Review Article

An Experimental Study: The Effect of *Garcinia kola* on Superoxide Dismutase, Catalase, and Lipid Peroxidation Levels in the Kidney of Mice

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Abstract

Garcinia kola is a tropical plant with various traditional medicinal uses, and its potential effects on antioxidant enzymes and lipid peroxidation make it a subject of interest for scientific investigation. This study was conducted over 2 months and 6 days to investigate the effect of consuming *G. kola* on catalase and superoxide dismutase (SOD) activities and lipid peroxidation levels in the kidney of mice. Twelve female albino mice were divided into four groups, each consisting of three. Group 1 (control) received only growers mash, while groups A, B, and C were fed diets containing 1%, 3%, and 6% *G. kola*, respectively. The study revealed that mice fed with 1% *G. kola* exhibited a significant increase in SOD activity in their kidneys compared to other experimental groups. However, no significant changes were observed in catalase activity and lipid peroxidation levels among all the experimental groups. These findings suggest that dietary consumption of *G. kola* appears to be nontoxic to mice.

Keywords: Antioxidant enzyme, bitter kola, catalase, *Garcinia kola*, kidney, lipid peroxidation, mice, phytochemicals, superoxide dismutase

Introduction

Garcinia kola, commonly known as bitter kola, is a tropical plant belonging to the Guttiferae family.[1] It is native to the forests of West and Central Africa and is widely cultivated and distributed in the region. This medium-sized tree can grow up to 12 m in height. Bitter kola has a long history of traditional medicinal use among various indigenous communities in Africa, and its medicinal properties have led to its inclusion in numerous remedies and practices.[2] Among its reported medicinal uses, bitter kola is known for its purgative, antiparasitic, and antimicrobial properties.[3,4] It has been employed to treat respiratory ailments such as bronchitis and throat infections, alleviate colic, cure head or chest colds, and relieve coughs. In addition, bitter kola has been used in traditional medicine to address liver disorders and serve as a traditional chewing stick. The potential health-promoting effects of bitter kola are attributed to its active constituents, including phytochemicals, vitamins, and minerals.[5]

Furthermore, bitter kola has been studied for its diverse pharmacological activities, such as anti-inflammatory, antimicrobial, antiviral, antidiabetic, and anti-hepatotoxic effects. These properties have garnered attention from researchers and practitioners, prompting further investigations into their potential medicinal applications.[6-9] Due to its widespread utilization as a medicinal agent and its significant role in traditional practices, there is a growing need to study bitter kola's safety profile and potential toxicity. Understanding its impact on various physiological processes and organs, such as the kidney, can provide valuable insights for optimizing its medicinal use and ensuring the well-being of individuals relying on its traditional benefits.

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The present study aims to evaluate the effect of *G. kola* on the activities of SOD and catalase, as well as the levels of lipid peroxidation, in the kidneys of mice. This study holds importance as medicinal agents, including plant-based ones, are commonly metabolized in the liver or kidney, making these organs crucial for assessing potential toxicity. Examining the activities of these enzymes and lipid peroxidation offers valuable insights into how bitter kola affects kidney health.

Literature Review

History of medical plants

Since the dawn of humanity, the fear of illness and mortality, coupled with the need for sustenance and well-being, has driven people from all corners of the world to explore the offerings of nature.[10,11] Through trial and error, our ancestors discovered the healing powers of plants and their potential to protect and enhance health. This profound wisdom led to the emergence of medicinal plant use across cultures and civilizations.

One of the earliest documented instances of herbal medicine dates back to ancient China, between 2730 and 3000 before common era (BCE).[12] The *Hydnocarpus gaertn* species produced chaulmoogra oil, renowned for its effectiveness in treating leprosy. Unfortunately, the identity of the pioneer who harnessed this medicinal potential remains unknown.[13,14] In parallel, ancient Egyptian records inscribed on the Ebers papyrus around 1500 BCE highlight the use of opium poppy seeds and castor oil for therapeutic purposes. Throughout history, prominent figures such as Hippocrates, often called the father of medicine (460 BCE), documented their knowledge of medicinal plants. Among his descriptions were herbs such as opium mint, sage, rosemary, and *Verbena*, showcasing the widespread use and appreciation of these natural remedies.[15]

During the middle ages, the writings of Galen (AD 131) gained popularity as he treated various ailments with herbal remedies.[16,17] In addition, in Europe during the 14th century, the doctrine of signatures (1496–1541) influenced the use of medicinal plants.[18] According to this doctrine, plants were believed to have specific features bestowed by God, indicating their suitability for treating particular diseases or body parts.[19]

Medicinal plants: Definitions and applications

The World Health Organization consultative group defined medicinal plants as those containing substances useful for therapeutic purposes or as precursors for synthesizing beneficial drugs. These plants can be utilized in several ways:

Botanical preparations

Medicinal plant parts or whole plants are used directly in traditional remedies. An example is using cascara bark in herbal medicine.[20]

Extraction of pure substances

Medicinal plants are a source of essential compounds used directly for medical purposes or as starting materials in the hemi-synthesis of medicinal compounds. Ginger, used as a spice and for medicinal purposes, exemplifies this category.

Microscopic plants

Certain fungi and actinomycetes are sources for isolating drugs, especially antibiotics.

Fiber plants

Plants such as cotton, besides their primary use as fibers, have applications in surgical dressing.

Medicinal plants in therapy: Phytochemistry, pharmacognosy, and horticulture

The research on medicinal plants primarily focuses on three interconnected areas: phytochemistry, pharmacognosy, and horticulture.[21] In phytochemistry, scientists explore medicinal plants for their bioactive compounds, which are then isolated and subjected to detailed structural analysis. Pharmacognosy, on the other hand, involves evaluating the bioactivity of these compounds and identifying their potential modes of action and target sites within the body. This understanding aids in optimizing the use of medicinal plants in various therapeutic approaches. Horticulture research related to medicinal plants aims to develop optimal cultivation methods. This is crucial to ensure a stable and sustainable supply of medicinal plant materials while maintaining the plants' potency and efficacy.

Medicinal plants boast diverse chemical compositions, each contributing to their unique therapeutic effects.[22] In contrast, primary products such as carbohydrates, lipids, proteins, heme, chlorophyll, and nucleic acids are common to all plants. They are essential in their growth and development. Many phytomedicines exert their benefits through the synergistic or additive actions of several chemical compounds, acting on specific target sites associated with physiological processes.[23] This multifaceted approach can reduce potential side effects compared to relying on a single-dominant compound.

Garcinia kola: A potent medicinal plant

G. kola, commonly known as bitter kola, is a medium-sized tree found in various forests throughout West and Central Africa.[24] Its fruit is reddish, yellowish, or orange, and each fruit contains 2–4 yellow seeds embedded in a sour-tasting pulp.[25] Notably, the flowering period spans December to January, with fruit maturation between June and August. This remarkable plant contains several active constituents, including phenols, flavonoids, alkaloids, tannins, and saponins. In addition, it boasts a rich vitamin composition, including thiamin (Vitamin B1), riboflavin (Vitamin B2), niacin (nicotinic acid), and ascorbic acid (Vitamin C). Its mineral content encompasses essential macroelements such as magnesium, calcium, potassium, phosphorus, and sodium and microelements such as iron, zinc, copper, and manganese.[26]

G. kola has been highly valued for its versatile medicinal applications. Its seeds are chewed as an aphrodisiac and are used to treat cough, dysentery, and chest colds in traditional

herbal medicine. Moreover, it holds promise as a valuable raw material for the pharmaceutical industry. The stem bark serves as a purgative, the powdered bark as a treatment for malignant tumors, and the sap for curing skin diseases caused by parasites. In addition, *G. kola* seeds are employed in the treatment of cholic disorders, head or chest colds, and conditions such as cirrhosis and hepatitis, mainly due to their potential anti-inflammatory, antibacterial, and anti-fungal properties.[27]

Antioxidant enzymes: Catalase and superoxide dismutase

In the quest to understand medicinal plants' biochemical aspects, two vital antioxidant enzymes stand out: catalase and superoxide dismutase (SOD).

Catalase (EC.1.11.1.6)

Catalase is a hemoprotein containing four heme groups, and its primary function involves the breakdown of hydrogen peroxide (H_2O_2) into water and oxygen. It can use one molecule of H_2O_2 as a substrate electron donor and another molecule of H_2O_2 as an oxidant or electron acceptor.[28]

$$2\,H_2O_2 \xrightarrow{\text{catalase}} 2H_20 + 0_2$$

This essential antioxidant enzyme is found in various tissues, including blood, bone marrow, mucous membranes, kidney, and liver. It neutralizes H_2O_2 formed during cellular metabolic processes, protecting the cell from oxidative damage. Factors affecting catalase activity include substrate concentration, temperature, pH, salt concentration, the presence of inhibitors, and the presence of activators.

Substrate concentration

If all other conditions are constant, the reaction rate should increase with substrate concentration. At very low values of the substrate, the reaction rate will increase very rapidly. At higher substrate concentrations, the rate begins to level off. Eventually, the maximum rate of that reaction will be achieved, and a further increase in substrate concentration will have no effect.

Temperature

In general, chemical reactions speed up as the temperature is raised. When the temperature increases, more reacting molecules have the kinetic energy required to undergo the reaction.[29]

pH measures a solution's acidity or hydrogen ion concentration.[30] As the pH drops into the acidic range, an enzyme gains hydrogen ions from the solution. The enzyme loses hydrogen ions to the solution as the pH moves into the basic range. In both cases, the changes produced in the chemical bonds of the enzyme molecule result in a change in conformation that decreases enzyme activity.

Salt concentration

Every enzyme has an optimal salt concentration, in which it can catalyze reactions. Too high or too low, a salt concentration will denature the enzyme.[31]

Presence of inhibitors

A molecule that interacts with the enzyme and decreases its activity is an inhibitor.[32]

Presence of activators

An activator is a molecule that interacts with an enzyme and increases its activity.[33]

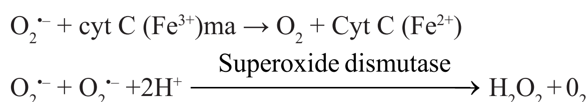
Superoxide dismutase (SOD, EC 1.15.1.1)

SOD plays a critical role in combating the harmful effects of superoxide radicals (O2•−), converting them into less harmful H_2O_2 and oxygen.[34] SOD is present in aerobic cells' cytosol and mitochondria and acts as a first-line defense against oxidative stress. It possesses a catalytic mechanism involving copper and zinc ions coordinated with specific amino acid residues, facilitating the dismutation of superoxide radicals.

Superoxide is formed when reduced flavins present, for example, in xanthine oxidase - are reoxidized univalent by molecular oxygen.

$$\text{Enz-flavin -}H_2 + 0_2 \text{ - Enz-flavin-H} + O_2^{\bullet -} + H^+$$

Superoxide can reduce oxidized cytochrome C or be removed by SOD.



$$O_2^{\bullet -} + \text{cyt C } (Fe^{3+})\text{ma} \rightarrow O_2 + \text{Cyt C } (Fe^{2+})$$

$$O_2^{\bullet -} + O_2^{\bullet -} + 2H^+ \xrightarrow{\text{Superoxide dismutase}} H_2O_2 + 0_2$$

Figure 1 presents the proposed catalytic mechanism of SOD. A copper ion at the active site cycles between +1 and +2 oxidation states. The histidine side chain coordinated to Cu^{2+} during part of the cycle is also bounded to Zn^{2+}. The other key participant is an arginine side chain. The active site of the cytosolic enzyme in eukaryotes contains a copper and a zinc ion coordinated to the side chain of a histidine residue [Figure 1]. The negatively charged so is guided electrostatically to a very positively charged catalytic site at the bottom of a channel. $O_2^{\bullet -}$ binds to Cu^{2+} and the guanidino group of an arginine residue. An electron is transferred from SO to a cupric ion to form Cu^+ and O_2, which is released. A second SO enters the active site and binds to Cu^+ arginine and H_30^+. The bound $O_2^{\bullet -}$ acquires an electron from Cu^+ and two protons from its binding partners to form H_2O_2 and regenerate the Cu^{2+} state of the enzyme. The enzyme occurs in all major aerobic tissues in the mitochondria and the cytosol. Although animal exposure to an atmosphere of 100% oxygen causes an adaptive increase in SOD, particularly in the lung.

Lipid peroxidation and antioxidant defense

Lipid peroxidation is a chain reaction responsible for the oxidative damage of lipids, leading to various health issues and aging. Peroxidation occurs when free radicals, such as ROO•, RO•, and OH•, attack fatty acids containing methylene-interrupted double bonds. To counteract lipid peroxidation, the body relies on antioxidants, which can be preventive or chain-breaking.[35,36]

The reaction is imitated by an existing free radical (X), light, or metal ions. Malondialdehyde (MDA) is only formed by fatty

acids with three or more double bonds. It is used as a measure of lipid peroxidation together with ethane from the terminal two carbon of w_6 fatty acids and pentane from the terminal five carbons of we fatty acid [Figure 2]. Lipid peroxidation is a chain reaction providing a continuous supply of free radicals that initiate further peroxidation.[37,38] The whole process can be depicted as follows:

1. Initiation
   $ROOH + Metal^{(n)+} \rightarrow ROO^{\bullet} + Metal^{(n-1)} + H^{+}$
   $X^{\bullet} + RH \rightarrow R^{\bullet} + XH$
2. Propagation
   $R^{\bullet} + 0_2 \rightarrow ROO^{\bullet}$
   $ROO^{\bullet} + RH \rightarrow ROOH + R^{\bullet}$ etc.
3. Termination
   $ROO^{\bullet\circ} + ROO^{\bullet} \rightarrow ROOR + 0_2$
   $ROO^{\bullet} + R^{\bullet} \rightarrow ROOR$
   $R^{\bullet} + R^{\bullet} \rightarrow RR$

To control and reduce lipid peroxidation, both humans in their activities and nature invoke the use of antioxidants.[39] Preventive antioxidants, such as catalase, reduce the rate of chain initiation, while chain-breaking antioxidants, such as SOD, interfere with chain propagation.[40,41] These antioxidant enzymes work together to maintain a delicate balance, safeguarding cells from oxidative stress, and preserving overall health.

Materials and Methods

Materials

In the experiment, the following apparatus/instruments were used: A spectrophotometer (UV 7504) from Lengguang Tech. China, a Centrifuge from Remi's Motors Ltd., test tubes, test tube holders, a water bath, an electronic weighing balance (Setra BL-4105), needles and syringes, cotton wool, hand gloves, a measuring cylinder, beakers, a desiccator, graduated pipettes, ethylenediaminetetraacetic acid (EDTA) free tubes, bitter kola extract, chloroform, and conical flasks.

The required chemicals and their manufacturers were carbonated buffer from British Drug House (BDH) in Poole,

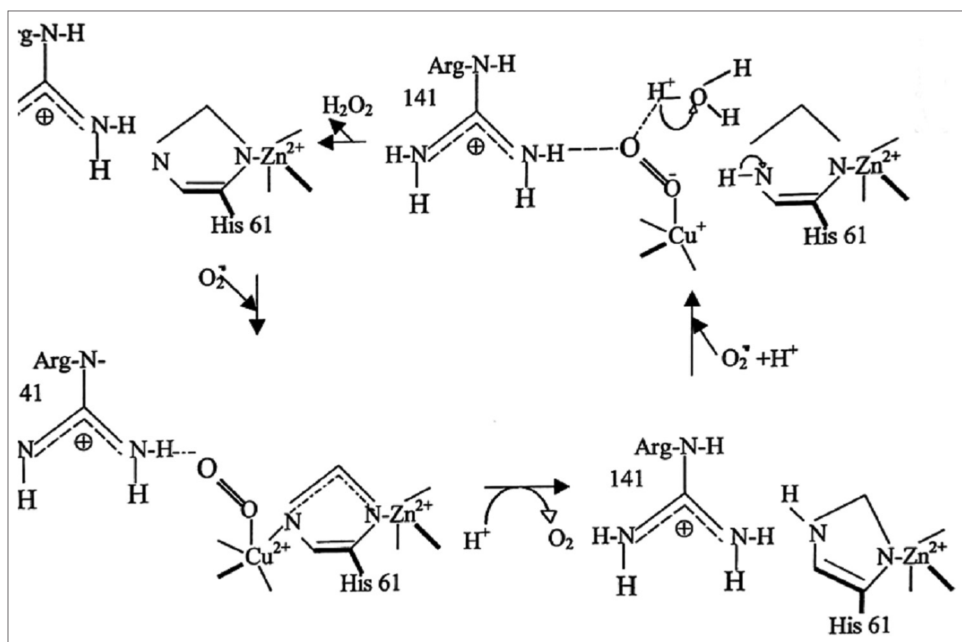


Figure 1: Proposed catalytic mechanism of superoxide dismutase (Stryer, 2000)

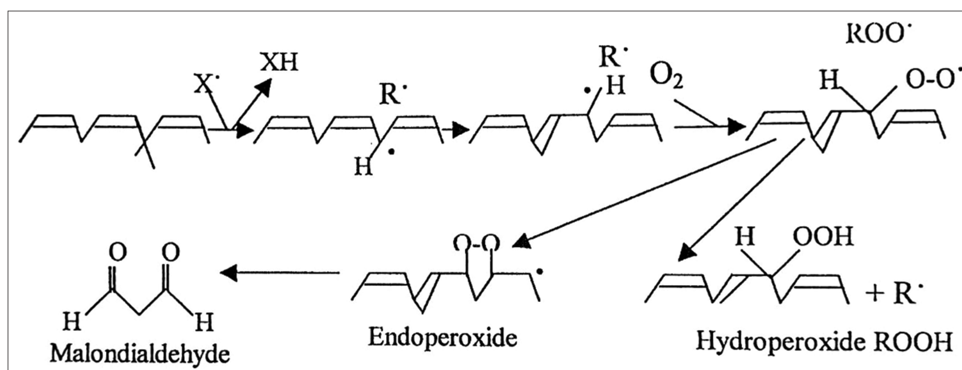


Figure 2: Lipid peroxidation

England, hydrochloric acid (HCI) from May and Baker in Dagenham, England, phosphate buffer from May and Baker in Dagenham, England, epinephrine from BDH in Poole, England, tetraoxosulfate (IV) acid (H2SO4) from May and Baker in Dagenham, England, saline solution from the Biochemistry Department, Delta State University, Abraka, thiobarbituric acid (TBA) from Mark, New Jessy, USA, trichloroacetic acid from BDH in Poole, England, and potassium permanganate from May and Baker Ltd. in Dagenham, England.

For the experimental animals, 12 female albino mice were purchased from Lagos University Teaching Hospital with an average weight of 50g. These mice were bred in the animal house of Delta State University. They were kept in standard mice cages and allowed to acclimatize to the laboratory conditions for 2 weeks.

Methods

The seeds of *G.kola* were purchased from Abraka market, Ethiopia East Local Government Area of Delta State, Nigeria. Ripe seeds of *G. kola*, weighing 1 kg, were sun-dried and ground into a uniform powder using a Thomas Wiley machine. The resulting powder was stored in air-tight bottles until it was required for analysis.

For the treatment of experimental animals, 12 female albino mice were divided into four groups, with each group consisting of three mice. Group I (control) was given 100g growers mash only, while groups 2, 3, and 4 were fed with 1%, 3%, and 6% *G. kola* in their diets, respectively. The exposure period lasted for 2 months and 6 days.

After the treatment period, each mouse was sacrificed, and their kidneys were collected into EDTA containers, weighed, and stored at -20°C until further use. To prepare the tissue samples, a 10% homogenate of the kidney from each mouse was created using 0.9% NaCl solution under cooled conditions. The homogenate was centrifuged at 3,000g for 10 min, and the obtained supernatant was used for biochemical analysis.

Biochemical assay

Assay for superoxide dismutase

The activity of SOD was assayed by the method of Misra and Fridovich (1972).

Principle

The assay of SOD is an indirect method based on the inhibitory effect of SOD on the initial rate of epinephrine autoxidation, which is derived from the reaction proposed by Misra and Fridovich (1972)[42] for the base-catalyzed autoxidation of epinephrine. Thus, if RH_3 represents epinephrine and R represents adrenochrome. The following reactions represent the chain reaction, which might occur at high pH.

$$RH^-_3 + Me^n \rightarrow RH_3 + Me^{n-1} \quad (a)$$

$$RH_3 + 0_2 \rightarrow RH_2 + 0^-_2 + H^+ \quad (b)$$

$$RH_3 + 0_2 + H^+ \rightarrow RH + H_2O_2 \quad (c)$$

$$RH + 0_2 \rightarrow R + 0^-_2 + H^+ \quad (d)$$

$$RH^-_3 + 0^-_2 + 2H^+ \rightarrow RH_3 + H_2O_2 \quad (e)$$

In this way, one initiating event was shown as the univalent oxidation of an epinephrine anion by a metal caution reaction (a) or by a superoxide anion reaction (e), which starts a chain of reactions, in which O_2 is a propagative species. It is clear that SOD should strongly inhibit this mechanism. At lower pH, the organic radical generated by the initial event could lead to adrenochrome formation by a series of dismutation reactions such as:

$$RH_3 + RH_3 \rightarrow RH_2 + RH_4 \quad (f)$$

$$RH_3 + RH_2 \rightarrow RH + RH_4 \quad (g)$$

$$RH + RH \rightarrow R + RH_2 \quad (h)$$

In this case, SOD cannot inhibit adrenochrome formation. The reduced metal generated in reaction (a) could, in any case, be oxidized by reaction with oxygen.

$$Me^{n-1} + 0_2 \rightarrow Me^n + 0_2 \quad (i)$$

The oxygen radical generated by reaction (i) could either dismutase or react with epinephrine in reaction (b).[42]

Procedure

The assay was performed by adding 0.2 ml of the supernatant to 2.5 ml, 0.5 M carbonate buffer pH 10.2. The reaction was started by adding 0.3 ml of freshly prepared 0.3 mM epinephrine as the substrate to the buffer supernatant mixture and was quickly mixed by inversion. The reference cuvette contained 2.5 ml of the buffer, 0.3 ml of the substrate, and 0.2 ml of distilled H_2O. The increase in absorbance at 480 nm due to the adrenochrome formed was monitored every 30 s for 150 s. One unit of SOD activity is given as the amount of SOD activity necessary to cause 50% inhibition of the oxidation of epinephrine to adrenochrome for 9 min.

Blank is prepared by adding 2.5 ml of carbonate buffer to 0.3 ml of epinephrine and 0.2 ml of distilled water in place of the sample, i.e., the supernatant. Enzyme activity can be expressed as a percentage inhibition, as shown in Equation 1.

$$100 - 100 \times \frac{\text{Epinephrine in the presence of SOD}}{\text{Epinephrine in the absence of SOD}} \quad (1)$$

Assay for catalase activity

The activity of this enzyme was determined according to the method of Cohen, Dembiec, and Marcus (1970).[43]

Principle

The principle of the assay is derived from the fact that in the UV range, H_2O_2 shows a continual increase with decreasing wavelengths. The absorbance decomposition of H_2O_2 can thus be followed directly by the increase in extinction at 240nM (E240 = 40 $cm^3/\mu mol$)—the difference in extinction, E240 per unit of time, measures catalase activity.

Procedure

Excess potassium permanganate is added to the assay, and then, the residual untreated permanganate is measured

spectrophotometrically. It has been shown that the decomposition of H_2O_2 by catalase follows first-order kinetics.[44] The assay was performed by adding 0.5 ml of the samples into cold test tubes while the blank contained 0.6 ml of distilled water. Enzymatic reactions were initiated by adding sequentially at fixed intervals 5.0 ml of the samples into hard test tubes. At the same time, the blank contained 0.5 ml of distilled water. Enzymatic reactions were initiated by adding sequentially at fixed intervals 5.0 ml of cold 30mM $H_2O_{2,}$ which were mixed thoroughly by inversion. After exactly 3 min, the response was stopped sequentially at the same fixed intervals by rapidly adding 1m 6N H_2SO4 and was also mixed quickly by inversion. To the enzyme samples and blank taken one at a time, 7.0 ml 0.01N KmnO4 was added, mixed thoroughly, and read at 480mM within 30–60 s in a spectrophotometer. The spectrophotometer standard was prepared by adding 7.0 ml of 0.01N $KmnO_4$ to a mixture of 5.5 ml 0.05M phosphate buffer pH 7.0 and 1.0 ml 6N H_2S0_4. The spectrophotometer was zeroed with distilled water; under the conditions described, the decomposition of H_2O_2 by catalase follows first-order kinetics given by the equation.

$$K = \text{Log}\frac{S_0}{S_3} \times \frac{2.3}{t} \quad (2)$$

Where X is the first-order rate constant, t is the time interval over which the reaction is measured (3 min), S_0 is the substrate concentration at zero time, and S_3 is the substrate concentration at 3 min.

To obtain S_0 (expressed in absorbance unit), the absorbance of the reaction system of the blank (SB) was subtracted from the spectrophotometric standard (St). Moreover, to obtain S_3, the absorbance of the reaction sample (SA) was subtracted from St, as shown in Equation 3.

$$S_3 = St - SA \quad (3)$$

Assay for the level of lipid peroxidation

The level of lipid peroxidation in the kidney was determined by this method of Hunter.[45]

Principle

MDA, formed from the breakdown of polyunsaturated fatty acids, served as a convenient index for determining the extent of the peroxidation reaction. MDA has been identified as the product of lipid peroxidation that reacts with TBA to give a red species absorbing at 535 m.

Procedure

0.5 ml of sample and 3.5 ml of stock reagent were mixed in a corked test tube and heated for 15 min in a boiling water bath. After cooling at room temperature, the precipitate was removed by centrifugation 1000xg for 10 min, and the absorbance of the supernatant was measured at 532 nm against the blank containing all the reagents except the sample.

Calculation

The MDA concentration of the sample was calculated from the absorbance using an extinction coefficient of $1.56 \times 10^5 m^{-\prime}$cm and according to the method of Adam-Vizi and Seregi.[46]

$$\text{Lipid peroxidation} = \frac{A \times Vm \times I}{\text{Molar absorbency index} \times Vs \times X^1 \text{ for MDA}} \quad (4)$$

Where A = absorbance, Vm = Volume of the reaction mixture (5ml), Vs = Volume of the sample (0.5 ml), Molar absorbency index = 1.56×10^5 m^{-1} cm^{-1}, and X^1 = gram of sample in the reaction mixture.

Results

The result of the analysis carried out is presented in Tables 1-3. Table 1 shows the effect of *G. kola* on the kidney SOD activity of mice. The result indicates that the activity of SOD in the kidney of mice fed 1% *G. kola* was significantly increased compared to the other experimental groups.

Table 2 represents the effect of *G.kola* kidney catalase activity in mice. No significant change was observed in the activity of catalase in the kidneys of mice in all the experimental groups.

Table 3 presents the effect of *G. kola* on mice's kidney lipid peroxidation levels. No significant change was observed in the level of lipid peroxidation in the kidneys of mice in all the experimental groups.

Discussion

G. kola, a remarkable medicinal plant, has garnered attention for its multifaceted properties. Studies have revealed that *G. kola* exhibits anti-inflammatory, antimicrobial, and antiviral activities, underscoring its potential therapeutic value. The complex chemical composition of *G. kola* comprises bioflavonoids, prenylated benzophenones, and xanthones, as highlighted by Terashima, Aqil, and Niwa (1995).[47] Among these compounds, bioflavonoids are particularly prominent. At

Table 1: Effects of *Garcinia Kola* on superoxide dismutase activity in the kidney of mice

	Experimental groups			
Parameters	0%	1%	3%	6%
SOD units/g tissue	15.16±6.45[a]	23.18±0.288[a]	11.64±0.1439[a]	11.59±0.1599[a]

Results are expressed as mean±SD. Mean of the same row followed by different letters differ significantly ($P<0.05$). SOD: Superoxide dismutase, SD: Standard deviation

Table 2: Effect of *Garcinia Kola* on catalyst activity in the kidney of mice

	Experimental groups			
Parameters	0%	1%	3%	6%
Catalase units/g tissue	0.316±0.039[a]	0.247±0.149[a]	0.305±0.052[a]	0.213±0.138[a]

Results are expressed as mean±SD. The mean of the same row followed by the same letters shows no significant difference ($P<0.05$). SD: Standard deviation

Table 3: Effect of *Garcinia Kola* on the level of liquid peroxidation in the kidney of mice

	Experimental groups			
Parameters	**0%**	**1%**	**3%**	**6%**
Liquid peroxidation MDA/g wet tissue	68.24±1.28[a]	95.49±51.16[a]	55.95±10.14[a]	58.32±0.61[a]

Results are expressed as mean±SD. The mean of the same row followed by the same letters shows no significant difference ($P<0.05$). MDA: Malondialdehyde, SD: Standard deviation

the same time, kolaviron, consisting of GB-1, GB-2, and kola flavanones, contributes significantly to *G. kola's* medicinal attributes.

Remarkably, *G. kola's* effects extend to the kidneys, as indicated by histological changes observed by Braide and Grill (1990).[48] These findings suggest that kolaviron might protect against carcinogens and drug-induced oxidative and membrane damage, potentially playing a role in the chemotherapy of kidney diseases. To delve further into the biochemical impact of *G. kola*, a thorough analysis was conducted on its effects on SOD, catalase, and lipid peroxidation levels. Remarkably, mice fed with 1% *G. kola* exhibited increased kidney SOD activity, indicating oxidative stress within this group.[49] However, no significant changes in this parameter were observed at higher concentrations. Similar outcomes have been reported in other studies where initial toxicity at lower concentrations of toxicants was reversed when higher concentrations were administered.

The lack of significant difference in SOD and catalase activities among the various groups of mice is expected, given that both enzymes work in tandem. This observation may also be linked to the observed lack of significant difference in lipid peroxidation levels in the respective mouse groups. It is worth noting that an inverse relationship has been reported between SOD and lipid peroxidation.[50] Importantly, the present study concludes that consuming various doses of *G. kola* is nontoxic to mice, particularly at lower doses, as there were no significant alterations in the parameters studied.

Conclusion

In summary, the research on *G. kola* underscores its remarkable potential as a nontoxic medicinal plant, offering a plethora of therapeutic benefits. The documented anti-inflammatory, antimicrobial, and antiviral properties highlight its suitability for various health applications. The diverse chemical composition, including prominent bioflavonoids and the essential kolaviron, contributes to its extensive medicinal attributes. Moreover, the protective effects of *G. kola* on the kidneys, evident from histological changes, open up possibilities for combating kidney diseases and oxidative damage. The biochemical analysis further substantiates *G. kola's* role in modulating antioxidant enzyme activities, notably SOD and catalase and regulating lipid peroxidation levels.

Given the nontoxicity observed in mice, *G. kola* shows promise as a safe botanical remedy. This favorable profile may hold significant potential for human consumption, warranting further exploration for its application in human health care. Ultimately, the findings highlight the enduring relevance of traditional medicinal plants such as *G. kola* in modern medicine and reinforce their role as valuable resources for promoting health and well-being.

Recommendation

In light of the valuable medicinal properties and diverse chemical composition of *G. kola*, it is imperative to advocate for and invest in further research on this remarkable plant. The existing knowledge about its biological effects, such as anti-inflammatory, antimicrobial, and antiviral activities, represents only the tip of the iceberg. Comprehensive studies are essential to unlock the full potential of *G. kola* and harness its benefits for human health and various industries.

Scientists can unravel the intricate mechanisms underlying the plant's therapeutic effects by conducting in-depth research. Exploring its bioactive compounds, including bioflavonoids, prenylated benzophenones, and xanthones, could reveal new insights into how these components interact with biological systems and contribute to their medicinal attributes. In addition, unraveling the role of kolaviron, consisting of GB-1, GB-2, and kola flavanones, is crucial in understanding the plant's holistic medicinal properties.

As the current research indicates a potential inverse relationship between SOD and lipid peroxidation, a comprehensive exploration of the interplay between antioxidant enzymes and lipid peroxidation in response to *G. kola* consumption is warranted. Further investigations could unveil intricate biochemical pathways and shed light on potential strategies for managing oxidative stress-related diseases. In addition, exploring the potential synergistic effects of *G. kola* with conventional medications or other medicinal plants could lead to novel therapeutic combinations, enhancing the efficacy and safety of treatment options.

In conclusion, the call for further research on *G. kola* is paramount to unlocking its full therapeutic potential and contributing to the advancement of modern medicine. Exploring the depths of its biological effects, chemical composition, safety profile, and potential synergies will allow us to tap into the abundant advantages of this natural resource, enhancing human health and well-being. The thorough investigation of *G. kola* holds the potential to revolutionize drug development, nutrition, and complementary medicine, bringing benefits to individuals and societies across the globe.

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Conflicts of interest

There are no conflicts of interest.

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