



ISSN No: 0975-7384
CODEN(USA): JCPRC5

J. Chem. Pharm. Res., 2011, 3(6):238-242

Protective effect of *Tagetes erecta* against lead acetate induced oxidative stress in male albino rats

Reka, B* and Anuradha, R

PG and Research Department of Biochemistry, S.T.E.T. Women's College, Sundarakkottai, Mannargudi, Tamilnadu, India.

ABSTRACT

*Lead intoxication may initiate many disorders in humans and animals. This study investigated the role of *Tagetes erecta* in protecting rats against lead exposures. The results showed that the administration of *Tagetes erecta* efficiently protected albino rats against the lead caused injury, as revealed by some improvement in the enzymatic antioxidant (SOD, CAT and GPx), nonenzymatic antioxidant (GSH, Vit-E and Vit-C) activities. Thus, this suggests the possibility of *T. erecta* usefulness in limiting toxicant induced by environmental heavy metals.*

Key words: *Tagetes erecta*, Lead acetate, oxidative stress, kidney.

INTRODUCTION

Environmental pollution is the contamination of the ecosystem that causes instability, disorder, and harm on discomfort to the physical systems or living organisms. Environmental factors have important links with infectious as well as non-infectious disease of both acute and chronic nature [1]. Lead is a non-threshold multi-targeted toxicant that causes alterations in different organs of the body, including the kidney [2,3]. The absorbed lead is conjugated in the liver and passed to the kidney, where a small quantity is excreted in urine and the rest accumulates in various body organs and interferes with their functions, specially the kidney as a target site for lead toxicity [2,4]. Lead many undesired effects, including neurological [5], Behavioral [6], renal [7], hepatic [8] and reproductive disfunctions [9].

Tagetes erecta L. (Asteraceae) the Mexican marigold, also called Aztec marigold, is a species of the genus *Tagetes* native to Mexico and Central America. Despite its being native to the

Americas, it is often called African marigold. This plant reaches heights of between 50 and 100 cm [10]. Different parts of this plant including flower are used in folk medicine to cure various diseases like fever, epileptic fits (Ayurveda), astringent, carminative, stomachic, scabies and liver complaints and also employed in diseases of the eyes[11].The leaves are good for piles, kidney troubles and muscular pain. Their juice is used for earache and ophthalmia. The flower is bitter astringent,carminative, stomachic, good for the teeth and the gums; The flowers are employed in disease of the eyes and for unhealthy ulcers, internally they are said to purify the blood; their juice is given as remedy for bleeding piles [12].The study aimed to evaluate the influence of lead acetate toxicity in renal tissue of albino rats and to estimate the productive role of the ethanol extract of *Tagetes erecta* flowers against this induced toxicity.

EXPERIMENTAL SECTION

Drug and Reagents

Lead acetate was used as the nephrotoxicity inducer in animals and was procured from pharmacy at Thanjavur. All other chemicals used for the experimental were of analytical grade.

Plant material

The fresh flowers of *Tagetes erecta* was collected from the local gardens of STET Women's college, Mannargudi, Tamilnadu, India and vocchur specimens are deposited in the STET Herbarium at the Department of Botany and Microbiology, S.T.E.T. Women's College, Mannargudi, Tamilnadu, India. Measured amount of air dried powdered plant material of *Tagetes erecta* flowers were separately soxhlet extracted with 50 ml of 70 % ethanol. The ethanol mixture were evaporated at 55°C by using heating mantle, the collected extract were subjected for nephrotoxicity studies.

Animals

Male albino rats (130gm- 150gm) were purchased from animal house, Thanjavur. The rats were housed in polypropylene cages and kept under standard laboratory conditions (temperature 25±2⁰C; natural light-dark cycle). The animals were kept individually for feeding in pellet diet (Sai Durga feeds and foods, Chennai, India) with water *ad libitum*. The rat feed contained 20-21% crude protein, 4-5% ether extract, 4% crude fiber, 8% ash, 1-2% Calcium and 0-6% phosphorous.

Experimental design

In this experiment, a total of 24 rats were used. The rats were randomly divided into 4 groups of 6 rats in each group.

Group – I : Rats were kept as control

Group-II : Rats were given lead acetate 160mg/kg body weight orally for 21 days.

Group –III : Rats were given ethanol extract of tagetes erecta flowers 100mg/kg body weight orally for 21days.

Group –IV : Rats were given tagetes erecta (100mg/kg body weight) along with lead acetate orally for 21days.

Biochemical Assay

At the end of the experimental period, animals in different groups were sacrificed by direct cardiac puncture under diethyl ether anaesthesia. The kidney tissues were dissected out, weighed and washed using ice cold saline solution. Tissues were minced and homogenized (50% w/v) in

sodium phosphate buffer (0.05 M; pH 7.0) and centrifuged at $700 \times g$ for 10 min at 4°C . The resulting supernatant was used for various biochemical assays. Level of SOD was assayed by the method of Kakkar [13] and the activity of CAT by the method of Sinha [14] and the activity of GPx by the method of Rotruck [15], GSH by the method of Ellman [16]. Vitamin C was measured according to the method of Roje [17] and Vitamin E in tissues by the method of Zaspel and Csallany [18].

Statistical analysis

Data were analyzed by the one way analysis of variance (ANOVA) followed by Duncan's Multiple Range Test (DMRT) using the statistics software package SPSS for Windows, V.13.0; Chicago, IL, USA.

RESULTS AND DISCUSSION

The levels of enzymatic and nonenzymatic antioxidant systems in the kidney tissue of rats treated with lead acetate were significantly ($p < 0.05$) lesser than control rats (Table 1 and Figure 1). Administration of *Tagetes erecta* to lead treated rats significantly ($p < 0.05$) increased the level of enzymatic and nonenzymatic antioxidants in tissues. In contrast, lead with *Tagetes erecta* treated rats showed a significant ($p < 0.05$) increase in the activities of these antioxidants.

Table 1: Changes in the activities of enzymatic and nonenzymatic antioxidant rats

Groups	Group I	Group II	Group III	Group IV
SOD	9.68±0.95 ^c	7.76±1.36 ^a	9.86±1.72 ^c	7.99±0.50 ^b
CAT	76.26±1.54 ^c	58.34±1.42 ^a	75.62±1.52 ^c	68.40±1.22 ^b
GPx	9.59±1.27 ^c	5.66±1.28 ^a	9.52±1.37 ^c	6.56±1.30 ^b
GSH	100.89±1.98 ^c	85.38±1.32 ^a	99.36±1.48 ^c	90.33±3.14 ^b
Vitamin E	124±3.74 ^c	87±5.09 ^a	113.25±2.06 ^b	104.25±2.5 ^b
Vitamin C	56±5.16 ^c	39±4.96 ^a	57±0.81 ^c	44.5±2.64 ^b

Values are mean \pm SD for 6 rats in each groups. ^{a-c} In each rows means with different superscript letter different significantly at $P < 0.05$ (DMRT).

SOD-Enzyme concentration required to inhibit the chromogen produced by 50 % in one minute under standard condition, CAT- μmole of H_2O_2 consumed / minute, GPx- μg of GSH utilized / minute, GSH-mg/g tissue, Vitamin-E-mg/dl tissue, Vitamin-C-mg/dl tissue

The decreased activity of SOD, CAT and GPx in kidney tissues during chronic administration of lead. This decrease could be due to a feedback inhibition or oxidative inactivation of enzyme protein due to excess ROS generation. The generation of H_2O_2 may also lead to inactivation of this enzyme [19] but the enzymatic antioxidants of SOD, CAT and GPx activities were increased in *T. erecta* treatment. This effect could be attributed to a donation of hydrogen, to an electron reduction and / or direct scavenger activities. Krinsky [20] reported that tissue GSH levels are significantly reduced by oxidative stress and proposed that impairment of antioxidant defense mechanisms could permit enhanced free radical induced tissue damage. The activity of GPx in the study were lowered on lead acetate treatment which may attributed to the unavailability of GSH. Vitamin E is one of the major chain breaking lipophilic antioxidants. It inhibits ROS-induced generation of lipid peroxy radicals thereby protecting cells from lipid peroxidation [21]. Vitamin C is a potent scavenger of reactive oxygen species in plasma and extracellular compartments of the kidney. It scavenges and destroys free radicals in combination with vitamin E and glutathione [22]. The drastic decrease of vitamin C in lead induced toxicity indicates

increased oxidative stress, free radical formation and simultaneous damage of the plasma membrane. The result of the present study indicated that the levels antioxidant enzymes got increased when the animals were administered with *T.erecta*, and *T.erecta* along with lead acetate with significant elevations in the activities of SOD, CAT, GPx, GSH, Vitamin –E and Vitamin-C in the kidney tissue when compared to those of the kidney tissue when compared to those of the unsupplemented lead acetate treated rats.

The study showed that the exposure to lead acetate could generate free radicals which resulted in the decreased level of antioxidant levels. The biochemical alterations during *T.erecta* treated in lead treated rats may be due to Presence of natural antioxidants and Free radical scavenging activity, antioxidant property and health protecting potential of *T.erecta*. Further molecular studies would be required to confirm the beneficial effects of *T.erecta* extract in lead acetate induced toxicity.

Acknowledgements

The authors are grateful to the management of STET Women's college, Mannargudi for their encouragement and support and also thankful to Dr.P.Krishnamoorthy, Assistant Professor, Dept.of Zoology for providing laboratory facilities.

REFERENCES

- [1].Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJL. *Lancet*. **2002**; 360:1347-1360.
- [2].Jarrar BM. *Ann.Saudi.Med*. **2003**; 23:10-15.
- [3].Senapati SK, Dey S, Dwivedi SK, Swarup D. *J.Ethanopharmacol*. **2001**; 76:229-232.
- [4].Victory W, Miller CR,Goyer RA. *J.Lab.Clin.Med*. **1986**; 107:129-135.
- [5].Royce ES, Herdert L, Needle man E. *ATSDR*, **1990**; P:2-8.
- [6].Shafiq-ur-Rehman S. *Arch. Environ Contam. Toxicol*. **1991**; 20:527-530.
- [7].Vyskocil A, Pancl J, Tuml M , Ettlerova E, Semecky V , Kasparova L ,Lauwerys R , Bernard A. *J.Appl.Toxicol*. **1989** ; 9(6):395-400.
- [8].Honchel R , Marsano LJ, Cohen D, Shedlofsky S, McClair CJ. *J.Lab clin.Med*. **1991**;117(3):202-208.
- [9].Marchelewicz M, Protasouicki M, Rozewicka L, Piasecka M, Laszczynska M. *Folio histo chem Cytoviol*. **1993**;31(2): 55-62.
- [10].Kirtikar KR and Basu BD. *Indian Medicinal Plants*. Second edn,Vol.iii.Lalit Mohan Basu, Allahabad, Uttar Pradesh, India,**1987**; 1385-1386.
- [11].Ghani A.*Medicinal plants of Bangladesh chemical constituents and uses*, 2nd ed., Asiatic society of Bangladesh, Dhaka. **1998**; 301-302.
- [12].Venkataraman K. 20th. Ind Sci. Congress, Patna. **1993**.
- [13].Kakkar P, Das B, Viswanathan PN. *Ind J Biochem Biophys* .**1984**; 21: 130-132.
- [14].Sinha AK. *Anal Biochem*. **1972**; 47: 389-394.
- [15].Rotruck JT, Pope AL, Ganther HE. *Science*, **1973** ;179: 588-590.
- [16].Ellman GL. *Arch. Biochem. Biophys*. **1951**; 82 : 70-77.
- [17].Roje JH and Kuther CA. *J Biol Chem*.**1943**;147:399.
- [18].Zaspel BJ, Csallany AS. *Anal Biochem*. **1983** ;130;146-150.
- [19].Ohata Y, Kongo-Nishimura M, and Mastura T. *J. Pineal Res*, **2004**; 36:10-17.

[20].Krinsky NI, Russett MD, Handelman GJ, Snodderly DM. *J Nutr.* **1990**; 120:1654-1662.

[21].Esterbauer H, Gebicki J, and Puhl H. *Free Rad. Bio. Med.***1992**;13: 431-490.

[22].Inoue M. Protective mechanisms against reactive oxygen species. In: Arias IM, Boyer, JL, Chisari FV, Fausto N, Schachter D, Shafritz DA, Editors, *The liver: biology and pathobiology IV* edn. Philadelphia; Lippincott Williams and Wilkins: **2001**; 282-290.