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Original Research Article

RENOPROTECTIVE, ANTIDYSLIPIDEMIC, AND ANTIOXIDANT POTENTIALS OF *Picralima nitida* PHENOLIC LEAF EXTRACT IN STREPTOZOTOCIN-INDUCED DIABETIC RATS

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ABSTRACT

Complications of diabetes pose significant health challenges and emphasize the need for effective and specific interventions. This study evaluated the renoprotective, antidyslipidemic, and antioxidant effects of the phenolic fraction of *Picralima nitida* leaf extract in streptozotocin (STZ)-induced diabetic Wistar rats. To obtain a phenolic-rich fraction, fresh leaves were dried, ground, and extracted using hydro-methanolic maceration and solvent partitioning. A single STZ dosage of 50 mg/kg was used to induce diabetes in adult Wistar rats, who were then divided into six groups: normal control, diabetic control, glibenclamide (3 mg/kg), and phenolic extract at 200, 400, and 600 mg/kg. For 14 days, treatments were given orally. Standard spectrophotometric techniques were used to measure oxidative stress indicators, lipid profiles, and renal indices. One-way ANOVA and the Tukey post hoc test were used to examine the data at $p < 0.05$. The results indicate that phenolic extract improved all indicators in a dose-dependent manner. 600 mg/kg dosage restored Na^+, K^+, and Cl^- while considerably lowering urea and creatinine to levels statistically equivalent to normal controls ($p > 0.05$). Phenolic extract at 400 and 600 mg/kg significantly reduced total cholesterol, low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), and triacylglycerol (TAG) ($p < 0.05$), significantly correcting lipid abnormalities. While malondialdehyde (MDA) levels decreased dose-dependently, with 400 mg/kg correcting lipid peroxidation, antioxidant enzymes superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were significantly enhanced in all treatment groups ($p < 0.05$). Therefore, the phenolic extract of *Picralima nitida* leaf extract has proven to exhibit strong renoprotective, antihyperlipidemic, and antioxidant properties that are on par with or better than glibenclamide.

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Picralima nitida, diabetes mellitus, streptozotocin-induced diabetes, phenolic extract, renoprotective activity, antidyslipidemic activity.

INTRODUCTION

Diabetes is a serious worldwide health issue that affects millions of people of all ages. It happens when the body cannot effectively control blood sugar levels because to either inadequate insulin usage (Type 2) or insufficient insulin synthesis by the pancreas (Type 1) [1–3]. Over time, elevated blood sugar can damage vital organs including the liver and kidneys in addition to boosting the formation of harmful chemicals called free radicals, which results in oxidative stress [4–7]. Diabetes has been identified as one of the leading causes of mortality globally due to its rising occurrence, which affected an estimated 285 million people in 2010 and is predicted to reach 439 million by 2030 [4, 5], it

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has been cited as one of the main causes of death worldwide [4]. Type 2 diabetes (T2D) accounts for more than 90% of cases [6], leading to insulin resistance, metabolic dysregulation, and β-cell dysfunction [7]. The multi-organ issues linked to diabetes, which are mostly brought on by oxidative stress, chronic inflammation, and metabolic abnormalities, include hepatic, renal, and cardiovascular dysfunction [8–10].

The liver is crucial for regulating how fats and carbohydrates are metabolized. Hepatocellular oxidative stress, inflammation, and mitochondrial dysfunction are all brought on by persistent hyperglycemia in diabetes, which damages the liver's structure and function [11]. Important biomarkers that indicate the condition of the liver in diabetes patients include elevated blood liver enzymes including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), as well as anomalies in bilirubin and plasma protein levels [8,12]. These biomarkers serve as sensitive indicators of hepatic integrity and are crucial for evaluating the hepatoprotective potential of therapy treatments. Diabetic nephropathy (DN) is a common result of oxidative stress, inflammation, and microvascular damage brought on by hyperglycemia [13]. Diabetes has been linked to elevated blood urea and creatinine levels, electrolyte imbalances (such Na^+), and histological changes including glomerulosclerosis and tubular damage [13,14].

Oxidative stress is a critical role in the development of DN and is indicated by increased reactive oxygen species (ROS), lipid peroxidation, and the depletion of antioxidant defenses such as glutathione peroxidase (GPx), catalase (CAT), and superoxide dismutase (SOD) [2,15–17]. Monitoring oxidative stress indicators and renal function can help identify the extent of diabetic kidney damage and the efficacy of preventive interventions. Additionally, diabetes disrupts lipid metabolism, leading to dyslipidemia, a condition marked by elevated total cholesterol (TC), triglycerides (TAG), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C) and decreased high-density lipoprotein cholesterol (HDL-C) [18–20]. These lipid abnormalities impair hepatic and renal function, exacerbate oxidative stress, and raise the risk of cardiovascular disease.

Blood glucose monitoring, medication, lifestyle modifications, handling complications, and more are all part of treating diabetes [21]. Despite their effectiveness, conventional antidiabetic medications are frequently restricted in low- and middle-income countries due to their high cost, side effects, and limited accessibility [22]. Interest in therapeutic plants high in bioactive phytochemicals has increased as a result. Long before modern drugs were created, plants were the primary source of therapeutic remedies in traditional medicine [23]. *Picralima nitida*, commonly known as "Ogbono bitter" or "Akuamma," is a West African medicinal plant that has long been used to cure malaria, fever, gastrointestinal problems, and metabolic disorder [24–29]. Instead of depending on whole-plant preparations, current research is increasingly concentrated on specific phytochemicals and their impact in a few key organs and indicators. Therefore, we evaluated the renoprotective, antidyslipidemic, and antioxidant potentials of *Picralima nitida* phenolic leaf extract in streptozotocin-induced diabetic rats.

MATERIALS AND METHODS

Plant Material

Picralima nitida fresh leaves was collected from Eziobodo, Owerri, Imo State. A voucher specimen was created and placed in the departmental herbarium under the voucher number ESUT/AB/PN/2025/017 for future reference after the plant was identified and verified at the Department of Applied Biology, Enugu State University of Science and Technology (ESUT), Enugu, Nigeria. The leaves were washed to get rid of dirt and debris, let to air dry for fourteen days at room temperature (25 to 28 °C), and then ground into a fine powder using a clean electric grinder. Until extraction, the powdered material was kept in sealed containers.

Experimental Animals

The adult Wistar rats (150–200 g) were housed in standard plastic cages with a 12-hour light/dark cycle, a temperature of 22–26°C, and a relative humidity of 50–60% were employed to house the animals. They had unlimited access to clean water and normal rat pellets. Prior to testing, all animals were acclimated for seven days. Institutional and international standards for the care and use of laboratory animals were met by ethical handling and procedures.

Chemicals, Drugs and Equipment

Reputable analytical vendors provided methanol, n-hexane, ethyl acetate, ferric chloride reagent, and streptozotocin (STZ), which was typically supplied by Sigma-Aldrich (USA). The standard antidiabetic medication, glibenclamide, was likewise acquired from Sigma-Aldrich (USA). Every chemical and solvent utilized was of analytical quality. A rotary evaporator (Buchi Rotavapor R-300, Switzerland), digital weighing balance (Ohaus Pioneer PA214, USA), water bath (Memmert WNB 14, Germany), hot-air oven (Gallenkamp OV-160, United Kingdom), UV-visible spectrophotometer (Shimadzu UV-1800, Japan), glucometer (Accu-Chek Active, Roche Diagnostics, Germany), automated hematology analyzer (Sysmex KX-21N, Japan), and standard extraction glassware (Pyrex borosilicate, Corning Inc., USA).

Preparation of *Picralima nitida* Leaf Material

The sample was weighed and prepared for solvent extraction using hydro-methanolic maceration to maximize recovery of phenolic constituents. The dried powdered leaves were sieved to guarantee uniform particle size and kept in clean, airtight containers to prevent moisture absorption before extraction.

Extraction and Bioactive Constituents of Phenolic Fraction from *P. nitida* Leaf

Using techniques from [30], henolic compounds were extracted using a modified hydro-methanolic maceration and solvent-partitioning approach. To improve solute–solvent interaction, the powdered leaf material was macerated in 70–80% methanol for 48–72 hours while being stirred periodically. To create a semi-solid extract, the mixture was filtered, and the filtrate was concentrated at 40–50°C under low pressure using a rotary evaporator. After suspending the concentrated extract in distilled water, nonpolar components were removed by defatting it with n-hexane. Ethyl acetate was then used to partition the aqueous phase in order to extract medium-polar phenolic chemicals. The mixed ethyl acetate fractions were dried out and kept in amber vials at 4°C. The ferric chloride test was used to validate the presence of phenolic compounds in the extract, as indicated by distinctive color changes. The phenolic fraction's percentage yield was calculated in relation to the plant material's original dry weight.

Induction of Diabetes

Following the acclimation phase, streptozotocin (STZ) was used to produce experimental diabetes. Animals were given a single intraperitoneal injection of STZ at a dosage of 50 mg/kg body weight, freshly dissolved in cold citrate buffer (0.1 M, pH 4.5), after fasting for ten to twelve hours. For the first twenty-four hours following induction, animals were given 5% glucose solution in their drinking water to reduce early hypoglycemia. A digital glucometer was used to test fasting blood glucose by tail-vein sample 72 hours after introduction. Rats with fasting glucose levels more than 250 mg/dL were diagnosed with diabetes and included in further experiments.

Experimental Design

Animals were randomly assigned into six groups of three rats each. The grouping and treatment protocols were as follows:

- **Group A**: Normal control (distilled water + standard feed)
- **Group B**: Diabetic control (STZ-induced; no treatment)
- **Group C**: Diabetic + Glibenclamide (3 mg/kg, orally)
- **Group D**: Diabetic + Phenolic extract (200 mg/kg)
- **Group E**: Diabetic + Phenolic extract (400 mg/kg)
- **Group F**: Diabetic + Phenolic extract (600 mg/kg)

Treatments were administered once daily for 14 days via oral gavage. Body weight and fasting blood glucose were monitored at predetermined intervals. At the end of treatment, animals were fasted overnight and sacrificed under mild anesthesia for blood and organ collection.

Ethical Statement

Ethical approval for this study was obtained from the Research Ethics Committee of Enugu State University of Science and Technology (ESUT), Enugu, Nigeria, with approval number ESUT/REC/2025/049. All procedures were conducted in accordance with institutional and international guidelines for the care and use of experimental subjects.

Analysis of Lipid Profile

In a controlled laboratory environment, the lipid profile was ascertained using standardized enzymatic colorimetric techniques as outlined in [31]. sing the cholesterol oxidase–peroxidase aminoantipyrine (CHOD–PAP) method, which involves reacting serum samples with enzymatic reagents to create a quinoneimine chromogen and measuring absorbance spectrophotometrically at 500 nm against a reagent blank, the concentration of total cholesterol (TC) was determined. Following the enzymatic hydrolysis of triglycerides to glycerol, the glycerol-3-phosphate oxidase–peroxidase (GPO–PAP) technique was used to quantify triglycerides (TG), with absorbance measured at 500 nm. Following centrifugation and the precipitation of apoB-containing lipoproteins using a phosphotungstate–magnesium chloride reagent, high-density lipoprotein cholesterol (HDL-C) was measured using the same CHOD–PAP technique as for total cholesterol.

For fasting blood samples with triglycerides < 400 mg/dL, the Friedewald equation was used to compute low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) from the observed lipid fractions, as indicated in Equations 1 and 2, respectively.

$$\text{LDL-C} = \text{TC} - \text{HDL} - \text{C} - \left(\frac{TG}{5}\right) \quad (1)$$

$$\text{VLDL-C} = \frac{TG}{5} \quad (2)$$

Where: LDL-C is Low-Density Lipoprotein Cholesterol, TC is Total Cholesterol, HDL-C is High-Density Lipoprotein Cholesterol, TG = Triglycerides, VLDL-C = Very Low-Density Lipoprotein Cholesterol. All absorbance readings were taken using a calibrated UV–Visible spectrophotometer under standardized laboratory conditions to ensure analytical precision.

Analysis of Renal Function

Serum urea, creatinine, sodium, potassium, chloride, and bicarbonate were measured using recognized clinical chemistry techniques to assess renal function. An enzymatic urease-based technique was used to measure urea [32]. The kinetic Jaffé reaction was used to quantify creatinine [33]. The ion-selective electrode (ISE) technique, which enables direct potentiometric determination of ion activity in biological fluids in accordance with internationally standardized clinical chemistry protocols (IFCC/CLSI-recommended procedures for electrolyte analysis), was used to measure serum electrolytes (Na^+, K^+, and Cl^-). Every test was carried out in compliance with the manufacturer's calibration and quality control protocols.

Renal Biomarker Evaluation

Standard enzymatic and colorimetric clinical chemistry techniques were used to measure serum urea and creatinine

values in compliance with the procedure in [34]. The urease–GLDH technique, a standardized enzymatic test that is widely recognized as a reference method in clinical biochemistry and is detailed in the Tietz Textbook of Clinical Chemistry and Molecular Diagnostics (6th ed.), was used to detect urea. The kinetic Jaffé reaction, a traditional reference technique approved by the International Federation of Clinical Chemistry (IFCC) for regular laboratory usage, was used to assay creatinine.

Oxidative Stress Markers

In cold phosphate buffer (0.1 M, pH 7.4), liver and kidney tissues were homogenized. Using standard spectrophotometric techniques, antioxidant indices such as glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD) were assessed [35]. The thiobarbituric acid reactive substances test (TBARS) was used to quantify malondialdehyde (MDA), a marker of lipid peroxidation.

Statistical Analysis

The data were presented as mean ± SD. One-way ANOVA was used for statistical comparisons, and Tukey's test were used to identify group differences. Fasting blood glucose and other time-dependent variables were analyzed using repeated-measures ANOVA. $P < 0.05$ was considered significant. IBM SPSS Statistics (version 23) was used for all statistical analysis.

RESULTS

Effects of phenolic extract of *P. nitida* on renal function parameters of diabetic rats

Figure 1-6 illustrate the impact of *P. nitida* phenolic extract on renal function markers in diabetic rats. Renal impairment was confirmed by the diabetes control group's considerably higher urea levels ($p < 0.05$) as compared to the normal control group (Fig. 1). The 600 mg/kg phenolic extract had the most impact, even when compared to glibenclamide, and the difference with the normal control was not significant ($P>0.05$). All treatment groups showed substantial decreases in urea compared to the diabetic control ($P<0.05$). Glibenclamide and all phenol-treated groups significantly reduced creatinine levels ($P<0.05$), with the Phenol (400 and 600 mg/kg) doses statistically indistinguishable from the normal control ($P>0.05$). In terms of creatinine levels, the diabetic control was significantly higher than the normal rats ($P<0.05$) (Fig. 2).

Electrolyte balance was also significantly altered by diabetes. Serum sodium, potassium, and chloride levels were significantly reduced in the diabetic control group, showing clear separation from the normal control ($p < 0.05$). When compared to the diabetic control, treatment with glibenclamide and all phenolic extract dosages significantly improved Na^+, K^+, and Cl^- levels ($P<0.05$). Phenol (600 mg/kg) outperformed other treatment groups, including glibenclamide, although there was no significant difference between them ($P>0.05$). Sodium concentrations in the treated groups exhibited a partial but considerable recovery ($P<0.05$). All treatment groups showed a substantial dose-dependent improvement in potassium levels ($P<0.05$), with values significantly higher than glibenclamide and closer to normal rats at the 400 and 600 mg/kg phenol dosages.

Chloride levels were considerably restored in all treatment groups ($P<0.05$), with 600 mg/kg producing the most therapeutic effect, compared to glibenclamide, and the difference with the normal control was negligible ($P>0.05$). There was little difference in serum bicarbonate (HCO_3^-) across the groups. There was a minor but significant difference ($P<0.05$) between the diabetes control group and the normal control. All treated groups showed a substantial improvement when compared to the diabetes control ($P<0.05$), although there was no significant recovery ($p >0.05$) across the treatment groups to the normal control.

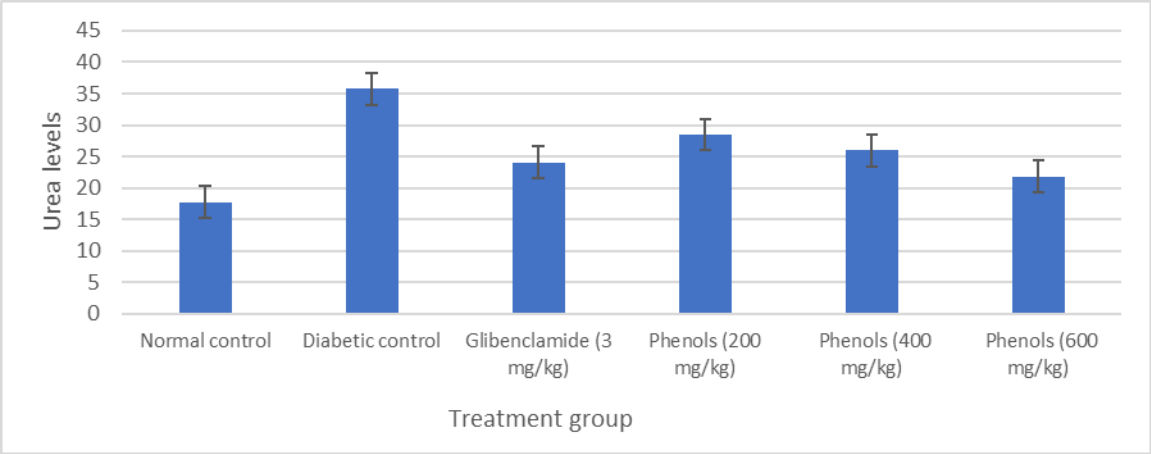

Figure 1: Effect of *Picralima nitida* phenolic leaf extract on urea level of diabetic Wistar rats

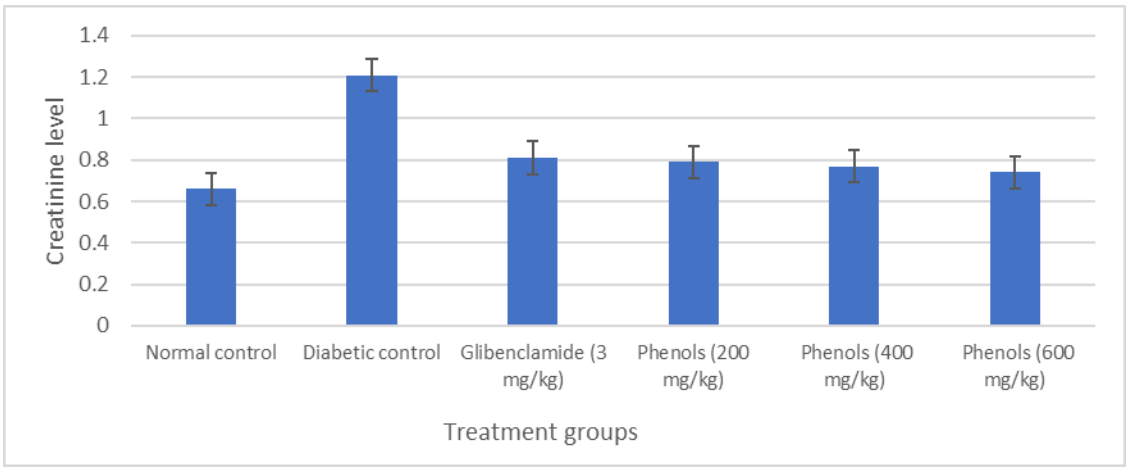

Figure 2: Effect of *P. nitida* phenolic leaf extract on creatinine level of diabetic Wistar rats

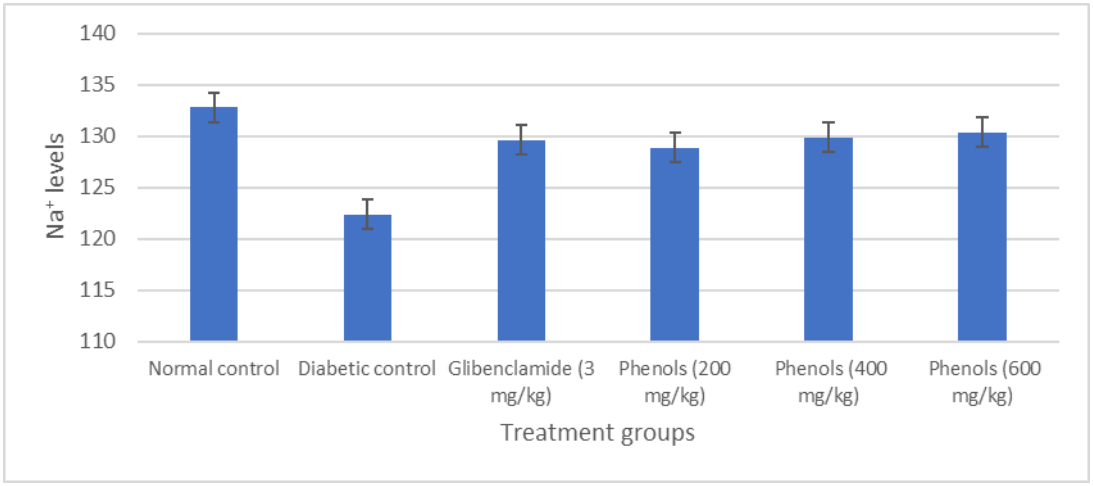

Figure 3: Effect of *P. nitida* phenolic leaf extract on Na^+ level of diabetic Wistar rats

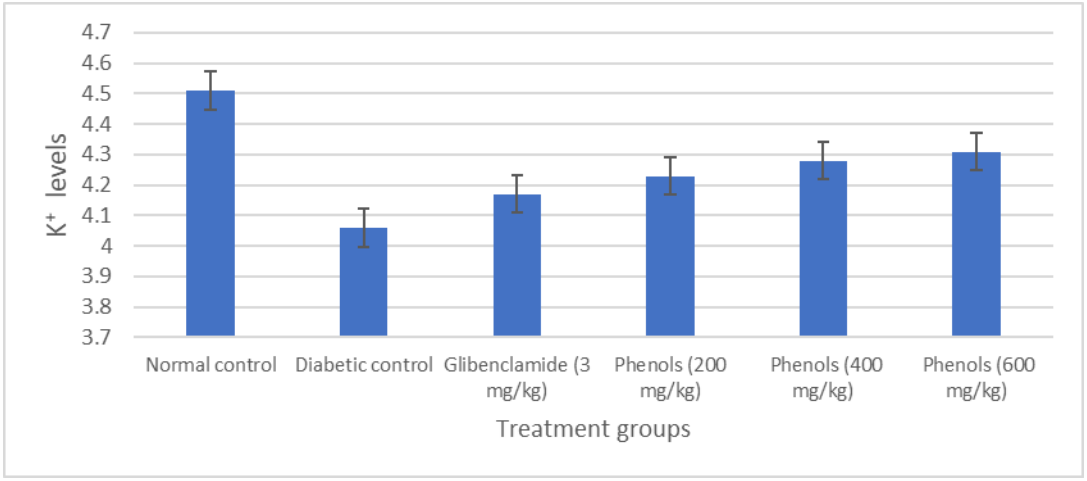

Figure 4: Effect of *P. nitida* phenolic leaf extract on K^+ level of diabetic Wistar rats

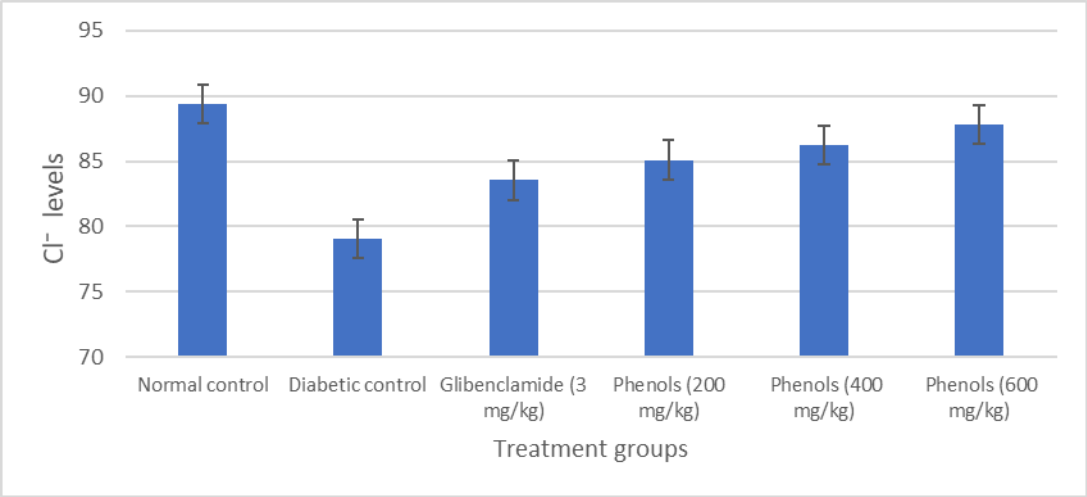

Figure 5: Effect of *P. nitida* phenolic leaf extract on Cl^- level of diabetic Wistar rats

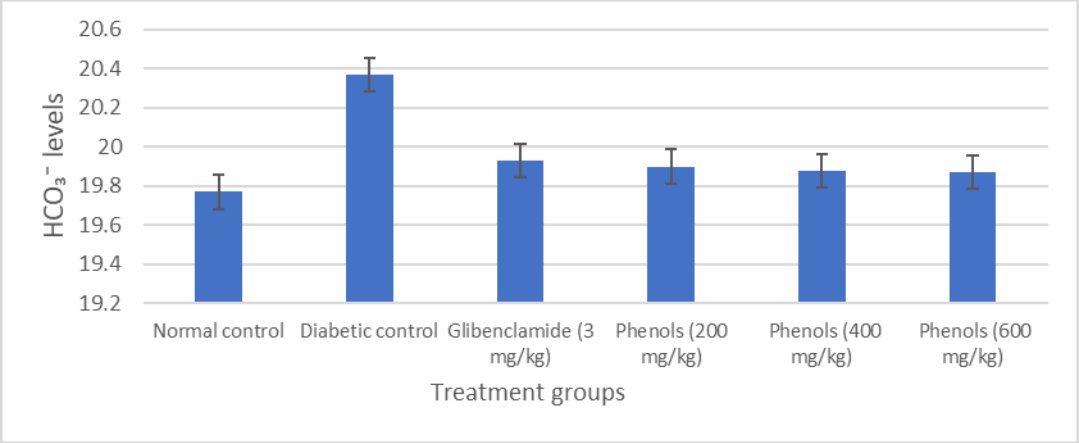

Figure 6: Effect of *P. nitida* phenolic leaf extract on HCO_3^- level of diabetic Wistar rats

Effects of phenolic extract on lipid profile parameters in diabetic rats

Figures. 7-11 showed the impact of the phenolic extract on the lipid profile in diabetic Wistar rats. The diabetic group had considerably higher total cholesterol than the normal control group (MD = 15.967, $p < 0.001$). In comparison to diabetic controls, administration of glibenclamide and phenolic extract at 200, 400, and 600 mg/kg substantially decreased total cholesterol (mean differences = 11.30, 9.30, 8.50, and 8.50, respectively, all $p < 0.01$). Although levels did not fully recover to normal, phenol 400 and 600 mg/kg both had the greatest restorative effect (Fig. 7). The diabetes control group's HDL-C levels were considerably lower ($P<0.05$), whereas the treatment groups' improvements were not statistically significant when compared to the diabetic and normal control groups ($P<0.05$).

In comparison to normal controls, diabetic controls had significantly higher triglyceride (TAG) levels (MD = 23.467, $p < 0.001$). At all concentration, phenolic extract treatments showed a substantial decrease ($P<0.05$), especially at 200 mg/kg, where they considerably outperformed the Glibenclamide ($P<0.05$). VLDL-C levels were considerably higher in the diabetic controls compared to normal, and there were significant reductions with glibenclamide and phenolic therapies ($P<0.05$). Glibenclamide considerably outperformed all phenol extract dosages ($P<0.05$), with the phenolic extract group performing best at 400 and 600 mg/kg. LDL-C levels were significantly higher in diabetic controls relative to normal controls ($F(5,12) = 34.686$, $p < 0.001$, mean difference = 15.170, $p < 0.001$), and treatment with phenolic extract at all tested doses effectively lowered LDL-C (mean differences ranging from 6.973 to 8.197, all $p < 0.05$), comparable to glibenclamide effects.

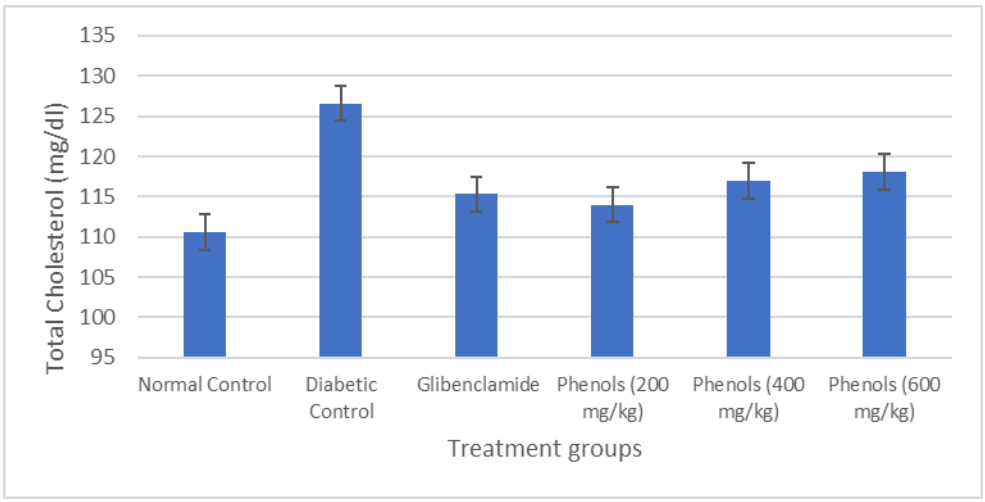

Figure 7: Effect of *Picralima nitida* phenolic leaf extract on total cholesterol level of diabetic Wistar rats

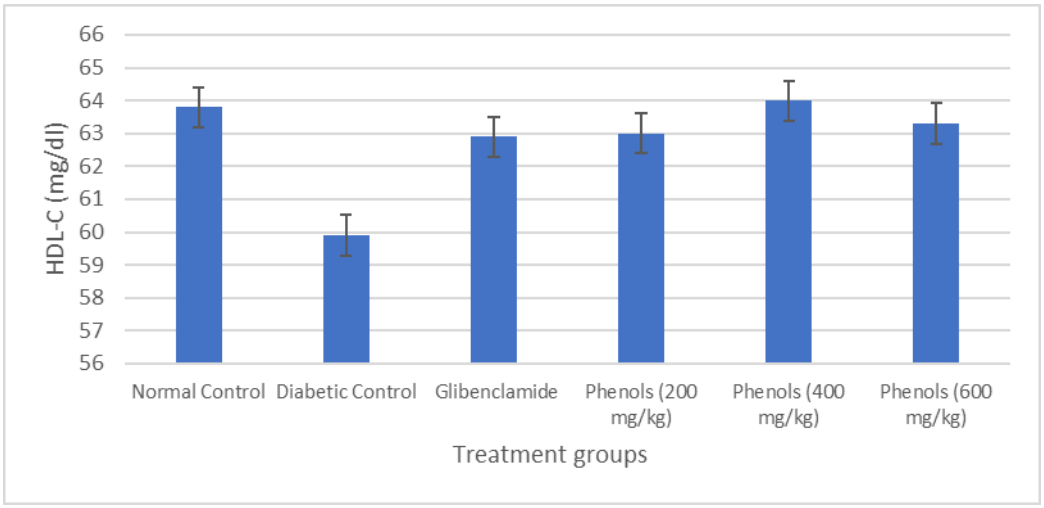

Figure 8: Effect of *Picralima nitida* phenolic leaf extract on HDL-C level of diabetic Wistar rats

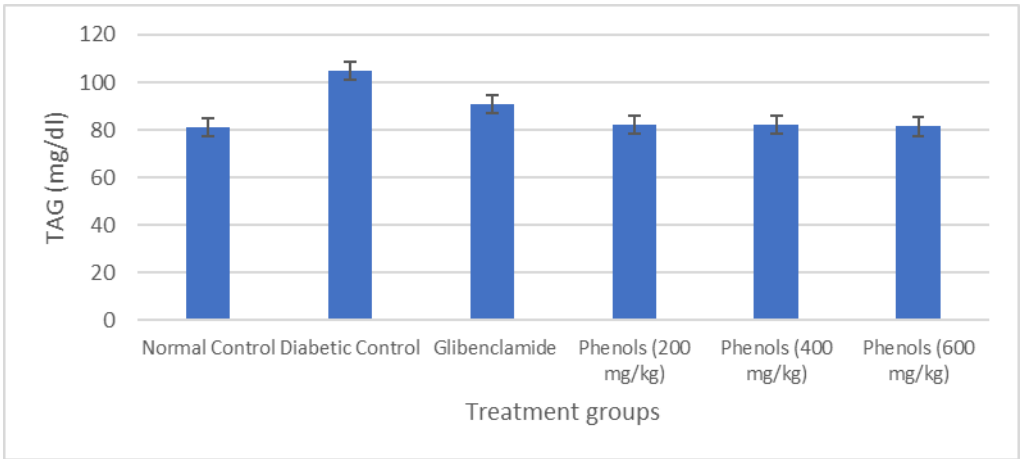

Figure 9: Effect of *Picralima nitida* phenolic leaf extract on TAG level of diabetic Wistar rats

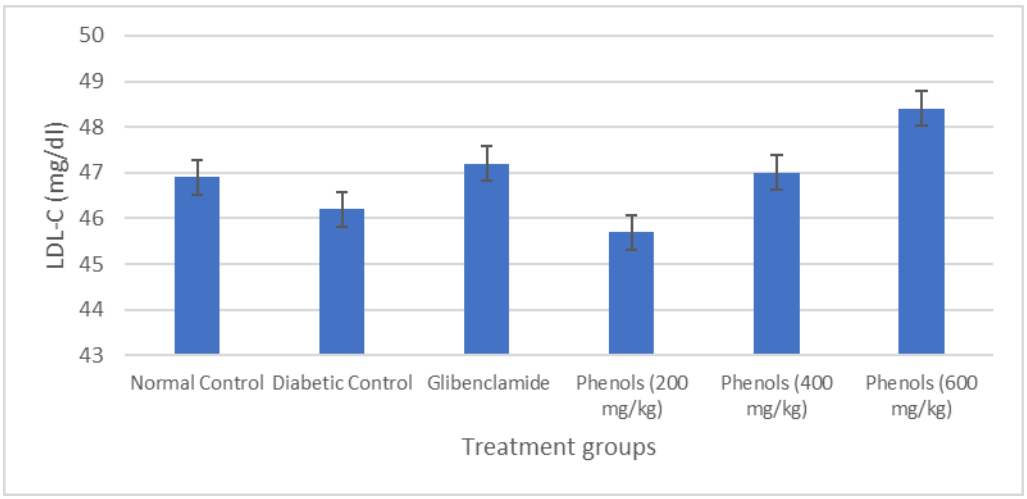

Figure 10: Effect of *Picralima nitida* phenolic leaf extract on LDL-C level of diabetic Wistar rats

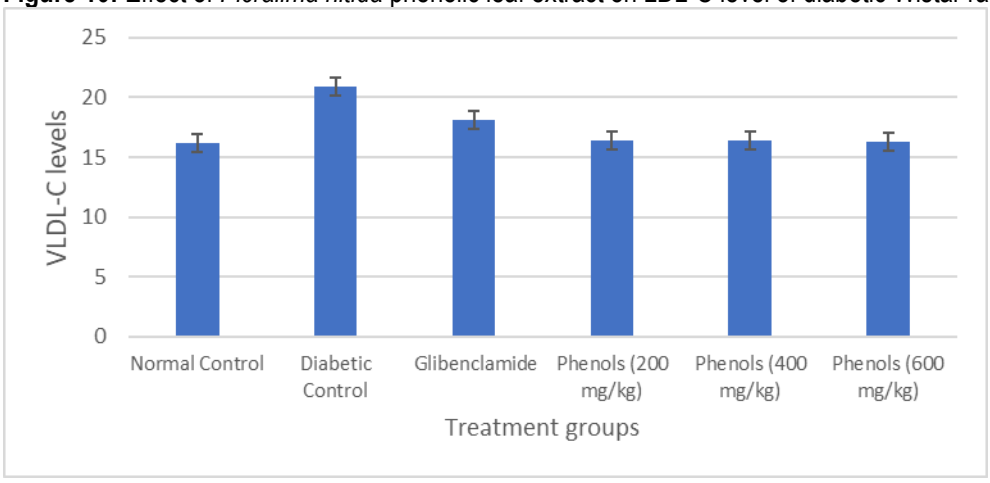

Figure 11: Effect of *Picralima nitida* phenolic leaf extract on VLDL-C level of diabetic Wistar rats

Effects of phenolic extract of *P. nitida* on serum antioxidant parameters in diabetic rats

Figures 12-14 shows the effect of *P. nitida* phenolic extract affected blood antioxidant markers in diabetic rats. GPx activity varied considerably between groups ($F(5,12) = 82.688$, $p < 0.05$), with GPx levels in diabetic control rats being significantly lower than in normal controls ($P<0.05$). The results showed that glibenclamide therapy increased GPx activity in comparison to diabetic controls, however this difference was not statistically significant ($P<0.05$). Phenolic extract significantly improved all treatment groups ($P<0.05$), and levels reverted to normal at 200 and 400 mg/kg with no significant difference from the normal control ($P>0.05$) (Fig. 12). Group differences in SOD activity were significant ($F(5,12) = 170.109$, $p < 0.05$).

When compared to normal controls, diabetic control rats showed substantially lower SOD activity ($P<0.05$). Glibenclamide administration had no significant effect on SOD activity ($P>0.05$), but phenolic extract (200, 400, and 600 mg/kg) significantly increased SOD activity in comparison to diabetic controls ($P<0.05$), with the 400 and 600 mg/kg phenol doses exceeding the normal controls with no significant difference between them ($p>0.05$). Additionally, there was a significant difference in CAT activity across groups ($F(5,12) = 32.388$, $p<0.05$). Glibenclamide considerably increased the reduced CAT activity in diabetic rats (p 0.05), although it was still significantly different from the normal control ($P<0.05$). 200, 400, and 600 mg/kg of phenolic treatments ($p<0.05$). There was no significant alteration in the CAT activity levels across the treatment groups. When compared to normal controls, diabetic controls had considerably higher MDA levels ($p<0.05$). MDA concentrations were significantly decreased by treatment with glibenclamide and phenolic extract (200, 400, and 600 mg/kg) in a dose-dependent manner; the 400 mg/kg phenol dosages produced reductions similar to normal controls ($p<0.05$), with no significant difference ($P>0.05$).

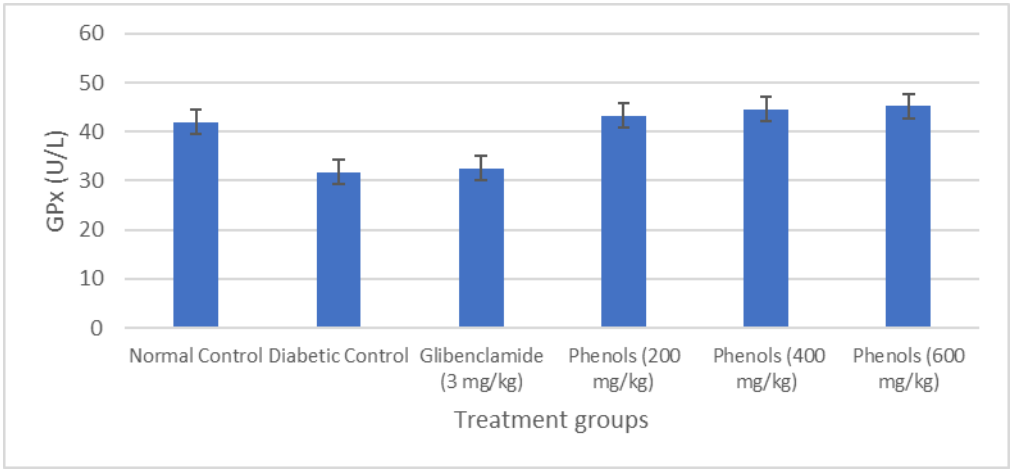

Figure 12: Effect of *Picralima nitida* phenolic leaf extract on GPx level of diabetic Wistar rats

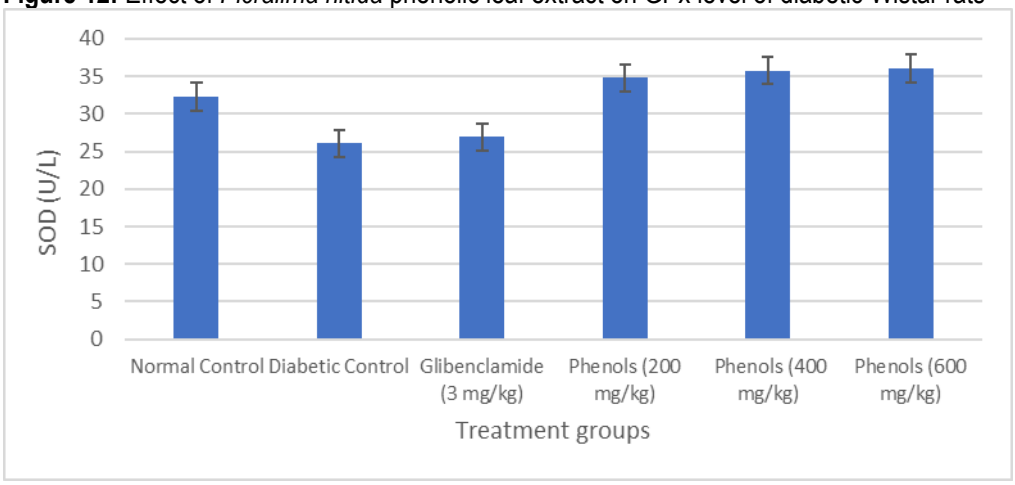

Figure 13: Effect of *P. nitida* phenolic leaf extract on SOD level of diabetic Wistar rats

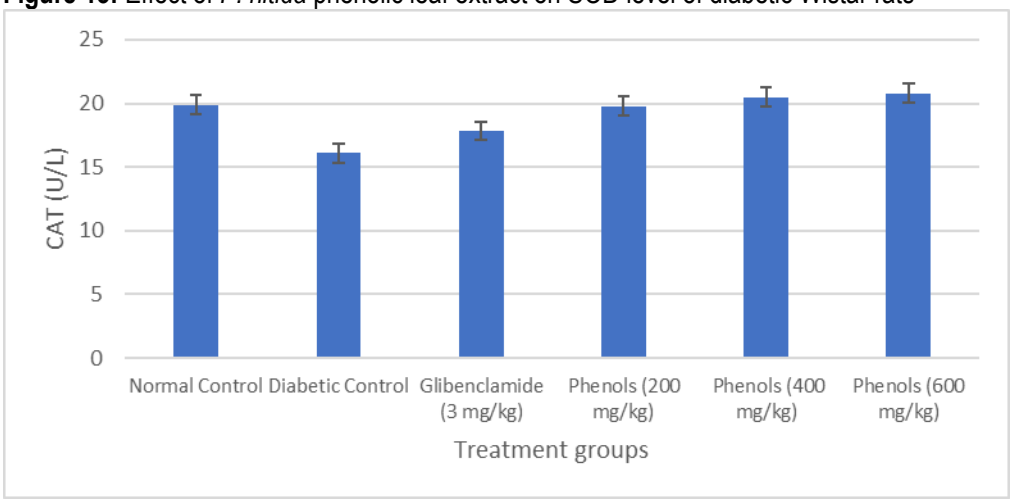

Figure 14: Effect of *P. nitida* phenolic leaf extract on CAT level of diabetic Wistar rats

DISCUSSION

The liver is essential for maintaining stable blood glucose levels and promoting general energy balance [11,36]. it plays a key role in lipid management, which involves absorbing fats, breaking them down, and repackaging them into forms that may be transported to other bodily tissues [37]. Typically, these functions rely on appropriate insulin signaling. Non-alcoholic fatty liver disease (NAFLD) develops in people with type 2 diabetes as a result of these metabolic pathways being disturbed as the body becomes less sensitive to insulin [11]. Significant dyslipidemia, elevated total cholesterol, triglycerides, VLDL-C, LDL-C, and decreased HDL-C were all caused by diabetes in this research. This pattern is linked to rapid atherogenesis and is commonly seen in insulin resistance and deficient situations. These results are consistent with those of De. Campos *Picralima nitida*. [38] who discovered that *P. nitida* seed extract improved HDL, decreased oxidative stress indicators (MDA), increased antioxidant enzymes (SOD, CAT), and decreased total cholesterol, triglycerides, and LDL.

When compared to normal controls, the diabetes control group's serum urea and creatinine levels were significantly higher, suggesting reduced renal excretory function that is consistent with early diabetic renal impairment. Acute or chronic kidney disease is frequently associated with elevated urea and creatinine, which are recognized indicators of decreased glomerular filtration [39]. The renoprotective action of the extract, as described in [39], is supported by our finding that glibenclamide and all dosages of the phenolic extract considerably decreased both urea and creatinine, with the 600 mg/kg dose returning values to levels not statistically different from normal. Through oxidative stress, advanced glycation end products, and inflammatory signaling, hyperglycemia causes glomerular hyperfiltration, mesangial expansion, and tubular injury. These processes raise serum urea and creatinine and, if persistent, lead to diabetic nephropathy and chronic kidney disease. According to [40], *P. nitida* phenolics' capacity to lower these indicators points to the maintenance of filtration function and the reduction of oxidative and inflammatory damage to renal parenchyma.

Following treatment with phenolic extract, improvements in Na^+, K^+, and Cl^- are likely due to reduced tubular oxidative damage, enhanced tubular reabsorption/secretion, and reduced osmotic diuresis (glycaemic control). This outcome is similar to that of Radajewska *Picralima nitida*. [41]. Significantly lower SOD, GPx, and CAT activity as well as noticeably higher MDA levels were seen in the diabetic control rats, suggesting enhanced lipid peroxidation and compromised antioxidant defense as reported in [42]. This is consistent with the results of Olufunsho *Picralima nitida*. [39] who found that phenolic therapy reduced MDA toward baseline and restored antioxidant enzyme activity in a dose-dependent manner. Interestingly, GPx, SOD, and CAT returned to statistically indistinguishable levels after several phenolic dosages. These results are in line with mechanistic and meta-analytic studies showing that phenolic compounds have antioxidant qualities that reduce lipid peroxidation and restore enzyme functioning, and that type 2 diabetes is associated with increased oxidative stress [43]. By scavenging reactive oxygen species, boosting endogenous antioxidant defenses, and reducing lipid peroxidation, phenolic compounds shield cellular and microvascular structures, including renal glomeruli and tubules.

CONCLUSION

This study showed that phenolic extract of *P. nitida* significantly reduced total cholesterol, triglycerides, LDL-C, and VLDL-C together with a rise in HDL-C, indicating robust antihyperlipidemic action. Antioxidant assessments also reveal reduced lipid peroxidation and increased SOD, CAT, and GPx activity, suggesting that oxidative stress has been lessened. Overall, this work has demonstrated the ability of *P. nitida* phenolic extract to reduce oxidative, metabolic, and renal deficiencies.

ACKNOWLEDGMENT

The Department of Biotechnology and the Laboratory Animal Facility at Enugu State University of Technology, Nigeria, are greatly appreciated by the authors for providing the necessary infrastructure and technical assistance needed to carry out this study successfully.

AUTHORS' CONTRIBUTION

The study was conceived and designed by J.C.E., who also carried out the experiments, evaluated the data, and wrote the report. I.O. oversaw the research, helped design the study, and critically edited the text to provide significant intellectual value. The final text was reviewed and approved by both authors.

CONFLICT OF INTEREST

The author declares that there are no known financial or personal conflicts of interest that could have influenced the work reported in this manuscript.

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