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# A COMPARATIVE STUDY OF ANTIOXIDANT LEVELS IN FARMERS (SMOKERS AND NON-SMOKERS) EXPOSED TO MONOCROTOPHOS

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**ABSTRACT:** Despite extensive research in understanding toxic effects of monocrotophos, some uncertainty exists. The results showed a decrease in SOD activity both in smoker and non-smoker exposed groups when compared to the control group in the present study. The mean SOD activity in non-smoker control group was  $1263\pm7.22$  and the same has decreased to  $496.0\pm9.123$  in the non-smoker exposed group. Similarly a decrease in SOD activity was observed in smoker exposed group. While SOD activity in control group is  $1258.3\pm12.79$  it has decreased to  $496.0\pm9.12$  in the smokers exposed to monocrotophos pesticide. The results showed a decrease in GPX activity both in smoker and non-smoker exposed groups when compared to the control group. The mean GPX activity in non-smoker control group was  $20.62\pm4.14$  and the same has decreased to  $10.75\pm2.87$  in the non-smoker exposed group.

Key words: monocrotophos, smokers, non smokers, antioxidant levels

## **INTRODUCTION**

The biochemical changes induced after exposure to pesticides or their active metabolites include target cell/receptor binding, protein and DNA adduct formation, and induction or inhibition of enzymes (Heinzow and McLean, 1994). Oxidative stress is induced by pesticides, either by free radicals formation or by alteration in antioxidant defence mechanisms, including detoxification and scavenging enzymes (Abdollahi et al., 2004). Oxidative stress has been reported to play an important role in the toxicity of various pesticides, including organochlorines, organophosphates (OPs) (Ranjbar et al., 2002), carbamates and pyrethroids (Kale et al., 1999). The higher oxidative stress in pesticide sprayers is evidenced by altered activities of cellular enzymes (Prakasam et al., 2001). In blood, normal erythrocyte function depends on the intactness of cell membrane which is the target for many toxic factors including pesticides. Agrawal et al., (1991) have reported that erythrocyte reduced glutathione (GSH) together with glutathione peroxidase (GPx), glutathione reductase (GR), glutathione S-transferase (GST), gamma-glutamyl transferase (GGT), superoxide dismutase (SOD) and catalase (CAT) efficiently scavenge toxic free radicals and are partly responsible for protection against lipid peroxidation due to acute/chronic pesticide exposure. Although a huge number of studies have addressed the association between exposure to pesticides and generation of oxidative stress, they are quite heterogeneous. These studies have examined different tissues - including blood and exposure doses and conditions (either acute or chronic). The available data on experimental animals (Thapar et al., 2002; John et al., 2001) and "in vitro" studies (Singh et al., 2006) indicate that the enzymes associated with antioxidant defence mechanisms are altered under the influence of pesticides. However, these studies have led to controversial results since either increased or decreased activities of antioxidant enzymes were reported. When increased activities are found they might result from an activation of the compensatory mechanism leading to the induction of free radical scavenging enzymes to counteract the oxidative stress generated by pesticides. In contrast, the decrease in antioxidant enzymes has been interpreted as an indirect inhibition of the enzymes resulting from the binding of oxidative molecules produced during pesticide metabolism. Unfortunately, scarce studies have been performed in humans long-term exposed to pesticides, some of them included very few subjects (Panemangalore et al., 1999).

### **MATERIAL AND METHODS**

In this study 600 risk prone Male subjects were randomly selected from Medak district of Andhra Pradesh during 2007-09 and 2009-10 respectively. All the selected subjects were given an informed written consent by informing the objectives of the study.

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The subjects selected for this study falls in the age group of 20-50 years and belong to the same socio economic status. 110 Agricultural workers were recruited from 600 risk prone subjects for the study. 120 Similarly age sex matched Male subjects who did not have any pesticidal exposure were also selected as controls for comparison. The entire experimental was approved by the institutional ethical committee and utmost care was taken during the experimental procedure. Blood samples were collected by puncturing the anticubital vein into evacuated tubes combining heparin solution as anticoagulant. Lipid peroxidation is assessed by spectophotometrically (Templar et al. 1999 and Bhuyan et al, 1986). Serum proteins were precipitated by adding 1ml of 10% homogenate was added to 1 ml of 20% TCA and rehomogenized using glass homogenizer. The sample was then heated in a water bath at 70°C for 10 min after which it was cooled to room temperature and centrifuged at 30,000 g for 10 minutes at  $25^{\circ}$ C. A 400µl of aliquot of the protein free supernatant was mixed with 200µl of 0.5% (w/v) aqueous TBA reagent in a test tube and covered with a glass marble and heated in a boiling water bath for 10 minutes after which the tubes were cooled to room temperature and absorbance of pink coloured trimethane condensation product was measured at 533nm in a spectrophotometer. SOD activity in the blood was determined by indirect method based on the ability of the enzyme to inhibit  $O_2^-$  dependent auto oxidation of pyrogallol (Marklund & Marklund, 1974). Glutathione peroxidase activity in the blood was measured by the method of Martinez, et al. (Martinez, et al., 1979). The method followed by Habig et al., (1974) was used for the measurement of Glutathione-S-Transferase.

## **RESULTS AND DISCUSSION**

The results on red cell SOD activity in non-smoker and smoker workers occupationally exposed to moncrotophos are presented in Tables SOD activity is expressed as U/g Hb. The results showed a decrease in SOD activity both in smoker and non-smoker exposed groups when compared to the control group. The mean SOD activity in non-smoker control group was  $1263\pm7.22$  and the same has decreased to  $496.0\pm9.123$  in the non-smoker exposed group. Similarly a decrease in SOD activity was observed in smoker exposed group. While SOD activity in control group is  $1258.3\pm12.79$  it has decreased to  $496.0\pm9.12$  in the smokers exposed to monocrotophos pesticide. The effect of duration of exposure on the SOD activity was studied both in the smoker and non-smoker exposed groups. The results clearly showed decrease in the SOD activity in the sprayers with the increase in duration of exposure .and occupational pesticide sprayers exposed to more than 15 years. There is a gradual decrease in the SOD activity is Statistically significant between control and exposed groups .The age wise distribution of SOD activity both in controls and exposed groups showed a decrease with increase in the age. While the SOD activity was 934.45  $\pm$  4.559, in the age group of 20-30 years with activity being  $868.8\pm17.3$  in the occupational sprayers of aged between 41-50 years. The results were analyzed statistically and the values of statistical analysis indicates the effect of age on SOD was statistically significant.

		Mean	<b>F-value</b>	p- value
SOD	Control	1263.2±7.22	12709.48	.000
	Non-Smokers	868.8 .± 17.03		
GPX	Control	20.62±4.14	110.540	.000
	Non-Smokers	10.75±2.87		
LPO	Control	1.918±0.508	18.513	.000
	Non-Smokers	2.237±0.493		
GST	Control	1.12±0.34	7.239	.000
	Non-Smokers	4.53±0.94		

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<b>Table-1: Antioxidant</b>	<b>Enzymatic Non-Sm</b>	okers/Control Levels	in occupational spra	ayers (Non-smokers)

#### Table-2: Antioxidant Enzymatic Levels in Occupational Sprayers of Smokers

	•	Mean	F-value	p- value
SOD	Control	1258.3±12.79	22974.118	.000
	Smokers	496.0±9.123		
GPX -	Control	16.34±3.080	16.554	.000
	Smokers	13.04±2.145		
LPO -	Control	2.823±0.393	36.011	.000
	Smokers	4.286±0.159		
GST	Control	1.12±0.66	5.11	.998
	Smokers	2.71±0.92		

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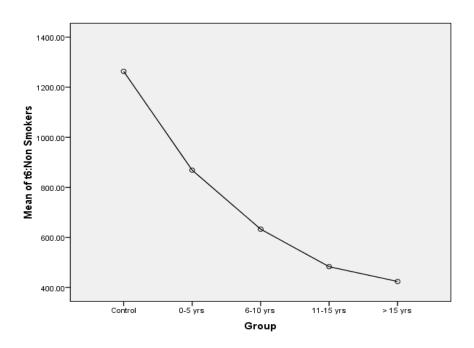
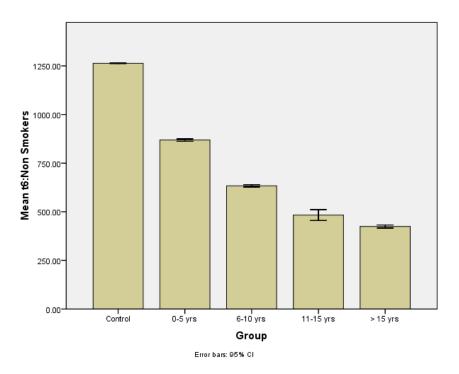
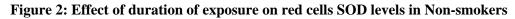


Figure 1: Effect of duration of exposure on red cells SOD levels in Non-smokers.





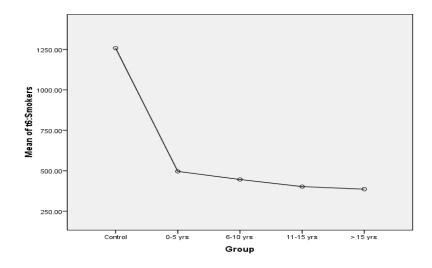


Figure 3: Effect of duration of exposure on red cells SOD levels in study group.

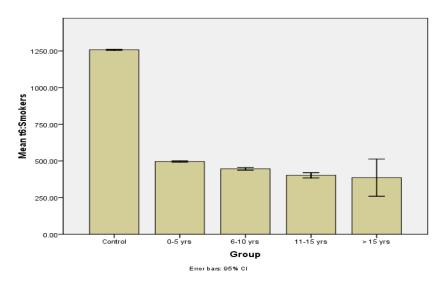
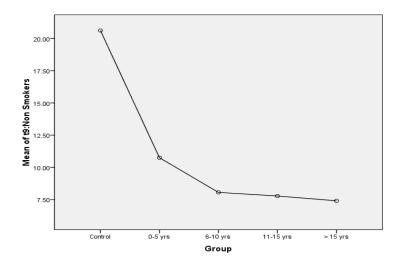
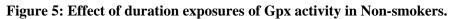


Figure 4: Effect of duration of exposure on red cells SOD levels in study group.





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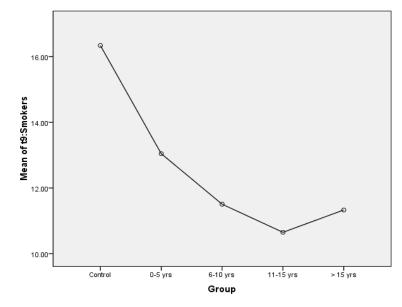


Figure 6: Effect of duration of exposure on Gpx activity in study group.

## REFERENCES

- Abdollahi, M, Ranjbar, A, Shadnia, S, Nikfar, S, Rezaiee, A, (2004). Pesticides and oxidative stress: a review. Med. Sci. Monit. '0, RA141-RA147.
- Agrawal, D., Sultana, R, Gupta, G.S., (1991). Oxidative damage and changes in the glutathione redox system in erythrocytes from rats treated with hexachlorocyclohexane. Food Chem. Toxicol. 29, 459-462.
- Heinzow, B.G.J., McLean, A., (1994). Critical evaluation of current concepts in exposure assessment. Clin. Chem. 40, 1368-1375.
- John S,Kale M,Rathore N and Bhatnagar D(2001).Protective effect of vitamin E in dimethoate and malathion induced oxidative stress in rat erythrocytes. J. Nutr. Biochem.12, 500-504.
- Kale, M., Rathore, N., John, S., Bhatnagar, D., 1999. Lipid peroxidative damage on pyrethroid exposure and alterations in antioxidant status in rat erythrocytes: a possible involvement of reactive oxygen species. Toxicol. Lett. 105, 197-205
- Panemangalore, M., Dowla, H.A., Byers, M.E., (1999). Occupational exposure to agricultural chemicals: effect on the activities of some enzymes in the blood of farm workers. Int. Arch. Occup. Environ. Health 72, 84-88.
- Prakasam A and Sethupathy S (2001).Vitamin E supplementation on biochemical changes observed in agricultural workers exposed to different classes of pesticides .Indian J.Clin.Biochem.16, 185-189.
- Ranjbar A, Solhi H, Jalali F, Susnabdi A, Rezale A and Abdollahi M (2004). Oxidative stress in acute human poisoning with org ative stress in acute human poisoning with organophosphorus insecticides; a case control study. Env Toxicol & Pharmac., 20:88-91.
- Singh,N.P.,McCoy,M.T.,R.R.and Schneider, E.L (1988). A simple technique for quantitation of low levels of DNA damage in individual cells .Exp.Cell Res.,175, 184-191.
- Thapar A, Sandhir R and Kiran R (2002). Acephate induced oxidative damage in erythrocytes .Indian J. Exp.Biol.40, 963-966.