



E-ISSN: 2278-4136
P-ISSN: 2349-8234
JPP 2019; SP5: 327-329

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(Special Issue- 5)
International Conference on
“Food Security through Agriculture & Allied Sciences”
(May 27-29, 2019)

Effect of *Withania somnifera* powder against lead-induced toxicity in albino rats

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Abstract

This work has been conducted to evaluate the effect of *Withania somnifera* (WS) powder on Lead (Pb) Induced toxicity in Wistar albino rats. Twenty four albino rats were divided into group I as control; Lead Acetate (PbAc) 1000mg/kg was orally given to group II, and group III was orally treated with *Withania somnifera* powder (500mg/kg) along with lead acetate (1000 mg/ kg). Oral administration of PbAc for 28 days resulted in a significant decrease in TEC, TLC and Hb%, significant increase of AST, ALT, ALP, BUN and serum creatinine and lead accumulation in tissues. There was significant decrease in total serum protein and serum albumin level after oral administration of PbAc to the rats. Treatment with WS powder @ 500 mg/kg significantly increased the reduced level of TEC, TLC and Hb% and restored altered haematological levels as compared to PbAc treated group. It also significantly decreased the elevated level of AST, ALT, ALP, BUN and serum creatinine. It simultaneously increased the level of total serum protein and total albumin. There was no significant differences in lead concentration in tissues when compared with group II. The study concludes that supplementation of powder of *Withania somnifera* daily P.O for 28 days has provided mild protection against lead induced toxicity.

Keywords: Lead, serum bio-marker, *Withania somnifera* powder, rats

Introduction

Heavy metal intoxication is great threat for environment and population. Lead is considered as one of the most hazardous and cumulative environmental pollutants that affect all biological systems through exposure from air, water and food sources. Lead is also known as “Salt of Saturn”, Plumbous acetate, neutral lead acetate and acetic acid salt. Many people who are exposed to gasoline, paints and exhaust fumes from automobile through inhalation, oral or dermal route have suffered from lots of health problems (Ademuyiwa *et al.*, 1994) [1]. Lead is known to induce a broad range of physiological, biochemical, and behavioural dysfunctions in laboratory animals and humans (Goyer, 1996) [7] including central and peripheral nervous systems (Bressler *et al.*, 1991) [5], cardiovascular system (Khalil-Manesh *et al.*, 1993) [11], hemopoietic system and kidneys (Baykov *et al.*, 1996) [4], liver (Sharma *et al.*, 1980) [19], and male (Lancranjan *et al.*, 1975) [13] and female reproductive systems (Ronis *et al.*, 1998) [18]. *Withania somnifera* commonly known as Ashwagandha, ‘Indian Ginseng’ or Winter cherry is an important Indian medicinal plant that has been widely used in Ayurvedic and indigenous medicine for over 3,000 years. It is widely claimed to have potent immunomodulator, anti-inflammatory, antibacterial, neuroprotective, aphrodisiac, sedative, rejuvenative (Mirjalili, *et al.* 2009) [14], antioxidant properties and possess free radical scavenging activity (Panda and Kar, 1997) [15].

This study has been conducted to evaluate the protective activity of *Withania somnifera* plant powder against lead induced toxicity in wistar rats.

Materials and Methods

Twenty four Wistar Albino rats were grouped as control group -I, group -II rats were given Lead acetate (PbAc) @ 1000 mg/kg daily oral for 28 days and group -III rats were treated with *Withania somnifera* powder @ 500 mg/kg along with PbAc @ 1000 mg/kg daily oral for 28 days. Lead acetate is dissolved in distilled water and given orally, root powder of *Withania somnifera* was used along with gum acacia for oral administration.

Haematological parameters *viz.*, Total erythrocyte count (TEC), Total leucocyte count (TLC) and haemoglobin % (Hb%) were estimated, ALT (Alanine amino transferase), AST (Aspartate amino transferase), ALP (alkaline phosphatase), BUN (blood urea nitrogen) and creatinine were analysed. Total Pb concentration in tissues (liver, kidney, heart, brain and lungs) were quantified in AAS. Statistical analysis was done by ANOVA using SPSS software version 17.0.

Results and Discussion

There was significant reduction in the TEC, TLC and Hb% in Group – II as compared with control Group – I (Table 1). In Group – III there was significant increase in the altered levels of TEC, TLC and Hb% as compared to Group –II. The results obtained in the present study were in agreement with the results by Ibrahim *et al.* (2012) [10]. In the present experiment the haematological alteration might be due to effect of lead on activity of γ - aminolevulinic acid dehydratase (ALAD), key enzyme of haem synthesis. Moreover, lead also inhibits the conversion of

coproporphyrinogen III to protoporphyrin IX leading to reduction in haemoglobin production and shorten life span of erythrocytes (Klassen *et al.* 2001) [12]. The increase in altered haematological levels in Group- III as compared to Group –II might be due to protective activity of *Withania somnifera*. There was significant increase in ALT & AST level in PbAc treated Group–II as compared to control Group (Table 1). The results obtained in the study were correlating with the results of Ibrahim *et al.* (2012) [10]. Gutierrez *et al.* (1992) [8] found that LC₅₀ of lead acetate (100 μ mol) caused significant leakage of ALT & AST into the medium. This indicated that leakage of cytoplasmic enzymes as indicator of cellular injury produced by heavy metals. Also a significant increase in ALP level in group - II was observed in the present study. Similar finding was observed by Suradkar *et al.* (2009) [22] and Sujatha *et al.* (2011) [21]. There was significant decrease in ALT, AST & ALP levels when co-treated with PbAc and WS in group– III (Table 1) may be due to potent hepatoprotective and nephroprotective activity of *Withania somnifera* (Harikrishnan *et al.* 2008) [9].

Table 1: Effect of treatment groups on various parameters (Mean \pm SEM) (n=8)

Parameters	Group I	Group II	Group III
TEC (10 ⁶ / μ l)	8.48 \pm 0.37	6.33** \pm 0.18	7.46 \pm 0.45 ^{NS}
TLC (10 ³ / μ l)	10.06 \pm 0.35	8.36** \pm 0.39	9.79 ^{A*} \pm 0.40
Hb (mg %)	15.44 \pm 0.25	10.65** \pm 0.68	11.39 ^{A*} \pm 0.46
ALT (I.U/L)	38.74 \pm 1.91	73.90** \pm 1.76	52.46 ^{A**} \pm 1.81
AST (I.U/L)	182.13 \pm 4.11	225.18** \pm 3.38	193.37 ^{A**} \pm 1.53
ALP (I.U/L)	119.97 \pm 1.09	182.44** \pm 6.24	171.16 ^{A*} \pm 8.69
BUN (mg/dl)	24.24 \pm 1.41	51.59** \pm 3.58	35.57 ^{A*} \pm 1.72
Creatinine(mg/dl)	0.37 \pm 0.02	1.22** \pm 0.03	0.78 ^{A**} \pm 0.02
Total Protein (g/dl)	5.78 \pm 0.51	2.41* \pm 0.24	2.51 \pm 0.39 ^{NS}
Serum Albumin (g/dl)	3.22 \pm 0.14	2.18* \pm 0.28	2.93 \pm 0.14 ^{NS}
Liver (ppm)	0.08 \pm 0.02	34.76** \pm 2.54	31.79 \pm 2.47 ^{NS}
Kidney (ppm)	0.13 \pm 0.02	64.38** \pm 2.45	60.0 \pm 3.09 ^{NS}
Heart (ppm)	0.07 \pm 0.01	35.14** \pm 1.24	31.40 \pm 1.95 ^{NS}
Brain (ppm)	0.08 \pm 0.02	36.21** \pm 1.92	31.91 \pm 2.86 ^{NS}
Lung (ppm)	0.07 \pm 0.01	33.20** \pm 1.80	31.47 \pm 1.04 ^{NS}

^{A*} $P < 0.05$, ^{A**} $P < 0.01$; statistically significant when compared to group – II.

^{**} $P < 0.01$: statistically significant when compared to control group - I.

^{NS}: statistically non-significant when compared with group – II.

An increased level of BUN and creatinine in group – II (Table 1) was observed as compared to control group. Similar results were observed by Sujatha *et al.* (2011) [21] and Ahmod *et al.* (2011). The increase of creatinine concentration might be due to loss of 50% of kidney function and considered as functional evidence of lead induced nephrotoxicity (Qu *et al.* 2002) [17]. This may be attributed to the mechanism of action of lead-induced kidney damage due to increased production of reactive oxygen species (Upasani and Balaraman 2003) [23]. Harikrishnan *et al.* (2008) [9] reported the reduced BUN and creatinine levels after oral administration of *Withania somnifera* indicating the nephroprotective effect against toxicants.

A decreased level of both total serum protein and serum albumin in group-II was observed as compared to control group (Table 1). The results were in correlation with the results obtained by Suradkar *et al.* (2009) [22] and Ibrahim *et al.* (2012) [10]. This alteration in protein patterns which might be due to binding of lead to albumin (Stone and Soares, 1976) [20]. Hypoproteinaemia with simultaneous reduction in serum albumin level in lead-induced animals was likely to be due to

marked destruction and disintegration of parenchymatous tissues. The increase level of total serum protein and serum albumin in group III might be due to hepato and nephro-protective effects on liver and kidney respectively.

In the present study, there was significant increase in Pb concentration level in liver, kidneys, heart, brain and lungs in group-II as compared to control group. The result correlated with previous studied by Patra and Swarup, (2000) [16] and Ghoniem *et al.* (2012) [6]. Akan *et al.* (2010) [3] reported the concentrations of all the metals including Pb was increased in the liver, kidney and meat of beef, mutton and chevon. *Withania somnifera* evoked a significant amelioration due to only of its antioxidant activity.

The study concluded that exposure to lead for 28 days causes altered metabolism in the body. The altered serum bio-marker levels will be responsible for the hepatotoxic and nephrotoxic actions of lead. Treatment with plant powder of *Withania somnifera* @ 500 mg/kg showed mild protective effect against lead toxicity by reducing only the increased levels of ALT, AST, BUN and creatinine indicating its protective activity, without restoring other parameters.

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