International Journal of Basic Science and Technology
June, Volume 11, Number 2, Pages 187 - 196 http://www.ijbst.fuotuoke.edu.ng
https://10.5281/zenodo.16673246 ISSN 2488-8648

Neuroprotective Effects of *Elaeis guineensis* (Red Palm Oil) Against Potassium Bromate-Induced Oxidative Stress and Neurotoxicity in Male Wistar Rats

[1]Adeogun, E.F. and [2]Omotayo, B. I.*
[1] Department of Biochemistry, Faculty of Life Sciences, University of Benin, Benin City, Nigeria
[2] Department of Biochemistry, Faculty of Science, Federal University Otuoke, Nigeria.

Article Information
Article # 100279
Received date:4th April. 2025
Revision:28th April 2025
Acceptance:14th May 2025
Published: 7th June, 2025

Key Words
Neuroprotective Effects
Elaeis guineensis
Potassium Bromate
Oxidative Stress

Abstract

Potassium bromate ($KBrO_3$), a food additive and environmental contaminant, is a known neurotoxin whose deleterious effects are primarily mediated through the induction of oxidative stress. *Elaeis guineensis* (red palm oil) is a natural product exceptionally rich in antioxidants, including tocotrienols and carotenoids. This study was designed to investigate the potential neuroprotective effects of *Elaeis guineensis* fruit oil against the neurobiochemical toxicity induced by oral administration of potassium bromate in male Wistar rats. Twenty-four male Wistar rats were randomly divided into four groups (n=6): a control group, a group receiving potassium bromate (100 mg/kg) only, 2 groups pre-treated with *Elaeis guineensis* oil (2 ml/kg and 4ml/kg) prior to potassium bromate administration. Following a 21-day administration period, brain tissues were homogenized and analyzed for key biochemical markers, including glutathione (GSH), malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), acetylcholinesterase (AChE), and nitric oxide (NO). Administration of potassium bromate alone resulted in a statistically significant ($p<0.05$) decrease in the brain concentrations of GSH, CAT, SOD, and AChE, coupled with a significant elevation in MDA and NO levels when compared to the control group. However, pre-treatment with *Elaeis guineensis* fruit oil significantly ameliorated these adverse effects, restoring the measured biochemical parameters to levels comparable with the normal control group. The findings demonstrate that *E. guineensis* fruit oil possesses potent neuroprotective properties against potassium bromate-induced toxicity. This protective capacity is likely attributable to its strong free-radical scavenging and antioxidant activities, which mitigate oxidative neuronal damage.

Corresponding Author: Omotayo, B. I.; ilesanmiob@fuotuoke.edu.ng

Introduction

The health challenge due to neuronal damage is enormous; the nervous system coordinates the various biochemical and physiological activities occurring in the body, thus any activity that alters the function of the nervous system is debilitating. The body is exposed to various environmental toxic chemicals that are harmful to the brain. Most of these toxic chemicals exert their harmful effect through oxidative stress, a process whereby the defence system of the body is compromised, leading to the generation of highly reactive species (Adewumi *et al*., 2017). This process often destroys the integrity of cells and tissues, including the brain (Ajarem *et al*. 2016; Shanmugavel *et al*. 2020). One of the major chemicals that human is exposed to is potassium bromate. In Nigeria, one of the major sources of exposure is in the baking industry, where it is used as an additive in bead making. Despite the attempt by the food regulatory agencies to discourage the use of bromate in the food industry (Chauhan and Jain 2016). The mechanism of $KBrO_3$ toxicity is mainly through the production of free radicals that are highly reactive within the biological systems. Increased production of these radicals puts pressure on the host antioxidant molecules, overwhelming them (Magomya and Yebpella, 2020; Olusola *et al*.,2024). This established mechanism of toxicity has provided a window of intervention by sourcing external antioxidant boosters to counter and eliminate the toxic effects of $KBrO_3$ in the brain (Ajarem *et al.,* 2016). The major antioxidant molecule in the brain is reduced glutathione, which provides electrons to radicals and makes them less reactive, thereby preventing their damage to brain cells and tissues (Ji *et al*., 2023). Thus, any activities that reduce the level of GSH can trigger brain injury and death. Lipid peroxidation is another process that is reported to be elevated in brain cells following oxidative stress. It involves the oxidation of lipids in the cell membrane, resulting in the loss of cellular integrity (Wang *et al*. 2023). One of the products of lipid peroxidation is malondialdehyde (MDA), and it is one

https://10.5281/zenodo.16673246 ISSN 2488-8648

of the major biomarkers used to evaluate oxidative stress. Apart from GSH, other endogenous antioxidants include superoxide dismutase and catalase, which are involved in neutralizing ROS (Ischiropoulos *et al*., 2003). Acetylcholinesterase plays an important role in neuronal architecture and efficiency. It acts as a neurotransmitter in the cognitive function of the brain; thus, any activities that alter its process can be a reflection of neuronal damage (Ilesanmi *et al*., 2022). Nitric oxide is a signalling molecule that performs several biochemical and physiological functions in the body; however, when it is elevated, it can be implicated in oxidative stress (Pacher *et al*., 2007). In an attempt to counter the toxic effects of $KBrO_3$, several antidotes have been investigated and prescribed as a remedy. *E. guineesis* fruit oil, commonly called palm oil in Nigeria, is widely produced in Nigeria, especially in the South-South region, such as Bayelsa and Edo states. It is a plant rich in antioxidant and anti-inflammatory natural compounds and has been prescribed as an antidote against poison and infection (Obahiagbon 2012). It is characterized by a rich biochemical composition that includes a high concentration of tocopherols and tocotrienols, which are different forms of vitamin E (Loganathan *et al*., 2017). It also contains significant amounts of carotenoids, precursors to vitamin A, as well as other bioactive compounds such as phytosterols and squalene. The presence of these compounds, particularly the vitamin E isoforms and carotenoids, positions palm oil as a potential source of protection against oxidative stress-related diseases, including those affecting the nervous system. Given that potassium bromate induces neurotoxicity through the generation of ROS (Ajarem *et al*., 2016), the inherent antioxidant capacity of palm oil suggests a plausible mechanism by which it could counteract these harmful effects in the brain. Existing research provides further support for the neuroprotective potential of palm oil or its components in various neurological contexts. For instance, studies have explored the effects of palm oil and its tocotrienol-rich fraction against stroke and in animal models of Alzheimer's disease, suggesting benefits in cognitive function and reduction of neurodegenerative markers (Sharif *et al*., 2025; Moorthy and Radhakrishnan, 2024). These findings, obtained in different models of neurological insult, strengthen the rationale for investigating the efficacy of palm oil against potassium bromate-induced neurotoxicity, as oxidative stress is a common underlying mechanism in many of these conditions. This research was designed to provide scientific evidence by investigating the potential of *Elaeis guineensis* fruit oil to counter the toxic effects of potassium bromate-induced brain injury in male Wistar rats.

Materials and Methods

Chemicals and Reagents

Potassium bromate (Sigma Aldrich, Germany) was supplied by a certified chemical reagent supplier in Ibadan, Oyo state, Nigeria. Crude, unrefined *E. guineensis* fruit oil (red palm oil) was obtained from a production company in Ogbia Local Government, Bayelsa State, Nigeria. All other chemicals and reagents used for the biochemical assays were of high purity and sourced from reputable suppliers.

Experimental Animals

Twenty (20) male Wistar rats, weighing 165 ±15g were used for the experiment. The animals were housed in clean cages under standard laboratory conditions, with a 12-hour light/dark cycle, a temperature of 25 ± 2°C, and free access to standard pellet feed and water. The rats were acclimatized to the laboratory environment for two weeks before the commencement of the experiment. All animal handling procedures were conducted following institutional and international guidelines for the care and use of laboratory animals.

Experimental Design

The rats were randomly divided into four groups (n=5 per group) for the study, which lasted for 21 days.

Group 1 (Control): Received distilled water orally once daily.

Group 2 ($KBrO_3$ only): Animals were orally administered 100 mg/kg of potassium bromate for 14 days.

Group 3 (*E. guineensis* + $KBrO_3$): animals were orally administered 2 ml/kg of *E. guineensis* fruit oil for 7 days before co-administration with $KBrO_3$ for 14 more days.

Group 4 (*E. guineensis* + $KBrO_3$): animals were orally administered 4 ml/kg of *E. guineensis* fruit oil for 7 days before co-administration with $KBrO_3$ for 14 more days.

E. guineensis oil was administered orally via gavage, and potassium bromate was dissolved in water to ensure accurate dosage. The duration of 21 days was chosen based on prior studies investigating chronic exposure to neurotoxic substances in rodents (Zubaidi *et al.,* 2021).

Sample Collection and Preparation

The male Wistar rats were sacrificed via cervical dislocation after mild anaesthesia, 24 hours after the last administration. The brain was excised, rinsed and weighed before homogenizing in phosphate buffer saline solution (0.1M, pH=7.4). The homogenized brain was centrifuged at 16000rpm for 10 minutes to obtain a clear supernatant. Each centrifuged plain tube was collected into another plain tube labelled accordingly. The separated samples were kept frozen

International Journal of Basic Science and Technology
June, Volume 11, Number 2, Pages 187 - 196 http://www.ijbst.fuotuoke.edu.ng
https://10.5281/zenodo.16673246 ISSN 2488-8648

in a refrigerator until needed for various biochemical assays.

Biochemical Assays

The brain supernatant was used to determine the concentration or activity of the following biochemical markers using established standard laboratory protocols:

The cerebral cortex malondialdehyde (MDA) concentrations and index of lipid peroxidation were spectrophotometrically determined according to the method of Draper and Hadley (1990). Catalase (CAT) activity was assayed by the decomposition of hydrogen peroxide according to the method of Claiborne (1984). Superoxide dismutase (SOD) activity was determined by the method of Misra and Fridovich (1972). AChE activity in the cerebral cortex homogenates was measured by the method of Lombardi *et al*. (1999). Nitrite assay was done using Griess reagent with some modifications of the method of Green *et al*. (1982). The total protein levels were measured by an enzymatic colourimetric kit (Wako Chemicals USA, Inc.).

Statistical Analysis

Data obtained from the experiment were analysed using one-way analysis of variance (ANOVA) using the statistical package for social sciences version 23 (SPSS 23) and presented as means ± standard error of mean (SEM). Values at ($p < 0.05$) were regarded as significant in comparison with the appropriate controls.

Results

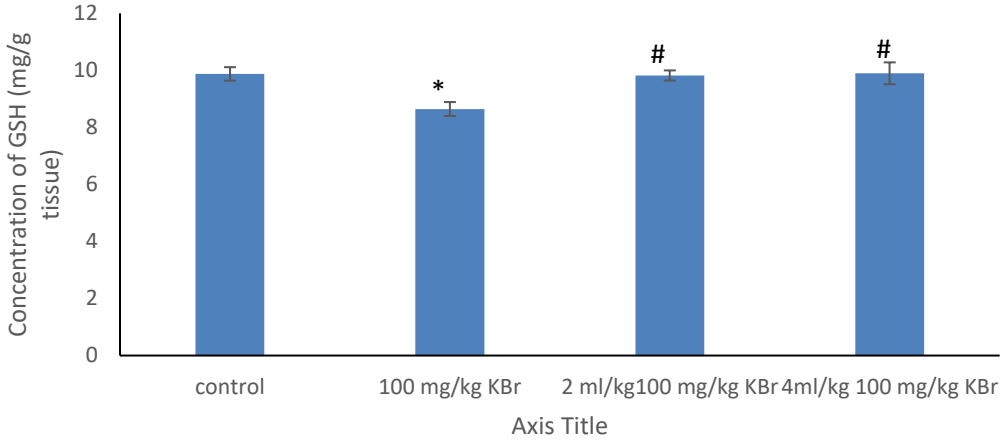

Figure 1: Effect of *E. guineensis* fruit oil on glutathione (GSH) concentration in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment).

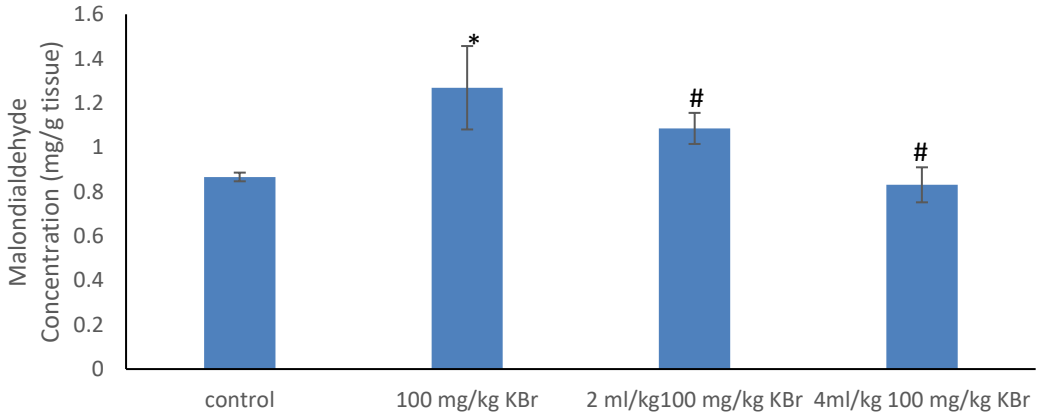

Figure 2: Effect of *E. guineensis* fruit oil on malondialdehyde (MDA) concentration in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment)

International Journal of Basic Science and Technology
June, Volume 11, Number 2, Pages 187 - 196 http://www.ijbst.fuotuoke.edu.ng
https://10.5281/zenodo.16673246 ISSN 2488-8648

The results presented in Figure 1 show a marked and statistically significant ($p<0.05$) decrease in the concentration of glutathione (GSH) in the brain tissue of rats administered only potassium bromate (Group 2) when compared to the normal control group (Group 1). In contrast, the group pre-treated with *E. guineensis* fruit oil before $KBrO_3$ administration (Group 3) exhibited a significant increase in GSH concentration, bringing it to a level comparable to that of the control group. The group that received only *E. guineensis* oil (Group 4) showed GSH levels similar to the control group, indicating that the oil itself does not adversely affect GSH stores.

As depicted in Figure 2, the administration of potassium bromate alone (Group 2) led to a significant ($p<0.05$) elevation in the levels of malondialdehyde (MDA), a marker of lipid peroxidation, in the brain homogenates compared to the control group (Group 1). Conversely, the co-administration of *E. guineensis* fruit oil with $KBrO_3$ (Group 3) resulted in a significant reduction of the elevated MDA levels, restoring them to near-normal values observed in the control group. The MDA concentration in the group treated with only *E. guineensis* oil (Group 4) was not significantly different from the control group.

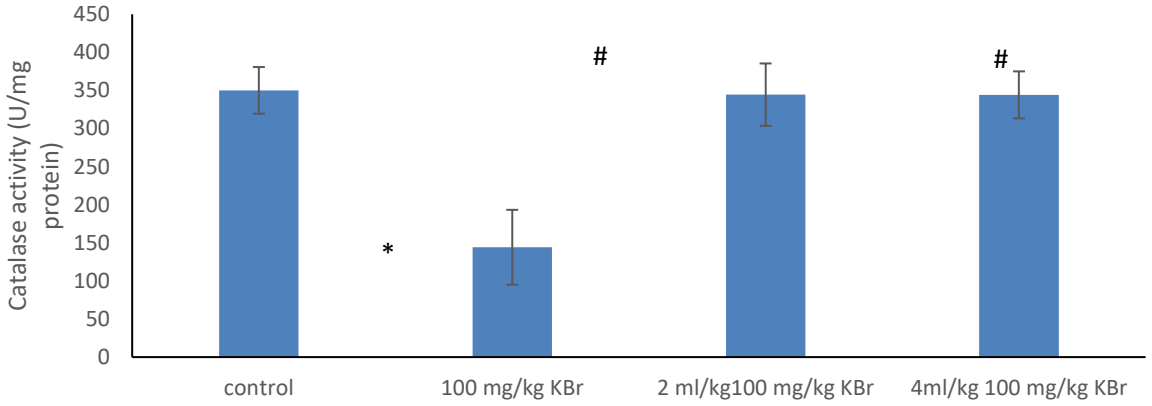

Figure 3: Effect of *E. guineensis* fruit oil on catalase activity in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment).

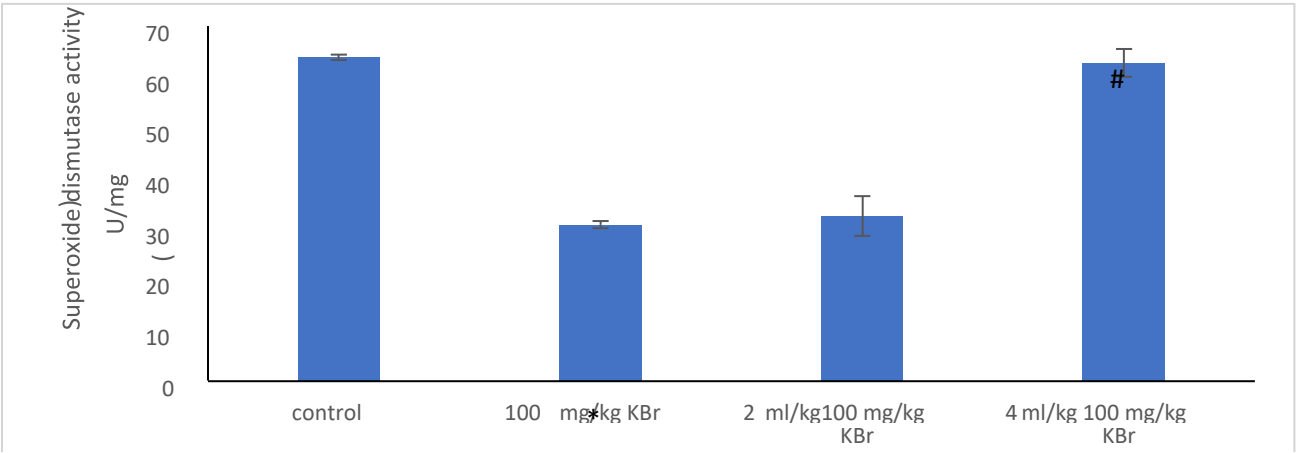

Figure 4: Effect of *E. guineensis* fruit oil on superoxide dismutase activity in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment).

Figure 3 illustrates the activity of the antioxidant enzyme catalase. The $KBrO_3$-only treated group (Group 2) showed a statistically significant ($p<0.05$) inhibition of catalase activity in the brain when

International Journal of Basic Science and Technology
June, Volume 11, Number 2, Pages 187 - 196 http://www.ijbst.fuotuoke.edu.ng
https://10.5281/zenodo.16673246 ISSN 2488-8648

compared to the control group (Group 1). However, in the group that received *E. guineensis* fruit oil before $KBrO_3$ (Group 3), the activity of catalase was significantly preserved and was not statistically different from the control group. The *E. guineensis* oil-only group (Group 4) maintained normal catalase activity.

The data in Figure 4 reveal that the administration of potassium bromate (Group 2) caused a significant ($p<0.05$) decline in the activity of brain superoxide dismutase relative to the control group (Group 1). Pre-treatment with *E. guineensis* fruit oil (Group 3) effectively prevented this decline, maintaining SOD activity at levels comparable to the normal control group. The SOD activity in the group receiving only *E. guineensis* oil (Group 4) remained within the normal range.

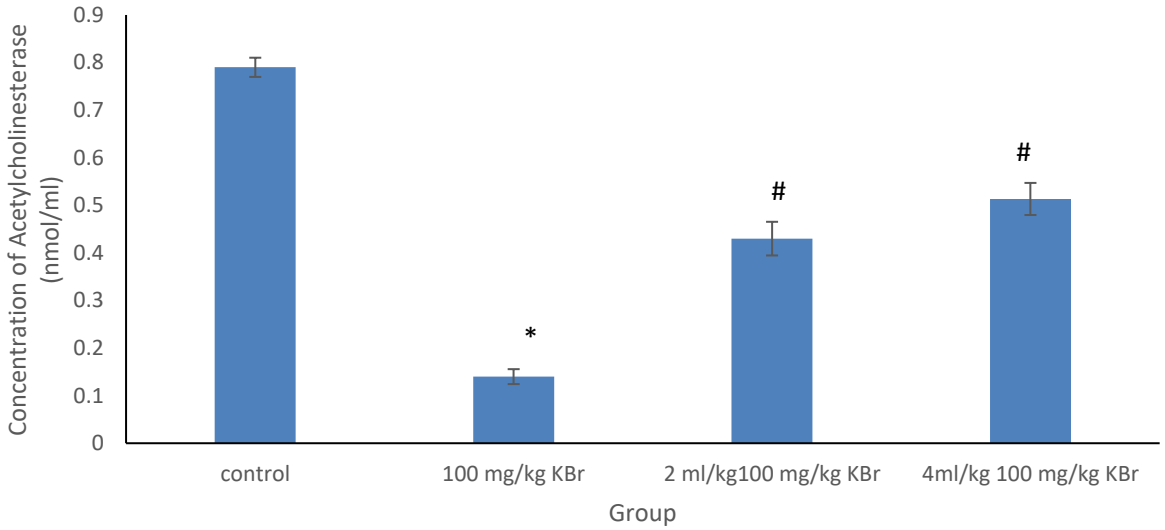

Figure 5: Effect of *E. guineensis* fruit oil on the activity of acetylcholinesterase in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment).

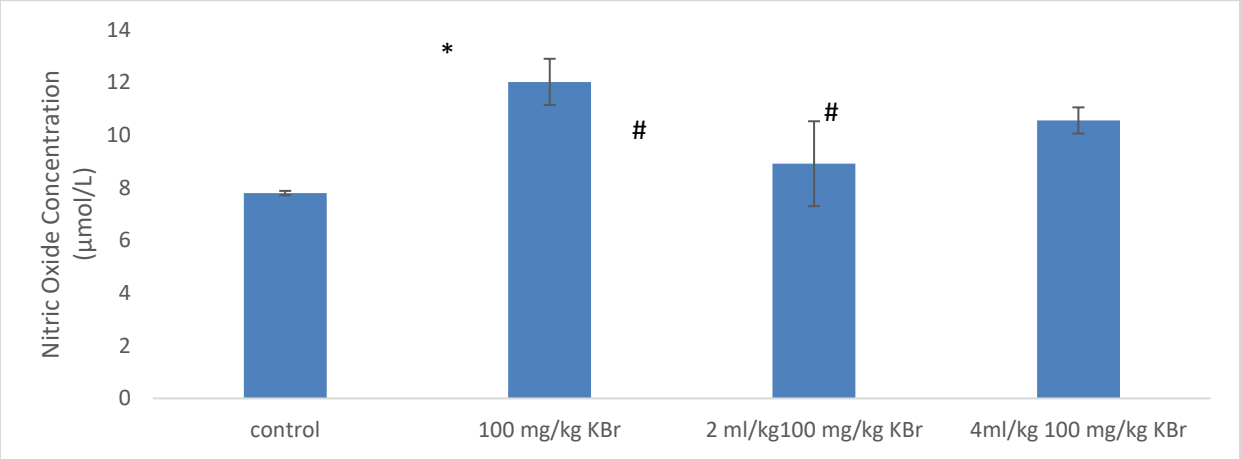

Figure 6: Effect of *E. guineensis* fruit oil on nitric oxide concentration in the brain of male Wistar rats orally administered potassium bromate ($KBrO_3$). Results are expressed as mean±standard deviation for 5 animals. *P<0.05 significantly different (Control vs $KBrO_3$); # P<0.05 significantly different ($KBrO_3$ vs treatment).

As shown in Figure 5, the potassium bromate-treated group (Group 2) displayed a significant ($p<0.05$) reduction in the concentration of acetylcholinesterase (AChE) in the brain compared to the control group (Group 1). The administration of *E. guineensis* fruit oil in conjunction with $KBrO_3$ (Group 3) was able to ameliorate this effect, resulting in AChE levels that were significantly higher than the $KBrO_3$-only group

https://10.5281/zenodo.16673246 ISSN 2488-8648

and close to those of the control group. The group receiving only the oil (Group 4) showed no significant change in AChE concentration.

The results in Figure 6 indicate that oral administration of potassium bromate (Group 2) led to a significant ($p<0.05$) increase in the concentration of nitric oxide (NO) in the brain tissue compared to the control group (Group 1). In the group pre-treated with *E. guineensis* fruit oil (Group 3), this $KBrO_3$-induced increase in NO was significantly attenuated, with levels returning close to the baseline observed in the control group. The NO concentration in the group treated solely with *E. guineensis* oil (Group 4) was similar to that of the control group.

Discussion

The results of this study, which demonstrate the neurotoxic effects of potassium bromate ($KBrO_3$) and the protective role of *E. guineensis* fruit oil, are strongly supported by and consistent with a significant body of existing scientific literature. The findings for each measured parameter align with the established mechanisms of oxidative stress and antioxidant-based neuroprotection (Akkoyun *et al*., 2023; Saad *et al*., 2018). The core finding that $KBrO_3$ induces oxidative stress in the brain is well-documented (Watanabe *et al*., 2004; Al-Mareed *et al*., 2022). Numerous studies have established that $KBrO_3$ administration in animal models leads to a significant imbalance in the redox state of various tissues, including the brain (Watanabe *et al*., 2004; Al-Mareed *et al*., 2022; Aygörmez *et al*., 2025; Akkoyun *et al*., 2023; Al-Mareed *et al*., 2022). The observed decrease in glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD) in the $KBrO_3$ -only group (Figures 1, 3, and 4) is a classic hallmark of severe oxidative stress. These molecules constitute the first line of endogenous antioxidant defence (Ighodaro and Akinloye, 2018. First line defence antioxidants-superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX): Their fundamental role in the entire antioxidant defence grid. $KBrO_3$ generates excessive reactive oxygen species (ROS), which consume GSH and inactivate crucial enzymes like CAT and SOD (Watanabe *et al*., 2004; Al-Mareed *et al*., 2022). This finding is in direct agreement with research by Akkoyun *et al*. (2023), who reported significantly lower SOD and CAT activity in the brains of rats exposed to $KBrO_3$. Similarly, Agu *et al*. (2023) found that $KBrO_3$ suppressed and diminished antioxidant enzyme activities in the brains of Wistar rats. The significant elevation of malondialdehyde (MDA) in the $KBrO_3$ group (Figure 2) indicates widespread damage to the lipid-rich membranes of brain cells. This is a direct consequence of ROS attacking polyunsaturated fatty acids (Giro *et al*., 2023). This result is highly consistent with multiple studies. For instance, the aforementioned study by Akkoyun *et al*. (2023) also found that MDA levels in the $KBrO_3$-exposed group were significantly higher than in the control group. This finding is a cornerstone of $KBrO_3$ toxicology and has been replicated in numerous other studies investigating its effects on various organs (Watanabe *et al*., 2004; Al-Mareed *et al*., 2022;Aygörmez *et al*., 2025; Akkoyun *et al*., 2023). The reduction in acetylcholinesterase (AChE) activity (Figure 5) aligns with findings from other neurotoxicity studies. Agu *et al*. (2023) also reported diminished AChE activity following $KBrO_3$ administration.[8] Oxidative damage to the enzyme's structure or altered gene expression due to cellular stress can lead to this reduction, impairing cholinergic neurotransmission, which is vital for cognitive functions. The increase in nitric oxide (NO) concentration (Figure 6) is also a consistent finding. While NO is a critical signalling molecule at physiological levels, its overproduction during oxidative stress leads to the formation of highly damaging peroxynitrite radicals when it reacts with superoxide Pacher *et al*., 2027. Agu *et al*. (2023) also documented a significant increase in NO as an oxidative stress biomarker in $KBrO_3$-treated rats. This elevation contributes to both further oxidative damage and neuroinflammation.

The ameliorative effects of *E. guineensis* fruit oil observed in this study (Group 3) are scientifically plausible and supported by research on its key antioxidant components. Red palm oil is the richest natural source of tocotrienols and is also high in carotenoids. The ability of the oil to restore GSH, CAT, and SOD levels while reducing MDA (Figures 1-4) is directly attributable to the potent antioxidant properties of its constituents, particularly tocotrienols and carotenoids. These compounds can directly scavenge free radicals, thereby sparing the endogenous antioxidant defences and preventing lipid peroxidation. A study published in *Sains Malaysian* (2010) highlighted that tocotrienols, in particular, are effective in protecting neurons from oxidative injuries. Research published in *PMC* on the neuroprotective effects of tocotrienol-rich fraction (TRF) from palm oil explicitly states that TRF possesses potent antioxidant and neuroprotective activities, diminishing cytotoxicity and recovering mitochondrial injury due to elevated oxidative stress. The oil's ability to normalize AChE and NO levels (Figures 5 and 6) demonstrates a broader protective effect beyond simple antioxidant action. By mitigating the initial oxidative insult from $KBrO_3$, the oil prevents the downstream pathological consequences, such as enzymatic dysfunction and neuroinflammatory responses. Leow *et al*., in their study on oil palm phenolics, found that these compounds not only have

https://10.5281/zenodo.16673246 ISSN 2488-8648

neuroprotective effects but also downregulate genes involved in inflammation, which is consistent with the observed reduction in NO (Ibrahim *et al*., 2020).

The results presented in the figures are in strong concordance with the established scientific consensus. The pattern of toxicity induced by potassium bromates characterized by depleted antioxidant defences, increased lipid peroxidation, and disruption of neurochemical markers, is a recurring theme in toxicological literature. Similarly, the reversal of these toxic effects by an antioxidant-rich substance like *E. guineensis* fruit oil follows a well-established principle of neuroprotection. Therefore, the findings of this study serve to reinforce the existing knowledge regarding the mechanisms of $KBrO_3$ neurotoxicity and highlight the therapeutic potential of palm oil in mitigating such oxidative damage.

Further research can be conducted to investigate the long-term effects of E. guineensis fruit oil and its potential interactions with other neurotoxicants. Also, a detailed investigation into the specific molecular mechanisms by which the active compounds in E. guineensis fruit oil exert neuroprotective effects may help clarify their role in mitigating oxidative damage.

Funding: The research was self-funded

Institutional Review Board: To ensure animal welfare, the study adhered to the guidelines outlined in the Helsinki Declaration of 1975. All animals used were healthy. The experimental design received approval (code ART2023008) from the Federal University Otuoke's ethical committee on animal research and treatment (ART). The experiments were conducted in the Department of Biochemistry's animal house between May and July 2022.

Conflicts of Interest: None.

Data Availability: It will be made available on request

Acknowledgements: The authors wish to appreciate all the students who assisted in the execution of the project

References

Adewunmi, R., Ilesanmi, O. B., Crown, O. O., Komolafe, K. C., Akinmoladun, A. C., Olaleye, T. M., and Akindahunsi, A. A. (2018). Attenuation of KCN-induced neurotoxicity by solvent fractions of Antiaris africana leaf. *Europe J Med Plant*, *23*(2), 1-11.

Ajarem, J. S., Altoom, N. G., Allam, A. A., Maodaa, S. N., and Mahmoud, A. M. (2016). Oral administration of potassium bromate induces neurobehavioral changes, alters cerebral neurotransmitters level and impairs brain tissue of Swiss mice. *Behavioral and Brain Functions*, *12*(1), 14.

Akkoyun, H. T., Uyar, A., Bayramoglu Akkoyun, M., Bengü, A. Ş., Melek, Ş., Karagözoğlu, F., and Erdem, S. A. (2023). The protective effect of arbutin against potassium bromate-induced oxidative damage in the rat brain. *Journal of Biochemical and Molecular Toxicology*, *37*(2), e23248.

Al-Mareed, A. A., Farah, M. A., Al-Anazi, K. M., Hailan, W. A., and Ali, M. A. (2022). Potassium bromate-induced oxidative stress, genotoxicity and cytotoxicity in the blood and liver cells of mice. *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, *878*, 503481.

Aygörmez, S., Küçükler, S., Akaras, N., Maraşli, Ş., and Kandemir, F. M. (2025). Investigation of the effects of morin on potassium bromate-induced brain damage in rats via different pathways with biochemical and histopathological methods. *Food and Chemical Toxicology*, 115466.

Chauhan, D., and Jain, P. (2016). A scientific study of genotoxic-carcinogenic impacts of Potassium Bromate as food additive on human health. *International Research Journal of Engineering and Technology*, *3*(6), 1136-1139.

Claiborne, A. (1985). Catalase activity. In R. A. Greenwald (Ed.), *CRC handbook of methods in oxygen radical research* (pp. 283–284). CRC Press.

Draper, H. H., and Hadley, H. (1990). Malondialdehyde determination as index of lipid peroxidation. *Methods in Enzymology*, *86*, 421–431. https://doi.org/10.1016/0076-6879(90)86135-i

Green, L. C., Wagner, D. A., Glogowski, J., Skipper, P. L., Wishnok, J. S., and Tannenbaum, S. R. (1982). Analysis of nitrate, nitrite, and [15N] nitrate in biological fluids. *Analytical Biochemistry*, *126*(1), 131–138. https://doi.org/10.1016/0003-2697(82)90118-x

Haro Girón, S., Monserrat Sanz, J., Ortega, M. A., Garcia-Montero, C., Fraile-Martínez, O., Gómez-Lahoz, A. M., Boaru, D. L., de Leon-Oliva, D., Guijarro, L. G., Atienza-Perez, M., Diaz, D., Lopez-Dolado, E., and Álvarez-Mon, M. (2023). Prognostic Value of Malondialdehyde (MDA) in the Temporal Progression of Chronic Spinal Cord Injury. *Journal of Personalized Medicine*, *13*(4), 626. https://doi.org/10.3390/jpm13040626

Ibrahim, N., Muhammad Ismail Tadj, N. B., Rahman Sarker, M. M., and Naina Mohamed, I. (2020). The potential mechanisms of the neuroprotective actions of oil palm phenolics: Implications for neurodegenerative diseases. *Molecules*, *25*(21), 5159. https://doi.org/10.3390/molecules25215159

https://10.5281/zenodo.16673246 ISSN 2488-8648

Ilesanmi, O. B., Akinmoladun, A. C., Elusiyan, C. A., Ogungbe, I. V., Olugbade, T. A., and Olaleye, M. T. (2022). Neuroprotective flavonoids of the leaf of Antiaris africana Englea against cyanide toxicity. *Journal of ethnopharmacology*, *282*, 114592.

Ischiropoulos, H., and Beckman, J. S. (2003). Oxidative stress and nitration in neurodegeneration: Cause, effect or association? *Journal of Clinical Investigation*, *111*(2), 163–169.

Ji, T., Zheng, L., Wu, J., Duan, M., Liu, Q., Liu, P., Shen, C., Liu, J., Ye, Q., Wen, J., *et al*. (2023). The thioesterase APT1 is a bidirectional adjustment redox sensor. *Nature Communications*, *14*(1), 2807.

Loganathan, R., Subramaniam, K. M., Radhakrishnan, A. K., Choo, Y.-M., and Teng, K.-T. (2017). Health-promoting effects of red palm oil: Evidence from animal and human studies. *Nutrition Reviews*, *75*(2), 98–113.

Lombardi, D., Gordon, K. L., Polinsky, P., Suga, S., Schwartz, S. M., and Johnson, R. J. (1999). Salt-sensitive hypertension develops after short-term exposure to angiotensin II. *Hypertension*, *33*(4), 1013–1019. https://doi.org/10.1161/01.hyp.33.4.1013

Magomya, A. M., Yebpella, G. G., Okpaegbe, U. C., and Nwunuji, P. C. (2020). Analysis of potassium bromate in bread and flour samples sold in Jalingo Metropolis, Northern Nigeria. *IOSR Journal of Environmental Science, Toxicology and Food Technology*, *14*(2), 1-5.

Misra, H. P., and Fridovich, I. (1972). The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. *Journal of Biological Chemistry*, *247*(10), 3170–3175.

Moorthy, M., and Radhakrishnan, A. K. (2024). Health benefits of palm tocotrienol-rich fraction: A systematic review of randomized controlled trials. *Nutrition Reviews*, *83*(2), 307–328.

Musa, I., Khaza'ai, H., Abdul Mutalib, M. S., Yusuf, F., Sanusi, J., and Chang, S. K. (2017). Effects of oil palm tocotrienol rich fraction on the viability and morphology of astrocytes injured with glutamate. *Food Bioscience*, *20*, 168–177.

Obahiagbon, F. I. (2012). A review: Aspects of the African oil Palm (*Elaeis guineensis* Jacq.) and the implications of its bioactives in human health. *American Journal of Biochemistry and Molecular Biology*, *2*(2), 106–119.

Olusola, J. A., Aturamu, A. O., Akeju, K. F., Ogunsakin, R. E., and Awotoye, O. O. (2024). Assessment of health risks from potassium bromate and trace metals in bread consumption in Ado Ekiti, Southwest, Nigeria. *Journal of Trace Elements and Minerals*, *8*, 100158.

Pacher, P., Beckman, J. S., and Liaudet, L. (2007). Nitric oxide and peroxynitrite in health and disease. *Physiological Reviews*, *87*(1), 315–424.

Saad, H. B., Driss, D., Jaballi, I., Ghozzi, H., Boudawara, O., Droguet, M., ... and Amara, I. B. (2018). Potassium bromate-induced changes in the adult mouse cerebellum are ameliorated by vanillin. *Biomedical and Environmental Sciences*, *31*(2), 115–125.

Shanmugavel, G., Muthu, M., and Valli, R. (2020). Potassium bromate: Effects on bread components, health, environment and method of analysis: A review. *Food Chemistry*, *311*, 125920.

Sharif, R., Wai, M. K., Choon, O. T., Hafid, S. R. A., and Lee, T. Y. (2025). Tocotrienol-enriched beverage enhances psychological well-being, antioxidant defense, and genomic stability in older adults: A randomized controlled trial. *Nutrients*, *17*(13), 2179.

Wang, Y., Wu, J., Wang, J., He, L., Lai, H., Zhang, T., Wang, X., and Li, W. (2023). Mitochondrial oxidative stress in brain microvascular endothelial cells: Triggering blood-brain barrier disruption. *Mitochondrion*, *69*, 71–82.

Watanabe, S., Tajima, Y., Yamaguchi, T., and Fukui, T. (2004). Potassium bromate-induced hyperuricemia stimulates acute kidney damage and oxidative stress. *Journal of Health Science*, *50*(6), 647–653.

Zubaidi, S. N. C. M., Halim, S., Kasini, Z. M., Fazleen, N. E., Thiruselvam, V., and Rajagopal, V. Red Palm Oil Prevents Congestive Impairment In A Rat Model Of D-Galactose-Induced Aging. *Journal of Cellular & Molecular Anesthesia*, *6*(4), 294-305.