Ameliorated effect of *Ocimum sanctum* (OS) herbal leaf extract on experimental chronic lead toxicity induced residues in Wistar albino rats


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Lead that contaminates the environment is largely air-borne but is redeposited by dust in to soil and water and is taken up by or exists on the surface of plants which are grazed by livestock (Bolter *et al.*, 1975). The absorbed lead is conjugated in the liver and passed to the kidney where a small quantity is excreted in urine and rest accumulates in various body organs and interferes with their function. Because of its persistence in the environment, exposure to lead has become a major public health concern (Vaglenov *et al.*, 2001). Lead damages cellular material and alters cellular genetics and produces oxidative damage. It causes increased production of free radicals and decreased availability of anti oxidant reserves to respond to the resultant damage. The present investigation was undertaken to evaluate ameliorated effect of *Ocimum sanctum* on lead residues in different organs in wistar rats.

The present research work was carried out in the Department of Veterinary Pathology, College of Veterinary Science, Tirupati during the year 2010. This study was conducted on 216 healthy adult male Wistar albino rats weighing more than 150g. All the rats were housed comfortably in standard rat cages at 25° ±1° C and a 12:12 hour interval light/dark cycle throughout the experimental period of 12 weeks and provided *ad libitum* feed and water. After 10 days of acclimatization the animals were randomly divided into six groups. Lead acetate [(CCH\textsubscript{3}COO\textsubscript{2})\textsubscript{2}Pb 3H\textsubscript{2}O, M.W = 379.33] with a Laboratory Reagent grade procured from the Qualigens Fine Chemicals, Bombay) was given orally after mixing in double distilled water to rats at the dose rate of 1/10 LD\textsubscript{50} (60mg/ Kg b.wt/3 days in a week), 1/20 LD\textsubscript{50} (30mg/ Kg b.wt/3 days in a week) respectively to the groups II and III. In addition to lead acetate, *Ocimum sanctum* herbal product (*Ocimum sanctum* (OS) from the Natural Remedies Pvt. Ltd. Benguluru) was given orally at a dose rate of 400 mg/ Kg b.wt./ 3 days a week to the groups IV and V to study ameliorative effect. Group I and VI were kept as distilled water control and *Ocimum* control. Rats from each group were randomly sacrificed at fortnight intervals after starting the experiment *i.e.*, 2\textsuperscript{nd}, 4\textsuperscript{th}, 6\textsuperscript{th}, 9\textsuperscript{th}, 10\textsuperscript{th} and 12 weeks necropsy done and organs were collected.

At the day of each sacrifice, the pooled blood about 5ml was collected into heparinized vials for the BP\textsubscript{b} (Blood lead) estimation and tissue samples about 5g of liver, kidney, muscle and testis were collected and stored at -20° C until use. All these samples were subjected to acid digestion method as described by Kolmer *et al.*, (1951). Further all these samples were subjected to blood lead and tissue lead
estimation by atomic absorption spectrophotometer following method of Perkin – Elmer 3100 (USA), double beam AAS with hollow cathode lamp of lead.

In the present study Mean and S.E values of lead residual levels in different organs of animals of different experimental groups are given in Table 1&Fig.1-5. Dose dependent significant (P<0.05) increase was noticed in blood lead levels in lead treated groups (Group II & III) when compared to the control (Group I) and the values were dose and duration dependent. In OS ameliorated groups, a non significant decrease was noticed in blood lead level as a dose dependent manner. Upadhyay (1988) observed significant increase in blood lead level in lead intoxicated calves. Dev et al., (1990) reported a significant increase in BPb in goats fed with lead @ 27.15 mg / Kg bwt for 56 days (or) till death. The present findings might be due to release of lead from soft tissues when animals are exposed to longer period.

In the present investigation, kidneys had highest residual levels of lead followed by liver, testis and muscle in lead treated animals(Group II & III) in a dose dependent manner when compared to control group (Group I). In OS ameliorated groups (Group IV & V) a non significant decrease was observed in lead concentration in these organs. Allcroft (1950) reported higher concentration of lead in kidney and liver. Clausen et al., (1980) estimated maximum levels of lead in kidney followed by liver and blood in case of lead encephalopathy in cattle. Zmudski et al., (1983) recorded higher concentration in kidney than in liver and spleen of calves given @10 mg of lead acetate / kg bwt for 20 days. Upadhyay et al., (1990) found the highest concentration in kidney followed by liver, lung and spleen. Vandana et al., (2008) recorded highest concentration of lead in kidney, followed by liver and low levels of accumulation was found in testes and ovaries of quails in a dose dependents manner, when fed with three different doses (0.5 ppm, 1.25 ppm and 2.50 ppm) for 21 days.

In OS treated groups, non significant decrease in lead concentration in blood and tissues were noticed when compared to lead toxicated groups. This might be due to reduced lead absorption in intestine or reduced deposition of lead in different organs and enhanced excretion of lead by the kidney. No reports were available to compare these results. In farm animals major portion of the gut absorbed lead is deposited into the skeletal system. Initially it accumulated in the bone until the possible threshold was reached then it is distributed to other tissues especially kidney where turnover rate was slow. A few adverse effects of lead may be noticed when stored in bone. But when it is distributed to other system symptoms of plumbism occur (Neathery and Miller, 1975).

CONCLUSION

The present work revealed that highest lead concentration was found in kidney followed by other organs and when compared to control increased lead concentration was observed in blood of treated rats. In Ocimum treated groups, non significant decrease in lead concentration in blood and tissues were noticed when compared to lead toxicated groups. This might be due to reduced lead absorption in intestine or reduced deposition of lead in different organs and enhanced excretion of lead by the kidney.
Table 1: Mean and S.E values of lead residual levels in different organs of animals of different experimental groups

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Group IV</th>
<th>Group V</th>
<th>Group VI</th>
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<tr>
<td><strong>Blood (µg/ ml)</strong></td>
<td>0.01&lt;sup&gt;bc&lt;/sup&gt; ±0.00</td>
<td>0.08&lt;sup&gt;a&lt;/sup&gt; ±2.29</td>
<td>0.06&lt;sup&gt;a&lt;/sup&gt; ±0.01</td>
<td>0.07&lt;sup&gt;a&lt;/sup&gt; ±0.01</td>
<td>0.05&lt;sup&gt;a&lt;/sup&gt; ±0.00</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt; ±0.00</td>
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<tr>
<td><strong>Kidney (µg/ g)</strong></td>
<td>0.02&lt;sup&gt;bc&lt;/sup&gt; ±0.00</td>
<td>8.72&lt;sup&gt;a&lt;/sup&gt; ±1.87</td>
<td>6.97&lt;sup&gt;a&lt;/sup&gt; ±1.58</td>
<td>7.86&lt;sup&gt;a&lt;/sup&gt; ±1.66</td>
<td>6.00&lt;sup&gt;a&lt;/sup&gt; ±1.34</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt; ±0.00</td>
</tr>
<tr>
<td><strong>Liver (µg/ g)</strong></td>
<td>0.01&lt;sup&gt;bc&lt;/sup&gt; ±0.00</td>
<td>3.30&lt;sup&gt;a&lt;/sup&gt; ±0.602</td>
<td>2.77&lt;sup&gt;a&lt;/sup&gt; ±0.47</td>
<td>3.08&lt;sup&gt;a&lt;/sup&gt; ±0.56</td>
<td>2.40&lt;sup&gt;a&lt;/sup&gt; ±0.40</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt; ±0.00</td>
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<tr>
<td><strong>Testis (µg/ g)</strong></td>
<td>0.01&lt;sup&gt;d&lt;/sup&gt; ±0.00</td>
<td>0.50&lt;sup&gt;a&lt;/sup&gt; ±0.06</td>
<td>0.42&lt;sup&gt;ab&lt;/sup&gt; ±0.05</td>
<td>0.45&lt;sup&gt;a&lt;/sup&gt; ±0.05</td>
<td>0.36&lt;sup&gt;b&lt;/sup&gt; ±0.04</td>
<td>0.01&lt;sup&gt;cd&lt;/sup&gt; ±0.00</td>
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<tr>
<td><strong>Muscle (µg/ g)</strong></td>
<td>0.01&lt;sup&gt;bc&lt;/sup&gt; ±0.00</td>
<td>0.46&lt;sup&gt;a&lt;/sup&gt; ±0.16</td>
<td>0.34&lt;sup&gt;a&lt;/sup&gt; ±0.12</td>
<td>0.38&lt;sup&gt;a&lt;/sup&gt; ±0.13</td>
<td>0.24&lt;sup&gt;abc&lt;/sup&gt; ±0.09</td>
<td>0.01&lt;sup&gt;c&lt;/sup&gt; ±0.00</td>
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Mean values with different superscripts differ significantly (p < 0.05)
S.E – Standard error

Fig.1: Mean values of Lead residual levels in Blood (µg/ ml) in animals of different experimental groups
Fig. 2: Mean values of Lead residual levels in kidney (μg/g) in animals of different experimental groups

Fig. 3: Mean values of Lead residual levels in liver (μg/g) in animals of different experimental groups
Fig. 4: Mean values of Lead residual levels in testis (µg/ g) in animals of different experimental groups

Fig. 5: Mean values of Lead residual levels in muscle (µg/ g) in animals of different experimental groups
REFERENCES


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