

Plasma Aspartate Aminotransferase Activity in the Catfish *Clarias albopunctatus* Exposed to Sublethal Zinc and Mercury

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Received: 3 January 1999/Accepted: 12 July 1999

Aspartate aminotransferase (ASAT), like alanine aminotransferase, is a key enzyme in the protein to carbohydrate metabolism. It has wide distribution in both mammalian and fish tissues (Eze 1983; Gaudet et al., 1975). The enzyme may leak into the plasma following reservoir tissue damage or dysfunction. Hence, the assay has become an indispensable tool in the clinical determination of the pathological conditions of the reservoir tissues and organs (LaDue et al. 1954).

The aquatic ecosystem remained the major recipient of industrial wastes like metallic irons, industrial solvents and contaminated run-off from the land without regard to the health of the aquatic ecosystem. Today, as result of increasing urbanization, expanding industrial and mining activities as well as modern agricultural enterprises, heavy metals in aquatic ecosystem are found in excess of natural load.

Sometimes, death of the aquatic living resources such as fish may not occur but they suffer physiological perturbations like altered enzyme activity due mainly to high load of these environmental anthropogenic agents (Racicoot et al. 1975). However, massive mortality of fish and other living aquatic resources do occur with its attendants public health problems. With increasing awareness of the impact of these pollutants, especially in developing economies, it is essential that sensitive biochemical and/or physiological parameters which could act as early warning signals of pollution at sublethal levels be studied. Hence, changes in the activity of enzymes (Kristofferson et al., 1974) or other biomarkers (Moore and Simpson, 1992) have been studied as possible tools for aquatic toxicological research. This study was undertaken with a view to using changes in the activity of plasma aspartate aminotransferase in the fish, *C. albopunctatus*, as a low cost biodiagnostic tool in aquatic ichthyotoxicological work.

MATERIALS AND METHODS

One hundred and five fish (mean weight 28.72 ± 2.90 g) used in the study were acclimatized for 14 days in the laboratory. The fish was randomly distributed into seven treatment groups (T_1 - T_7). Each treatment group was further subdivided into three replicate experiments of five fish per replicate. Treatments T_1 , T_2 , and T_3 were exposed to 50, 100 and 150 µg Zn L^{-1} (as Zinc sulphate), respectively. Treatment T_4 , T_5 and T_6 were exposed to 50, 100 and 150 µgHg L^{-1} (as mercuric chloride), respectively. The seventh treatment (T_7 was exposed to deionized tap water only as the control.

The fish were fed once daily at 3% body weight at 180°hr for the 21 days the experiment lasted. The static bioassay renewal method in which both the deionized tap water and metallic ions were changed completely every two days was used.

At the end of the experiment, the fish were anaesthetized in 100 ppm tricaine methane sulphonate (MS222). The blood was collected by the caudal puncture method. Only the unhaemolysed blood was used for the enzyme assay. The plasma aspartate aminotransferase activity was calorimetrically determined by the method of Reitman and Frankel(1957) as described by Bergmeyer and Brent (1975). The data from the replicate experiments were pooled and analyzed using analysis of variance and the least-square regression methods.

RESULTS AND DISCUSSION

The dose selectin in this study was informed by the reported levels of the metals in the surface sediments in Nigeria and West African subregion as well as on the Federal Environmental Protection Agency (FEPA) standards. The concentration of zinc in the surface sediments in some areas of Lagos Lagoon was 466.1 and 445.9 µgg⁻¹ (Okoye et al., 1991). In the Niger Delta (Nigeria) and River Wiwi (Ghana) the amount of mercury in the surface sediments were 0.33 and 0.21 µgl⁻¹, respectively (Kakulu and Osibanjo 1988; Biney and Beeko, 1991). The statutory allowed limits in the Nigerian water for zinc and mercury are 3.0 and 0.05 mgl⁻¹ respectively (FEPA, 1991).

Our data showed that plasma aspartate aminotransferase (ASAT) activity was elevated in *Clarias albopunctatus*, when exposed to both zinc and

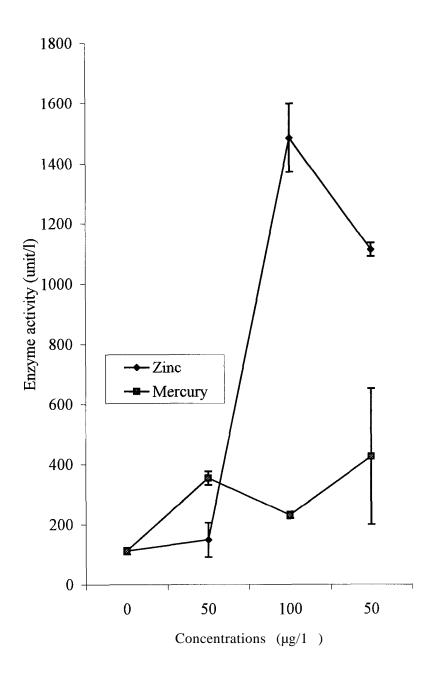


Figure 1: Effects of sublethal concentrations of zinc and mercury on the plasma aspartate amino transferase activity in Clarias albopunctatus (mean \pm 80SD) .

mercury (Fig 1). The ASAT activity was 149.0 ± 0.71 and $1485.0\pm1.41UL^{-1}$ In the fish exposed to 50, $100\mu gZnl^{-1}$, respectively. At $150~\mu gZnl^{-1}$ the enzyme activity was $1113.0\pm028~UL^{-1}$.

The enzyme activity in the different groups was significantly different (P < 0.05).

The mean enzyme activity in the control group was $112.5\pm0.14~UL^{-1}$. There was a significant positive correlation between the enzyme activity and test zinc concentrations (r = 0.8099). Compared with the control (112.5 μL^{-1}), the ASAT activity increased by 1.36, 13.26 and 9.94 folds in the fish exposed to 50, 100 and 150 μg Zn L^{-2} , respectively. The enzyme activity in the control differed (P<0.05) from those in the treatment groups exposed to zinc.

When the fish was exposed to 50, 100 and 150 μ g Hg L⁻¹, the enzyme activities were 354.0±0.28, 231.5±0.14, and 425.0±2.83UL⁻¹, respectively. Like in the groups exposed to zinc, the ASAT activity differed (P < 0.05) in the treatment groups exposed to mercury. There was also a significant positive correlation between the enzyme activity and the test mercury concentrations (r = 0.7639). Compared with the control, the ASAT activity was increased by 3.16, 2.06 and 3.80 folds when the fish was exposed to the 50, 100, and 150 μ g Hg L⁻¹, respectively. The mean enzyme activity in the control group was generally lower than those in the treatment groups exposed to either zinc or mercury. The mean ASAT activity was generally higher in the groups exposed to zinc.

The observed increase in the serum aspartate aminotransferase activity in the fish exposed to both zinc and mercury agreed with earlier reports on the effects of chemical irritants, industrial effluents and heavy metals on serum enzymes of fish. In the report of Michael et al. (1987) serum transaminase activity was elavated when *Clarias lazera* was exposed to nitrite. Similarly, carbon tetrachloride (Dimman and Berstein, 1968; Racicoot et al., 1975) methyl parathion (Rao and Rao, 1984) and copper (Oluah, 1998). Zinc and mercury (Hilmy et al., 1981; Oluah and Amalu, 1998) have been reported to cause increase in the serum transaminase activity in fish and mammals.

The activity of serum aminotransferases and other enzymes, particularly lactate dehydrogenase are good correlates of the health of the reservoir organs (LaDue et al., 1954). Since there exist kinetic equilibrium between

plasma and tissue levels of aspartate aminotransferase and other enzymes, any increase in the plasma levels of these enzymes may likely be due to imbalances int he physiology and/or anatomy of the reservoir tissues. Thus, the observed increase in the ASAT activity in C. albopunctatus exposed to these metals may be due to organ damage and/or malfunctin following exposure to zinc and mercury. This appears a plausible explanation on account of the liver and kidney damage (reservoir tissues) observed when the fish was exposed to similar concentrations of zinc and mercury (Chiedo, 1994). This is also consistent with the report of Hilmy et al. (1981) that elavated serum aspartate aminotransferase in C. lazera exposed to mercury would be the result of damage to liver parenchyma. The observed increased in the plasma ASAT activity in the fish could be a manifestation of the general adaptive response in animals. Thus, plasma ASAT, like plasma alanine aminotransferase aminotransferase elarlier implicated (Nomivama et al., 1973; Hilmy et al., 1981; Oluah and Amalu 1998) could be used as biomarkers of metal contamination.

Other enzymes that have been implicated as biomarkers of hevy metal pollution include Cu- and Zn-superoxide dismutases (Picket and Lu, 1989) and alkaline phosphatase (Hilmy et al., 1981). The results of this study suggest that the routine assay of serum aspartate aminotransferase activity in fish promises to be a good tool in ichthyo-toxicological research, aquatic impact assessment, diagnosis of onset of pollution or even the assessment of remediation actions in the aquatic ecosystem.

ACKNOWLEDGEMENT I thank Dr. H. M. G. Ezenwaji for his critical

review of the manuscript. My sincere gratitude also goes to Mr. S. Ali, of Department of Zoology, for helping in the routine changing of the water in the aquaria.

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