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(RESEARCH ARTICLE)

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Lauric acid protects the cerebral cortex and hippocampus of adult male Wistar rat with lead induced neurotoxicity: Evidence of its anti-neuroinflammatory and anti-oxidative properties

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Abstract

***Background*:** The present study investigated the protective effect of lauric acid on the hippocampal and cerebral cortex of male wistar rats with lead-induced neuroinflammation and oxidative stress.

***Methodology*:** Thirty-five adult male wistar rats (150 – 200g) were divided into seven groups of experimental protocols: Food only, Pb^{2+} only, Lauric acid (LA) only, (500mg/kg LA + Pb^{2+}), (1000mg/kg LA + Pb^{2+}), (2000mg/kg LA + Pb^{2+}), and (0.2mg/kg Donepezil + Pb^{2+}). Cerebral cortex and hippocampus were protected against Pb^{2+} by oral pretreatment of lauric acid daily for 14 days, before oral inducement of neurotoxicity with 120mg/kg of Pb^{2+} daily for 7 days. After the experimental period, the animals were sacrificed via cervical dislocation, and blood samples were collected for neuroinflammatory and oxidative stress assays. Perfusion was made, and the brains were harvested, rinsed in normal saline, fixed in 10% formal saline, and subjected to histological studies.

***Results*:** There is a statistically significant increase ($p < 0.05$) in the serum level of TNF and IL-17, MDA with decreased SOD, Catalase in the Pb^{2+} treated animals without lauric acid intervention when compared with pretreatment with lauric acid and donepezil before inducing neuroinflammation and oxidative stress. Pretreatment with lauric acid significantly ($p < 0.05$) decreased the serum level of TNF, IL-17 and MDA, but significantly increased ($p < 0.05$) SOD and Catalase. This biochemical fact is supported by the histological results.

***Conclusion*:** There were comparable degrees of abatement as evidenced by the biochemical results, normal appearance of the pyramidal cells, with conspicuous apical and basal dendrites, and no signs of degeneration in the histology. Therefore, lauric acid has justified in this study that well-protected cerebral cortex and hippocampus may be the indication of its anti-inflammatory and antioxidative properties. Meanwhile, it is worthwhile to consider this aspect at a deeper level of investigation using different animal models and methods.

Keywords*:* Anti-Neuroinflammatory; Anti-oxidative; Lauric Acid; Cerebral Cortex; Hippocampus

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1 Introduction

Chronic neuroinflammation is a prominent risk factor for neurological dysfunction and progression of dementia, including Alzheimer's disease(AD), and other neurological diseases, Ezugwu et al., 2021; Hampel et al., 2020; Ashayeri et al., 2021; Li et al., 2021; Teleanu et al., 2022; Brockie et al., 2021; Kashif et al., 2021; Imam et al., 2016; Ezugwu et al., 2022. In rats, lead toxicity causes acute neuroinflammation and oxidative stress, which gradually progress to chronic neuroinflammation, that eventually results to neurodegeneration, Ezugwu et al., 2021; Ashayeri et al., 2020; Alvi et al., 2021; Panahi et al., 2019; Liu et al., 2021; Khan et al., 2020; Iqubal et al., 2020; Issa et al., 2020; Ahmed et al., 2013; Bhattacharyya and Bhunia, 2021. Consequently, learning deficits and memory loss occur, Habas, 2013; Amanzadeh et al., 2019; Taejib et al., 2022; Balogun et al., 2021. Of course, this can eventually disrupt, or even kill neurons, which are cells that transmit and process signals in the brain and other parts of the nervous system, Jarsky et al., 2005; Gorji, 2022; Ben-Azu et al., 2020; Taejib et al.,; Shi et al., 2012. Increased levels of heavy metals like Pb^{2+} in the body causes neurotoxicity, Zhang et al., 2017. Humans have been exposed to Pb^{2+} since ancient times. Lead is known to be a neurotoxicant that competes with and impairs calcium ion signaling in neuronal processes (Thangarajan et al., 2018). It degenerates and inhibits the differentiation of neurons, suppresses long-term potentiation (LTP), alters the secretion of neurotransmitters (Thangarajan et al., 2018) and triggers the production of β-amyloid proteins, Gundersen, 2021; Udomruk et al., 2021. Inflammatory cytokines like TNF-α and IL-1β can kill off dopaminergic neurons by damaging mitochondria and inducing cell death. Post-mortem analyses indicate that cytokines, including TNF-α and IL-1β, are significantly elevated in the area of the substantia nigra where maximal destruction of vulnerable dopaminergic neurons occurs in PD patients, which alters the secretion of neurotransmitters (Gundersen, 2021; Udomruk et al., 2021) and triggers the production of β-amyloid proteins. Oxidative stress has been linked with a variety of diseases, being involved in the debut and/or progress of several neurodegenerative disorders, (Teleanu et al., 2022). Findings correlate the overproduction of reactive oxygen species with the pathophysiology of Alzheimer's disease, Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis, (Teleanu et al., 2022).Lead ($Pb^{2+)}$ is one of the well-known environmental toxins that causes neuroinflammation and oxidative stress, affecting neurodevelopment and mental health, Zhang et al., 2017. Lead is a heavy metal that is widely used in various forms despite well-documented reports of its toxic effects, Ezugwu et al., 2021; Ezugwu et al., 2022; Thangarajan et al., 2018. Lead, as a neurotoxicant, crosses the blood-brain barrier to cause oxidative stress, (Barkur and Bairy, 2015; Ezugwu et al., 2022), morphologic damage, neurodegeneration and cognitive impairment in the brain, particularly that of the developing brain, 2015; Zhang et al., 2017.

The increasing popularity of lauric acid has led to new research in its clinical application apart from its role as a functional food oil; it has been shown that coconut oil has a lot of health benefits, Ezugwu et al., 2022; Zicker et al., 2018; Kappally, et al., 2015. Lauric acid has been shown to possess antihypertensive, antimicrobial, and anti-inflammatory properties, Kappally et al., 2015. Lauric acid (LA), a medium-chain triglyceride (MCT), with a melting point of (43.0 to 44.2oC), is widely acknowledged as a "healthier" saturated fat. MCT molecules can be rapidly absorbed by the body because of their ability to hydrolyze completely into fatty acids and glycerol by pancreatic lipase, Lappano et al., 2017. Lauric acid promotes neuronal maturation mediated by astrocytes in primary cortical cultures, Shingo and Hiroshi, 2020. Astrocyte-conditioned medium after LA treatment also enhanced the presynaptic protein levels in cortical neuron cultures, Cassidy et al., 2020. The intake of middle chain fatty acids (MCFAs) as a ketogenic diet has been suggested to have a therapeutic effect on mood disorders and cognitive dysfunction (Ahmed et al., 2021; Augustin et al., 2018). MCFAs such as capric acid, caprylic acid, and lauric acid (LA) are catabolized to ketone bodies. Interestingly, MCFAs not only provide ketone bodies but also protect cortical neurons against amyloid β-induced toxicity. Lauric acid prevents stress-induced depressive- and anxiety-like behaviors in rodents, González-Fuentes et al., 2018; Da Silva et al., 2018.

2 Materials and method

2.1 Materials

2.1.1 Experimental animals

Thirty-five (35) adult male wistar rats (150 – 205g) were procured and caged in a well ventilated animal house. They were divided into seven groups of experimental protocols (Table 2.2.1). Each group had five (5) rats. The experimental period lasted for 21 days, after which the animals were sacrificed via cervical dislocation, and blood samples were collected for neuroinflammatory and oxidative stress assays. Perfusion was made, and the brains were harvested, rinsed in normal saline, weighed, fixed in 10% formal saline, and subjected to histological studies.

2.1.2 Extracts, Drugs and Chemicals

All the extracts, drugs and chemicals used in this study were purchased from Nigerian markets, Nigerian chemical stores and Pharmacy, and they are of analytical standard.

2.1.2.1 Extract and Chemical

Extracts and chemicals were purchased from Okey Scientific Store, Enugu main market, Enugu, Enugu State, Nigeria. They were authenticated at the department of Applied Biochemistry, University of Nigeria, Nsukka.

2.1.2.2 Donepezil

Donepezil was purchased from Care-Root Pharmacy and Stores, Trans-Ekulu, Enugu, Enugu State of Nigeria.

Table 1 Experimental Protocol

Treatment	**Administration**
Food only	Received food and normal saline only
Control: Lead (Pb^{2+})	Received 120 mg/kg Pb^{2+} daily for 7 days
1000mg/kg Lauric acid (LA)	Received 1000mg/kg lauric acid for 14 days. Then, normal saline daily for 7 days.
500mg/kg LA + 120mg/kg Pb^{2+}	Received 500mg/kg lauric acid for 14 days. Then, 120 mg/kg Pb^{2+} daily for 7 days.
1000mg/kg LA + 120mg/kg Pb^{2+}	Received 1000mg/kg lauric acid for 14 days. Then, 120 mg/kg Pb^{2+} daily for 7 days.
2000mg/kg LA + 120mg/kg Pb^{2+}	Received 2000mg/kg lauric acid for 14 days. Then, 120 mg/kg Pb^{2+} daily for 7 days.
0.2mg/kg Donepezil + 120mg/kg Pb^{2+}	Received 0.2mg/kg donepezil for 14 days. Then, 120 mg/kg Pb^{2+} daily for 7 days.

2.2 Neuroinflammatory and Oxidative Stress Biomarkers

2.2.1 Determination of interleukin-17 (IL-17)

Test principle:

Sandwich-ELISA principle was used to determine the activities of interleukin-17 (IL-17) in the serum.

2.2.2 Determination of Tumor necrosis factor-alpha (TNF-alpha)

The method was based on the use of two monoclonal antibodies against TNF-alpha, one "capture" antibody and one labeled with biotin, in a "sandwich type" assay format.

2.2.3 Determination of superoxide dismutase activity

Superoxide Dismutase (SOD) activity was determined by a colorimetric method described by Fridovich, (1989).

Malondialdehyde (MDA): Was determined by a colorimetric method described by Fridovich, (1989).

2.2.4 Determination of catalase activity

Catalase activity was determined using the method described by Sinha (1972).

2.3 Wistar Rats Sacrifice

After 24 hours following the last administration, the animals were sacrificed by cervical dislocation, and the blood samples were collected via orbital puncture from each rat and put in a plain blood specimen bottle for biochemical

assay. The brain tissues were removed and fixed in buffered formalin solution. The specimens were taken to the histology laboratory for tissue processing (Highab et al., 2018).

2.4 Haematoxylin and Eosin (H and E) Cresyl Fast Violet Staining Method

The methods of H and E and CFV staining were carried out according (Highab et al., 2018). Sections of the tissues were viewed under a light microscope, and photomicrographs were made using a digital am scope (MD 900) microscope.

2.5 Data Analysis

Data obtained were expressed as mean ± SEM (standard error of mean). One-way analysis of variance (ANOVA) was used to compare the mean differences. Tukey's post hoc test was done where the result was significant. P-value less than to 0.05 was considered statistically significant. All results were analyzed using the Statistical Package for Social Sciences (SPSS version 22).

3 Results

3.1 Body and Brain Weight Results

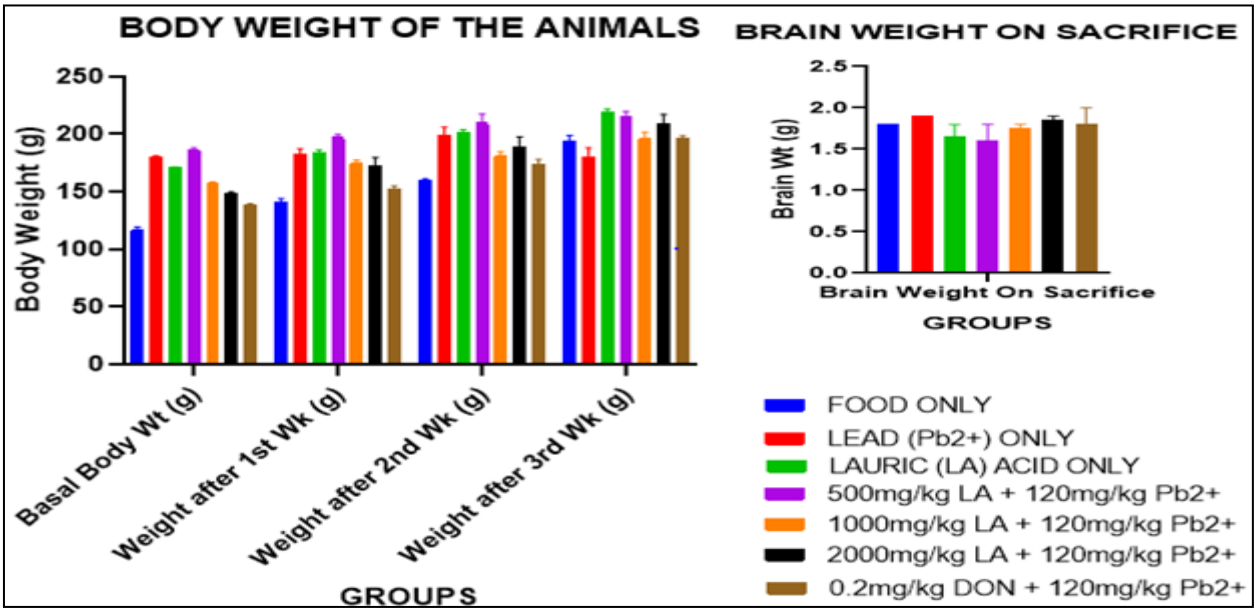


Figure 1 Body and brain weight of the animals

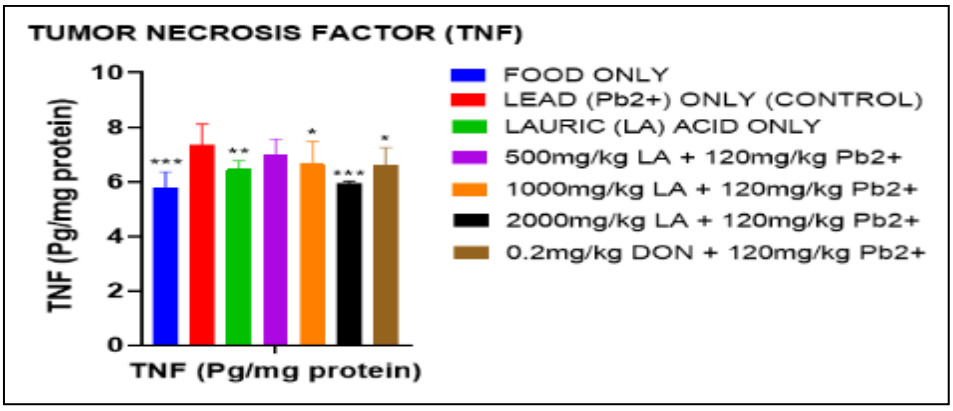
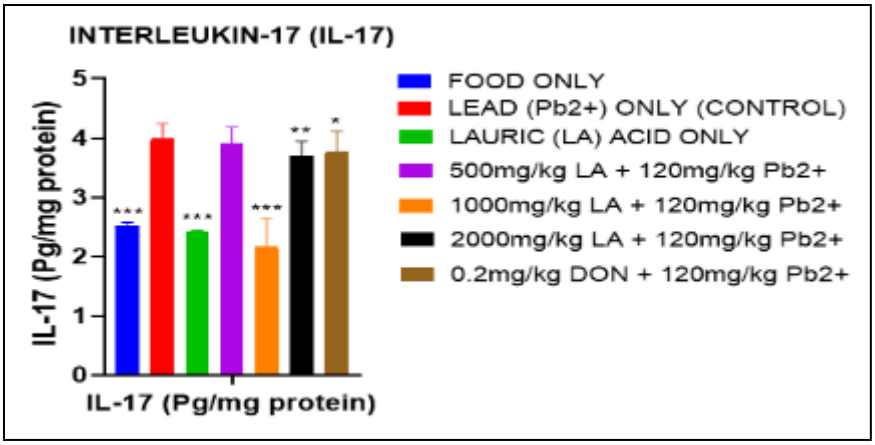


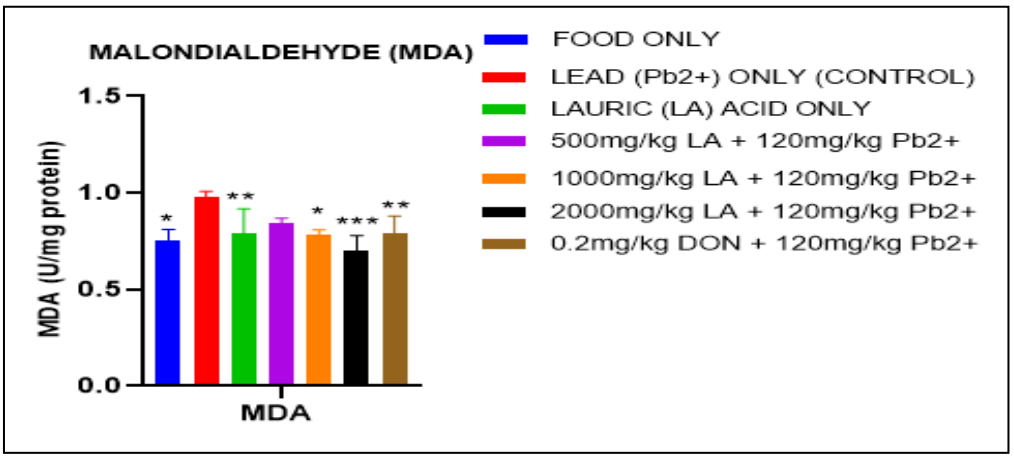
Figure 2 Tumor Necrosis Factor (TNF)

Values are mean ± SEM; n = 5 in each group, (P < 0.05) = Statistically significant.; * Significant when compared to the control at P < 0.05; ** Significant when compared to the control at P < 0.01; *** Significant when compared to the control at P < 0.001



Values are mean ± SEM; n = 5 in each group, (P < 0.05) = Statistically significant.; * Significant when compared to the control at P < 0.05; ** Significant when compared to the control at P < 0.01; *** Significant when compared to the control at P < 0.001

Figure 3 Interleukin-17 (IL-17)



Values are mean ± SEM; n = 5 in each group, (P < 0.05) = Statistically significant.; * Significant when compared to the control at P < 0.05; ** Significant when compared to the control at P < 0.01; *** Significant when compared to the control at P < 0.001

Figure 4 Malondialdehyde (MDA)

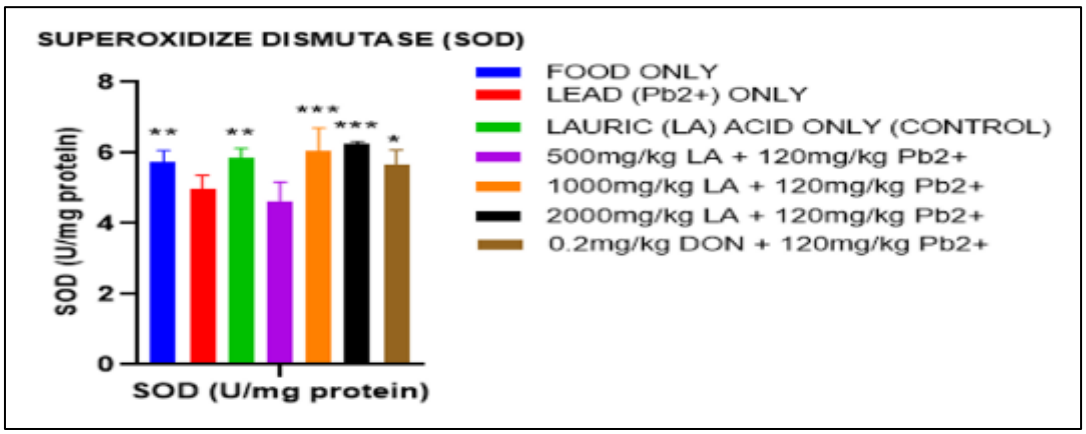
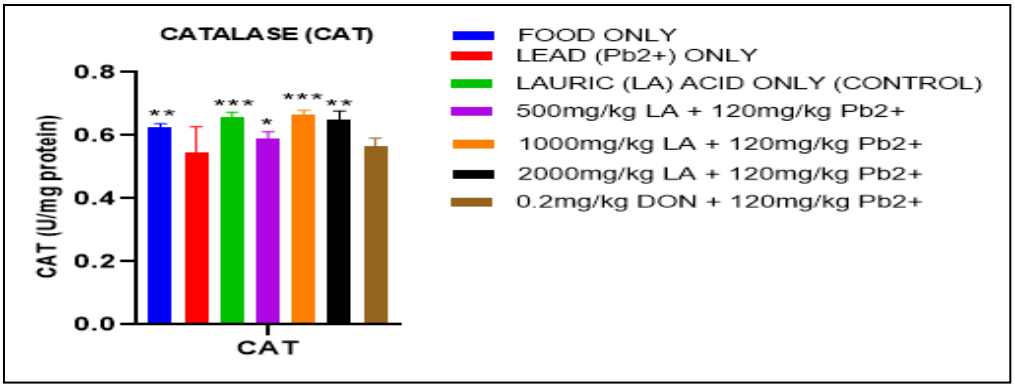


Figure 5 Superoxide Dismutase (SOD)

Values are mean ± SEM; n = 5 in each group, (P < 0.05) = Statistically significant.; * Significant when compared to the control at P < 0.05; ** Significant when compared to the control at P < 0.01; *** Significant when compared to the control at P < 0.001



Values are mean ± SEM; n = 5 in each group, (P < 0.05) = Statistically significant.; * Significant when compared to the control at P < 0.05; ** Significant when compared to the control at P < 0.01; *** Significant when compared to the control at P < 0.001

Figure 6 Catalase

3.1.1 Histology

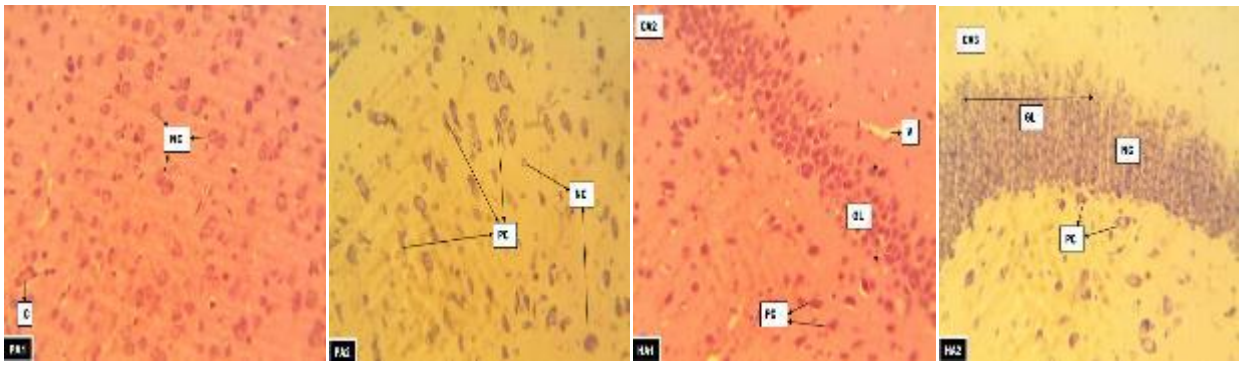


Figure 7 PA1&PA2 The prefrontal cortex normal control presents a normal neurons and shows normal appearance of neuroglial cells (NC); intensively stained normal pyramidal cells (PC) with conspicuous apical and basal dendrites, and no signs of degeneration (H&E stain, X400 and CFV stain, X400).

HA1&HA2: The hippocampus of normal control shows well-defined glandular cell layer (GL) with numerous neroglial cells (NC). Note the well-defined and packed pyramidal cells (PC) in the glandular cell layer (GL) of connus ammonus (CA3) with cytoplasmic Nissl granules. Neurons have features of characteristic large multipolar neurons (H&E stain, X400 and CFV X400).

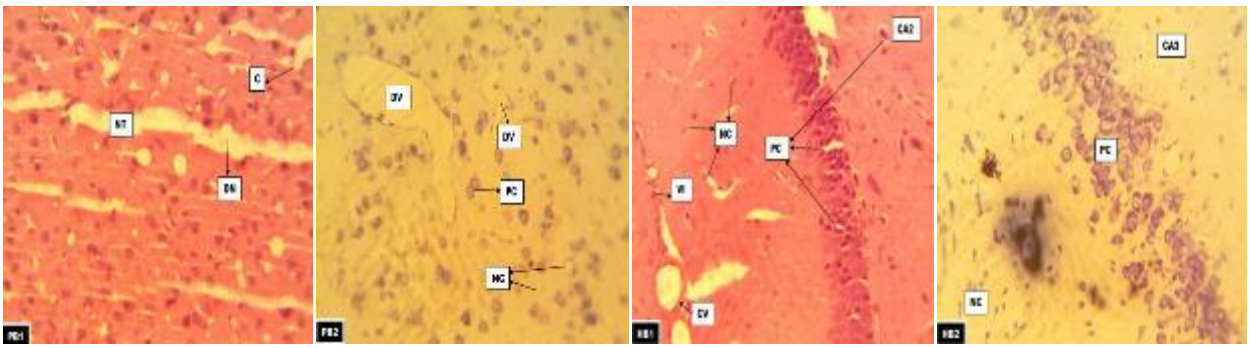


Figure 8 PB1&PB2 The prefrontal cortex (PFC) of Group B induced with lead without treatment (positive control) shows cells with condensed irregular nuclei and large degeneration and vacuolation (DV), and necrosis of neuroglial cells (NC), abnormal appearance of pyknotic pyramidal cells (PC) with chromatolysis as well as reduced apical and basal dendrites. There is a general sign of histopathological changes. (H&E X400 and CFV stain, X400).

HB1&HB2: The hippocampus of Group B induced with lead without treatment (positive control) shows shrunken neuroglial cells (NC) and necrosis of all layers with a zone of vacuolation deep to the pyramidal layer. The pyramidal

cells (PC) present chromatolysis and pyknosis with very reduced apical and basal dendrites. The pyramidal cells in the CA3 region of the hippocampus show pycnotic pyramidal cells organized in a cluster and apoptotic CA3 cells appear to be poorly stained as a sign of degenerative changes. The cellular assortment of the CA3 region of the hippocampus alongside the polymorphic layer adjoining it on either side appears distorted. (H&E X400 and CFV X400).

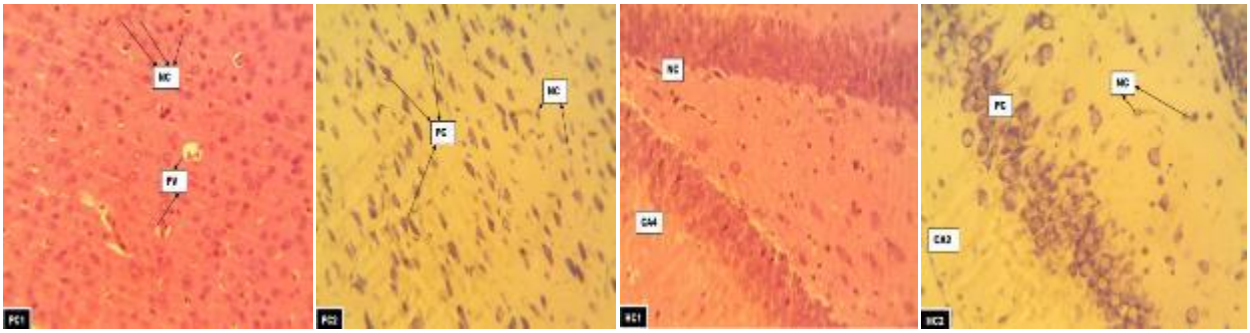


Figure 9 PC1&PC2 The prefrontal cortex treated with lauric acid only (negative control) presents normal neurons generally and shows normal appearance of neuroglial cells (NC); also intensively stained normal high density pyramidal cells (PC) with conspicuous apical and basal dendrites and no sign of degeneration (CFV stain, X400).

HC1&HC2: The hippocampus treated with lauric acid and normal saline (negative control for protective) presents a granular layer that is spaced out from CA2 to CA3 regions. presents normal neurons generally, and shows normal appearance of neuroglial cells (NC); also intensively stained normal high density pyramidal cells (PC) with conspicuous central nuclei, apical and basal dendrites, and no sign of degeneration (H&E stain, X400 and CFV stain, X400).

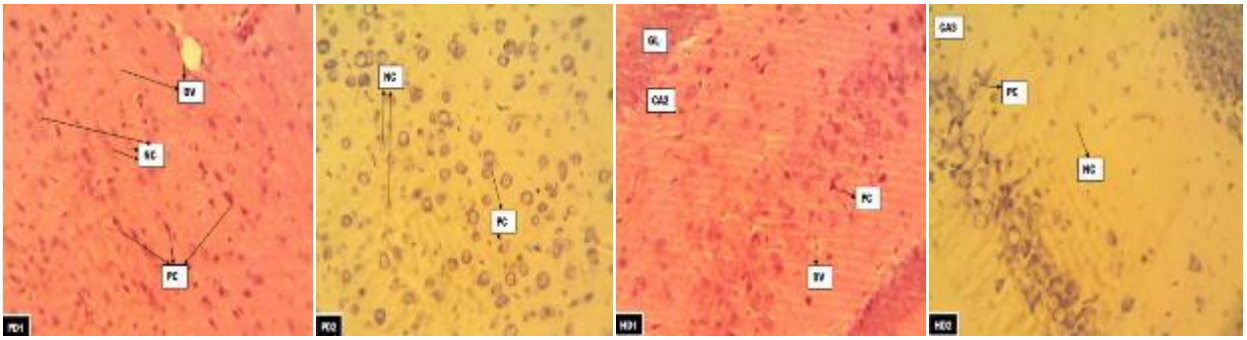


Figure 10 PD1&PD2 The prefrontal cortex treated with 500mg/kg (low dose) lauric acid and induced with lead (protective test) shows cells with condensed irregular nuclei and no peripheral vacuolation, and necrosis of neuroglial cells (NC), abnormal appearance of pyknotic pyramidal cells (PC) with chromatolysis as well as reduced apical and basal dendrites. There is a mild sign of histopathological changes (H&E stain, X400 and CFV stain, X400).

HD1&HD2: The hippocampus treated with 500mg/kg (low dose) lauric acid and induced with lead (protective test) generally shows mild chromatolysis (there are few chromatolytic cells). But the pyramidal cells (PC) present normal apical and basal dendrites in the granular cell layer. There is a decreased number of neuroglial cells (NC) especially in the molecular cell layer. (H&E stain, X400 and CFV stain, X400)

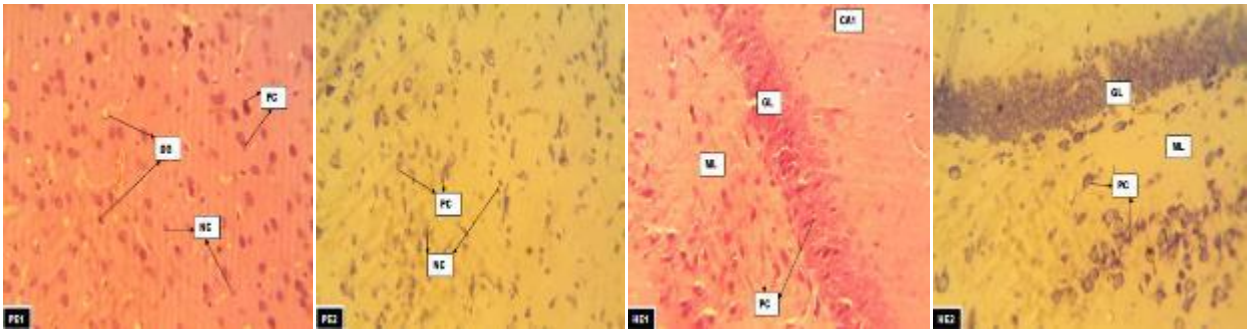


Figure 11 PE1&PE2 The prefrontal cortex treated with 1000mg/kg (medium dose) lauric acid and induced with lead (protective test) presents normal neurons generally, and shows normal appearance of neuroglial cells (NC); also intensively stained normal moderate density pyramidal cells (PC) with conspicuous apical and basal dendrites, and no sign of conspicuous degeneration (H&E stain, X400 and CFV stain, X400).

HE1&HE2: The hippocampus was treated with 1000mg/kg (medium dose) lauric acid and induced with lead (protective test) showed well-defined pyramidal cells (PC) with well-defined apical and basal dendrites in various layers (GL and ML). There is no sign of chromatolysis and pykinosis (H&E stain, X400 and CFV stain, X400).

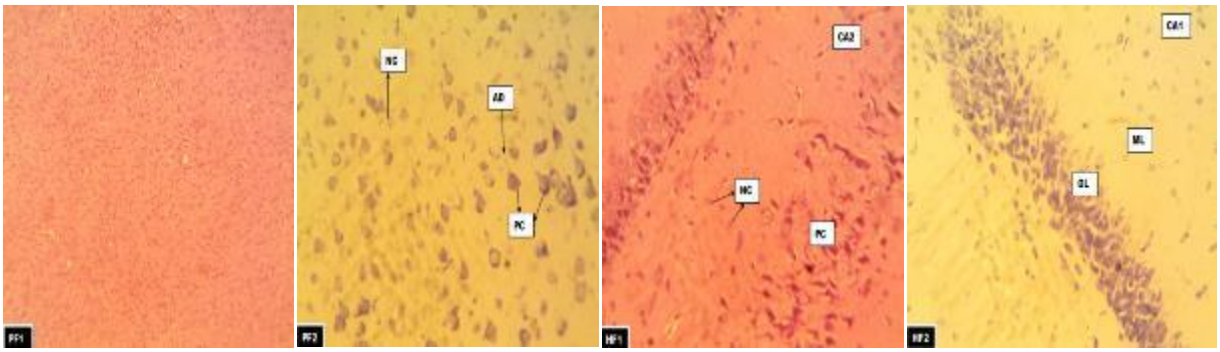


Figure 12 PF1&PF2 The prefrontal cortex treated with 2000mg/kg (high dose) lauric acid and induced with lead (protective test) showed increased cellular density and normal appearance of neuroglial cells (NC). Moreover intensively stained high density pyramidal cells (PC) with conspicuous apical (AD) and basal dendrites, and no signs of chromatolysis, pyknosis, or degeneration. (H&E stain, X100 and CFV stain, X400).

HF1&HF2: The hippocampus of Group F treated with 2000mg/kg (high dose) lauric acid and induced with lead (protective test) showed well-packed pyramidal cells in the granular layer (GL) with no obvious necrosis, chromatolysis and pykinosis. Moreover there is no vacuolation in the glial cells of the molecular layer (ML) and granular layer (GL), hence no obvious histopathology (H&E stain, X400 and CFV stain, X400).

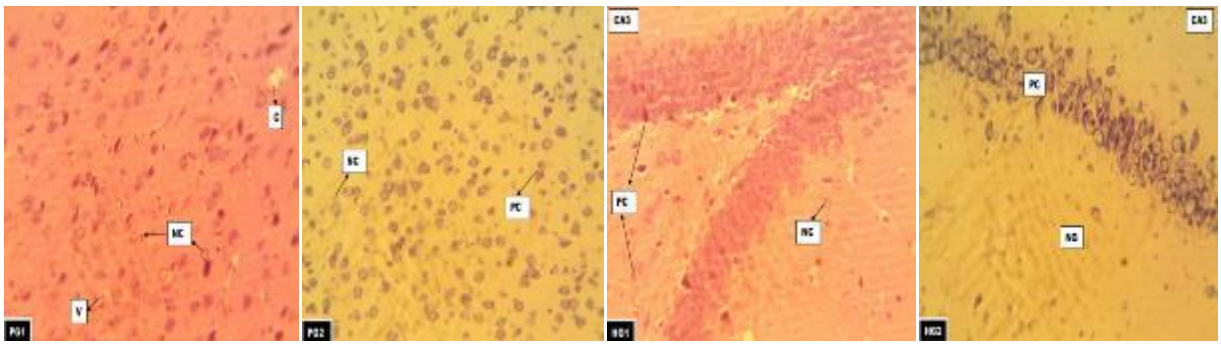


Figure 13 PG1&PG2 The prefrontal cortex treated with 0.2mg/kg of Donepezil and induced with lead (protective test) shows increased cellular density and normal appearance of neuroglial cells (NC), no sign of chromatolysis and pyknosis or degeneration but there is reduced apical dendrite, (H&E stain, X400 and CFV stain, X400).

HG1&HG2: The prefrontal cortex treated with 0.2mg/kg of Donepezil and induced with lead (protective test) shows distinctly stained pyramidal cells with apical and basal dendrites projecting out of the cell body, (H&E stain, X400 and CFV stain, X400).

4 Discussion

The body weights (Figure 1) of rats treated with (Pb^{2+}) significantly ($p < 0.05$) reduced when compared to the normal control, donepezil, and lauric acid groups. Our results are in agreement with Ahmed et al., 2013; Kumawat et al., 2014, who reported that mice ingested 1/20 LD50 of (Pb^{2+}) showed significant reduction in weight. In the same trend, Flora et al. (2012); Kasten-Jolly et al., (2012) reported a significant reduction ($p<0.01$) of mean body weight of mice exposed to Pb^{2+}. There is significantly ($p < 0.05$) decreased levels of SOD, and catalase in the Pb^{2+} treated rats, and increased level in lauric acid treated rats, which indicate that lauric acid has the potential that may have protected the hippocampus and cerebral cortex pyramidal cells from oxidative stress. This is in agreement with the reports of Imam et al., 2016 that SOD and Catalase decrease with oxidative stress. Also, there is significantly ($p < 0.05$) increased levels of MDA in the Pb^{2+} treated rats, and decreased level in lauric acid treated rats, There is positive correlation of oxidative stress with MDA, Imam et al., 2016. This is also in accordance with (Dayrit, 2015) report on the anti-oxidative activity of lauric acid. There is significantly ($p < 0.05$) decreased serum level of IL-17 and TNF in the lauric acid treated rats when compared to the Pb^{2+} treated rats. There is positive correlation of tumor necrosis factor (TNF) and interleukin-17 (IL-17) with neuroinflammation. The above results indicate that the lauric acid has the potential that may have prevented the neuroinflammatory and oxidative activies in the nervous system. The administration of lauric acid may have prevented neuroinflammation as evidenced by the decreased level of TNF and IL-17 when compared with the lead acetate treated group. This is in accordance with the report of Ashayeri et al., 2020, that proinflammatory cytokines like tumor necrosis factor (TNF) and interleukin-17 (IL-17) levels increase with neuroinflammation. Moreover, Lingling et al., 2022; Khalil et al., 2018, reported a similar cases on the relationship between neurofilaments and neurological disorders. Teleanu et al., 2022 reported that decreased level of proinflammatory cytokines like IL-17, TNF has a negative correlation with neurotoxicity and increased level has a positive correlation.

So the lauric acid from coconut oil has the potential that may have protected the brain from the neurotoxicity. Abd Rashed et al., 2021, and Ahmad et al., 2015, reported that some essential oil have pharmacological and neuroprotective properties. So our report is in accordance with Abd Rashed et al., 2021, and Ahmad et al., 2015, that lauric acid as an essential medium chain fatty acid (MCFA) has a potential that may have protected the pyramidal cells from neurological assault. There is a significant ($p < 0.05$) increase in the number of pyramidal cells in normal control, lauric acid, and donepezil treated rats when compared to Pb^{2+} treated rats. The histological reports of normal control, lauric acid, and donepezil treated rats presented good appearance of neuroglial cells (NC); intensively stained normal pyramidal cells (PC) with conspicuous apical and basal dendrites and no signs of degeneration when compared to the Pb^{2+} treated. These indicate that lauric acid has the potential that may have protected the hippocampal and cerebral cortex from neuroinflammation and oxidative stress. This is in agreement with the earlier report by Abd Rashed et al., 2021, that essential oils like lauric acid have a neuroprotective remedy for neurodegenerative diseases. This overall result is in agreement with Ezigbo and Mbaegbu, 2016, report on the medicinal benefits of lauric acid, and also in agreement with Abd Rashed et al., 2021, on the pharmacological and neuroprotective benefit of essential oil like lauric acid. The histological reports of normal control present normal appearance of neuroglial cells (NC); intensively stained normal pyramidal cells (PC) with conspicuous apical and basal dendrites and no signs of degeneration when compared to the Pb^{2+} treated rats without lauric acid protection (See plate 1-7: PA to PG and HA to HG). These indicate that lauric acid has potential that may have protected the hippocampal and cerebral cortex from neuroinflammation and oxidative stress. This is in agreement with the earlier report by Abd Rashed et al., 2021, that essential oils like lauric acid have an anti-inflammatory remedy for neurotoxicity. These overall results are in agreement with Ezigbo and Mbaegbu, 2016, report on the medicinal benefits of lauric acid, and also in agreement with Abuhamdah et al., 2015, report on the pharmacological and neuroprotective benefit of essential oil like lauric acid.

5 Conclusion

The results of this study suggest that lauric acid has therapeutic potential against neuroinflammation and oxidative stress associated with demyelination and neurotoxicity of hippocampal and cerebral cortex due to lead (Pb^{2+}) poison. The mechanism underlying this protection seems to involve, in addition to improved permeability of lauric acid across lipid bilayers and blood-brain barrier, and also as a ketogenic molecule. Several lines of data support this hypothesis, for example, cognitive disruption, such as that seen in humans including schizophrenic patients is known to involve alterations in the pro-inflammatory cytokines and anti-oxidants.

Findings

- There are balance in the concentration levels of biochemical parameters: TNF, IL-17, CAT, SOD, and MDA with lauric acid pretreatment.
- Lauric acid prevented neuroinflammation, oxidative stress, and histological alterations in the hippocampal and cerebral cortex, which indicates that lauric acid is a promising neurological remedy.

Recommendation

We, hereby recommend that lauric acid should be considered into clinical trial, to see if it can be helpful to people or patients with neurological disorders. Also, further studies on the exact molecular mechanisms responsible for these actions of lauric acid in the nervous system should be carried out. Meanwhile, it is worthwhile to consider this aspect at a deeper level of investigation using different animal models and methods.

Contribution to Knowledge

- Molecular mechanism associated with neuroinflammation and oxidative stress have been elucidated in this study.
- Lauric acid has anti-neuroinflammatory and anti-oxidant potential.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

Funding

Funded by individuals.

Dada availability

All data are available at, Department of Anatomy, University of Nigeria.

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