ASSESSMENT OF LIPID PEROXIDATION AND ANTIOXIDANT ACTIVITY IN *Leucas aspera* ON ALUMINIUM TOXICITY

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ABSTRACT

In the present study to investigate the lipid peroxidation and antioxidant activity of Leucas aspera in aluminium toxicity in rats. Lipid Peroxidation, Catalase, Hydrogen peroxide oxidoreductase Superoxide dismutase, glutathione S-transferase activities, Reduced Glutathione, Vitamin – C content in the tissue samples were analysed. Exposure to aluminium has been found to cause alteration in antioxidants parameters studied in the present investigation indicating the possible adverse impacts. Simultaneous treatment with aqueous or alcohol extract of the leaves of Leucas aspera has restored the antioxidants in most instances. This includes their beneficial influence on antioxidant to nullify the adverse effects of aluminium on tissue antioxidants changes.

Keywords: *Leucas aspera,* Aluminium, Antioxidants, Free radicals

INTRODUCTION

Excessive free radicals produced in the tissues are detrimental to normal functioning and forms the basis for many pathological alterations and tissue damage. Aluminium is reported to induce oxidative stress in cultured rat hippocampal neurons and reduce their survival (Xie *et al.,* 1996). Assessment of free radicals production in excess beyond the scavenging capability of antioxidants present in the tissue investigated by estimating the extent of by lipid peroxidation has shown the impact of aluminium to vary in different tissues studied. In liver lipid peroxidation was not increased during exposure to aluminium as well as treatment with extracts. In fact a decrease though not statistically significant has been observed during treatment with extracts.

Aluminium is the third most common element of the earth's crust. Much of the aluminium-containing compounds in the environment exist in insoluble forms and are not readily available to biotic species (Clauberg and Joshi, (1993). It exists as tightly bound to oxygen and silicon as aluminosilicate in the earth's crust. It is a chemically reactive metal and does not occur naturally in the elemental form (Wisniewski et al. 1986). In the biosphere it exists in forms of low bioavailability to biological species. This general low bio-availabiliy and the presence of physiological barriers in organisms restrict the deleterious interactions of

aluminium within biological systems. In the present study to investigate the lipid peroxidation and antioxidant activity of *Leucas aspera* in aluminium toxicity in rats.

MATERIALS AND METHODS

Animals

Both male and female Wistar albino rats weighing about 120–150g have been use for the study and procured from Central Animal House Facility (CAHF) of Dr. ALM.P.G.IBMS, University of Madras, Taramani campus, Chennai, India. The animals were housed in autoclavable polypropylene cages over husk beddings under controlled environment: Temperature (23 ± 4) and Humidity (50-70%) in a 12h light and dark cycle. The rats were fed with a commercial pellet diet (M/s Hindustan foods Ltd., Bangalore, India) and water available ad libitum.

Group I : Administered with 1 ml of double distilled water by oral route.

Group II : Received aluminum chloride alone (300 mg/kg body weight)

Group III : Received aqueous extract alone (400 mg/ kg body weight) dissolved in water daily by oral route.

Group IV : Received alcohol extract alone (400 mg/ kg body weight) dissolved in water daily by oral route.

Group V : Received aluminum chloride (300 mg/kg body Weight) +aqueous extract (400 mg/ kg body weight) dissolved in water daily by oral route

Group VI : Received aluminum chloride (300 mg/kg body Weight) + Alcohol extract (400 mg/ kg body weight) dissolved in water daily by oral route

Tissue homogenate

Immediately after blood collecting, the animals were sacrificed by cervical dislocation and the liver was dissected out, washed with ice-cold physiological saline. The required amount was weighed and homogenized using a Teflon homogenizer. Tissue homogenate was prepared in 0.1 M Tris Hcl buffer (pH 7.4) and used for the estimation of various biochemical parameters.

Biochemical estimation

The status of Lipid Peroxidation in tissue homogenates was estimated according to the method of Ohkawa *et al*, (1979). The activity of Catalase (Hydrogen peroxide: Hydrogen peroxide oxidoreductase - EC.1.11.1.6.) in the tissues were estimated by the method of Sinha, (1972). Superoxide dismutase activity in the tissue homogenate was estimated by the method of Marklund and Marklund, (1974). The activity of glutathione S-transferase was assayed by the method of Habig *et al.*, (1974). The Reduced Glutathione content in the tissue homogenates was estimated by the method of Ellman, (1959) with slight modification (Beutler *et al.*, 1963). The Vitamin – C content in the tissue samples was determined according to the method of Omaye *et al*, (1979).

RESULT

Table 1a: Effect of *Leucas aspera* and aluminium on Tissues Lipid peroxidation (LPO) in *Wistar albino* rats (ηM of MDA formed /mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	90.64 ± 20.79	85.36 ± 18.75	77.55 ± 19.03	72.88 ± 15.42	94.55 ± 21.70	64.28 ± 20.83
2. Kidney	104.85 ± 28.25	107.27 ± 24.02	74.23 ± 22.25	76.38 ± 14.53	92.16 ± 11.48	69.78 ± 16.37
3. Heart	169.82 ± 28.67	84.21*** ± 17.61	69.08*** ± 19.35	90.69*** ± 24.15	83.69*** ± 24.07	57.24 ± 24.70
4. Lung	119.85 ± 15.66	146.98 ± 21.40	68.37** ± 19.00	87.67 ± 21.14	58.27*** ± 24.94	97.19 ± 18.35
5. Spleen	122.29 ± 26.70	126.38 ± 27.46	112.44 ± 15.80	89.97 ± 16.45	114.14 ± 23.42	82.91 ± 21.82
6. Brain (Cortex)	101.14 ± 21.10	188.02*** ± 30.64	187.28*** ± 22.36	123.46 ± 28.41	131.19 ± 21.69	201.17*** ± 26.16
7. Blood Serum	99.04 ± 8.87	49.23*** ± 16.86	43.78*** ± 14.86	18.37*** ± 3.16	33.22*** ± 9.50	38.03*** ± 15.19

Table 1b: Effect of *Leucas aspera* and aluminium on Tissues Lipid peroxidation (LPO) in *Wistar albino* rats - 90 Days study (Percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	-5.83	-14.45	-19.59	+4.32	-29.08
2. Kidney	-	+2.30	-29.21	-27.15	-12.10	-33.45
3. Heart	-	-50.41	-59.32	-46.59	-50.72	-66.29
4. Lung	-	+22.64	-42.95	-26.85	-51.38	-18.91
5. Spleen	-	+3.35	-8.05	-26.43	-6.66	-32.21
6.Brain (Cortex)	-	+85.91	+85.18	+22.08	+29.72	+98.91
7. Blood Serum	-	-50.29	-55.80	-81.45	-66.46	-61.60

Percent change calculated by keeping the control value as 100%

Figure -1a

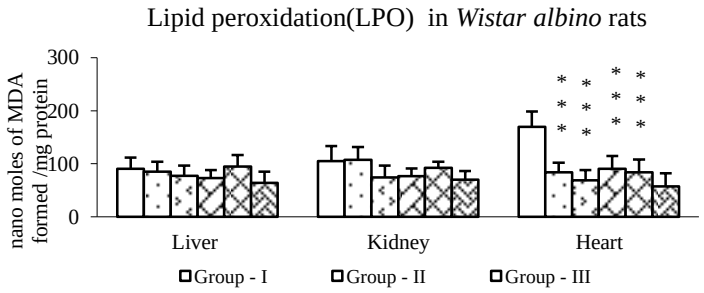

Figure -1b

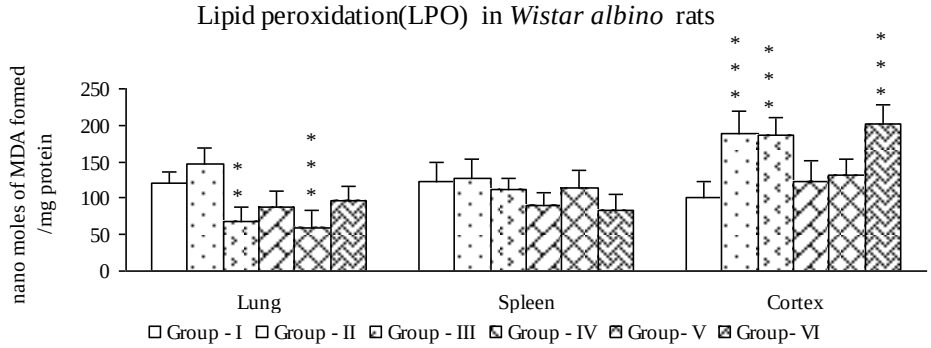

Figure -1c

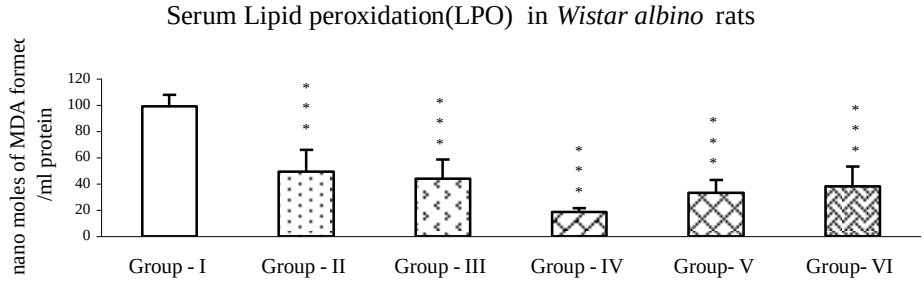

Figure -1: Effect of *Leucas aspera* and aluminium on Tissue Lipid peroxidation (LPO) in *Wistar albino* rats (ηM of MDA formed /mg protein)

Table 2a: Effect of *Leucas aspera* and aluminium on Tissues Catalase (CAT) activity in *Wistar albino* rats (µM of H_2O_2 Consumed/min /mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	27.60 ± 4.92	27.31 ± 5.60	28.65 ± 8.07	26.21 ± 5.68	25.66 ± 6.19	31.73 ± 8.69
2. Kidney	55.15 ± 15.72	36.24 ± 6.17	32.55** ± 5.70	47.40 ± 10.72	66.11 ± 14.28	35.68* ± 9.17

3. Heart	98.16 ± 20.62	122.62 ± 22.05	89.93 ± 23.84	137.28 ± 28.72	170.00*** ± 29.52	118.02 ± 12.73
4. Lung	91.05 ± 14.30	87.01 ± 17.59	71.73 ± 12.77	87.55 ± 19.64	116.05 ± 19.83	109.59 ± 19.77
5. Spleen	65.41 ± 11.24	72.34 ± 15.57	67.84 ± 15.54	68.30 ± 21.58	79.56 ± 18.32	68.02 ± 7.77
6. Brain (Cortex)	66.03 ± 19.13	102.16 ± 26.62	95.82* ± 20.80	96.20 ± 15.26	93.68 ± 14.44	178.58*** ± 20.84
7. Blood Serum	63.32 ± 14.49	50.97 ± 13.50	76.63 ± 11.35	61.80 ± 12.91	84.63 ± 15.59	87.08 ± 15.64

n=6 Mean ± SD *p<0.05 ** p < 0.01 *** p < 0.001
Compared with control group

Table 2b: Effect of *Leucas aspera* and aluminium on Tissues Catalase (CAT) activity in *Wistar albino* rats - 90 Days study (Percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	-1.05	+3.80	-5.04	-7.05	+14.97
2. Kidney	-	-34.29	-40.98	-14.06	+19.88	-35.30
3. Heart	-	+24.92	-8.39	+39.85	+73.18	+20.23
4. Lung	-	-4.44	-21.21	-3.84	+27.46	+20.36
5. Spleen	-	+10.59	+3.71	+4.42	+21.63	+3.99
6. Brain (Cortex)	-	+54.71	+45.11	+45.68	+41.87	+170.45
7. Blood Serum	-	-19.50	+21.03	-2.40	+33.66	+37.53

Percent change calculated by keeping the control value as 100%

Figure -2a

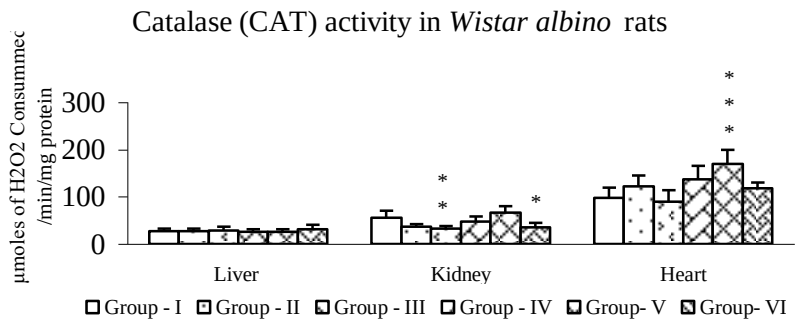

Figure -2b

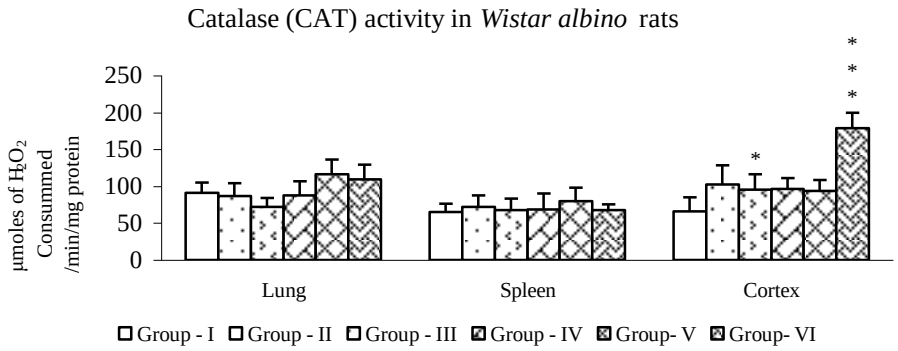

Figure -2c

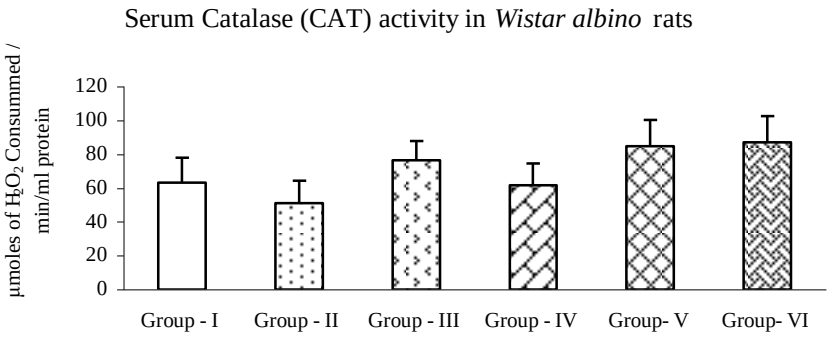

Figure -2: Effect of *Leucas aspera* and aluminium on tissue Catalase (CAT) activity in *Wistar albino* rats (µM of H_2O_2 Consumed/min /mg protein)

Table 3a:Effect of *Leucas aspera* and aluminium on Tissues Superoxide Dismutase (SOD) activity in *Wistar albino* rats (Units /mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	0.419 ± 0.100	0.321 ± 0.072	0.584 * ± 0.078	0.358 ± 0.056	0.331 ± 0.080	0.247** ± 0.081
2. Kidney	0.509 ± 0.084	0.345* ± 0.070	0.429 ± 0.137	0.291*** ± 0.084	0.337 * ± 0.053	0.229 ± 0.062
3. Heart	0.611 ± 0.114	0.421 * ± 0.103	0.550 ± 0.082	0.435 * ± 0.099	0.532 ± 0.0710	0.368*** ± 0.098
4. Lung	0.431 ± 0.064	0.354 ± 0.066	0.374 ± 0.131	0.231** ± 0.082	0.317 ± 0.089	0.317 ± 0.066
5. Spleen	0.266 ± 0.049	0.245 ± 0.034	0.281 ± 0.048	0.235 ± 0.078	0.173 ± 0.084	0.243 ± 0.053

6. Brain (Cortex)	0.216 ± 0.095	0.304 ± 0.080	0.425** ± 0.108	0.303 ± 0.072	0.185 ± 0.068	0.489*** ± 0.062
7. Blood Serum	0.474 ± 0.107	0.325 ± 0.096	0.405 ± 0.109	0.306* ± 0.050	0.394 ± 0.075	0.451 ± 0.104

n=6 Mean ± SD *p<0.05 ** p < 0.01 *** p < 0.001
Compared with control group

Table 3b: Effect of *Leucas aspera* and aluminium on Tissues Superoxide Dismutase (SOD) activity in *Wistar albino* rats - 90 Days study (percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	-23.35	+39.31	-14.60	-20.99	-40.99
2. Kidney	-	-32.19	-15.67	-42.84	-33.84	-54.95
3. Heart	-	-31.17	-10.02	-28.82	-13.04	-39.78
4. Lung	-	-17.99	-13.40	-46.54	-26.48	-26.57
5. Spleen	-	-7.99	+5.43	-11.97	-35.23	-8.63
6. Brain (Cortex)	-	+40.65	+96.60	+40.29	-14.66	+126.14
7. Blood Serum	-	-31.39	-14.71	-35.44	-16.88	-4.83

Percent change calculated by keeping the control value as 100%

Figure -3a

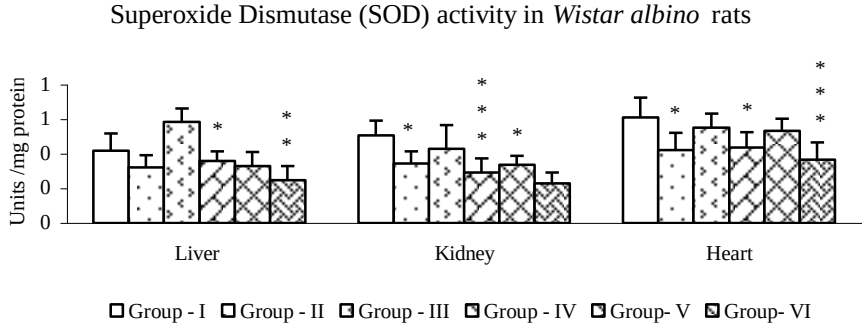

Figure -3b

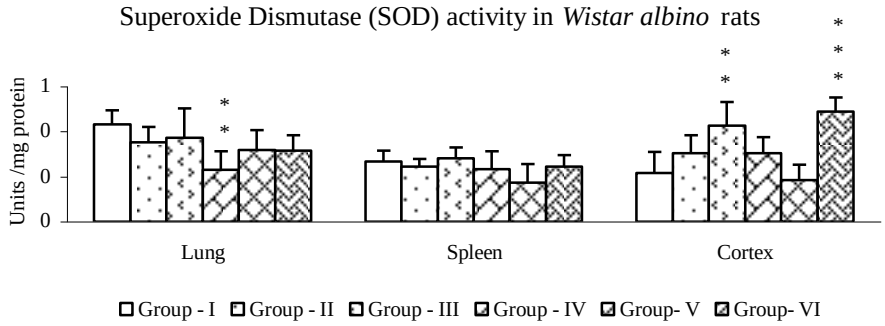

Figure -3c

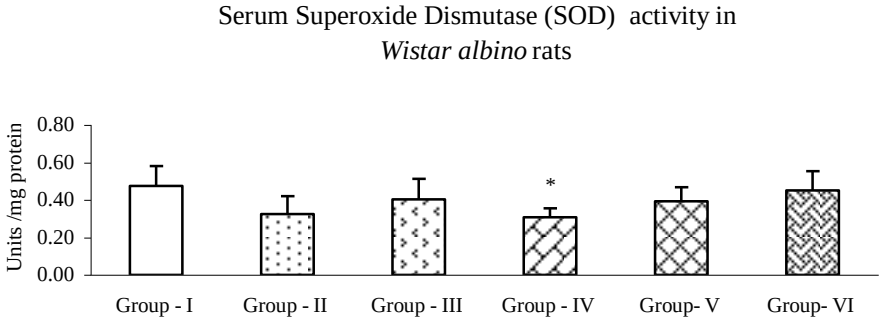

Figure -3: Effect of *Leucas aspera* and aluminium on Tissues Superoxide Dismutase (SOD) in *Wistar albino* rats activity (Units /mg protein).

Table 4a:Effect of *Leucas aspera* and aluminium on Tissues Glutathione-S-Transferase (GST) activity in *Wistar albino* rats (µM of GSH & CDNB conjugate formed/ min /mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	1.119 ± 0.350	1.040 ± 0.341	1.041 ± 0.326	1.507 ± 0.457	1.674 ± 0.436	1.944** ± 0.307
2. Kidney	0.155 ± 0.047	0.966*** ± 0.204	0.217 ± 0.075	0.339 ± 0.066	0.202 ± 0.034	0.536*** ± 0.151
3. Heart	0.439 ± 0.131	0.770*** ± 0.132	0.518 ± 0.095	0.408 ± 0.078	0.852*** ± 0.120	0.609* ± 0.122
4. Lung	1.232 ± 0.222	0.891* ± 0.232	0.404*** ± 0.149	0.870** ± 0.099	0.715*** ± 0.160	0.805** ± 0.111
5. Spleen	0.315 ± 0.076	0.553*** ± 0.133	0.309 ± 0.072	0.337 ± 0.052	0.351 ± 0.078	0.415 ± 0.115
6. Brain (Cortex)	0.180 ± 0.051	0.403** ± 0.089	0.524*** ± 0.075	0.407** ± 0.061	0.368* ± 0.100	0.652*** ± 0.143

7. Blood Serum	0.159 ± 0.023	0.461*** ± 0.138	0.152 ± 0.037	0.201 ± 0.032	0.246 ± 0.026	0.403*** ± 0.056

n=6 Mean ± SD *p<0.05 ** p < 0.01 *** p < 0.001
Compared with control group

Table 4b: Effect of *Leucas aspera* and aluminium on Tissues Glutathione-S-Transferase (GST) activity in *Wistar albino* rats- 90 Days study (Percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	-7.03	-6.91	+34.70	+49.64	+73.77
2. Kidney	-	+522.97	+40.24	+118.73	+30.20	+245.84
3. Heart	-	+75.53	+18.19	-7.01	+94.18	+38.75
4. Lung	-	-27.68	-67.24	-29.40	-42.01	-34.71
5. Spleen	-	+75.60	-1.82	+7.04	+11.56	+31.61
6. Brain (Cortex)	-	+123.79	+190.94	+125.72	+103.94	+261.67
7. Blood Serum	-	+189.46	-4.49	+26.34	+54.67	+153.13

Percent change calculated by keeping the control value as 100%

Figure -4a

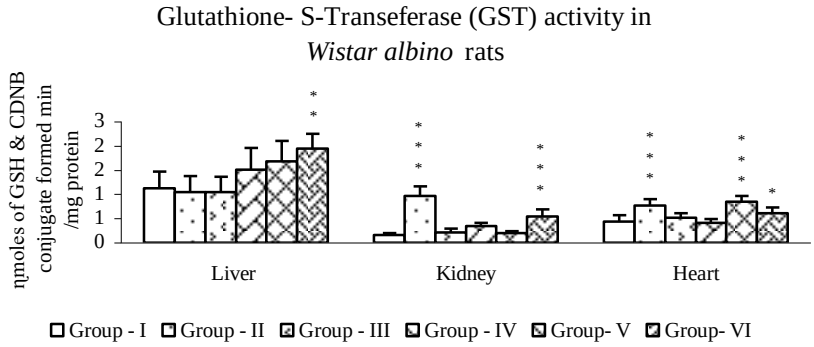

Figure -4b

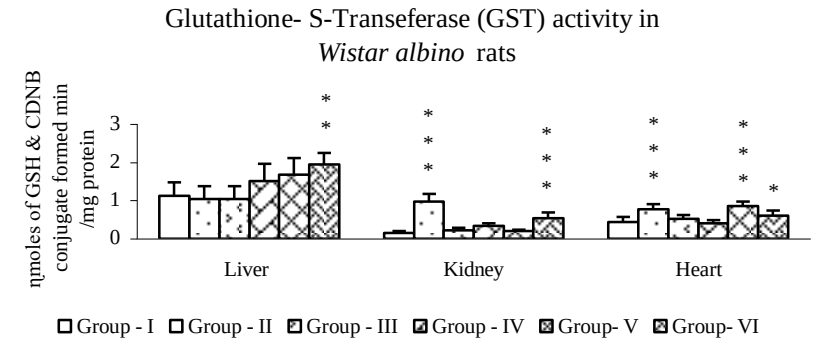

Figure -4c

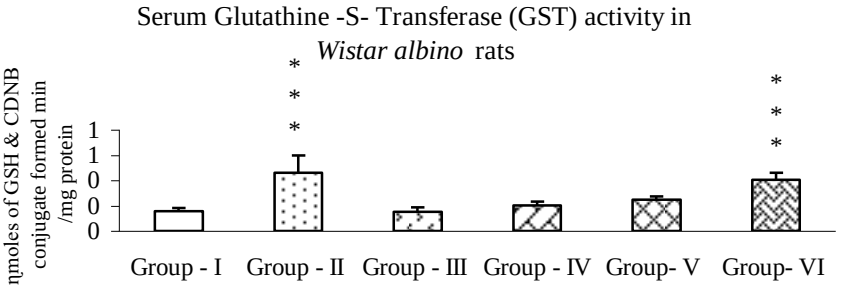

Figure -4: Effect of *Leucas aspera* and aluminium on Tissue Glutathione-S-Transferase (GST) activity in *Wistar albino* rats (µM of GSH & CDNB conjugate formed/ min /mg protein).

Table 5a: Effect of *Leucas aspera* and aluminium on Tissues Reduced Glutathione (GSH) activity in *Wistar albino* rats (µg of GSH/min /mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	5.59 ± 1.61	7.06 ± 1.33	18.22 ** ± 3.16	29.00*** ± 7.12	28.49 *** ±7.04	40.46*** ± 7.69
2. Kidney	11.19 ±2.66	13.76 ± 3.12	20.36 ± 4.03	40.89*** ± 6.38	23.40** ± 6.48	39.13*** ± 7.10
3. Heart	14.06 ± 5.26	27.71 ± 6.45	38.71*** ± 7.42	78.80*** ± 9.73	47.98*** ± 9.37	71.29 ± 9.09
4. Lung	13.01 ± 2.34	21.14 ± 6.74	25.55 ± 5.38	52.56*** ± 9.24	34.02*** ± 9.16	68.49*** ± 9.47
5. Spleen	9.95 ± 2.31	16.91 ± 3.99	19.48 ± 4.25	44.13*** ± 10.57	25.92** ± 5.47	30.76*** ± 8.54

6. Brain (Cortex)	11.40 ± 3.55	24.22 ± 10.16	34.24*** ± 10.90	11.97 ± 6.09	34.21*** ± 7.09	55.99*** ± 9.16
7. Blood Serum	14.02 ± 3.86	14.61 ± 1.68	20.29 ± 4.94	29.52*** ± 4.52	37.16*** ± 5.22	26.07*** ± 5.04

n=6 Mean ± SD ** p < 0.01 *** p < 0.001
Compared with control group

Table 5b: Effect of *Leucas aspera* and aluminium on Tissues Reduced Glutathione (GSH) activity in *Wistar albino* rats - 90 Days study (Percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	+26.43	+226.06	+419.09	+410.02	+624.16
2. Kidney	-	+22.99	+82.03	+265.52	+109.21	+249.79
3. Heart	-	+97.07	+175.32	+460.42	+241.23	+407.02
4. Lung	-	+62.47	+96.35	+303.94	+161.46	+426.38
5. Spleen	-	+69.98	+95.82	+343.62	+160.52	+209.23
6. Brain (Cortex)	-	+112.51	+200.36	+5.05	+200.12	+391.19
7. Blood Serum	-	+4.16	+44.70	+110.49	+164.99	+85.91

Percent change calculated by keeping the control value as 100%

Figure -5a

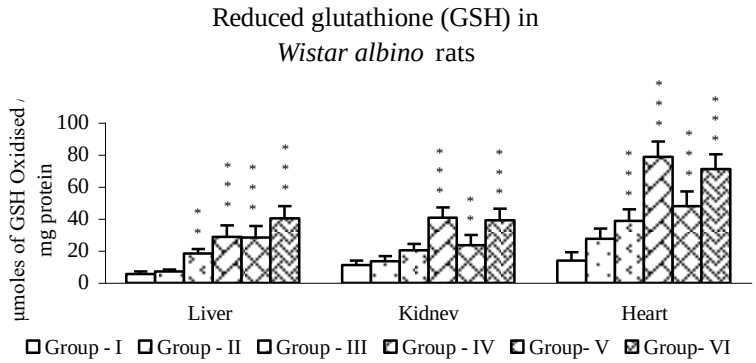

Figure -5b

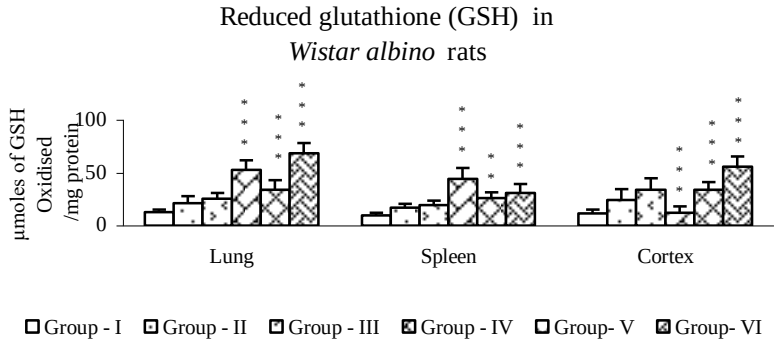

Figure -5c

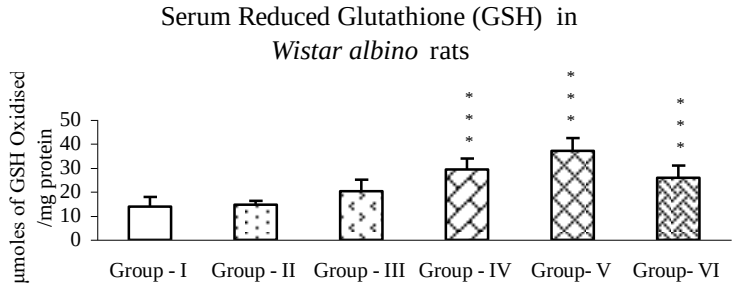

Figure -5: Effect of *Leucas aspera* and aluminium on Tissue Reduced Glutathione (GSH) level in *Wistar albino* rats (µM of GSH Oxidised /mg protein)

Table 6a: Effect of *Leucas aspera* and aluminium on Tissue biochemical Vitamin-C level in *Wistar albino* rats (µg / mg protein)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	0.379 ± 0.041	0.372 ± 0.033	0.582*** ± 0.031	0.402 ± 0.030	0.290*** ± 0.033	0.213*** ± 0.021
2. Kidney	0.262 ± 0.025	0.232 ± 0.012	0.515*** ± 0.020	0.370*** ± 0.024	0.204** ± 0.029	0.393*** ± 0.027
3. Heart	0.420 ± 0.024	0.171*** ± 0.014	0.278* ± 0.017	0.241** ± 0.025	0.156*** ± 0.024	0.436 ± 0.170
4. Lung	0.599 ± 0.020	0.888*** ± 0.015	0.638 ± 0.027	0.632 ± 0.029	0.422*** ± 0.034	0.652 ± 0.093
5. Spleen	0.892 ± 0.077	0.914 ± 0.063	0.642*** ± 0.109	0.821 ± 0.096	0.605*** ± 0.117	0.898 ± 0.093
6. Brain (Cortex)	1.303 ± 0.354	1.338 ± 0.216	1.306 ± 0.250	1.242 ± 0.138	1.053 ± 0.13	1.077 ± 0.345

7. Blood Serum	6.46 ± 1.07	6.52 ± 1.11	5.45 ± 1.24	3.09*** ± 0.55	4.93 ±0.86	5.70 ± 0.89

n=6 Mean ± SD *p<0.05 ** p < 0.01 *** p < 0.001
Compared with control group

Table 6b: Effect of *Leucas aspera* and aluminium on Tissue biochemical Vitamin-C level in *Wistar albino* rats-90 days study (Percent change)

Tissues	Group I	Group II	Group III	Group IV	Group V	Group VI
1. Liver	-	-1.77	+53.47	+6.13	-23.39	-43.77
2. Kidney	-	-11.72	+96.31	+40.92	-22.14	+49.81
3. Heart	-	-59.31	-33.68	-42.51	-62.84	+3.83
4. Lung	-	+48.19	+6.46	+5.45	-29.52	+8.75
5. Spleen	-	+2.44	-28.06	-7.96	-32.11	+0.66
6. Brain (Cortex)	-	+2.70	+0.21	-4.68	-19.17	-17.31
7. Blood Serum	-	+0.86	-15.64	-52.12	-23.68	-11.82

Percent change calculated by keeping the control value as 100%

Figure -6a

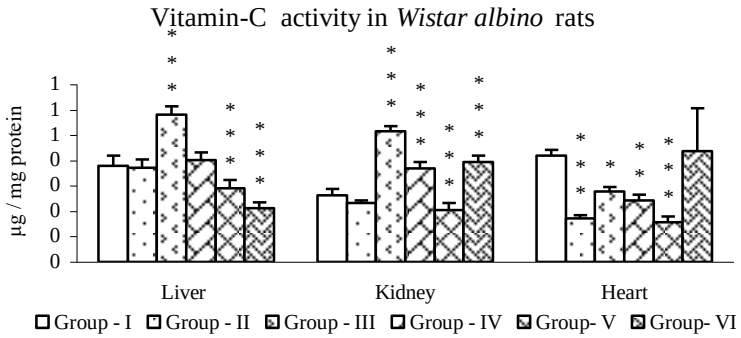

Figure -6b

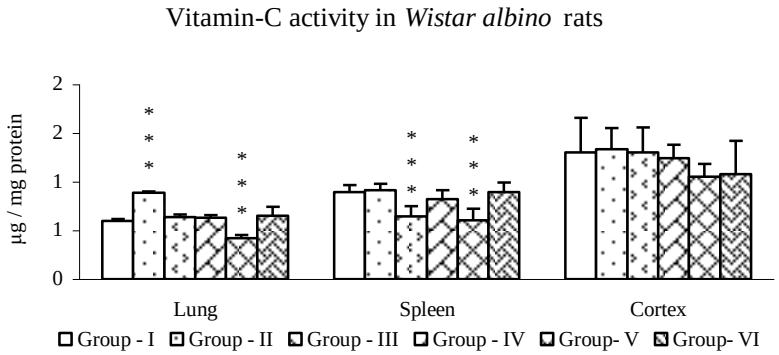

Figure -6c

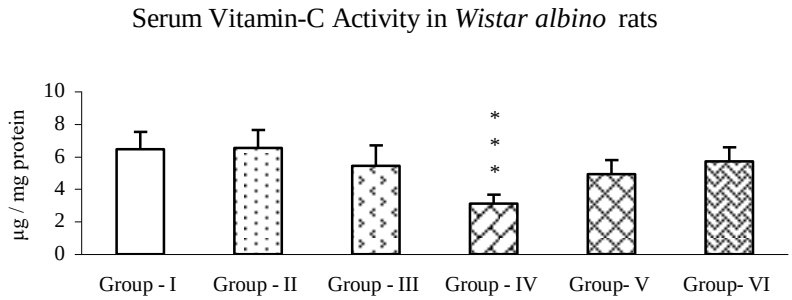

Figure -6: Effect of *Leucas aspera* and aluminium on Tissue Biochemical Vitamin-C level in *Wistar albino* rats (µg / mg protein)

DISCUSSION

Assessment of free radicals production in excess beyond the scavenging capability of antioxidants present in the tissue investigated by estimating the extent of by lipid peroxidation has shown the impact of aluminium to vary in different tissues studied. In liver lipid peroxidation was not increased during exposure to aluminium as well as treatment with extracts. In fact a decrease though not statistically significant has been observed during treatment with extracts. Free radical production in cardiac tissue seems to be lesser during exposure to aluminium as well as treatment with the extracts as there was a significant reduction of LPO during individual as well as combined treatment (El-Demerdash, 2004). One possible explanation for this could be adequate and constant perfusion of this tissue with blood.

In most of the tissues GST increased during exposure to aluminium. Both the extracts increased the brain GST and such an increase was seen in most of the tissues with administration of alcohol extract (AlE) of *Leucas aspera* leaves along with $AlCl_3$ (El-Demerdash, 2004). However a decreased was seen in lung tissue during treatment with aluminium as well as the extracts either individually or in combination.

While exposure to aluminium resulted in a decrease in SOD in most of the tissues, it has been observed to be significant in kidney and heart. This influence of aluminium was not suppressed by co-administration of the extracts (Banks and Kastin, 1983). While an increase was seen with administration of AqE in liver and brain, a decrease was seen with administration of alcohol extract (AlE) of leaves in most of the tissues. There was no significant change in reduced glutathione in tissues during exposure to aluminium while the extracts have increased it in most of the tissues. This increase was seen during combined treatment with $AlCl_3$ also (Riihimaki *et al.*, 2000). Vit C content was not altered in the tissues except an increase in lungs and decrease in heart during aluminium exposure (Clauberg and Joshi, (1993). Treatment with the extracts increased the vit C content in liver and kidney. The negative effect of alumnium on heart was inhibited by AlE. It is to be noticed that the extracts have been found to contain Vit C and the tissue content of it has been found to be increased only in liver and kidney.

As in the case of liver, LPO was not increased in the kidneys also due to treatment with AlCl3 and decrease of the same was observed during treatment with the extracts and during combined administration of aluminium and extracts. Free radical production in cardiac tissue seems to be lesser during exposure to aluminium as well as treatment with the extracts as there was a significant reduction of LPO during individual as well as combined treatment. One possible explanation for this could be adequate and constant perfusion of this tissue with blood (El-Demerdash *et al*., 2004).

Lungs seem to be vulnerable to excessive free radical production during aluminium exposure as an increase in LPO was noticed in this tissue. Treatment with the extracts resulted in decrease of LPO, and with AlE this was significantly reduced. The extracts have the potential to combat the influence of aluminium induced free radical production in this tissue as fall in LPO was noticed during combined treatment. AqE is found to be more effective in this aspect as a significant reduction was seen during combined treatment with it and AlCl3.As lungs are the place for exchange of gases especially oxygen and carbon dioxide, the free radical production due to aluminium exposure gains toxicological importance and the beneficial influence of the extracts in suppressing this effect of aluminium can have therapeutic utility (Fatma *et al*., 2004).

No significant change in tissue LPO was seen in spleen which is an important component of reticulo-endothelial system. A discernible decrease of LPO was observed in this tissue after treatment with the AlE. This effect of AlE seems to persist during exposure to aluminium as the reduction in LPO was seen after combined treatment with AlE and $AlCl_3$.

Influence of aluminium on brain has investigated by many investigators. A significant increase in LPO recorded in this tissue indicates the toxic potential of aluminium. While treatment with the AqE also resulted in a similar increase, there was no significant change after treatment with AlE. However the change induced by aluminium was reduced to conspicuous extent by AqE and not by AlE, indicating the potential of AqE to suppress aluminium induced free radical production in brain (Riihimaki *et al.*, 2000).

Serum LPO was found to be significantly decreased during exposure to aluminium as well as treatment with the extracts. The extent of decrease was more with the extracts and found to be persistent during exposure to aluminium also. Thus free radical production has been found to be increased in lungs and brain tissues, and the extracts with inherent potential to inhibit this were found to suppress the influence of aluminium.

Exposure to aluminium has not resulted in any significant change in the catalase activity in the tissues. Treatment with AqE and co-administration of AlCl3 with AlE increased the enzyme activity in brain Similarly co-administration of AlCl3 and AqE increased the catalase activity in heart. Treatment with AqE as well as co-administration of AlE along with AlCl3 resulted in a decrease in the kidney catalase. Thus the extracts caused a positive impact on catalase activity in brain and heart tissues. Though an inhibition was seen in kidney, assessment of the overall impact on tissue andtioxidants needs to be considered. In most of the tissues GST increased during exposure to aluminium. Both the extracts increased the brain GST and such an increase was seen in most of the tissues with administration of AlE along with AlCl3 . However a decreased was seen in lung tissue during treatment with aluminium as well as the extracts either individually or in combination (Banks and Kastin, 1983).

While exposure to aluminium resulted in a decrease in SOD in most of the tissues, it has been observed to be significant in kidney and heart. This influence of aluminium was not suppressed by coadministration of the extracts. While an increase was seen with administration of AqE in liver and brain, a decrease was seen with administration of AlE in most of the tissues. There was no significant change in reduced glutathione in tissues during exposure to aluminium while the extracts have increased it in most of the tissues. This increase was seen during combined treatment with AlCl3 also (Clauberg and Joshi, (1993). Vit C content was not altered in the tissues except an increase in lungs and decrease in heart during aluminium exposure. Treatment with the extracts increased the vit C content in liver and kidney. The negative effect of alumnium on heart was inhibited by AlE. It is to be noticed that the extracts have been found to contain Vit C and the tissue content of it has been found to be increased only in liver and kidney. Inferences that can be drawn from the foregoing discussion on free radical production and tissue antioxidants are: (a) the Aluminium causes an increased free radical production as exemplified by increased LPO in lungs and brain. This influence of aluminum has been found to be effectively inhibited by the extract especially AqE. (b) The increased GST seen during exposure to aluminium could be considered as a consequence to combat the free radical production. (c) However, exposure to aluminium seems to have direct suppressive impact on tissue antioxidants also as a decrease in SOD was noticed in most of the tissues, (d) Increased catalase activity in brain and heart, an increase in reduced glutathione in most of the tissues and increase in Vit C content in liver and kidney caused by the extracts indicate their possible usefulness in curtailing the effects of aluminium induced free radical production (El-Demerdash *et al*., 2004). The antioxidant activity of Leucas *aspera* due to the presence of flavonoids (Meghashri et al., 2010).

CONCLUSION

Exposure to aluminium has been found to cause alteration in antioxidants parameters studied in the present investigation indicating the possible adverse impacts. Simultaneous treatment with aqueous or alcohol extract of the leaves of *Leucas aspera* has restored the antioxidants in most instances. This includes their beneficial influence on antioxidant to nullify the adverse effects of aluminium on tissue antioxidants changes.

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