RESEARCH PAPER

Acute Toxicity Assessment Of Hydroalcoholic Fruit Extract Of Garcinia Lanceifolia Roxb. In Wistar Rats

Jay Kumar[1], Nisha Sharma[1], Kalpana[1], Pratima Katiyar[1], Ajay Kumar Singh[1], Chaynika Kala[2], Ashish Kar[3], Prakash Chandra Gupta[*1]

[1]*Schoolof Pharmaceutical Sciences, Chhatrapati Shahu Ji Maharaj University Kanpur, India 208024*
[2]*Pathology Department, Ganesh Shankar Vidyarthi Memorial Medical College, Kanpur, India 208002*
[3]*The Energy and Resources Institute (TERI), Northeastern Regional Centre, Guwahati-781036 Assam, India*

ABSTRACT

Objectives: Garcinia lanceifolia Roxb. Var. Oxyphylla (Clusiaceae), an important ethnomedicinal plant, widely used as medicine for jaundice, diarrhea, gastrointestinal and urinary problems. The aim of the study was to evaluate the acute oral toxicity and safety profile of the hydroalcoholic fruit extract of Garcinia lanceifolia Roxb. in Wistar rats.

Methods: Garcinia lanceifolia fruit extract (GLE) was administered orally to rats in graded doses up to 2000 mg/kg body weight. The animals were observed for 14 days to assess clinical signs of toxicity, behavioral changes, morbidity, and mortality. Body weight, food intake, and water consumption were recorded throughout the study. At the end of the experimental period, hematological and biochemical parameters were analyzed, and vital organs were examined histopathologically to assess any systemic toxicity.

Results: No mortality or significant behavioral abnormalities were observed at any of the tested doses. Body weight gain, food consumption, and water intake were comparable to the control group. Hematological values remained within normal physiological ranges, indicating no adverse effects on blood components. Biochemical analysis showed only significant fluctuations in ALT and cholesterol levels at the highest dose (2000 mg/kg), but all the biochemical parameters remained within reference limits. Histopsathological evaluation of the liver, kidney, and spleen revealed preserved organ architecture with only mild, non-specific changes.

Conclusion: The hydroalcoholic fruit extract of Garcinia lanceifolia demonstrated no significant acute toxicity in rats up to the dose of 2000 mg/kg. The findings indicate a wide margin of safety and support the safe oral use of the fruit extract, providing a basis for further pharmacological studies.

Keywords: Garcinia lanceifolia , acute toxicity, histopathology

How to cite this article: Kumar J, Sharma N, Kalpana, Katiyar P, Singh AK, Kala C, Kar A, Gupta PC, Acute Toxicity Assessment Of Hydroalcoholic Fruit Extract Of Garcinia Lanceifolia Roxb. In Wistar Rats..Int J Drug Deliv Technol. 2026;16 (2s): 673-679; DOI: 10.25258/ijddt.16. 673-679

Source of support:. None

Conflict of interest: None

INTRODUCTION

The genus *Garcinia* (Clusiaceae) comprises of more than 200 species widely distributed in tropical Asia, Africa and the Americas and is a rich source of bioactive secondary metabolites like xanthones, benzophenones, garcinol, and organic acids such as hydroxycitric acid [1]. Herbal medicines derived from fruits of the genus Garcinia are increasingly employed worldwide for their diverse pharmacological activities, including hepatoprotective, anti-inflammatory, antiulcer, and metabolic regulatory effects. These pharmacological activities have driven renewed interest in *Garcinia* species as sources of nutraceuticals and lead compounds for drug development [2,3]. *Garcinia lanceifolia Roxb. Var. Oxyphylla* is an important medicinal plant of northeastern India and commonly known as "Rupahi-thekera" (Assamese). Various ethnic communities of northeast India use the fruit of plant for various diseases and disorders like stomache, dysentery, dyspepsia and gastrointestinal and urinary problem. Polyphenol rich *G. lanceifolia* fruit with various mineral possess potent antioxoidant, antiobesity and hypolipidemic properties. Fruits are also consumed as a traditional food and pickle ingredient in several communities [4-6]. Given the growing interest in utilizing *Garcinia* fruits as potential nutraceuticals or therapeutic adjuncts, it is essential to establish their safety profile particularly acute toxicity before further development [7]. Acute oral toxicity studies provide first-line information on hazard potential, approximate lethal-dose ranges, and early indicators of target-organ toxicity. Although some toxicological investigation has been conducted on stem and bark of *G. lanceifolia, but* comprehensive acute toxicity data specifically for fruits which may differ chemically from leaves or bark and more likely to be used in food and nutraceutical application remain limited. It is necessary to evaluate the acute oral safety of fruit extract in experimental animals because fruit phytoconstituents (such as organic acids, xanthones, and garcinol isomers) can affect both pharmacological activity and toxicity [8,9].

Therefore, the present study was undertaken to generate guideline-concordant acute oral toxicity data of *G. lanceifolia fruit*. Using OECD guideline, we evaluated the single-dose oral safety of fruit extract by monitoring mortality, clinical signs, body-weight changes, gross pathology and organ weights data that will establish an initial safety profile, inform dose selection for repeated-dose studies, and support any future preclinical development of *G. lanceifolia* fruit derived preparations.

**Author for Correspondence: herbalprakash@yahoo.com*

MATERIALS AND METHODS

Collection and identification of plant material

Fresh fruits of *G. lanceifolia* were collected from Ashrang village, Dimahasao district, Assam, India. The plant specimen was identified, authenticated by Taxonomist, Central Ayurveda Research Institute, Guwahati, India and voucher specimen was deposited in departmental herbarium section for future reference.

Preparation of extract

The collected fresh fruits of *G. lanceifolia* were cut into small pieces. The pulp of the fruits was air-dried under the controlled conditions and ground to the powder before extraction.

Powdered air-dried material (350 g) was macerated in 70% ethyl alcohol (3 L, four times) followed by filtration done twice through a muslin cloth and filter paper simultaneously. The procedure was repeated and all the filtrates were combined. The pooled filtrate was evaporated at 40°C under low pressure in a rotavapor (Bucchi, New Castle) and then lyophilized to obtain a hydroalcoholic extract of *G. lanceifolia* (GLE). The yield of GLE was 7.42 % w/v.

Experimental animals

Healthy female rats were procured from Laboratory Animal House Facility, CSIR-Indian Institute of Toxicology Research, Lucknow, India. All the animals were acclimatized for at least one week in animal house under standard conditions of temperature of (25 ± 2°C), relative humidity 45-55% and 12 h light/dark cycle. The animals fed with standard rodent feed (Ashirwad, India) and water. The rats were randomly assigned to different experimental groups, each containing five rats. All the animal experiments were conducted in compliance with the CCSEA guidelines for the care and use of experimental animals and with the prior permission from Institutional Animal Ethics Committee of University Institute of Pharmacy, Chhatrapati Shahu Ji Maharaj University (CSJMU), Kanpur.

Acute toxicity Study

OECD guideline nos. 420 were used to conduct single dosage toxicity tests [10]. This investigation employed 4 groups of rats to look into any possible harmful effects or variations in normal behavior. The acute toxicity of *G. lanceifolia* fruit extract was investigated by administering different concentrations of GLE (500, 1000 and 2000 mg/kg body weight) orally using gavage needle to first three different groups of animals. Prior to the experiment, the animals were fasted for 24 h though water was allowed *ad libitum.* The fourth group was taken as negative control and given as vehicle (1% CMC in distilled water).

On day of study, the animals were weighed and the test drug was administered after the 24-hour fasting. Food may be withheld for further 3–4 hours after the substance has been administered.

Bodyweight measurement and cage side observation

Each animal was closely observed after the drug administration and special attention was given for the first 4-6 hours after the dosing. The animals were closely observed daily twice (morning & evening) for a total of 14 days for overt signs of toxicity, morbidity and mortality. In addition, each animal was removed from its cage and a physical examination of each animal were systematically recorded daily for any changes in skin, fur, eyes, respiratory function, autonomic effects such as salivation, diarrhea, urination and central nerve effects including tremors and convulsions, and general behavior [11,12]. The body weights of all the animals were measured at regular interval throughout the study period by using calibrated balance. Subsequent weight changes were calculated and documented.

Consumption of food and water

The amount of food and water consumption of each animal of both control and test groups were calculated daily from the amount of food and water delivered and the amount left after 24 hours during the study period [12,13].

Relative and absolute organ weight

All organs of experimental animals were collected using standardised surgical procedures at the end of the acute toxicity trial (14 days). Organs such as the liver, spleen and kidney, were removed, cleansed with saline, weighed, and kept in 10% formalin for histopathological investigations [13,14].

Heamatology

At the end of toxicity study, the blood sample collected in the heparinized centrifuge tubes was analysed using an automated hematology analyzer. Parameters evaluated include hemoglobin (HGB), erythrocyte count (RBC), hemat-ocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), platelet count and leucocyte count (WBC) [13,14].

Biochemical analysis

For serum separation, the blood samples were collected (after 14 days) in centrifuge tubes and immediately the serum was separated by centrifugation at 3000 rpm for 15 min. The separated serum sample were transferred into dry, clean serum tubes, stoppered and stored in the refrigerator at –80 °C till it was used for testing. The biochemical parameters analyzed included creatinine (CREA), urea, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total cholesterol and electrolytes [15].

Histopathological observation

The harvested vital organs (liver, kidney, spleen) were microscopically examined. The morphology of the internal organs was visually observed for any signs of toxicity. These organs were stained with hematoxylin and eosin (H&E) stain. Abnormalities were categorized as mild,

moderate, and severe [16].

Statistical analysis

Data were presented as mean ± SEM and analyzed by Graph pad prism 5. In acute toxicity groups, mean differences were calculated by a Student t-test. Differences between groups were considered significant at $p < 0.05$.

RESULTS

Cage side observation

Oral administration of GLE up to dose of 2000 mg/kg in experimental rats did not produce significant changes in behavior, skin, breathing, faeces, postural irregularities, impairment in water consumption and food intake and hair loss. No animal mortality was detected in the groups treated with *G. lanceifolia* fruit extract.

Body weight measurement

The mean body weight of rats was recorded daily for a period of 14 consecutive days. The CMC (negative control) group exhibited slight gain in body weight relative to the other three rat groups administered *G. lanceifolia* fruit extract. Body weight measurement in animals is shown in Table 1.

Table 1 A. Mean body weight measurements in the test and control group for 14 days.

Group	Day 0 (g)	1st week (g)	2nd week (g)
Control (1% CMC)	201.6 ± 2.78	211.6±2.40	221.8±2.28
GLE 500 mg/kg	199.0± 2.30	207.8±1.64	217.3±1.60
GLE 1000 mg/kg	200.6 ± 2.39	209.6±2.02	218.6±2.028
GLE 2000 mg/kg	199.0 ± 2.98	208.2±2.44	217.8±2.15

Values are expressed as mean ± SEM of five rats in each group. No statistically significant ($p > 0.05$) difference was observed between test and control groups.

Food and water consumption

Daily measurements of food and water consumption were conducted by assessing the quantity supplied and the remaining amount after 24 hours. The amounts consumed were calculated from total intake and average relative body weight of preceding time interval. Each data point represents an equal sample size. The data presented in table 2 indicated no statistically significant difference from the negative control (1% CMC), $p > 0.05$.

Table 2 Food intake of rats after 14 days' exposure of test and control groups

Group	Day 0	1st week	2nd week
Control (1% CMC)	13.22 ± 0.36	13.93 ± 0.44	14.65 ± 2.45
GLE 500 mg/kg	13.59 ± 0.26	14.38± 0.37	14.95 ± 0.34
GLE 1000 mg/kg	13.21± 0.31	13.98 ± 0.41	14.75 ± 0.23
GLE 2000 mg/kg	13.67 ± 0.67	14.48 ± 0.42	15.03 ± 0.40

Values are expressed as mean ± SEM of five rats in each group. No statistically significant ($p > 0.05$) difference was observed between test and control groups.

Absolute and relative organ weight

At the end of the 14-day acute toxicity test period, the absolute and relative organ weights of the rats were measured. There were no statistically significant differences observed among groups, $p > 0.05$.

Complete blood count (CBC) test

Hematological data from rats for 13 parameters were analyzed at the end of the acute toxicology test period (14 days). RBC-related parameters (HB, Total RBC, HCT, MCV, MCHC, MCH) show mild dose-related fluctuations, particularly at 1000 mg/kg, where hemoglobin and RBC count slightly drop. However, all remain within normal reference limits, suggesting no overt anemia or hemolysis. WBC and differential counts are stable across all doses, indicating no significant immune suppression or stimulation. Platelet counts remain unchanged, suggesting no hematopoietic toxicity affecting thrombopoiesis. No marked hematological toxicity is observed up to the highest tested dose (2000 mg/kg). The hematological results of the control and GLE treated rats are given in Table 3

Table 3 Hematological parameters of acute toxicity study

Biological marker	Unit	Control (1% CMC)	GLE 500 mg/kg	GLE 1000 mg/kg	GLE 2000 mg/kg
HB	(g/dl)	14.5	13.91	13.41	14.1
TOTAL RBC	X(10 ^ 6) / L	8.47	8.21	7.91	8.41
HCT	%	54.1	54.23	53.80	54.2
MCV	FL	63.9	62.9	63.1	63.8
MCHC	(g/dl)	26.8	26.4	26.2	26.5

PLATELET COUNT	X(10 ^ 3) / L	644	642	641	640
WBC COUNT	X(10 ^ 3) / L	7.63	7.53	7.62	7.61
NEUTROPHILS	%	53.4	53.2	53.3	53.2
LYMHOCYTES	%	22.0	22.1	22.2	22.0
MONOCYTES	%	0.5	0.5	0.5	0.6
EOSINOPHILS	%	7.4	7.2	7.4	7.3
MCH	Pg	17.9	17.4	17.5	17.7
BASOPHILS	%	16.7	16.1	16.2	16.3

HB: Hemoglobin, RBC: Red Blood Cell Count, WBC: White Blood Cell Count, MCV: Mean Corpuscular, Volume, MCH: Mean Cell Hemoglobin, MCHC: Mean Corpuscular Hemoglobin Concentration. Values are expressed as mean ± SEM of five rats in each group. No statistically significant ($p > 0.05$) difference was observed between test and control groups.

Biochemical analysis

Table 4 indicated the changes in readings for the diverse biochemical indicators across the majority of the rat groups. Significant increase was observed in some parameters such as ALT, and cholesterol at highest dose level

Table 4 Biochemical parameters of acute toxicity study

Parameter	Control (1% CMC)	GLE 500 mg/kg	GLE 1000 mg/kg	GLE 2000 mg/kg
Creatinine (mg/dL)	0.56 ± 0.06	0.42 ± 0.12	0.30 ± 0.01	0.69 ± 0.10
Urea (mg/dL)	42.13 ± 4.42	48.70 ± 5.81	46.90 ± 6.48	42.40 ± 2.95
ALT (U/L)	80.00 ± 6.81	61.33 ± 10.17	69.33 ± 8.11	122.7 ± 19.46*
AST (U/L)	140.30 ± 22.60	136.70 ± 20.93	124.00 ± 4.16	134.00 ± 6.43
Total Cholesterol (mg/dL)	50.00 ± 7.37	35.67 ± 0.88	43.33 ± 9.33	71.00 ± 22.27*
Na^+ (mmol/L)	105.00 ± 1.80	124.70 ± 1.07	129.60 ± 0.41	151.00 ± 0.56
K^+ (mmol/L)	6.97 ± 0.05	6.53 ± 0.56	6.12 ± 0.15	6.08 ± 0.09
Cl^- (mmol/L)	109.60 ± 5.11	151.00 ± 2.56*	159.20 ± 3.63*	106.80 ± 0.84

Values are expressed as mean ± SEM of five rats in each group. No statistically significant ($p > 0.05$) difference was observed between test and control groups. For a given dose,* indicate a significant difference at $p < 0.05$.

Histopathological observation

Histopathology has long been regarded as the most reliable method for determining the level of no-observed-adverse effects [17]. Histopathological analysis of liver, kidney, and spleen sections from normal and acute toxicity groups was conducted on the final day of the experimental period, with results presented in Figure 1.

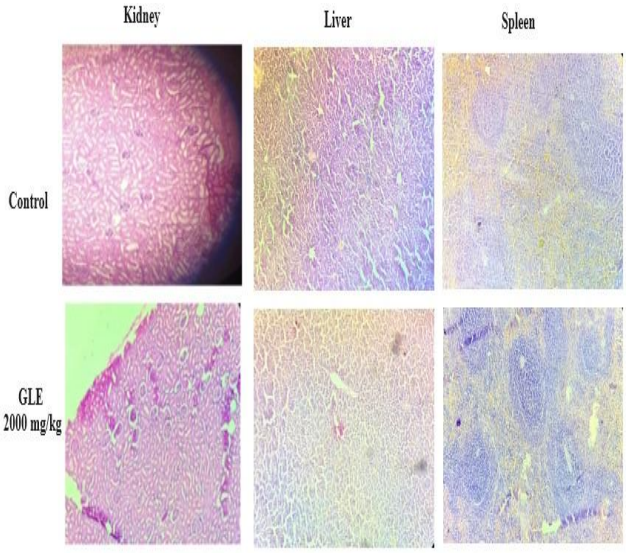

Fig. 1 Representative images of kidney, liver and spleen of rats at from control and 2,000 mg/kg of GLE

The renal cortex is visible in the kidney tissue section, with several well-preserved glomeruli encased in Bowman's

capsules and encircled by proximal and distal convoluted tubules. There is no indication of glomerular architectural deterioration, hypercellularity, or sclerosis. Tubular dilatation, necrosis, or degeneration are not evident, and the tubules retain their normal epithelial lining. There are no indications of fibrosis, inflammation, or edema in the interstitial spaces, which seem normal. No discernible harmful effects on renal tissue are implied by the statement for the treated group.

The microscopic section of the liver in image appears to show a generally preserved hepatic lobular architecture with distinct cords of hepatocytes separated by sinusoids. The central veins and portal triads are visible, and there is no prominent evidence of massive necrosis, steatosis, or marked inflammatory infiltration. Hepatocytes arranged in cords, sinusoidal spaces intact, central vein visible. **No obvious toxic pathological changes:** No extensive hepatocellular degeneration, necrosis, or fatty infiltration seen in this field.

The splenic architecture is discernible with identifiable **white pulp** (lymphoid follicles) and **red pulp** regions. However, the demarcation between these zones is **less distinct** compared to a normal control spleen. The **red pulp** exhibits: **Diffuse yellowish-brown pigment deposits**, likely representing **hemosiderin**, suggestive of increased erythrocyte breakdown and iron storage. Mild **vascular congestion** with dilated sinusoids. The **white pulp** shows, Slight **lymphoid depletion** in some follicles. No marked hyperplasia or germinal center formation. No evidence of necrosis, granulomatous inflammation, or neoplastic changes is observed in this field. The observed changes **mild lymphoid depletion, hemosiderin deposition, and congestion** are **consistent with a mild toxic effect** on the spleen, potentially secondary to **erythrocyte damage, increased hemolysis, or altered splenic blood flow**.While not overtly severe, these alterations may indicate **subclinical splenic stress** due to the administered test substance, particularly if absent in concurrent controls.

DISCUSSION

Plant derived natural remedy, has gained widespread popularity in primary healthcare globally. Herbal sources derived from medicinal plants are considered safe and devoid of adverse health effects, leading to their widespread usage in self-medication [18]. Nonetheless, due to the absence of validated scientific research on the toxicity and detrimental effects of these medicines, additional investigations are necessary, which also pertains to Garcinia species. During this 14-day acute toxicity assessment, rats administered *Garcinia lanceifolia* fruit extract at doses of 500, 1000, and 2000 mg/kg exhibited no mortality, and all subjects displayed no signs of toxicity. Our findings regarding cage-side functional observations, including respiratory effects and impacts on the autonomic and neurological systems in high-dose group animals, revealed no significant alterations in animal behavior. The *Garcinia lanceifolia* fruit extract, irrespective of the dosage administered, did not influence the body weight of the rats and resulted in no significant alterations in their food and water intake. The consumption of food and water demonstrated normal metabolic activity in the animals [19], indicating that the single oral administration of *G. lanceifolia* extract did not impede the growth of rats. The rise in body weight may be attributed to the accumulation of body fat. Our findings suggest that no significant alterations were seen in the absolute organ weights of the liver, kidneys, and spleen of rats administered with *G. lanceifolia* extract compared to the negative control group. The biochemistry results indicated little alterations in the biochemical parameters of the *G. lanceifolia* extract-treated group. Only a significant increase in ALT levels was seen at the maximum dosage (2000 mg/kg body weight) of *G. lanceifolia* extract.

The effect of the *G. lanceifolia* extract at doses of 500 mg/kg, 1000 mg/kg, and 2000 mg/kg on various hematological parameters in treated animals compared to control showed that hemoglobin (HB) levels remained relatively stable across all treated groups, ranging from 13.91 to 14.1 g/dl, compared to the control value of 14.5 g/dl, suggesting no significant impact on hemoglobin concentration. Total RBC count showed a slight, non-significant decrease in a dose-dependent manner, from 8.47×10^6/L in control to 8.41×10^6/L at the highest dose (2000 mg/kg), but remained within the normal range. Hematocrit (HCT) values remained consistent across all groups (54.23–54.2%), indicating no notable changes in the volume percentage of red blood cells. MCV, MCH, and MCHC values showed minimal variation across groups, with MCV ranging from 62.9–63.8 FL; MCH from 17.4–17.7 pg, and MCHC from 26.4–26.5 g/dl, indicating stability in red blood cell indices.Rats given 500 mg/kg, 1000 mg/kg, and 2000 mg/kg doses of the test extract did not exhibit any appreciable changes in their hematological analysis as compared to the normal control group. All treated groups' hemoglobin, total red blood cell count, hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) levels stayed within the normal physiological range. Comparable differences between the control and treatment groups were also not observed in the platelet count, total WBC count, or differential leukocyte counts (neutrophils, lymphocytes, monocytes, eosinophils, and basophils). Platelet counts remained comparable across all groups (~$642–640\times10^3$/L), indicating no thrombocytopenic or thrombocytosis effects due to treatment. Total WBC count and differential leukocyte counts (neutrophils, lymphocytes, monocytes, eosinophils, and basophils) showed no significant variations between the treated and control groups: WBC count: $7.53–7.61\times10^3$/L; Neutrophils: ~53%; Lymphocytes: ~22%; Monocytes: 0.5–0.6%; Eosinophils: 7.2–7.3%; Basophils: 16.1–16.3%.

Plasma creatinine concentrations are utilized to assess glomerular filtration rate [20] . The recommended range for creatinine is 0.2 to 0.8 mg/dl [21]. In present investigation, creatinine levels of all acute toxicity group were within the normal physiological range and did not differ statistically

significantly from the control ($p > 0.05$), as indicated by the lack of histological alterations in the kidney.

This suggests that neither renal nor glomerular filtration were hampered by the extract. There was no discernible difference in the 500 mg/kg and 1000 mg/kg groups for the liver enzymes ALT and AST ($p > 0.05$), suggesting that there was no hepatocellular damage at these dosages. At the maximum dosage, however, a significant increase in ALT was noted at 2000 mg/kg, indicating mild hepatic stress. Usually, elevated ALT is linked to hepatocyte leakage, which could be a sign of early cellular irritation from an excessive dosage [22]. The fact that AST levels stayed constant in spite of this suggests that any liver damage was not very serious. At 500 mg/kg and 1000 mg/kg doses, serum total cholesterol significantly decreased, indicating a potential hypolipidemic impact of the extract, perhaps as a result of phytosterols or flavonoids affecting lipid metabolism. On the other hand, a rise in cholesterol at 2000 mg/kg can be the result of a biochemical reaction brought on by stress or a dose-related change. In terms of electrolytes, both groups' levels of Na^+ and K^+ stayed within normal physiological bounds, suggesting that electrolyte balance was maintained ($p > 0.05$). But at the 500 mg/kg and 1000 mg/kg doses, Cl^- levels were noticeably higher ($p < 0.05$), which might be related to a small acid-base adjustment but did not result in any toxicity symptoms. Histological analysis of kidney, liver and spleen samples treated with *G. lanceifolia* extract, stained with hematoxylin and eosin (H&E), demonstrated no aberrant morphological characteristics across all extract groups.

CONCLUSION

In conclusion, the acute toxicity investigation indicated that GLE possesses an LD50 value exceeding the test dose of 2,000 mg/kg. The oral administration of GLE (500, 1000, and 2000 mg/kg body weight) to Wistar rats over a period of 14 days did not result in treatment-related mortality or adverse effects in any of the experimental animals. The statistical analysis of the acute toxicity study groups demonstrates that there were no significant alterations in body weight, food and drink intake, or absolute and relative organ weights. Furthermore, more of the hematology parameters and serum biochemical tests carried out on the investigational animals were within the reference range. This study provides valuable data on the toxicity profile of *G. lanceifolia*. Further studies may focus on chronic toxicity studies of *G. lanceifolia* in order to evaluate its long-term effects.

ACKNOWLEDGEMENT:

The authors wish to *acknowledge* the financial *support* provided by the Department of Biotechnology, Government of India.

CONFLICT OF INTEREST:

The authors declare no conflict of interest

REFERENCE

1. Fengke Lin, Zihao Liu, Zhongxin, Duan, Rui Zhao, Binsheng, Luo. 2025. An overview of Garcinia species research based on bibliometric analysis using VOSviewer, CiteSpace and HistCite. Applied Food Research, 5(2), 101392.
2. Espirito Santo BLSD, Santana LF, Kato Junior WH. 2020. Medicinal potential of Garcinia species and theircompounds. Molecules, 25(19), 4513.1706
3. Gupta PC, Kar A, Sharma N, Singh PK, Goswami NK, Kumar S. 2021. Protective effect of standardised fruit extract of Garcinia cowa Roxb. ex Choisy against ethanol induced gastric mucosal lesions in Wistar rats. Annals of Medicine, 53(1), 1696-1708.
4. Baruah A. 2007. Ethnomedicinal plants of India. Aavishkar Publisher and Distributors, Jaipur.
5. Bora NS, Kakoti BB, Bairy P, Gogoi B. 2014. Garcinia lanceifolia Roxb., an endemic medicinal plant of Assam, relieves pain and delays nociceptive response: An assay for its analgesic and anti-inflammatory activity. International Journal of Pharmaceutical Sciences and Drug Research, 6, 216–219.
6. Gupta PC, Kar A, Sharma N, Sethi N, Pandey P, Saharia D, Goswami NK. 2019. Pharmacognostic and physicochemical Studies of Garcinia lanceifolia Roxb. Var. Oxyphylla. Research Journal of Pharmcy and Technology, 12(2), 706-710.
7. Chowdhury T, Handique PJ. 2012. Evaluation of antibacterial activity and phytochemical activity of Garcinia lanceifolia Roxb. Indian Journal of Pharmaceutical Science and Research, 3, 1663–1667.
8. Kar A, Borkakoti S, Borthakur SK. 2008. Extended distribution of Garcinia species in Sonitpur district, Assam. Pleione, 2, 165–170.
9. Bora NS, Kakoti BB, Yadav P, Gogoi B, Borah S. 2017. Phyto-physicochemical, acute and subacute toxicity studies of Garcinia lanceifolia Roxb.- A rare ethnomedicinal plant of Assam, India. Indian Journal of Natural Products and Resources, 8(4), 360-369.
10. OECD, 2001. OECD Guideline for Testing of Chemicals. Acute Oral Toxicity-Fixed Dose Procedure. OECD/OEDC.
11. Obici S, Otobone JF, da Silva Sela, VR, Ishida K, da Silva JC, Nakamura CV, Cortez D.A.G., Audi EA. 2008. Preliminary toxicity study of dichloromethane extract of Kielmeyera coriacea stems in mice and rats. Journal of Ethnopharmacology, 115, 131–139.
12. Kumarnsit, Ekkasit, Niwat Keawpradub, and Watcharin Nuankaew. 2006. "Acute and long-term effects of alkaloid extract of Mitragyna speciosa on food and water intake and body weight in rats." Fitoterapia, 77, 339-345.
13. Demma J, Gebre-Mariam T, Asres K, Evgetie W, Engindawork E. (2006). Toxicological study on Glinus lotoides: a traditionally used technical herb in ethiopia.

Journal of Ethnopharmacology, 111, 451–457.
14. Tan PV, Mezui C, Enow-Orock G, Njikam N, Dimo T, Bitolog P. 2008. Teratogenic effects, acute and sub chronic toxicity of the leaf aqueous extract of Ocimum suave Wild (Lamiaceae) in rats. Journal of Ethnopharmacology, 115, 232–237.
15. Mukinda JT, Syce JA. 2007. Acute and cronic toxicity of the aqueous extract of Artemisia afra in Rodent. Journal of Ethnopharmacology. 111, 138–144.
16. Jain A, Soni M, Deb L, Jain A, Rout SP, Gupta VB, Krishna KL. 2008. Antioxidant and hepatoprotective activity of ethanolic and aqueous extract of Momordica dioica Roxb leaves. Journal of Ethnopharmacology, 115, 61–66.
17. Dorato MA, Engelhardt JA. 2005. The no-observed-adverse-effectlevel in drug safety evaluations: use, issues, and definition(s). Regul Toxicol Pharmacol, 42:265–74.
18. Vaghasiya YK, Shukla VJ, Chanda SV. 2020. Acute oral toxicity study of pluchea arguta boiss extract in mice. J Pharmacol Toxicol, 6, 113–23.
19. Mukinda JT, Syce JA. 2007. Acute and cronic toxicity of the aqueous extract of Artemisia afra in Rodent. Journal of Ethnopharmacology, 111, 138–144.
20. Mpio I, Laville M, Hadj-Aïssa A, Fauvel JP. 2003. Predicted creatinine clearance to evaluate glomerular filtration rate in black Caribbean subjects. Nephrol Dial Transplant, 18:1307–10.
21. Loha M, Mulu A, Abay SM, Ergete W, Geleta B. 2019. Acute and subacute toxicity of methanol extract of Syzygium guineense leaves on the histology of the liver and kidney and biochemical compositions of blood in rats. Evid Based Complement Altern Med, 2019; 2019.
22. Atici S, Cinel I, Cinel L, Doruk N, Eskandari G, Oral U. 2005. Liver and kidney toxicity in chronic use of opioids: An experimental long-term treatment model. Journal of Bioscience, 30, 245–252.