Research Article
Volume 5:S2, 2021

Journal of Morphology and Anatomy

ISSN: 2684-4265 Open Access

Neurodegenerative Changes in the Cerebral Cortex of Adult Wistar Rats Following Lead Induced Oxidative Damage

Ajibade AJ* and Akinrinade SO

Department of Anatomy, Ladoke Akintola University of Technology, Ogbomoso, Nigeria

Abstract

Lead is among the chemical compounds that have been reported to cause devastating problem worldwide. The primary site of action of lead is the central nervous system. This study investigated some effects of lead acetate on the cerebral cortex of adult Wistar rats. Thirty six adult Wistarrat of both sexes weighing between 120 g-250 g were randomly grouped into four groups of nine animals per group. Group A rats which was the control was maintained on standard feed and distilled water for 28 days, group B rats were treated with 10 mg/kg of lead acetate for 28 days, group C rats were treated with 20 mg/kg of lead acetate for 28 days and group D rats were also treated with 40 mg/kg of lead for 28 days. The lead acetate solution was administered orally on daily basis. The weights of the Wistar rats was recorded on weekly basis using a sensitive balance (before and during the weeks of administration). On the 28th day of the treatment the Wistar rats in group A, B, C and D were sacrificed by cervical dislocation, the brain was removed and weighed immediately using a sensitive balance, part of the brain was collected and homogenized for biochemical analysis for MDA.GSH and NO, the remaining part was then fixed in 10% formol calcium, the tissue was processed and sectioned at 5µm and stained with Haematoxylin and Eosin for histological study. The morphometric result showed that the mean body weights of the Wistar rats reduced significantly ($P<0.05$) in group B,C and D which received 10 mg/kg, 20 mg/kg and 40 mg/kg of lead acetate respectively as compared with Group A which reduced insignificantly ($P>0.05$). The brain weights in group B, C and D also decreased insignificantly ($P>0.05$) when compared with group A (control group). In the biochemical analysis there was statistically significant increase ($p<0.05$) in the level of Malondialdehyde (MDA) and Nitric Acid (NO) and statistically decreased level of Glutathione (GSH) in group B, C and D as compared with group A. Histological study of the cerebral cortex revealed that the cerebral cortical layers in group B, C and D appeared distorted and degenerated and in a dose dependent manner as compared with group A which shows a normal cerebral cortical architecture. This study concluded that lead has a neurotoxic effect on the cerebral cortex of adult Wistar rats which may adversely affect some cerebral functions

Keywords: Lead acetate • Cerebral cortex • Morphometric • Histological changes • Neurodegeneration • MDA • NO • GSH

Introduction

Man is exposed to various types of environmental contaminants at different stages of his life span, of which many of them are harmful. Lead is a heavy soft metal which occurs in nature either as an oxide or as a salt. Lead is a ubiquitous environmental and industrial pollutant that has been detected in every environmental and biological system. It came into use very early in the history of civilization and its poisonous effects were discovered.

Lead (Pb) is ubiquitous and one of the earliest metals discovered by the human race. Unique properties of lead, like softness, high malleability, ductility, low melting point and resistance to corrosion, have resulted in its widespread usage in different industries like automobiles, paint, ceramics, plastics, etc. This in turn has led to a manifold rise in the occurrence of free lead in biological systems and the inert environment [1].

Lead is considered as one of the most hazards and cumulative environmental pollutants that affect all biological systems through exposure to air, water, and food sources [2]. The exposure to lead induces clinic pathological changes through toxicity occurring in kidney and endocrine system [3]. A high level of lead in animals resulted in reproductive failure [4]. Lead is one of the global environmental pollutants, mainly found widely in industrial regions, as such that animals can easily be exposed to lead. Lead poisoning particularly in animals can be found from numerous sources in the general environment, and this can be traced back from contamination of feed, and soil from industrial pollution and agricultural practices. Furthermore, consuming high amount of lead has resulted in poor performance, poisoning, and death in animals [5]. Accumulated lead is toxic in most of its chemical forms, whether it is inhaled or ingested in water or feed. The extent to which orally administered lead absorbed into the host is small. However, due to its slow rate of elimination, harmful levels of lead can accumulate in tissues after prolonged exposure to low quantities [6].

The cerebrum is the largest part of the brain consisting of two parts and is separated by a deep midline sagittal fissure, the longitudinal cerebral fissure. The fissure contains the sickle-shaped fold of dura mater, the falxcerebri, and the anterior cerebral arteries. In the depths of the fissure, the great commissure, the corpus callosum, connects the hemispheres across the midline [7]. Each cerebral hemisphere is divided for descriptive purposes into four lobes, each of which is related to, but the boundaries of which do not correspond to, the overlying bones of the same name. From a superior view, the cerebrum is essentially divided into quarters by the median longitudinal cerebral fissure and the coronal central sulcus. The central sulcus separates the frontal lobes (anteriorly) from the parietal lobes (posteriorly) [8].

Materials and Methods

The Animal house of Anatomy Department of the Faculty of Basic Medical Sciences, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria served as the location for this study. Thirty-six healthy adult Wistar rats of both sexes weighing 120 g-250 g were obtained from the Animal House of the Department of Human Anatomy, Faculty of Basic Medical Sciences, Ladoke Akintola University Of Technology Ogbomoso. The animals were kept and maintained in the laboratory for two weeks to acclimatize prior to the study. The animals were treated in accordance with the 'Guide for the Care and use of Laboratory animals' complied by the National Academy of Science and Published by the National Institute of Health 1985. The body weighs of the experimental animals were monitored and weighed with a sensitive weighing scale and the values were recorded. The body weights were taken during acclimatization periods and during administration so as to monitor and study the effect of administration of

***Address for Correspondence:** Dr. Ajibade AJ, Department of Anatomy, Ladoke Akintola University of Technology, Ogbomoso, Nigeria; Email: adeshinaajibade@yahoo.co.uk*

Received: August 17, 2021; **Accepted:** August 31, 2021; **Published:** September 07, 2021

lead on the weight of the adult Wistar rats. The experimental animals were divided into four groups based on their weight range:

Group A (Control Group): were given normal feed and distilled water dosage for 28days

Group B: were given 10 mg/kg of lead and normal feed for 28 days

Group C: were given 20 mg/kg of lead and normal feed for 28 days

Group D: were given 40 mg/kg of lead and normal feed for 28 days

Lead administration to the rats was done using oral route of administration. The animals were sacrificed on the 28th day of the experiment by cervical dislocation which temporarily rendered the animals unconscious. The cereberal cortex of each rat was carefully harvested and weighted with a sensitive balance, part of it was homogenized for biochemical analysis (GSH, MDA and NO), and the remaining part was then fixed in 10% formol saline.

The statistical analysis of the results obtained in this study was evaluated and tested for significance using Student's t-test. If P-value of t-test was less than 0.05 (P<0.05), then result was significant. If P-value of the t-test was greater than 0.05 (P>0.05), then that means that the result is not significant. All data were expressed as Mean ± SEM.

Results

The table revealed the body weights of Wistar rats which started decreasing from the control group (A) which decreased from mean value of 223.3 ± 8.94 in the beginning to 199 ± 7.67 at the end (Table 1).

Group B which received a low dose of lead acetate (10 mg/kg) shows insignificant decreased (P>0.05) in body weights of the Wistar rats from mean value of 225.3 ± 7.42 in the beginning to 187.7 ± 11.59 at the end as compared with the control (group A).

Group C which received a medium dose of lead acetate (20 mg/kg) shows statistical significant decreased (P<0.05) in body weights of the Wistar rats from mean value of 187.60 ± 6.27 in the beginning to 186.30 ± 5.18 at the end when compared with the control (Group A). Group D which received a high dose of lead acetate (40 mg/kg) shows statistical significant decreased (P<0.05) in body weights of Wistar rats from mean value of 152.20 ± 12.38 in the beginning to 147.10 ± 5.29 at the end when compared with the control (Group A).

The graph above shows insignificant reduction (P>0.05) in the body weights of Wistar rats in group A and statistical significant reduction (P<0.05) in group B, C and D during the administration of lead (Figure 1).

It shows a decrease in final body weights of the animals in all groups. The initial and final weights in Group B is not statistically significant (P>0.05) when compared with group A, the initial weight in Group C shows a statistically significant (P<0.05) decrease when compared with Group A, and the initial and final weight in Group D shows a statistically significant (P<0.05) decrease when compared with Group A which was the control group (Table 2).

Graph shows a significant reduction (P<0.05) in the initial and final weights in group B, C and D as compared with group A (Figure 2).

It shows insignificant (P>0.05) decrease in the brain weight in Group B, C and D as compared with Group A (Table 3).

The graph shows insignificant (P>0.05) decrease in brain weight in Group B, C and D as compared with Group A (Figure 3).

The table revealed increase in the level of MDA in the treated groups when compared with the control, it increased significantly (P<0.05) from 0.92 ± 0.11 to 1.45 ± 0.10 in group B, 1.40 ± 0.08 in group C and 1.34 ± 0.02 in group D.

The level of GSH reduced significantly (P<0.05) in the treated groups compared with the control, it reduced from 5.54 ± 0.69 to 3.38 ± 0.62 in group B, 3.21 ± 0.36 in group C and 2.72 ± 0.02 in group D.

The level of NO increased significantly (P<0.05) in the treated groups compared with the control, it increased from 10.06 ± 0.79 to 13.21 ± 0.72 in group B, 14.59 ± 0.47 in group C and 16.14 ± 1.06 in group D (Table 4).

Table 1. Showing the mean ± sem of the body weights of Wistar rats before and during administration.

Period/Week	Group A	Group B (10 mg/kg)	Group C (20 mg/kg)	Group D (40 mg/kg)
Week 0	223.30 ± 8.94	225.30 ± 7.42	187.60 ± 6.27**	152.2 0 ± 12.38***
Week 1	229.30 ± 8.77	220.50 ± 7.49	186.40 ± 8.79**	142.30 ± 5.75**
Week 2	226.0 ± 8.45	203.70 ± 17.15	194.50 ± 7.38*	155.30 ± 5.06***
Week 3	216.40 ± 8.69	200.00 ± 9.09	178.00 ± 8.75**	141.50 ± 5.44**
Week 4	199.70 ± 7.67	187.70 ± 11.59	186.30 ± 5.18	147.10 ± 5.29***

Note: P<0.05, values greater than 0.05 were considered insignificant while values less than 0.05 were considered significant (*). Values were expressed as mean ± Standard error of mean.

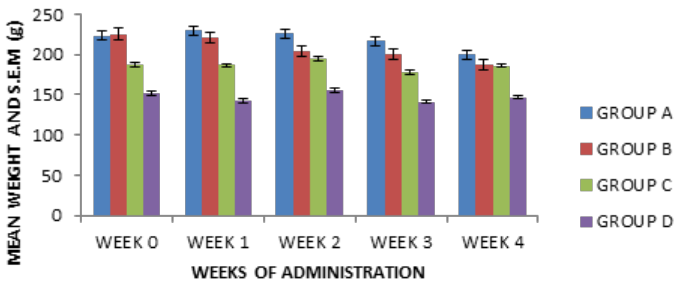

Figure 1. Histogram showing changes in the average body weights from the beginning of the administration to the end. Note: The graph above shows insignificant reduction (P>0.05) in the body weights of Wistar rats in group A and statistical significant reduction (P<0.05) in group B, C and D during the administration of lead.

Table 2. Showing the initial and final body weights analysis of Wistar rats.

Groups	Initial weight (g)	Final weight (g)	% Weight gain or loss
A	223.30 ± 8.94	199.70 ± 7.670	-23.6
B	225.30 ± 7.42	187.70 ± 11.59	-37.6
C	187.60 ± 6.27**	186.30 ± 5.18	-1.3
D	152.20 ± 12.38***	147.10 ± 5.29***	-5.1

Note: P<0.05, values greater than 0.05 were considered insignificant while values less than 0.05 were considered significant (*). Values were expressed as mean ± Standard error of mean.

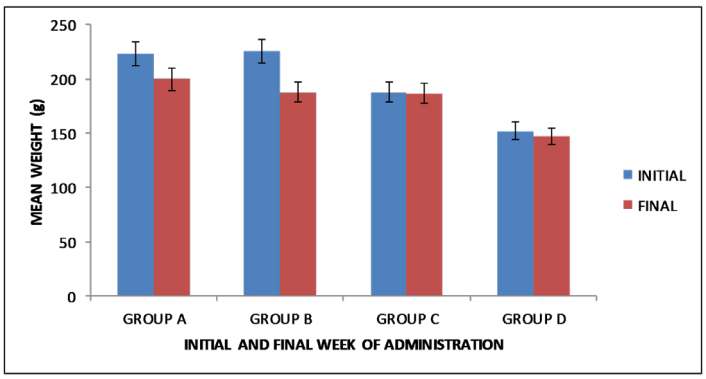

Figure 2. Histogram showing changes in initial and final body weights. Note: Graph above shows a significant reduction (P<0.05) in the initial and final weights in group B, C and D as compared with group A.

Table 3. Showing the mean ± sem of brain weights of adult wistar rats after administration of lead acetate.

Groups	Weight (g)	Relative weight of brain (%)
A (control)	2.05 ± 0.14	1.02
B (10 mg/kg)	1.72 ± 0.05	0.92
C (20 mg/kg)	1.57 ± 0.32	0.84
D (40 mg/kg)	1.53 ± 0.05	1.04

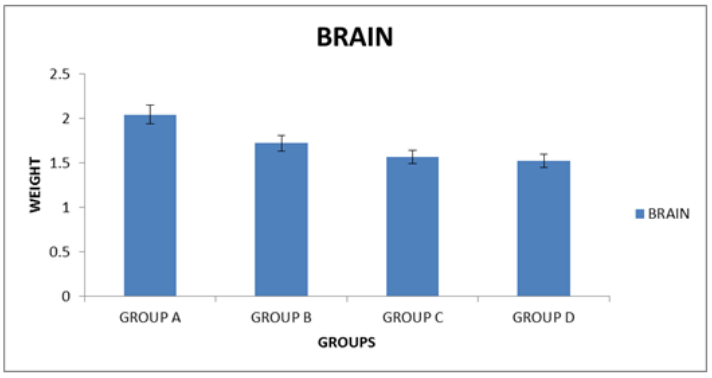

Figure 3. Histogram showing the mean ± sem weights of brain. Note: The graph above shows insignificant (P>0.05) decrease in brain weight in Group B, C and D as compared with Group A.

Table 4. Showing the effect of lead acetate on the levels of MDA, GSH and NO in the brain.

Groups	MDA	GSH	NO
A	0.92 ± 0.11	5.54 ± 0.69	10.06 ± 0.79
B	1.45 ± 0.10**	3.38 ± 0.62**	13.21 ± 0.72*
C	1.4 ± 0.08**	3.21 ± 0.36*	14.59 ± 0.47**
D	1.34 ± 0.02**	2.72 ± 0.02**	16.14 ± 1.06**

Note: P<0.05, values greater than 0.05 were considered insignificant while values less than 0.05 were considered significant (*). Values are expressed as mean ± Standard error of mean.

Abbreviations: Malondialdehyde (MDA); Glutathione (GSH); Nitric Oxide (NO).

The graph shows statistically significance (P<0.05) increase in the level of MDA in group B, C and D when compared with group A.

Group B, C and D shows a statistical reduction (P<0.05) in the level of GSH as compared with group A.

Group B, C and D shows a statistical reduction (P<0.05) in the level of NO as compared with Group A (Figure 4).

Histological observation

Representative micrographs of H and E staining showing the general cytoarchitecture of the cerebral cortex in Wistar rats in group A (control), Group B (Treated with 10 mg/kg of lead acetate for 28 days), Group C

(Treated with 20 mg/kg of lead acetate for 28 days), Group D (Treated with 40 mg/kg of lead acetate for 28 days). Magnification: X40, X100 respectively.

Normal histological features of the prefrontal cortex in group A characterized by large pyramidal neurons with long axons that extend well from the soma to adjacent neurons within the neuropil. Apical and basal dendrites extend from the well delineated soma of the pyramidal neurons in this group.

Group C-D treatments caused degenerative changes in the cortex that was characterized by clustered pyknotic pyramidal neurons that appear with fragmented cytoplasm and condensed nuclei within soma (red arrow). Perineural spaces can be seen surrounding degenerating neurons (red arrows) Axons and dendrites are scarcely appreciable around neurons in this groups.

Plate A: photomicrographs showing a normal histological feature of the cerebral cortex, characterized by large pyramidal cell, with long axons that extends well from the delineated soma of the pyramidal neurons, normal molecular layers and external granular layer also appear normal. (H and E X40, X100) (Figure 5).

Plate B: photomicrographs shows a slightly mild generative changes in the pyramidal cell, which appear slightly distorted with loss of their process, mild generative changes occur in the cytoplasm with condensed nuclei (red arrow) , molecular layer is similar to that of group A (H and E X40, X100) (Figure 6).

Plate C: Photomicrographs shows loss of pyramidal cells due to degeneration (red arrow), leading to plenty of perineural spaces (blue arrow), molecular layers appear unorganized with lots of spaces, cell distortion was very obvious. (H and EX40, X100) (Figure 7).

Plate D: photomicrographs shows severe degeneration in fragmented cytoplasm, condensed nuclei within soma, very large and numerous perineural spaces (red arrow) surrounds the degenerating neuron, spaces within the pyramidal cells and granular layer is observed. (H and EX40, X100) (Figure 8).

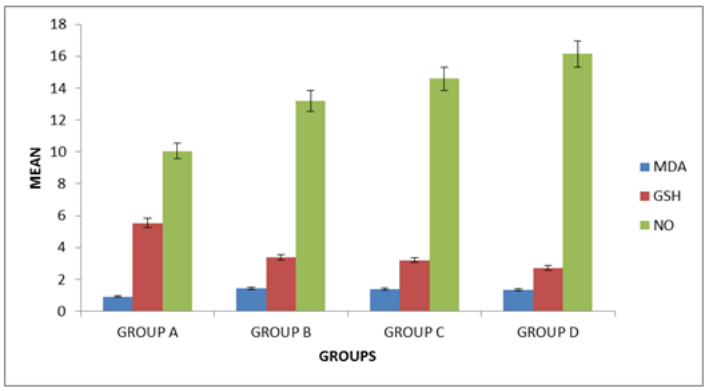

Figure 4. Histogram showing the changes in level of MDA, GSH and NO. Note: The graph above shows statistically significance (P<0.05) increase in the level of MDA in group B, C and D when compared with group A. Group B, C and D shows a statistical reduction (P<0.05) in the level of GSH as compared with group A. Group B, C and D shows a statistical reduction (P<0.05) in the level of NO as compared with Group A.

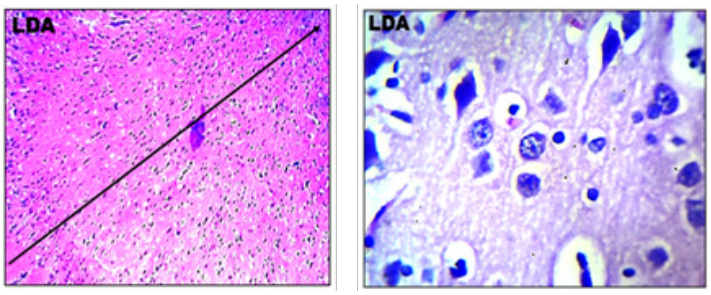

Figure 5. Control group.

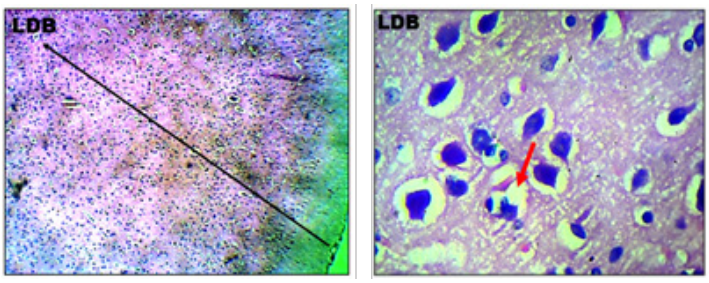

Figure 6. Treated with 10 mg/kg of lead acetate for 28 days.

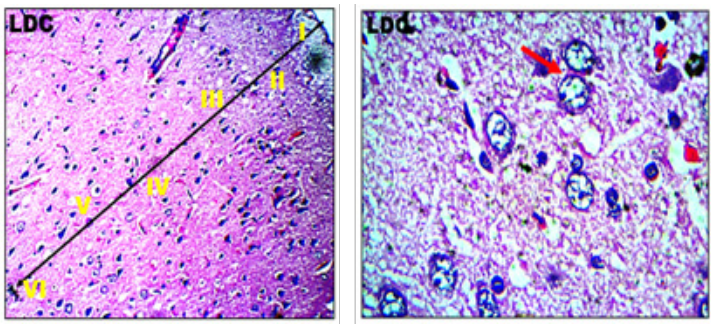

Figure 7. Treated with 20 mg/kg of lead acetate for 28 days.

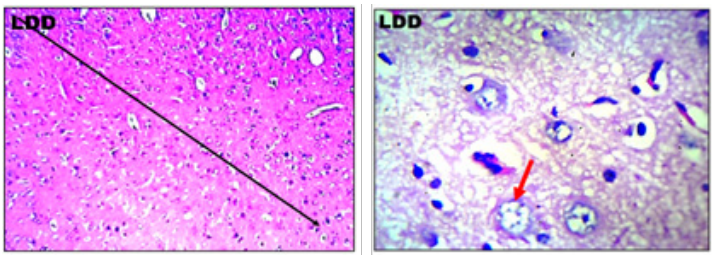

Figure 8. Treated with 40 mg/kg of lead acetate for 28 days.

Discussion

This study investigated the histomorphological effects of lead acetate on the cerebral cortex of adult Wistar rats, result of body weights analysis showed an insignificant increase (P>0.05) in the final total body weights of rats in group A, there was also an insignificant reduction (P>0.05) in body weights of rats in group. By which received 10 mg of lead acetate as compared with group A. Group C and D rats which received 20 mg/kg and 40 mg/kg of lead acetate respectively reduced significantly (P<0.05) as compared with group A. The loss in the body weights in this study may be explained on the basis of anorexia (loss of appetite) which is induced by heavy metal ingestion comparing the result obtained with the previous work [9]. Another possible explanation for the loss of body weights may be the decreased muscle mass and cachexia due to the oxidative stress induced by lead. Previous report has indicated that heavy metal toxicity is associated with oxidative stress [2]. Which is according to associated with muscle wasting and cachexia leading to low body weights as compared with the previous work done by [10] Rats treated with lead acetate has been reported to show an insignificant decreased (P>0.05) in their brain weights compared to group A (control) although not dose dependent, however reported that lead has a negative effect on brain weights when he compared the lead treated rats with the control after sacrificed.

The results of biochemical parameters investigated shows a significant increased (P<0.05) in level of MDA (Malondialdehyde) in group B, C And D that received 10 mg/kg, 20 mg/kg and 40 mg/kg of lead acetate respectively as compared with group A and this findings is supported by a work previously carried out [11,12]. The level of NO (nitric Oxide) in group B,C and D that received 10 mg/kg, 20 mg/kg and 40 mg/kg of lead acetate also increased significantly (P<0.05) as compared with group A. The level of GSH (Glutathione) in group B,C and D that received 10 mg/kg, 20 mg/kg and 40 mg/kg of lead acetate increased significantly (P<0.05) as compared with group A. MDA and NO are indicator of oxidative stress while GSH has a role as an antioxidant by reducing free radicals directly or as a cofactor of antioxidant enzymes such as glutathione peroxidase and glutathione transhydrogenase. However the results of this biochemical assay has a similarity with the previous work done by [13].

Malondialdehyde (MDA) is a compound that describes the activity of free radicals in cells so that it is used as one of the indications of oxidative stress caused by free radicals [14]. Another study reinforces this statement by stating that the mediator Malondialdehyde (MDA) is a final product of fat peroxidation which is used as a biological biomarker of fat peroxidation and can describe the degree of oxidative stress [15].

Glutathione is a tripeptide consisting of glutamic acid, cysteine, and glycine. The compound has a Sulfhydryl /Thiol group (-SH) found in the amino acid cysteine. The sulfhydryl group causes GSH to act as a powerful electron donor (nucleophile) in counteracting free radicals. GSH works to counteract these free radicals to prevent or reduce cell damage the significant reduction in GSH level in a dose dependent manner in this study maybe associated with its free radicals scavenging characteristics nature of GSH in relation to increase lipid peroxidation.

MDA, NO and GSH levels have a strong significant correlation and have a reciprocal Relationship which means that the higher the MDA and NO level, the lower the level of (GSH) glutathione in the body. This has similarities with another study which states that there is an increase in MDA levels and a decrease in Glutathione levels in cement workers [16]. Conversely, MDA, NO and GSH have a strong correlation with the free radicals increase generation of free radicals through Lead metaboloism that becomes free radicals (MDA,NO), the free radicals will directly interfere with the work of biotransformation enzymes (detoxification) of GSH.

Microscopic examination of the cerebral cortex revealed a normal tissue architecture in group A(control group), B (treated with 10 mg of lead acetate) shows slight and mild distortion in the architecture of the cerebral cortex, group C and D that received 20 mg and 40 mg of lead acetate respectively shows a more prominent and significant damage in the cerebral cortical layers, it was also observed that this damage on rats brain in various groups was dose dependent which has been similarly reported by previous studies [17]. Additionally, the findings from this study have indicated that lead acetate can induce diffusion and widespread neurosis of cortical neurons.

In the control (Group A) at the magnification of X40; neurons and glia are observable as they are distributed in a somewhat particulate pattern across the layers of the cerebral cortex. Large pyramidal neurons are relatively prominent. At the 100X magnification neurons appear morphologically normal and healthy as they are predominantly monomorphic within layers and/or regions. Glia- astrocytes especially, population also supports the above observation as astrocyte population and morphology appear normal. Oligodendrocyte and microglia morphology also appear normal. These observations also rule out any possibility of gliosis- an important

indication or neuronal damage or 'ill health'. There is no vacoulation in the neuropil- ruling out neuronal loss or myelin or cell processes damage. The control group therefore presents a healthy and histomorphologically normal cerebral cortex. In Group B, C and D, neurons are barely distinct and observable at the lower\Magnification (X40) was a sign of cytological disruption. At the higher magnification(X100), it is clear that the vacoulations are due to extensive damage which is dose dependent not only to neurons but also to the glia and the extensive processes of the cells. Individually, the neurons appear to be undergoing degeneration as cell bodies are poorly stained, with distorted morphology exhibiting features of acute eosinophilic neuron degeneration [18].

Glial astrocyte population appears to have increased in reactions to neuronal damage. Oligodendrocytes are almost not distinguishable; a sign that suggest axonal degeneration. Microglias too are barely distinguishable. The observed vacoulations are therefore due to extensive loss of adjacent neurons, their processes and surrounding supportive glia cells. This is a clear indication of lead toxicity deleterious effects as it is also seen in the biochemical analysis results where lead caused increase in the level of MDA, NO and a decrease in the amount of the antioxidant GSH. This is associated with previous work done by [19,20].

Conclusion

In conclusion, the findings of this study shows that exposure to lead acetate could be a risk factor inducing cellular damage and neuradegeneration in diseases. Neurodegeneration is the progressive loss of construction or capacity of neurons, which may at last include cell passing. Numerous neurodegenerative illnesses, for example, amyotrophic sidelong sclerosis, various sclerosis, Parkinson's infection, Alzheimer's sickness, Huntington's illness, and prion sicknesses-happen because of neurodegenerative cycles. Neurodegeneration can be found in the mind at various degrees of neuronal hardware, going from atomic to systemic. In the search for viable medicines (instead of palliative consideration), examiners utilize creature models of infection to test expected remedial specialists. Model creatures give a reasonable and moderately fast intend to perform two primary capacities: target distinguishing proof and target validation.

References

1. MFlora, Swaran JS, Flora Govinder and Saxena Geetu. “Environmental Occurrence, Health Effects and Management of Lead Poisoning.” In: Chemistry, Analytical Aspects, Environmental Impact and Health Effects. José SC, José S, (eds), Philadelphia, United States. (2006): 158-228.
2. Patra, Ramesh Chandra, Rautray Amiya K and Swarup D. “Oxidative Stress in Lead and Cadmium Toxicity and its Amelioration.” *J Vet Intern Med* 11 (2011): 457327.
3. Jadhav, Sachin H, Sarkar Souvendra Nath, Patil Rajreddy and Tripathi Harish Chandra. “Effects of Subchronic Exposure via Drinking Water to a Mixture of Eight Water Contaminating Metals: A Biochemical and Histopathological Study in Male Rats.” *Arch Environ Con Toxicol* 53 (2007): 667-677.
4. Ahamed, Maqusood and Siddiqui Mohd K Kaleem J Javed. “Environmental Lead Toxicity and Nutritional Factors.” *Clin Nutr* 26 (2007): 400-408.
5. McDowell, LR. ‘‘Minerals in Animal and Human Nutrition’’. *Philadelphia, United States*. (2003)
6. Ercal, N, Gurer Orhan Hande and Aykin Burns Nukhet. “Toxic Metals and Oxidative Stress Part. I: Mechanisms Involved in Metal-Induced Oxidative Damage.” *Curr Top Med Chem* 1 (2001): 529-539.
7. Richard S Snell. “Clinical Neuroanatomy for Medical Students.” *Philadelphia: Lippincott Williams and Wilkins*, (2001).
8. Moore, Keith L, Dally Arthur F and Agur Anne MR. “Clinically Oriented Anatomy.” *Philadelphia: Lippincott Williams and Wilkins*, (2010).
9. Klaassen, CD. “Heavy Metals and Heavy-Metal Antagonists.” In: Goodman and Gilman’s Pharmacological Basis of Therapeutics. McGraw-Hill, (eds), New York. (2001).
10. Canli, Mustaffa. “The Relationships between Heavy Metal (Cd, Cr, Cu, Fe, Pb, Zn) Levels and the Size of Six Mediterranean Fish Species.’’ *Environ Pollut* 121 (2003): 129-136.
11. Kumawat, Manjulata, Sharma Tarun Kumar, Singh Ishwar and Singh Neelima, et al. “Antioxidant Enzymes and Lipid Peroxidation in Type 2 Diabetes Mellitus Patients with and without Nephropathy.” *N Am J Med Sci* 5 (2013): 213-219.
12. Aydin Ahmet, Orhan Hilmi, Sayal Ahmet, and Ozata Metin, et al. “Oxidative Stress and Nitric Oxide Related Parameters in Type II Diabetes Mellitus: Effects of Glycemic Control.” *Clin Biochem* 34 (2001): 65-70.
13. Shraideh, Ziad, Badran Darwish, Hunaiti Abdelrahim and Battah Abdelkader. “Association between Occupational Lead Exposure and Plasma Levels of Selected Oxidative Stress-Related Parameters in Jordanian Automobile Workers.” I*nter J Occup Med Environ Health* 31 (2018): 517-525.
14. Asni E, Harahap IP, Prijanti AR and Wanandi S I, et al. “Effect of Hypoxia on Malondialdehyde Levels, Reduced Glutathione and Catalase Activity of Rat Kidneys.” *Indonesian Medicine Magazine*, 2009.
15. Rahardjani, Kamilah Budhi. “Relationship between Malondialdehyde (MDA) and Outcome of Neonatorum Sepsis.” *Sari Pediatri* 12 (2016): 82-87.
16. Orman, Ayse, Kahraman Ahmet, Çakar Hamdullah, and Ellidokuz Hulya, et al. “Plasma Malondialdehyde and Erythrocyte Glutathione Levels in Workers with Cement Dust-Exposure Silicosis.” *Toxicology* 207 (2005): 15-20.
17. Sanders, Talia, Liu Yiming, Buchner Virginia and Tchounwou Paul B. “Neurotoxic Effects and Biomarkers of Lead Exposure: A Review.” *Res Environ Health* 24 (2009): 15-45.
18. Garman, Robert H. “Histology of the Central Nervous System.” *Toxicol Pathol* 39 (2011): 22-35.
19. Owolabi, Joshua. “Moringaoleifera’s Ameliorates Lead Toxicity in Cerebral Cortex.” (2014).
20. Landrigan, Phillip J, Clyde B Schechter, Jeffrey M Lipton and Marianne C Fahs, et al.“Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities.” *Environ Health Perspect* 110 (2002): 721-728.

How to cite this article: AJ, Ajibade and Akinrinade SO “Neurodegenerative Changes in the Cerebral Cortex of Adult Wistar Rats Following Lead Induced Oxidative Damage” *J Morphol Anat* 5 (2021): 001.