### Available online <u>www.jocpr.com</u>

# Journal of Chemical and Pharmaceutical Research, 2012, 4(1):72-74



Research Article

ISSN: 0975-7384 CODEN(USA): JCPRC5

## Protective Effect of *Solanum nigrum* and *Solanum trilobatum* Aqueous Leaf Extract on Lead Induced Neurotoxicity in Albino mice

### Poo. Chinthana and T. Ananthi\*

PG Department of Biochemistry, S.T.E.T Women's College, Mannargudi, Tiruvarur, Tamil Nadu

#### ABSTRACT

Protective effect of an aqueous leaf extract of Solanum nigrum and Solanum trilobatum extract was examined against lead acetate Swiss albino mice. The oral administration of the above extract for 30 days against lead acetate affected mice significantly increased the levels of antioxidants (SOD,CAT, GPx) and decreased the level of lipid peroxidation (LPO). The results of the present study, for the first time, provide clear evidence of defence provided by S.nigrum and S.trilobatum extracts against lead acetate induced toxicity in brains of albino mice.

Key words: Solanum nigrum, Solanum trilobatum, lipid peroxidation, lead acetate, catalase.

#### INTRODUCTION

Modernization and industrial revolution has resulted in environmental pollution which is a great cause of concern these days. There is an explosion of global awareness concerning increasing imbalances in natural ecosystem. Therefore various measures are being taken to correct the root cause of the imbalances. As one of the aspects of the body's natural ecosystem, it is interestingly being realized now that majority of the diseases or disorders are mainly due to the imbalance between preoxidant and antioxidant homeostatic phenomena in the body. Preoxidant conditions dominate either due to increased generation of free radicals.

Heavy metals produce toxicity by forming complexes or ligands with organic compounds. These modified biological molecules lose their ability to function properly and result in malfunctioning, or death of the affected cells. The most common groups involved in ligand formation are oxygen, sulphur and nitrogen. When metals bind to these groups, they might make inactive important enzyme systems or affect protein structure <sup>1</sup>.

Lead (II) acetate is a chemical compound. It is a white crystalline substance with a sweetish taste. It is made by treating litharge (Lead (II) oxide ) with acetic acid. Like other lead compounds, it is very toxic. Lead acetate poisoning leads to the accumulation of toxins in our tissues and brain, causing neurological disorders, and even can lead to autoimmune dirsorders. Lead acetate collides with tiny molecules of our body, creating new free radicals. There could be chain reaction of millions of free radicals produced form one impact. Lead acetate binds with sulphur groups or proteins and inactivates them. Lead suppresses neuron clusters in the brain, hindering brain development in children by stunting the mapping of sensory nerves <sup>2</sup>. Now many attempts have been made to overcome heavy metal poisoning.

Herbs are staging a comeback and herbal renaissance is happening all over the globe. The herbal products today symbolize safety in contrast to the synthetics that are regarded as unsafe to human and environment. Although herbs had been priced for their medicinal, flavoring and aromatic qualities for centuries, the synthetic products of the modern age surpassed their importance, for a while. However, the blind dependence on synthetics is over and people are returning to the naturals with hope of safety and security <sup>3</sup>. The present study has been undertaken to investigate the protective effect of *Solanum nigrum* and *Solanum trilobatum* against lead induced neurotoxicity in albino mice.

#### **EXPERIMENTAL SECTION**

#### **Extraction of plant materials**

The dried leaves of both *Solanum nigrum* and *Solanum trilobatum* were procured from the local market and was shade dried for seven days. The dried leaves were extracted by soxhlet extractor using different solvents. Ethanol, Choloroform and aquous solvents were used for extraction. The aqeous extract was chosen for pharmacological study and other solvent extracts were used for phytochemical study.

#### **Qualitative Phytochemical Screening**

Phytochemical screening of the plant extract was carried out as per the standard methods and tests given to decipher the presence or absence of various phytocompounds <sup>4</sup>.

#### Pharmacological studies

Albino mice of both sexes weighing about 25-30 grams were used as experimental animals. They were reared in a well ventilated animal room. Separate poly propylene cages covered with stainless steel meshes were used for rearing. The animals were acclimatized in laboratory condition for 10 days and were fed with normal rodent diet (pellet diet), water was given *ad libitum*. After complete acclimatization the animals were primarily grouped 6 groups each containing 6 animals.

#### **Experimental Design**

Group- I served as control; Group- II served as an experimental control (Lead acetate induced); Group –III contains animals treated with aqueous leaf extract of *Solanum nigrum*; Group –IV contains animals treated with aqueous leaf extract of *Solanum rilobatum*; Group – V contains animals co- treated with aqueous leaf extract of *Solanum nigrum* and lead acetate; Group- VI contains animals co- treated with aqueous leaf extract of *Solanum trilobatum* and lead acetate.

The treatment for the animals belonging to group III and group IV were carried out for 30 days and the dosage of the extract fed was 200mg/kg body weight. The animals of group I and II were fed with normal diet during the treatment period. The animals belonging to group V and VI treated with plant extract and lead acetate simultaneously for 30 days. The dosage of lead acetate concentration was 10mg/kg body weight. Then during 31<sup>st</sup> day the animals were sacrificed after 6 hours of fasting. Blood was collected, serum separated and brain dissected out washed in saline and preserved in 10% formalin for histological studies and part of it was homogenized with suitable buffer for tissue antioxidant enzymes analysis.<sup>5, 6, 7, and 8</sup>

#### **Statistical Analysis**

Data for protective effect was expressed as Mean  $\pm$  S.D from 6 rats in each group. The protective effect were analyzed statistically using one way analysis of variance (ANOVA) followed by Tunkey's multiple comparison test. The minimum level of significance was fixed at p $\leq$ 0.05.

#### **RESULTS AND DISCUSSION**

Phytochemical screening of various extracts of *Solanum nigrum* and *Solanum trilobatum* revealed the presence of secondary metabolites such as steroids, triterpenoids, flavonoids, tannins, sugars. (Table. I).

Solvents used	Solanum nigrum	Solanum trilobatum		
Choloroform	Sugars, tanning ganoning storoids tritornonoids	Steroids, triterpenoids, sugars, Reducing		
	Sugars, taminis, saponnis, steroids, interpenoids	sugars, tannins		
Ethanol	Tannins, amino acids anthroquinones, triterpenoids,	Triterpenoids, phenolic compounds,		
	sugars, phenolic compounds	tannins, anthroquinone, amino acids		
Water	Sugars, phenolic compounds, flavonoids, saponins,	Saponins, tannins, anthroquinones		
	tannins			

#### Table:1 Results of phytochemical screening of Solanum nigrum and Solanum trilobatum

In the present study, treatment with *Solanum nigrum* and *Solanum trilobatum* extracts elevated the antioxidant enzyme levels thus exhibited cytoprotective effect in the brain tissue. Aqueous extracts of these plants significantly inhibited lead induced lipid peroxidation in vivo and superoxide generation in vivo<sup>9.</sup>

The levels of Catalase, superoxide dismutase and Glutathione peroxidase were found to be decreased very much in case of lead induced heavy metal toxic groups when compared to the treatment groups GV and GVI. The results

obtained were quite similar to that of a protein isolated from *Cajanus indicus* that protected thio acetamide included cytotoxicity in neuronal cells because of free radical scavenging activity<sup>10</sup>. *Solanum trilobatum* is rich in antioxidants and its effect is much greater when compared to *Solanum nigrum*.

Table:2 Changes in the level of	f antioxidant enzymes in control and	other experimental groups
---------------------------------	--------------------------------------	---------------------------

Groups	SOD (mg/Protein)	Catalase(mg/Protein	GPx (nmol NADPH consumed min–1 mg/Protein)	LPO (nmol malondialdehyde mg /protein 30 min-1)
Group I	2.90±0.12	35.10±0.02	10.02±0.27	16.05±0.56
Group II	$1.80\pm0.08$	23.07±1.04	6.88±0.72	$9.65 \pm 0.22$
Group III	2.73±0.14*	35.06±0.09**	9.56±0.23*	14.32 ±0.68*
Group IV	2.77±0.18*	35.82±0.02**	9.73±0.05*	$14.55 \pm 0.01*$
Group V	2.59±0.70**	32.61±0.18*	9.49±0.81*	14.26± 0.16*
Group VI	2.61±0.20*	33.11±0.23*	9.69±0.65*	15.09± 0.25*

Values are expressed in mean  $\pm$  SD, Values are statistically significant at \*  $P \le 0.05$  as compared with group II significant \*\* Not significant ( $P \ge 0.05$ )

#### CONCLUSION

The present findings demonstrated the antioxidant effect of *Solanum nigrum* and *Solanum trilobatum* aqeous leaf extract against lead induced heavy metal toxicity. According to traditional indigenous medicinal systems of India. These plants have got several medicinal effects without producing any severe side effects. In the absence of any reliable modern medicine these plant could be very well used neuroprotectant against lead induced toxicity. However this is only a tentative research and more work is to be done to identify the extract phytochemical responsible for this curative effect.

#### REFERENCES

[1] Ferner DJ. Heavy metals Toxicity. Emedical Journal. 2(5); 2001:1-3

[2] Minnema D. Neurotoxic metals and Neuronal Siginalling process in the volnacube brain and environmental risks, V(2).

[3] Kritikar KR, Basu BD and Chopra Indian Medicinal Plants, Oriental Enterprises, Dehradun.

[4] Trease GE and Evans WC. Pharmacology 11<sup>th</sup> Ed. Bailliere Tindall Ltd, London. **1978**: 60-75.

[5] Claiborn A. Catalase activity. In Green Wald RA ed. Handbook of methods for oxygen radical research. Boca Raton, FL: CRC press. **1985**: 283-84.

[6] Mohandas J, Marshal JJ and Duggin GG. Cancer research. 44;1984: 86-91.

[7] Utely HC, Bernehein F and Hochslein P. Achieves of Biochemistry and Biophysics. 118; 1967: 29-32.

[8] Beauchamp C and Fridovich I. Annals of Biochemistry. 44: 1971: 276-87.

[9] Sini S and Devi KS. Pharmaceutical Biology. 42(6); 2005: 462-466.

[10] Sarkar K, Ghosh A and Sil P. *Hepatol. Res.*, 33; **2005**: 39-49.