Effect of antox on hippocampal structure in male albino rats exposed to lead toxicity: histological and biochemical study

Omayma K. Helal¹, Ayman M. Mousa¹ and Nadra Kandeel²

Introduction

Lead is considered as one of the most dangerous causes of environmental pollution that poses a great danger to the health of people. The central nervous system, especially the areas undergoing postnatal development such as the hippocampus, is considered the most susceptible region to the hazards of lead.

Aim of the study

The present study was carried out to determine the effects of subclinical lead exposure on the hippocampus of albino rats and the ameliorating effect of antox on the affected group.

Materials and methods

Thirty-two male albino rats were divided into four groups (eight rats each). The first (control) group received distilled water orally, the second group received antox 10 mg/kg/day orally, the third group received 0.5% lead acetate in distilled water, and the fourth group received both antox and lead acetate at the same dose and through the same route for 2 months. Brain sections were prepared for histological (H&E), immunohistochemical (for detection of anti-glial fibrillary acidic protein), and electron microscopic studies. Blood samples were examined for estimation of superoxide dismutase, catalase, and serum malondialdehyde.

Results

The pyramidal cell layer in the hippocampus of the lead-treated group decreased significantly in number with the appearance of some degenerated cells. Numerous dilated blood capillaries were detected in the lead-treated group. There was a significant increase \( (P < 0.001) \) in the intensity of staining of astrocytes with glial fibrillary acidic protein in the lead-treated group. On electron microscopic study, there were degenerated nerve cells and fibers with a deformed myelin sheath. Oligodendrocytes showed an irregular dark nucleus and clumped chromatin. Antox ameliorated the toxic effects of lead for all histological results. Biochemical studies revealed a significant decrease in both superoxide dismutase and catalase, whereas malondialdehyde showed a significant increase in the lead-treated group and improved with the combination of antox and lead.

Conclusion

The present study showed a considerable ameliorating effect of antox on the structure of the hippocampus subjected to subclinical lead exposure.

Keywords:
antox, hippocampus, lead

Original article

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a number of other brain regions including the amygdala and nucleus accumbens are involved in the formation of nondeclarative memories. The formation of all these memories and learning process is based on the creation and remodeling of synapses [4].

A high level of lead exposure exerts serious clinical effects including neuroencephalopathy and peripheral neuropathy but the incidence of high lead exposure in human is now rare. Subclinical poisoning from chronic chemical agents such as heavy metals induces oxidative stress leading to the generation of more free radicals and alteration in antioxidants or oxygen-free radical-scavenging enzymes [6].

Antox is a multivitamin compound formed of ascorbic acid C, vitamin A acetate, vitamin E, and selenium. Protective effect of these vitamins that represent strong antioxidants and free radical scavenging activity [7].

Consequently, the aim of the present work was to determine the effects of subclinical lead exposure on the histological structure of the hippocampus in albino rats, as well as the ameliorating effect of antox on the activity of antioxidant enzymes [superoxide dismutase (SOD), catalase (CAT)] and also on the concentration of malondialdehyde (MDA) in the serum of these rats.

Materials and methods
Thirty-two male albino rats aged 2 weeks weighing 100–200 g were housed at room temperature and had free access to food and water ad libitum for 2 months [8], and were divided into four groups (eight rats each):

(1) Group I was the control group, which received distilled water orally.

(2) Group II was gavaged with antox dissolved in distilled water at a dose of 10 mg/kg/day [9] (Mebaco Arab Company for Pharmaceutical and Medicinal Plants, Cairo, Egypt).

(3) Group III was supplied with lead acetate powder [Cl(OCO)2Pb·3H2O] dissolved in distilled water at a concentration of 0.5% as the sole source of drinking water for rats [10] (Algomeria Company, Algomeria, Co. Pharmaceuticals & Chemicals, Cairo, Egypt).

(4) Group IV was treated by both lead acetate and antox at the same dose and using the same route.

Two months from the start of the experiment, 50% of the animals of each group were sacrificed. The brain of each animal was divided into two cerebral hemispheres, fixed in 10% formal saline, processed, and embedded in paraffin wax. Sections were prepared for histological (H&E) [11] and immunohistochemical [glial fibrillary acidic protein (GFAP) for astroglia] studies [11].

The rest of the animals of each group (50%) were anesthetized and perfused transcardially for 20 min with freshly prepared Karnovsky’s fixative (4% paraformaldehyde and 1% glutaraldehyde in 0.1 mol/l phosphate buffer at pH 7.4 and 4–10°C). Each brain was separated from the spinal cord by a perpendicular cut at the level of the obex, hemisectioned by a midline incision through the corpus callosum, and then a 4–5-mm-thick coronal slab of the right cerebral hemisphere was obtained at the level of the rostral limit of the anterior commissure and the caudal end of the median eminence. Each slab contained the parietal cortex and the anterior portion of the dorsal hippocampus. These small pieces were fixed in 2.5% glutaraldehyde, buffered in 0.1 mol/l sodium cacodylate, and then postfixed in 1% osmium tetroxide for ultrastructural study [10].

Slabs contained the parietal cortex and the anterior portions of the dorsal hippocampus were obtained. Small pieces were fixed in 2.5% glutaraldehyde, buffered in 0.1 mol/l sodium cacodylate, and then postfixed in 1% osmium tetroxide for ultrastructural study [12]. Sections were prepared, examined, and photographed at the Electron Microscope Research Laboratory, National Research Center, using JEOL (Musashino 3-chome Akishima Tokyo 196-8558, Japan) transmission electron microscopy.

Biochemical study
Blood samples were taken from the medial epicanthus for determination of lead blood levels using an atomic absorption spectrophotometer model 215, VGB 1996. The lead levels in the blood were reported in mg/dl. Serum biochemical investigation: SOD activity was determined using the epinephrine method [13]. CAT activity was also measured using the epinephrine method [14]. MDA was assayed colorimetrically, whereas MDA was determined using 1 ml of trichloroacetic acid (10%) and 1 ml of thiobarbituric acid (0.67). Consequently, they were heated in a boiling water bath for 30 min. Thiobarbituric acid-reactive substances were determined by absorbance at 535 nm and expressed as MDA formed [15].

Statistical study
Five fields from different H&E-stained sections of each rat in all groups were examined to count the number of abnormal and total nerve cells, and the percentage of abnormal cells in the three layers of hippocampus was determined using the image analyser Leica Q500 MC (Instruments Pty Ltd, North Ryde, NSW, Australia) program at the Faculty of Science, Al-Azhar University.

The area percentages of positive GFAP immunoreactive stain in astrocytes were measured in 10 images for the first, second, third, and fourth groups using Image-Pro Plus program version 6.0 (Media Cybernetics Inc., Bethesda, Maryland, USA).

All data are expressed as mean ± SD and statistically analyzed by Graph Pad Prism 4 (Inc. 2236 Avenida de la Playa, La Jolla, CA 92037, USA) Computerized program using one-way analysis of variance, followed by the Tukey–Kramer posttest to compare statistical differences between all groups at $P < 0.05$. 

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**Results**

**Histological study**

Light microscopic results of H&E-stained sections in both I and II groups revealed that the C-shaped hippocampus was composed of three areas: CA1, CA2, and CA3. Each area appears in three layers; polymorphic, pyramidal, and molecular. Narrow hippocampal sulcus, a part of the parahippocampal gyrus, and dentate gyrus were seen (Fig. 1). The pyramidal nerve cells appeared as large triangular cells with large vesicular nuclei and prominent processes (Fig. 2).

Immunohistochemical results for GFAP-stained sections of groups I and II revealed that area CA1 had a cytoplasmic brown reaction in large branched astrocytes dispersed among different cell layers of the hippocampus (Fig. 3).

Electron microscopic results revealed nerve cells with large lucent nuclei. Their cytoplasm had mitochondria and rough endoplasmic reticulum (rER). Also, dendritic processes were demonstrated in the neuropil (Fig. 4). Large oligodendrocytes were demonstrated with an irregular electron-dense nucleus, a tongue-like process, an electron-dense cytoplasm, and distended parallel cisternae of rER. Myelinated axons with regular contours were demonstrated in the surrounding neuropil (Fig. 5).

Group III: Examination of H&E-stained sections of lead-treated albino rats revealed a significant increase in shrunken nerve cells with a darkly stained cytoplasm and lost nuclear details (Fig. 6) in comparison with that in Fig. 2.

Immunohistochemical results for GFAP-stained sections revealed a positive expression of GFAP as dark brown cytoplasmic granules in astrocytes (Fig. 7) in comparison with that in the control group (Fig. 3).

Electron microscopic results revealed nerve cells with large ovoid nuclei. Their cytoplasm was scanty, with small aggregated mitochondria and few cisternae of rER. Some cells had irregular dark nuclei (Fig. 8) compared with the control group (Fig. 4). Oligodendrocytes with a large irregular dark nucleus and clumped chromatin were seen. Congested small blood vessels were also detected and the surrounding neuropil had irregular myelinated axons with separation and discontinuity of the myelin sheath (Fig. 9).

Dark oligodendrocytes had an electron-dense cytoplasm with small vacuoles. Apoptotic cells with an electron-dense cytoplasm were also seen (Fig. 10).

Group IV: Examination of H&E-stained sections in the hippocampus of lead-treated and antox-treated rat indicated a significant increase in pyramidal nerve cells compared with the lead-treated group with large vesicular nuclei. Few shrunken nerve cells with a dark cytoplasm and lost nuclear details appeared. Numerous blood capillaries were also seen (Fig. 11).

There was a significant increase in the mean % number of abnormal cells after lead intake in comparison with groups I and II ($P < 0.001$). However, antox with lead in group IV resulted in a significant decrease in mean % number of abnormal cells as compared with group III, whereas it was greater than those in groups I and II (Table 1 and Histogram 1).

Immunohistochemical results for GFAP-stained sections revealed a positive expression to GFAP in astrocytes, which showed a significant decrease in intensity (Fig. 12) compared with the lead-treated group (Fig. 7).

The mean area % of a positive GFAP immunoreactive stain in astrocytes for groups I, II, III, and IV was represented, respectively. There was a significant increase in positive staining to GFAP in astrocytes of the lead-treated group (Fig. 8) in comparison with that in the control (Fig. 3) and antox in the lead-treated group (Fig. 14).

In the present study, there was a significant increase in area % for GFAP expression in the lead-treated group ($P < 0.001$) compared with groups I and II. Coadministration of antox and lead resulted in a marked reduction in area % for GFAP expression in comparison with group III, although it was still at a significantly higher level than that in groups I and II (Table 1 and Histogram 2).

Electron microscopic results revealed nerve cells with ovoid electron-lucent nuclei. Dendritic processes were demonstrated in the surrounding neuropil (Fig. 13). Oligodendrocytes with dense irregular nuclei and an electron-dense cytoplasm were present. The surrounding neuropil showed some outfolded myelinated axons (Fig. 14).

Biochemical study in Table 2 shows a significant increase of lead in the lead-treated group (third) group and the lead+antox (fourth) group compared with the control group $P ≤ 0.001$, whereas the SOD level showed a significant increase on using antox alone in the second group $99.47 ± 3.38$ compared with the control group $89.27 ± 3029$ and decreased significantly in the group administered lead-poisoned water to record $73.05 ± 2.55$ to raise up to $82.52 ± 5.21$ in the fourth group but kept significantly lower than control with $P ≤ 0.001$.

CAT showed a significant reduction to $55.23 ± 2.12$ in the third group compared with $66.12 ± 2.06$ in the control group and $77.64 ± 1.35$ in the second group and increased to $60.06 ± 1.37$ when lead was combined with antox, with $P ≤ 0.001$.

MDA showed a significant increase to $11.25 ± 0.15$ in the serum of the third group compared with $8.51 ± 0.5$ in the control group and $7.21 ± 0.14$ in the second group, which decreased to $9.22 ± 0.32$ in the fourth group but remained higher than the control $P ≤ 0.001$. 

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**Table 1**

<table>
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<td>18.23 ± 2.12</td>
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<tr>
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<td>Antox</td>
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**Histogram 1**

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<td>16.75 ± 3.21</td>
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<td>None</td>
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<tr>
<td>III</td>
<td>Lead</td>
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<td>21.34 ± 2.06</td>
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<tr>
<td>IV</td>
<td>Lead</td>
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**Histogram 2**

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<td>None</td>
<td>16.75 ± 3.21</td>
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<tr>
<td>III</td>
<td>Lead</td>
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<tr>
<td>IV</td>
<td>Lead</td>
<td>Antox</td>
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Figure 1.

A photomicrograph of a section in the hippocampus of a control rat showing a C-shaped hippocampus composed of three areas: CA1, CA2, and CA3. Each area appears in three layers: polymorphic (PP), pyramidal (P), and molecular (M). Narrow hippocampal sulcus (arrow), a part of the parahippocampal gyrus (PH), and dentate gyrus (DG). (A) area CA1 that magnified. 

H&E, × 40.

Figure 2.

A photomicrograph of a section in the hippocampus of a control rat showing three layers: polymorphic (PP), pyramidal (P), and molecular (M) in area CA1. The pyramidal nerve cells appear as large triangular cells (arrow) that represent the principal cell layer in the hippocampus with large vesicular nuclei (double arrow).

H&E, × 400.

Figure 3.

A photomicrograph of a section in the hippocampus of a control rat showing part of area CA1 with a positive expression of glial fibrillary acidic protein (GFAP) in large branched astrocytes with cytoplasmic brown granules (arrow). Astrocytes are dispersed among different cell layers of the hippocampus.

GFAP immunostain, × 400.

Figure 4.

An electron micrograph in the hippocampus of a control rat showing two nerve cells with a large lucent nucleus (N), mitochondria (m), and rough endoplasmic reticulum (r). The surrounding neuropil has wide dendritic processes (d).

× 8000.

Figure 5.

An electron micrograph in the hippocampus of a control rat showing a large oligodendrocyte (O) with an electron-dense irregular nucleus (n), dark cytoplasm, a tongue-like cytoplasmic process, and distended parallel cisternae of rough endoplasmic reticulum (r). Numerous myelinated axons (ma) with a regular contour can be seen in the surrounding neuropil.

× 8000.

Figure 6.

A photomicrograph of a section in the hippocampus of lead-treated rats showing three layers: polymorphic (pp), pyramidal (p), and molecular (M) in area CA1. There are a few normal dispersed nerve cells (arrow) with a vesicular nucleus (N). Numerous shrunken pyramidal nerve cells with a darkly stained cytoplasm and lost nuclear details (double arrow). Many small capillaries can be seen (C), Numerous shrunken pyramidal nerve cells (V).

H&E, × 400.
Figure 7. A photomicrograph of a section in the hippocampus of a lead-treated rat, showing a part of area CA1 with dense positive brown expression of glial fibrillary acidic protein (GFAP) in branched astrocytes (arrow) dispersed among different cell layers of the hippocampus. GFAP immunostain, ×400.

Figure 8. An electron micrograph of a section in the hippocampus of lead-treated rats showing two nerve cells with large ovoid nuclei (N), scanty and vacuolated cytoplasm (v) with small aggregated mitochondria (m), and few cisternae of rough endoplasmic reticulum (r). A pyknotic cell with an irregular dark nucleus can also be seen (l). ×10 000.

Figure 9. An electron micrograph of a section in the hippocampus of a lead-treated rat showing an oligodendrocyte (O) with a large irregular dark nucleus and clumped chromatin. Congested small blood vessels (C) and irregular myelinated axons (ma) with separation (arrow head) and discontinuity of myelin (arrow) are present in the surrounding neuropil. ×8000.

Figure 10. An electron micrograph of a section in the hippocampus of a lead-treated rat showing a dark oligodendrocyte (O) with an electron-dense cytoplasm containing small vacuoles (v). An apoptotic cell with an electron-dense nucleus and a dark cytoplasm (ap) is present. ×6000.

Figure 11. A photomicrograph of a section in the hippocampus of a lead-treated and antox-treated rat, showing three layers: polymorphic (pp), pyramidal (p), and molecular (M) in area CA1. The pyramidal nerve cells appear with large vesicular nuclei (N). A few pyramidal nerve cells have small dark pyknotic nuclei with lost nuclear details (arrow). Some blood capillaries (C) can be seen. H&E, ×400.
Table 1. Percentage number of abnormal cells and area % for glial fibrillary acidic protein expression in the hippocampus of normal, antox-treated, lead acetate-exposed, and antox + lead acetate-administered rats

<table>
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<tr>
<th>Parameters</th>
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<th>Percentage number of abnormal cells (mean ± SD)</th>
<th>Area % for GFAP expression (mean ± SD)</th>
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<td></td>
<td>GI</td>
<td>2.4% ± 0.374b</td>
<td>1.3 ± 0.003b</td>
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<tr>
<td></td>
<td>GII</td>
<td>2.9 ± 0.386b</td>
<td>1.3 ± 0.003b</td>
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<tr>
<td></td>
<td>GIII</td>
<td>92.1 ± 0.942a∗</td>
<td>7.5 ± 0.001a∗</td>
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<td>GIV</td>
<td>17.5 ± 0.868a,b</td>
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*Significant difference from GI.

aSignificant difference from GII.
bSignificant difference from GIII.

Figure 12. A photomicrograph of a section in the hippocampus of a lead-treated and antox-treated rat showing a part of area CA1 with a dense positive brown expression to glial fibrillary acidic protein (GFAP) in astrocytes that appear as large branched cells (arrow) dispersed among different cell layers of the hippocampus. GFAP immunostain, ×400.

Figure 13. An electron micrograph of a section in the hippocampus of a lead-treated and antox-treated rat showing two nerve cells with ovoid nuclei (N). The surrounding neuropil has smooth outlined dendrites (d). ×10 000.

Figure 14. An electron micrograph of a section in the hippocampus of a lead-treated and antox-treated rat showing nearly normal oligodendrocytes (O) with a dense irregular nucleus (n) and an electron-dense cytoplasm. The surrounding neuropil shows some outfolded mylinated axons (ma) and numerous electron-lucent spaces (S). ×8000.
Table 2. Statistical comparison between means ± SD values of superoxide dismutase, catalase, and malondialdehyde serum level among the four studied groups using the one-way (analysis of variance) test; *P ≤ 0.005 indicates statistically significant difference

<table>
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<td>BLL (mg/dl)</td>
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<td>SOD (U/l)</td>
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<td>CAT (U/l)</td>
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<td>MDA (mmol/ml)</td>
<td>8.51 ± 0.5</td>
<td>7.21 ± 0.14</td>
<td>11.25 ± 0.15*</td>
<td>9.22 ± 0.32</td>
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CAT, catalase; MDA, malondialdehyde; SOD, superoxide dismutase, BLL, blood lead level
* Significant difference

Histogram 2.

Area % for glial fibrillary acidic protein expression in the hippocampus of normal, antox-treated, lead acetate-exposed, and antox+lead acetate-administered rats. *Significant difference from GI, **Significant difference from GII. *Significant difference from GIII.

Discussion

The CNS is considered as the primary target of low-level lead exposure as the developing brain appears to be especially vulnerable to lead neurotoxicity. Lead neurotoxicity causes impairment of cognitive abilities and a significant delay in behavioral development, especially in children. Thus, rats aged 2 weeks were used in this study. The intellectual deficits persist throughout life [16]. The hippocampus was reported to continue its development after birth; thus, it was chosen as the target site in this study. Moreover, lead acetate and lead phosphate are listed as carcinogens whereas inorganic lead compounds are classified as 2B chemicals [17].

The current study was performed to investigate the histological (H&E), immunohistochemical (GFAP), and ultrastructural features of the hippocampus and also biochemical reactions to determine the effects of lead exposure and the effectiveness of antox as an antioxidant in ameliorating such toxic effects.

Light microscopic examination of lead-administered groups in this study revealed that the number of pyramidal cells was significantly decreased compared with the control with the appearance of some degenerated cells with pyknotic nuclei. This was in agreement with the results of some authors, who attributed these results to the direct effect of lead on the hippocampal neurons [18]. These results were further reinforced by the work of others who found that lead was deposited in the hippocampus and cerebellum seven times more than the rest of the brain regions. The selective vulnerability of these cells could be related to its early development [19].

Several cells are responsible for an increase in neuronal connections and synaptic contacts. Various aspects of integration, plasticity, learning, and memory have been assigned to these particular properties of these cells, playing an important role in behavior [20].

Also, the lead-administered group showed some effects on the blood vessels, where numerous small capillaries were observed. However, there was no detectable hemorrhage. These findings indicated that the blood vessels and the BBB were affected due to lead exposure. This was comonitant with researchers who reported that lead had a greater affinity for endothelial cells that form part of the BBB and also the glial cells, mainly astrocytes, which are an important constituent of the BBB, might be the primary target of lead [21].

In the present study, the effect of lead on the astrocytes was studied using immunohistochemical labeling with GFAP antibodies. There was a significant increase in the intensity of cytoplasmic staining of astrocytes in the hippocampus of lead-administered rats in comparison with that of the control, antox, and lead+antox groups. Also, this response of astrocytes (gliosis) was a response to insults of lead typically characterized by an increase in its cytoskeletal intermediate filaments, which consist of polymerized GFAPs. The morphological changes in astrocytes that were observed in this study could be suspected to occur because the astrocytes have a greater capacity for reactive responses to toxic insults than neurons, as the neurons do not proliferate in response to injury [22].

These astrocytic morphological alterations were explained by some researchers as hyperactivity of cells that attempt at first to accommodate the toxic effect of metal and then form more glutathione to bind to it in an attempt to protect neurons from its hazardous effects [23]. This shows the protective role of antox against the harmful changes produced by lead. These findings were in agreement with another study that found a transient increase in the GFAP immunoreactivity in astrocytes of the hippocampus after 2 months of lead administration [24]. Although astrocytes are considered more resistant...
to the toxic effects of lead with chronic low doses of exposure, the acute high doses might cause failure of astrocytic resistance and the death of both neurons and astrocytes [25].

In the current work, electron microscopic study of the lead-administered group showed that some pyknotic nerve cells and myelinated nerve fibers had a disrupted contour. Some axons were distended with loss of the neuroplasm and degenerated organelles. However, the myelin sheaths around the deformed axons appeared abnormal, showing separation, discontinuity, and outfolding. These demonstrated changes were in agreement with the observations of other researchers, who suggested that chronic lead exposure may have an impact on brain development by impairing myelin production due to perturbation of the early development of oligodendroglial progenitors. Also, lead may exert a direct effect on the myelin sheath and alter its stability both by altering the lipid contents of membrane and by interfering directly with the lamellar structure. Wherever, galactolipids are the major lipid components of the myelin and myelin-competent oligodendrocytes, lead decreased the levels of galactolipids and theoligodendrocyte marker CNPase (2,3-cyclic nucleotide 3-phosphohydrolase). It is further suggested that the perturbation of the galactolipid pathway during developmental maturation of the oligodendrocytes may be a mechanism responsible for lead-induced neurotoxicity [26].

Also, oligodendrocytes in the hippocampus showed an irregular dense nucleus with clumped chromatin and a dense cytoplasm that contained numerous vacuoles. These findings were in agreement with the findings of some authors, who obtained similar results in their experimental work to detect the effect of lead exposure on these cells in neonatal rats [27].

The mechanisms of lead neurotoxicity are still not fully understood and cannot be tied together by a single unifying mechanism [28]. Another work reported that lead had direct and indirect neurotoxic actions but the ability of lead to substitute for calcium was a major common factor in many of its toxic actions. The direct neurotoxic actions of lead include apoptosis and disturbance of neurotransmitter storage or release processes [29].

Lead exerts a toxic effect on astrocytes, oligodendrocytes, and blood vessels, causing its congestion. Lead exposure delays the differentiation of glial progenitors and causes hypomyelination and demyelination [30].

In the present study, as can be seen from the biochemical results of SOD, CAT, and MAD levels, the ameliorative effect of antox on lead toxicity coincided with the results of some authors, who reported that the antioxidant antox led to an improvement in both histological and biochemical alterations of rats induced by the toxic herbicide paraquat [7,31]. The ameliorative potential of selenium in arsenic-mediated inhibition of ache revealed a positive role of selenium [20]. It was reported that lead increased the level of lipid peroxidation [17]. It can lead to inhibition of the activities of antioxidant enzymes, including glutathione peroxidase, CAT, and SOD [32]. Furthermore, the generation of reactive oxygen species and stimulation of lipid peroxidation allows the depletion of antioxidant reserves, which was postulated to be a major contributor to lead-exposure-related diseases [33].

Conclusion
All the previous data suggest that lead is one of the most widespread environmental toxins affecting young age persons today. Antox had a protective role against lead toxicity. Thus, it can be recommended as an important antioxidant drug in ameliorating the effects of lead toxicity as lead remains an environmental health concern. Efforts toward prevention of further contamination are reasonable and numerous steps should be taken to prevent occupational exposure to this metal.

Acknowledgements
There is no conflict of interest to declare.

References
تأثير الأنتوكس على تركيب الهيبوكامبس فى ذكور الفئران البيضاء المعرضة للتسمم بالرصاص (دراسة هستولوجية وبيوكيميائية)

أمية كامل هلال، أيمن محمد موسي و نادره قنديل

قسم الأنسجة وبيولوجيا الخلية - كلية الطب البشرى - جامعة بنها - وقسم الطبي الشرعي والسموم - كلية الطب البشرى - جامعة الزقازيق

يعتبر الرصاص من اخطر اسباب التلوث البيئى التى تهدد صحة البشر. وقد وجد أن الجهاز العصبى المركزى - خاصة المناطق التي تتطور بعد الولادة مثل الهيبوكامبس - من أكثر المناطق حساسية لذلك.

وقد استهدف هذا البحث دراسة الدور التحسينى لبعض مضادات الأكسدة (أنتوكس) على التغيرات الهستولوجية التي تحدث في الهيبوكامبس لذكور الفئران البيضاء نتيجة تعرضها لمياه ملوثة من الرصاص وعلى مستوى الإنزيمات المضادة للأكسدة.

أجريت دراسة على عينات من الفئران البيضاء مكونة من ثمانية فئران:

المجموعة الأولى: (الضابطة) تلقت الفئران الطعام والماء التنقيط.

المجموعة الثانية: أعطيت الفئران عقار الأنتوكس عن طريق الفم بجرعات 0.5% لمدة شهرين.

المجموعة الثالثة: تناولت الفئران ماء الشرب ملوثا بخلايا الرصاص بنسبة 0.5% لمدة شهرين.

المجموعة الرابعة: تناولت الفئران ماء الشرب ملوثا بخلايا الرصاص وأعطيت أيضاً عقار الأنتوكس بنفس الجرعات السابقة لمدة شهرين.

وقد أظهرت النتائج الهستولوجية وبيوكيميائية بين المجموعة الضابطة ومجموعة الأنتوكس تأثيراً واضحاً على الخلايا الهيبارية في الهيبوكامبس مع تدهور بعض الخلايا العصبية المتشكلة مع وجود كثرة واسعة للخلايا العصبية المتشكلة ونقص واضح للخلايا العصبية المتميزة.

وباستخدام الميكرسكوب الإلكتروني وجدت زيادة في نسبة الخلايا العصبية المتشكلة مع تدهور عدد الخلايا العصبية المتميزة ونسبة الخلايا العصبية المتشكلة.

ومقارنة نتائج المجموعات لم يكن هناك إختلافاً في النتائج الهستولوجية وبيوكيميائية بين المجموعات الداعمة ومجموعة الأنتوكس. ولقد لوحظ تأثيراً واضحاً للرصاص على الخلايا الخبيثة حيث نقص ظاهر في عدد الخلايا الهيبارية في الهيبوكامبس مع ظهور بعض الخلايا العصبية المتشكلة مع وجود كثرة واسعة للخلايا العصبية المتشكلة.

ووجدت زيادة في نسبة الخلايا العصبية المتشكلة مع تدهور عدد الخلايا العصبية المتميزة ونسبة الخلايا العصبية المتشكلة.

وقد أظهرت نتائج الأنتوكس تأثيراً إيجابياً على الخلايا العصبية والهليوبلازمية ونسبة الخلايا العصبية المتشكلة.

وباستخدام الميكرسكوب الإلكتروني وجدت زيادة في نسبة الخلايا العصبية المتشكلة مع تدهور عدد الخلايا العصبية المتميزة ونسبة الخلايا العصبية المتشكلة.

وقد لوحظ تأثيراً إيجابياً على الخلايا العصبية والهليوبلازمية ونسبة الخلايا العصبية المتشكلة.

وقد لوحظ تأثيراً إيجابياً على الخلايا العصبية والهليوبلازمية ونسبة الخلايا العصبية المتشكلة.

وهذا يؤكد أن تأثير الأنتوكس على تركيب الهيبوكامبس لدى الفئران البيضاء يمكن تحسينه عن طريق استخدام مضادات الأكسدة. وتشير نتائج الدراسة إلى أن تأثير الرصاص على تركيب الهيبوكامبس يمكن تحسينه عن طريق استخدام مضادات الأكسدة.