RESULTS AND DISCUSSION
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ANTIOXIDANT ENZYMES ASSAY

RESULTS

In the present study, the antioxidant enzyme activities are as follow.

Glutathione (GSH) level (µg of GSH/mg protein) was decreased in lead exposed groups when compared to control group. Group-I (control) 1.0550±0.0269, Group-II (exposed to lead toxicity/one week) 0.9533±0.0411 and Group-IV (exposed to lead toxicity/three weeks) 0.8850±0.0716 (Table-I). Treatment with ginger ethanolic extract increased the level of GSH. Group-III (exposed to lead toxicity cum treated with ginger extract/one week) 1.0083±0.0339 and Group-V (exposed to lead toxicity cum treated with ginger extract/three weeks) 0.9483±0.0501 (Table-II and Table-III).

Glutathione peroxidase (GPX) activity (µg of GSH consumed/mg protein) was decreased in lead exposed groups when compared to control group. Group-I (control) 1.3417±0.0498, Group-II (exposed to lead toxicity/one week) 1.2550±0.0373 and Group-IV (exposed to lead toxicity/three weeks) 1.0833±0.3789 (Table-I). Treatment with ginger ethanolic extract increased the activity of GPX. Group-III (exposed to lead toxicity cum treated with ginger extract/one week) 1.3033±0.0330 and Group-V (exposed to lead toxicity cum treated with ginger extract/three weeks) 1.2317±0.0273 (Table-II and Table-III).

Glutathione-S-transferase activity (µm of GSH-CDNB formed/min/mg protein) was decreased in lead exposed groups when compared to control group. Group-I (control) 19.0467±0.4462, Group-II (exposed to lead toxicity/one week) 17.1383±0.2639 and Group-IV (exposed to lead toxicity/three weeks) 21.1383±0.2639 and Group-IV
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(exposed to lead toxicity/three weeks) 15.0267± 0.3136(Table-I). Treatment with ginger ethanolic extract increased the activity of GST. Group-III (exposed to lead toxicity cum treated with ginger extract/one week) 17.4383±0.3478 and Group-V (exposed to lead toxicity cum treated with ginger extract/three weeks) 15.5500± 0.2707 (Table-II and Table-III).

Catalase (CAT) activity (m moles of H$_2$O$_2$ decomposed/ min/mg protein) was decreased in lead exposed groups when compared to control group. Group-I (control) 40.3933±0.3342, Group-II (exposed to lead toxicity/one week) 37.0850±0.2704 and Group-IV (exposed to lead toxicity/three weeks) 32.9883± 0.4156 (Table-I). Treatment with ginger ethanolic extract increased the activity of CAT. Group-III (exposed to lead toxicity cum treated with ginger extract/one week) 38.6867±0.9565 and Group-V (exposed to lead toxicity cum treated with ginger extract) 35.1367±0.4400 (Table-II and Table-III).
**Table-I**

Comparison of Glutathione (GSH) levels and activities of Glutathione peroxidase (GPX), Glutathione-S-transferase (GST) and Catalase (CAT) in Control (Group-I), One week lead exposed (Group-II) and Three weeks lead exposed (Group-IV).

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>Group-I</th>
<th>Group-II</th>
<th>Group-IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSH (µg of GSH/mg protein)</td>
<td>1.0550±0.0269</td>
<td>0.9533±0.0411</td>
<td>0.8850±0.0716</td>
</tr>
<tr>
<td>GPX (µg of GSH consumed/mg protein)</td>
<td>1.3417±0.0498</td>
<td>1.2550±0.0373</td>
<td>1.0833±0.3789</td>
</tr>
<tr>
<td>GST (µm of GSH-CDNB formed/min/mg protein)</td>
<td>19.0467±0.4462</td>
<td>17.1383±0.2639</td>
<td>15.0267±0.3136</td>
</tr>
<tr>
<td>Catalse (m moles of H₂O₂ decomposed/min/mg protein)</td>
<td>40.3933±0.3342</td>
<td>37.0850±0.2704</td>
<td>32.9883±0.4156</td>
</tr>
</tbody>
</table>

- The values are significantly different at P<0.05
- The values indicated by different letters are significantly different and those indicated by same letter are not significantly different.
# Table-II

Comparison of Glutathione (GSH) levels and activities of Glutathione peroxidase (GPX), Glutathione-S-transferase (GST) and Catalase (CAT) in Control (Group-I), One week lead exposed (Group-II) and one week lead exposed cum treated with ginger ethanolic extract (Group-III)

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>Group-I</th>
<th>Group-II</th>
<th>Group-III</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSH (μg of GSH/mg protein)</td>
<td>a</td>
<td>b</td>
<td>b</td>
</tr>
<tr>
<td>GPX (μg of GSH consumed/mg protein)</td>
<td>a</td>
<td>a</td>
<td>a</td>
</tr>
<tr>
<td>GST (μM of GSH-CDNB formed/min/mg protein)</td>
<td>a</td>
<td>b</td>
<td>b</td>
</tr>
<tr>
<td>Catalse (m moles of H₂O₂ decomposed/min/mg protein)</td>
<td>a</td>
<td>b</td>
<td>c</td>
</tr>
</tbody>
</table>

- The values are significantly different at P<0.05
- The values indicated by different letters are significantly different and those indicated by same letter are not significantly different.
TABLE-III

Comparison of Glutathione (GSH) levels and activities of Glutathione peroxidase (GPX), Glutathione-S-transferase (GST) and Catalase (CAT) in Control (Group-I), Three weeks lead exposed (Group-IV) and Three weeks lead exposed cum treated with ginger ethanolic extract (Group-V).

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>Group-I</th>
<th>Group-IV</th>
<th>Group-V</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSH</td>
<td>a</td>
<td>b</td>
<td>c</td>
</tr>
<tr>
<td>(µg of GSH/mg protein)</td>
<td>1.0550±0.0269</td>
<td>0.8850±0.0716</td>
<td>0.9483±0.0501</td>
</tr>
<tr>
<td>GPX</td>
<td>a</td>
<td>b</td>
<td>b</td>
</tr>
<tr>
<td>(µg of GSH consumed/mg protein)</td>
<td>1.3417±0.0498</td>
<td>1.0833±0.3789</td>
<td>1.2317±0.0273</td>
</tr>
<tr>
<td>GST</td>
<td>a</td>
<td>b</td>
<td>c</td>
</tr>
<tr>
<td>(µm of GSH-CDNB formed/min/mg protein)</td>
<td>19.0467±0.4462</td>
<td>15.0267±0.3136</td>
<td>15.5500±0.2707</td>
</tr>
<tr>
<td>Catalse</td>
<td>a</td>
<td>c</td>
<td>c</td>
</tr>
<tr>
<td>m moles of H₂O₂ decomposed/min/mg protein</td>
<td>40.3933±0.3342</td>
<td>32.9883±0.4156</td>
<td>35.1367±0.4400</td>
</tr>
</tbody>
</table>

- The values are significantly different at P<0.05
- The values indicated by different letters are significantly different and those indicated by same letter are not significantly different.
DISCUSSION

Lead is a non essential toxic heavy metal widely distributed in the environment and a chronic exposure to low levels of lead induces a broad range of physiological, biochemical and behavioral dysfunctions (Koller, 1990; Feldman and White, 1992; Yokoyama et al., 1997). Its toxicity is not well understood and various mechanisms have been suggested to explain it. These suggestions include disturbances in the mineral metabolism, demyelization of nerve tissues and inactivation of several enzymes (Ercal et al., 2001). Moreover recent studies have proposed that one possible mechanism of lead toxicity is the disturbance of prooxidant and antioxidant balance by generation of reactive oxygen species (ROS) (Gurer and Ercal 2000; Wang et al., 2001). This can evoke the oxidative damage of critical biomolecules such as lipids, proteins and DNA. It has also been reported that lead exposure has a dose response relationship with changes in antioxidant enzyme levels and their activities (Adonaylo and Oteiza, 1999).

Reactive oxygen species (ROS) are an entire class of highly reactive molecules derived from the metabolism of oxygen. ROS, including superoxide radicals, hydroxyl radicals and hydrogen peroxide are often generated by products of biological reactions or from exogenous factors. In vivo, some of these ROS play positive roles in cell physiology; however, they may also cause great damage to cell membrane lipid peroxidation, decreased membrane fluidity, and DNA mutations leading to cancer, degenerative, and other diseases (Cerruti 1991; Harman, 1994; Ames, 1998; Frinkel and Holbrook, 2000).

The cellular free radical scavengers and antioxidant enzymes normally protects the cell from toxic effect of ROS which include
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glutathione (GSH) and GSH dependent antioxidant scavenging systems viz., glutathione reductase (GR), glutathione peroxidase (GPX), glutathione-s-transferase (GST) and GSH independent antioxidant enzymes viz., superoxide dismutase (SOD) and catalase (CAT) (Simmon, 1984; Halliwell et al., 1984). Oxidative stress occurs when there is imbalance between free radical production reactions and scavenging capacity of antioxidative defense mechanism of the organisms. Enzymatic antioxidant systems SOD, CAT, GR, GPX, and GST functions by direct or sequential removal of ROS there by terminating their activities (Sies, 1991).

Mammalian cells possess elaborate defense mechanisms for radical detoxification. Key metabolic steps are Superoxide dismutase (SOD), Catalase (CAT) and Glutathione peroxidase (GPX), which destroy toxic peroxides. In addition to antioxidant enzymes, nonenzymatic molecules, including thioredoxin, thiols and disulfide bonding play important roles in antioxidant defense systems. Some of the compounds are of an exogenous nature and are obtained from food, such as α-tocopherol, β-carotene and ascorbic acid and such micronutrient elements as zinc and selenium (Halliwell and Gutteridge, 1998).

Antioxidant based drugs/formulations for the prevention and treatment of complex diseases like atherosclerosis, stroke, diabetes, Alzheimer’s disease and cancer have appeared during the last three decades (Devasagayam et al., 2004). Several herbs and spices have been reported to exhibit antioxidant activity, including rose marry, sage, thyme, mutmeg, turmeric, white pepper, chill pepper, ginger and several Chinese medicinal plant extracts (Kikuzaki et al., 1991; Jitoe et al., 1992; Kikuzaki et al., 1993; Lee et al., 2003). The majority of the active antioxidant compounds are flavonoids, isoflavones, flavones, anthocyanins, coumarins, lignans, catechins and isocatechins. In addition
to the above compounds found in natural foods, vitamin C and E, β-carotene and α-tocopherol are known to possess antioxidant potential (Prior, 2003; Cai et al., 2004; Kaur and Kapoor, 2007).

Glutathione a tripeptide and an essential bio-factor synthesized in all living cells. It functions mainly as an effective intracellular reductant (Beckman and Ames, 1998). It protects cells from free radical mediated damage caused by drugs and also by ionizing radiation. It forms an important substrate for GPX, GST and several other enzymes, which are involved in free radical scavenging. It can act as a nonenzymatic antioxidant by direct interaction of the SH group with ROS or it can be involved in the enzymatic detoxification reactions for ROS as a cofactor (Ding, Gonic and Vaziri, 2000). Resistance of many cells against oxidative stress is associated with high intracellular levels of GSH (Meistar, 1991; Estrela et al., 1995). GSH and thiol redox status regulates expression of genes involved in the pathogenesis of different diseases, including cancer, diabetes (Sen and Packer, 1996). In present study GSH levels were decreased in lead induced rat group when compared to control group. The GSH levels of control is 1.0550 µg of GSH/mg protein, and in one week lead exposed group is 0.9533 µg of GSH/mg protein, and in three weeks lead exposed group is 0.8850 µg of GSH/mg protein. Several studies supporting that lead toxicity decreases GSH levels (Bechara, 2004; Patra, Swarup and Dwidedi, 2001; Gurer, Ozgunes, Oztezcan and Ercal, 1999; Sivaprasad, Nagaraj and Varalakshmi, 2003). In the groups to which lead toxic induced and also treated with ginger ethanolic extract, GSH levels were increased when compared to only lead induced but not treat with ginger ethanolic extract. In one week lead induced and also treated with ginger ethanolic extract group, GSH level is 1.0083 µg of GSH/mg protein (0.9533 µg of GSH/mg protein in one week lead exposed but not treated with ethanolic ginger extract group) and in three weeks lead exposed and treated with ginger ethanolic extract group,
RESULTS AND DISCUSSION

GSH level is 0.9483 µg of GSH/mg protein (0.8850 µg of GSH/mg protein in three weeks lead exposed but not treated with ginger ethanolic extract group). As the GSH is substrate for GPX, GST and several other enzymes, it is believed that GSH level is also increased when these enzymes activity is increased. Several studies strengthening that GSH dependent antioxidant activity is increased by ginger extracts (Kikuzaki and Nakatani 1993; Lee and Ahn, 1985).

Glutathione peroxidase (GPX) is an antioxidant enzyme. This enzyme catalyzes the hydrogen peroxide-reduced glutathione reaction. The end products of this reaction are water and oxidized glutathione (Mehler, 1986). Glutathione peroxidase is an important component in the enzymatic defense system against the increase of free radicals (Crack et al., 2001; Klivenyi et al., 2000). Peroxides produced in a cell can be detoxified by the action of GPX (glutathione peroxidase) and catalase (CAT). GPX has a complementary catalytic activity with catalase. The Km value of H₂O₂ of GPX 0.25m mol/L is lower than that of CAT 25 m mol/L, providing a preferential pathway for the degradation of low concentration of H₂O₂ present in intact cell (Wohaieb and Godin, 1987). GPX catalyses the reduction of peroxides with GSH to form GSSG and the reduction product of H₂O₂ (Chance et al., 1979). This enzyme is specific for its hydrogen donor GSH, and non specific for the hydrogen peroxides ranging from H₂O₂ to organic peroxides (Freeman and Crapo, 1982). Thus offering a major defending role in cells against peroxidative damage of complex biochemical compounds such as lipids and nucleic acids (Meister and Anderson, 1983: Udhaybandyo padhay et al., 1999). In present study, the activity of glutathione peroxidase in control is 1.3417 µg of GSH consumed/min/mg protein, and in one week lead exposed group is 1.2550 µg of GSH consumed/min/mg protein and in three weeks lead exposed group is 1.0833 µg of GSH consumed/min/mg protein. The decrease in GPX activity due to lead toxicity has the support of literature.
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(Farmand et al., 2005; MC Gowan, Donaldson, 1986; Sugawara et al., 1991). In the group of lead induced and treated with ginger extract for one week group, the GPX activity is 1.3033 μg of GSH consumed/min/mg protein and in the group of lead induced and treated with ginger extract for three weeks, the GPX activity is 1.2317 μg of GSH consumed/min/mg protein. Literature supporting that ginger can promote glutathione peroxidase, as one of the antioxidant activity (Kikuzaki and Nakatani, 1993; Lee and Ahn, 1985).

The glutathione-s-transferase (GST) are an important enzymatic system of the cellular mechanism of detoxification that protects cells against reactive oxygen metabolites due to the conjugation of glutathione with electrophilic compounds. They show a broad specificity for organic hydroperoxides but not for H$_2$O$_2$ (Bruce et al., 1982). Enzymes of GST family involve in the cytosolic detoxification of a range of xenobiotic compounds by conjugation to glutathione which is essential in the maintenance of normal physiological processes (Hayes and Pulford, 1995; Deneke and Fanburge, 1989). In the present study the activity of GST is found as 19.0467 μ moles of GSH-CDNB formed/min/mg protein in control and 17.1383 μ moles of GSH-CDNB formed/min/mg protein in one week lead induced group and 15.0267 μ moles of GSH-CDNB formed/min/mg protein in three weeks lead induced group. Lead toxicity reduced the activity of GST (Planas-Bhone, Elizade, 1992; Adonaylo and Oteiza, 1999). GST activity is increased in groups of lead induced and treated with ginger extract when compared to the only lead induced groups but not treated. GST activity in one week lead induced and treated with ginger ethanolic extract group is 17.4383 μ moles of GSH-CDNB formed/min/mg protein, and in three weeks lead induced and treated with ginger ethanolic extract group is 15.5500 μ moles of GSH-CDNB formed/min/mg protein. There is a suitable literature supporting the
promotion of GST activity by ginger (Debanka Sekhar Misra et al., 2005; ICMR Bulletin 2003; Kikuzaki and Nakatani, 1993).

Catalase (CAT) is a metalloprotein, it detoxifies enzymatically the peroxides and superoxides and superoxide anion. Reducing the hydrogen peroxide, catalase protects the tissues from highly reactive hydroxyl radicals. Catalase decomposes the $\text{H}_2\text{O}_2$ to $\text{H}_2\text{O}$ and $\text{O}_2$. CAT contains haem as the prosthetic group, this antioxidant enzyme is widely distributed in all animal tissues. Studies have shown that the administration of catalase results in protection against $\text{H}_2\text{O}_2$ mediated Lipid Peroxidation (LPO). In present study the control group catalase activity is 40.3933 and that of one week lead exposed group is 37.0850 and that of three weeks lead exposed group is 32.9883 m moles of $\text{H}_2\text{O}_2$ decomposed/min/mg protein. The decrease in CAT activity may be due to the inhibition of haembiosynthesis of catalase by lead (Patil et al., 2006). In some other ways CAT activity may be reduced, decrease of CAT activity due to lead toxicity gained the support of literature (Imran Khan Mohammad et al., 2008; Vaunzuela et al., 1989). Whereas CAT activity in ginger ethanolic extract treated for lead toxicity groups, in one week is 38.6867 and in three weeks 35.1367 m moles of $\text{H}_2\text{O}_2$ decomposed/min/mg protein. Ginger promoted the CAT activity and literature support is there strengthening such an activity of ginger (Debanka Sekhar Misra et al., 2005; Kikuzaki and Nakatani, 1993).
Figure-1

Comparison of Glutathione (GSH) levels in Control (Group-I), One week lead exposed (Group-II) and Three weeks lead exposed (Group-IV).
Figure-2

Comparison of Glutathione peroxidase (GPX) activity in Control (Group-I), One week lead exposed (Group-II) and Three weeks lead exposed (Group-IV).

![GLUTATHIONE PEROXIDASE (GPX)](image-url)

- **Group-I**: 1.3417 micro g of GSH consumed/mg protein
- **Group-II**: 1.255 micro g of GSH consumed/mg protein
- **Group-IV**: 1.0833 micro g of GSH consumed/mg protein
Figure-3

Comparison of Glutathione-S-transferase (GST) activity in Control (Group-I), One week lead exposed (Group-II) and Three weeks lead exposed (Group-IV)

![Graph showing GST activity in different groups](image-url)
Figure-4

Comparison of Catalase (CAT) activity in Control (Group-I), One week lead exposed (Group-II) and Three weeks lead exposed (Group-IV).
Figure-5

Comparison of Glutathione (GSH) levels in Control (Group-I), One week lead exposed (Group-II) and one week lead exposed cum treated with ginger ethanolic extract (Group-III)
Figure-6

Comparison of Glutathione peroxidase (GPX) activity in Control (Group-I), One week lead exposed (Group-II) and one week lead exposed cum treated with ginger ethanolic extract (Group-III)
Figure-7

Comparison of Glutathione-S-transferase (GST) activity in Control (Group-I), One week lead exposed (Group-II) and one week lead exposed cum treated with ginger ethanolic extract (Group-III)
RESULTS

The normal structure of the kidney of albino rat comprised of numerous functional excretory units, the nephrons. Each nephron consisted of a renal corpuscle, a coiled uniferous tubule and haemopoietic tissue. The renal corpuscles were having distinct glomerulus and Bowman's capsule. The Bowman's capsule had two epithelial layers, inner squamodial and outer cuboidal. Bowman's capsule was followed by a tubular neck, proximal tubule, distal tubule and collecting tubule. The brush borders of these tubules were not clearly seen. The haemopoietic tissue occupied with intertubular spaces (Plate-I A and Plate-II A).

On exposure to the lead toxicity, the kidney of albino rat for one week showed architectural changes of glomerulus and tubular epithelial cells. The changes observed were dispersed in intra tubular region (Plate-I B). Whereas in kidney of one week lead exposed cum treated with ginger ethanolic extract rat's kidney, mild changes were observed than the kidney of one week exposed but not treated. Necrotic regions were observed and it seems to be fatty changes in between glomeruli and intratubular region. Mild hemorrhages were seen in glomeruli (Plate-I C).

On exposure to lead for three weeks, the kidney of rat showed severe changes than that of one week exposed. Infiltration of inflammatory cells was seen in intertubular region. Tubular epithelial cells review degenerative changes with mild necrotic changes (Plate-II B). Whereas in three weeks exposed to lead toxicity cum treated with ginger ethanolic extract showed lesser changes than three weeks exposed to lead but not treated. Atrophic glomeruli with enlarged and surrounded tubules were seen and more infiltration was observed in between intratubules and mild hemorrhages were observed (Plate-II C).
DISCUSSION

The architectural dynamics of a tissue is very essential for maintaining the tissue integrity and for effective physiological, biochemical and metabolic functions. The cellular and sub cellular constituents of tissue in terms of size, shape, number and position play an important role in the physiological and metabolic functions. Therefore, the histological structure of tissue in an animal has a profound influence on its function. Histology, the study of micro anatomy of specific tissues has been successfully employed as a diagnostic tool in medical and veterinary sciences since the first cellular investigations carried out in the nineteenth century (Virchow, 1858). The knowledge of the histology is useful to distinguish normal cells from abnormal or diseased ones, which helps in diagnosis of many diseases (Majumdar, 1980).

In present study, due to lead toxicity histological changes in kidney were observed in all lead induced and also in lead induced cum ginger ethanolic extract treated groups of one week and three weeks.

In one week lead exposed, kidney showed architectural changes of glomerulus and tubular epithelial cells were dispersed in intratubular region (Plate-I B). Whereas in the kidney of one week lead exposed cum ginger ethanolic extract treated, lesser histological changes were observed. In it fatty changes were observed in between glomeruli and intratubular region. And mild hemarages were seen in glomeruli (Plate-I C). The given ginger ethanolic extract seems to be mitigated the damage by lead.
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In three weeks lead exposed kidney, severe histological changes were observed than the one week lead exposed. In it infiltration of inflammatory cells was seen in intertubular region. Tubular epithelial cells review degenerative changes with mild necrotic changes (Plate-II B). In the three weeks lead exposed cum ginger ethanolic extract treated, mild changes were observed than untreated of three weeks. Atrophic glomeruli with enlarged and surrounded tubules were seen and more infiltration was observed in between intratubules and mild hemarrages were observed (Plate-II C). It is believed that ginger extract mitigating the damage by lead toxicity.

There are several reports that reveal, lead induces kidney organelles damage and its function on its accumulation in kidney. Lead poisoning not only causes renal dysfunction, but also liver cirrhosis, damage to the central nervous system and anemia (Sheffield et al., 2001 Damek-Poprawa and Sawicka-Kapusta, 2004).

Most of the lead entering the systemic circulation by injection invades the reticuloendothelial circulation represented by bone marrow, spleen and liver. In contrast, that entering the gut wall goes to bone and kidney (Blaxter, 1950).

Environmental lead poisoning is an increasing health burden and chronic exposure to high levels of lead leads to adverse effects on renal function and the hematopoietic system in both animals and humans. Chronic accumulation of lead in the body eventually leads to impairment in renal function (Restek-Samarzija et al., 1996).

Lead potentially induces oxidative stress and causes pathological changes of tissues like kidney (Ercal et al., 2000; Farmand et al., 2005; Khalil et al., 1992).
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Exposure to high lead levels can produce renal tubular damage with glycosuria and aminoaciduria (Loghman-Adam, 1997; Ehrlich, Robins, Jordan et al., 1998; Gerhard son, Chettle, Englyst, et al., 1992). The toxic effects of lead on the kidney appear to be primarily localized in the proximal tubule (Goyer and Ryne, 1973; Murakami et al., 1983).

Lead has commonly been thought to induce renal disease by causing direct tubular damage. Experimental lead toxicity can be associated with proximal tubular injury with characteristic intra nuclear inclusions (Mahaffey, Capar, Galden, Fowler, 1998).

Physiological studies of lead transport in the kidney have shown that this metal is taken up in proximal tubule cells (Vander et al., 1977; Vander et al., 1979). Brush border membrane vesicle transport studies have shown that lead is taken up by extensive membrane binding and possibly by a passive transport mechanism (Victery et al., 1984).

The marked ultra structural alterations due to lead intoxication were seen mainly in mitochondria. These organelles have shown toxic effects and the results indicated clearly that the mitochondria are highly susceptible to toxic injury of lead. The swollen mitochondria due to lead intoxication might be an indication to the overall swelling of injured cells. Mitochondria swelling is thought to be related with a change in osmolarity that leads to an influx of salts and water into the inner mitochondrial membrane, which becomes distended while the outer membrane eventually ruptures due to osmotic swelling (King et al., 1983). Renal proximal tubule cell mitochondria have long been known for their sensitivity to lead (Goyer, 1968; Oskarsson and Fowler, 1985).
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Decreased in the specific activities of mitochondrial based heme pathway enzymes have also been reported (Oskarsson and Fowler, 1985).

The histological studies and antioxidant enzyme studies when the lead treated rats exposed to ginger extract showed that there is some recovery from lead toxicity. Suggestions that the natural remedies are safe for human consumption without side effects and also our studies proved that the toxic levels of metals can be reduced to considerable extent.