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# Sodium Cyanide Caused Biochemical Modulations in Liver of Fish Catla Catla

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***Abstract:*** *The primary aim of this study is to evaluate the pernicious effects of sodium cyanide (NaCN) on some biochemical aspects in the liver of fish Catla catla. Fish which were exposed to a sublethal concentration (0.2 mg/L) of sodium cyanide, for a period of 15 and 30 days and further allowed to undergo a recovery of 30 days, resulted in variation of levels of protein, free amino acid and protease enzyme activity with respect to control. The changes were in a manner that suggested the possible existence an oscillatory phase in biochemical modulations towards a more synthetic phase leading to the establishment of recuperation and adaptation phenomena. As, sodium cyanide is being a toxic component needs attention towards its neutralization prior to its disposal. Hence, it is inferred that necessary wariness should be taken during its discarding, as it possesses a serious threat to fish.*

***Keywords:*** *Catla catla, Sodium cyanide, Biochemical Indices, Adaptation phenomena, Proteolytic activity.*

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## I. Introduction

Cyanide is known to be one of the most toxic pollutants around the globe which is used for various industrial processes [1]. It is used in the production of plastic, rubber and several chemicals, also in jewellery, metal plating, leather processing, fertilizer production, pesticide and insecticide production, and in gold and silver mining premises. About 2-3 million tons of cyanide is produced annually in industry and some areas of the cyanide leaks into water and its concentration in natural water might be in such a wide range as 0.01-10.0 mg/L. Leakage of cyanide into waste water affects first the aquatic species and subsequently affects human health considerably through food chain. Fish and other aquatic organisms are especially susceptible to cyanide exposure [2].

It is an extremely destructive chemical which can kill variety of organisms irrespective of being targeted or non-targeted. In addition, it is also known to be a suicidal as well as a chemical warfare agent [3]. Cyanide and cyanogenic compounds commonly exist in the environment and are known to be highly toxic to the aquatic organisms, mainly due to the formation of metal complexes. Cyanide is ubiquitous in the environment; irrespective of its origin, only free cyanide is considered to be a biologically meaningful expression of cyanide toxicity and is the primary toxic agent. The cyanide metal complex is known to occur with Fe3+ ion, which in turn inhibits respiration and thereby restricting phosphorylation process [4]. Cyanide is known to contaminate aquatic environment that threatens the survival of aquatic organisms [5].

Many studies showed that freshwater fishes are the most cyanide-sensitive. Free cyanide at concentration of >5g/l can cause negative impact on the swimming and reproduction of fish. While, at concentration of >20g/l, the cyanide induces high fish mortality [6, 7]. Cyanide existsin water in the form of free state ($CN^-$ and HCN), simple cyanide (e.g., NaCN), complex cyanide and total cyanide. Almost all cyanide persists in the undissociated form (HCN) in pH below 7. But at a pH value of 11, all of the cyanide appears as the free (CN) ion [8]. Toxicity of cyanide is primarily determined by the concentration of undissociated HCN in the water column (even in case of simple cyanides and metal cyanide complexes). Hydrocyanic acid (HCN) is also more toxic than the free cyanide and the degree of its toxicity depends on the solubility and the dissociation rate to form free cyanide[9].

The toxicity of cyanide can be influenced by a variety of factors including concentration of cyanide, environmental temperature, dissolved oxygen content, pre-exposure and age on fishes [6]. Its high toxicity of its high potency caused a respiratory poison in all aerobic forms of life [7, 8]. Acute doses of cyanide caused fatal changes, due to the glaring susceptibility of the nerve cells of the respiratory centre to hypoxia [9]. Chronic cyanide intoxication has been incriminated in numerous syndromes, such as tropical ataxic neuropathy [10] and goitre [11].

The toxicity of cyanide is attributed to the presence of HCN derived from dissociation of the complexes that penetrate cell walls [12] and cause fish mortality [13]. When the fish is subjected to direct contact with sodium cyanide, it shows some behavioural changes during its movement [14, 15]. Such behaviours can be used as biological indicators which provide a unique perspective linking the physiology and ecology of an organism and its environment [16]. Few studies have been done to evaluate the effect of cyanide on behaviour of fish. One of those studies, showed that sodium cyanide has tremendous effect on the behaviour of *Clarias gariepinus* resulting from depression of the central nervous system which may be attributed to the combination of lactate acidosis with cytotoxic hypoxia [17].

A number of studies have resolved the toxicity of cyanide that exposure have led to altered levels in the enzyme activities of liver [18-20]. Industries dealing with metal plating and finishing, mining and extraction of metals such as gold and silver, production of synthetic fibres and the processing of coal produce large aggregation of cyanide containing wastes [21]. Sodium cyanide toxicity by referring to histo-architechtural changes in fish have also been reported by various authors [22].

Some other studies have been carried out to evaluate the effect of the sodium cyanide on the activity of adenosine triphosphate (ATPase) enzyme. This complex enzyme plays a central role in the physiological process as energy transducers by coupling chemical reaction [23, 24]. ATPases require Na+/K+, Mg2+ and Ca2+ ions for their activity and cleavage of ATP to ADP/AMP and inorganic phosphate with liberation of energy [25, 26]. The studies revealed that the reduction of ATPase may cause disturbances in cellular metabolism, leading to histotoxic hypoxia in fish. Concerning the effect of cyanide, free cyanide ions can pass through the gill membranes but it can act as a metabolic inhibitor that prevents re-synthesis of adenosine triphosphate (ATP) in the axon. So, cyanide is expected to reduce the efflux of ions to a very low value [27]. Therefore, the enzymatic activities can be used as physiological indicators to detect damages within different organs related to water contamination [28].

The concentration and rate of transformation of cyanide in tissues is dependent on the initial concentration in the sample, storage time and the preservation method (e.g., addition of sodium fluoride to sample), storage condition (e.g., temperature) of the sample, types of cyanide compound and pH. There are no reports of cyanide biomagnifications or cycling in living organisms, probably owing to its rapid detoxification [29]. So that the opinions were divided between those that believe that cyanide is quickly metabolized and excreted in a matter of hours [30] and those that believe that cyanide is retained for longer time periods [31].

As the fresh water fishes are the most sensitive to cyanide [32], an attempt has been made in the present study to evaluate the impact of sub lethal concentration of sodium cyanide on protein levels, free amino acids and protease enzyme activity in liver of fish, *Catla catla.*

## II. Materials And Methods

**Collection of Test animal and maintenance of fish**

Healthy fresh water fish *Catla catla* were procured from the A.P. State Fisheries breeding Centre, Kalyani dam, near Tirupati, India and were acclimatized to laboratory conditions for two weeks at room temperature 26±2°C. Further they were held in dechlorinated tap water in large cement aquaria which was previously washed with potassium permanganate to free the walls from any microbial growth. Fish were fed regularly and 12h : 12h light and period was maintained daily during acclimation period. At least $3/4^{th}$ water was exchanged regularly, and the water quality parameters are checked and maintained at a constant level. Physico-chemical characteristics of water were analyzed following standard methods mentioned as mentioned in APHA [33] and were found as follows: temperature 26 ± 2 °C; pH 7.5± 0.2; dissolved oxygen 7.6 ± 0.6 mg/L; total hardness 28.6 ± 2.1 mg as CaCO3/L; salinity 0; specific gravity 1.002; conductivity less than 16 µS/cm; calcium 16.47 ± 0.82 mg/L; phosphate 0.3 ± 0.004 µg/L and magnesium, 0.9 ± 0.4 mg/L.

**Experimental Fish**

The fish, *Catla catla*, were grouped into four different groups namely: **Group** 1 (control, without adding sodium cyanide), **Group 2** (15 day exposure), **Group 3** (30 day exposure) and **Group 4** (30 day recovery period). Each group was maintained in triplicate and consisted of 20 individual fishes each.

**Preparation of stock solution and exposed to fishes to chemical**

The experimental chemical Sodium cyanide of 95% purity was procured from the Loba Chemie Pvt. Ltd., Mumbai, India. Stock solution was prepared by dissolving sodium cyanide in glass distilled water in a standard volumetric flask. Water at least $3/4^{th}$ was every day was exchanged over test periods. The fish were exposed in batches of 20 to a fixed sub lethal concentration of sodium cyanide with 20 L of water in three replicates for each concentration. $1/5^{th}$ of the LC50 (LC50 = 2mg/L) calculated as 0.2mg/L was selected as sub lethal concentration for further studies and the duration of exposure were 15 and 30 days. Further, the

experimental fish were allowed to undergo a recovery period of 30 days. At the end of 15 and 30 days and recovery of 30 days, experimental fish were sacrificed used for further biochemical studies.

**Biochemical studies**

**Estimation of soluble, structural and total proteins:** The soluble, structural and total proteins in liver were estimated using the Folin-phenol reagent method as described by Lowry et al., [34].

**Estimation of Free amino acid:** Free amino acid level in the liver was estimated by the ninhydrin method as described by Moore and Stein [35]. The free amino acid levels are expressed as mg amino acid nitrogen released/g wet wt. of the liver.

**Estimation of protease activity:** Protease activity in the liver was estimated using the ninhydrin method as described by Davis and Smith [36]. The protease activity is expressed as µ moles amino acid nitrogen released/mg protein/h.

## III. Results And Discussion

In the present study, an attempt was made to examine the toxic effects of sodium cyanide on some biochemical aspects in liver of fish *Catla catla* under sub lethal exposure. Significant changes were observed in the total protein levels of the all groups of exposed fish. Tables 1 & 2 represents the depleted protein levels and elevation of protease enzyme activity and that of free amino acids levels in the liver of the fish exposed to 0.2mg/L of sodium cyanide ($1/5^{th}$ LC50). The total, structural and soluble protein levels were found to decrease until 10th day in fish exposed to sodium cyanide. The maximum decrease of total protein content was recorded in Group 2, that suggests the direct tissue damage caused by sodium cyanide to the fish. However, in Group 3, a gradual increase in protein content and decrease in free amino acid and protease enzyme activity was also recorded. Similar results were also observed in Group 4 fish, indicating the ability to overcome the stress induced by sodium cyanide. However, complete recovery of animal is not possible. Similar levels of free amino acids and the activity of protease enzyme, the same were found to increase with increase in the duration of exposure until $15^{th}$ day. It is indicated that the fish were able to overcome the stress by $30^{th}$ day which was indicated by restoration of the levels of free amino acid and protease enzyme which tend to move towards normalcy (Table 2). Further, no changes are identified for any of the above aspects in the liver of control fish. To have a more precise understanding of the variation among soluble and structural proteins, the ratio of Soluble to Total (Sop/Tp), Structural to total (Stp/Tp) and soluble to structural (Sop/Stp) were calculated. The Stp/Tp values are correspondingly are higher over Sop/Tp and Sop/Stp values. These ratios are clearly indicated that recycling of soluble proteins was substantially higher compared with structural proteins.

Protein synthesis is one of the most important processes responsible for maintaining the physiological balance in the body of animals. Proteins are indeed of primary and paramount importance not only because of their peculiars but also because of the fact that they appear to confer their biological specificity among various type of cells [37]. The degradation of protein levels and increased proteolytic activity suggests the possibilities of correlation and probable utilization of their products for metabolic process thereby causing damage to tissues, this finding is supported by Mastan and Ramayya [38] who also reported similar kind of changes. From the previous studies it evident that aquatic environment is continuously being contaminated with toxic chemicals from industrial, agricultural and domestic activities [39-45] among which, sodium cyanide is also considered as potentially toxic substance. These results for protein levels obtained from the present investigation are in agreement with the earlier reports of Khalid Abdullah Al-Ghanim [46] who reported similar findings in *C. Carpio* exposed to cypermethrin. Kumar and Gopal [47] reported that the liver protein in fishes was depleted, exposed to different toxicants of indusatrial effluent, indicates the conversion of tissue protein into soluble portion is set off due to the toxicant stress, depending on which the soluble portion may be channelized to blood stream for use. The increased energy demand during stress is compensated by catabolising of the proteins which may be in turn triggered by enzymatic activities [48]. In long term exposure to sodium cyanide much of the energy must have been used up to compensate the stress, hence the depletion in the protein content is observed. Few of the earlier reports reported that an alteration of protein metabolism were observed in fish exposed to various toxicants including metals, industrial effluents and pesticides, [49]. In the studies also, similar trend is observed, it certainly supports the outcome of the present study.

Durga and Veeriah [50] observed the changes in protein metabolism due to effect of toxic stress induced by cypermethrin on the fish *L. rohita* that manifested the decline in total protein level and increase of free amino acid level, this supports the present investigation. The increased free amino acid level suggests tissue damage probably due to the increased proteolysis activity due to sodium cyanide toxicity. The decrease of protein levels might be attributed to the increased energy demands, which is caused by sub lethal concentration of sodium cyanide. The increased amount of free amino acid might also be correlated with the increased proteolytic activity which was the responsible factor that caused the breakdown of protein molecule to yield free amino acid subunits [51].

On the other hand, the elevated levels of free amino acids can be utilized for energy production by feeding them in to the TCA cycle through aminotransferase reactions supporting the view of Imtiyaz et al. [51]. The quality of protein is dependent on the frequency of protein synthesis, or on frequency of its humiliation. The quality of the protein might also be affected due to impaired incorporation of amino acids into polypeptide chain [52] suggested that the fish exposed to a toxicant may recompense any viable protein loss by increasing its protein synthesis. David et al. [53] summarized that compensatory production of enzymes lost as result of tissue necrosis or to meet increased demand to detoxify the toxic compound might have necessitated enhanced synthesis of enzyme proteins. Increased free amino acid levels are resulting the breakdown of protein for energy and impaired incorporation of amino acids in protein synthesis. The sub lethal concentration of lead nitrate induced hormonal imbalance is evident, because it could be direct or indirect affect on the tissue protein levels [54].

It can be concluded that the decreased trend of protein content in liver of *Catla catla* in the present study might be due to metabolic utilization of keto acids in the synthesis of glucose or for the osmotic and ionic regulation as reported by Janardana Reddy et al. (55), Venktramana et al., [56], Muley [57], Janardana Reddy[58] and Suneetha [59]. Tilak et al., [60] explained the reduction in levels of protein content of liver of *C. punctatus* due to toxic stress exerted by fenvalerate. A decrease in total, structural and soluble proteins and an increase in free amino acid and protease activity levels in contrast to protein decrement were observed in 7 and 15 days of exposure of malathion, but on 30 days of exposure all the values got nearer to normalcy [61] and these reports are also in agreement with the present study.

The result of the present study showed that when the fish were exposed to malathion, the protein content were found to have decreased. The present decrease was found to be higher in all exposure periods in liver tissue. The reduction of protein may be due to proteolysis and increased metabolism under toxicant stress [62] and these reports are also in concurrence with the present study.

## IV. Conclusion

The experimental study is inferred that sodium cyanide is toxic to the freshwater fish *Catla catla*, when the fish is subjected to at least sub-lethal concentration and at a low concentration of 0.2mg/L ($1/5^{th}$ LC50), as it can sabotage the levels of biochemical content in the liver of the fish. It is further suggested that adequate care be taken to detoxify sodium cyanide before it is discharged into water bodies as it seriously threatens the survival of the present experimental model. More studies should be focused on the cyanide metabolism of fish. Increasing the awareness of fishermen about the dangers of using sodium cyanide for fishing that have a serious hazardous impact on the fishery and fish production as well to the human beings.

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**Table 1: Variations in Protein levels in the liver of the fish, *Catla catla* exposed to sub-lethal concentration of Sodium cyanide.**

| Parameter | Control | Exposed | | Recovery |
|---|---|---|---|---|
| | | 15 days | 30 days | 30 days |
| Soluble Protein (SoP) | 24.52 ±1.16 | 21.66 ± 1.26 | 19.28 ±1.25 | 22.46 ±1.59 |
| %change | ---- | - 11.66 | - 21.37 | -8.40 |
| Structural Protein (StP) | 45.79 ±2.15 | 41.35 ±2.57 | 40.66 ±1.85 | 42.65 ±1.25 |
| %change | ---- | -9.69 | -11.20 | -6.86 |
| Total Protein (TP) | 68.61 ±1.16 | 62.35 ±1.08 | 60.95 ±1.09 | 64.34 ±1.29 |
| %change | ---- | -9.12 | - 11.16 | - 6.22 |
| SoP/TP | 0.328 | 0.346 | 0.378 | 0.395 |
| %change | ---- | 5.48 | 15.24 | 20.42 |
| StP/TP | 0.728 | 0.716 | 0.694 | 0.682 |
| %change | ---- | -1.64 | -4.67 | -6.31 |
| SoP/StP | 0.481 | 0.516 | 0.528 | 0.549 |
| %change | ---- | 7.27 | 9.77 | 14.13 |

Means ± SD of 6 individual observations.
Values are significant at $\leq 0.05$level from each other.

**Table 2: Protease activity (µM of amino acids/mg protein/h) and free amino acid (mg/g wet wt.) in the liver of the fish, *Catla catla* following exposure to sublethal concentration (0.2mg/L, 1/5th LC50) of Sodium cyanide.**

| Parameter | Control | Exposed | | Recovery |
|---|---|---|---|---|
| | | 15 days | 30 days | 30 days |
| Protease activity | 4.58 ±0.16 | 4.69 ± 0.32 | 4.82 ±0.25 | 4.62 ±0.52 |
| %change | ---- | 2.40 | 5.24 | 0.87 |
| Free Amino acid levels (FAA) | 4.79 ±2.15 | 4.96 ±2.57 | 5.36 ±1.85 11.89 | 5.15 ±1.25 7.51 |
| %change | ---- | 3.54 | | |

Means ± SD of 6 individual observations.
Values are significant at $\leq 0.05$level from each other.

S. Janardana Reddy. "Sodium Cyanide Caused Biochemical Modulations In Liver Of Fish Catla Catla." IOSR Journal of Pharmacy and Biological Sciences (IOSR-JPBS) 13.6 (2018): 49-54.