INTRODUCTION

There has been increasing concern about the rapidly rising levels of environmental chemicals, especially heavy metals. Lead (Pb) is the most common nonessential heavy metal in the environment, having a toxic effect in humans. Humans have been using lead since ancient times, but the total quantity of lead used in the 20th century is far greater than the total consumption in all the previous periods. Although lead is one of the most useful metal, it is also one of the most toxic metals, producing severe organ damage in animals and humans (Humphreys, 1991; Spivey, 2007; Téllez-Rojo et al., 2004). Several studies have reported that lead toxicity is associated with impaired functioning of brain, liver, kidney, testes and the hematopoietic system (Bellinger, 2008; El-Nekeety et al., 2009; Sivaprasad et al., 2004). Prolonged exposure to low level of environmental lead has been proved as a risk factor for neurological, cardiovascular and reproductive disorders. Toxicity of lead is closely related to its accumulation in many tissues inside the body and its interference with the bioelements, that will hamper several physiological and biochemical processes (Berrahal et al., 2007; Pande et al., 2001).

Research studies indicate that cell damage mediated by increased production of free radicals can be involved...
in the pathology associated with lead (Erkal et al., 2001; Patra et al., 2001). Lead is known to cause oxidative damage in several tissues by bringing about imbalance in the generation and removal of reactive oxygen species (Adonaylo and Oteiza, 1999; Gurer et al., 1998; Sharma et al., 2010). Although the exact mechanisms by which lead induces oxidative damage in various organ systems are not completely explained, evidence indicates that multiple complex mechanisms are involved. Any compound that causes oxidative stress, does so by accelerating the generation of pro-oxidants and at the same time reducing antioxidant defense of the cells. Studies have reported that exposure to lead will lead to the disruption of reducing status of a tissue and form reactive oxygen species, which will damage the essential biomolecules such as protein, lipids and DNA (Flora et al., 2004; Stohs and Bagchi, 1995). Recent in vivo studies in lead exposed animals and workers showed the generation of reactive oxygen species, stimulation of lipid peroxidation and decreased antioxidant defense system, supporting the role of oxidative stress in lead toxicity (Bolin et al., 2006; Patrick, 2006; Sandhir and Gill, 1995). In the event that the generation of reactive oxygen species is mainly implicated in lead toxicity, a therapeutic strategy to increase the antioxidant defense system of the body may be of help for long-term effective treatment of lead poisoning. Several anti-oxidative approaches have been proposed therapeutically, including supplementation with antioxidant and up-regulation of endogenous anti-oxidative defense system for lead-induced oxidative stress in various organ systems. The proposed mechanism of action of these antioxidants is still indistinct. These antioxidants are believed to protect the cells from the influence of oxidative damage by scavenging the free radicals generation and inhibiting lipid peroxidation.

*Etlingera elatior* is a coarse herb, growing in clumps, native to Indonesia, Malaysia and Thailand. The plant is commonly known as the ‘torch ginger’ and is one of the 15 species of *Etlingera* sp. identified in Malaysia (Lim, 2001). More than 15 species of *Etlingera* plants have been recorded in Peninsular Malaysia (Chan et al., 2007). The young shoots and flower buds of the plants are consumed raw by indigenous communities in Malaysia and Thailand. Inflorescence of *E. elatior* is used for flavoring the food and also as ornamentals. The flowers and flower buds are commonly used in Malaysian dishes such as, Penang laksa, nasi kerabu and nasi ulam (Chan et al., 2007; Khaw, 2001; Larsen et al., 1999). *E. elatior* has been well known for its medicinal properties among indigenous communities in Malaysia. Leaves of *E. elatior* reported to have highest total phenolic content (TPC), ascorbic acid equivalent antioxidant capacity and tyrosine inhibiting activities (Chan et al., 2008). All the available studies on *E. elatior* have been conducted in vitro and there are no in vivo studies on the effects of this plant extract and its constituents on the oxidative stress parameters. Hence, the present study was taken up with an objective to look into the possible protective effect of extract of *E. elatior* against lead – induced liver toxicity in rats.

**MATERIALS AND METHODS**

**Plant extraction**

In the present study, we used the inflorescence of the *Etlingera elatior* plant because; it is this part of the plant that is eaten by the local population. Twelve kg of *Etlingera elatior* inflorescence was collected from a nursery in Sungai Buloh, Kuala Lumpur, Malaysia, and was authenticated by Department of Horticulture, University Putra Malaysia. Inflorescence of *Etlingera elatior* was washed in running tap water three times and cut into 3 cm pieces and again washed and soaked in running tap water for 5 min and air dried. The fresh dried petals were powdered and extracted with 70% ethanol. The mixture was filtered, evaporated in vacuum evaporator and lyophilized. The final product was a pink-purplish fine powder that bears the ginger aroma. The powder was stored in 50 ml polypropylene tubes away from direct light sources at 4°C till further use. The extract was evaluated for the presence of different classes of phytochemical compounds such as alkaloids, phenolic compounds, flavonoids, tannins, and proanthocynadins using standard procedures of analysis (Khan and Gilani, 2008).

**Experimental animals**

Three months old male Sprague Dawley rats weighing 180-200 g were purchased from University Kebangsaan Malaysia (UKM), Kuala Lumpur, Malaysia, and housed under standard laboratory conditions (25 ± 2°C; 12 hr light and dark cycles). The rats had access to an animal diet and tap water *ad libitum*. The rats were placed in polypropylene cages with three animals per cage and were allowed to acclimatize one week prior to treatment. All the experimental protocols conducted were in accordance with the internationally accepted principles for laboratory animal use and care and Institutional animal care and use committee and the study got approval from the Research and Ethics committee.

The rats were randomly divided into two groups, control (n = 8) and experimental group (n = 24). The rats in the experimental groups were divided into three groups; *E. elatior* alone, lead acetate alone, and lead acetate with
E. elatior. E. elatior extract was administered daily in the morning to non-fasted rats. At the end of 21 days, the rats were sacrificed and dissected. Blood and liver tissue samples were taken for biochemical and histopathological investigations.

Control group received ad libitum food and water. Rats in lead alone and lead with E. elatior group received lead acetate in their drinking water (500 ppm) for 21 days (Bokara et al., 2009). E. elatior treatment groups received once a day E. elatior extract at a dose of 300 mg/kg body weight. E. elatior extract was diluted with distilled water to the desired concentration (300 mg/kg body weight) and the extract was, force fed with feeding tube (0.5 ml/rat/day). This dose was chosen based on the result of the experiment on optimum dose with significant antioxidant properties, done in our laboratory.

Liver collected from each rat was weighed and washed immediately with ice cold saline. Liver homogenates were prepared in cold Tris-HCl (5 mmol/l, pH 7.4), using glass homogenizer. The samples were centrifuged at 10,000 rpm, for 7 min at 4°C and the supernatant was collected and stored at −80°C till further analysis. All the enzyme analyses were done within one week of collecting the samples. From the homogenate samples, lipid hydroperoxides (LPO), total antioxidants (TA), protein carbonyl content (PCC), superoxide dismutase (SOD), glutathione peroxidase (GPX) and glutathione S-transferase (GST) levels were assayed using ELISA kits (Cayman Chemical Company Ann Arbor, MI, USA and Pierce Biotechnology, Rockford, IL, USA). Protein levels of the samples were estimated by protein assay kits from Cayman Chemicals and Pierce Biotechnology.

For histopathology, liver tissues collected from each rats were stored in formaldehyde solution until processing. For block preparation, liver were dissected out and processed using a graded ethanol series and embedded in paraffin. Four micrometer paraffin sections were cut and stained with haematoxylin and eosin for light microscopic examination. The sections were evaluated by hemorrhage, vacuolar degeneration, necrosis, congestion and dilatation of sinusoids. Severity of changes were graded using a scale of no change (−), mild changes (+), moderate changes (+++) and severe changes (++++).

**Statistical analysis**

All the results were expressed as means with standard deviation. Graph Pad prism 5.0 software was used for statistical analyses. Comparison between all the groups were done using Kruskal Wallis one way analysis of variance test. Pair wise comparison between the different groups was done using Mann-Whitney-U test and a value of p < 0.05 was considered as statistically significant.

**RESULTS**

**BLL**

A significant increase (p < 0.05) in BLL was seen in the lead alone group compared to control and E. elatior groups. A significant decrease in BLL with 300 mg of E. elatior was recorded after 21 days (Table 1).

**Serum enzymes**

There was a significant heaptotoxicity with lead -acetate in drinking water for 21 days as evidenced by a significant increase (p < 0.05) in serum ALP, ALT, and AST levels. Treatment with E. elatior extract alone did not change these enzymes compared to control rats. Concurrent treatment with E. elatior significantly reduced (p < 0.05) ALP, ALT and AST level. Lead with E. elatior group had significantly higher (p < 0.05) serum ALP, ALT and AST levels than control group (Table 2).

**Hepatic lipid peroxidation**

To evaluate the effects of E. elatior treatment on lead acetate induced hepatic lipid peroxidation, we estimated the lipid hydroperoxide level and PCC in the liver homogenate. Hepatic lipid hydroperoxide levels increased significantly (p < 0.05) in the lead alone treatment group. A significant decrease in hepatic LPO was observed in concurrent treatment with E. elatior group (p < 0.05).

<table>
<thead>
<tr>
<th>Table 1. BLL in lead-induced hepatotoxicity</th>
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<tbody>
<tr>
<td>Groups</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>E. elatior</td>
</tr>
<tr>
<td>Lead Acetate</td>
</tr>
<tr>
<td>Lead with E. elatior</td>
</tr>
</tbody>
</table>

Within each row, means superscript with different letters are significantly different (p < 0.05).

*Significantly different from control - p < 0.05.

*Significantly different from lead alone treatment group - p < 0.05.

*Significantly different from lead + EE group - p < 0.05.
Treatment with *E. elatior* was able to reduce the serum LPO levels significantly (p < 0.05) to control levels. *E. elatior* alone group had significantly lower lipid hydroperoxides (p < 0.05) than control group (Table 3). Concurrent *E. elatior* treatment resulted in a significant decrease in PCC in liver when compared to lead alone group (p < 0.05) (Table 3).

**Hepatic antioxidant enzyme activities**

Super oxide dismutase, GPX, glutathione S - transferase and TA were measured as an index of antioxidant status in liver. The levels of SOD, GPX, GST and TA were significantly decreased (p < 0.05) in liver homogenates, compared to normal control and *E. elatior* alone groups. More significant difference in GST and GPX levels were recorded with lead treated rats than TA and SOD levels. Rats receiving 300 mg/kg of *E. elatior* alone did not show statistically significant difference in TA and antioxidant enzyme levels. Treatment with *E. elatior* to lead acetate drinking rats resulted in significant reversal of lead-induced alterations in these enzymes (p < 0.05). TA and SOD, GPX and GST levels increased significantly with concurrent treatment with *E. elatior*. But the levels of these antioxidant enzymes and TA were still significantly lower (p < 0.05) than normal control levels (Table 3).

### Table 2. Effects of lead acetate and *E. elatior* on serum enzyme levels in rats

<table>
<thead>
<tr>
<th>Groups</th>
<th>ALP (U/l)</th>
<th>ALT (U/l)</th>
<th>AST (U/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>128.12 ± 4.11</td>
<td>55.52 ± 2.41</td>
<td>72.11 ± 3.77</td>
</tr>
<tr>
<td><em>E. elatior</em></td>
<td>120.17 ± 3.99&lt;sup&gt;c&lt;/sup&gt;</td>
<td>52.12 ± 6.44&lt;sup&gt;c&lt;/sup&gt;</td>
<td>64.11 ± 2.55&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Lead acetate</td>
<td>241.22 ± 6.71&lt;sup&gt;c&lt;/sup&gt;</td>
<td>84.47 ± 4.89&lt;sup&gt;c&lt;/sup&gt;</td>
<td>108.88 ± 6.14&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Lead with <em>E. elatior</em></td>
<td>184.11 ± 8.44&lt;sup&gt;b&lt;/sup&gt;</td>
<td>66.19 ± 3.18&lt;sup&gt;b&lt;/sup&gt;</td>
<td>89.71 ± 8.17&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Within each row, means superscript with different letters are significantly different (p < 0.05).

<sup>a</sup> Significantly different from control - p < 0.05.
<sup>b</sup> Significantly different from lead alone treatment group - p < 0.05.
<sup>c</sup> Significantly different from lead + EE group - p < 0.05.

### Table 3. Effects of lead acetate and *E. elatior* on liver oxidative parameters in rats (Mean ± S.D.)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th><em>E. elatior</em></th>
<th>Lead Acetate</th>
<th>Lead with <em>E. elatior</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>LPO (nmol/mg protein)</td>
<td>3.23 ± 0.42</td>
<td>2.082 ± 0.043&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>5.129 ± 0.419&lt;sup&gt;c&lt;/sup&gt;</td>
<td>3.175 ± 0.621&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>PCC (nmol/mg protein)</td>
<td>12.098 ± 0.089</td>
<td>11.032 ± 0.312&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>18.421 ± 0.649&lt;sup&gt;c&lt;/sup&gt;</td>
<td>13.932 ± 0.541&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>TA (µmol/mg protein)</td>
<td>4.375 ± 0.143</td>
<td>4.103 ± 0.616&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.603 ± 0.115&lt;sup&gt;c&lt;/sup&gt;</td>
<td>3.711 ± 0.119&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>SOD (U/mg protein)</td>
<td>2.117 ± 0.442</td>
<td>2.354 ± 0.598&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>0.993 ± 0.054&lt;sup&gt;c&lt;/sup&gt;</td>
<td>2.116 ± 0.141&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>GPX (nmol/mg protein)</td>
<td>5.111 ± 0.277</td>
<td>5.209 ± 0.012&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.006 ± 0.305&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.843 ± 0.083&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>GST (µmol/h/mg protein)</td>
<td>5.555 ± 0.176</td>
<td>4.737 ± 0.531&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.051 ± 0.052&lt;sup&gt;c&lt;/sup&gt;</td>
<td>3.991 ± 0.115&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Within each row, means superscript with different letters are significantly different (p < 0.05).

<sup>a</sup> Significantly different from control - p < 0.05.
<sup>b</sup> Significantly different from lead alone treatment group - p < 0.05.
<sup>c</sup> Significantly different from lead + EE group - p < 0.05.
Histopathological changes

Control and *E. elatior* treatment showed normal arrangements of hepatocytes with normal nucleus and adequate cytoplasm, and there were clear sinusoids in most of the parts. There were no fatty changes or signs of necrosis. Microscopic observations showed that lead acetate for 21 days caused significant fatty changes in hepatic parenchyma and there was degeneration of hepatocytes. There were also nuclear pyknosis and extensive vascular congestion with lead toxicity in liver. Dilatation of sinusoids and red blood cells pooling were also seen in liver sinusoids with lead treatment. However, concurrent treatment with *E. elatior* had significant reduction of lead induced changes. Treatment of lead drinking rats with *E. elatior* from first day was able to reduce the hepatocyte damage of lead toxicity. There were many areas with healthy hepatocytes. Not much of necrotic changes were seen in lead with *E. elatior* groups. Vacuolar degeneration and vascular congestion was also significantly reduced compared to lead lone treated groups (Table 4; Figs 1, 2, 3 and 4).

**DISCUSSION**

The results of the present study showed that lead acetate in drinking water for 21 days resulted in severe hepato-
Table 4. Histopathological changes in liver of rats exposed to lead-acetate and E. elatior

<table>
<thead>
<tr>
<th>Groups</th>
<th>Vascular changes</th>
<th>Necrosis</th>
<th>Infiltration</th>
<th>Dilatation of sinusoids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>E. elatior</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Lead acetate</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Lead with E. elatior</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

− Nil; + Mild; ++ Moderate; +++ Severe

totoxicity. The selective dose of lead acetate used in the current study was based on previous literature (Aungst et al., 1981; Bokara et al., 2009). A significant increase in serum AST, ALT and ALP with lead treatment confirmed the severe hepatic damage caused by lead acetate. Liver enzymes such as AST, ALT, and ALP are marker enzymes for liver function and hepatic integrity. These enzymes are the markers for hepatotoxicity; damage to hepatocyte membrane will cause the release of many of these enzymes into circulation. The observed increase in ALP, AST and ALT confirm the earlier observations (Sharma et al., 2009, 2010). Damage to hepatic structural integrity induced by lead acetate is further supported by our histopathological study, where severe hepatocyte damage, vascular congestion and necrosis with fatty changes were seen after 21 days of lead ingestion. Similar changes were also reported earlier (Mudipall, 2007; Daggett et al., 1998).

There was increase in LPO and PCC in liver homogenates and decrease in TA and antioxidant enzymes like SOD, GPX and GST. This observation support the findings of several earlier studies, who reported perturbations in antioxidant enzyme activities in lead treated animals (Mudipall, 2007; Bolin et al., 2006; Sandhir and Gill, 1995) and lead exposed workers (Sugawara et al., 1991). These findings suggest that there is the major role of oxidative stress in the pathophysiology of lead toxicity.

Lead is known to produce oxidative damage in various organs by increasing lipid peroxidation (Adonaylo and Oteiza, 1999; Gurer et al., 1998). Lipid peroxidation will inactivate cell constituents by oxidation and ultimately lead to loss of membrane integrity (El-Nekeety et al., 2009; Flora et al., 2003). The observed increased LPO in the current study could be because of lead induced formation of free radicals and also through exhaustion of antioxidants, leading to oxidative stress (Abdel-Wahhab et al., 2008; Shabani and Rabbani, 2000). Protein modifications elicited by direct oxidative attack lead to the formation of protein carbonyl derivatives and PCC is the most commonly used biomarker for protein oxidation (Halliwell, 1996; Slater, 1985). The observed increase in PCC in lead exposed rats confirms the oxidative stress induced by lead acetate in hepatocytes.

Lead is known to cause oxidative damage in liver, brain, testis and kidney by enhancing lipid peroxidation (Halliwell and Gutteridge, 1989; Hamadouche et al., 2009; Landrigan et al., 2000; Sharma et al., 2010). Lipid hydroxyl peroxides are formed due to oxidation of lipid and cholesterol containing cellular molecules. These include cell membrane phospholipids, lipoproteins, glycolipid, cholesterol and other lipid-containing structures (Mudipall, 2007; Porter et al., 1995). Hepatic LPO levels increased significantly in lead poisoning and this could be due to lead induced inhibition of radical scavenging enzymes like GST and SOD. This indirectly causes ROS to accumulate in hepatocytes and cause increased oxidation (Ribarov and Bochev, 1982). Enzymes like GPX which converts peroxides into alcohol and water (Ursini et al., 1985) were also inhibited as observed in the present study, confirming that LPO once formed is not readily removed during lead toxicity.

Treatment with lead acetate significantly decreased the activities of SOD, GPX, GST and TA level. These results are in agreement with previous reports (Flora et al., 2007; Newairy and Abdou, 2009). The decrease in GPX level in the liver with lead acetate exposure could be due to lead’s displacement of the selenocysteine group from the active site in the liver tissue. The selenium containing functional group at the active site of GPX is responsible for it catalytic functions (Forstrom et al., 1978). Glutathione S - transferase catalyses a conjugation reaction, that adds a reduced glutathione to electrophilic groups on the molecule. This makes the original molecule, more soluble (Habig et al., 1974; Leaver and George, 1998). GST is also neutralizing oxidative molecules like ROS. The significant reduction in GST levels observed after lead acetate exposure could be due to lead acetate affects on sulphydryl groups as lead is known to be able to bind irreversibly with - SH groups on proteins, rendering them useless (Mudipall, 2007; Flora et al., 2007; Gurer and Erkal, 2000; Hsu and Guo, 2002; Newairy and Abdou, 2009).

Superoxide dismutases are a group of enzymes which catalyzes the conversion of superoxide anion (O₂⁻) to hydrogen peroxide (H₂O₂). The functional groups of the few types of SOD consist of transitional metals like copper/zinc, manganese, and iron (Maier and Chan, 2002; Sandstrom et al., 1994). Our results showed that there
was a significant reduction in super oxide dismutase level in the liver after three weeks of lead exposure, confirming the oxidant effects of lead acetate in hepatic tissue. Super oxide dismutase, GPX and GST enzymes take part in maintaining glutathione homeostasis in the tissues. These antioxidant enzymes are involved in the defense system against free radical mediated tissue or cellular damage after lead exposure (Arai et al., 1999; Sharma et al., 2010; Yiin and Lin, 1995). The observed decrease in liver antioxidants and decrease in TA confirm the lead acetate induced depletion of antioxidants.

In the present study, administration of *Etlingera elatior* alone significantly increased the hepatic antioxidant enzymes. There was also reversal of lead - induced changes in serum AST, ALT and ALP levels after treatment with *E. elatior*. The observed decrease in these serum marker enzymes shows that *E. elatior* preserves the structural integrity of liver against lead-induced damage. Treatment of *E. elatior* extract along with lead acetate treatment decreased the lead-induced changes in LPO and antioxidant enzyme levels. We have also observed a significant decrease in TA level in the liver after lead acetate and *E. elatior* treatment significantly reversed the changes in TA. This along with the changes in other antioxidant enzymes (SOD, GPx and GST) after *E. elatior* treatment strongly supports that *E. elatior* has powerful antioxidant properties against lead induced free radical damage in the liver (Arai et al., 1999; Newairy and Abdou, 2009).

There are very few reports available about antioxidant effects of *E. elatior* extract, and majority of them are in vitro studies (Habsah et al., 2005; Mian and Mohamed, 2001). Although the flower and flower shoots of this plant have been used for food, there are no studies on the possible antioxidant effect of *E. elatior* flower extract. This is the first research study on the effects of *E. elatior* on lead-induced changes in serum hepatotoxicity marker enzymes and hepatic oxidative stress markers. Habsah et al. (2000) reported that crude dichloromethane and methanol extracts of *E. elatior* possessed antioxidant activity and antitumor promoting activity. Chan et al. (2008) compared 26 different species of gingers for their anti-oxidative properties, and they have reported that, *E. elatior* ranked highest in TPC and ascorbic acid equivalent antioxidant capability (AEAC). The observed antioxidant activities of *E. elatior* flower extract against lead-induced hepatotoxicity could be attributed to the various antioxidant compounds present in the extract. Phytochemical screening of *Etlingera elatior* inflorescence extract showed that the extract had significantly higher quantities of phenolic compounds, flavonoids and flavones. The free radical scavenging effects of *E. elatior* extract could be attributed to its higher flavonoids and flavones contents. From the qualitative study on *Etlingera elatior*, Williams and Harbone (1977) isolated kaempferol 3-glucuronide, quercetin 3-glucuronide, quercetin 3-glucoside and quercetin 3-rhamnose compounds from the extract of leaves and rhizomes of this plants. These are bioactive flavonoid compounds, able to scavenge free radicals and increasing the synthesis and release of the antioxidant enzymes. In a more recent qualitative study, Habsah et al. (2005) identified two diarylheptanoids, namely 1,7-bis(4-hydroxyphenyl)-2,4,6-heptatrienone and 16-hydroxylabda-8 (17),11,13-trien-15,16-olide. The presence of these antioxidant compounds could have been the cause for observed protection of liver from lead-induced oxidative damage. The most important observation from this study on the antioxidant effects of inflorescence extract of *E. elatior* is that, the treatment nearly normalized the alterations of liver antioxidant enzymes, LPO and PCC. BLL showed that lead alone treatment group had a significantly higher BLL than concurrent exposure to *E. elatior* and lead groups. Lead, after its absorption to blood is carried to various tissues and more than 90% of blood lead is transported in the erythrocytes as lead phosphate (Freeman, 1970; Georing, 1993). This increased the BLL in lead-ingested rats, as there was daily ingestion in drinking water for three weeks, which was sufficient to maintain this elevated BLL to cause toxic effects. The observed decrease in BLL in *E. elatior* with lead acetate treatment group suggests the possible lead chelating effect of *E. elatior* extract. However, this property of *Etlingera elatior* needs further elaborate study.

Thus, in conclusion, the present study, confirmed that *E. elatior* has a powerful antioxidant effects against lead-induced hepatotoxicity. Treatment with *E. elatior* to lead - ingesting rats was able to reduce the hepatic lipid hydroperoxidation and PCC and increased total antioxidant levels and antioxidant enzymes. The protective effect of *E. elatior* was also confirmed by histopathological observations. There was also a reduction in blood lead content after *E. elatior* treatment supporting the possible lead-chelating effects of this plant extract. Thus, *E. elatior* has hepatoprotective effect against lead toxicity in rats. Further studies are being done to look into the different antioxidant constituents from this plant extract that has potent therapeutic effect against lead induced hepatotoxicity.

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