The Journal of Anatomical Sciences
Email: journalofanatomicalsciences@gmail.com

J. Anat Sci 17(2) June

Submitted: April 19th, 2026
Revised: June 22nd, 2026
Accepted: June 26th, 2026

Antidiabetic and Pancreatoprotective Effects of Hexane Seed Extract of *Garcinia kola* in Alloxan-Induced Diabetic Wistar Rats: Histological and Glycaemic Evidence

Vincent Uchenna Uche [1a], John Chukwuebuka Abraham [2b*], Clinton Ogbonnaya Njoku [1c], Michael Arinze Epete [1], Albert Nkereuwem Eteudo [1], Ifeanyichukwu Maxwell Ama [1d], Monday Nelly Chima [1e], Emmanuel Tochukwu Ekuma [3f], Ugochi Okeh[4]

[1]Department of Anatomy, Ebonyi State University; [2]Department of Radiography, Evangel University Akaeze; [3]Biochemistry Unit, Science Laboratory Technology Department, Akanu Ibiam Federal Polytechnic Unwana; [4]Department of General Studies, Federal College of Agriculture, Ishiagu, Ebonyi State

ORCIDs: [a]0009-0007-6924-0159; [b]0000-0001-5146-6714; [c]0000-0001-6082-9177; [d]0009-0000-3449-393x; [e]0009-0006-4794-0712; [f]0000-0002-2025-5364.

***Corresponding Author:** Email: abrahamjohnebukac@gmail.com; Tel +2348030562255

ABSTRACT

Diabetes mellitus is estimated to affect over 589 million globally, with projections reaching 853 million by 2050, and a higher burden reported in sub-Saharan Africa. The disease is characterized by destruction of pancreatic β-cells and persistent hyperglycemia. Limitations associated with conventional antidiabetic drugs in low-resource settings emphasize the need for accessible phytotherapeutic alternatives. *Garcinia kola*, a medicinal plant native to West Africa, has shown antidiabetic activity in preclinical studies; however, its hexane seed fraction remains poorly investigated. This study evaluated the effects of graded doses of hexane seed extract of *Garcinia kola* on fasting blood glucose and pancreatic histoarchitecture in alloxan-induced diabetic Wistar rats. Thirty-six rats were divided into six groups (n=6): normal control, diabetic untreated, metformin (500 mg/kg), and extract-treated groups (100, 300, and 500 mg/kg). Diabetes was induced intraperitoneally using alloxan (150 mg/kg) and confirmed after 72 hours at ≥180 mg/dL. Treatments were administered orally for 28 days. Fasting blood glucose was measured at baseline, post-induction, and on days 7, 14, and 21. Pancreatic tissues were examined using hematoxylin and eosin staining. Data were analyzed using one-way ANOVA at $p<0.05$. By day 21, the 300 mg/kg and 500 mg/kg groups showed near-normal glucose levels of 90.67±16.04 mg/dL and 87.67±22.19 mg/dL, respectively, compared with the metformin group (409.33±64.66 mg/dL) and untreated diabetic group (231.33±46.49 mg/dL). Histological analysis showed regeneration of islets of Langerhans and partial recovery of pancreatic structure in higher-dose groups. Overall, the extract demonstrated dose-dependent antihyperglycemic and pancreatic-protective effects, supporting further investigation into its potential therapeutic use.

Keywords: *Garcinia kola*, Hexane, Alloxan, Diabetes Mellitus, Pancreas

INTRODUCTION

Diabetes mellitus (DM) is a long-term metabolic condition resulting from impaired insulin production, reduced insulin effectiveness, or both, leading to prolonged elevations in blood glucose levels and disturbances in carbohydrate, lipid, and protein metabolism 1. Diabetes prevalence has risen markedly worldwide and is projected to continue increasing through 2050[2]. The burden is growing fastest in resource-limited regions, while improvements in treatment access remain uneven, creating significant disparities in diabetes management and healthcare outcomes [3]. Within Nigeria, evidence synthesized from studies involving 124,876 individuals estimated the prevalence of Type 2 diabetes mellitus at 7.0%, suggesting that about 8.02 million people are affected by the condition nationwide. Additionally, the country has one of the highest reported rates of intermediate hyperglycemia globally, with a prevalence of 40.5%, indicating a substantial population at risk of developing diabetes[4].

The pancreas occupies a central role in DM pathogenesis. As the principal endocrine organ regulating insulin secretion through its islets of Langerhans, the pancreas is severely vulnerable to cytotoxic insults that selectively destroy β-cells. Studies conducted previously show that alloxan-induced diabetic models indicate that alloxan enters pancreatic β-cells through GLUT2 transporters and triggers reactive oxygen species production, leading to oxidative injury, loss of β-cell function, impaired insulin secretion, and prolonged hyperglycaemia[5,6]. Although progressive islet necrosis, inflammatory cell infiltration, and acinar disruption collectively impair both endocrine and exocrine pancreatic function[7].

Previous reports have indicated that although conventional antidiabetic therapies effectively manage hyperglycemia, they do not promote pancreatic β-cell regeneration and may be constrained by adverse effects, high treatment costs, and limited accessibility, particularly in low-resource settings[8,9].

However, *Garcinia kola* Heckel, a member of the Clusiaceae (formerly Guttiferae) family, is commonly known as bitter kola[10], is an evergreen tree indigenous to the moist lowland forests of West and Central Africa. In Nigerian ethnomedicine, the seed is designated "Aki Ilu" among the Igbo, "Orogbo" among the Yoruba, and "Namijin Gworo" among the Hausa. It has been used traditionally for the management of metabolic diseases, including diabetes, liver disorders, and respiratory conditions [11, 12]. The seed is richly endowed with bioactive secondary metabolites, including biflavonoids, most notably kolaviron, as well as benzophenones, xanthones, triterpenoids, phytosterols, and species unique compounds, including garcinianin, kolanone, and garcinoic acid [13, 14]. Kolaviron, the most extensively studied constituent, has demonstrated the capacity to suppress hyperglycemia and protect islet architecture in streptozotocin-induced diabetic rats [15], and to reduce oxidative hepatic damage through up-regulation of antioxidant enzymes[16]. Ethanolic and methanolic extracts of *Garcinia kola* are well documented in the literature; however, the hexane fraction, which contains mainly non-polar compounds like phytosterols, terpenoids, fixed oils, garcinoic acid, and kolanone, remains less explored[17]. Studies have reported that the hexane fraction of *Garcinia kola* significantly reduced fasting blood glucose and maintained inflammatory biomarker profiles comparable to insulin-treated controls in streptozotocin-induced diabetic rats. Consistent with these findings, active subfractions of *G. kola* have also been shown to improve glycemic control and prevent diabetes-associated motor deficits in streptozotocin-induced diabetic mice[17,18]. This study was designed to evaluate the antidiabetic effects of the hexane extract of Garcina kola in alloxan-induced diabetic rats.

MATERIALS AND METHODS

Plant authentication and extract preparation

Garcinia kola seeds were obtained from the International Market in Abakaliki, Ebonyi State, Nigeria, and their identity was confirmed at the Herbarium of the Department of Applied Biology, Ebonyi State University (EBSU). The seeds were manually dehulled, cut into smaller pieces, and dried under ambient laboratory conditions. After drying, they were pulverized into a fine powder using a mechanical grinder. A 500 g portion of the powdered material was soaked in 2.5 L of n-hexane and left to extract for 72 hours, with intermittent agitation every six hours to enhance solvent penetration. The resulting mixture was filtered using Whatman No. 1 filter paper, and the filtrate was concentrated by allowing the solvent to evaporate at room temperature. This process yielded a viscous crude hexane extract, which was subsequently stored in a refrigerator until required for further analysis.

Drug and chemical

Alloxan monohydrate (Sigma-Aldrich 8269) and metformin were procured from reputable pharmaceutical suppliers. All reagents and medications employed in this investigation were of analytical grade and used in accordance with the manufacturers' instructions and recommended handling procedures.

Animal procurement and housing

Thirty-six adult Wistar albino rats (200–260 g) were procured from the Faculty of Basic Medical Sciences Animal Facility at Ebonyi State University. Only male rats were used to eliminate hormonal variations associated with the female reproductive cycle. The Experimental animals were kept in well-ventilated aluminum cages under a 12-hour light/12-hour dark photoperiod at ambient room temperature. They had unrestricted access to standard pelleted commercial feed produced by Chikun Feed (Nigeria Ltd.) and clean tap water throughout the study. Before the experiment, the animals were allowed to acclimate to the laboratory environment for one week."

Ethical approval

Ethical approval for this study was granted by the Research and Ethics Committee of the Faculty of Basic Medical Sciences (FREC), Ebonyi State University, Abakaliki, Nigeria, under reference number MPC/2502/30/014. All experimental procedures complied with internationally accepted standards for the care and use of laboratory animals.

Induction of diabetes

After the one-week adaptation period, diabetes was experimentally induced in the designated groups by administering a single intraperitoneal dose of alloxan monohydrate (150 mg/kg body weight). Before induction, the animals were deprived of food for at least 6 hours. Seventy-two hours after alloxan administration, fasting blood glucose concentrations were determined using an Accu-Chek Active glucometer manufactured by Roche (Germany). Animals exhibiting fasting blood glucose values of 180 mg/dL or higher were classified as diabetic and selected for subsequent experimental procedures.

Experimental design

The present study investigated the antihyperglycemic and pancreatic-protective effects of hexane seed extract of *Garcinia kola* in a Wistar rat model of alloxan-induced diabetes. Thirty-six adult male rats were randomly allocated into six experimental groups (n = 6 per group). Diabetes was induced in groups G2, G3, G5, and G6 through a single intraperitoneal administration of alloxan monohydrate (150 mg/kg body weight), while groups G1 and G4 were maintained as non-diabetic controls. Hyperglycemia

was confirmed 72 hours after induction, before the initiation of treatment. Group G1 received only standard feed and water, whereas G2 served as the diabetic untreated control. Group G3 was treated orally with metformin (500 mg/kg/day) via gastric gavage. Groups G4 to G6 received oral administration of *Garcinia kola* hexane extract at doses of 100, 300, and 500 mg/kg, respectively. Treatments were administered once daily for 28 consecutive days. The experimental design and treatment allocation are presented in Table 1.

Animal sacrifice

At the conclusion of the 28-day study period, the animals were humanely euthanized. A midline incision was made through the abdominal region to expose the internal organs. The pancreas was then carefully removed, cleared of surrounding connective and adipose tissues, and promptly preserved in 10% formalin saline for subsequent histological examination.

Histological procedures

Pancreatic tissues were immobilized in 10% formol-saline fixative, passed through progressive ethanol concentrations for dehydration (50%, 70%, 90%, and absolute alcohol) for 45 minutes each, cleared in three changes of xylene for 45 minutes each, and impregnated in three changes of molten paraffin wax at 60°C for 30 minutes each. Tissues were embedded in paraffin, sectioned at 3 µm with a rotary microtome, placed onto glass microscope slides, and subjected to H&E staining (hematoxylin and eosin). Stained sections were observed and photographed using a light microscope.

Statistical analysis

Results are reported as mean ± SD. Group differences were assessed by applying one-way analysis of variance (ANOVA), and post-hoc tests were conducted afterward and analyzed using SPSS version 20.0 (IBM Corp., Armonk, NY). A p-value below 0.05 was considered statistically significant.

RESULTS

Fasting blood glucose

Table 1 presents the fasting blood glucose values across all six groups at each of the five time points. Baseline glucose levels were homogeneous across all groups, ranging from 68.00±11.14 mg/dL to 78.33±10.12mg/dL, with no statistically significant inter-group differences ($F = 0.846$, $p = 0.543$), confirming pre-experimental uniformity. Following alloxan induction, Groups 2 through 6 demonstrated significant hyperglycemia relative to group 1 ($F = 7.951$, $p = 0.002$). Group 3 (metformin) recorded the highest value while Group 6 recorded the lowest among induced groups, with no significant difference between. By day 7 (Table 2), Group 6 demonstrated the most pronounced glucose reduction, with no significant difference from Group 1 or 2. Groups 3, 4, and 5 remained markedly hyperglycemic. At day 14 Groups 5 and 6 showed glucose concentrations not significantly different from Group 1, while Group 3 remained grossly elevated. By day 21 of treatment, Groups 5 and 6 achieved near-normoglycemia (Table 2). In stark contrast, the metformin group (G3) recorded the highest blood glucose of any treatment group, significantly greater than both Groups 1 and 2. The 100 mg/kg dose remained substantially hyperglycemic on day 21, not significantly different from Group 2.

Pancreatic Histopathology

Histological examination of H&E-stained pancreatic tissues demonstrated clear differences among experimental groups, ranging from normal architecture to varying degrees of injury and regeneration following alloxan induction and subsequent treatments.

Group 1 (non-diabetic control) demonstrated normal pancreatic histoarchitecture with well-defined, active islets of Langerhans and intact pancreatic acini, while Group 2 (untreated diabetic) showed moderate pancreatic degeneration with moderate areas of necrosis, marked infiltration of inflammatory cells, and poorly perfused acini, consistent with alloxan-induced β-cell cytotoxicity (Figure 1).

Group 3 showed normal pancreatic architecture with islets of Langerhans (IL) surrounded by intact pancreatic acini (A). The cellular morphology appears preserved, with no evidence of inflammation, necrosis, or structural distortion, indicating normal endocrine and exocrine pancreatic function. In contrast, Group 4 (alloxan-induced) sections demonstrated marked pathological changes. There is evident degeneration of pancreatic tissue characterized by poorly perfused and disrupted acinar cells, along with areas of necrosis (NA/N). The islets of Langerhans appear reduced in size and cellular integrity, suggesting β-cell damage. Prominent infiltration of inflammatory cells (IIC) further indicates ongoing tissue injury and inflammatory response. These findings are consistent with alloxan-mediated selective destruction of pancreatic β-cells and resultant oxidative stress–induced damage. In G5, there is partial recovery of pancreatic architecture, with reduced inflammatory infiltration and improved acinar organization, although some residual tissue distortion persists. This suggests a moderate protective or regenerative effect of the intervention. In G6, more pronounced restoration is observed, with relatively well-preserved islets of Langerhans and improved cellular architecture. The reduction in inflammatory cells and normalization of tissue structure indicate a stronger protective or antidiabetic effect compared to untreated diabetic groups. The histological findings suggest that alloxan induces significant pancreatic damage, while the treatment groups demonstrate varying degrees of histological recovery, with G6 showing the most notable

improvement in pancreatic integrity (Figure 2).

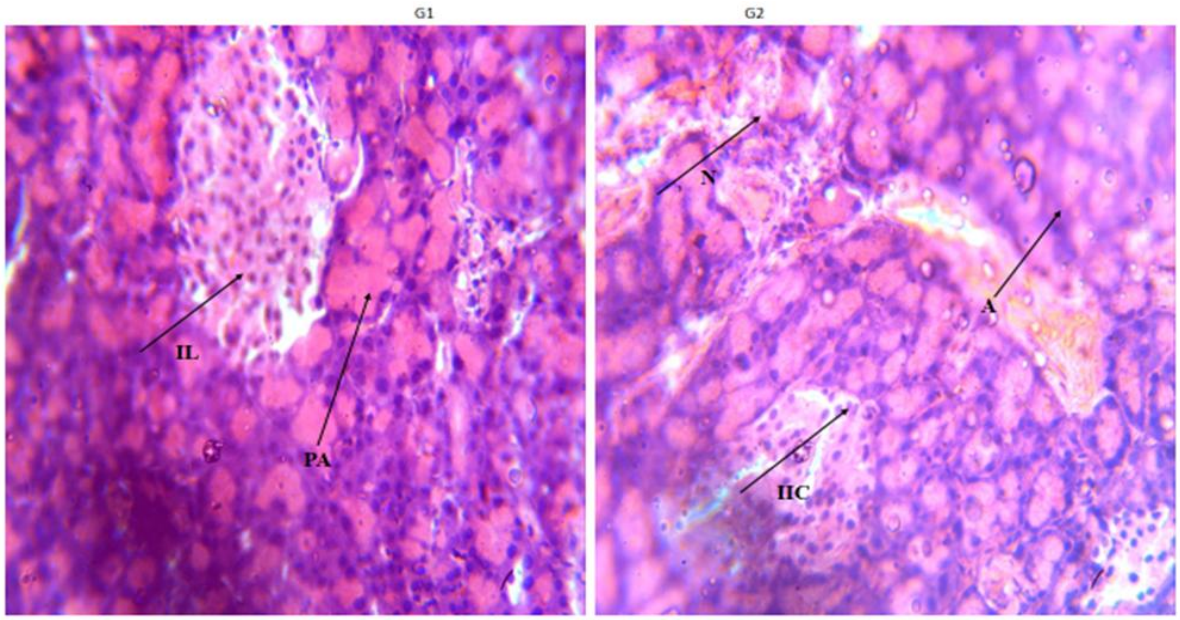

Figure 1: Photomicrographs of pancreatic sections (H&E stain, ×400). Group 1 (Control) shows normal pancreatic histoarchitecture with clearly visible, active Islets of Langerhans (IL) and well-preserved pancreatic acini (PA). Group 2 (alloxan-induced diabetic animals) demonstrates moderate degenerative changes characterized by areas of necrosis (N), infiltration of inflammatory cells (IIC), and poorly perfused pancreatic acini (A).

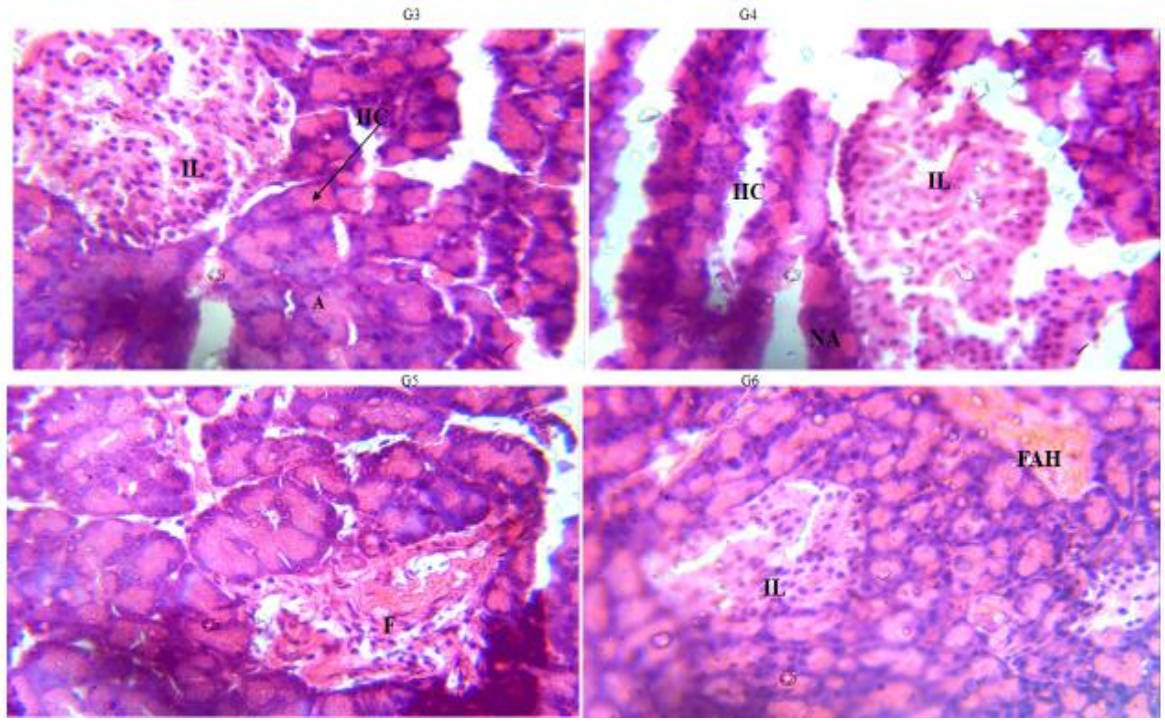

Figure 2: Pancreatic sections: G3 (metformin) with moderate IL and inflammation; G4 (*Garcinia kola* 100 mg/kg) mild regeneration with IL and necrotic acini; G5 (300 mg/kg) mild regeneration with fibrosis and necrosis; G6 (500 mg/kg) moderate regeneration with active IL and hemorrhagic areas, indicating dose-dependent histological improvement.

DISCUSSION

The findings of this study indicate that the n-hexane fraction obtained from Garcinia kola seeds progressively enhanced glucose-lowering activity and pancreatic tissue recovery with increasing dosage in Wistar rats with diabetes experimentally induced by alloxan. By day 21, animals receiving the two higher extract doses attained fasting blood glucose levels that did not differ significantly from those of the normal control group, whereas the lowest dose showed little improvement and remained comparable to the untreated diabetic group, suggesting limited therapeutic effectiveness at that dose. These results may have important implications for future therapeutic development, particularly in view of the rising worldwide prevalence of diabetes mellitus and the limited access to conventional diabetes medications in many parts of sub-Saharan Africa[8].

An important observation from the results was that the metformin-treated group exhibited markedly elevated blood glucose levels following induction but before treatment when compared with the other diabetic groups, even though glucose values were comparable among all experimental groups before alloxan administration. This suggests that, due to random allocation, the animals in the metformin group experienced greater damage to pancreatic β-cells following alloxan exposure than animals assigned to the remaining diabetic groups. Since metformin exerts its glucose-lowering effect largely through enhancement of insulin sensitivity and relies on the presence of remaining insulin-producing capacity, its effectiveness may have been reduced in animals with more extensive β-cell destruction[21], its therapeutic effectiveness would likely be reduced in animals experiencing greater destruction of pancreatic β-cells, irrespective of the dose administered. This initial difference between groups, rather than a true absence of glucose-lowering potential, is the most likely explanation for the less favorable pattern of blood glucose reduction observed in the metformin-treated animals. Therefore, this outcome should be interpreted as a consequence of the group assignment process within the study and not as evidence against the well-documented therapeutic effectiveness of metformin. Subsequent studies should consider stratifying or re-randomizing animals according to post-induction glucose levels or employing statistical approaches that account for initial glucose differences to minimize the influence of this source of bias.

By contrast, the higher-dose extract groups did not carry this baseline disadvantage, and their progressive glycemic improvement from day 7 onward is consistent with Osinubi[20], who demonstrated that the methanolic extract of *Garcinia kola* seeds significantly lowered blood glucose in alloxan-induced diabetic rats, and Idris *et al.*[17], who reported comparable glycemic reduction with the hexane fraction of *G. kola* in streptozotocin-induced diabetic rats. The extract appears to act through mechanisms that are at least partly insulin-independent, potentially including inhibition of α-glucosidase and α-amylase activity, stimulation of residual β-cell regeneration, and enhancement of peripheral glucose uptake, consistent with the pharmacological profile described for kolaviron and related biflavonoid constituents of *Garcinia kola* by Iwu *et al.*[22] and Salau *et al.*[23]. Non-polar constituents concentrated in the hexane fraction, including terpenoids, phytosterols, and long-chain fatty acids, may additionally contribute to insulin sensitisation and β-cell membrane stabilization. The dose-response pattern observed, with an incomplete response at the lowest dose and near-normoglycemia at the two higher doses, is consistent with reports by Kanedi *et al.*[24] and Adeyemi[25] that higher doses of *Garcinia kola* seed preparations produce the most pronounced antidiabetic effects, supporting a threshold-dependent relationship between bioactive constituent exposure and recovery from alloxan-induced β-cell injury.

The histological findings broadly corroborate the glycemic data, with one notable exception that merits cautious interpretation. The non-diabetic control retained normal islet and acinar architecture, while the untreated diabetic group showed degeneration, necrosis, and inflammatory infiltration consistent with alloxan-induced cytotoxicity. The two higher-dose extract groups showed the most advanced histological recovery, with active, morphologically preserved islets, paralleling their favorable glycemic outcomes. The metformin group, however, presented a discordant picture: its islets of Langerhans appeared histologically active and regenerated, yet acinar perfusion remained poor and inflammatory infiltration persisted, and this morphological recovery was not matched by a corresponding improvement in fasting blood glucose. This discordance suggests that structural islet recovery, as assessed by light microscopy, does not necessarily indicate restored functional insulin secretory capacity; a histologically intact-looking islet may still be functionally compromised. These observations align with Adaramoye[14], who demonstrated that kolaviron suppressed hyperglycemia and preserved islet architecture in streptozotocin-induced diabetic rats through antioxidant and anti-inflammatory mechanisms.

This study has limitations that should be acknowledged. Allocating animals to groups before alloxan induction, rather than stratifying by post-induction glycemic severity, introduced the baseline imbalance discussed above and complicates direct efficacy comparisons between the metformin and extract groups. The alloxan model predominantly replicates Type 1-like insulin-deficient diabetes and may not fully recapitulate Type 2 diabetes mellitus, which constitutes the overwhelming majority of

clinical cases; future work using a high-fat diet/low-dose streptozotocin model would strengthen translational relevance. The mechanistic pathways underlying the observed antidiabetic activity, including α-glucosidase inhibition, GLUT4 translocation, and β-cell regenerative signaling, were not directly assayed and represent important avenues for future investigation. Phytochemical standardization of the hexane fraction using HPLC and LC-MS/MS is also recommended to identify the specific bioactive constituents responsible for the observed effects[13].

CONCLUSION

The hexane seed extract of *Garcinia kola* produced progressive antidiabetic activity and enhanced pancreatic tissue repair with increasing dose in alloxan-induced diabetic Wistar rats, and higher treatment doses reduced fasting blood glucose to values not significantly different from those of the normal control group and facilitated regeneration of pancreatic islet cells by day 21. These findings offer experimental evidence for additional studies on its bioactive constituents, mode of action, and safety profile to determine its potential as an adjunct therapy for diabetes management.

REFERENCES

1. American Diabetes Association. Standards of medical care in diabetes—2021. Diabetes Care. 2021;44(Suppl 1):S1–S232.
2. Duncan BB, Magliano DJ, Boyko EJ. IDF Diabetes Atlas 11th edition 2025: global prevalence and projections for 2050. Nephrol Dial Transplant. 2025;41(1):7–9.
3. Liu J, Wang Y, Zhang L, Dang S. Global trends and inequalities in diabetes prevalence and treatment coverage among adults aged ≥45 years, 1990–2040: insights from the NCD-RisC database. Diabetes Metab Res Rev. 2026;42(4):e70164.
4. Olamoyegun MA, Alare K, Afolabi SA, Aderinto N, Adeyemi T. A systematic review and meta-analysis of the prevalence and risk factors of type 2 diabetes mellitus in Nigeria. Clin Diabetes Endocrinol. 2024;10(1):43.
5. Lenzen S. The mechanisms of alloxan- and streptozotocin-induced diabetes. Diabetologia. 2008;51(2):216–226.
6. Ardestani A, Maedler K. MST1 deletion protects β-cells in a mouse model of diabetes. Nutr Diabetes. 2022;12:7.
7. Cheekati RR, Rao SA, Vijayaraghavan R. A histological study of alloxan-induced diabetes on experimental male Wistar rats. Natl J Physiol Pharm Pharmacol. 2017;7(12):1329–1334.
8. Nasri H, Rafieian-Kopaei M. Metformin: current knowledge. J Res Med Sci. 2024;29:6.
9. Uti DE, Atangwho IJ, Alum EU, Egba SI, Ugwu OP, Ikechukwu GC. Natural antidiabetic agents: current evidence and development pathways from medicinal plants to clinical use. Nat Prod Commun. 2025;20(3):1934578X251323393.
10. Buba CI, Okhale SE, Muazzam I. Garcinia kola: the phytochemistry, pharmacology and therapeutic applications. Int J Pharmacogn. 2016;3(2):67–81.
11. Chinedu SN, Olatunde OA, Eddie-Amadi BK, Iheagwam FN. Column chromatographic purification of kolaviron from Garcinia kola seeds. Br J Med Med Res. 2013;3(3):651–659.
12. Ebomoyi MI, Okojie OH. Phytotherapeutic perspective of Garcinia kola (bitter kola): a review. Int J Nat Prod Res. 2012;2(1):1–7.
13. Tauchen J, Frankova A, Manourova A, Valterova I, Lojka B, Leuner O. Garcinia kola: a critical review on chemistry and pharmacology of an important West African medicinal plant. Phytochem Rev. 2023;23:1-47.
14. Adaramoye OA, Farombi EO, Adeyemi EO, Emerole GO. Comparative study of the antioxidant properties of flavonoids of Garcinia kola seeds in some rat tissues. Pak J Med Sci. 2005;21(3):331–339.
15. Adaramoye OA. Antidiabetic effect of kolaviron, a bioflavonoid complex isolated from Garcinia kola seeds, in Wistar rats. Afr Health Sci. 2012;12(4):498–506.
16. Owumi SE, Adedara IA, Farombi EO, Oyelere AK. Protective effects of kolaviron on lipopolysaccharide-induced hepatic and renal toxicity in rats. Hum Exp Toxicol. 2015;34(6):553–563.
17. Idris AE, Seke Etet PF, Saeed AA, Farahna M, Satti GMH, AlShammari SZ, *et al.* Evaluation of metabolic, antioxidant and anti-inflammatory effects of Garcinia kola on diabetic rats. Saudi J Biol Sci. 2020;27(12):3641–3646.
18. Seke Etet PF, Hamza MA, El-Tahir A, Vecchio L, Osman SY, Satti GMH, *et al*. An eluate of the medicinal plant Garcinia kola displays strong antidiabetic and neuroprotective properties in streptozotocin-induced diabetic mice. Evid Based Complement Alternat Med. 2022;2022:8708961.
19. Abboud AM. A modified protocol of the alloxan technique for the induction of diabetes mellitus in Wistar rats. Med Vet UFRPE. 2020;14(4):315–318.
20. Osinubi AAA. Effects of Garcinia kola and chlorpropamide (standard) in alloxan-induced diabetic male Sprague-Dawley rats.

Med J Islam World Acad Sci. 2005;15(3):105–110.
21. Bailey CJ. Biguanides and NIDDM. Diabetes Care. 1992;15(6):755–772.
22. Iwu MM, Igboko OA, Okunji CO, Tempesta MS. Antidiabetic and aldose reductase activities of biflavanones of Garcinia kola. J Pharm Pharmacol. 1990;42(4):290–292.
23. Salau VF, Erukainure OL, Ibeji CU, Olasehinde TA, Koorbanally NA, Islam MS. Kolaviron abrogated pathological abnormalities in brain tissues of diabetic rats by modulating crucial neuro-inflammatory and cholinergic enzymes. Metab Brain Dis. 2020;35(2):369–380.
24. Kanedi M, Widodo N, Busman H, Habibah D. Garcinia kola seed extract as an antidiabetic for alloxan-induced mice. Int J Agric Environ Biores. 2022;7(3):1–9.
25. Udenze ECC, Braide VB, Okwesilieze CN, Akuodor GC, Odey MO. The effects of gavage treatment with Garcinia kola seeds on biochemical markers of liver functionality in diabetic rats. Ann Biol Res. 2012;3(9):4601–4608.