



## Pelagia Research Library

European Journal of Experimental Biology, 2012, 2 (2):404-409



# The effect of chronic ingestion of crude garcinia kola on the histology of the liver

<sup>1</sup>Charity U Osifo, <sup>1,3</sup>Uwaifoh Akpamu, <sup>2</sup>Charlse I Idehen, <sup>1</sup>Williams A Adisa and <sup>4</sup>Kerry E Azeke

<sup>1</sup>Department of Physiology, Faculty of Basic Medical Sciences; Ambrose Alli University, Ekpoma, Edo State, Nigeria

<sup>2</sup>Histopathology, Faculty of Clinical Sciences, College of Medicine, Ambrose Alli University, Ekpoma, Edo State, Nigeria

<sup>3</sup>Anthonio Research Center, Ekpoma, Edo State, Nigeria

<sup>4</sup>Accident and Emergency Unit, Irrua Specialist Teaching Hospital, Irrua- Edo, Nigeria

---

## ABSTRACT

Different quantities of *Garcinia kola* in form of determined doses in mg/kg body weight was given to different groups of rabbits daily for a period of six weeks, to assess possible adverse effects of chronic administration on the histology of the liver. 1200mg/kg, 1500mg/kg and 1800mg/kg of the reconstituted powdered *Garcinia kola* were administered to the rabbits in the test groups B, C and D respectively while A (control) received normal saline. Slide photomicrographs of the control (group A) shows normal histological hepatocytes. Although photomicrographs of the tests groups (B, C and D) presented mild cellular edema, this was not of any pathological significant because it was also observed in the control (group A). The elicitation of no observable histo-pathological effects by *Garcinia kola* on the histology of the liver is a reflection of its hepatic safety in healthy condition. However, it toxic dose requires investigation and thus the needs for further studies.

**Key words:** Medicinal plants, *Garcinia kola*, Histology, Liver, Male, Rabbits.

---

## INTRODUCTION

With the shifting of attention from synthetic drugs to natural plant products, plants and plant extracts which are known to provide a source of inspiration for novel drug compounds are now been used for enhancing organs and body systems performance in man and animals [1, 2]. In recent times, researchers have been motivated to consider the effects of a number of medicinal plants that are believed to possess therapeutic properties for a number of body tissues, organs and systems. One of such plants that has gained much attention is *Garcinia Kola* (*G. Kola*), commonly known as bitter kola or false kola and by several ethnic communities in Nigeria as- 'Adu' in Esan, 'Miji-goro' in Hausa, "Akilu" or "Ugolo" by the Igbos, "Orogbo" in Yoruba. According to Uko *et al.* [3], *G. Kola* is

referred to as “male kola” because of its perceived aphrodisiac activity. Scientifically, the potentials of *G. kola* as a therapeutic agent have been reported and these include anti-inflammatory, anti-microbial, anti-diabetic, anti-parasitic, antiviral properties [4, 5, 6, 7, 8, 9, 10], antiulcer properties [11], hepato-protective [6, 12, 13, 14], antithrombotic [15], hypoglycaemic (anti-diabetic), broncho-dilatation, antispasmodic effect on smooth muscle [1, 16, 17], poison antidote, treatment of diarrhea, hepatitis, asthma, common cold, cough, dysmenorrhoea, anti-anemia [18, 19, 20] and anti-obesity [21]. Despite these potential beneficial therapeutic effects of *G. kola*, there have been some scientific reports of its adverse effects too [5, 22, 23].

Fernandez-Checa and Kaplowitz [24] reported that every drug is associated with hepatotoxicity almost certainly due to the ability to generate free radicals and disturbance in hepatocyte biochemistry. In addition, the liver plays a role in regulating various physiological processes in the body among which is the metabolism of substances ingested by human [25, 26, 36]. In relation to these facts, it becomes rationale to hypothesize that the liver might be subjected to a variety of diseases and disorders from excessive consumption of *G. Kola*. Reason due to the mass-consumption rate considering the benefits attached. This study was designed to investigate the effects of chronic administration of *G. kola* seed on the histology of the liver of rabbits.

## MATERIALS AND METHODS

**Plant of study:** The seeds of *G. kola* were obtained from a local market in Ireukpen, Ekpoma, Edo State, Nigeria. The coat of the seeds were removed and subsequently cut into pieces to increase its surface area for drying which was carried out under the hot sun. Grinding of the dried pieces into fine powder followed this procedure and finally the resultant *G. kola* powder was measured using Electric Balance (Denver Company USA -200398. 1REV.CXP-3000). The measurement was done separately, each weighed sample being packed in a drug envelope and stored in a dry glass bottle to keep it dry.

**Animals:** Twenty-four Male adult rabbits of comparable weight purchased from Aduwawa cattle market, Benin City, were used for the study. The rabbits were randomly divided into four groups, namely, group A (control; n = 6), group B, C and D (test groups; n =6 each). They were housed in separately labeled wooden cages and allowed acclimatization for a period of 10 days. During this period, they had *ad libitum* access to water and standard laboratory animal feed from Bendel Feed and Flour Mill, Ewu, Edo State, Nigeria. The cages were swept clean every morning and the animal’s feet and head examined regularly for evidences of infection, such as sore feet, sore mouth and discharges from their eyes and nose.

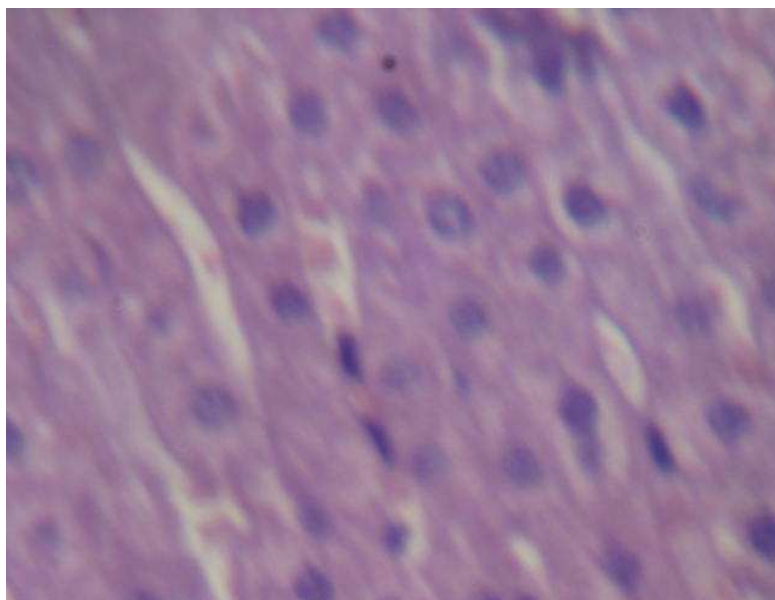
**Treatment with plant material:** The difference in feed composition between the control and test animals was that the later was supplemented with the test material (dried and milled seed of *Garcinia kola*). The weighted packed sample of *G.Kola* which was aliquots was reconstituted with distilled water to obtain suspensions of appropriate concentrations for oral administration. Animals in all groups, except the control which received normal saline, received by gavage graded concentrations of *G.kola* powder (suspended in distilled water) daily for Forty-two days. Three doses; 1200mg/kg B.W, 1500mg/kg B.W and 1800mg/kg, of the reconstituted powdered *G.kola* were administered to the test groups B, C and D respectively. These values are chosen based on comparable information from previous work [27].

**Histological studies:** At termination of the experiment, all the rabbits fasted for 12 hours and the livers were dissected out and immediately fixed in 10% formal saline. Using standard tissue processor, the tissues were then dehydrated in ascending grades of alcohol: 70, 95% and absolute alcohol in 2 changes each. After which clearing was done with xylene/ absolute alcohol (50:50 v/v ratio). This was followed by infiltration in molten paraffin wax at 60°C in 2 changes. They were further processed for staining with haematoxyline and eosin (H&E) as described by John et al. [28]. Photomicrographs of the slides (x 40) were taken for histological examination and the slides in tests were compared to that of the control for histological variations.

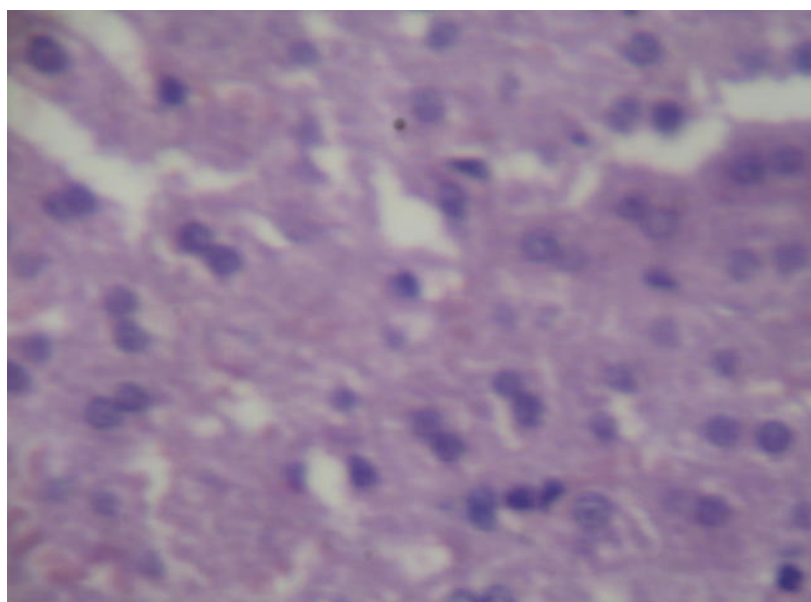
## RESULTS

Figure 1 shows a section of the liver of rabbit in control group (not fed G. Kola). Normal hepatocytes were observed in the livers of rabbits in this group. Figure 2, 3 and 4 shows the liver section from rabbits that received varies doses

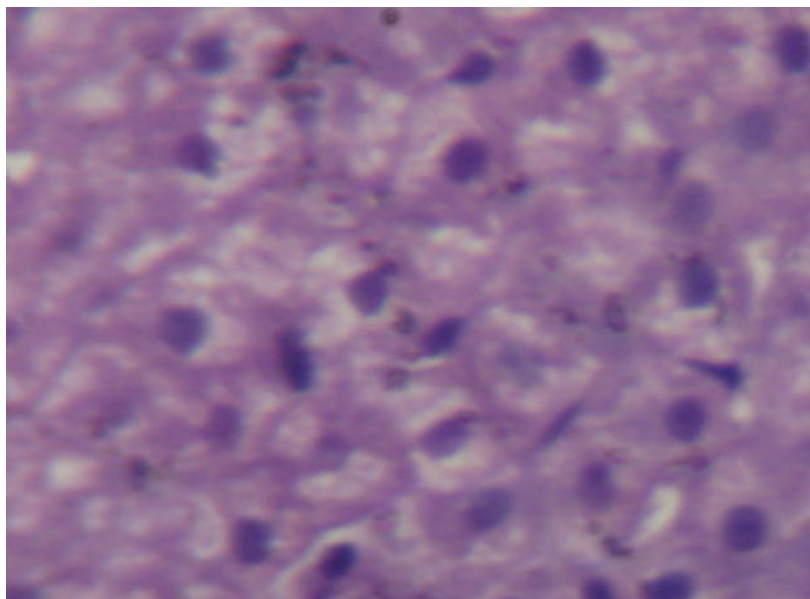
of G.kola each from group B, C and D respectively. There were also normal hepatocytes and cell cords. Histological observations show no evidence of cellular damage in the treatment groups treated with varies doses of G. Kola compared to the control. However, mild cellular edema was observed in test groups and the control group which received normal saline.



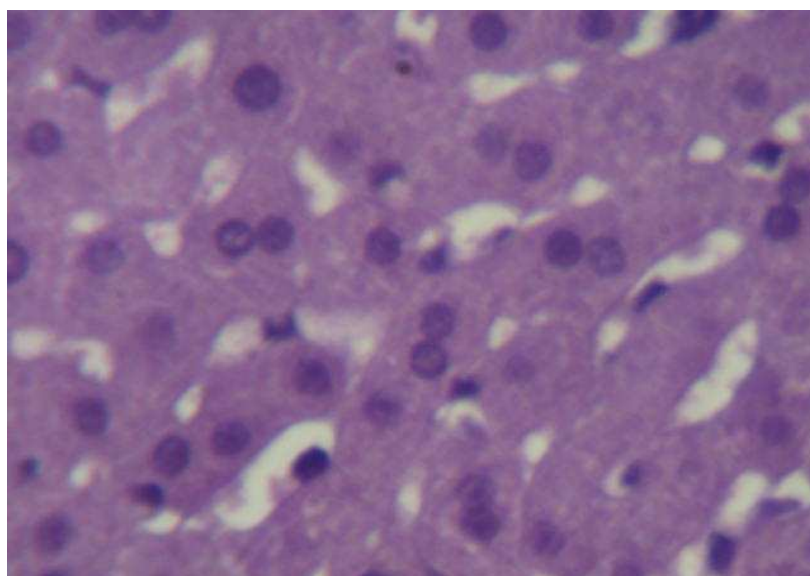
**Figure 1.** Section from the liver of rabbit which received water only and no G. Kola (control, group A). (H&E x40).  
*It shows normal hepatocytes and mild cellular edema.*



**Figure 2.** Section from the liver of rabbit which received 1200mg/bw of crude G. Kola (test group B). (H&E x40).  
*It shows normal hepatocytes and mild cellular edema.*



**Figure 3.** Section from the liver of rabbit which received 1500mg/bw of crude *G. Kola* (test group C). (H&E x40).  
*It shows normal hepatocytes and mild cellular edema.*



**Figure 4.** Section from the liver of rabbit which received 1800mg/bw of crude *G. Kola* (test group D). (H&E x40).  
*It shows normal hepatocytes and mild cellular edema.*

#### DISCUSSION

It can be seen from the tissue micrographs that the histology of the liver of rabbits fed varies doses of *G. Kola* for six weeks was not affected. The absent of evidence of cellular damage in the present study agrees with the study of Uko et al. [3]. However, earlier findings have reported contradictory observations [5, 22, 23, 29]. Specifically, Virk and Menke [29] reported prolonged ingestion of *G. kola* to cause degenerative changes in the liver while Bradie and Gill [5] reported numerous intracytoplasmic vacuoles in hepatocytes and others reporting adverse effects with *G. kola* ingestion [22, 23]. In a recent study, the liver of dogs fed ethanolic extract of *Garcinia kola* (500mg/Kg and

1000mg/Kg) for six weeks, showed severe widespread vacuolar degeneration of hepatocytes, multifocal centrilobular hepatocellular necrosis, mononuclear cell aggregations, cellular infiltration including neutrophils, lymphocytes and macrophages, mild periportal fibrosis and Kupffer cell proliferation [30]. Interestingly, the study of Uko *et al.*, [3] reported no microscopic histological alteration in the liver of rats fed *G. Kola* for 70 days even when there was non-significant reduction in the mass of the liver. He then concluded that the difference in histological differences from other studies may possibly be due to non-challenge of the liver of the rats with high doses of *G. Kola*. However, in the present study where far higher dosage was applied there was no pathological alteration in the cyto-architectures of the liver. Although photomicrographs of the test groups (B, C and D) presented mild cellular edema, this was not of any pathological significance because it was also observed in the control (group A). The differences of this study from other studies where alteration in hepatocytes have been reported, are the non-challenge of the animal under study with hepatotoxic substances prior to administration of *G. kola* and the use of the whole *G. Kola* seeds as against other studies where its extracts (ethanolic or methanolic) are used. The dissimilarities in histological observation in this study from other studies may possibly be due to non-challenge of the liver with hepatotoxins prior to the administration of *G. Kola* and or the use of whole seeds as against extracts. Previous studies have shown promising results of *G. Kola* in the evaluation of anti-hepatic drugs for the treatment of hepatotoxicity, induced in laboratory animals in various experimental models [6, 13, 31, 32, 33]. In addition, researchers have reported its antioxidant and scavenging properties *in vitro* and *in vivo* [34, 35]. The absence of cellular damage in the liver with chronic ingestion of *G. Kola*, may therefore be owed to its antioxidants and scavenging potentials.

Conclusively, the elicitation of non-pathological significant effects of crude ingestion of *G. Kola* on the architecture of the liver hepatocytes is a reflection of its normal hepatic metabolism and safety and may be owed to its antioxidant and scavenging properties. However, more specific studies to ascertain the effect of *G. Kola* on the liver and other organs of the body cannot be overemphasized. In addition, its toxic dose requires investigation and thus the need for further studies.

#### Acknowledgments

The authors are grateful to Dr. A.O. Nwaopara of the Department of Anatomy, College of Medicine, Ambrose Alli University, for his technical assistance. We also recognize our individual just efforts toward the completion of the research work.

#### REFERENCES

- [1] MM Iwu, AR Duncan, CO Okunji. In: J. Janick (ed). Perspectives on new crops and new uses. Alexandria, ASHS Press, **1999**. Pp.457-62.
- [2] AA Dada, M Ikuerowo. *African Journal of Agricultural Research*. **2009**; 4 (4); 344 - 347.
- [3] OJ Uko, A Usman, AM Ataja. *Vet. Arhiv*. **2001**; 71, 287-297.
- [4] MM Iwu. In: Cody V, Middleton E, Harbone JB (ed). Plant Flavonoids in Biology and Medicine. New York: Alan R. Liss. **1986**; 485 – 8.
- [5] VB Braide, V Grill. *Gegenbaurs Morphol. Jahrb*. **1989**; 136 (1): 95-101.
- [6] A Akintonwa, AR Essien. *J. Ethnopharmacol*. **1990**; 29 (2): 207-211.
- [7] NN Orie, EU Ekon. *East Afri. Med J*. **1993**; 70:143 – 145.
- [8] K Matsumoto, Y Akao, E Kobayashi, T Ito, K Ohguchi, T Tanaka, M Iinuma, Y Nozawa. *Biol Pharm Bull*.**2003**; 26:569–71.
- [9] GO Ezeifeke, MU Orji, TI Mbata, AO Patrick. *Biotechnology*,**2004**; 3(1):41-43.
- [10] OS Adedeji, GO Farimu, SA Amen, JB Olayemi. *J. Anim. Vet. Adv*. **2006**; 5(3): 184 – 187.
- [11] GF Ibronke, SB Oldeye, O Balogun, A Aremu. *Phytotherapy research*.**1997**; 11(4): 312-313.
- [12] MM Iwu, OA Igboko, UA Onwuchekwa, CO Okunji. *J Ethnopharmacol*.**1987**; 21:127–38.
- [13] OE Farmobi, JG Tahntering, AO Agbooola, JO Nwankwo, GO Emerole. *Food Chem. Toxicol*.**2000**; 38: 535-541.
- [14] JO Nwankwo, JG Tahnteng, GO Emerole. *Eur. J. Cancer Prev*. **2000**; 9: 35-61.
- [15] OA Olajide. *Phytother. Res*. **1999**;13 (3): 231 - 2.
- [16] MM Iwu. *Experimentia*;**1985**; 41, 699 -700.
- [17] VB Braide. *Fitoterapia LX*: **1989**; 123- 129.



- 
- [18] EH Holmes. *Liberia Pharm. J.* **2001**; 8: 1877-1878.
- [19] WH Lewis. *Plants Effecting Mainsheath*. New York: John Wiley – Int. Pub. **1977**; 231 –232.
- [20] JM Dalziel. *Useful plants of tropical Africa* London. *Crown Agents*, **1956**; 10: 612-617.
- [21] UC Osifo, U Akpamu, HO Otamere, CN Ekhaton. *Archives of Applied Science Research*, **2011**, 3 (5):526-531.
- [22] SE Atawodi, P Mende, B Phndsteiq, R Preussmann, B Spiegelhalder. *Food chemical toxicology*.**1995**; 33: 8, 625-630.
- [23] AK Akinloye, OO Igbarha, MO Olaniyi, OO Alaka, BO Oke. *Trop. Vet.* **2000**, 18:49-54.
- [24] JC Fernandez-Checa, N Kaplowitz. *Toxicol. Applied Pharm.***2005**; 204: 263 – 273.
- [25] K Sembulingam, P Sembulligan. *Essentials of Medical Physiology*. 4<sup>th</sup> Ed. Jaypee brothers Medical Publishers Ltd. **2006**.
- [26] AC Guyton, JE Hall. *Text Book of Medical Physiology*. W.B sanders company Philadelphia. Pp. 861-864.
- [27] AA Ahumibe, BV Braide. *Nigerian Journal of Physiological Sciences*. **2009**; 24 (1): 47-52.
- [28] DM John, S Alan, RT David. *Theory and practice of histological technique*. Churchill Livingstone, London, 3rd Ed. **1990**; Pp .112.
- [29] AS Virk, KH Menke. *Anim. Res. Dev.***1986**; 24:7-22.
- [30] HO Nottidge, TO Omobowale, VO Taiwo, MA Omotoso. *Int. J. Morphol.* **2008**; 26(4):1067-1072.
- [31] MM Iwu, OA Igboko, CO Okunji, MS Tempesta. *J. Pharm. Pharmacol.***1990**; 42: 290-292.
- [32] EO Farombi. *Pharmacol Res*, **2000**; 42. 75.
- [33] EO Farombi, BF Adepoju, OE Ola-Davies, GO Emerole. *Mutat Res*, **2001**; 483. S106.
- [34] EO Farombi, OO Akanni, GO Emerole. *Pharm Biol*, **2002a**; 40. 107.
- [35] EO Farombi, TO Akuru, MC Alabi. *Pharmacol Res*,**2002b**; 45. 63.
- [36] KC Patrick-Iwuanyanwu, MO Wegwu, T Maknmoor. *EJEB*. **2011**; 1(1): 128-138.