



Impact of Industrial Solid Waste Of A Chlor-Alkali Industry On The AChE Activity Of Estuarine Fishes Collected from the Rushikulya Estuary and Its Ecological Significance.

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Abstract

Pollution of the aquatic environments by industrial wastes is not a new phenomenon. It's an old age practice. Knowingly most of the industries were discharging and dumping their wastes in the environmental segments. The physico-chemical variables of the discharged wastes from the industry were untreated and had higher level in each variable more than the stipulated concentration and level recommended by EPA, DOE, PCB and Environmental Act and Environmental schedule. The mercury concentration in sediment sample was found to be 489.62 ± 36.54 mg of Hg l⁻¹ and the mercury concentration decreased to 226.12 ± 9.25 mg of Hg l⁻¹ because of leaching at the solid waste dumping site. This warrants attention. No significant relationship exists between residual mercury concentration in fish body with either body length or weight of the fish collected from the contaminated estuary. Residual mercury in fish body depends on the availability and retention time of the fishes at the contaminated site. Autopsy studies revealed that the liver and brain of exposed fish were congested, pale and tender. All the exposed fish appeared lethargic after exposure to the SWE. The major clinical symptoms such as inappetance and ataxia appeared after 2 to 3 days exposure. At higher concentrations of the SWE, the exposed fish showed erratic movement leading to collision to inner side of the aquarium. At higher exposure periods, the exposed fish appeared lethargic and irregular swimming activity was observed when compared to control fish. Fish death started in exposed aquarium after 20days of exposure. The depletion in AChE enzyme activity in contaminated fishes can be linked to residual mercury impact on those tissues. The depression in active metabolism in exposed fishes might be due to depletion in respiratory metabolism, depletion in AChE activity and the erratic behavior of the contaminated fishes can be related to depletion in enzyme activity induced by residual mercury absorbed from the environment.

Keywords: Chlor-alkali industry, Solid waste, Mercury, fish, *Tilapia*, AChE activity.

Introduction

The Chlor-alkali industry was dumping its solid wastes on the riverside (bank of the river) and effluents initially directly for around 60years and later indirectly for around 20years, contaminating both aquatic and terrestrial ecosystems. The industry discharges its effluents through effluent canal. The sediments collected from effluent canal, treatment pond-1 and treatment pond-2, were dumped nearer to the Rushikulya River bank. Lechate chemicals and rain washed chemicals from solid waste dumps enter into the river and finally reach to the estuary and ultimately enter into the Sea (Priyadarsan & Panigrahi, 2024a,b). The photographs clearly indicated that the solid waste dumping site is not a safer place, as most of the domesticated animals graze in that area. All grazing plants contained mercury absorbed from the dumping site. These grazing animals also drink the effluent water as no fresh water

was available in the vicinity. Earlier Panigrahi (1980) reported presence of mercury in the milk of the cows roaming in the contaminated site. This mercury ultimately finds a way to enter into human body. Hence, this project was planned to study the impact of lechate chemicals of the solid waste of a Chlor-alkali industry on the AChE activity of estuarine fishes collected from the estuary.

Materials & method

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 effluent into the river directly (initially) and the solid waste collected from the effluent canal was dumped near the banks of Rushikulya river. The sediments collected from the effluent canal containing huge amount of mercury was removed periodically and dumped in and around the industry as huge deposits. During rainy season, flood water carries the dumped solid waste in to river Rushikulya contaminating the river water, Rushikulya estuary and Bay of Bengal. The discharged untreated effluent enters into the River, Rushikulya. The industry is located at $84^{\circ} 53'E$ longitude and $19^{\circ} 16'N$ latitude.



(Arc GIS explore expanded photograph showing the area and site map of M/S. Jayashree Chemicals Pvt. Ltd, located Rushukulya River and Rushikulya estuary at Ganjam near Bay of Bengal.



(Photographs showing the location of the Chloro-alkali industry, solid waste dumping site; industry and effluent stocking pond)

The temperature of the SWE prepared in the laboratory was $28.2^{\circ}C$. The pH of the SWE was alkaline and the value recorded was 9.4 ± 0.4 . The alkalinity was 241.8 ± 18.5 as $CaCO_3$ in $mg\ l^{-1}$. The hardness of the SWE sample was 392.2 ± 12.6 as $CaCO_3$ in $mg\ l^{-1}$. The chlorinity was $1018.6 \pm 24.6 mg\ l^{-1}$. The dissolved oxygen content was low and ranged within $2.2 \pm 0.4 mg\ l^{-1}$. The suspended solids were low but significant and the value was $102.5 \pm 6.4 mg\ l^{-1}$.

The physico-chemical analysis was carried out by following the APHA (1995) technical manual. The AChE activity was estimated following the protocol of Sigma Technical bulletin (USA). Body weight was measured by a electrical top pan balance. The length of the fish was measured by centimeter scale. Ten ml of the SWE was taken and acid digestion mixture (Conc. H_2SO_4 :Conc. HNO_3) was added and digested in a Bethge's apparatus (Wanntorp and Dyfverman, 1955). Effluent, water and sediment samples were digested following the same technique. Mercury concentration analysis was carried out in a Mercury Analyser following the detailed procedure mentioned in the ECIL manual for estimation of mercury. Total mercury level was expressed as mg of $Hg\ l^{-1}$ in the effluent sample. Live collected fish were immediately dissected on site and the tissues like brain, liver and muscle were separated carefully and were kept in Sucrose –Ringer's solution in small tubes, kept in ice bath, brought to the laboratory immediately for analysis. Few experiments were conducted in a nearby place (college), where instruments were kept for analysis (Priyadarsan & Panigrahi, 2018).

Test solution: The solid waste was collected from the solid waste dumping site and also from the sediments collected from the effluent canal and brought to the laboratory in glass jars and kept in the

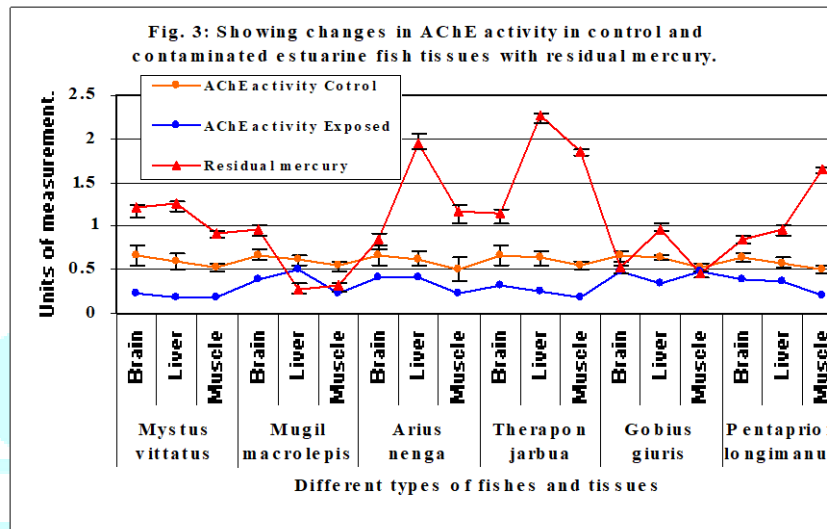
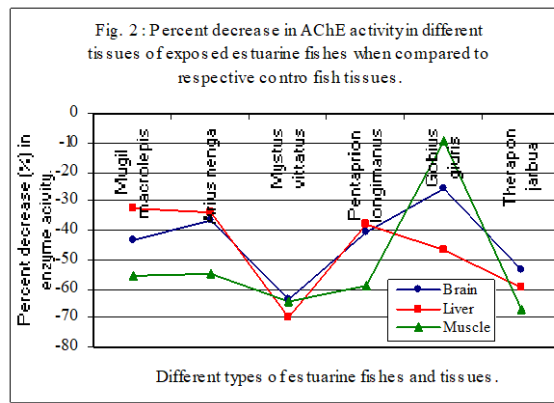
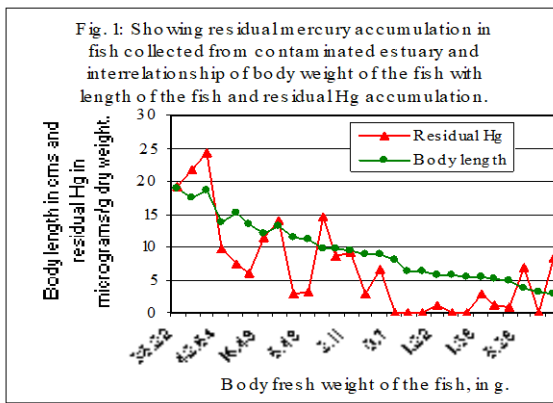
refrigerator for experimental use. The solid waste was air dried in shade, powdered and sieved. One kg of solid waste powder was mixed with 2liters of distilled water and stirred for 12hrs. Allowed to rest for 12hrs. The process was repeated for 15 days. After 15days the mixture was allowed to rest and the supernatant was carefully decanted and the extract was preserved in the fridge for use. The body weight of both control and exposed fish was measured by top pan balance. The fishes were not disturbed or excited. They were allowed to remain undisturbed. All the obtained values were statistically analysed.

Table: Analysis of solid waste extract used in this study (Priyadarsan, 2024)

Solid Waste Extract (SWE)	
pH	- 8.3±0.2
Phosphate	- 21.6 ± 3.4 mg l ⁻¹
Chloride	- 18.2± 1.4g l ⁻¹
Calcium	- 88± 11 mg l ⁻¹
Magnesium	- 21.1 ± 3.6 mg l ⁻¹
Sodium	- 5.2 ± 0.8 g l ⁻¹
Potassium	- 12.2 ± 16.6 mg l ⁻¹
Total nitrogen	- 4.5 ± 1.3mg l ⁻¹
Mercury	- 9.75 mg l ⁻¹

Results

Acute toxicity testing of the SWE revealed the following information. After 24h of exposure the lethal concentration values were, LC₀- 5.4%SWE, LC₁₀-6.2%SWE, LC₅₀- 8.4%SWE, LC₉₀-9.1%SWE and LC₁₀₀-9.9%SWE; after 48hrs, the LC values were LC₀- 5.4%SWE, LC₁₀-6.1%SWE, LC₅₀-6.6%SWE, LC₉₀-8.8%SWE and LC₁₀₀-9.7%SWE; after 72hrs, the LC values were LC₀- 5.1%SWE, LC₁₀-5.9%SWE, LC₅₀- 6.4% SWE, LC₉₀-8.5%SWE and LC₁₀₀-9.6%SWE; after 96hrs, the LC values were LC₀- 4.5%SWE, LC₁₀-5.6%SWE, LC₅₀- 6.3%SWE, LC₉₀-8.1%SWE and LC₁₀₀-9.4%SWE. After 28days of exposure in chronic poisoning, the LC values were LC₀- 2.85%SWE, LC₁₀-3.11%SWE, LC₅₀- 3.95%SWE, LC₉₀-4.68%SWE and LC₁₀₀-4.95%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. Autopsy studies revealed that the liver and brain of exposed fish were congested, pale and tender. All the exposed fish appeared lethargic after exposure to the SWE. The major clinical symptoms such as inappetance and ataxia appeared after 2 to 3 days exposure. At higher concentrations of the SWE, the exposed fish showed erratic movement leading to collision to inner side of the aquarium. At higher exposure periods, the exposed fish appeared lethargic and irregular swimming activity was observed when compared to control fish. Fish death started in exposed aquarium after 20days of exposure. The figure represents the relationship of between fish weight & length of different fishes collected from the estuary & whole body residual mercury accumulation. The figure clearly indicated the existence of a significant positive correlation between body length and body weight of the fish, except few exceptions. It was observed that the residual mercury accumulations in fishes are mostly weight dependent and indirectly length dependent. With the increase in body weight of the fishes, the residual mercury accumulation increased showing a positive correlation ($r= 0.989$; $p\leq 0.01$). However, with the increase in body length of the fishes, the residual mercury accumulation increased and showed a positive correlation ($r= 0.978$; $p\leq 0.05$). Both the parameters showed a link but the only difference was the level of significance. The changes in body length and body weight are time dependent and increases with time. The size of the fish also depends on environmental parameters and totally genetic. Hence uniform biological rule of growth cannot be adopted as a standard protocol for estimations, measurements and interpretation to understand a biological mechanism might be related to environmental stress. The residual accumulation of mercury in fish is definitely time dependent. The figures (Fig.1) however indicated that residual accumulation occurred in fish body and the correlation is positive for both the parameters but not significant.



The fish having 53.6g fresh weight accumulated 21.8mg of Hg kg⁻¹ dry weight having 17.4cm length. Whereas, the fish having 35.65g fresh weight accumulated 24.2mg of Hg kg⁻¹ dry weight having 18.5cm length. The fish having 7.2g fresh weight accumulated 14.6mg of Hg kg⁻¹ dry weight having 9.7cm length. The fish having 2.1g fresh weight accumulated 9.1mg of Hg kg⁻¹ dry weight having 9.3cm length. The fish having 1.36g fresh weight accumulated 2.85mg of Hg kg⁻¹ dry weight having 5.3cm length. Small fishes ranging between 1.2g to 0.4g weight did not show any residual mercury. In field conditions when the area is open and free movement of fishes occur, it is really difficult to assess the residual accumulation and interpret with either weight or length of the fish. It may so happen during fish catch, large fishes might have reached the site from sea without getting contaminated and the fisherman catches those fishes for us for analysis. Residual accumulation in static ecosystems is easy to assess but in dynamic ecosystems, difficult to assess. Similar interpretations are also equally valid when we correlate fish length with residual accumulation (Fig.1). The idea was to establish the relationship of the parameters. Whether body length of the fish can indicate residual accumulation or the body weight can indicate residual accumulation. Fig.8 represents the observation of residual mercury concentration in fish body collected from the estuary. The figure represents the relationship between fish length and residual mercury accumulation. Result showed species dependent accumulation of mercury in different tissues because *Arius nenga*, 9-10 cm in length accumulated mercury to the tune of 5 mg kg⁻¹ fresh wt, whereas other fishes of nearly same size accumulated mercury only to a level of 2.15 mg kg⁻¹ fresh fish (*Therapon jarbua.*, 8.5-9 cm) 2.18 mg kg⁻¹ fresh wt (*P. longimanus*, 7.8 cm), 1.35 mg kg⁻¹ fresh wt (*E. lineolata*, 8.9 cm), etc. Fishes of the same species but of different sizes showed a decreasing trend of accumulation in their muscle tissues with increase in the size. For example, in *M. macrolepis* when the length was 10-11 cm the mercury concentration was 0.26mg kg⁻¹ fresh wt whereas with increase in the length to 28-28.5 cm, the concentration decreased to 0.06 mg kg⁻¹ fresh wt. Similarly, in *A. nenga* the concentration of mercury was 5 mg kg⁻¹ fresh wt. in fish measuring 9-10 cm in length however, the concentration decreased to 1.5 mg kg⁻¹ fresh wt in fish of greater length (19.5-20.5 cm). Similar trend was observed in case of *T. jarbua*. One exception was *S. sihama* where the concentration increased with increase in the size. Fig.3 represent the changes in AChE activity of brain, liver and muscle of control fish collected from Gopalpur sea and exposed fish collected from contaminated estuary. In case of *Mugil macrolepis*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissue, when compared to its respective control tissue. The AChE activity declined from 0.67±0.11 to 0.38 ±0.04 μmole of Ach hydrolysed g⁻¹ hr⁻¹ in brain tissue; from 0.61±0.05 to 0.51±0.06 μmole of

ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.54 ± 0.06 to 0.24 ± 0.04 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Muscle tissue showed the highest depletion in the AChE activity, when compared to brain and liver tissues of the contaminated fish. Brain tissue showed 43.3% decrease, liver tissue showed 16.4% decrease and muscle tissue showed 55.6% decrease, when compared to its respective control tissues. Fig.3 represent the changes in AChE activity of brain, liver and muscle of control fish collected from Gopalpur sea and exposed fish collected from contaminated estuary. In case of *Arius nenga*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.66 ± 0.11 to 0.42 ± 0.08 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in brain tissue; from 0.62 ± 0.08 to 0.41 ± 0.12 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.51 ± 0.14 to 0.23 ± 0.07 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Muscle tissues showed the highest depletion in the AChE activity, when compared to liver and brain tissues of the contaminated fish. Brain tissues showed 36.4% decrease, liver tissues showed 33.9% decrease and muscle tissues showed 54.9% decrease, when compared to its respective control tissues. Table represent the changes in AChE activity of brain, liver and muscle of control fish collect ed from Gopalpur sea and exposed fish collected from contaminated estuary. In case of *Mystus vittatus*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.66 ± 0.11 to 0.24 ± 0.11 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in brain tissue, from 0.59 ± 0.09 to 0.18 ± 0.03 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.53 ± 0.04 to 0.19 ± 0.04 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Liver showed the highest depletion in the AChE activity, when compared to muscle and brain tissue of the contaminated fish. Brain tissues showed 63.6% decrease, liver tissues showed 69.5% decrease and muscle tissues showed 64.2% decrease, when compared to its respective control tissue. Table-4 represent the change in AChE activity of brain, liver and muscle of control fish collected from Gopalpur Sea and exposed fish collected from contamination estuary. In case of *Pentaprion longimamus*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissue. The AChE activity declined from 0.64 ± 0.05 to 0.38 ± 0.03 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.51 ± 0.04 to 0.21 ± 0.02 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Muscle tissue showed the highest depletion in the AChE activity, when compared to brain and liver tissues of the contaminated fish. Brain tissues showed 40.6% decrease, liver tissue showed 37.9% decrease and muscle tissue showed 58.8% decrease, when compared to its respective control. Table represent the changes in AChE activity of brain, liver and muscle of control fish collected from Gopaspur Sea and exposed fish collected from contaminated estuary. In case of *Trachinocephalus myops*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.69 ± 0.07 to 0.31 ± 0.11 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in brain tissue; from 0.63 ± 0.03 to 0.34 ± 0.09 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.54 ± 0.06 to 0.41 ± 0.04 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Brain tissue showed the highest depletion in the AChE activity, when compared to liver and muscle tissues of the contaminated fish. Brain tissues showed 55.1% decrease, liver tissue showed 46.0% decrease and muscle tissues showed 24.1% decrease, when compared to its respective control tissues, Table represent the changes in AChE activity of brain, liver and muscle of control fish collected from Gopalpur sea and exposed fish collected from contamination estuary. In case of *Gobius giuris*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.66 ± 0.06 to 0.49 ± 0.03 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in brain tissue; from 0.64 ± 0.3 to 0.34 ± 0.07 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.53 ± 0.04 to 0.08 ± 0.02 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Liver tissue showed the highest depletion in the AChE activity, when compared to brain and muscle tissue of the contaminated fish. Brain tissues showed 25.8% decrease, liver tissues showed 46.9% decrease and muscle tissues showed 9.4% decrease, when compared to its respective control tissues. In case of *Therapon jarbua*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.67 ± 0.11 to 0.31 ± 0.06 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in brain tissue; from 0.64 ± 0.08 to 0.26 ± 0.03 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in liver tissue and from 0.55 ± 0.04 to 0.18 ± 0.02 μ mole of ACh hydrolysed $g^{-1} hr^{-1}$ in muscle tissue. Muscle tissue showed the highest depletion in the AChE activity, when compared to liver and brain tissues of the contamination fish. Brain tissues showed 53.7% decrease, liver tissues showed 59.4% decrease and muscle tissues showed 67.3% decrease, when compared to its respective control tissues In case of *Lutianus johnil*, the

AChE activity significantly declined in exposed fish brain, liver and muscle tissue, when compared to its respective control tissues. The AChE activity declined from 0.68 ± 0.07 to $0.34 \pm 0.08 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in brain tissue; from 0.66 ± 0.05 to $0.36 \pm 0.05 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in liver tissue and from 0.51 ± 0.04 to $0.32 \pm 0.04 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in muscle tissue. Brain tissue showed the highest depletion in the AChE activity, when compared to liver and muscle tissues of the contaminated fish. Brain tissues showed 50.0% decrease, liver tissues showed 45.5% decrease and muscle tissues showed 37.3% decrease, when compared to its respective control tissues. In case of *Scatophagus argus*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.68 ± 0.13 to $0.52 \pm 0.21 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in brain tissues; from 0.61 ± 0.12 to $0.383 \pm 0.08 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in liver tissue and from 0.49 ± 0.09 to $0.22 \pm 0.04 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in muscle tissue. Muscle tissues showed their highest depletion in the AChE activity, when compared to liver and brain tissues of the contaminated fish. Brain tissues showed 23.5% decrease, liver tissue showed 37.7% decrease and muscle tissues showed 55.1% decrease, when compared to its respective control tissues. Fig.3 represent the changes in AChE activity of brain, liver and muscle of control fish collected from Gopalpur sea and exposed fish collected from contamination estuary. In case of *Equula lineolata*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.68 ± 0.09 to $0.19 \pm 0.04 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in brain tissue; from 0.66 ± 0.07 to $0.22 \pm 0.06 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in liver tissue and from 0.56 ± 0.05 to $0.17 \pm 0.04 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in muscle tissue. Brain tissue showed the lowest depletion in the AChE activity, when compared to muscle and liver tissues of the contaminated fish. Brain tissues showed 72.1% decrease, liver tissue showed 66.7% decrease and muscle tissue showed 69.6% decrease, when compared to its respective control tissues. In case of *Sillago sihama*, the AChE activity significantly declined in exposed fish brain, liver and muscle tissues, when compared to its respective control tissues. The AChE activity declined from 0.68 ± 0.12 to $0.38 \pm 0.16 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in brain tissue; from 0.62 ± 0.14 to $0.39 \pm 0.21 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in liver tissue and from 0.4 ± 0.09 to $0.24 \pm 0.14 \mu\text{mole of ACh hydrolysed g}^{-1} \text{ hr}^{-1}$ in muscle tissue. Muscle tissue showed the highest depletion in the AChE activity, when compared to brain and liver tissues of the contaminated fish. Brain tissues showed 44.1% decrease, liver tissues showed 37.1% decrease and muscle tissues showed 47.8% decrease, when compared to its respective control tissues. Mercury is deadly toxic and affects the neurotransmission of SWE exposed fishes.

Discussion

The leachate leaching from solid waste dump site showed serious impact on estuarine fishes and because of the wastes discharge on this site, the breeding of marine fishes was seriously affected. The impact became more serious because of mercury concentration in the environment. The other physico-chemical variable had no effect because of dilution factor. The mercury concentration in sediment sample was found to be $489.62 \pm 36.54 \text{ mg of Hg l}^{-1}$ at station-V in 2013 and the mercury concentration decreased to $226.12 \pm 9.25 \text{ mg of Hg l}^{-1}$ at station-V in 2014. From residual mercury stand point, insignificant amount of mercury was recorded in stations VI to VIII, when compared to other 5 stations located either in the upstream or downstream from the junction point where effluent joins the Rushikulya River (Priyadarsan and Panigrahi, 2024a,b). No significant relationship exists between residual mercury concentration in fish body with either body length or weight of the fish collected from the contaminated estuary. Residual mercury in fish body depends on the availability and retention time of the fishes at the contaminated site. The physico-chemical analysis of the effluent, solid waste discharged from the industry showed very high level of mercury. The solid waste leachate leaching from solid waste dump site, effluent leachate from effluent stocking pond raised the mercury level in different stations located in and around the industry. Leachate 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 $9.75 \text{ mg of Hg l}^{-1}$ (Priyadarsan, 2024). 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. Autopsy studies revealed that the liver and brain of exposed fish were congested, pale and

tender. All the exposed fish appeared lethargic after exposure to the SWE. The major clinical symptoms such as inappetance and ataxia appeared after 2 to 3 days exposure. At higher concentrations of the SWE, the exposed fish showed erratic movement leading to collision to inner side of the aquarium. At higher exposure periods, the exposed fish appeared lethargic and irregular swimming activity was observed when compared to control fish. Fish death started in exposed aquarium after 20 days of exposure. The ions studied showed significant decrease in all tissues of the exposed fish, when compared to the control fish tissues indicating ionic imbalance responsible for the erratic behavior of the SWE exposed fish. 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. In field conditions when the area is open and free movement of fishes occur, it is really difficult to assess the residual accumulation and interpret with either weight or length of the fish. It may so happen during fish catch, large fishes might have reached the site from sea without getting contaminated and the fisherman catches those fishes for us for analysis. Residual accumulation in static ecosystems is easy to assess but in dynamic ecosystems, difficult to assess. Similar interpretations are also equally valid when we correlate fish length with residual accumulation (Fig.1). The idea was to establish the relationship of the parameters. Whether body length of the fish can indicate residual accumulation or the body weight can indicate residual accumulation. Fig.8 represents the observation of residual mercury concentration in fish body collected from the estuary. The figure represents the relationship between fish length and residual mercury accumulation. Result showed species dependent accumulation of mercury in different tissues. Fishes of the same species but of different sizes showed a decreasing trend of accumulation in their muscle tissues with increase in the size. Mercury (Hg) is a contaminant found in ecosystems globally. Although inorganic and insoluble Hg has relatively low bioavailability, methyl mercury (MeHg) is a neurotoxic chemical which can biomagnify rapidly to elevated concentrations in biota such that concentrations in top trophic predators can threaten both ecological and human health (Boudou *et al.*, 1991 and Mason *et al.*, 1996). Human activities have greatly increased the emissions of mercury into the environment over the past century (Mason *et al.*, 1994). Mercury can be biomagnified at all levels of aquatic food chains (Mason *et al.*, 1995), and high levels of mercury have been detected in fish, especially in the organic form. This imposes a great threat on human health through seafood consumption (Wu and Wang, 2011). The same author also pointed out that toxic responses are specific to metals and species, but the sub-cellular basis underlying such inter-species and inter-metal differences was not clear. The depletion in enzyme activity like ATPase and AChE in contaminated fishes can be linked to residual mercury impact in those tissues. The depression in active metabolism in contaminated / exposed fishes might be due to depletion in respiratory metabolism and the erratic behavior of the contaminated fishes can be related to depletion in enzyme activity induced by residual mercury absorbed from the environment. 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 macrophage (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 sulfhydryl 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, AChE 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 (Priyadarsan, 2024; Panigrahi & Misra, 1978,1980). This stimulus can be physical or chemical impact on the animal system.

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Conflict of interest

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

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