IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Eco-Physiological Changes Induced By Solid Waste Extract Of A Chlor-Alkali Industry On A Fresh Water Fish Under Controlled Conditions.

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Abstract

Lechate collected from the dumping site and contaminated area at different time periods indicated significant difference in chemical composition and mercury concentration. Hence, it was planned to prepare the extract by taking the solid waste from the dumping site and preparing the extract in the laboratory, now known as solid waste extract (SWE) for the experimental purpose. The solid waste extract contained 9.75 mg of Hg l^{-1}. This prepared solid waste extract was considered equivalent to the leached chemicals leaching in rainy season and entering into neighboring ponds and crop fields and water bodies like Rushikulya River and estuary. The ATPase activity in SWE exposed fish brain, liver and muscle tissues showed 41.03%, 63.9%, and 49.6% decrease over respective control values, where the SWE exposed fish liver was more damaged than other tissues tested. Significant depression in ATPase enzyme activity may severely affect the ion transport mechanism of the exposed fish body when compared to control fish body. The exposed fishes were when transferred to SWE free medium the enzyme activity showed no recovery at all rather further depletion was noted. During recovery period the exposed brain tissue was drastically affected, might be due to mobilization and transport of more amount of residual mercury from the body tissues to the brain. The changes observed in exposed liver and muscle tissues were interesting and the observed changes were not statistically significant. Significant differences in ion content of different tissues of the exposed fish when compared to control fish clearly indicated the nature of the toxicant and effects caused by the SWE toxicant on the nervous tissue, synaptic transmission, nerve impulse generation, ionic balance and membrane transport system of the exposed fish. The exposed fishes did not recover when transferred to SWE free medium. The enzyme activity showed no recovery in liver, brain and muscle tissues but little variation during recovery was not statistically significant. Exposed liver tissues showed the highest damage when compared to brain and muscle tissues of the exposed fish, when compared to control fish tissues.

Keywords: Chlor-alkali industry, Solid waste, SWExtract, fish, *Tilapia*, ATPase**.**

Introduction

Tilapia fish is a mouth breeder and breeds 2-3 times a year, handy to handle in laboratory conditions, better survival rate in laboratory conditions and bimodal feeding habit and gaseous exchange make the fish as an excellent material for toxicity testing and to study the changes occurring in physiological parameters. The entire idea of selecting solid waste and fish for the present study came to our mind from local complaints lodged by the villagers. The report that change of fish colour from white to black and death of number of fishes both in ponds and the river prompted us to think of poisoning due to leaching of chemicals from solid waste particularly in rainy season. Series of regular field assessments were conducted monthly and seasonally to assess the potentiality of the situation and changes in few physico-chemical parameters and most important to assess the mercury load in different stations of the contaminated area both upstream and down stream of the river. In field experiments, estuarine fishes were collected from the Rushikulya River, Rushikulya estuary to study bioaccumulation of mercury in whole fish and in different organs of the fish. No information was available on the direct effect of the solid waste / effluent of the industry on freshwater fishes. The solid waste and effluents of the industry directly enter along with rain runoff water and by

leaching enter into neighboring ponds and contaminated the ponds, where this *Tilapia* fish was mass cultured. Hence, this project was designed for instant information on the issue. The present piece of work was planned first to analyze the solid waste and effluent of the industry, movement of mercury in the course of effluent movement, junction point where effluent joins the Rushikulya River, Rushikulya estuary; residual mercury in the solid waste dumps and residual mercury in fishes collected from the contaminated estuary and Rushikulya River near the industrial site at Ganjam, Odisha. It was observed that solid waste leachates from solid waste dumps are transported to the River Rushikulya and the lechates also enter into the surrounding crop fields and ponds. It was also observed that the fish populations inhabiting the nearby ponds suffer from pollution stress. A pilot experimental study in the laboratory for toxicity testing of the SWE (solid waste extract) of the chlor-alkali industry on a fresh water fish was under taken to understand the impact of mercury contained solid waste extract on fishes and secondly, to study the effect of the leached chemicals of the solid waste known as solid waste extract (SWE) of a chlor-alkali industry on a fresh water fish, *Tilapia mossambica*, Peters and its possible eco-toxicological significance.

Materials and methods:

Location of the industry: The Chlor-alkali industry M/S Jayashree Chemicals Pvt. Ltd. is situated on the side of National Highway-16 at Ganjam. The industry is located very close to Ganjam Township; district Ganjam, Odisha state, India. The industry is located on the Bank of River Rushikulya discharging its solid waste into the river directly (initially) and dumped near Rushikulya river. The leached waste enter into the river. The industry is located at at 84^0 53'E longitude and 19^0 16'N latitude.

Test fish:

Oreochromis mossambicus, Peters [***Sarotherodon mossambica***, Peters or ***Tilapia mossambica***, Peters; (popularly known as ***Tilapia*** fish)] was collected, acclimatized in the laboratory.

(Photograph of *Tilapia mossambica*, Peters, Lateral view)

Maintenance of fish in the aquarium:

Tilapia fish was collected from a local nursery of Government Fishery Department, Berhampur. Fishes were allowed to acclimatize for 15days with normal dietary supplement in aquarium. Air was bubbled to maintain 85% air saturation value. The water quality was monitored on regular basis. Chlorine free water was used in the aquarium. The food habit was slowly changes from earthworm pieces to goat liver and then to sliced boiled eggs (only white portion) during holding and experimental period. To prevent infection before any experiment, dilute $KMnO_4$ (1%) washing of fishes was carried out.

Table- Water quality of control and solid waste extract (SWE) exposed aquarium during holding and experiments in the laboratory controlled conditions. (Transparency was measured in terms of optical density (OD) at 540 nm taking double glass distilled water as standard).

Water Quality	Control	Exposed (SWE)
Colour	Transparent	Grayish
pH	7.1 ± 0.3	7.8 ± 0.4
Temperature, ^oC	26 ± 2	26 ± 2
Illumination, Lux	2200 ± 200	2200 ± 200
Total hardness, mg l^{-1}	76.5 ± 3.4	115.1 ± 8.5
Specific conductivity,	2.54 x 100µmho	4.52 x 100µmho
Transparency, at 540nm	0.01 - 0.025 (OD)	0.07 - 0.105 (OD)
Hg in the medium, mg l^{-1}	00	0.195±0.024

The mortality rate of test-fish was studied following the method described by Panigrahi (1980). Graded series of concentrations (micro range, middle range and mega range) of solid waste extract (SWE) was prepared in small 2liter rectangular jars. Ten healthy acclimatized *Tilapia* fishes were allowed to live in those jars. Over crowding of fishes were avoided. The control and exposed fishes were fed daily and timely.

Observation on the toxicity of the industrial effluent was made at 24, 48, 72, 96 hours and 28 days after the experimental animals were first exposed to the solid waste extract prepared in the laboratory. Individuals showing no respiratory movements, no opercular movements and no response to a tactile stimulus were recorded as dead, and were immediately removed. The test fishes were exposed to maximum allowable concentration (MAC) of the solid waste extract, where no death was noticed during experimentation and the value was presented in percentage (%).

Experimental study of SWE:

The physico-chemical analysis of solid waste extract was carried out following the methods as described in Analytical Methods manual (APHA, 1995). The sediment and solid waste collected from the site were air dried and powdered in a grinder. Dried solid waste sample was weighed in a top pan balance and digested in a Bethge's apparatus (Wanntorp and Dyfverman, 1955). One kg of solid waste powder was taken in a mixer and 2liters of distilled water was added. The mixture was homogenized in the mixer for 15days continuously at 12hrs interval. The mixture was stirred for 1 hr and the content was allowed to rest for 1hr. The process was repeated for everyday for 15days. After 15^{th} day of stirring, the whole content was allowed to settle and the supernatant was decanted carefully. The decanted supernatant was the prepared solid waste extract (SWE) used for the experimental purpose. Effluent, SWE and sediment samples were also digested following the same technique. Mercury concentration analysis was carried out in a Mercury Analyser following the detailed procedure earlier. Total mercury level was expressed as mg of Hg l^{-1} in solid waste extract sample.

Test solution:

One kg of the air dried solid waste was taken in a jar and two liters of distilled water was added and continuously stirred for 2days followed by intermittent stirring for another 13days. Then the contents were allowed to settle and the supernatant is decanted carefully, which is now known as SWE (Solid waste extract) to a container and kept in the refrigerator for use. The experiments were conducted in chlorine-free tap water at a room temperature of 26±2^{o}C (Priyadasan and Panigrahi, 2018).

The fishes were sacrificed at 7 days interval (both control and SWE exposed). The fish was dissected and different parts like brain, liver; muscle & gill were carefully removed and separated. Brain, liver and muscle tissues were removed, kept in watch glasses, and weighed separately. The tissues were kept in ice-cold 0.25M sucrose. Known weights of the tissues were homogenized, centrifuged and the supernatant was taken for testing. Inorganic phosphate produced in the reaction was measured by the method of Fiske and Subbarow (1925) as modified by Martinek (1970). Protein was estimated by the procedure developed by Lowry *et al*. (1951), using a spectrophotometer. The enzyme activity was presented as µmoles of ip liberated mg^{-1} of protein h^{-1}. Fishes were sacrificed and dissected after 28 days of exposure. Different tissues of control and SWE exposed fishes were dissected out washed and weighed. Care was taken during autopsy and tissues were cleaned and separated. The observed data was statistically analyzed.

Results

The solid waste extract prepared in the laboratory as mentioned in the material and method chapter was considered equivalent to the lechate leaking from the solid waste dumps located nearer to the industry. The lechate leach to the peripheral area and contaminate ground water and also water bodies like small ponds. The lechate also flow directly into the River Rushikulya mostly in rainy season. In addition, during rainy season, the solid wastes were washed away into the river bed. Many a times during rainy season, the flood water carries all the dumped solid waste deposits into the sea, passing through the estuary. Hence, it was felt necessary to study the lechate of the solid waste. The detailed study indicated the presence of mercury in the lechate. Lechate collected from the dumping site and contaminated area at different time periods indicated significant difference in chemical composition and mercury concentration. Hence, it was planned to prepare the extract by taking the solid waste from the dumping site and preparing the extract in the laboratory, now known as solid waste extract (SWE) for the experimental purpose.

Table-A1: Showing the values of acute toxicity and chronic test. Acute toxicity:

Lethal concentration values	Toxicant (SWE) concentration, Dilution percentage (%)				
	Time period in hours				Days
	24h	48h	72h	96h	28days
LC_0	5.4	5.4	5.1	4.5	2.85
LC_{10}	6.2	6.1	5.9	5.6	3.11
LC_{50}	8.4	6.6	6.4	6.3	3.95
LC_{90}	9.1	8.8	8.5	8.1	4.68
LC_{100}	9.9	9.7	9.6	9.4	4.95
MAC	6.5	6.3	5.9	6.1	2.8

Chronic toxicity test:

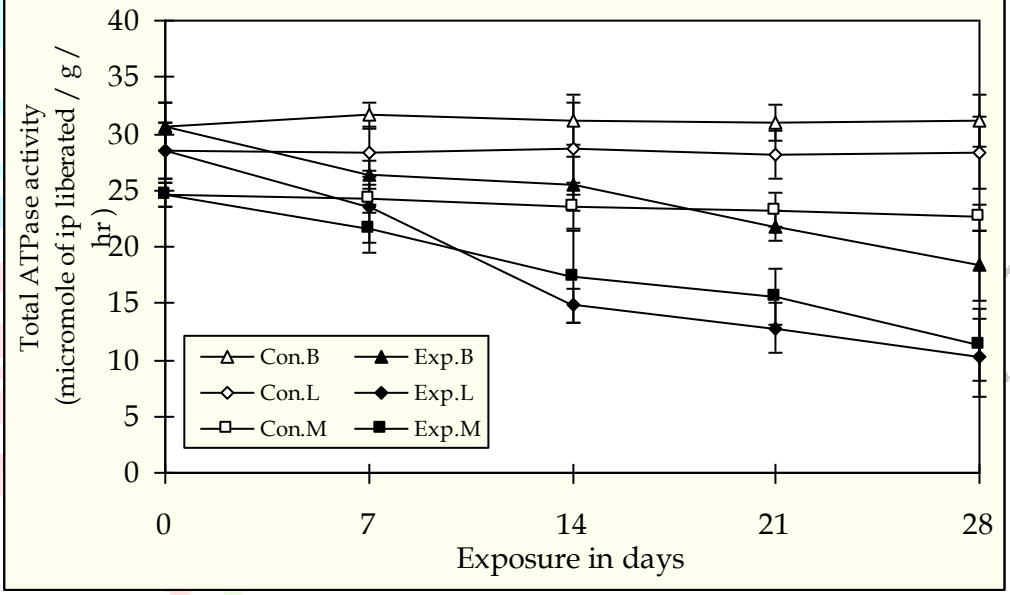
MAC value of Solid Waste Extract (SWE) as obtained from toxicity testing.	2.85% of SWE	30days
Used concentration of SWE for experiments	2.8% of SWE	28days

Observation on the toxicity of the industrial solid waste was made at 24, 48, 72, 96 hours and 28 days after the experimental animals were first exposed to the solid waste extract prepared in the laboratory. Individuals showing no respiratory movements, no opercular movements and no response to a tactile stimulus were recorded as dead, and were immediately removed from the jars and solution of the jar was immediately changed to avoid any infection and release of decomposition wastes from the dead fishes. No death of fishes was recorded in the control set jars. No of fishes died in each jar and death time period was recorded in each jar for 24, 48, 72, and 96hours. After this acute toxicity period, experimental fishes were allowed for 30days further exposure for studying impact of chronic exposure of the toxicant on the freshwater fish, *Tilapia*. Acute toxicity testing revealed the following information. After 24h of exposure the lethal concentration values were, LC_0- 5.4%SWE, LC_{10}-6.2%SWE, LC_{50}- 8.4%SWE, LC_{90}-9.1%SWE and LC_{100}-9.9%SWE; after 48hrs, After 28days of exposure in chronic poisoning, the LC values were LC_0-2.85%SWE, LC_{10}-3.11%SWE, LC_{50}- 3.95%SWE, LC_{90}-4.68%SWE and LC_{100}-4.95%SWE. The deduced MAC values were 5.6%SWE after 24h, 5.3%SWE after 48h, 5.1%SWE after 72h, 4.9%SWE after 96hours and the MAC value after 28days was 2.8% SWE

The observed changes like severe impact of SWE on the ventilation rate, whole body oxygen uptake and tissues slice respiration of SWE exposed fish when compared to control fish, it was planned to study ATPase activity of different tissues and related ion concentration studies of control and SWE exposed fish under laboratory controlled conditions. The changes in total ATPase activity in brain, liver and muscle of control and SWE exposed fish at different days of exposure and recovery and the percent changes were shown in Fig. -E-18. The ATPase enzyme activity ranged between 30.6 ± 2.1 to 31.6 ± 1.4 µmole ip liberated/mg of protein/hr during the entire period of experimentation in the control brain tissue (Fig.-E18). In the SWE exposed fish brain, the enzyme activity decreased from 31.6 ± 1.2µmole ip liberated/mg of protein/hr to 26.4±1.2µmole ip liberated /mg of protein/hr after 7days of exposure; from 31.2±2.2µmole ip liberated/mg of protein/hr to 25.5 ± 2.4µmole ip liberated/mg of protein/hr after 14days of exposure; from 30.9 ± 1.6µmole ip liberated/mg of protein/hr to 21.8 ± 1.3µmole ip liberated/mg of protein/hr after 21days of exposure and from 31.2±2.3µmole ip liberated/mg of protein/hr to 18.4 ± 3.1µmole ip liberated / mg of protein / hr after 28days of exposure (Fig. E-18). The enzyme activity in SWE exposed fish brain tissue declined by 16.5%, 18.3% 29.4% and 41.03% on 7, 14, 21 and 28 day of exposure respectively. During recovery studies, the ATPase activity in exposed fish brain instead of showing any signs of recovery further declined from 32.1±4.5µmole ip liberated/mg of protein/hr to 14.6±3.8µmole ip liberated/mg of protein/hr on 14^{th} day of recovery & from 32.2± 3.6µmole ip liberated/mg of protein/hr to 11.5±2.2µmole ip liberated/mg of protein/hr on 28^{th} day of recovery. The enzyme activity decreased by 54.5% on 14^{th} day recovery and decreased by 64.2% over the 28^{th} day exposure value, after 28 days of recovery, indicating no recovery at all (Table-E-1). The exposed fish brain enzyme activity could not recover even partly during recovery period but showed 13.5% and 23.3% further depletion on 14^{th} and 28^{th} day of recovery. No recovery in the exposed fish brain enzyme activity as marked on 14d and 28d of recovery indicated the acute toxic nature of the toxicant.

The ATPase enzyme activity ranged between 28.2±2.1 to 28.7±4.1µmole ip liberated/mg of protein/hr during the entire period of experimentation in the control liver tissue (Fig.-E18). In the SWE exposed fish liver, the enzyme activity decreased from 28.4± 2.2µmole ip liberated/mg of protein/hr to 23.6 ± 3.2µmole ip liberated/mg of protein/hr after 7days of exposure; from 28.7±4.1µmole ip liberated/mg of protein/hr to 14.8±1.5µmole ip liberated/mg of protein/hr after 14days of exposure; from 28.2±2.1µmole ip liberated/mg of protein/hr to 12.8±2.2µmole ip liberated/mg of protein/hr after 21days of exposure and from 28.3±3.2µmole ip liberated/mg of protein/hr to 10.2±3.4µmole ip liberated/mg of protein/hr after 28days of exposure (Fig.- E-18). The data indicated that the liver of the SWE exposed fish was damaged maximum when compared to SWE exposed fish muscle and brain tissues of the SWE exposed fish. The ATPase activity in exposed fish liver instead of showing any signs of recovery further declined from 28.4±3.6µmole ip liberated/mg of protein/hr to 9.9 ± 2.1µmole ip liberated/mg of protein/hr on 14th day of recovery and from 28.6±1.8µmole ip liberated /mg of protein/hr to 10.2±1.6µmole ip liberated/mg of protein/hr on 28th day of recovery. The enzyme activity decreased by 65.1% on 14th day recovery and decreased by 64.3% over the 28th day exposure value, after 28 days of recovery, indicating no recovery at all. The exposed fish liver enzyme activity could not recover even partly during recovery period but showed 1.2% and 0.37% further depletion on 14th and 28th day of recovery. No recovery in the exposed fish liver enzyme activity as marked on 14d and 28d of recovery indicated the acute toxic nature of the toxicant.

Fig.E-18: Changes in total ATPase activity in brain, liver and muscle of control and SWE exposed fish, at different days of exposure.



The ATPase enzyme activity ranged between 22.6±1.2 to 24.6±1.1µmole of ip liberated mg^{-1} of protein hr^{-1} during the entire period of experimentation in the control muscle tissue (Fig.-E18). In the SWE exposed fish muscle, the enzyme activity decreased from 24.2±1.6µmole ip liberated/mg of protein/hr to 21.6±2.2µmole ip liberated/mg of protein/hr after 7days of exposure; from 23.6±2.1µmole ip liberated/mg of protein/hr to 17.4±4.2µmole ip liberated / mg of protein/hr after 14days of exposure; from 23.2±1.5µmole ip liberated/mg of protein/hr to 15.6±2.5µmole ip liberated/mg of protein/hr after 21days of exposure and from 22.6±1.2µmole ip liberated/mg of protein/hr to 11.4±3.2µmole ip liberated/mg of protein/hr after 28days of exposure in exposed muscle tissues (Fig.- E-18). The enzyme activity in SWE exposed fish muscle tissue declined by 10.7%, 26.3%, 32.8% & 49.6% on 7, 14, 21 & 28 day of exposure respectively. The data indicated that the muscle of the SWE exposed fish was damaged more when compared to SWE exposed fish brain tissue. The exposed fish brain was least affected when compared to fish liver and muscle.

Table-E-1: Showing percent changes (decrease) in ATPase activity in exposed fish tissues when compared to respective control values. Data calculated from the mean values.

Tissues	Exposure period in days					Recovery in days	
	0	7	14	21	28	14	28
Brain	0	16.45	18.27	29.4	41.03	54.52	64.28
Liver	0	16.9	48.43	54.61	63.96	65.14	64.33
Muscle	0	10.74	26.27	32.76	49.56	48.21	53.98
Percent recovery during recovery period. Data calculated from 28d exposure value					Brain	13.47	23.25
					Liver	1.18	0.37
					Muscle	1.35	4.42

The ATPase activity in exposed fish muscle instead of showing any signs of recovery further declined from 22.4±3.4µmole ip liberated/mg of protein/hr to 11.6±2.6µmole ip liberated/mg of protein/hr on 14th day of recovery and from 22.6± 3.8µmole ip liberated/mg of protein/hr to 10.4±2.8µmole ip liberated/mg of protein/hr on 28th day of recovery. The enzyme activity decreased by 65.1% on 14th day recovery and decreased by 64.3% over the 28th day exposure value, after 28 days of recovery, indicating no recovery at all. The exposed fish muscle enzyme activity could not recover even partly during recovery period but showed 1.4% and 4.4% further depletion on 14th and 28th day of recovery. No recovery in the exposed fish muscle enzyme activity as marked on 14d and 28d of recovery indicated the acute toxic nature of the toxicant. Significant depression in ATPase enzyme activity may severely affect the ion transport mechanism of the contaminated fish body in comparison to control fish body. The correlation coefficient analysis between days of exposure and total ATPase activity in control fish brain showed a positive but non-significant correlation, whereas, the exposed fish brain showed negative correlation ($r = -0.976$, $P \leq 0.01$). The control set showed positive but insignificant correlation. The percent change in the enzyme activity in the exposed fish brain when compared to control fish brain showed a positive significant correlation ($r = 0.974$, $P \leq 0.01$) with the increase in exposure period. The analysis of variance ratio test indicated the existence significant difference between rows and non-significant difference between columns. The correlation coefficient analysis between days of exposure and the total ATPase of liver slices of the control fish did not show any significant correlation, whereas the exposed fish liver showed a negative correlation and significant ($r = -0.971$, $P \leq 0.01$) with the increase in exposure period. The percent decrease in ATPase activity showed a positive, significant correlation ($r = 0.973$, $P \leq 0.01$). The analysis of variance ratio test indicated significant difference between rows and significant difference between columns. The control muscle did not show any significant correlation between exposure period and enzyme activity. However, the muscle of the exposed fish showed negative and significant correlation ($r = -0.988$, $P \leq 0.01$). The percent change in the enzyme activity showed negative and significant correlation with the exposure period. The analysis of variance ratio test for muscle showed significant difference between rows and significant difference between columns. The exposed fishes were when transferred to SWE free medium the enzyme activity showed no recovery at all rather further depletion was noted. During recovery period the exposed brain tissue was drastically affected, might be due to mobilization and transport of more amount of residual mercury from the body tissues to the brain. The changes observed in exposed liver and muscle tissues were interesting and the observed changes were not statistically significant. Liver and muscle showed the highest virtual recovery not by further increase but by lower range depletion when compared to brain tissues. Significant differences in ion content of different tissues of the exposed fish clearly indicated the nature of the toxicant and effects caused by the SWE toxicant on the nervous tissue, synaptic transmission, nerve impulse generation, ionic balance and membrane transport system of the exposed fish. The exposed fishes did not recover when transferred to SWE free medium. The enzyme activity showed no recovery in liver, brain and muscle tissues but little variation during recovery was not statistically significant. Exposed liver tissues showed the highest damage when compared to other tissues studied in exposed fish, when compared to control fish tissues.

Discussion

Long-term exposure of animals to toxicants might cause pathological changes in addition to physiological and biochemical changes. The rapid absorption of the toxicant (industrial effluent) through the gill, skin and gastro-intestinal tract of fish was well evident in the observed exposed fish. Similar findings and trends were also reported earlier in mercury intoxication (Panigrahi, 1980). The observed depletion in metabolic activity in exposed fish indicate probable damage caused to respiratory system, and inhibition of enzymes or an important system, was totally acceptable and agree with the findings of Panigrahi (1980). Mishra (2013) and Panda *et al.*, (2017) reported the effect of red mud waste and red mud waste extract on fresh water fishes whole body oxygen uptake separately and also indicated that these wastes depress active

metabolism and the exposed fish intake of oxygen decreases significantly. Panigrahi & Misra (1978, 1980) and Panigrahi (1980) reported depletion in oxygen uptake in mercury exposed fishes under laboratory experimental conditions. Our findings agree with the findings of above authors. Residual mercury in exposed fish tissues depleted the respiration rate of tissues, which ultimately reflected on the whole body oxygen uptake and ventilation rate when compared to control fish tissues as reported by Priyadasan and Panigrahi (2018). The reports of Panigrahi and Mishra (1978a&b) agree with our findings except that we conducted the experiment by taking the solid waste extract containing mercury for our experiment where the impact was synergistic effect of all chemicals present in the extract in addition to mercury. If we compare the impact of our study with the findings of Panigrahi and Misra (1978a&b), the observed effects reported were the impact of mercury as mercuric nitrate, a single chemical on fresh water fishes. The same authors also reported residual mercury accumulation in fish tissues and depletion of tissue slice respiration in different tissues (Panigrahi, 1980) and correlated the impact to be an effect of mercury poisoning. We got similar results and agree with the findings of Panigrahi and Misra (1978a&b) and Panigrahi (1980).

Considerable information are available pertaining to residual toxicity levels in fresh water, estuarine and marine fishes but relatively very little work has been done on the mechanism of toxic action of mercurial compounds especially on studies concerning active transport across cellular membranes. Jackim (1974) reported significant depression of (Na^+, K^+) ATPase activity to be associated with higher absorption and accumulation of mercury. Na^+, K^+-ATPase is well known to play an important role in nerve impulse generation and synaptic transmission (Ahuja and Subramanyam, 1978). The same author also reported the indication of suppressed or stimulated enzyme activity was caused when organisms exposed to minor doses of metals. Studies have shown that cadmium ion has damaging effects on respiration and ATPase activity of the pulmonary alveolar microphage (Cross *et al*., 1970). Panigrahi (1980) reported depression of ATPase activity in vivo and in vitro in freshwater fishes following inorganic mercury intoxication. Panigrahi (1980) reported a similar trend in freshwater fishes exposed to mercury based fungicide. Metals can combine with enzymes in many ways among which are sulfahydryl binding, chelation and salt formation. A good number of references are available pertaining to the inhibitions of ATPase activity in fish by polychloride biphenyls (Desaiah *et al*., 1972), Toxaphene (Desaiah and Koch, 1975); DDT (Desaiah *et al*., 1975) and by Kepone and Mirex (Desaiah *et al*., 1975 and 1977). Earlier it was thought that the ATPases might be involved in the transport of ions in the nerve and interfere with a variety of membrane linked functions. This earlier thought is now a reality, where ATPase and ions play an important role in nerve impulse generation and synaptic transmission from PNS to CNS and vice versa to respond to external stimulus. This stimulus can be physical or chemical impact on the animal system.

Acknowledgement

Authors wish to thank authorities of Berhampur University for providing necessary laboratory facilities and working place in the department.

Conflict of interest

The authors declare that they have no conflict of interest for this publication.

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