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An Insight into Effects of Pollution in Aquatic Environment through Effects of Nickel Accumulation on Catalase Activity of Clarias Gariepinus

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Abstract

Aquatic ecosystem is the natural habitat of fish and water pollution that directly affect underwater fish and human beings are indirectly at high risk by consuming these fish. This study investigated the effects of nickel accumulation on catalase activity Clarias gariepinus. The level nickel bioaccumulations were determined after the exposure of the fish sample to concentrations of nickel using of Principle of Atomic Absorption Spectrophotometer (AAS 240 FS) and the effects of accumulation assessed using catalase as a bioindicator. The results indicated that a short-term exposure to nickel induced stress reaction in fish as there was an increase in the catalase activity. Thus exposure of fish and other aquatic organisms to toxicants such as nickel could lead to physiological stress.

Keywords: Pollution; Heavy metals; Nickel; Clarias gariepinus; Atomic absorption spectrophotometer; Toxicant; Physiological stress

Introduction

Aquatic ecosystem is the natural habitat of fish and water pollution that directly affect underwater fish and human beings are indirectly at high risk by consuming these fish [1,2]. Industrial, agricultural and domestic wastes discharge in water bodies leads to the pollution of these bodies through ecological and environmental hazards [3,4]. Humans and animals health in water ecosystem are threatened by production of high contents of metal by these pollutants (Sekabira et al., 2010). Effluents can accumulate in living tissues for longer period, but fish is being negatively influenced than other species [5]. Zinc (Zn), Copper (Cu), and Nickel (Ni) are essential ions for the maintenance requirements of fauna and flora [6]. Bioaccumulation of these pollutants has adverse effects for biota and the aquatic life [7,8]. Some heavy metals act as a cofactor or as chelates for vital metabolic reactions. Even at low concentrations heavy metals still exert their deleterious effects. Accumulation of heavy metals by aquatic organisms leads to the production of Reactive Oxygen Species (ROS). The reactive oxygen species such as hydrogen peroxide, superoxide radical and hydroxyl radicals cause oxidative stress in living organisms [9,10]. There would be development of the defensive mechanisms for Reactive Oxygen Species (ROS) in living bodies. Antioxidant enzymes such as Super Oxide Dismutase (SOD), catalase (CAT) and POD functions in a synchronized way to counteract oxidative stress [11]. Antioxidant enzymes in liver, gills and kidney provide defensive mechanism against Reactive Oxygen Species (ROS), but liver is the main metabolic center that takes part in detoxification (Bagnyukova et al., 2006). The contaminants from water enters in fish through food, non-food particles, skin, gills and water then absorbed in blood and passed to liver for transformation or storage [5,12]. An examination of the *in vitro* accumulation of toxicants such as nickel will give an insight into the possible effects and consequences in the aquatic environment.

Materials and Methods

Fingerlings of Clarias gariepinus of similar age (4 weeks old) were collected from Applied Biology and Biotechnology local laboratory of Enugu State University of Science and Technology, Enugu State. The test animals (fingerlings) were allowed to acclimatize to laboratory conditions (28°C ± 20°C, RH 70% ± 2%) for a period of seven days before they were used in bioassays. During the acclimatization, the fingerlings were fed with 0.5 mm clarias feed. Water in the plastic tanks was changed once every two days to prevent accumulation of wastes and decaying of food particles. Feeding was discontinued 12 hours prior to commencement of bio-assays. Clarias gariepinus (Juveniles) were exposed to sub-lethal concentrations of heavy nickel salt for a period of 7 days in

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Table 1: Concentrations of nickel in the flesh of the fish after seven (7) days of exposure.

Conc. introduced ($mgkg^{-1}$)	Conc. Accumulated (mkg^{-1}) M + SD
Control	0.000 ± 0.000
1	0.000 ± 0.000
2	0.061 ± 0.000
3	0.342 ± 0.395
4	0.431 ± 0.498

single metal bioassays. These metal salts were made up to 1 liter with de-ionized portable water. In the procedure followed each test media was changed into a fresh solution at the same concentration of heavy metal salt or untreated control as the case may be, once every two days and transferring the same exposed test animals into the freshly prepared test media over the 7 days period of experimentation. On the seventh day, two Clarias gariepinus were randomly selected from both treated and untreated test media and dissected. The muscle (edible part) was carefully extracted, kept in separate bottles and preserved in the refrigerator ready for digestion (for heavy metal analysis) and homogenization (for biochemical assay). The digest of the test sample were assayed for the presence of heavy metals using atomic absorption spectrophotometer spectra AA model number 240 FS under the appropriate wavelength and detection limit for the nickel. Further analysis was done to determine the catalase activity in the extracted muscles of the fish.

Statistical analysis

After checking for normality and homoscedasticity, the results obtained from each group and each exposure period were cross compared by parametric (ANOVA) or non-parametric analysis of variance and differences among treatment groups were identified respectively. Results are presented as mean+SD, values of $p<0.05$ were considered significant.

Results

Concentrations of nickel in the flesh of the fish after seven (7) days of exposure shown in Table 1 and Concentrations of nickel in introduced and the corresponding activity shown in Table 2, Concentrations in the flesh of fish and the corresponding catalase activity shown in Table 3.

Discussion

The Tables 1 and 3 show results from the study. There was a higher accumulation for high concentrations of nickel introduced and higher catalase activity as the concentration of nickel introduced increased and accumulated. A regular trend was generally observed in the mortality rate which increases with increased concentration. At the early stage (i.e., the first 24 hr) of the toxicants introduction, all the fishes survive initial attack. This may be due to their protective adaptations and the hardy nature of Clarias gariepinus. During the second renewal (48 hr exposure) some damages or injuries were noticeable particularly amongst some fishes in the highest concentration (3 mg and 4 mg). With progressive exposure of 72 hr, deaths becomes inevitable even at lower concentrations. This could be due to stress and cumulative impact of nickel toxicity. Apart from least concentration (1 mg), death, though at different rates, were recorded at every other concentration. Factors contributing to nickel toxicity to aquatic life include the species, pH, water hardness and other environmental factors.

Table 2: Concentrations of nickel in introduced and the corresponding activity.

Conc. Introduced ($mgkg^{-1}$)	Cat. activity ($\mu molmin^{-1}$)
Control	0.000
1	6.000
2	6.900
3	9.500
4	10.600

Table 3: Concentrations in the flesh of fish and the corresponding catalase activity.

Conc. Accumulated (mkg^{-1}) M+SD	Cat. activity ($\mu molmin^{-1}$)
0.000 ± 0.000	6
0.061 ± 0.000	6.9
0.342 ± 0.395	9.5
0.43 ± 0.498	10.6

Physiological functions are generally are affected by contaminant exposures, with fish exhibiting adaptive responses on the first contact with environmental pollutants. Physiological adaptation such as spine curling and longitudinal movement of the fish was observed. This may be due to loss of static and homeostatic balance at high concentrations of nickel which made the fish wriggle and lead to its ultimate mortality. Thus, swimming performance is considered one of the measures which could serve as possible sensitive indicator of sub-lethal toxic exposure. This kind of behavioural abnormality has been reported in various fish species on exposure to metals behavioural changes usually occur much earlier than mortality. Blockage of nervous transmission between the nervous system and various effectors sites may lead to nervous impairment, enzyme dysfunction may cause paralysis and depression of respiratory centre, and alteration of pathway which yields energy results in energy depletion all lead to physiological adaptations exhibited by fish exposed to heavy metals like nickel. However, in this result a short-term exposure to high levels of nickel induced stress reaction in fish. The minor changes at lower concentration of toxicants in fish behaviour implied a transient stress which resulted in osmotic imbalance. However, greater changes observed showed that stress could reduce the immune potential of fish. This reduced immunological status which persisted resulted in higher mortality especially at higher concentrations. Thus, it seems that even an incidental toxic stress may result in a considerable increase in susceptibility of fish to infections.

Conclusion

In conclusion, bioaccumulation is not a valid criterion for judging the ecotoxicity of nickel substances because nickel is an essential element for many organisms and these organisms would suffer if they did not have the ability to accumulate and utilize nickel. Hence, good knowledge of fish response to various stressors will be of greater help in improving production of fish and in providing information on ways of effectively controlling and monitoring stress in aquaculture.

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