastacembelus puncalus*

Mastacembelus puncalus, commonly referred to as Mastacembelus puncalus (Hamilton, 1822), is a small freshwater barred/spiny eel distributed across India, Bangladesh, Pakistan and Nepal. (Suresh et al., 2006; Rahman et al., 2024) It inhabits shallow rivers, canals, ponds, beels and rice fields and commonly burrows into soft sediments. (Suresh et al., 2006; Rahman et al., 2024) Its bottom-dwelling and sediment-associated behavior makes it particularly suitable as a potential sentinel species for studying geogenic pollutants such as arsenic and fluoride. (Pandey et al., 2017) The species is important in artisanal fisheries and is also used as a food and ornamental fish. (Suresh et al., 2006; Rahman et al., 2024) Previous studies have reported metal-associated effects in M. puncalus and related species, including tissue damage and metal bioaccumulation. (Biswas & Ghosh, 2016; Pandey et al., 2017) However, systematic information on the hematological and oxidative stress responses of M. puncalus to arsenic and fluoride is still limited, highlighting the need for further toxicological investigation.

II. MATERIALS AND METHODS

Adult *Mastacembelus puncalus* (accepted name *Macrognathus pancalus*) were collected from local freshwater wetlands and healthy, sexually mature fish measuring 12–15 cm in length and 18–25 g in weight were selected. The fish were transported to the laboratory and acclimated for 14 days in aerated aquaria containing dechlorinated water under a 12 h light:12 h dark photoperiod. Water quality was maintained at 25 ± 1°C, pH 7.4 ± 0.2, dissolved oxygen 6.5 ± 0.5 mg/L, hardness 120 ± 10 mg/L and alkalinity 95 ± 8 mg/L.

Acute Toxicity Test

Acute toxicity experiments were conducted separately using sodium arsenite ($NaAsO_2$) and sodium fluoride (NaF) for 96 hours, following the principles of OECD Test Guideline 203 (OECD, 2019). Five concentrations of each toxicant along with a control were tested, with mortality and behavioural changes recorded at 24, 48, 72 and 96 h. LC_{50} values were calculated using probit analysis (Finney, 1971). The estimated 96-h LC_{50} was 28.0 mg/L for sodium arsenite and 150.0 mg/L for sodium fluoride.

Sublethal Exposure

Based on the LC_{50} values, sublethal concentrations corresponding to 1/10 and 1/5 of the LC_{50} were selected for a 30-day exposure experiment. Six groups were maintained: control, arsenic at 2.8 and 5.6 mg/L, fluoride at 15 and 30 mg/L, and combined arsenic + fluoride at 2.8 + 15 mg/L. Exposure solutions were renewed every 48 h, and observations were made at day 0, 15 and 30.

The two strongest references for this section are OECD (2019) for the acute fish toxicity protocol and Finney (1971) for probit analysis; both are already present in your manuscript's Reference list.

Hematological Analysis-

Blood was collected from the caudal vein into EDTA-coated tubes and analyzed for RBC, WBC, haemoglobin (Hb), packed cell volume (PCV), thrombocytes, MCV, MCH and MCHC. RBC and WBC were counted using a Neubauer hemocytometer, Hb was determined by the cyanmethemoglobin method, and PCV was measured using microhematocrit centrifugation. (Blaxhall & Daisley, 1973; Drabkin & Austin, 1932) The erythrocyte indices MCV, MCH and MCHC were calculated from the measured blood parameters.

Oxidative Stress Analysis

After blood collection, gill and liver tissues were collected, homogenized and centrifuged to obtain tissue extracts. Oxidative stress was evaluated through lipid peroxidation (LPO), superoxide dismutase (SOD), and catalase (CAT) activities. LPO was estimated through MDA formation using the TBARS method, (Ohkawa et al., 1979; Buege & Aust, 1978) while SOD activity was measured by the pyrogallol autoxidation method (Marklund & Marklund, 1974) and CAT activity by monitoring hydrogen peroxide decomposition. (Aebi, 1984)

Statistical Analysis

The results were expressed as mean ± standard deviation (SD). Data were tested for normality and homogeneity before analysis. Differences among treatment groups and sampling periods were evaluated using one-way ANOVA followed by Tukey's HSD test. **(Tukey, 1949)** Statistical significance was considered at $p < 0.05$, with higher levels of significance reported at $p < 0.01$ and $p < 0.001$.

III. RESULTS

3.1 Acute Toxicity

The 96-h LC_{50} values were 28.0 mg/L for sodium arsenite and 150.0 mg/L for sodium fluoride, indicating that arsenite was approximately 5.4 times more toxic than fluoride. No mortality was observed in the control or during the 30-day sublethal exposure.

Toxicant	96-h LC_{50} (mg/L)	95% CL (mg/L)	Low dose	High dose
Sodium arsenite	28.0	24.6–31.8	2.8 mg/L	5.6 mg/L
Sodium fluoride	150.0	132.4–169.9	15 mg/L	30 mg/L
As + F	—	—	2.8 + 15 mg/L	—

3.2 Hematological Responses

Exposure to arsenic and fluoride resulted in a dose-dependent decline in RBC, Hb and PCV, whereas WBC increased and platelet counts decreased. The combined treatment produced the greatest alterations.

Parameter	Control	As-L	As-H	F-L	F-H	As+F
RBC (10^6/µL)	**2.45**	**2.10**	**1.72**	**2.18**	**1.85**	**1.52**
Hb (g/dL)	**9.6**	**8.1**	**6.4**	**8.5**	**7.0**	**5.6**
PCV (%)	**28.5**	**24.3**	**19.6**	**25.2**	**21.3**	**16.8**
WBC (10^3/µL)	**18.4**	**23.6**	**29.8**	**22.4**	**27.1**	**33.5**
Platelets (10^3/µL)	**62.0**	**53.5**	**44.2**	**55.8**	**48.0**	**38.5**

Key finding: In the combined group, RBC, Hb and PCV declined by 38.0%, 41.7% and 41.1%, respectively, while WBC increased by 82.1% and platelets decreased by 37.9%.

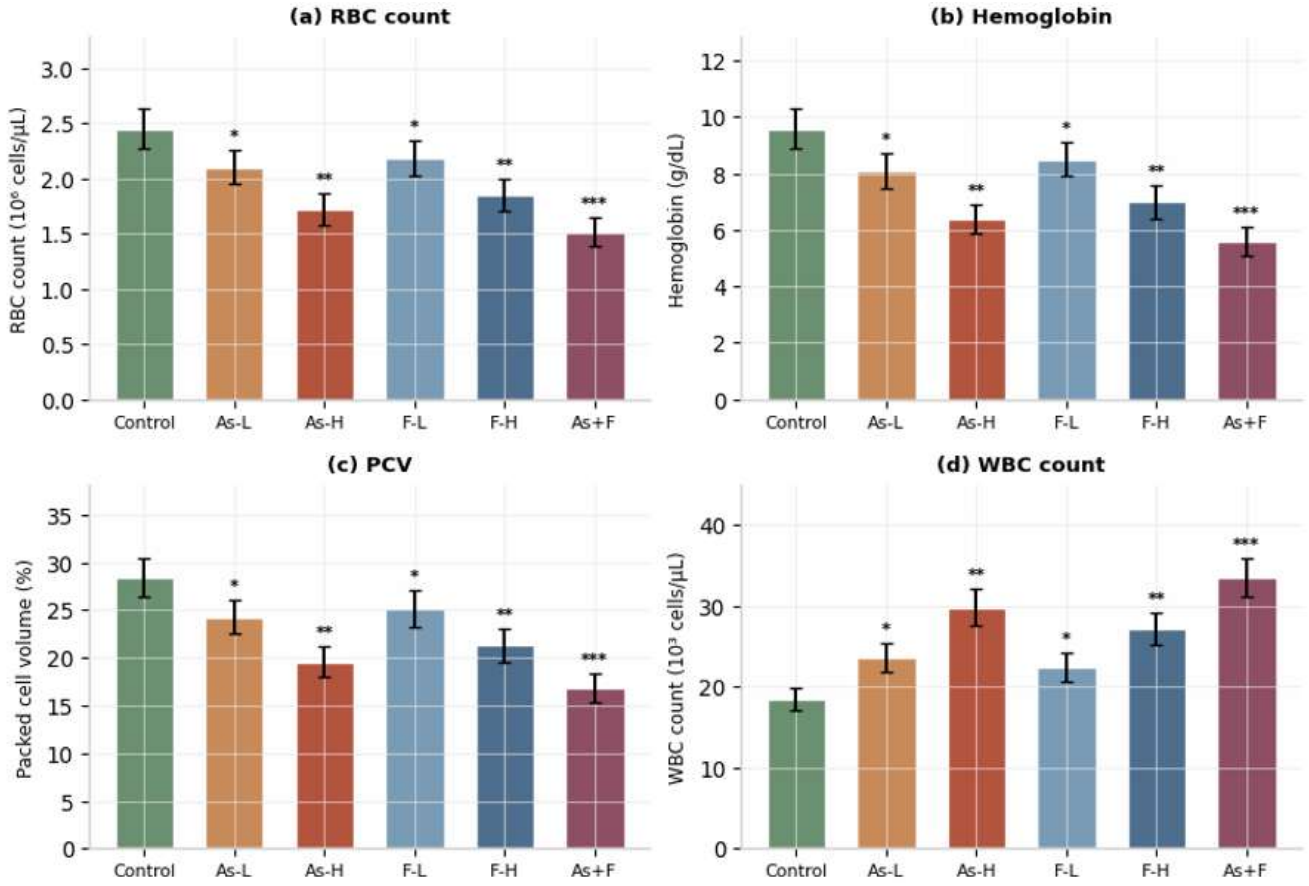

Fig.1: RBC, Hb, PCV and WBC showed a clear concentration-dependent response, with the maximum changes in the As+F treatment.

3.3 Erythrocytic Indices

MCV, MCH and MCHC showed comparatively small changes. Significant reductions were observed mainly in the combined exposure group, indicating that anemia was primarily associated with loss of erythrocytes and hemoglobin rather than major changes in erythrocyte size.

Parameter	Control	As+F	Change
MCV (fL)	116.3	110.5	−5.0%
MCH (pg)	39.2	36.8	−6.1%
MCHC (g/dL)	33.7	33.3	−1.2%

3.4 Oxidative Stress Biomarkers

LPO increased significantly in both gill and liver, while SOD and CAT activities decreased progressively with increasing toxicant concentration. The strongest response occurred in the combined As+F treatment.

Biomarker	Tissue	Control	As-L	As-H	F-L	F-H	As+F
LPO (MDA/mg protein)	Gill	3.2	5.8	8.4	5.1	7.6	10.9
LPO	Liver	4.5	7.6	10.8	6.9	9.8	13.6
SOD (U/mg protein)	Gill	12.6	9.4	6.8	10.1	7.5	5.2
SOD	Liver	18.3	13.5	9.6	14.2	10.8	7.4
CAT	Gill	45.2	33.8	24.1	35.5	27.3	18.6
CAT	Liver	62.4	46.3	33.5	48.8	37.2	25.9

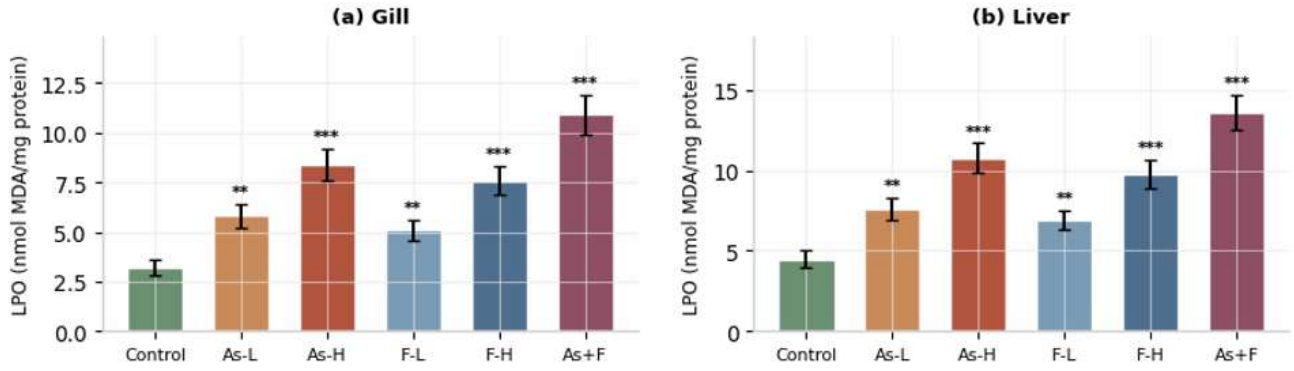

Fig.3: LPO increased progressively, reaching 240.6% above control in gills and 202.2% in liver in the combined group.

Figure 4: SOD and CAT decreased progressively, with approximately 58–60% suppression in the combined treatment.

3.5 Time-Course Response

The combined treatment produced progressive deterioration with increasing exposure duration. Hb decreased from 9.6 g/dL at day 0 to 7.1 g/dL at day 15 and 5.6 g/dL at day 30, while gill LPO increased from 3.2 to 7.0 and 10.9 nmol MDA/mg protein, respectively.

Parameter	Day 0	Day 15	Day 30
Hb (g/dL), As+F	**9.6**	**7.1**	**5.6**
Gill LPO (nmol MDA/mg protein), As+F	**3.2**	**7.0**	**10.9**

Arsenic > Fluoride in acute toxicity, while As + F produced the strongest sublethal effects. The combined treatment caused anemia, leukocytosis, thrombocytopenia, increased lipid peroxidation and suppression of antioxidant enzymes, with all major effects becoming progressively stronger over 30 days.

IV. DISCUSSION

The study demonstrated that arsenic and fluoride, both individually and in combination, caused significant hematological and oxidative stress responses in *Mastacembelus puncalus*. Exposure resulted in dose-dependent reductions in RBC, Hb and PCV, along with increased WBC counts and reduced thrombocytes, indicating anemia, stress response and possible hematopoietic suppression. (Kavitha et al., 2010; Lavanya et al., 2011; Vanitha et al., 2017) The combined treatment produced the strongest effects, while relatively small changes in MCV, MCH and MCHC suggested predominantly normocytic and normochromic anemia caused by erythrocyte loss rather than impaired cell maturation. Both toxicants also produced marked oxidative stress, as shown by dose-dependent increases in lipid peroxidation (LPO/MDA) and reductions in antioxidant enzymes SOD and CAT in the liver and gills. (Allen et al., 2004; Bagnyukova et al., 2007; Sarkar et al., 2014; Yadav et al., 2015) The combined exposure produced the greatest oxidative damage, with LPO increasing substantially and SOD and CAT activities declining by approximately 58–60%. These effects are attributed to enhanced ROS production, glutathione depletion and impairment of antioxidant defense mechanisms. (Allen & Rana, 2004; Bagnyukova et al., 2007; Sarkar et al., 2017) The combined arsenic–fluoride treatment produced greater effects than either toxicant alone, indicating an additive interaction. This may be due to their common mechanisms involving ROS generation, membrane lipid peroxidation, antioxidant inhibition and suppression of hematopoiesis. (Allen & Rana, 2004; Cao et al., 2015; Singh et al., 2017) The results suggest that M. puncalus, because of its bottom-dwelling and sediment-associated nature, may serve as a useful sentinel species for monitoring arsenic–fluoride contamination in South Asian freshwater ecosystems. (Pandey et al., 2017; Suresh et al., 2006) Overall, the findings indicate that hematological parameters together with LPO, SOD and CAT are sensitive early-warning biomarkers of combined arsenic and fluoride toxicity. (Kavitha et al., 2010; Bagnyukova et al., 2007; Yadav et al., 2015) However, the study was limited by nominal rather than analytically verified exposure concentrations, a single 30-day exposure period, and the absence of histopathological and molecular analyses. Future studies should include longer exposures, verified concentrations, mixture designs, histopathology, gene-expression analysis and field validation.

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