**Evaluation of *Garcinia mangostana* seed oil based feed in male rats.**

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**Abstract:** Evaluation of the nutritional and probable toxicological effect of *Garcinia mangostana* (GM) seed oil-based formulated diet on some biochemical parameters of selected rat tissue and serum was studied. The physicochemical properties of the seed oils gave acid value of 4.58±0.49 mg KOH/g, the iodine value of 53.65±2.34 mg I2/100g, peroxide value of 3.27±0.07 mEq / kg and saponification value 134.0±6.56mgKOH/ 100g. The smoke and flash points were 170 and 215 oC. Animals were placed in four groups of 6 rats each, group 1 had 10 % vegetable oil (VO), Group 2 had10 % GM, group 3 had 5 % GM and 5 %VO and group 4 had 3 % GM and 7 % VO. All animals had increase in body weight of over 30%, with group 4 having the highest gain in weight of 38.5%. The liver and brain weight were significantly decreased (p<0.05) in group 2 compared to control and Group 4 rats had significantly increased (p<0.05) organ weights relative to control. The serum triglyceride, cholesterol and high density lipoprotein cholesterol of the rats on GM seed oil increased but the artherogenic index decreased, specifically *Garcinia mangostana* decreased the atherogenic index by over 200 % compared to VO. 10 % GM seed oil feeding for eight weeks caused significant (p<0.05) increase in SOD and MDA, significantly (p<0.05) decreased catalase activity while GSH concentration was not statistically significant. GM oil fed animals had serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities decreased compared to control and histopathology result showed normal tissue architecture. The results from this study show GM oil is edible and 5 % *G. Mangostana* seed oil based feed was identified to have better effects in this experiment.

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1. **Introduction**

Fats and oils are an essential part of the diet as they are the source of the essential fatty acids and also provide twice as much energy as carbohydrates. Common edible oils world-wide are from rape seed, sunflower, olive, walnut, corn while in Nigeria we have palm oil (*Eleasis guineensis*) in South Eastern part majorly and ground nut (*Arachis hypogaea*) from the fast disappearing ground nut pyramids. Vast majority of the consumable oils are from cultivated plants. As a result of the ever increasing human population and the demand for oil in human and animal nourishment, industrial usage, the need for oil has been on the increase. Oils are expensive, especially the healthier variety with mono and poly unsaturated double bonds. The common sources of edible vegetable oils are becoming scarce and there is therefore the need for suitable, healthy and non-toxic alternatives from the wide array of trees in both the humid and semi-arid tropics for consumption (Odoemelam, 2005). Tropical seeds, either wild or cultivated offer a convenient and cheap means of providing nourishments in terms of vitamins, minerals, lipid, protein and carbohydrate, especially in rural populations (Eromosele *et al*., 1991; Glew *et al*., 2005).

*Garcinia mangostana* L. (mangosteen, Clusiaceae) is commonly known as Mangosteen. It is a tall tree which grows to about 9-18 m, with a straight trunk and rounded dense crown and the fruiting period is between July to December (Harborne and Baxter, 1993). Mangosteen has been used as a medicinal plant especially in the management of inflammation, skin infections and wound healing (Obolskiy *et al*., 2009). Other applications include the therapy of various conditions such as dysentery, different urinary disorders, cystitis and gonorrhoea (Pedraza-Chaverri *et al*., 2009). The pericarp of the fruit is used abdominal pain, diarrhoea, dysentery and skin diseases. Chromatographic separation of dimethylene chloride extract of the pericarp led to the isolation of two new highly oxygenated prenylated xanthones and some of the xanthones have shown activity in cancer cell lines comparable with standard drug (Jung *et al.,* 2006; Zhang *et al.*, 2010). Xantones from Mangosteen have been reported to be able to prevent atherosclerosis (William *et al*., 1995; Sargowo and Setiawan, 2012; Adiputro *et al*., 2013).

The antioxidant present in Mangosteen and the lipid modulatory effects in hyperlipidermia and the cardioprotective properties led us to investigate the effect of *Garcinia mangostana* seed oils compounded feed and its continual ingestion for eight weeks on various biochemical parameters such as lipid profile, antioxidant status of brain and liver post mitochondrial fractions as well as possible sign of toxicity in male rats while using commercially available oil as standard for comparison.

1. **Materials and Methods**

Seeds of *Garcinia mangostana* were harvested from Botanical Garden in University of Ibadan, Ibadan, identified by Mr D. Esimekhuai at the Herbarium of the Department of Botany, University of Ibadan, Ibadan Nigeria and voucher specimens were deposited at the Departmental Herbarium. The fresh fruits were room dried in Nutritional Biochemistry Laboratory. Dried seeds were ground to coarse texture using the dry cup of domestic Kenwood blender and subjected to Soxhlet extraction using *n*Hexane for 6 hours to obtain the seed oil. The impure oil was purified by adding 0.73 % NaCl solution to the oil in a separating funnel, the lower aqueous layer was removed and the organic phase was further purified thrice using chloroform, methanol, 0.58 % sodium chloride solution (5:48:47 v/v) to obtain the pure oil.

Physical analysis was carried out on the oil to check for its colour and to observe if it was in solid, semi-solid or liquid form at room temperature andthe density was also compared with other edible oils. Thermal stability parameter is suitable for deep frying, acid value is a measure of the age, quality and suitability for consumption and stability, the degree of unsaturation, the amount of free fatty acid in a given amount of fat/oil were done by measuring the peroxide number, free fatty acid content, saponification and iodine values using methods described by AOAC (1990) and the results are on Table 1.

Soybean oil sold as Grand vegetable oil, a product of Grand Cereals and Oil mills Limited, Bukuru, Jos, Nigeria, yellow maize (*Zea mays*) whole grains and soy beans were purchased from Bodija Market, Ibadan, Nigeria. These food items were cleaned out by hand picking and winnowing. Vitamin mix was obtained from BASF Aktiengesellschaft, Ludwigshafen, Germany and experimental diets were compounded using method of Ellison and Riddle, (1961) as shown on Table 2. Briefly, Corn starch was used as carbohydrate source, it was cleaned, sundried and milled. Soybean served as protein source, it was dehusked prior to making it into a fine flour. Mineral mix was from Sigma-Aldrich Co. Ltd., Poole Dorset, UK. The different food items of the diet were thoroughly mixed and made into pellets for easy handling by animals and was thoroughly oven dried in Biochemistry Department to prevent mould growth. Feeds were stored in air tight bags at 4 °C to prevent microbial contamination and auto-oxidation of the oil (Oladiji *et al*., 2010).

Feed compounding table using commercial vegetable oil and varying percentage of *Garcinia mangostana* seed oil

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Composition** | **Test Feed %** | **3 % GM** | **5 % GM** | **10 % GM** |
| **Soybean** | 27 | 27 | 27 | 27 |
| **Corn starch** | 59 | 59 | 59 | 59 |
| **Vitamin mix** | 4 | 4 | 4 | 4 |
| **Seed oil** | Nil | 3 | 5 | 10 |
| **Vegetable oil** | 10 | 7 | 5 | Nil |

Twenty four male albino rats (Wistar strain) weighing between 123 g and 143 g were obtained from the animal house in the Physiology Department Animal house, University of Ibadan, Ibadan. The rats were transferred to experimental animal house in Biochemistry Department, University of Ibadan and were allowed to acclimatize for two weeks. Rats were maintained on the standard rat chow (Ladokun Feed, Ibadan) with water *ad libitum* in the Biochemistry Department Animal house under normal room temperature before the commencement of the experiment.

Rats were distributed randomly into four different groups of six animals each. The control group received compounded diet prepared with 10 % commercially available Grand soybean oil purchased from Bodija market, Ibadan. All animals had 10 % oil in their different formulated feed except that the proportion of the different seed oils and Grand vegetable oil varied.

Group1: (10 % VO); Group2: (10 % GM); Group 3: (5 % GM + 5 % V.O.) and Group 4: (3 % GM + 7 % V.O.)

Animals were fed seed oil compounded feed for eight week and were weighed 24 hours after the last administration.

The animals were fasted overnight and were sacrificed by cervical dislocation. Serum was obtained from animals by using 2 ml syringe to obtain blood by cardiac puncture into clean sample bottles without anticoagulant, the bottles were left to stand for 1 h for complete coagulation. The clotted samples were spun at 3000 rpm for 10 minutes, the supernatant serum was removed and it was stored at 4 °C for analysis. The brain and liver were quickly removed, washed with 1.15 % KCl, homogenized in 56 mMTris-HCl buffer (pH 7.4) containing 1.15 % potassium chloride and the homogenate was centrifuged at 10,000 rpm for 15 minutes at 4 °C. Supernatant was stored at 0 °C until needed. Small pieces of liver and brain sections were fixed in 10 % formal saline and sent to Veterinary Anatomy Department, University of Ibadan, Ibadan for histopathological examination.

Quantification of the protein was by Biuret method (Gornal *et al*.*,* 1949) with bovine serum albumin (BSA) as standard. Lipid peroxidation (LPO) was assayed by measuring thiobarbituric acid reactive substances (TBARS) using the colorimetric assay reaction of the lipid peroxidation product malondialdehyde (MDA) with thiobarbituric acid (TBA) to form a pink precipitate, which was read at 532 nm by spectrophotometry. Catalase (CAT) activity was done by measuring the rate of decomposition of hydrogen peroxide at 570 nm as described by Sinha, (1971). Reduced glutathione (GSH) level was determined by measuring the rate of formation of chromphoric product in a reaction between DTNB (5,5´-dithiobis- (2-nitrbenzoic acid) and free sulphydryl groups at 412 nm (Beutler *et al*., 1963). Superoxide dismutase (SOD) activity was assayed using the method of Misra and Fridovich (1972), cholesterol was determined using Randox kit. The lipoproteins were assayed using enzymatic colorimetric method for very low density lipoprotein (VLDL) and low density lipoprotein (LDL) by precipitation using phosphotungistic acid and magnesium chloride. After centrifugation at 3000 rpm for 10 min at 25 °C, the clear supernatant contained high density lipoprotein (HDL) fraction using HDL-cholesterol precipitant kit. The LDL-cholesterol (LDL-c) was calculated using the formulae of Friedwald *et al*., (1972). Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were assayed using Randox kits.

Data was expressed as mean ± S.D of six determinations, except for the physicochemical properties which was done in triplicate and were analysed using one way analysis of variance (ANOVA) and complimented with student t-test. Values for p < 0.05 were considered to be statistically significant.

1. **Results**

Physiochemical properties of *Garcinia mangostana* seed oil is shown in table 1 and effect of *Garcinia mangostana* seed oil compounded feed compared to control (10 % vegetable oil) on the body and organ weights are shown in table 2. There was significant (p<0.05) increase in the body weight of animals fed with 3 % and 5 % *Garcinia mangostana* compared to 10 % *Garcinia mangostana* oil (group 2). 5 % *Garcinia mangostana* caused the lowest increase in body weight of 32 %, while animals on 3 % *Garcinia mangostana* had the highest gain in body weight of 38.3 %. There was a significant (P<0.05) difference in the weight of liver of rats on 3 % and 5 % *Garcinia mangostana* oil feed *when* compared with the control. However, in the brain, there was significant (P<0.05) decrease in weight of the animals fed 10 % GM compared to control, in 5 % GM, the brain was significantly increased compared to 10 % GM and incidentally rats on 3 % GM with highest percentage gain in weight had the least brain size.

Table 3 has data for results obtained for serum lipids in animals feed varying percentages of *Garcinia mangostana* seed oils for eight weeks. The result shows that rats fed with seed oil based formulated feed had their total cholesterol, HDL-c and triglyceride increased significantly (p<0.05) when compared with the control. 3 % and 5 % *Garcinia mangostana* rats had reduced total cholesterol levels compared to animals on 10 % GM. 10 % *Garcinia mangostana* and other animals on GM seed oil based feed had significantly elevated HDL-c levels compared to control. Serum HDL-c level was higher in animals fed with seed oil diet, especially in animals fed with *Garcinia mangostana* which had over 170 % increase when compared to control (10 % VO) rats. The level of LDL-c in the rats fed with *Garcinia mangostana* (10 %) based formulated feed decreased significantly (p<0.05) when compared with control. LDL-c levels decreased significantly (p<0.05) in the groups fed with 10 % and 3 % *Garcinia mangostana*.

Levels of antioxidants in the brain and liver homogenate of rats on *G. mangostana* seed oil formulated diet for eight weeks are shown on Tables 4 and 5. GM seed oil feed for eight weeks caused significant (p<0.05) increase in the activity of SOD in the liver and brain of rats on 10 % *G. mangostana* fed group compared to control, however 3 % *G. mangostana*, had slightly lowered SOD activity in both liver and the brain compared to control. *G. mangostana* seed oil feeding also caused a significant reduction in liver MDA relative to control but in the brain, 3 % and 5 % *G. mangostana* seed oil fed rats had significantly elevated MDA compared to control. GSH was not statistically significant in the test groups compared to control in both liver and brain. Liver and brain catalase activity in 10 % *G. mangostana* were significantly (p<0.05) decreased when compared with the control. 3 % and 5% *G. mangostana* had significantly (p<0.05) increased CAT activity in both brain and liver tissue when compared to 10 % *G. Mangostana* (group 2). Generally, seed oil fed groups had significantly decreased (p<0.05) catalase activity in both liver and brain tissue homogenate compared to control.

Table 6 has the data obtained for the activities of Aspartate amino-transferase (AST) and Alanine aminotransferase (ALT) in the serum and tissue protein concentration in the brain and liver of rats fed control diet and GM seed oil diet for eight weeks. In this study, there were no significant differences in protein concentrations in liver and brain of rats in test groups compared to control group. ALT decreased significantly (p<0.05) in GM oil fed rats when compared with control, there was over 100 % decrease in ALT in rats given 10% GM oil diet compared to the VO group. Similarly AST decreased in GM fed rats, there was a significant (p<0.05) decrease in 3 % and 5 % GM seed oil fed groups compared to both control and rats on 10% GM.

1. **Discussion**

Lipids are an essential component of a balanced diet. Fats and oils further serve as concentrated stores of calories and fat soluble vitamins such as vitamins A, D, E and K. We have saturated, and unsaturated lipids and they could be classified based on degree of unsaturation. The essential fatty acids are precursors for important hormones and prostaglandins. They have organoleptic properties and serve to make foods more palatable and are important in the maintenance of membrane fluidity and function. Chemical composition of fats and oils, processes involved in their purification, as well as chemical modification of the lipids are essential for health benefits, especially as some contain phytotoxins (Asif *et al*., 2010). Some seed oil contains toxic anti-nutritional factors thus the need for safety evaluation, purification, and effect on both growth and development becomes important.

Thermal stability and physicochemical analysis of *Garcinia mangostana* seed oil is on (Table 1) Smoke and flashpoints (174 and 215 °C) indicate that the seed oil could be used at relatively high temperatures as in deep frying. Solidification temperature is low and oil was in soft solid forms in the early hours of the morning. Age, stability on storage over time, quality and suitability for consumption can be determined from the acid value (Kardash and Tur’yan, 2005; Akubugwo, 2008). Low acid value (4.58±0.49) of the seed oils indicates reduced susceptibility to hydrolysis of oils and the acid values obtained for *Garcinia mangostana* seed oil is within the allowable limits for edible oils and compares favourably with common edible oils such as sesame, soybean, sunflower and rape oils (Eckey, 1954). Iodine values of oils, is a measure of unsaturation and it depicts reduced susceptibility to hydrolysis and oxidative spoilage on storage (Popoola and Yangomodou, 2006). The iodine value of 53.65±2.34 mg I2/100g oil, places *Garcinia mangostana* seed oil as non-drying type and the peroxide value of 3.27±0.07mEq / kg oil further supports the lowered tendency of the oil to go rancid and the values are within limits for commonly consumed oils such as palm, coconut, groundnut and soybean with 4.0; 10.0; 18.2 and 7.6 (mEq / kg oil) respectively (Oladiji *et al*., 2009). The saponification value (134.0±6.56) is low, thus *Garcinia mangostana* seed oil is not suitable for soap production but compares favourably with edible oils; with reduced tendency towards oxidative spoilage.

All test groups of animals in the study had increments in body weight in the eight week duration of the study. Body weight in all tested animals on *Garcinia mangostana* based seed oil diet was comparable to control on vegetable oil. The increase in weight rats on 3 % *Garcinia mangostana* was highest with 38 % maybe due to increased fed intake or water. The liver of *Garcinia mangostana* fed rats were bigger possibly due to greater increase in body weight, interestingly only in 5 % *Garcinia mangostana* fed rats was the brain significantly increased possibly due to a high proportion of fat in 5 % compounded feed compared to 10 % vegetable oil only. *Garcinia mangostana* based diet which caused both increase in body weight and visceral organs was probably more palatable and acceptable to the rats

Serum lipid profiles data are essential biomarkers for assessing health risk in obesity, diabetes and coronary heart diseases (CHD) (Cain, 2007). Diet induced alterations in serum lipids of animals could provide useful information on the effect of diet on lipid metabolism as well as predisposition of the heart to atherosclerosis and other CHD (Visavadiya and Narasimacharya, 2005; Abolaji *et al*., 2007). Saturated fatty acids and those with varying degree of unsaturation in the diet have been shown to affect both serum lipid profile and lipid peroxidation (Gilani *et al*., 2002). Rats on test oil had elevated cholesterol and triglyceride levels relative to control, the same animals had significantly increased HDL-c and subsequently lower artherogenic index (Table 3). Increase in the serum HDL-c of rats maintained on 10 % GM shows that the oil might be good for consumption. HDL-c is considered as good cholesterol, since it is an essential transporter of cholesterol from cells and arteries to the liver for catabolism (Lacko *et al*., 2000). Cardiovascular disease (CVD) patients are known to have markedly elevated levels of triglycerides and reduced HDL-c levels (Patsch, 1993). Furthermore, the triglyceride and LDL-c levels in the same group of rats when compared with the control further suggested that *Garcinia mangostana* oil offered protection against cardiovascular diseases especially in the 10 % GM feed rats which had 200 % decrease in artherogenic index value.

Peroxide value of *Garcinia mangostana* oil (Table 1), showed that the oils might be susceptible to oxidative damage and could have fewer unsaturated bonds, hence we evaluated the radical generation, radical scavenging and probable radical induced damaged potential that may be attribute to the seed oil intake in both the brain and liver tissues. The body antioxidant system plays an effective role in protecting the various biological tissues below a critical threshold of reactive oxygen species, thus preventing organ damage (Oschsendorf, 1999). *Garcinia mangostana* seed oil based formulated feed elicited significant reduction in GSH in the liver and the brain when compared with control. Reduction in GSH concentration in these organs in the animal fed *Garcinia mangostana* based formulated feed may be due to GSH dependent antioxidant enzymes challenging a prevailing oxidative stress under the influence of reactive oxygen species. Superoxide dismutase (SOD) an enzyme which generates H2O2, in the brain of animals with reduced GSH concentration, the activity of SOD was comparable to those rats maintained on vegetable oil only. All *Garcina mangostana* seed oil-based fed animals had increased SOD activities in the liver. This reflects that the *Garcinia mangostana* is not toxic to liver and brain and that the rats on GM must generate H202, in the brains through other mechanisms apart from via SOD. GM could be pro-oxidant since catalase activities were significantly increased in both liver and brain of the same set of animals when compared with the control while the activities in the same organs of animals maintained on 10 % *Garcinia mangostana* was comparable with control.

Alanine and aspartate aminotransferases (ALT and AST) occupy a central position in the metabolism of amino acids as they help in the retention of amino groups during the breakdown of amino acid and are involved in the biochemical regulation of amino acid pool. Hence, the aminotransferases provide necessary intermediate to predict possible toxicity in some organs such as liver cytolysis and the heart of animals as these compounds are only released into the blood stream after tissue injury (Rahman *et al*., 2001; Shahjahan *et al*., 2004). Measurement of the activities of these enzymes is of clinical and toxicological significance, since the enzymes will only be released into cellular flow when liver is damage. There was reduction in the activities of these enzymes in the serum of the animals maintained on GM formulated diets (Table 6). Hence, the effect of the GM compounded feed continual ingestion for eight weeks has insignificant toxicological effect on the liver.

Histological results of this study showed no lesions in liver of all the experimental animals. It could be concluded that the oil of *G. mangostana* seed has no deleterious effects on animal model. 5 % *G. mangostana* seed oil based feed was identified to have better effects in this experiment.

**Table 1: Physicochemical properties of *Garcinia mangostana* seed oils**

|  |  |
| --- | --- |
| **Parameter** | ***Garcinia mangostana*** |
| **Saponification value (mgKOH/100g)** | 134.0±6.56 |
| **Iodine value (mg I2/100g oil)** | 53.6±2.34 |
| **Peroxide value (mEq / kg oil)** | 3.27±0.07 |
| **Acid value (mg KOH/ g oil)** | 4.58±0.49 |
| **Specific gravity (g/ml)** | 0.98 |
| **Solidification point (°C)** | 18-21 |
| **Smoke point (°C)** | 174 |
| **Flash point (°C)** | 215 |

**Table 2**: **Effect of *Garcinia mangostana* seed oil based formulated feed on body weight and organ weight of rats for eight weeks**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Treatment Group** | **Initial weight(g)** | **Final weight (g)** | **% Weight gain** | **Liver (g)** | **Brain (g)** |
| **Group1**  **(10% VO)** | 123.33±1.66 | 193.33±6.66 | 36.20 | 5.693±0.046 | 2.15±0.712 |
| **Group 2**  **(10% GM)** | 126.66±1.66 | 195.0±12.58a | 35.0 | 5.630±0.076a | 2.11±0.272a |
| **Group 3**  **(5% GM)** | 143.33±3.33 | 210.0±0.00b | 32 | 6.37±0.56b | 2.20±0.240b |
| **Group 4**  **(3% GM)** | 123.33±1.66 | 200.0±0.00b | 38.3 | 6.69±0.248b | 2.04±0,014b |

Values shown are mean ±S.D. (n = 6). Mean differences are significant (*P* < 0.05) when compared with: a control group, b 10 % GM

**Table 3: Effect of *Garcinia mangostana* seed oil based formulated feed onserum lipid profile of experimental animals.**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Treatment Group** | **Total cholesterol**  **(U/L)** | **Triglyceride**  **(U/L)** | **HDL-c**  **(U/L)** | **LDL-c**  **(U/L)** | **Artherogenic index**  **LDL-c/HDL-c** |
| **Group1**  **(10 % VO)** | 84.1269±1.66 | 67.77±1.53 | 5.51±1.57 | 1.23±0.06 | 0.2244 |
| **Group 2**  **(10% GM)** | 99.85±3.68a | 84.71±1.29a | 14.17±0.45a | 1.05±0.14a | 0.07441a |
| **Group 3 (5 % GM)** | 85.7143±0.49b | 75.35±11.19 | 13.38±1.98 | 1.31±0.01 | 0.09807 |
| **Group 4**  **(3 % GM )** | 84.9528±0.38 | 74.89±1.44b | 9.18±0.26 | 0.97±0.04 | 0.106367 |

Values shown are mean ±S.D. (n = 6). Mean differences are significant (*P* < 0.05) when compared with: a control group, b 10 % GM

**Table 4: Effect of *Garciniamangostana* seed oil based formulated feed onSuperoxide dismutase, catalase activities, glutathione content and lipid peroxidation in the liver**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Treatment Group** | **Liver SOD (µg/mg protein)** | **Liver CAT (µg/mg protein)** | **Liver GSH (µg/mg protein)** | **Liver MDA (µg/mg protein)** |
| **Group 1**  **(10 % VO)** | 0.46 ± 0.023 | 279.31±4.04 | 118.33±0.60 | 2.20±0.14 |
| **Group 2**  **(10 % GM)** | 0.66 ±0.014a | 209.67±2.33a | 119.33±1.30 | 1.62±0.02a |
| **Group 3**  **(5 % GM)** | 0.84 ±0.46b | 322.10±10.61b | 115.66±0.72 | 1.40±0.096b |
| **Group 4**  **(3 % GM)** | 0.49 ±0.011b | 286.81±4.58b | 119.16±1.16 | 1.43±0.064b |

Values shown are mean ±S.D. (n = 6). Mean differences are significant (*P* < 0.05) when compared with: a control group, b 10 % GM

**Table 5: Effect of *Garcinia mangostana* seed oil based formulated feed on Superoxidedismutase, catalase activities, glutathione content and lipid peroxidation in the brain**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Treatment group** | **Brain SOD (µg/mg protein)** | **Brain CAT (µg/mg protein)** | **Brain GSH (µg/mg protein)** | **Brain MDA (µg/mg protein)** |
| **Group 1**  **(10 % VO)** | 1.14±0.44 | 189.27±25.56 | 119.83±0.833 | 0.56±0.014 |
| **Group 2**  **(10 % GM)** | 1.27±0.23a | 126.08±10.67a | 115.85±1.45 | 0.54±0.122a |
| **Group 3**  **(5 % GM)** | 1.32±0.65b | 214.33±11.45b | 112.83±0.88 | 1.26±0.133b |
| **Group 4**  **(3 % GM)** | 1.12±0.19 | 223.10±4.75b | 118.16±1.01 | 1.13±0.043b |

Values shown are mean ±S.D. (n = 6). Mean differences are significant (*P* < 0.05) when compared with: a control group, b 10 % GM

**Table 6: Effect of *Garcinia mangostana* seed oil based formulated feed on serum ALT, AST, liver and brain protein**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Treatment group** | **ALT(U/L)** | **AST (U/L)** | **Liver protein**  **(µg/ml)** | **Brain protein**  **(µg/ml)** |
| **Group 1**  **(10 % VO)** | 13.39±2.165 | 84.05±1.04 | 2.12±0.005 | 0.105±0.005 |
| **Group 2**  **(10 % GM)** | 4.52±0.117a | 82.02±0.38a | 2.32±0.158 | 0.115±0.0046 |
| **Group 3**  **(5 % GM)** | 5.17±0.103b | 72.43±1.18b | 2.31±0.152 | 0.123±0.022 |
| **Group 4**  **(3 % GM)** | 2.85±0.103b | 69.33±1.88b | 1.57±0.046 | 0.142±0.015 |

Values shown are mean ±S.D. (n = 6). Mean differences are significant (*P* < 0.05) when compared with: a control group, b 10 % GM

**Histopathology Results**

Photomicrographs Of Haematoxylin And Eosin Stained Liver Section.

 

**A**

**B**

 

**C**

**D**

(A) Group 1(control rats receiving 10 % VO diet): No visible lesion seen

(B) Group 2 (rats receiving GM 10 % diet): No visible lesions seen

(C) Group 3(rats receiving GM 5 % diet): No visible lesion seen

(D.) Group 4 (rats receiving GM 3 % diet): No visible lesion seen

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**References**

1. Odeomelam SA. Proximate composition and selected physicochemical properties of seeds of African Oil Beans (*Pentaclethramacrophylla*) Pak. J. Nutr 2005; 4:382-383.
2. Eromosele IC, EromoseleCO, Kuzhkuha DM. Evaluation of mineral elements andascorbic acid contents in fruits of some wild plants. Plant Foods Hum. Nutr 1991; 41: 151-154.
3. Glew RS, Dorothy J, Chang LT, Huang YS, Millson M., Glew RH. Nutrient content of four edible wild plants from West Africa. Plant Foods Hum. Nutr 2005; 60:187-193.
4. Harborne JB, Baxter H. 1993. Phytochemical Dictionary. Taylor & Francis, London. 791pp.
5. [Obolskiy D](http://www.ncbi.nlm.nih.gov/pubmed?term=Obolskiy%20D%5BAuthor%5D&cauthor=true&cauthor_uid=19172667), [Pischel I](http://www.ncbi.nlm.nih.gov/pubmed?term=Pischel%20I%5BAuthor%5D&cauthor=true&cauthor_uid=19172667), [Siriwatanametanon N](http://www.ncbi.nlm.nih.gov/pubmed?term=Siriwatanametanon%20N%5BAuthor%5D&cauthor=true&cauthor_uid=19172667), [Heinrich M](http://www.ncbi.nlm.nih.gov/pubmed?term=Heinrich%20M%5BAuthor%5D&cauthor=true&cauthor_uid=19172667). *Garciniamangostana* L.: a phytochemical and pharmacological review. P[hytother Res.](http://www.ncbi.nlm.nih.gov/pubmed/19172667) 2009; 3(8):1047-65.
6. [Pedraza-Chaverri J](http://www.ncbi.nlm.nih.gov/pubmed?term=Pedraza-Chaverri%20J%5BAuthor%5D&cauthor=true&cauthor_uid=18725264), [Cárdenas-Rodríguez N](http://www.ncbi.nlm.nih.gov/pubmed?term=C%C3%A1rdenas-Rodr%C3%ADguez%20N%5BAuthor%5D&cauthor=true&cauthor_uid=18725264), [Orozco-Ibarra M](http://www.ncbi.nlm.nih.gov/pubmed?term=Orozco-Ibarra%20M%5BAuthor%5D&cauthor=true&cauthor_uid=18725264), [Pérez-Rojas JM](http://www.ncbi.nlm.nih.gov/pubmed?term=P%C3%A9rez-Rojas%20JM%5BAuthor%5D&cauthor=true&cauthor_uid=18725264). Medicinal properties of mangosteen (*Garciniamangostana*). Food Chemical Toxicol. 2008 46(10):3227-3239.
7. [Jung HA](http://www.ncbi.nlm.nih.gov/pubmed?term=Jung%20HA%5BAuthor%5D&cauthor=true&cauthor_uid=16536578), [Su BN](http://www.ncbi.nlm.nih.gov/pubmed?term=Su%20BN%5BAuthor%5D&cauthor=true&cauthor_uid=16536578), [Keller WJ](http://www.ncbi.nlm.nih.gov/pubmed?term=Keller%20WJ%5BAuthor%5D&cauthor=true&cauthor_uid=16536578), [Mehta RG](http://www.ncbi.nlm.nih.gov/pubmed?term=Mehta%20RG%5BAuthor%5D&cauthor=true&cauthor_uid=16536578), [Kinghorn AD](http://www.ncbi.nlm.nih.gov/pubmed?term=Kinghorn%20AD%5BAuthor%5D&cauthor=true&cauthor_uid=16536578). Antioxidant xanthones from the pericarp of *Garciniamangostana* (Mangosteen). [J Agric Food Chem.](http://www.ncbi.nlm.nih.gov/pubmed/16536578) 2006; 54(6):2077-2082.
8. [Zhang Y](http://www.ncbi.nlm.nih.gov/pubmed?term=Zhang%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=20176087), [Song Z](http://www.ncbi.nlm.nih.gov/pubmed?term=Song%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=20176087), [Hao J](http://www.ncbi.nlm.nih.gov/pubmed?term=Hao%20J%5BAuthor%5D&cauthor=true&cauthor_uid=20176087), [Qiu S](http://www.ncbi.nlm.nih.gov/pubmed?term=Qiu%20S%5BAuthor%5D&cauthor=true&cauthor_uid=20176087), [Xu Z](http://www.ncbi.nlm.nih.gov/pubmed?term=Xu%20Z%5BAuthor%5D&cauthor=true&cauthor_uid=20176087). Two new prenylatedxanthones and a new prenylatedtetrahydroxanthone from the pericarp of *Garciniamangostana*. [Fitoterapia](http://www.ncbi.nlm.nih.gov/pubmed/20176087) 2010;81(6):595-599.
9. Williams P, Ongsakul M, Proudfoot J, Croft K, Beilin L. Mangostin inhibits the oxidative modification of human low density lipoprotein. *Free Radic Res*. 1995;23(2):175–184.
10. Sargowo D, Setiawan M. Effect of extract from pericarp of mangosteen as antioxidant rats models of atherosclerosis. JurnalKardiologi 2012;33:71.
11. Adiputro DL, Khotimah H, Widodo MA, Romdoni R, Sargowo D. Effects of ethanolic extracts of *Garciniamangostana* fruit pericarp on oxidant-antioxidant status and foam cells in atherosclerotic rats. OxidAntioxid Med Sci. 2013;2:61–64.
12. A.O.A.C. (1990) A.O.A.C., Official methods of analysis (15thed) Association of Official analytical chemists. Washington D.C U.S.A.
13. Oladiji AT, Abodunrin TP,Yakubu MT. Toxicological evaluation of *Tetracarpediumconophorum* nut-based diet in rats. Food and Chem Tox. 2010; (48):898-902.
14. Gornall AC, Bardawill EJ, David MM. Determination of serum proteins by means of Biuret reaction. J. Biol. Chem. 1949; 177: 364-365.
15. Sinha KA. Colorimetric Assay of Catalase. Anal Biochem1971; 47: 389 – 394.
16. Beutler E, Duron O, Kelly BM. Improved method for the determination of blood glutathione. Lab. Clin. Med*.* 1963; 61: 882-888.
17. Misra HP, Fridovich I. The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. J. Biol Chem 1972; 24: 3170-3175.
18. Friedewald WT, Levy R.I, Fredrickson DS. Estimation of the concentration of Low- density lipoprotein cholesterol in plasma without use of the preparative ultra-centrifuge. Clin. Chem. 1972; 18: 499– 502.
19. Asif M. A review on purification and chemical modification of oils and fats T. Ph. Res 2010; 4:16-27.
20. Kardash E, Tur’yan YI. Acid value determination in vegetable oils by indirect titration in aqueous alcohol media. Croatia Chem Acta 2005;78:99-103.
21. Akubugwo IE, Chinyere GC, Ugbogu, AE. Comparative studies on oils from some common plant seeds in Nigeria. Pak J Nutr 2008; 7:570-573.
22. Eckey EW. Vegetable fats and oils. American Chemical Society Monograph (1951) Series No 123. Reinhold Publishing Corporation, New York.
23. Popoola TOS, Yangomodou OD. Extraction, properties and utilization potentails of cassava seed oil. Biotechnology 2006; 5:38-41.
24. Oladiji AT, Shoremekun Kl, Yakubu MT. Physicochemical properties of the oil from *Blighiasapida* and toxicological evaluation of the oil-based diet in rat J. Med Food 2009; 12(5): 1-9.
25. Visavadiya NP, Narasimacharya AV. Hypolipidemic and antioxidant activities of *Asparagus racemosus* in hypercholesterolamic rats. Indian J. Pharmacol. 2005; 37 (6): 376–380.
26. Abolaji AO, Adebayo AH, OdesanmiOS. Effect of ethalonic extract of *Parinaripolyandra* (Rosaceae) on serum lipid profile and some electrolytes in pregnant rabbits. Res. J. Med. Plants 2007; 1 (4), 121-127.
27. Gilani GS, Ratnayaka WMN, Brooks SPJ, Botting HG, Plouffe LJ, Lampi BJ. Effects of dietary protein and fat on cholesterol and fat metabolism in rats. Nutr. Res. 2002; 22.
28. Lacko AG, Barter P, Ehnholm C, Van Tol A. An international symposium on basic aspects of HDL metabolism and disease prevention. J. Lipid Res. 2000; 41: 1695–1699.
29. Patsch JR. 1993. Plasma triglycerides and high-density lipoprotein. In: Catapano, A.I., Bernini, F., Corsini, A. (Eds.), High-density Lipoprotein: Physiopathology and Clinical Relevance. Raven Press Ltd., New York, 1993; page 139.
30. Ochsendorf FR. Infections in the male genital tract and reactive oxygen species. Human Reprod. Update 1999; 5: 399-420.
31. Rahman MF, Siddiqui MK, Jamil K. Effects of vepacide (*Azadirachtaindica*) on aspartate and alanine aminotransferase profiles in sub-chronic study with rats. Hum. Exp. Toxicol. 2001; 20: 243–249.
32. Shahjahan M, Sabitha KE, Jamu M, Shyamala-Devi CS. 2004. Effect of *Solanumtrilobatum* against carbon tetrachloride induced hepatic damage in albino rats. Indian J. Med. Res. 2004; 120: 194–198.

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