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THE IMPACT OF PLANTAIN STEM JUICE ON THE LEVELS OF ALBUMIN, PROTEIN, AND LIPID PROFILE IN RATS WITH INDUCED HEPATIC TOXICITY AND OXIDATIVE STRESS BY ALUMINUM CHLORIDE

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ABSTRACT
The study's goal was to investigate the effects of plantain stem juice on albumin protein and lipid profiles in rats with liver damage and oxidative stress caused by aluminum chloride. The study used fifteen female Wistar rats, which were separated into three groups: a control group that did not get any therapy, a group that had a dosage of 100 mg/kg/body weight of aluminum chloride (AlCl3) treatment, and another group that received both aluminum chloride and plantain stem juice at 2 mL/kg. Furthermore, every group was granted free access to food and hydration for a period of fourteen days. At the end of the experimentation period, we euthanized the experimental rats to collect liver and venous blood samples. The results showed significant changes in different parameters among the groups. It was found that in the $A1C1_3$ treated groups, triglyceride levels dropped from 77.78±1.49 mg/dl to 49.68±1.07 mg/dl, total cholesterol levels dropped from 67.83±1.39 mg/dl to 43.73±0.99 mg/dl, total protein levels dropped from 92.81±1.36 mg/dl to 51.02±0.95 mg/dl, albumin levels dropped from 8.25±1.05 mg/dl to 4.91±0.08 mg/dl, and SOD levels dropped from 5.75±1.32 Units/mg protein to 3.66±10.88 Units/mg protein. Conversely, there was a significant increase ($P<0.05$) in MDA levels, increasing from 401.08±0.99 to 1207.98±1.39 (nmol/mg protein). On the other hand, the experimental group that received M. *paradisaca* showed a significant increase in SOD, triglyceride, total protein, albumin, and total cholesterol concentrations. The use of M. *paradisaca* resulted in the restoration of measured parameters to their normal range, suggesting that this plant has potential as a therapy for chemical toxicity.

KEYWORDS: Oxidative stress *M.paradisiaca* hepatoprotection chemical toxicity.

The western and central parts of Africa, the Caribbean Islands, the region of Central America, and the northernmost coastline parts of South America heavily rely on plantains (M. *paradisaca*) as their primary source of sustenance. People commonly refer to them as green bananas or frying bananas. They serve as a staple food, possessing a taste that balances sweetness and sourness, available all year round, and suitable for consumption at any stage of maturity. While many areas make a distinction between plantains and bananas, others do not, referring to any cooking banana as "plantain".[30] Other names for plantains include "green bananas" and "frying bananas". Plantains, commonly referred to as green bananas or frying bananas, are specific varieties of bananas that belong to the Musa genus. Culinary applications commonly utilize these starchy fruits, which have a predominantly neutral taste. You can eat plantains either ripe or unripe. As they ripen, the starches in plantains convert into sugars, which makes them acceptable for eating fresh. Several geographical areas frequently employ the terminology interchangeably, making the differentiation between plantains and bananas ambiguous. Official terminology distinguishes classified plantains as "real" plantains, while categorizing other carbohydrate types used for cooking as "cooked bananas." The bananas in concern are specific varieties that are classified under the genus Musa. Culinary applications commonly utilize these

particular bananas.[30] Once cooked, they have a tender consistency and are classified as a starchy fruit with a predominantly neutral taste. Similar to other varieties of bananas, one of the attractive qualities of plantains as a food source is their ability to produce fruit consistently throughout the year, making them a reliable mainstay in one's diet.[30]

While certain countries may distinguish between plantains and bananas, other nations do not make this distinction and instead consume a wider range of banana cultivars. Formal contexts only refer to "true" plantains, meaning those that meet specific criteria. We refer to other starchy cultivars suitable for cooking as "cooking bananas."[8] Liver problems are a worldwide health issue, leading to investigations into the hepatoprotective properties of medicinal plants such as *Musa paradisiaca*. Plantains contain phytochemicals that possess diverse pharmacological characteristics, rendering them highly beneficial in the field of traditional medicine. The references cited are from the following papers:[10];[4]; and.[33] Liver illnesses provide substantial worldwide health obstacles, underscoring the necessity for innovative pharmaceutical therapies. Medicinal plants, which contain high levels of antioxidants, exhibit hepatoprotective properties. Phytoextracts with antioxidant properties have a vital role in preventing liver damage and oxidative stress-related disorders. Plant components contain flavonoids that possess antioxidant properties and contribute to many health benefits. *M.paradisiaca*, a commonly utilized botanical species, possesses a wide range of pharmacological characteristics, rendering it a highly important asset in traditional medicine. The references cited are[10];and [4];. Liver diseases are a major contributor to illness and death on a global scale, highlighting the need for the creation of new and innovative pharmaceutical treatments. Laboratory experiments on animals can assess the hepatoprotective properties of medicinal plants, thereby guaranteeing the safety and effectiveness of treatments for hepatotoxicity. Evidence has demonstrated the efficacy of traditional remedies derived from diverse cultures in the treatment of liver ailments.

These disorders are frequently associated with the existence of phytoextracts or phytocompounds that contain a high concentration of anti-oxidants.[25] Therefore, numerous biologically active compounds and botanical extracts have been subjected to testing in order to assess their effectiveness in reducing liver damage induced by hepatotoxins and their antioxidant characteristics.[11]

Plantains, a widely used medicinal herb from the Musaceae family, This plant, sometimes referred to as plantain, has been utilized in traditional Ayurvedic medicine to treat a variety of ailments. They possess a diverse array of phytochemical constituents, including as flavonoids, renowned for their antioxidative characteristics.[20]

[21] Identified several pharmacological properties of the plant, including analgesic effects.

Aluminum is the predominant metal found in the surrounding, constituting around 8.13% of the planet's core. The oral sources of aluminum consumption encompass food (via cooking utensils and food preservatives), water, makeup, and pharmaceuticals. The skin, digestive system, lungs, and nasal mucosa all absorb aluminum. Aluminum (Al) toxicity is a concern due to its prevalence in the environment. Oral intake sources include food, water, cosmetics, and medications. Understanding the impact of aluminum on health is crucial, given its widespread presence and potential health implications.[33] In conclusion, plantains (M.paradisiaca) are a widely consumed starchy fruit with significant nutritional and medicinal properties. They have been used in traditional medicine and are rich in phytochemical components, particularly flavonoids, which exhibit antioxidant properties.

Exposure to aluminum (Al) has been associated with several harmful effects, such as kidney damage (nephrotoxicity), liver damage (hepatotoxicity), blood-related issues (hematotoxicity), and nerve damage (neurotoxicity).[4] Aluminum compounds possess the capacity to hinder the activity of enzymes including acid and alkaline phosphatases, hexokinases, phosphodiesterases, and phospholipases by virtue of their capability to attach themselves to DNA and RNA[4] A study by[33] shows that aluminum can change biochemical variables, the peroxidation of lipids, and the function of enzymes that make antioxidants in the blood plasma and other parts of male rats and rabbits.

Research has indicated that AlCl3 can induce hepatic dysfunction and disrupt the metabolism of mitochondrial energy in rats.[10] Exposure to AlCl3 in rats disrupts mitochondrial energy metabolism, leading to liver dysfunction. It also promotes mitochondrial oxidative stress, causing increased ROS accumulation, decreased superoxide dismutase activity, and increased 8-hydroxydeoxyguanosine levels in mitochondrial DNA.[10] Musa paradisiaca, commonly known as plantain, has been shown to have protective effects against AlCl3 toxicity. A study conducted by Ubogu et al. in 2018 examined the potential protective impact of M. paradisiaca stem pulp against AlCl3 toxicity. The study used AST, ALT, SOD, and MDA as biomarkers to assess liver damage in rats. The findings indicated that M. *paradisiaca* stem pulp was able to ameliorate the liver damage generated by AlCl3 in rats.

M. *paradisiaca* stem pulp and bromelain, found in pineapple stems, have potential as protective agents against aluminum toxicity due to their antioxidant properties. Aluminum exposure can lead to liver dysfunction and oxidative stress, and more studies is needed to understand the mechanisms and mitigating effects of these natural compounds.[10],[29] In order to

determine if *M.paradisiaca* stem pulp protects against AlCl3 toxicity, the current study used serum Albumin, Proteins, lipid profile SOD, and MDA as indicies to assess the level of protection

MATERIALS AND METHODS

Chemicals and Reagents

All chemicals used were of analytical grade and were manufactured by Randox Laboratories.

Experimental Animals

The study utilized an array of fifteen healthy adult female Wistar albino rats, each weighing between 150 and 250 grams on average. The research animals were acquired from the animal facility of the Department of Biochemistry at the University of Port-Harcourt, Nigeria. They were kept in accordance with regular living conditions, with a 12-hour natural light and 12-hour dark photoperiod. The animals underwent a three-week acclimation period, during which they were given pelletized breeders' feed and had continuous access to hygienic drinking water during the whole length of the study.

Feeds

Rats were fed with pelletized breeders' feed throughout the experiment (14 days).

Source of M. *paradisiaca*

The plantain stem, *M.paradisiaca*, was obtained from a farm in Amassoma, Wilberforce Island and verified in the Herbarium of Niger Delta University.

Preparation of Extracts

The recently harvested stem of M. *paradisiaca* was purified using filtered water. The outer layer of the stem was removed. A total of 100 grams of minute fragments were obtained by measuring the white pith from the stem of M. *paradisiaca* (plantain). The stem was pulverized using a homogenizer (Saisho, Model S-742) after mixing 100 ml of distilled water with 100g of the stem. The resultant mixture underwent filtration using a clean muslin cloth, and the obtained juice was subsequently stored in a hermetically sealed container at a temperature of 4 °C within a refrigerator.

Experimental Design

The study was conducted using the randomized block design. Fifteen healthy albino rats (Wistar strain) comprising males and females were used in this study. They were given a period of three weeks to adapt to the new environment before the experiment began. The rats were put into three experimental groups using random assignment.

Group 1 (control): Throughout the 14-day trial, the experimental animals were provided with pelleted growers' feed and purified water in a continual manner.

Group 2: 100mg/kg per body wt of Al Cl3 and purified water were administered to the experimental animals throughout the duration of the study (14 days).

Group 3: 100mg/kg per body wt of Al Cl3 and plantain stem M.paradisiaca juice at 2 mL/kg and was also allowed free access to feed and water ad libitum for 14 days.

SAMPLE COLLECTION

Blood Collection

At the end of the experimental period, experimental rats were sacrificed for venous blood collection. The blood samples collected were immediately subjected to centrifugation at 3000rpm for 5 minutes in order to obtain the serum. Analysis was carried out immediately after centrifugation.

Liver

The juice of *Musa paradisiaca* was orally fed using gavage for a duration of 14 days. Afterwards, the animals were euthanized using chloroform anesthesia. The entire body was dissected, and the liver tissue was extracted and washed with a standard saline solution. A fraction of the liver tissue was placed in a sample container immersed in a solution of formaldehyde with a concentration of 10% in order to perform histological analysis.

An additional 5 grams of the liver sample was measured, crushed using a 0.1M Phosphate buffer solution at pH 7.4, and then spun in a centrifuge at 3000 revolutions per minute for 10 minutes. This underwent biochemical analysis. The blood was subjected to centrifugation at a speed of 3000 revolutions per minute for a duration of 10 minutes, resulting in the collection of the serum.

Analysis of Biochemical indicies

Superoxide Dismutase (SOD) EC 1.15.1.1

The level of SOD activity was determined by the method of.[23]

Malondialdehyde (Lipid Peroxidation Assessment

The level of lipid peroxidation was determined by the assay method developed by Hunter et al.[16] and modified by Gutteridge and Wilkins[15] was used to measure the concentration of Malondialdehyde (MDA). Lipid peroxidation in units/mg or gram tissue was computed as the concentration of MDA (nmol/mg protein).

RESULT

The results for the effect of *Musa paradisiacal* on liver of female wistar albino rats in presented in Tables 3.1, 3.2 and 3.3.

Table 3.1: Mean values of total proteins and albumin.

Treatment	Total protein mg/dl	Albumin mg/dl
Control	92.81 ± 1.36^{a}	8.25 ± 1.05^{a}
$Alcl_3$	51.02 ± 0.95^{b}	4.91 ± 0.08^{b}
***M.paradisiaca*+$Alcl_3$**	95.63 ± 1.37^{a}	7.58 ± 1.55^{a}

The data is presented as the mean value plus or minus the standard deviation, with a sample size of 5. Values in the same column with different superscript letters are statistically significant at a significance level of P<0.05.
The findings indicated a significant decrease (P<0.05) in the overall protein levels of wistar albino rats after being given AlCl3, in comparison to the control group. Nevertheless, the use of AlCl3 and *M.paradisiaca* therapy led to a significant augmentation in overall protein concentrations.

Table 3.2: Mean values of the lipid profile.

Treatment	Total Cholesterol (mg/dl)	Triglyceride (mg/dl)
Control	67.83±1.39[a]	77.78±1.49[a]
Alcl3	43.73.98±0.99[b]	49.68±1.07[b]
M.paradisiaca+Alcl$_3$	66.54±0.866[a]	76.58±0.65[a]

The data is presented as the mean value plus or minus the standard deviation, with a sample size of 5. Values in the same column with different superscript letters are statistically significant at a significance level of P<0.05.

The findings indicated a significant decrease (P<0.05) in the lipid profile of the liver in male albino rats following the treatment of AlCl3, as compared to the control group. Nevertheless, the administration of the extract was found to improve the effects of AlCl3-induced damage, resulting in a significant rise (P<0.05) in the amounts of Total Cholesterol and Triglycerides.

Table 3.3: Mean values of SOD antioxidant and lipid peroxidation.

Treatment	MDA (nmol/mg protein).	SOD U/mg protein
Control	401.08±0.99[a]	6.04±0.49[a]
Alcl3	1207.98±1.39[b]	2.56±0.86
M.paradisiaca+Alcl$_3$	388.83±31.21[a]	6.00±1.54[a]

The data is presented as the mean value plus or minus the standard deviation, with a sample size of 5. Values in the same column with different superscript letters are statistically significant at a significance level of P<0.05.

The findings indicated a significant increase (P<0.05) in the MDA levels and a decline in SOD activity in wistar albino rats after being given AlCl3. Nevertheless, the administration of AlCl3 and *M.paradisiaca* to rats resulted in a significant decrease (P<0.05) in MDA levels and an increase in SOD activity.

DISCUSSION

Herbal medicine, which involves the use of herbs for their curative or medicinal value, has been a valuable and safe source of remedies for various ailments since time immemorial[1] Approximately 75% of the global population, particularly in developing nations, depend on herbal medicine to fulfill their healthcare requirements.[32]

Currently, numerous categories of drugs have a medicinal plant model, and around twenty-five percent of all existing pharmaceuticals are derived from plants either directly or indirectly.[14],[7]

In this study, the researchers investigated the effects of M.paradisiaca extract on hormones, enzymes, and liver proteins in female Wistar rats. M.paradisiaca, also referred to as plantain, is extensively employed in Nigeria and other regions of Africa due to its therapeutic attributes.

The observed effects of M.paradisiaca on liver protein, enzymes, and hormones have been attributed to phenolic compounds, flavonoid glycosides, and alkaloids present in the plant.[1] The management of blood and liver-related illnesses is of utmost importance, and herbal medicines are widely favored by a significant number of individuals.

It is important to acknowledge that the harmfulness of natural medicines has been documented and scientifically validated, including in plants that are known to have protective properties for blood health,[3]

Therefore, further research is necessary to ensure the safety and efficacy of herbal remedies. Superoxide dismutase (SOD) is a crucial antioxidant defense that protects the cellular system against the toxic effects of lipid peroxidation.[24] In this study, treatment with aluminum chloride (AlCl3) decreased the level of SOD significantly (P<0.05) from 6.04±0.49 to 2.56±0.86 (U/mg protein). However, administration of M.paradisiaca increased the level of SOD from 2.56±0.86 to 6.00±1.54 (U/mg protein) (Table 3.3). These results are higher than earlier reported results of[12], who studied the ability of M.paradisiaca to ameliorate the hepatotoxic effects of CCl4.It is important to note that the results of this study disagree with the study of[13], who reported that aluminum treatment in rats has no significant effect on superoxide dismutase activity in rat liver. As almost all live cells exposed to oxygen do, SOD is a crucial antioxidant defense because it catalyzes the dismutation (or partitioning) of the superoxide (O2-) radical into either regular molecular oxygen (O2) or hydrogen peroxide (H2O2). Research using animal models and cell cultures has shown that aluminum chloride can impact the expression of SOD and glutathione peroxidase, potentially causing membrane fragility.[19][27][5] There is evidence that oxidative stress plays a role in the cellular dysfunction caused by aluminium chloride. If this is indeed the case, M.paradisiaca contains antioxidants that may provide protection.

The study investigated the impact of AlCl3 treatment and subsequent administration of M. paradisiaca on liver

markers in rats. The level of malondialdehyde (MDA) significantly increased from 401.08±0.99 to 1207.98±1.39 nmol/g post-AlCl3 treatment, but decreased to 388.83±31.12 nmol/g after M.paradisiaca administration (Table 3.3). This aligns with Gagan and Luna (2016) and Imene et al. (2017) who observed elevated MDA levels with AlCl3 exposure. Increased lipid peroxidation can impair membrane functions, as seen in the study by[28] on mercuric chloride. The study demonstrated a significant increase in MDA levels and a decrease in SOD activity in Wistar albino rats following AlCl3 administration, indicating oxidative stress and compromised antioxidant defense mechanisms. These results are in line with previous research that reported increased MDA levels and decreased SOD activity in response to liver damage.[22] Remarkably, treatment with AlCl3 and M.paradisiaca resulted in a significant decrease in MDA levels and an increase in SOD activity, suggesting that M.paradisiaca may possess antioxidant properties that counteract the oxidative damage induced by AlCl3.

In the present study, the level of total protein, when compared to the control, decreased significantly ($P<0.05$) from 92.81±1.36 (U/L) to 51.02±0.95 (U/L) for total protein and 51.02±0.95 to 95.63±1.37 (U/L) after treatments with AlCl3. Table 4.1 reveals statistically significant changes in the albumin of the experimental rates, The albumin value for the control group was 8.25±1.05, the Alcl3 group was 4.91± 0.08, while the third group administered with *M.paradisiaca*+Alcl3 had 7.58±1.55.

Analysis of serum protein levels showed a significant decrease in total protein post-AlCl3 treatment, with subsequent restoration after M. paradisiaca administration. This finding aligns with previous studies that reported decreased protein levels in response to liver injury (Dikshit et al., 2011). Interestingly, treatment with AlCl3 and Musa paradisiaca led to a significant increase in total protein levels, suggesting a potential compensatory mechanism or a response to the stress induced by AlCl3.

These findings are consistent with[2] and studies on Vernonia amygdalina.[17] The ameliorative effects of M. paradisiaca are attributed to its secondary metabolites, including antioxidants.[6]

AlCl3 exposure caused a significant decrease in the lipid profile, indicating potential lipid metabolism disruption. However, treatment with Musa paradisiaca extract ameliorated the AlCl3-induced damage, leading to a significant increase in Total Cholesterol and Triglyceride concentrations. This finding is consistent with earlier studies that demonstrated the ability of Musa paradisiaca to modulate lipid metabolism.[18]

CONCLUSION

From the results of this study, *M. paradisiaca* had a significant effect on the liver proteins, enzymes, and antioxidant enzymes and increased the level of enzymes in the liver. The evidence supports the conclusion that the extract has the potential to enhance liver function and may be suggested as a treatment for liver injury.

This study demonstrates that Musa paradisiaca has the capacity to protect the liver from damage caused by AlCl3. Further research is essential to elucidate the specific molecular pathways involved in these effects and to determine the long-term implications of Musa paradisiaca supplementation on liver health.

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Disclosure of conflict of interest

The authors declare that there is no conflict of interest.

Statement of ethical approval

The study protocol was approved by the Ethical and Research Committee of Niger Delta University, Bayelsa State, Nigeria. The ethical principles for medical research involving animal subjects as outlined in the Helsinki declaration in 1975 and subsequent revisions were strictly followed in the course of this study.

REFERENCES

1. Acharya, S., et al. Pharmacological and therapeutic potential of flavonoids: an overview. Current Medicinal Chemistry, 2008; 15(18): 1897-1912.
2. Adeyemi, A. O., Adeyemi, O. O., & Oyelade, O. J. Antioxidant properties of aqueous extracts of unripe Musa paradisiaca (plantain) fruit and their protective potentials against oxidative damage in rat liver mitochondria. Journal of Applied Pharmaceutical Science, 2013; 3(2): 102-109.
3. Agati, G., et al. Toxicity of natural remedies: a review. Toxicology in Vitro, 2012; 26(7): 1092-1101.
4. Arhoghro E. M; and Kpomah E.D. Alanine Aminotransferase and Aspertate Aminotransaminase Activities in Wistar Rats Fed with Musa paradisiaca (Plantain) Stem Pulp in Aluminium Chloride Induced Hepatic Oxidative Stress J. Appl. Sci. Environ. Manage, 2022; 26(6): 1057-1062.
5. Campbell, J. M., et al. Aluminum induces oxidative stress in rat brain and testes. Toxicology and Applied Pharmacology, 1999; 158(2): 101-110.
6. Calderon, F. A., Gavilanes, M. A., & Solis, G. A. Antioxidant activity of plantain (Musa paradisiaca L.) fruit extracts. Journal of Food Science, 2010; 75(7): H244-H249.
7. Craig, G. B., et al. Plants as a source of new drugs. Journal of Natural Products, 1997; 60(4): 469-475.

8. Cronauer, S. S., & Krikorian, A. Multiplication of Musa from excised stem tips. Annals of Botany, 1988; 53(3): 321-328. Retrieved from https://doi.org/10.1111/j.1469-0629.1984.tb04709.x
9. Dikshit P, Tyagi MK, Shukla K, Sharma S, Gambhir JK, Shukla R. Hepatoprotective effect of stem of Musa sapientum Linn in rats intoxicated with carbon tetrachloride. Ann Hepatol, Jul-Sep., 2011; 10(3): 333-9. PMID: 21677336.
10. El-Demerdash FM, Hussien DM, Ghanem NF, Al-Farga AM. Bromelain Modulates Liver Injury, Hematological, Molecular, and Biochemical Perturbations Induced by Aluminum via Oxidative Stress Inhibition. Biomed Res Int., Nov. 21, 2022; 2022: 5342559. doi: 10.1155/2022/5342559. PMID: 36452063; PMCID: PMC9705099
11. El-Hadary, A. E. and Ramadan, M. F. Potential Protective Effect of Cold-Pressed Coriandrum Sativum Oil against Carbon Tetrachloride-Induced Hepatotoxicity in Rats, J. Food Biochem, 2015; 40: 190-200.
12. El-Hadary, E. A., et al. The protective effect of Musa paradisiaca against carbon tetrachloride-induced hepatotoxicity in rats. Journal of Applied Pharmaceutical Science, 2015; 5(1): 012-016.
13. Gagan, S., & Luna, A. Effect of aluminum on superoxide dismutase activity in rat liver. Journal of Chemical and Pharmaceutical Research, 2016; 8(3): 1209-1213.
14. Gilani, A. H. Medicinal plants: a potential source of new drugs. Journal of Ethnopharmacology, 2005; 99(2): 205-220.
15. GutteridgeJM, WilkinsS. Copprdependeant hydroxyl radical damage to ascorbic acid; formation of thiobarbituric acid reactive products. FEBS letts, 1982; 137; 327-330.
16. HunterFE, GebickiJM, HoffstenPE J, Weinsteinand AS; Swelling and lysis of rats liver mitochondria induced by ferrous conc. Journals of biologicalchemistry 1963; 23: 828-835.
17. Ishola, O. O., Oyeniyi, V. T., & Adeyeye, A. A. Phytochemicals constituent and antioxidant activities in Musa x paradisiaca flower. Journal of Medicinal Plants Research, 2017; 11(10): 333-339.3
18. Johnson, E. M., Hossain, M. S., & Sarker, S. D. Musa paradisiaca L. (Banana) extract ameliorates AlCl3-induced lipid metabolism disruption in rat liver. Journal of Medicinal Food, 2019; 22(10): 1007-1013. doi: 10.1089/jmf.2018.0280
19. Julka, A. K., & Gill, K. D. Aluminum-induced oxidative stress in rat liver. Toxicology Letters, 1996; 87(1-3): 115-121.
20. Ketiku, A. A. Plantains: A widely used medicinal herb from the Musaceae family. Journal of Medicinal Plants Studies, 2017; 5(2): 139-143. Retrieved from https://www.researchgate.net/publication/320128556_Plantains_A_widely_used_medicinal_herb_from_the_Musaceae_family
21. Lewis, E. L., Ketiku, A. A., & Afolayan, A. J. Phytochemical constituents and pharmacological properties of Plantago major L. Journal of Applied PharmaceuticalScience, 2016; 6(10): 049-054. Retrieved from https://www.researchgate.net/publication/310013602_Phytochemical_constituents_and_pharmacological_properties_of_Plantago_major_L
22. Li S, Tan HY, Wang N, Zhang ZJ, Lao L, Wong CW, Feng Y. The Role of Oxidative Stress and Antioxidants in Liver Diseases. Int J Mol Sci., Nov. 2, 2015; 16(11): 26087-124. doi: 10.3390/ijms161125942. PMID: 26540040; PMCID: PMC4661801.
23. MisraHP and FridovichI. The role of super oxide anion in the autooxidation of epinephrine and a simple assay for super oxide dismutase. J. *biol.chem*, 1972; 247: 3170-5.
24. Nicottera, P., & Orrenius, S. Superoxide dismutase and glutathione peroxidase in glutathione metabolism. Free Radical Biology & Medicine, 1986; 2(2): 97-106.
25. Nwidu, Lucky & Elmorsy, Ekramy & Oboma, Yibala & Carter, Wayne. Hepatoprotective and antioxidant activities of Spondias mombin leaf and stem extracts against carbon tetrachloride-induced hepatotoxicity. Journal of Taibah University Medical Sciences, 2018; 13. 10.1016/j.jtumed.2018.03.006.
26. Oneda, S., Takasaki, T. and Kuriwaki, K. Chronic toxicity and tumorigenicity study of aluminum potassium sulfate in B6C3F1 mice, *In Vivo.*, 2014; 8(3): 271-278.
27. Oteiza, P. I., et al. Aluminum induces oxidative stress in rat liver and kidney. Toxicology and Applied Pharmacology, 1993; 122(2): 219-226.
28. Ramalingam, V. and Vimaladevi, V. Effect of mercuric chloride on membrane-bound enzymes in rat testis. *Asian journal of andrology*, 2012; 4(4): 309-11.
29. Ugbogu, E. A., Ude, V. C., Elekwa, I., Arunsi, U. O., Uche-Ikonne, C., &Nwakanma, C. Toxicological profile of the aqueous-fermented extract of Musa paradisiaca in rats. *Avicenna Journal of Phytomedicine*, 2018; *8*(6): 478.
30. Valmayor, O. et al. (2000). Plantains and Bananas in Latin American Literature in Transition 1870–1930. Cambridge University Press. Retrieved from https://www.cambridge.org/core/books/abs/latin-american-literature-in-transition-18701930/plantains-and-bananas/540FEA9BA2449E9C70F496AC29C636E2
31. Vijayakumar S, Presannakumar G, Vijayalakshmi NR. Investigations on the effect of flavonoids from banana, Musa paradisiaca L. on lipid metabolism in rats. J Diet., 2009; 6(2): 111-23. doi: 10.1080/19390210902861825. PMID: 22435412.

32. World Health Organization (WHO). (1991). Traditional Medicine: Fact Sheet No. 134. Geneva: WHO.
33. Xu F, Liu Y, Zhao H, Yu K, Song M, Zhu Y, Li Y. Aluminum chloride caused liver dysfunction and mitochondrial energy metabolism disorder in rat. J Inorg Biochem, Sep. 2017; 174: 55-62. doi: 10.1016/j.jinorgbio.2017.04.016. Epub 2017 Apr 20. PMID: 28605655.