Heavy metals are either simulators or inhibiting factors of life processes. The exposure of heavy metal induces the production of reactive oxygen species (ROS) such as superoxide radical, hydroxyl ion and hydrogen peroxide through oxidative stress mechanism. The assumption of oxidative stress as a mechanism in heavy metal such as lead toxicity suggests that antioxidants play an important role in therapy. Antioxidant is a substance that works in the biological systems in various ways such as removal of free radicals as scavengers or in the form of proteins that minimize the availability of prooxidant such as metal ions. Allium compounds were reported to have tremendous antioxidant property such as scavenging ROS, enhancing cellular antioxidant enzymes and helping to increase glutathione level. The present study was designed to ascertain the oxidative damages produced by lead poisoning due to daily consumption of lead acetate solution and to examine the curative effects of polar and non polar fractions of garlic and onion oils in order to combat lead toxicity in albino rats in comparison with vitamin E. Vitamin E is a nature’s major lipid phase chain breaking antioxidant that is a known standard antioxidant to protect biological membranes and lipoproteins from oxidative stress.

For the purpose of this study male albino Sprague – Dawley rats weighing 150- 200 g were housed, six per polypropylene cage and maintained under hygienic standard laboratory conditions at room temperature with 12 hour light / dark cycle. The animals were provided with pellet chow (Hindustan lever limited) and water ad-libitum. Polar and non polar fractions of garlic and onion oils, Vitamin E and lead acetate solution were orally fed to the rats at fixed doses daily by means of a stomach tube for a period as given below. Glycerol was the medium or vehicle for dissolving the oils and control groups also received the medium glycerol at the same dose. At the end of the experimental period the rats were left to fast overnight and sacrificed in the
morning and collected their blood and tissues such as liver, kidney and heart in clean dry test tubes kept in ice cold containers respectively for analysis of various parameters.

Analytical and biochemical methods used in this study were already discussed in detail in the second chapter.

**Experimental Design:**

Male albino (Sprague –Dawley) rats weighing between 150 -200g were divided into 8 groups of 6 rats each.

**Groups**

**Group I** : Normal control

**Group II** : Lead acetate control group (**Dose 10mg/kg/day**): Rats were orally fed with lead acetate for 1 month along with diet ad- libitum.

**Group III** : Lead acetate (Dose 10mg/kg/day) was orally fed for 1 month as above and left for 1 month without lead acetate administration but supplied with diet ad- libitum.

**TEST GROUPS**

**Group IV** : Lead acetate pretreated group, treated later with Polar fraction of garlic oil (**PFG**) for one month each:

Lead acetate (Dose 10mg/kg/day) was fed for 1 month and after that fed with polar fraction of garlic oil for another 1 month (Dose: 100mg/kg/day) along with diet ad- libitum.
**Group V** : Lead acetate pretreated group, treated later with Non polar fraction of garlic oil (NPFG) for one month each: Lead acetate was orally fed as above for 1 month and after that fed with non polar fraction of garlic oil (Dose: 100mg/kg/day) for another 1 month along with diet ad- libitum.

**Group VI** : Lead acetate pretreated group, treated later with Polar fraction of onion oil (PFO) for one month each

Lead acetate was orally fed as above for 1 month and after that fed with polar fraction of onion oil for another 1 month (Dose: 100mg/kg/day) along with diet ad- libitum.

**Group VII** : Lead acetate pretreated group, treated later with Non polar fraction of onion oil (NPFO) for one month each:

Lead acetate was fed as above for 1 month and after that fed with non polar fraction of onion oil (Dose: 100mg/kg/day) for another 1 month along with diet ad- libitum.

**Group VIII** : Lead acetate pretreated group, treated later with Vitamin E for one month each:

Lead acetate was fed as above for 1 month and after that fed with vitamin E (Dose: 100mg/kg/day) for another 1 month along with diet ad- libitum.
Results:

Table 1: Concentration of serum lead (Pb) is expressed as µg/ml and activities of blood δ- ALAD is expressed as IU/L

(Each value is the mean ± SD of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Lead (µg/ml)</th>
<th>δ- ALAD (IU/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>0.75±0.06</td>
<td>3.7 ± 0.33</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>1.43±0.13a#</td>
<td>2.29± 0.21a#</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>1.54±0.14a#,bNS</td>
<td>2.36± 0.22a#bNS</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>0.75±0.07bcf #,d*</td>
<td>3.49±0.32bcf#,d*</td>
</tr>
<tr>
<td>V</td>
<td>Do +NPFG</td>
<td>0.87±0.08bc#,f*</td>
<td>3.08±0.28bc#</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>0.71±0.07bcf,e•</td>
<td>3.28±0.29b,c,f#e•</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>0.89±0.08bc#</td>
<td>2.98±0.27bc#</td>
</tr>
<tr>
<td>VIII</td>
<td>Do + Vitamin E</td>
<td>0.98±0.09bc#</td>
<td>2.77±0.25bc•</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI,VII)

F value: Lead126.834, δ-ALAD 111.1

Concentrations of serum lead (Pb) and activities of δ- ALAD (Table 1) (Only significantly varied values are cited here)

Serum lead level increased very high significantly (p≤ 0.001) in rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats . There was no significant difference between the above groups II and III. On treatment with
polar and non polar fractions of garlic and onion oils or vitamin E, the groups of rats (groups IV to VIII) fed with lead acetate solution followed by treatment with the above 5 samples of drugs produced very highly significant decreases (p≤ 0.001) of serum lead level when compared to the values of the rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). However the significant difference is varying between the treated groups IV to V i.e. on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed only just significant (p≤0.05) decrease in serum lead level as compared to that of the rats fed with lead acetate solution and treated later with NPFG oil (group V). Further on treatment with PFO oil to the group of rats previously fed with lead acetate solution (group VI) showed highly significant (p≤0.01) decrease of serum lead level when compared to that of rats fed with lead acetate solution and treated later with NPFO oil (group VII). On treatment with PFG and PFO oils the groups of rats previously fed with lead acetate solution (group IV and VI) showed very highly significant (p≤0.001) decreases in serum lead level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). On treatment with NPFG oil to the group of rats previously fed with lead acetate solution (group V) showed only significant (p≤0.05) decrease in the serum lead level as compared to the rats fed with lead acetate solution and treated later with vitamin E (group VIII).

Activity of blood δ- ALAD decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month and left for another one month without lead administration (group III) as compared to that of the normal rats. Further there was no significant difference between the values of the values of the above groups II and III. Further on treatment with polar and non polar fractions of garlic and onion oils to the groups of rats previously fed with lead acetate solution (groups IV to VII) showed very high significantly (p≤
0.001) increases of blood d- ALAD activities when compared to the values of the rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). However on treatment with vitamin E to the groups of rats previously fed with lead acetate solution (group VIII) showed highly significant (p≤ 0.01) increases of blood d- ALAD activities when compared to the values of the rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil to the rats previously fed with lead acetate solution (group IV) showed only just significant (p≤0.05) increase of blood d- ALAD activity as compared to that of the rats fed with lead acetate solution and treated later with NPFG oil (group V). Further on treatment with PFG oil (group IV) to the rats previously fed with lead acetate solution showed very high significantly (p≤0.001) increase in blood d- ALAD activity as compared to that of the rats fed with lead acetate solution treated later with vitamin E (group VIII). However on treatment with PFO oil (group VI) to the rats previously fed with lead acetate solution showed highly significant (p≤0.01) increase in blood d- ALAD activity as compared to that of the rats fed with lead acetate solution treated later with vitamin E (group VIII).
Haematological parameters

**Table 2: Level of blood Hb is expressed as g%, RBC count is expressed as million/mm³ blood and WBC count is expressed as 10²/mm³ blood**

(Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Hb (g %)</th>
<th>RBC(million/mm³ blood)</th>
<th>WBC(10²/mm³ blood)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>13.66 ±1.25</td>
<td>9.07±0.83</td>
<td>11.22±1.03</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>11.24 ±1.03</td>
<td>6.11±0.56</td>
<td>5.27±0.48</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>11.44 ±1.04</td>
<td>6.29±0.57</td>
<td>4.43±0.41</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>12.98 ±1.18</td>
<td>8.29±0.76</td>
<td>10.77±0.98</td>
</tr>
<tr>
<td>V</td>
<td>Do +N PFG</td>
<td>12.96 ±1.18</td>
<td>7.99±0.73</td>
<td>8.09±0.74</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>13.88 ±1.27</td>
<td>8.76±0.80</td>
<td>8.42±0.77</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>13.06 ±1.19</td>
<td>8.14±0.74</td>
<td>8.35±0.76</td>
</tr>
<tr>
<td>VIII</td>
<td>Do+ Vitamin E</td>
<td>12.65 ±1.15</td>
<td>7.98±0.73</td>
<td>7.75±0.71</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI,VII)

F value: Hb 3.969, RBC 13.357, WBC 56.974

**Level of blood Hb, RBC count and WBC count (Table 2)** (Only significantly varied values are cited here)

Hb concentration decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) when compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with
PFG or NPFG oils or vitamin E (groups IV, V and VIII) showed only just significant (p ≤ 0.05) increases of Hb concentration as compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p ≤ 0.001) increase of Hb concentration when compared to the values of two groups of rats fed with lead acetate for one month (group II) and that fed with lead acetate but left as untreated group for one month (group III). Further on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed only highly significant (p ≤ 0.01) increase of Hb level as compared to the group of rats fed with lead acetate solution for one month (group II). However on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed only just significant increase of Hb level as compared to the group of rats fed with lead acetate but left as untreated group for one month (group III).

RBC count and WBC count decreased very high significantly (p ≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and that fed lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p ≤ 0.001) increases of RBC and WBC count as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p ≤ 0.001) increase in WBC count as compared to the values of two groups of rats fed with lead acetate solution and
later treated with NPFG oil (group V) or treated later with vitamin E (group VIII).

**Activities of toxicity marker enzymes**

*Table 3: Activities of serum and tissues alanine aminotransferases is expressed as IU/L for serum and µmol/min/100g for tissue*

(Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum (IU/L)</th>
<th>Liver(µmol/min/100g tissue)</th>
<th>Heart(µmol/min /100g tissue)</th>
<th>Kidney(µmol/min /100g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>12.55±1.14</td>
<td>89.22±8.14</td>
<td>162.75±14.85</td>
<td>115.70±10.56</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>30.52±2.79</td>
<td>54.37±4.96</td>
<td>61.84±5.64</td>
<td>62.72±5.72</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>30.67±2.79</td>
<td>54.37±4.96</td>
<td>66.02±6.03</td>
<td>64.12±5.85</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>15.33±1.39</td>
<td>82.26±7.51</td>
<td>120.39±10.99</td>
<td>101.68±9.28</td>
</tr>
<tr>
<td>V</td>
<td>Do +N PFG</td>
<td>16.73±1.53</td>
<td>82.25±7.51</td>
<td>106.33±9.70</td>
<td>100.38±9.16</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>19.52±1.78</td>
<td>75.28±6.87</td>
<td>132.01±12.05</td>
<td>83.64±7.63</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>20.91±1.90</td>
<td>73.89±6.74</td>
<td>127.75±11.66</td>
<td>82.24±7.51</td>
</tr>
<tr>
<td>VIII</td>
<td>Do+ Vitamin E</td>
<td>22.30±2.04</td>
<td>68.31±6.23</td>
<td>99.38±9.07</td>
<td>75.27±6.87</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

- * Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001
- a: Comparison between normal control and lead acetate fed group (I and II, III)
- b: Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
- c: Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
- d: Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
- e: Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
- f: Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V.VI, VII)

F value: Serum66.096, Liver 22.006, Heart 63.815, Kidney33.45

**Activities of serum and tissues alanine aminotransferases [ALT] (Table 3)** (Only significantly varied values are cited here)

The activities of serum ALT increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated
for another one month (group III) as compared to that of the normal rats. There was no significant difference in the serum ALT activities between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases of serum ALT activities as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG and NPFG oils the groups of rats previously fed with lead acetate solution (group IV and V) showed very highly significant (p≤0.001) decreases in serum ALT activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further the activity of serum ALT decreased just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFO oil (group VI) when compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The activities of liver, heart and kidney tissues ALT decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference in liver, heart and kidney tissue ALT activities between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases of tissue ALT activities as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further the activity of heart ALT increased just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as
compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Further on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (group IV and V) showed very highly significant (p≤0.001) increases in liver, heart and kidney tissue ALT activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Again the activities of heart ALT increased very high significantly (p≤0.001) in groups of rats fed with lead acetate solution and later treated with PFO and NPFO oils (group VI and VII) as compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

**Figure 1: Activities of serum and tissues aspartate amino transferases is expressed as IU/L for serum and µmol/min/100g for tissue**

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI, VII)

F value: Serum 11.378, Liver63.269, Heart 37.027, Kidney47.683
Activities of serum and tissues aspartate amino transferases [AST] (Figure 1) (Only significantly varied values are cited here)

The activities of serum AST increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference in the serum AST activity between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases of serum AST activities as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for one another month (group III). Further the activity of serum AST decreased just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) when compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The activities of liver, heart and kidney tissue AST decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference in the tissue AST activities between the values of the above the groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤0.001) increases of liver, heart and kidney tissue AST activities as compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further the activity of
liver AST increased only just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Similarly the activity of kidney AST increased only just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as compared to that of the rats fed with lead acetate solution and later treated with vitamin E. However on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤0.001) increase in liver AST activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant (p≤0.001) increase in liver AST activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed just significant (p≤0.05) increase in kidney AST activity as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII).
Oxidative stress parameters

Figure 2: Activities of serum and tissues catalase is expressed as units /mg protein

The activities of serum catalase decreased very high significantly (p ≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with
polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in serum catalase activity as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤ 0.001) increases in serum catalase activities when compared to the values of two groups of rats fed with lead acetate solution and later treated with NPFG oil and vitamin E (group V and VIII). Further the activity of serum catalase increased very high significantly (p≤ 0.001) in group of rats fed with lead acetate solution and later treated with PFO oil (group VI) when compared to the values of two groups of rats fed with lead acetate solution and later treated with NPFO oil and vitamin E (group VII and VIII).

The activities of liver, heart and kidney catalase decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the above values of the groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in liver, heart and kidney tissue catalase activities as compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤ 0.001) increase in kidney catalase and only just significant (p≤ 0.05) increase in heart catalase activity when compared to that of the rats fed with lead acetate solution and later treated with NPFG oil
(group V). Similarly on treatment with PFG oil the groups of rats previously fed with lead acetate solution (group IV) showed very highly significant increases in liver, heart and kidney tissue catalase activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant (p≤ 0.001) increase in liver catalase activity when compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Again the activities of heart and kidney catalase increased very high significantly (p≤ 0.001) in groups of rats fed with lead acetate solution and later treated with PFO oil (group VI) as compared with that of the rats fed with lead acetate solution and later treated NPFO oil (group VII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VII) showed very highly significant (p≤ 0.001) increase in liver and kidney tissue catalase activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed very highly significant (p≤ 0.001) increase in liver tissue catalase activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
Table 4: Activities of serum and tissues SOD is expressed as units*/mg protein

(Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum</th>
<th>Liver</th>
<th>Heart</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>2.21±0.20</td>
<td>3.86±0.36</td>
<td>4.56±0.42</td>
<td>3.24±0.03</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>0.47±0.44</td>
<td>2.14±0.19</td>
<td>2.39±0.22</td>
<td>0.94±0.09</td>
</tr>
<tr>
<td>III</td>
<td>Do + No drugs</td>
<td>0.54±0.49</td>
<td>2.01±0.18</td>
<td>2.45±0.22</td>
<td>0.98±0.09</td>
</tr>
<tr>
<td>IV</td>
<td>Do + PFG</td>
<td>1.41±0.13</td>
<td>3.54±0.32</td>
<td>4.55±0.42</td>
<td>2.23±0.21</td>
</tr>
<tr>
<td>V</td>
<td>Do + NPFG</td>
<td>1.02±0.09</td>
<td>3.27±0.29</td>
<td>3.99±0.36</td>
<td>1.56±0.14</td>
</tr>
<tr>
<td>VI</td>
<td>Do + PFO</td>
<td>1.52±0.14</td>
<td>4.49±0.41</td>
<td>4.92±0.45</td>
<td>2.71±0.25</td>
</tr>
<tr>
<td>VII</td>
<td>Do + NPFO</td>
<td>1.44±0.13</td>
<td>4.21±0.39</td>
<td>3.99±0.36</td>
<td>1.69±0.15</td>
</tr>
<tr>
<td>VIII</td>
<td>Do + Vitamin E</td>
<td>1.11±0.10</td>
<td>2.81±0.26</td>
<td>3.09±0.28</td>
<td>1.55±0.14</td>
</tr>
</tbody>
</table>

Units*: enzyme concentration required to inhibit the chromogen production by 50% in 1 minute

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V, VI, VII)

F value: Serum132.032, Liver 52.211, Heart46.786, Kidney172.510

Activities of serum and tissues SOD (Table 4) (Only significantly varied values are cited here)

The activities of serum SOD decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very high significantly (p≤ 0.001) increases of serum SOD
activities when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant ($p \leq 0.001$) increase in serum SOD activity when compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Again on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant ($p \leq 0.001$) increase in serum SOD activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant ($p \leq 0.001$) increases in serum SOD activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The activities of liver, heart and kidney tissue SOD decreased very high significantly ($p \leq 0.001$) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant ($p \leq 0.001$) increases of liver, heart and kidney tissue SOD activities when compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very high significantly ($p \leq 0.001$) increases in heart and kidney SOD activities when compared to that of the rats fed with lead acetate solution and later
treated with NPFG oil (group V). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant ($p \leq 0.001$) increases in liver, heart and kidney SOD activities when compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant ($p \leq 0.001$) increases in liver and heart SOD activity when compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further the activities of heart and kidney SOD increased very high significantly ($p \leq 0.001$) in the group of rats fed with lead acetate solution followed by treatment with PFO oil (group VI) as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant ($p \leq 0.001$) increases in liver, heart and kidney tissue SOD activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant ($p \leq 0.001$) increases in liver and heart tissue SOD activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
Figure 3: Activities of serum and tissues glutathione reductase is expressed as $10^{-2} \text{U}^*/\text{mg protein}$

![Graph showing the activities of serum and tissues glutathione reductase.](graph.png)

$1 \mu \text{m}$ of NADPH oxidized / minute

Significant level is determined by ANOVA

- Significant level $\leq 0.05$, • means high significant level $\leq 0.01$, # means very high significant level $\leq 0.001$

- a: Comparison between normal control and lead acetate fed group (I and II, III)
- b: Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
- c: Comparison between LA fed group with no treatment and treated groups (III and IV, V, VI, VII, VIII)
- d: Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
- e: Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
- f: Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V, VI, VII)

F value: Serum 28.546, Liver 71.199, Heart 60.449, Kidney 37.02

Activities of serum and tissues glutathione reductase (Figure 3) (Only significantly varied values are cited here)

The activities of serum glutathione reductase decreased very highly significantly ($p \leq 0.001$) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rat. There was no significant difference between the values
of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant ($p \leq 0.001$) increase in serum glutathione reductase activity when compared to the values of two groups of rats fed with lead acetate solution for 1 month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (group IV and V) showed only just significant ($p \leq 0.05$) increases in glutathione reductase activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant ($p \leq 0.001$) increase in serum glutathione reductase activity as compared that of the rats fed with lead acetate solution and later treated with NPFO oil.

The activities of liver, heart and kidney tissue glutathione reductase decreased very high significantly ($p \leq 0.001$) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant ($p \leq 0.001$) increases in liver, heart and kidney tissue glutathione reductase activities when compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed highly significant ($p \leq 0.01$) increases in liver, heart and kidney tissue glutathione reductase activities as
compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed highly significant (p≤0.01) increases in liver and kidney tissue glutathione reductase activities as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very high significantly (p≤0.001) increases in liver, heart and kidney glutathione reductase activities as compared that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p≤0.001) increases in liver and heart glutathione reductase activities as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Again the activities of liver, heart and kidney glutathione reductase increased very high significantly (p≤0.001) in group of rats fed with lead acetate solution and later treated with PFO oil (group VI) as compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
**Figure 4: Activities of serum and tissues glutathione peroxidase is expressed as 10⁻²U*/mg protein**

![Graph](image)

U*: 1 µm of NADPH oxidized / minute

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V, VI, VII)

F value: Serum 64.013, Liver 78.418, Heart21.972, Kidney18.903

Activities of serum and tissues glutathione peroxidase (Figure 4) (Only significantly varied values are cited here)

The activities of serum glutathione peroxidase decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in serum glutathione peroxidase activities when compared to the
values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤0.001) increase in serum glutathione peroxidase activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group V) showed highly significant (p≤0.01) increase in serum glutathione peroxidase activity as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The activities of liver, heart and kidney glutathione peroxidase decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in liver, heart and kidney tissue glutathione peroxidase activities when compared to the values of two groups of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further the activity of liver glutathione peroxidase increased just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤0.001) increases in liver and kidney tissue glutathione peroxidase activities as compared to that of
the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further the activity of kidney glutathione peroxidase increased just significantly (p≤0.05) in group of rats fed with lead acetate solution and later treated with NPFO oil (group VII) as compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

**Table 5:** Concentration of serum and tissues glutathione content is expressed as mM/100ml for Serum and mM/100g for tissue (Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum</th>
<th>Liver</th>
<th>Heart</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>15.22±1.39</td>
<td>287.91±26.27</td>
<td>209.25±19.10</td>
<td>175.18±15.99</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>8.24±0.75</td>
<td>182.66±16.67</td>
<td>144.85±13.23</td>
<td>95.76±8.74</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>7.29±0.67</td>
<td>182.11±16.61</td>
<td>144.36±13.17</td>
<td>96.54±8.81</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>11.93±1.09</td>
<td>299.80±27.35</td>
<td>189.74±17.32</td>
<td>182.41±16.65</td>
</tr>
<tr>
<td>V</td>
<td>Do +N PFG</td>
<td>10.65±0.97</td>
<td>257.65±23.52</td>
<td>183.64±16.76</td>
<td>163.83±14.95</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>12.81±1.17</td>
<td>243.09±22.19</td>
<td>219.43±20.01</td>
<td>199.33±18.18</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>11.56±1.06</td>
<td>210.71±19.23</td>
<td>202.20±18.43</td>
<td>181.07±16.52</td>
</tr>
<tr>
<td>VIII</td>
<td>Do+ Vitamin E</td>
<td>10.36±0.95</td>
<td>212.83±19.42</td>
<td>168.90±15.42</td>
<td>154.05±14.04</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI,VII)

F value: Serum35.886, Liver25.722, Heart 16.827, Kidney43.277

**Concentration of serum and tissues glutathione content** (Table 5) (Only significantly varied values are cited here)

Level of serum GSH content decreased very high significantly (p ≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and
also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference in the results between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very high significantly (p≤ 0.001) increases in serum glutathione content when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed only just significant (p≤0.05) increase in serum glutathione content as compared to that of the values of the two groups of the rats fed with lead acetate solution and later treated with NPFG oil and vitamin E respectively (group V and VIII). Again on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed just significant (p≤ 0.05) increase in serum GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The level of liver, heart and kidney GSH content decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to normal group I. There was no significant difference in the results between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in glutathione content in certain tissues (except liver glutathione in NPFO treated group and liver and heart tissues of glutathione content in vitamin E treated group) when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with
PFG oil the group of rats previously fed with lead acetate solution (group IV) showed highly significant (p≤ 0.01) increase in liver tissue glutathione content as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). However on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed just significant (p≤ 0.05) increase in kidney tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤ 0.001) increase in liver glutathione content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed highly significant (p≤ 0.01) increase in kidney tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Again on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed just significant (p≤ 0.05) increase in heart tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant (p≤ 0.001) increase in liver tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤ 0.05) increase in liver and kidney tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil fed group. Similarly on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤ 0.05) increase in liver tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed highly significant (p≤ 0.01)
increase in kidney tissue GSH content as compared to that of the rats fed with lead acetate solution and later treated with vitamin E.

**Table 6: Concentration of serum and tissues TBARS is expressed as mMol/100ml for serum as mMol/100g for tissues** (Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum</th>
<th>Liver</th>
<th>Heart</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>0.35±0.03</td>
<td>0.92±0.08</td>
<td>0.39±0.04</td>
<td>1.88±0.17</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>0.81±0.08</td>
<td>2.56±0.23</td>
<td>1.32±0.12</td>
<td>3.17±0.29</td>
</tr>
<tr>
<td>III</td>
<td>Do+ no drugs</td>
<td>0.83±0.08</td>
<td>2.62±0.24</td>
<td>1.32±0.12</td>
<td>3.37±0.31</td>
</tr>
<tr>
<td>IV</td>
<td>Do+ PFG</td>
<td>0.49±0.04</td>
<td>0.89±0.08</td>
<td>0.31±0.03</td>
<td>1.76±0.16</td>
</tr>
<tr>
<td>V</td>
<td>Do+NPFG</td>
<td>0.49±0.05</td>
<td>1.03±0.09</td>
<td>0.40±0.04</td>
<td>2.15±0.19</td>
</tr>
<tr>
<td>VI</td>
<td>Do+PFO</td>
<td>0.59±0.05</td>
<td>1.54±0.13</td>
<td>0.41±0.04</td>
<td>2.19±0.20</td>
</tr>
<tr>
<td>VII</td>
<td>Do+NPFO</td>
<td>0.65±0.06</td>
<td>1.64±0.14</td>
<td>0.43±0.04</td>
<td>2.52±0.23</td>
</tr>
<tr>
<td>VIII</td>
<td>Do+ Vitamin E</td>
<td>0.68±0.06</td>
<td>1.66±0.15</td>
<td>0.44±0.04</td>
<td>2.31±0.21</td>
</tr>
</tbody>
</table>

TBARS = Thiobarbituric acid reacting substances

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI,VII)

F value: Serum 49.250, Liver 111.518, Heart 240.736, Kidney 39.346

**Concentration of serum and tissues TBARS (Table 6)** (Only significantly varied values are cited here)

The concentration of serum TBARS increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats (group I). There was no significant difference between the values of the above groups II
and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in serum TBARS level when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution for one month but left as untreated group for another one month (group III). Further on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (group IV and V) showed very highly significant decreases in serum TBARS concentration when compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further the concentration of serum TBARS decreased highly significantly (p≤ 0.01) in group of rats fed with lead acetate solution and later treated with PFO oil (group VI) as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The concentration of liver, heart and kidney tissue TBARS also increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats. There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in liver, heart and kidney tissue TBARS level when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further the level of heart tissue TBARS decreased only just significantly (p≤0.05) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). However the level of kidney tissue TBARS decreased high significantly.
(p≤0.01) in the group of rats fed with lead acetate solution followed by treatment with PFG oil (group IV) as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Again on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant decreases in liver, heart and kidney tissue TBARS concentration as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Same effect with NPFG oil was found highly significant only with respect to liver tissue i.e. on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant decreases only in liver tissue TBARS concentration as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤0.05) decrease in kidney tissue TBARS level as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII). However on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p≤0.001) decrease in liver tissue TBARS level as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII).
Figure 5: Concentrations of serum vitamins E and C are expressed as mg/100ml

![Graph showing serum vitamins E and C concentrations]

The level of serum vitamin E decreased very high significantly (p ≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal rats (group I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non-polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p ≤ 0.001) increases of serum vitamin E level as compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further, on treatment with PFG oil, the group of rats previously fed with lead acetate solution (group IV) showed...
highly significant (p≤0.01) increases in serum vitamin E level as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Further on treatment with vitamin E the group of rats previously fed with lead acetate solution (group VIII) showed very highly significant (p≤0.001) increase in serum vitamin E level as compared to that groups of the rats fed with lead acetate solution and later treated with NPFG or PFO or NPFO oils (groups V, VI and VII).

Serum vitamin C level decreased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increase in serum vitamin C level when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (groups IV and V) showed very highly significant (p≤0.001) increases in serum vitamin C level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
Lipid profile:

**Table 7:** Concentration of serum and tissues total cholesterol is expressed as mg/dl for serum and mg/100g for tissues (Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum</th>
<th>Liver</th>
<th>Heart</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>76.40±6.97</td>
<td>353.59±32.27</td>
<td>235.78±21.51</td>
<td>367.45±33.52</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>109.49±9.99</td>
<td>540.57±49.33</td>
<td>443.71±40.48</td>
<td>568.08±51.84</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>112.02±10.21</td>
<td>533.57±48.69</td>
<td>415.97±37.96</td>
<td>540.78±49.35</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>73.85±6.74</td>
<td>342.86±30.79</td>
<td>114.38±10.44</td>
<td>298.12±27.20</td>
</tr>
<tr>
<td>V</td>
<td>Do +N PFG</td>
<td>80.22±7.31</td>
<td>356.96±32.57</td>
<td>153.67±14.02</td>
<td>325.86±29.74</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>78.95±7.20</td>
<td>350.12±31.95</td>
<td>142.13±12.96</td>
<td>318.93±29.11</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>85.31±7.78</td>
<td>408.96±37.32</td>
<td>208.11±18.99</td>
<td>367.45±33.53</td>
</tr>
<tr>
<td>VIII</td>
<td>Do +VitaminE</td>
<td>91.68±8.36</td>
<td>411.30±37.53</td>
<td>221.84±20.24</td>
<td>409.03±37.32</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V, VI, VII)

F value: Serum 19.708, Liver 27.226, Heart152.72, Kidney44.380

**Concentration of serum and tissues total cholesterol (Table 7)** (Only significantly varied values are cited here)

The concentration of serum total cholesterol increased very high significantly (p ≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference
between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in serum total cholesterol level when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤ 0.001) decrease in serum total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed only just significant (p≤ 0.05) decrease in serum total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed highly significant (p≤ 0.01) decrease in serum total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The concentration of liver, heart and kidney tissue total cholesterol increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in liver, heart and kidney tissue total cholesterol level when compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG
oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p ≤ 0.001) decreases in heart and kidney tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed only highly significant (p ≤ 0.01) decreases in liver tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant (p ≤ 0.001) decreases in heart and kidney tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed only just significant (p ≤ 0.05) decreases in liver tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Similarly on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very significant (p ≤ 0.001) decreases in heart and kidney tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed highly significant (p ≤ 0.01) decreases in liver tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Again on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed very highly significant (p ≤ 0.001) decreases in heart and kidney tissue total cholesterol level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
Table 8: Concentration of serum and tissues TAG is expressed as mg/dl for serum and for mg/100g for tissues (Each value is the Mean ± S.D of six rats)

<table>
<thead>
<tr>
<th>No.</th>
<th>Groups of rats</th>
<th>Serum</th>
<th>Liver</th>
<th>Heart</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>33.36±3.05</td>
<td>270.52±24.69</td>
<td>41.76±3.82</td>
<td>68.05±6.21</td>
</tr>
<tr>
<td>II</td>
<td>Lead acetate</td>
<td>80.57±6.87</td>
<td>612.29±55.88</td>
<td>95.66±8.74</td>
<td>93.98±8.56</td>
</tr>
<tr>
<td>III</td>
<td>Do +No drugs</td>
<td>74.03±6.75</td>
<td>594.87±54.29</td>
<td>94.70±8.64</td>
<td>100.19±9.15</td>
</tr>
<tr>
<td>IV</td>
<td>Do +PFG</td>
<td>32.55±2.97</td>
<td>228.36±20.84</td>
<td>40.93±3.73</td>
<td>48.31±4.42</td>
</tr>
<tr>
<td>V</td>
<td>Do +NPFG</td>
<td>41.48±3.78</td>
<td>232.24±21.19</td>
<td>40.46±3.69</td>
<td>61.16±5.58</td>
</tr>
<tr>
<td>VI</td>
<td>Do +PFO</td>
<td>42.29±3.85</td>
<td>219.87±20.07</td>
<td>36.28±3.31</td>
<td>46.64±4.27</td>
</tr>
<tr>
<td>VII</td>
<td>Do +NPFO</td>
<td>48.89±4.38</td>
<td>264.45±24.13</td>
<td>41.17±3.76</td>
<td>48.06±4.38</td>
</tr>
<tr>
<td>VIII</td>
<td>Do +Vitamin E</td>
<td>52.06±4.75</td>
<td>313.28±28.59</td>
<td>47.47±4.34</td>
<td>67.35±6.16</td>
</tr>
</tbody>
</table>

Significant level is determined by ANOVA

* Significant level ≤ 0.05, • means high significant level ≤ 0.01, # means very high significant level ≤ 0.001

a- Comparison between normal control and lead acetate fed group (I and II, III)
b- Comparison between LA fed group and treated groups (II and III, IV, V, VI, VII, VIII)
c- Comparison between LA fed group with no treatment and treated groups. (III and IV, V, VI, VII, VIII)
d- Comparison between PFG oil treated group and NPFG oil treated group (IV and V)
e- Comparison between PFO oil treated group and NPFO oil treated group (VI and VII)
f- Comparison between vitamin E treated group and different fractions of oils treated groups (VIII and IV, V,VI, VII)

F value: Serum 83.985, Liver 138.249, Heart 127.617, Kidney 63.69

Concentration of serum and tissues TAG (Table 8) (Only significantly varied values are cited here)

The level of serum TAG increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in serum
TAG level as compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed highly significant (p≤0.01) decrease of serum TAG level as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). However on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (group IV and V) showed very highly significant (p≤0.001) decreases in serum TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤0.05) decrease of serum TAG level as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII). Again on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p≤0.001) decrease of serum TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The level of liver, heart and kidney tissue TAG increased very high significantly (p≤ 0.001) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases in liver, heart and kidney tissue TAG level as compared to the values of two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly
significant (p≤0.001) decrease of kidney tissue TAG level as compared to that of the rats fed with lead acetate solution and later treated with NPFG oil (group V). Again on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤0.001) decreases in liver and kidney tissue TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However group IV showed only just significant decrease in heart TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed very highly significant (p≤0.001) decrease in liver tissue TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with NPFG oil the group of rats previously fed with lead acetate solution (group V) showed only just significant decrease in heart TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤0.05) decrease of liver TAG level as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VI). Again on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p≤0.001) decreases of liver, heart and kidney tissue TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed very highly significant (p≤ 0.001) decrease of kidney TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed only just significant (p≤ 0.05) decreases of liver and heart tissue TAG level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).
**Figure 6**: Concentrations of serum HDL and LDL fractions of cholesterol are expressed as mg/100ml

The level of serum HDL cholesterol decreased very high significantly \((p \leq 0.001)\) in the group of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to that of the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non-polar fractions of garlic and onion...
oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) increases in serum HDL cholesterol level as compared to the values of two groups of rats fed with lead acetate solution (group II) and also that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG oil the group of rats previously fed with lead acetate solution (group IV) showed very highly significant (p≤0.001) increase in serum HDL level as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further the level of serum HDL cholesterol increased just significantly (p≤0.05) in group of rats fed with lead acetate solution and later treated with NPFG oil (group V) as compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Again the level of serum HDL cholesterol increased highly significantly (p≤0.01) in group of rats fed with lead acetate solution and later treated with PFO oil (group VI) as compared with that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

The level of serum LDL cholesterol increased very high significantly (p≤ 0.001) in groups of rats fed with lead acetate solution for one month (group II) and also in that fed with lead acetate solution for one month but left as untreated for another one month (group III) as compared to the normal control group (I). There was no significant difference between the values of the above groups II and III. The groups of rats fed with lead acetate solution followed by treatment with polar and non polar fractions of garlic and onion oils or vitamin E (groups IV-VIII) showed very highly significant (p≤ 0.001) decreases of serum LDL cholesterol as compared to the values of the two groups of rats fed with lead acetate solution (group II) and that fed with lead acetate solution but left as untreated group for one month (group III). Further on treatment with PFG and NPFG oil the groups of rats previously fed with lead acetate solution (group IV and V) showed very highly significant (p≤0.001) decreases of serum LDL cholesterol as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). Further on treatment with PFO oil the
group of rats previously fed with lead acetate solution (group VI) showed only just significant (p≤0.05) decrease of serum LDL cholesterol as compared to that of the rats fed with lead acetate solution and later treated with NPFO oil (group VII). Further on treatment with PFO oil the group of rats previously fed with lead acetate solution (group VI) showed very highly significant (p≤0.001) decrease of serum LDL cholesterol as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII). However on treatment with NPFO oil the group of rats previously fed with lead acetate solution (group VII) showed only just significant (p≤0.05) decrease of serum LDL cholesterol as compared to that of the rats fed with lead acetate solution and later treated with vitamin E (group VIII).

**Histopathology of liver, heart and kidney:**

Histological studies showed that the control rats had normal hepatocytes and hepatic cord arranged radially around the central vein and centrally located nuclei. The normal control group revealed intact and distinct renal micro architecture. Lumen of the proximal convoluted, distal convoluted and collecting tubules were well demarcated with intact glomeruli. Normal architecture of the myocardium was observed that no evidence of microscopic changes in the control group.

Rats fed with lead acetate solution showed that the normal structural organization of the hepatic lobules was impaired and the characteristic cord like arrangement of the normal liver cells lost. The central and portal veins were congested. Central vein and sinusoids between hepatocytes were dialated. The same rats showed disruption of the normal architecture, vacuolar spaces and disrupted proximal and distal convoluted tubules and also distoration in the normal myofibrillar structure. On treatment with polar and non polar fractions of garlic and onion oils and vitamin E showed that most of these histopathological changes were diminished but some cells appeared with vacuolized cytoplasam.
Discussion:

Lead is a ubiquitous environmental and industrial pollutant that has been detected in almost all phases of biological system affecting a large segment of society. Treatment of lead toxicity should have different approaches; the currently used treatment for lead toxicity is to give metal chelating agents such as meso-2, 3 dimercapto succinic acid (DMSA) and mono isoamyl DMSA (MiA DMSA) which form an insoluble complex with lead. But all these chelators are potentially toxic and fail to remove lead from the all body tissues because they are only hydrophilic, not lipophilic. So they cannot cross the cell membrane for capturing intracellular lead. Recent trends in controlling and treating diseases favour natural antioxidants. Formulation of an effective economic way to recover from the toxicities induced by the lead is of particular importance for a healthy society. One of the molecular mechanisms of lead induced toxicities is mediated by oxidative stress. Antioxidants can ameliorate oxidative damage caused by ROS. Numerous studies support the idea that sulfur contained antioxidants protect against oxidative damage associated with disease development and progression. Researchers suggested protective role through multiple antioxidant mechanism such as ROS scavenging and metal binding. The allium plants such as garlic and onion have broad spectrum of activities with antioxidant properties. Both garlic and onion are economically suitable for people or societies and also these have biologically active lipophilic sulfur bearing compounds and these biologically active sulfur compounds can cross the cell membrane.

We have studied the oxidative damage produced by lead poisoning due to daily consumption of lead acetate solution in rats and compared it with the recovery during the administration of polar and non polar fractions of garlic and onion oils and vitamin E. Various aspects of toxicity manifestations such as hematological indices, lead level in serum, blood d-ALAD, antioxidant status, lipid peroxidation, lipid metabolism and histopathological changes of various
tissues were studied in detail. Our study indicates that the feeding of those fractions of garlic and onion oil and vitamin E leads to the recovery from lead acetate induced toxicity. When compared the curative efficiency, PFG oil fed rats showed faster recovery from lead toxicity. All the results correlated with our previous work done in MSc project and the data’s were published. In that work we observed that daily feeding of drinking water containing lead acetate to rats for a month produced certain deleterious alterations in the parameters of blood, serum and tissues, viz Hb, Pb, lipids, lipid peroxidation, vitamin C and E levels and enzyme activities of AST, ALT and catalase. Simultaneous feeding of either of the two antioxidants garlic oil and vitamin E at equal doses of 100 mg/Kg/day to the rats counteracted the deleterious effects of lead acetate toxicity significantly.

In the present study, feeding of lead acetate solution to experimental rats showed very highly significant decreases in Hb concentration, RBC and WBC counts when compared to that of the normal control rats. According to Swati N et al RBC, WBC, platelet counts, pcv and Hb concentrations decreased significantly after Nickel and Chromium treatment in albino rats. The simultaneous dietary garlic supplementation exhibited a protective role that combated nickel toxicity. This is in agreement with our study that on oral feeding of the antioxidants such as polar and non polar fractions of garlic and onion oils and vitamin E to the rats previously fed with lead acetate solution for a month showed significant increases in Hb concentration, RBC and WBC counts when compared to that of the lead acetate solution fed control group of rats. In another study by others mice treated with lead acetate showed severe toxic signs and significantly reduced total erythrocyte count, total leukocyte count, Hb and pcv, but when these mice were treated by garlic and vitamin B complex they showed almost normal levels of the above parameters. According to their findings garlic was more effective than vitamin B complex. This can be correlated well with our study that on treatment with garlic and onion oil polar fractions the groups of rats previously fed with lead acetate
solution showed a far better elevated levels of Hb, RBC and WBC counts when compared to that of the vitamin E treated rats. The haematological alteration might be due to the effects of the activity of the key enzyme for the synthesis such as d-ALAD. Lead also inhibits the conversion of coproporphyrinogen III to protoporphyrin IX leading to the reduction in Hb production and that can shorten the life span of RBC. Due to the binding of lead with RBC it leads to its destruction and fragility. Lead might induce oxidative stress by interacting with oxy Hb leading to peroxidative hemolysis in RBC membrane. These are the reasons behind the decreased hematological values in lead salt fed rats and mice. Lead directly affects the leucopoesis in lymphoid organs and decreases total leukocyte count\textsuperscript{231} in rats subjected to lead toxicity.

We observed that the activity of blood d-ALAD decreased very high significantly along with increased serum lead level in rats fed with lead acetate solution. Our results agree with those of Mohammad Hosein Noorimugahi et al\textsuperscript{232} that blood lead level is significantly higher in lead acetate intoxicated rats than that in the control group. This is also agreed with the study of Maged M Yassin\textsuperscript{233} that oral administration of lead acetate elevated blood lead level as compared to those of the control and sodium acetate groups but on treatment with crushed fresh garlic lobes, black seed or olive oil they all lowered blood lead level. Of these garlic showed the most effectiveness. Our study agreed with this work that on treatment with polar and non polar fractions of garlic and onion oils and vitamin E the groups of rats previously fed with lead acetate solution showed that the polar fractions of the oils very highly significantly decreased their serum lead level. Lesser grade of potency was shown by vitamin E and non polar fractions of garlic and onion oils. PFG showed the highest effect. This may be due to the fact that the polar fractions of the oils contain stronger detoxifying and antioxidant agents than the non polar fractions and vitamin E.

The mechanism of action of lead in the RBC is explained Nuran Ercal et al\textsuperscript{21} as follows d-ALAD and ferrochelatase are the two important enzymes
involved in heme synthesis. δ-ALAD is a Sulfhydryl (-SH group) containing enzyme and lead binds to its -SH group, making it inactive and thereby condensation of ALA is not possible due to the block of δ-ALAD activity by lead. Then δ-ALAD level becomes elevated in blood. This ALA undergoes enolization at physiological pH and then enolized ALA undergoes auto oxidation and generates super oxide anion. Proposed mechanisms for ALA oxidation and generation of free radicals are shown below

\[
\begin{align*}
\text{ALA (kenotic form)} & \quad \xrightarrow{\text{enolization}} \quad \text{ALA (enolic form)} \\
\text{ALA (enolic form)} + \quad \text{O}_2 & \quad \xrightarrow{\text{auto oxidation}} \quad \text{O}_2^- + \text{ALA}^+ + \text{H}^+ \\
2\text{O}_2^- + 2\text{H}^+ & \quad \xrightarrow{\text{SOD}} \quad \text{H}_2\text{O}_2 + \text{O}_2 \\
\text{H}_2\text{O}_2 + \text{Fe}^{2+} & \quad \xrightarrow{\text{H}_{2}\text{O}_{2}} \quad \text{OH}^- + \text{HO}^- + \text{Fe}^{3+}
\end{align*}
\]

The alliums contain biologically active lipophilic Sulfur compounds that can easily permeate through phospholipids membrane and reduces intracellular lead by removing it as PbS. This helps the tissues to reduce the lead burden following administration of these sulfur rich nutraceuticals. The mechanism of Allium sativum mediated chelation of lead is by the formation of ionic bonds between sulfur containing compounds and lead. The sulfur compounds such as allicin and thiosulfinates of garlic and onion act as active lewis acid with electron affinity and it can form compounds with positively charged ions i.e, lead that act as highly electropositive lewis base can initiate chelation of lead and increase the excretion of lead as their sulfides. Alternatively allium sulfides undergo very quick metabolism to give H\(_2\)S and this can remove Pb\(^{2+}\) from body fluids as PbS within a day.

\[
\text{H}_2\text{S} + \text{Pb. Ac} \quad \xrightarrow{\text{H}_{2}\text{S}} \quad \text{Acetic acid} + \text{PbS}
\]

According to Gehan AME Menoufy\(^{234}\) lead intoxication significantly increases the activities of SGOT and SGPT. Our study agreed with the findings of Gehan AME menoufy that the activities of serum ALT and AST increased and
tissue AST and ALT activities decreased very high significantly in rats fed with lead acetate solution. In another report SGPT and SGOT were increased in lead intoxication in mice and on treatment with garlic and vitamin B complex these values decreased to showing almost normal activity\(^\text{230}\). This is correlated with our work also that on treatment with antioxidants such as polar and non polar fractions of garlic and onion oils and vitamin E in groups of rats previously fed with lead acetate solution they showed significant decreases in serum AST and ALT activities and increased the activities of tissues AST and ALT. Polar fraction of garlic oil showed highly significant detoxification effect than any other sample of oils and vitamin E tested. This is also in agreement with MSH Khan et al\(^\text{230}\) report that explained AST and ALT are the enzymes seen in the cell cytoplasm under normal conditions. During injury or inflammation of cells such condition leads to the lack of cytoplasmic enzymes into the blood stream and thus the activities of serum ALT and AST are increased and tissues AST and ALT are decreased in cell injury due to toxicity\(^\text{234}\). Lead also causes cell lysis by affecting the K\(^+\) -Ca\(^{2+}\) channels that in turn leads to cytoskeleton alterations with increased susceptibility to lysis.

In our study the antioxidant enzymes such as catalase, SOD, glutathione reductase and glutathione peroxidase were decreased very high significantly in rats fed with lead acetate solution. Our study agree with the reports of other researchers also Ahamed R Ragab et al\(^\text{235}\) showed that lead acetate depressed the activities of SOD and Gpx and the report of Halyna Tkachenko et al\(^\text{236}\) states that lead intoxication also decreased the activities of glutathione reductase, glutathione peroxidase and SOD. Our work also correlates with the report of Swati N et al\(^\text{17}\) that erythrocyte SOD, glutathione peroxidase and catalase were significantly decreased by metallic ions of Nickel and chromium in rats. On simultaneous garlic supplementation in their diet the rats exhibited increased activities of these enzymes. Our study is well correlated with the above findings as in our results that the antioxidant enzymes such as SOD, catalase, glutathione reductase and peroxidase in
serum and tissues of the rats undergone lead toxicity significantly increased on treatment with polar and non polar fractions of garlic and onion oils and vitamin E. This is also in agreement with the study of Arti Sharma et al\textsuperscript{167} that aqueous and ethanolic extracts of garlic increased the depressed antioxidant enzymes SOD and catalase in lead nitrate toxicity and restored these deranged antioxidant enzymes to near normal level.

Our study also showed that rats fed with lead acetate solution decreased the level of non enzymatic antioxidant GSH in serum and tissues and that on treatment with antioxidants such as polar and non polar fractions of garlic and onion oils and vitamin E through feeding them significantly elevated the body GSH level. Our study is in agreement again with the findings of Artisharma et al\textsuperscript{167} that GSH can be restored along with antioxidant enzymes in lead nitrate induced toxicity in rats on orally feeding them with aqueous and ethanolic extracts of garlic which actually contain mostly the oil of garlic. Sudha K\textsuperscript{237} reported that in rats testes GSH level is decreased in arsenic induced toxicity and by the administration of α-tocopherol to these rats their testes significantly increased their GSH level. Therefore vitamin is definitely useful as allium oils in removing lead and arsenic toxicity in the body

In our findings serum and tissue lipid peroxidation level increased very high significantly in rats fed with lead acetate solution and on treatment of the groups of rats with polar and non polar fractions of garlic and onion oils and vitamin E respectively each of them significantly reduced the lipid peroxidation products. Our results are in agreement with that of the Halyana Tkaehenko et al\textsuperscript{236} in essence who reported that lipid peroxide level in the blood of rats fed with lead salt is significantly higher than the control and on treatment with arginine, it prevented the MDA production. Hande Gurer et al\textsuperscript{238} reported that lead exposed fischer 344 rats liver and kidney definitely lead to MDA production but on treatment of the rats with the antioxidant captopril it decreased the elevated MDA concentration. In yet another study Sadhana Shrivastava\textsuperscript{239} reported that garlic markedly attenuated the oxidative stress by
significantly reducing TBARS level in aluminium induced toxicity. Patra RC et al\textsuperscript{240} reported that vitamin E prevented lipo peroxide related lead toxicity. Lipid peroxidation is one of the main reasons of oxidative damage that plays an important role in the lipid toxicity of many xenobiotics. Lipid peroxidation is an outcome of the chain of events involving initiation, propagation and termination reactions. The lipid peroxides produced degraded into a variety of products including alkanals, hydroxyl alkanals, ketones and alkenes. All these products inactivate cell constituents by oxidations or it causes oxidative stress by undergoing radical chain reactions ultimately leading to loss of membrane integrity. Lipid peroxidation can also adversely affect the functions of membrane bound proteins such as enzymes and receptors\textsuperscript{226}.

In our study serum vitamin E and C levels decreased very high significantly in rats fed with lead acetate solution. Later on treatment with polar and non polar fractions of garlic and onion oils and vitamin E, all the five groups showed very highly significant increases of serum vitamin E and C levels .This is co related with Rekha B et al\textsuperscript{241} study that administration of Targeta erecta in lead exposed rats increased the non enzymatic antioxidants vitamin E and C levels. In our study it was also found that vitamin E treated group showed a higher significance in the values of vitamin E and C levels than allium oils treated groups. Vitamin E has a known protective action in membrane stability and it prevents membrane lipo proteins from oxidative damage. While vitamin C is a major water soluble antioxidant which acts directly by regenerating vitamin E. Vitamin C is an important scavenger of free radicals such as singlet oxygen, superoxide, hydroxyl, water soluble peroxyl radical and hypochlorous acid from extracellular fluids, trapping radicals and protecting membranes from peroxide damage\textsuperscript{242}.

We observed that feeding of lead acetate solution to rats increased the levels of serum, liver, heart and kidney tissue total cholesterol, TAG and LDL cholesterol while HDL cholesterol level decreased in these rats. Further on treatment with polar and non polar fractions of garlic and onion oils and
vitamin E showed very highly significant decreases of total cholesterol, TAG and LDL while HDL cholesterol increased in all these groups. Moreover polar fraction of garlic oil treated group showed higher significance in all these values when compared to other treated groups. Our results correlated with the study of Adeniyi TT et al that vitamin C and garlic decreased the level of cholesterol in lead exposed nephro toxic rats. In another study MAA Metwally reported that heavy metals induce rises in serum LDL, VLDL and TAG and a fall in serum HDL. Simultaneous garlic administration with the heavy metals improved in serum cholesterol, LDL, HDL, VLDL and TAG. All these agree with those of Artisharma et al that lead nitrate intake increased the mean values of cholesterol significantly in liver tissues. Lead salts mediated development of hypercholesterolemia involves the activation of cholesterol biosynthetic enzymes such as 3- OH- 3 methyl glutaryl Co A reductase (HMG Co A), farnesyl diphosphate synthase and squalene synthase and simultaneous suppression of cholesterol catabolic enzymes such as 7- alpha – hydroxylase. Garlic depressed the hepatic activities of lipogenic and cholesterogenic enzymes such as malic enzyme, fattyacid synthase, glucose – 6- phosphate dehydogenase and 3- OH- 3 methyl glutaryl Co A reductase. Further it also increased the excretion of cholesterol.

Therefore various researchers confirmed the detoxifying effects of garlic and alpha – tocopherol (vitamin E) in metal toxicity. As early as 1965 Petkov et al advocated the use of a Bulgarian drug ‘satlal’ as a remedy for lead poison in industries. Allium organic poly sulfur compounds are rich in ajoene type sulfides and they are present in allium oils and non toxic in minor doses. During these days our drinking water contains various metallic salts and insecticides as pollutants. We have to search the literature for more reports on the detoxifying nature of alliums not only on metallic salts but also on insecticides. Any way it is advisable to include onion and garlic in our diet to counteract pollution in general.