ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 3, Issue, 7, pp.128-130, July, 2011

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

IMPACT OF MERCURY ON THE ACETYLCHOLINESTERASE ACTIVITY IN MERCURY EXPOSED SPHAERODEMA RUSTICUM (HETEROPTERA: BELASTOMATIDAE)

V. Rajathi[1]* and Selvi Sabhanayagam[2]

[1]Department of Zoology, Government Arts College, C.Mutlur, Chidambaram-608102
[2]Department of Zoology, Annamalai University, Annamalai Nagar-608002

ARTICLE INFO

Article History:
Received 18th April, 2011
Received in revised form
12th May, 2011
Accepted 9th June, 2011
Published online 16th July 2011

Key words:
Mercury,
Brain test, *Sphaerodema rusticum.*

ABSTRACT

In the present study, an attempt has been made to analyze the changes in the acetylcholinesterase activity in the brain and testis of *Sphaerodema rusticum*. The sublethal concentration of mercury decreased the acetylcholinesterase activity in the brain and testis of *Sphaerodema rusticum.*

INTRODUCTION

Heavy metals enter into aquatic habitats by a number of routes and cause hazardues effect on their morphology and physiology. Heavy metal pollution of water is a major environmental problem facing the modern world (Shrivastava and Sathyanesan, 1987). Heavy metals have a unique property of accumulation over a period of time, along a food chain and a very high level can be accumulated in an organism from very low level concentration in water and sediments (Shrivastava and Sathyanesan, 1987; Bose *et al.,* 1994). Heavy metals are known for many years to produce toxic effects on the central nervous system. Their presence in the environment and at working places may pose serious health problem (Weiss and Simon, 1975). Heavy metal contaminants in aquatic ecosystems pose a serious environmental hazard because of their persistence and toxicity. Among myriad of heavy metal pollutants, mercury merits a special attention due to its potential health hazard to aquatic biota and human life in particular. Though mercury is one of the rare elements on earth's crust, it is found everywhere and everything (Weiss *et al.,* 1971. Mercury is distributed through the environment and it is derived from industrial processes, agricultural activities, burning of fossil fuels and weathering of geologic formations (WHO, 1989). It is an important pollutant when discharged into aquatic system can be ethylated and accumulated in most aquatic biota mush of the mercury in aquatic organisms is present in the organic from due to bimethylation particularly in anaerobic sediments. Mercury is the heavy metal that conducts electricity and combines easily with many metals. Forming alloys, called amalgams. Mercury containing compound have many uses including acting as effective pesticides, fungicides and preservations (ATSDR, 1999). Globally, detritions of the environment have increased the vulnerability of various populations to mercury from contaminants (Nriagu, 1988). Insect reproduction is an essential physiological process from the point of view of propagation and it has received relatively little direct attention perhaps due to the fact that its process so intimately associated with other system and are controlled by both intrinsic and extrinsic factors. In male insects, the reproductive organs are seen visibly affected either as a result of direct or by the indirect effect of chemicals. Induced sterility may be due to the complete cessation of spermatogenesis, resulting in the loss of fertility due to the presence of dominant lethal (Balakrishnan, 1990).

MATERIAL AND METHODS

The insects, *Sphaerodema rusticum* (Heteroptera: Belastomatidae) were collected from the local ponds and streams were maintained in the plastic trough at the laboratory temperature of 28±5. The insects were daily fed with mosquito larvae, pieces of earth worm and aquatic plants. The troughs were cleaned properly every alternative day changing the water. The insects were exposed to mercury (9ppm). The brain and testis were isolated and isolated tissues were used for the estimation of acetylcholinesterase (AchE) activity.

Corresponding author: sakipillai_zoo@yahoo.co.in

Estimation of acetylcholinesterase (AchE) activity

Acetylcholinesterase (AchE) activity was estimated by the following method of Metcalf (1951).All the tissues were isolated and 2% homogenate of the sample was prepared in 0.25 N cold sucrose solution and centrifuged at 3,000 rpm for 10 minutes. The clear supernatant was used for enzyme assay. 1.0 ml of clear supernatant was taken in a clean test tube and 1.0 ml of reaction mixture (0.1 M sodium potassium phosphate buffer and 0.008 M acetylcholine chloride in the ratio of 4:1) was added. The reaction was stopped after 30 minutes of incubation at 37°C by the addition of 2.0 ml of alkaline hydroxylamine hydrochloride followed by 1.0 ml of hydrochloric acid (1:1 ratio of HCL : water). The contents were shaken thoroughly and filtered. 2.5 ml of aliquot of the filterate was taken and 0.5 ml of clear 0.37 M ferric chloride solution was added to each aliquot. The intensity of the colour was measured at 545 nm in a UV-spectrophotometer against a reagent blank. The enzyme activity was calculated from the standard graph. The values are expressed as µmoles hydrolysed/mg.wet wt. of tissue/hr. The significance is analysed by using the student't' test.

RESULTS

In the normal brain tissue of insect the activity of acetyl cholinesterase was 4.317±0.004µm hydrolysed/mg protein/hr. During the mercury exposure the acetylcholinesterase activity is decreased (1.859±0.002µm hydrolysed/mg protein/hr). The percent change over the control was -57.09. In the normal testis of insect, the activity of acetylcholinesterase was 0.432±0.004 002µm hydrolysed/mg protein/hr. But in mercury exposed insect the activity of acetylcholinesterase was decreased upto 0.26±0.002002µm hydrolysed/mg protein/hr. The percent change over the control was -58.23.

Table 1.Activity of acetylcholinesterase (AchE) in brain and testis of Sphaerodema rusticum exposed to mercury (µ mole hydrolysed/mg/hr.)

Tissue	Control	Mercury Treated	Percent change over control
Brain	4.317±0.004	1.859±0.002	-57.09
Testis	0.62±0.004	0.26±0.002	-58.23

*Significance at 5 % of student 't ' test

DISCUSSION

Acetylcholinesterase (ACHE) is an enzyme and plays an important role in the control of normal cerebral activity by hydrolyzing and thereby terminating the action of the neurotransmitter (Sing *et al.,* 1979). It is a dependable indicator for assessing the effects of various xenobiotics (Mukhergee and Bhattacharya, 1974). Acetylcholinesterase is an important regulatory enzyme that controls the transmission of nerve impulses across cholinergic synapes by hydrolyzing to excitatory transmitted acetylcholine (Mitatrovic and Dettbarn, 1996). Acetylcholinestese (AchE) is a serine hydrolase, catalyzes the breakdown of the beurotrons mitter acetil cholbe into acetic acid and choline. This process involves the formation of a substrate enzyme complexes, followed by accentuation of the of the hydroxyl group. The inhibitory effects of AchE activity indicate that pollutions might interferon in the vital processes of energy metabolism of nerve cells (Nath and Kumar, 1999). The functional significance of cholinergic mechanisms in the nervous system has been reported by many investigators (Stavinoha et al., 1977; Sing and Sing, 2003). The heavy metals are widespread in the environment. These metals are accumulated in many tissue (Niemi *et al.,* 1991). The concentration of metals varies among the animal tissue depending on the route of uptake forms the metal and the physiological fate of the metal (Terry and William, 1994). In animals, heavy, metal accumulation causes atrophy, brain hemorrhaged and liver damage (Niemi *et al.,* 1991). The accumulation of the heavy meaty probably occurs through the process that exists for the essential metals. The heavy metals are known to induce cell injury in the tissues (Nolan and Shaikh, 1992).

In the present study, the level of acetylcholinesterase decreased in brain and testis tissue of *Sphaerodema rusticum* exposed to mercury. This result indicates that arsenic block the active center of enzyme and also drastically inhibits its de novo synthesis. The toxic effect of heavy metals on the necrotronsmitter may result from their action or sub cellular process such as interference with mechanism regulating calcium distribution in nerve terminals and anabolic effect that may occur as a result of impairment of energy production or inhibition of enzymes involved in the synthesis and storage of transmitters. Ferrando *et al.* (1992) base reported the bioaccumulation of insecticide after the acetylcholine and aacetylcholinesterase in the *Angulla angulla*. Nemscok *et al.,* (1990) reported that AchE causes death in higher vertebrates by blocking neurotransmission in the neuromuscular function. Radha Pant *et al.* (1982) observed the AchE was decreased in Philosania ricinilarval exposed to hexachlorobenzene.

Acknowledgement

The authors are thankful to Professor and Head, Department of Zoology, Annamalai University for providing necessary lab facilities to carry out the work successfully.

REFERENCES

Agency for toxic substances and Disease Registry (ATSDR). 1999. Toxicological profile for mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. www.atsdr.cdc.gov/toxprofiles/tp46.html

Balakrishnan, S.1990. Studies on the effect of Roger on the histology and histochemistry of the testis, fat body and accessory reproductive gland of adult male *Pherosophus lissoderus*. M.Phil Thesis. Annamalai University. Tamilnadu

Bose, S., Mukhopadhyay, B., Shibani Chaudhury and Bhattacharya. 1994. Correlation of metal distribution, reduced glutathione and metalothionein level in liver and kidney of rat. *Ind. J. Exp. Biol.,* 32:pp. 679-681.

Metcalf, R.L. 1951. Methods in biochemical analysis. (Glick, D. Eds.) Vol.V. Interscince publication. New York.44.

Mitatrovic, D. and Dettbarn, 1996. Modification of acetyl cholinesterase during adoptation to chronic, subacute paraoxon application in rat. *Toxicol. Appl. Pharmacol.,* 136: 20-28.

Nath, B.S. and Kumar, R.P.S. 1999. Toxic impact of organophosphorous insecticides on acetyl cholinesterase

activity in the silkworm. *Ecotoxicol. Environ. Saf.,* 42: 157-162.

Nemscok,J. Rakoncyzay, Z., Kasa,P Astalos,B and Azobo,A. 1990. Effect of methidation on distribution of molecular forms of acetylcholinesterase in carp as revealed by density gradine pest. *Biochem. Physiol.,* 37:140-144.

Niemi, A., Venalainen, E.R., Hirri, T., Hirn, J. and Karpanen, E. 1991. The lead, cadmium and mercury concentration in muscle, liver and kidney from gunia pigs and cattle during 1987-1988. *Zleberism Unters Forsch.,* 192: 427-429.

Nolan, C.V. and Shaikh, Z.A. 1992. Lead nephrotoxicity and associated disorders : Biochemical mechanisms. *Toxicol.,* 73: 127-146.

Nriagu, J.0.1979. The biogeochemistry of mercury in the eniornment. Elsevier / North Holland. Biomedical press, *Amsterdam*, 366-398

Radha Pant, Santhosh Kumar,K and geeta,J. 1982. Effect of feeding hexachlorobenzene and AchE to *Philosania ricinilarval*. *Curr. Sci.,* 51: 733-737.

Shrivastava, V.K. and Sathyanesan, A.G. 1987. Effect of lead nitrate on thyroid function of Indian Palm Squirrel, F. Pennanti (w). Bull. *Environ, contam. Toxicol.,* 38: 981-984.

Singh, H.C., Singh,R.H. and Udupa, K.N. 1979. Electric shock induced changes in free, bound total acetylcholine and acetylcholinesterase in different brain regions of rats. *Ind. J. Exp. Biol.,* 17(3): 304.

Singh, S.P. and Singh, R. 2003. Histoenzymological demonstration of alkaline phosphate, ATpase and acetylcholinesterase in the cerebellar cortex of desert bat, *Rhinopoma microphyllum*. *Biochem. Cell.Arch.,* 3, 53-56.

Stavinoha, W.B. Hosoy E, Maruyama Y. and Modak, A.T. 1977. Regional acetyl content in mouse brain after morphine tablet implantation. *Arch. Int. Pharmacodyn. Thet.,* 225, 58-63.

Terry, A.H. and William, G.B. 1994. Water, Air and Soil pollution 73: 265-274.

Weiss, H.V., Kodia M. and Guld berg, E.D. 1971. mercury in green land ice sheet. Evidence of recent input by man. *Science,* 174: 692.

Weiss,B. and W. Simon. 1975. Long term toxicity of mercury. In Behavioural Toxicology. Edited by. B. Weiss. V.G. Laties. Plenum press. New York, pp.429 - 439.

WHO, 1989. "Mercury – Environmental aspects". Environmental health Criteria 86, publication of WHO, Geneva ISBN. 92-4-154586.

****