ISSN: 0976-3031

Available Online at http://www.recentscientific.com

International Journal of Recent Scientific Research
Vol. 4, Issue, 9, pp.1323- 1326, September, 2013

International Journal of Recent Scientific Research

RESEARCH ARTICLE

EFFECT OF DIMETHOATE 30EC ON SOME HEMATOLOGICAL PARAMETERS OF ALBINO MICE FOLLOWING AN ORAL EXPOSURE

Mudasir Yasin and Poonam Sharma

Department Of Zoology Ground Floor Vigyan Bhawan Bundelkhand University Jhansi Utter Pardesh 284128

ARTICLE INFO

Article History:

Received 12th, August, 2013
Received in revised form 28th, August, 2013
Accepted 17th, September, 2013
Published online 30th September, 2013

Key words:
Organophosphorous compounds, Dimethoate, Hematological Parameters.

ABSTRACT

Organophorous compounds are widely used in industry, agriculture and for public health purposes. They are among the toxic compounds employed for insect control. The purpose of this work to study the hematological changes that might occurin the blood of mice as a result of acute Dimethoate intoxication. The results revealed that Dimethoate treated mice showed significant increase in RBCs, percent monocytes, percent basophills and percent neutrophills. However a significant decrease was observed in percent lymphocytes, hemoglobin count, PCV, MCV, MCH, MCHC and ESR. The number of eosonophils was increased insignificantly. When the Dimethoate treatment was stopped and the mice were allowed tometabolize the toxicant a significant recovery in the hematological parameters was observed showing the inbuilt capacity of detoxification.

INTRODUCTION

Now a days a frequent rise in the use of organophosphorous compounds in agriculture, industry and by house holders has been observed. Exposure of these pesticides in agriculture is one of the occupational hazards. These pesticides represent a group of pesticides that is widely used and has been shown to have toxic effects in man (Tsatsakis *et al.*, 1998; Betrosian *et al.*, 1995; De-Bleekeretal., 1993). Toxicity of organophosphorous pesticideany organs results in effects on many organs (Betrosian *et al.*, 1995), particularly the nervous system (Desi *et al.*, 1998; Nagymajtenyi *et al.*, 1998). These studies have shown that organophosphate intoxication caused abnormal EEGS and CNS changes charecterised by higher excitation state of the CNS and in some cases polyneuropathy.

Other systems that could be effected by organophosphorous intoxication include liver (Gomes *et al.*, 1999), immune system (Aly and El-Gend.,2000), Kidney (Kossmann *et al.*,1997) and reproductive system (Rawlings *et al.*,1998). In addition behavioural effects (Bazylewicz-Walczak *et al.*, 1999) and physiological dysfunction (Kedzierski., 1990) detected following chronic exposure to organophosphorous pesticides. Dimethoate is one of the most important organ ophosphorous insecticides and its poisoning is usually associated with the neuromuscular transmission block in both animals and humans (De-Bleeker *et al.*, 1993; Dongren *et al.*, 1999). Immunological effects due to Dimethoate have also been reported (Institoris *et al.*, 1999). Moreover Dimethoate increased the frequency of chromosomal abbrations and sister chromatid exchanges in peripheral lymphocytes from male pesticide applicators (Rupa *et al.*, 1991). Genotoxic effects of Dimethoate were also reported in mice (Hoda and Sinha., 1993). No fenotoxic effects were observed at doses upto 30mg/kg of Dimethoate (Srivastava and Raizada., 1996). Keeping this in mind our present study was planned to explore the effect of Dimethoate a commonly used Organophosphorous pesticide on some hematological parameters of mice and to find out the inbuilt capacity of mice for Dimethoate intoxication.

MATERIAL AND METHODS

Chemicals

Dimethoate 30 EC is an organophosphorous pesticide with a chemical formulla.

$$CH_3\ NHCOCH_2SP\ (OCH_3)_2$$

It has a stomach action and a cholinesterase inhibitor. It is of low persistence in the soil, water and environment with half lives of 4-16 days, may disappear from open waters due to microbial action or chemical degradation as photolysis and evaporation (Howard, 1991). Dimethoate was dissolved and diluted to the required doses using sunflower oil as a vehicle.

Animals Grouping and Treatment

Forty adult male Swiss Albino mice were used in the experiment. Animals were divided into eight groups of 5 mice each they were maintained in the animal house on daily observations and well fed by mouse chaw under good conditions of ventilation and at room temperature of 25 ± 2^0C, relative humidity of 50 ± 15 % and a normal photoperiod of 12 hours/day. At the end of 14 days mice from exposed Groups (I, II, III & IV) were taken in batches dissected, liver & kidneys were removed and fixed for histopathological investigations using 10% neutral formal saline. Tissues were processed by routine histological techniques, sectioned at 7 µm, stained with hematoxylin and eosin. Finally, stained sections were examined under the microscope and subsequently micrographs were taken. At the end of experimental

* Corresponding author: Mudasir Yasin
Department Of Zoology Ground Floor Vigyan Bhawan Bundelkhand university Jhansi Utter Pardesh 284128

period, mice from groups V, VI, VII and VIII were taken in batches dissected and examined for histopathological investigations.

The eight groups of 5 mice each were treated according to the following schedule

Group	Treatment Schedule
GroupI(Control)	pure sunflower oil for 14 days.
GroupII(exposed)	7mg/kg bw dimethoate along with vehicle for 14 days.
Group III(exposed)	14mg/kg dimethoate along with vehicle orally for 14 days.
Group IV(exposed)	28mg/kg dimethoate along with vehicle for 14 days.
Group V(Control)	Sunflower oil for 14 days and left without any treatment for next 14 days then sacrificed.
Group VI(Metabolized)	7mg/kg bw dimethoate for 14 days and left without any treatment for next 14 days then sacrificed.
Group VII(Metabolized)	14 mg/kg bw dimethoate for 14 days and left without any treatment for next 14 days then sacrificed.
Group VIII(Metabolized)	28 mg/kg bw dimethoate for 14 days and left without any treatment for next 14 days then sacrificed.

DISCUSSION

The hematological changes found in the blood of mice included an alteration in the hemoglobin content, hematocrit value, eosinophillia, differential leucocyte count (DLC), lymphocyte count, a little variation in the erythrocyte count, mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration and ESR, which seemed to be due to malabsorption of nutrients or due to the hyperactivity of the animal. Similarly Thanvel *et al.,* (1994) reported similar results when exposing the fish *Sarotherodon mossambicius* to dimecron.While Singh *et al.,*(1994) showed that another organophosphorous insecticide formothion gave a significant increase in the total erythrocyte count and hemoglobin content in fish. The reduction in hemoglobin may be resulted from the decrease in the formation of the globulin. Also the reduction in Hemoglobin may be due to the decreased RBCs resulting from the toxicity by the dimethoate. Linman (1975) and Zahran (1997) postulated that the destructive effect of the toxic substances on the erythrocytes increased directly the catabolism of hemoglobin. RBC count varied among different groups of the dimethote treated experimental animals.

Table 1 Hematological Changes observed in Dimethoate treated experimental Groups of Mice

Parameter	Group I	Group II	Group III	Group IV	Group V	Group VI	Group VII	Group VIII
Hemoglobin	13.46±0.45	11.92±1.23*	10.55±2.11*	9.20±1.97*	13.49±0.65	12.04±0.83*	10.95±1.06*	9.70±0.55*
RBC count	10.28±0.32	9.40±0.43*	8.80±0.59*	7.98±0.54*	10.30±0.44	9.46±0.35*	8.92±0.50*	8.12±0.71*
DLC (Lymphocytes)	67.70±0.61	62.20±1.15*	54.20±2.52*	43.65±1.92***	68.18±3.06	64.80±2.11*	58.37±2.68*	47.32±1.89***
DLC (Neutrophils)	28.20±0.42	30.60±1.238*	34.10±2.10*	39.40±1.84**	27.90±0.80	29.10±1.03*	32.60±2.16*	38.50±1.90**
DLC (Eosonophils)	1.10±0.22	2.50±0.47*	2.70±0.46*	3.20±0.61*	2.02±0.38	2.40±0.27*	2.56±0.13*	2.98±0.37*
DLC (Monocytes)	2.00±0.48	4.50±1.12*	8.60±1.96*	13.10±2.06***	1.90±0.57	3.70±0.23*	6.22±0.51*	10.70±1.20****
DLC (Basophils)	0.00±0.00	0.20±0.10*	0.40±0.22*	0.65±0.08*	0.00±0.00	0.00±0.00*	0.25±0.14*	0.50±0.11*
Packed Cell volume (PCV)	50.30±1.55	45.48±2.09*	41.06±1.20*	36.98±3.10*	50.42±2.13	45.74±1.46*	41.60±2.56*	37.56±1.98*
Mean corpuscular volume (MCV)	49.10±0.76	48.55±0.42*	47.12±0.22****	46.88±0.68****	49.18±0.16	48.72±0.32*	47.57±0.21**	47.91±0.51*
Mean corpuscular hemoglobin (MCH)	13.10±0.14	12.58±0.17*	11.88±0.18*	11.40±0.34**	13.16±0.17	12.66±0.27*	12.02±0.24*	11.59±0.04**
Mean corpuscular hemoglobin concentration (MCHC)	26.74±0.44	26.18±0.52*	25.48±1.02*	24.62±0.68*	26.29±0.23	26.29±0.81*	25.66±0.16*	24.78±0.60*
Erythocyte Sedimentation Rate (ESR)	28±1.00	27±2.00	25±1.00*	22±2.00*	28±0.00	27±1.00*	26±2.00*	23±1.00*

*=Non Significant(P>0.05) ** Significant(p<0.05) ***highlySignificant(P<0.01) ****Very Highly Significant(P<0.001)

RESULTS

Hematological changes were observed in the blood of the experimental animal and showed moderate changes when compared with those from the control group (Table 1). The results revealed that Dimethoate treated mice showed significant increase in RBCs, percent monocyte , percent basophills and percent neutrophills. However a significant decrease was observed in percent lymphocytes, hemoglobin count, PCV, MCV, MCH, MCHC and ESR. The number of eosonophils was increased insignificantly.

The decrease in the total RBCs may be due to the destructive effect of the toxicant as supported by Linman (1975) or may be due to the circulating failure as a result of inability to maintain circulatory blood volume due to the decrease in the developing stages of RBCs in hemopoitic tissues (EL-Feki; 1987).The decrease in RBCs count was also found to be caused by the drugs and infections by affecting the enzyme glucose 6 phosphate dehydrogenase (Genong, 1995). An obvious increase in neutrophils followed by monocytes, paralleled with a marked drop in lymphocytic percentage. These change could be explained by some researchers which found that the increase of WBCs was mainly pointed to the elevated neutrophills (Zahran, 1997) and

monocytes showed significant increase in their percentage due to their increased phagocytic activity towards destructive RBCs or may be due to innate defense capability of mice against toxins (Uhlir, 1991). Harding and Hogland (1984) concluded that the changes in monocytes number may be due to the engagement of these cells in the phagocytic process against different antigens. The increase monocyte number may be explained by the increase in hemopoitic activity after exocytosis which was done by monocytes after the discharge of its granules to lyse the antigens extracellularly (Norman et. al., 1973; Roitt et. al., 1982 and Dolen et, al., 1992). The changes in the differential leucocyte counts may be due to the changes in the immunological parameters during toxicity with dimethoate, this increase may be due to the increase in hemopoietic activity stimulated by loss of blood cells (Awad, 1992) as a result of hemolysis of RBCs.

On the other hand the increase of neutrophills was accompanied with a highly significant decrease in lymphocytes percentage in the treated groups of mice (Zahran et. al., 1995) reported that lymphocytes play the key role in all immune reactions and is always directed against the specific foreign antigens (Toxins) lymphocytes were significantly decreased in number in response to stressful condition after antigen entrance as reported (Sovenyl et. al., 1990) or may be due to the production of specific or non-specific antibodies against different antigens. Since lymphocytes are responsible for achieving the defense mechanism introduced into the body (El-Feki, 1987). Additionally lymphocytes migrate to the site of inflammations which may be resulted due to toxic effects of dimethoate as reported by Mahmoud (1995). Therefore its percentage in blood stream and population in the thymus, spleen and lymph nodes were decreased (Zahran 1997), because the circulation is not the essential site of WBCs, but only for their passage to infected organs and their circulation with lymph by filtration via blood capillaries.

A reduction in all these parameters was observed in the treatment groups of experimental animals. The reduction in PCV may be due to the decreased RBCs resulting from the toxicity by dimethoate. The blood indices, MCV, MCH were decreased compared with controlled groups which may be due to the failure in blood osmoregulation and plasma osmolarity (Wong and Davidson, 1983). We found that one of the effect of dimethoate toxicity was the production of ertythocytes with lower MCV, MCHC. MCV and MCHC are closely related to an adequate supply of Fe to hemoglobin (Mahieu, *et al.*,2000; Rao *et al.*,1983). It is established that the target sites of metals like Bi, in proteins and enzymes are both iron sites and Zn sites (Sadler *et al.*,1999) Bi also binds to serum transferase which are functional in iron transport to cells from blood (Hongzhe *et al.*, 2003). The fall of hemotocrit (PCV) is also a reason for the decreased erythocytic number (Harris *et al.*, 2002). In our study the significant fall of hemotocrit was observed at different doses. Thus the low values of hemotocrit in experimental mice might be due to low rate of RBCs. ESR was observed to decrease non-significantly as the dosage of dimethoate was increased. However there was a non significant increase in ESR as the dosage of toxicant was stopped. Which indicates the signs of recovery in the experimental organisms. When the mice were left without any treatment for next 14 days these hematological alterations (damages) were recovered to a little extent due to the self defensing mechanism of the body of an organism, showing a little bit recovering capacity.

References

Afifi, N.A. A. Ramadan, M.I. Abd El-Aziz and E.E. Saki, Influence of dimethoate on testicular and epididymal organs, testosterone plasma level and their tissue residues in rats, *Dtsch tierärztl Wschr* 98 (1991), pp. 419–423.

Amira M.S., F. Hamadi, B. Ghazi, J. Kamel, A. Emna and E. Feriel *et al.*, Effects of dimethoate on bone maturation of young rats during the suckling period, *Pesticide Biochem Physiol* 83 (2005), pp. 132–139.

Budrea C.H. and R.P. Singh, Effect of fenthion and dimethoate on reproduction in mouse, *Toxicol Appl Pharmacol* 26 (1976), pp. 29–34.

Courtney K.D., J.E. Andrews, J. Springer and L. Dalley, Teratogenic evaluation of the pesticides baygon, carbofuran, dimethoate, and EPN, *J Environ Sci Health B* 20 (1985), pp. 373–406.

Crissman, J.W. D.G. Goodman, P.K. Hildebrandt, R.R. Maronpot, D.A. Prater and J.H. Riley *et al.*, Best practice guideline: toxicologic histopathology, *Toxicol Pathol* 32 (2004), pp. 126–131

FAO/WHO. Pesticide residues in food. Food and agriculture organization plant production and protection paper no. 62, 1985.

Farag, A.T. T. Karkour and A. El Okazy, Teratogenic effect of orally administered technical dimethoate in rats, *Birth Defects Res Part B: Dev Reprod Toxicol* 77 (2006), pp. 40–46.

GIFAP. ‘‘Guidelines for Emergency Measures in Cases of Pesticide Poisoning.’’*International Group of National Associations of Manufacturers of Agrochemical Products*, Brussels, Belgium, (1984), 48p.

Howard, P.H. (Ed.). *Handbook of Environmental Fate and Exposure Data for Organic Chemicals.*Chelsea: Lewis Publishers, Vol. 3, 1991.

IARC Monograph on the evaluation of carcinogenic risk of chemicals to man Vol. 30 Miscellaneous pesticides, International Agency for Research on Cancer. Lyon, France. (1983).

IPCS/WHO. Classification of pesticides by hazard and guidelines to classification. Switzerland: Geneva; 2001.

IPCS/WHO. Environmental health criteria monograph “Dimethoate”. Geneva; 1991.

Kidd H. and D.R. James, Editors, *The agrochemicals handbook* (3rd ed.), Royal Society of Chemistry Information Services, Cambridge, UK (1991).

Mahadevaswami M.P. and B.B. Kaliwal, Evaluation of dimethoate toxicity on pregnancy in albino mice, *J Basic Clin Physiol Pharmacol* 15 (2004), pp. 211–221

Persis, V.T., and Kalaiarasi, J.M.V. “Histopathological Responses of *Mystus vittatus* to Chronic Sublethal and Acute Lethal Toxicity of an Organophosphate Pesticide.” *J. Expt Zoo India*, 4, No. 1(2001)

Reuber, M.D. Carcinogenicity of dimethoate, *Environ Res* 34 (1984), p. 193.

Sharkoori, A.R., Alam, J. and Ali, S.S. “Toxic Effects of Talstar: A New Synthetic Pyrethroid, on Blood and Liver of Rabbit.” *Pakistan J. Zool.*, 22, No. 3 (1990), 289-300.

Sharma Yukti, Bashir Somia, M. Irshad, S. Datta Gupta and T.D. Dogra, Effects of acute dimethoate administration on antioxidant status of liver and brain of experimental rats, *Toxicology* 206 (2005), pp. 49–57.

Sharma, Y. S. Bashir, M. Irshad, T.C. Nag and T.D. Dogra, Dimethoate-induced effects on antioxidant status of liver and

brain of rats following subchronic exposure, *Toxicology* 215 (2005), pp. 173–181.

Srivastava, M.K., Raizada, R.B., Development of technical dimethoate in rats: maternal and fetal toxicity evaluation. Indian J. Exp. Biol. (1996)34 (4), 329–333.

Thangavel, P., Baby Skaikila, I. and Ramaswamy, M. ''Synergestic Effect of Dimecroncuman L. on the Histology of Liver and Brain of *Sarotherodon mossambicus*''. *Jecobio*, 6, No. 3 (1994), 173-178.

Travlos, G.S. R.W. Morris, M.R. Elwell, A. Duke, S. Rosenblum and M.B. Thompson, Frequency and relationship of clinical chemistry and liver and kidney histopathology findings in 13-week toxicity studies in rats, *Toxicology* 107 (1996), pp. 17–29

Walsh, L.P. D.R. Webster and D.M. Stocco, Dimethoate inhibits steroidogenesis by disrupting transcription of steroidogenic acute regulatory (StAR) gene, *J Endocrinol* 167 (2000), pp. 253–263

****