Vol 4 Issue 1 Feb 2014

ISSN No : 2230-7850

International Multidisciplinary Research Journal

Indian Streams Research Journal

Welcome to ISRJ

RNI MAHMUL/2011/38595 **ISSN No.2230-7850**

Indian Streams Research Journal is a multidisciplinary research journal, published monthly in English, Hindi & Marathi Language. All research papers submitted to the journal will be double - blind peer reviewed referred by members of the editorial board.Readers will include investigator in universities, research institutes government and industry with research interest in the general subjects.

Address:-Ashok Yakkaldevi 258/34, Raviwar Peth, Solapur - 413 005 Maharashtra, India
Cell : 9595 359 435, Ph No: 02172372010 Email: ayisrj@yahoo.in Website: www.isrj.net

Indian Streams Research Journal
ISSN 2230-7850
Volume-4 | Issue-1 | Feb-2014
Available online at www.isrj.net

DIMETHOATE INDUCED OXIDATIVE STRESS IN HEART ALBINO RAT

R. Penchalamma , Ramesh Babu. Kand and Jacob Doss. P

Division of Toxicology, Dept. of Zoology, S.V. University , Tirupati, Andhra Pradesh, India

Abstract:-Now days, people's exposure to chemical compounds such as organphosphorous insecticide is continuously on the rise more and more. These compounds have induced an excessive production of free radicals which are responsible for several cell alterations in the organism. Recent investigations have proved the crucial role of nutritional antioxidants to prevent the damage caused by toxic compounds.Dimethoate (DM) is an organ phosphorous insecticide and acaricide used to kill mites and aphids among other insects and is applied on citrus, cotton, fruit, olives, potatoes, tea, tobacco and vegetables. The aim of the present work was to study biochemical changes that might occur in the heart of albino rats as a result of DM intoxication. In the present investigation the animals were treated with 1/10th of LD50of DM via oral gavage (34.5mg/kg body weight. The first group animals were considered as control animals. Second group of animals were treated with Dimethoate via oral gavage (34.5mg/kg body weight which is 1/10th of LD50) for 10 days, third and fourth groups of animals were administered for 20 and 30 days with an interval of 48h respectively. Total proteins showed decrement in the DM treated groups while all the other parameters selected in the present investigation showed an increment. The present findings indicate that chronic exposure to DM has clear toxic effect on the heart of albino rats.

Keywords: Dimethoate, Albino rat, Heart, Antioxidants.

INTRODUCTION

For several years, a special attention has been paid to oxidative stress situation of an excessive production of reactive oxygen species (e.g. the famous “free radical”) in the organism. A large number of experimental and epidemiological studies have indicated that the reactive oxygen species (ROS) contribute to organ injury in many systems (Halliwell et al., 1992cadet et al., 2002: Del Rio et al., 2005: Beaudeux et al., 2006: Goetz and Luch, 2008). Reactive oxygen species are constantly formed as a byproduct of normal metabolic reaction and their generation is accelerated by accidental exposure to occupational chemicals like pesticides.Organophospharus insecticide (OP) represents one group of pesticides that is widely used and has proved to have toxic effects in humans and animals (De-Bleecker et al., 1993: Betrosian et al., 1995: Tsatsakis et al., 1998: Hagar and Fahmy, 2002). Yahya et al., (2012) studies the effect of DM induced oxidative stress and morphological changes in the liver of guinea pig. Devi Sri Lakshmi kala et al., (2013) studied the effect of DM on different regions of the brain in the albino rat.

It is well known that pesticides, which we are widely used in agriculture, have many harmful effects on living organisms. Animals in the natural environment are usually exposed to low concentrations of xenobiotics, which are sub – lethal.Dimethoate (S- methylcarbamoyl-methyl-O,O- dimethyl phosphorodithioate) has been used for many years in many countries as a broad – spectrum insecticide.The antioxidant system an important role in protecting the biological system below a critical threshold of reactive oxygen species (ROS), thus preventing dysfunction. Antioxidant enzymes constitute a mutually superortive team of defence against ROS. It has been reported that increase in oxygen free radicals (OFR) can cause the destruction of all the cellular structure. The basis of Dimethoate toxicity as an organ phosphorous insticide (OPI) in the production of OFR may be due to: It's“redox- cycling” activity: it easily accepts an electron to from free radicals and then transfers them to oxygen to generate super oxide anions and hence hydrogen peroxide through dismutation reaction.Generation of free radicals probably because of the alteration in the normal homeostasis of the body resulting in antioxidants stress, if the requirement of continuousanti-oxidants is not maintained.SOD is an enzyme capable of removing superoxide radicals by catalyzing its dismutation to O2 plus H202 in the cell. When the concentrations of hydrogen peroxide in

the cell exceed the physio – biological level, catalase takes over the protective function and it to H20 and O2.When the generation of reactive free radicals overwhelms the anti-oxidant defence, lipidperoxidation of the cell membrane occurs. Acute poisoning by organ phosphorus (OP) compounds is a major global clinical problem, with thousands of deaths occurring every year. In recent years, the hazards of using these chemicals have been accentuated by the sharp rise in their use in agriculture and industry and by householders and governments. OP compounds are currently among the most frequently used pesticides worldwide (Heudorf et al., 2006). Toxicity of these OP compounds results in negative effects of many organs like liver, heart, kidney, nervous system and reproductive system (Ferah Sayim, 2007). Symptoms of acute poisoning of DM are common with all other OPs and revolve around cholinesterase inhibition. DM is highly mobile in the soil, labeled as a class II Moderately Toxic insecticide by (EPA) and is somewhat persistent, and is not often found in large quantities in water. It is however, highly toxic to honeybees, moderately to highly toxic to birds, and moderately toxic to aquatic organisms.DM is a widely used OP used to kill mites and aphids among other insects and is applied on citrus, cotton, fruit, olives, potatoes, tea, tobacco and vegetables. For humans, the main groups at risk of high rates of DM exposure are pesticide producers, pesticide workers and farm owners (Sharma et al., 2005).

Present study critically examines the magnitude and relationship of the metabolites enzymes involved in the antioxidant and histopathological changes in heart tissue of rats treated with sub lethaldoses of Dimethoate, since the farmers, pesticide applicators , industrial workers and other pesticide users will be exposed to the pesticides repeatedly.

MATERIAL AND METHODS

Test Chemical: Dimethoate Technical (94%) pure in crystalline form was obtained from Hyderabad chemical limited, Hyderabad A.P., India.

Animal model: Male adult Albino rat of 7 weeks old and aged 200 ± 20 g. were obtained from Indian Institute of Science (II.Sc.), Bangalore. They were housed at an ambient temperature 28 ± 2°C in a 12-h light/dark cycle and a minimum humidity of 40%. The animals had free access to commercial pellet diet supplied by Sai Durga Feeds and Foods, Bangalore, India and water ad libitum.

EXPERIMENTAL DESIGN:

All the male healthy adult male albino rats were randomly divided into four groups having with six rats per group. The first group animals were considered as control animals. Second group of animals were treated with Dimethoate via oral gavage (34.5mg/kg body weight which is 1/10th of LD50) for 10 days, third and fourth groups of animals were administered for 20 and 30 days with an interval of 48h respectively.

Biochemical Assays:

The activity of SOD was assayed by the reduction of nitro blue tetrazolium. Here the Superoxide was produced by riboflavin mediated photochemical reaction system. Superoxide dismutase activity was determined according to the method of Beachamp and Fridovich (1971). Catalase activity was measured by a slightly modified version of Aebi (1984) at room temperature.

Statistical treatment:

The data was subjected to statistical treatment. One way analysis of variance (ANOVA) and S-N-K tests were performed using SPSS (ver. 12) in the personal computer and $p < 0.05$ was considered as statistically significant.

RESULTS AND DISCUSSION:

The data presented in graphs showed the changes in SOD and CAT Activity. Gradual decreased in these enzymes activity was observed from single dose to multiple doses treated rats in dose dependant manner. The alterations in antioxidant enzyme activities were more pronounced in multiple dose treated Albino rats. The decrease in SOD and CAT markers of radical oxygen species generation, which indicates the Oxidative Stress induced byDimethoateThe increase in the activity of SOD and CAT found in our investigation was correlated with the findings of Homi Hercilia et al. (2002). Who reported the decreased SOD and Catalase activity levels in different brain regions during oxidative stress conditions. The Superoxide dismutase and Catalase activity levels were depleted significantly in different tissues during repeated exposure of rats to Deltamethrin (Manna et al. (2005). According to Gupta et al. (1999). continuous exposure of albino rats to quinophos up to PND 45, the SOD and CAT activities were decreased as 63% and 31% respectively.

According to Banerjee et al. (1999) pesticides may induce oxidative stress leading to generation of free radicals and alteration in antioxidant or oxygen free radicals scavenging enzyme system. SOD activity was significantly inhibited in both the brain and liver of albino rat during the development of behavioral tolerance to organophosphate compound phosphomidon

(Venkateswara Rao, 1993). In Rat heart tissues, pathological changes include Congestion (C), Slight Infiltration (SI), Infiltration (I), Rounded Nucleus (RN), Severe Necrosis in Cardiac Muscle Fiber(SNCMF) , Nucleus(N), Denaturated Nucleus (DN). So rat heart tissue is damaged with the pesticide effect and all tissues.Dimethoate effects on various enzymatic antioxidants such as SOD & Catalase. All the enzyme activities in the present study decreased as compared to their respective controls. This indicates the failure of antioxidant defense system to over the influx of ROS induced by Pesticide exposure.

REFERENCES

1.Aebi H, (1984). Catalase in vitro. Methods enzymol 105:121-6.
2.Banerjee BD, Seth V.Ahmad RS.(2001). Pesticide – induced oxidative stress: perspective and trands. Rev environ health 16:1.
3.Beyer WE.Fridovich I.(1987). Assaying for SOD activity some large consequences of minor changes in conditions. 161 : 559-66
4.Betrosian A, Balla M,Kafiri G,Kofinas G, Makri R,Kakouri A. (1995). Multiple systems organ failure from organophosphate poising.J Toxicol Clin toxicol 33(3):257-60.
5.Cadet J, Bellon S, Berger M,Bourdat AG, Douki T,DuarteV, (2002). Recent aspects of oxidative DNA damage: guanine lesions, measurement and substrate specificity of DNA repair glycosylases. Biol Chem 383:93.
6.De-Bleecker J, van-Den-Neucker K, Colradyn F, (1993).Intermediate syndromeinOrganophospharus poising: a prospective study. Crit Care Med 21(11): 1706-11.
7.Del Rio D, Stewart AJ, Pellegrini NA, (2005).review of recent studies on malonialdehyde as toxic molecule and biological marker of oxidative stress. Nutr Metab Cardiovasc Dis 15:316-28.
8.Devi Srilakshmi Kala S, Umakanthi V, Jayasudha M, Malleswari M, RavikanthSV and Jacob Doss P,(2013). Effect of Dimethoateon different regions of thebrain inAlbino rat. (In Press)
9.FerahSayim, (2007) Dimethoate-induced biochemical and histopathological changes in the liver of rats. Experimental and Toxicologic Pathology 59:237-243.
10.Fridovich I, Beachamp,(1995). Superoxide radical and superoxide dismutases. Annu.Rev.Biochem, 64-97.
11.Goetz ME, Luch A, (2008).Reactive species: a cell damaging rout assisting to chemical carcinogens. Cancer Lett 266: 73-83.
12.Halliwell B, Guteride JMC, Cross CE, (1992). Free radicals, antioxidants and human disease: where we now? J Lab Clin Med 119:598-620.
13.Hagar HH, Fahmy AH, (2002). A biochemical, histochemical and ultrastructural evaluation of the effect of Dimethoate intoxication on rat pancreass. Toxicol Lett 133:161-70.
14.Heudorf U, ButteW, Schulz C, Angerer J, (2006). Reference values for metabolites of pyrethroid and organophosphorus insecticides in urine for human bio monitoring in environmental medicine. Int J Hyg Environ Health, 209: 293-9.
15.Manna, S, BhattacharyyaD, MandalTKand Das S, (2005). Reported dose toxicity of Deltamethrin in rats. Indian journal of pharmacology, 37(3): 160-164.
16.Sharma Y, Bashir S, IrshadM,Dattagupta S,Dogra TD,(2005a). Effect of acute Dimethoate administration on antioxidant status of liver and brain of experimental rats, Toxicology, 206: 2005a, p. 49-57.
17.Sharma Y, BashirS,Irshad M, DattaguptaS,Dogra, TD, (2005b.). Dimethoate- induced effects on antioxidant status of liver and brain of rats following subchronic exposure, Toxicology, 215: 2005b, p. 173-181.
18.Venkata Swamy K, (1991). Neurochemical studies during the development of behavioral tolerance to organophosphate toxicity in albino rats. Ph.D Thesis, Sri Venkateswara University, Tirupati, India.
19.Yahya S, Al-Awthan Mohanmed A, Al-Douis Gamal H, El-Sokkary and Esam,(2012).Asian Journal of Biol. Sc. 5(1): 9-19.

Table 1.Changes in Superoxide Dismutase (SOD) activity (units of superoxide anion reduced/mg protein/min.) levels in different tissues of Albino rat exposed to sublethal dose of Dimethoate. Values in parentheses indicate percent change over control.

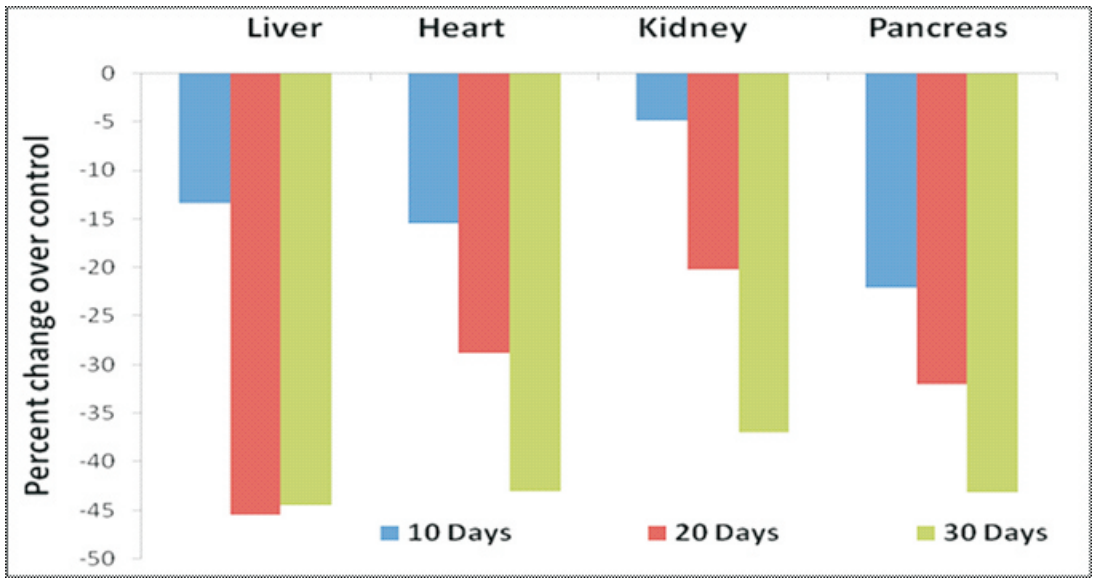

Table 2. Changes in Catalase activity (moles of H2O2 decomposed /mg protein/min) levels in different tissues of Albino rat exposed to sub lethal dose of Dimethoate.