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RESEARCH ARTICLE

IN-VITRO AND *IN-VIVO* STUDY OF TURMERIC-GINGER SYNERGY IN DIABETIC OXIDATIVE STRESS MANAGEMENT

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Abstract

Background and aim: Oxidative stress is a major factor in both the progression and the adverse effects of diabetes mellitus. Natural Bioactive substances like curcumin (derived from turmeric) and gingerol (from ginger) possess antioxidant properties that may offer therapeutic benefits by enhancing the body's defense mechanisms against oxidative damage.
This study investigates the synergistic antioxidant effects of co-administered curcumin and gingerol in managing oxidative stress associated with diabetes mellitus.
Method: Oxidative stress biomarkers including superoxide dismutase (SOD), malondialdehyde (MDA), catalase, and glutathione were evaluated in experimental rat groups treated with varying doses of curcumin and gingerol. Additionally, *in vitro* antioxidant activities of turmeric and ginger extracts were assessed using DPPH and FRAP assays.
Results: The combination of 400 mg/kg curcumin and gingerol significantly increased SOD (24.49±6.85 IU/g protein) and catalase (19.02±1.76 kU/g protein) activities ($p<0.05$) compared to other groups. Glutathione levels also rose notably (5.31±1.26 µg/L), indicating enhanced endogenous antioxidant capacity. Although MDA levels (75.05±24.76 nmol/g protein) were higher than in lower dose groups, they remained lower than in the 200 mg/kg group (135.35±8.51 nmol/g protein), suggesting a dose dependent response. *In vitro* assays confirmed strong radical scavenging and reducing activities, with turmeric slightly outperforming in FRAP, while ginger matched in DPPH scavenging.
Conclusion: Co-administration of curcumin and gingerol enhances antioxidant defences more effectively than individual treatments or metformin. This synergy presents a potential adjunct therapy for mitigating oxidative damage in diabetes and merits further clinical investigation.
Keywords: Antioxidant, curcumin, diabetes mellitus, gingerol, oxidative stress, synergistic effect.

INTRODUCTION

Diabetes mellitus is a long-term, progressive metabolic condition marked by high blood glucose levels, insulin resistance, and increased oxidative stress, all of which contribute to complications such as vascular damage, neuropathy, and organ dysfunction. Oxidative stress results from an imbalance between the production of reactive oxygen species (ROS) and the body's intrinsic antioxidant defense mechanisms, including superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH)[1]. In individuals with type 2 diabetes mellitus (T2DM), there is often an increase in lipid peroxidation indicators like malondialdehyde (MDA), accompanied by a reduction in the activity of key antioxidant enzymes such as SOD and CAT[2].

Against this backdrop, there is growing interest in plant-derived nutraceuticals as safer adjunct options to conventional therapy. Curcumin the principal curcuminoid in turmeric (Curcuma longa) has been clinically demonstrated to enhance serum total antioxidant capacity and SOD activity, while significantly reducing MDA levels in individuals with T2DM[2].

Ginger (*Zingiber officinale*) contains bioactive phytochemicals particularly gingerols and shogaols that exhibit Ginger exhibits strong antioxidant and anti-inflammatory properties. In studies involving diabetic animal models, supplementation with ginger at doses between 200 and 400 mg/kg per day has shown beneficial effects elevated SOD, CAT, and glutathione levels, while reducing MDA in liver, kidney, and neural tissues. The mechanistic underpinnings involve activation of Nrf2-mediated antioxidant pathways, suppression of NF-κB signaling, and regulation of lipid metabolism and glucose utilization.

While research has traditionally focused on single herb interventions, emerging data support synergistic anti-inflammatory and antioxidant interactions when turmeric and ginger are combined. *In vitro* studies using RAW 264.7 macrophages showed that combined turmeric-ginger (GT) extracts inhibited NF-κB translocation and miR-155-5p expression more effectively than either extract alone (e.g., reduced LPS-induced pro-inflammatory cytokines)[3].

Another *in vitro* investigation determined that mixtures of turmeric and ginger extract in optimized ratios (e.g., 5:2 w/w) significantly enhanced Nrf2 and HO-1 protein expression, leading to greater suppression Such as nitric oxide, tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) production than single extracts[3].

However, preclinical evidence evaluating turmeric–ginger synergy in diabetic oxidative stress models remains limited. Although curcumin or ginger alone have shown improvements in SOD, CAT, GSH, and reductions in MDA in diabetic rodents[2], little *in vivo* work has tested combination dosing to define potential synergistic antioxidant effects. One animal study combining aqueous ginger extract and curcumin reported superior reductions in hyperglycemia, HbA1c, dyslipidemia, and oxidative stress markers compared to either component alone but detailed dose–response data and multiple oxidative biomarkers were not systematically evaluated[4].

In this context, a comprehensive experimental design that integrates both *in vitro* antioxidant assays (e.g., DPPH, FRAP) and *in vivo* diabetic models evaluating biomarkers such as SOD, CAT, GSH, and MDA across varying combination doses are crucial. *In vitro* methods specifically highlight the direct free radical-scavenging and reducing capacities of turmeric and ginger extracts, while *in vivo* models (e.g., alloxan-induced diabetic rats) assess functional outcomes on oxidative biomarkers and tissue integrity (Table 1). Integrating both approaches fills a critical gap by demonstrating whether higher combination doses effectively enhance endogenous enzyme defences and suppress lipid peroxidation, potentially outperforming standard treatment such as metformin[5].

The current study seeks to assess the dose-dependent effects of curcumin and gingerol on oxidative stress and related biochemical markers in diabetic models *gingerol* combinations on key oxidative stress biomarkers: SOD, CAT, GSH, and MDA in Alloxan induced diabetic rat models, alongside *in vitro* assays (DPPH, FRAP) assessing radical scavenging and reducing capacity. We hypothesize that higher-dose combinations will synergistically enhance antioxidant enzyme activities and mitigate lipid peroxidation more effectively than either agent alone or metformin, and that *in vitro* assays will support these findings by demonstrating superior radical-scavenging and reducing power.

This integrated investigation spanning *in vitro* and *in vivo* modalities is poised to deliver deeper insight into turmeric–ginger synergy, informing future clinical translation as a complementary antioxidant strategy in diabetes management.

METHODS

Collection of plant material

The fresh rhizomes of turmeric (*Curcumalonga*) and ginger (*Zingiber officinale*) was harvested from National Root Crops Research Institute, Umudike, Abia State, Nigeria. The plant materials, the rhizomes were carefully washed under running tap water to eliminate soil and surface contaminants, then rinsed with distilled water. They were cut into smaller segments to enhance the drying process and subsequently air-dried at ambient temperature (25–28°C) for 10–14 days until a constant weight was achieved. Once fully dried, the samples were finely ground using a mechanical grinder, weighed and stored in airtight containers under at room temperature until they were extracted.

Plant Extraction

The authenticated plant materials were brought to the Biochemistry Department laboratory and air-dried for two weeks. After drying, the samples were ground into a fine powder using a Warring blender (Quilink QBL-20 L40 model). Methanolic extraction was performed following the method described by Achuba[5]. Specifically, 500 g of each powdered sample (turmeric and ginger) was soaked in 80% methanol and allowed to undergo fermentation for 72 hours. After the fermentation period, the mixtures were filtered sequentially using cotton wool and Whatman filter paper. The filtrates were then concentrated using a rotary evaporator and further dried in a water bath to yield a crude extract in powdered form.

Study Protocol

The study protocol adhered to the guidelines and declarations of Animal Research Ethics[6] and the World Medical Association[7] concerning the use of animals in biomedical research. It also complied with the animal rights regulations of Michael Okpara University of Agriculture, Umudike, Abia State.

Preparation of Extract for Animal Administration

A stock solution of the rhizome extract was prepared by dissolving 2 g of the crude methanol extract in 20 ml of water, resulting in a concentration of 100 mg/ml. Doses were administered individually based on the animals' body weight (mg/kg).

Animal Acclimatization

Twenty healthy male albino rats, weighing between 140 and 190 g, were obtained from the animal house of Dr. Daniel. The animals were housed in the Biochemistry Department's animal facility at Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria. They were allowed to acclimate to the new environment and kept in well-constructed stainless steel cages (30 x

40 x 80 cm) with wire mesh tops to ensure adequate ventilation and airflow. The cages were designed to be free of sharp edges and protrusions to prevent injuries or accidental entrapment of the animals' limbs.

Experimental Design

After acclimatization 36 healthy mature rats were selected for further experimentation. The animals were divided into six groups, each consisting of six rats. The groups were organized as follows: Six groups (n=6 each):

1. Normal control (vehicle);
2. Diabetic control (no treatment);
3. Diabetes + Metformin, 100 mg/kg
4. Diabetic + turmeric, 100 mg/kg + ginger, 100 mg/kg combined extract
5. Diabetic + turmeric, 200 mg/kg + ginger, 200 mg/kg combined extract
6. Diabetic + turmeric, 400 mg/kg + ginger, 400 mg/kg combined extract

Treatments were administered orally daily for 21 days. At study end, rats were euthanized; blood and tissues were collected for biochemical assays.

Biochemical Assays

Serum and tissue homogenates were analyzed for activities of antioxidant enzymes such as superoxide dismutase (SOD)[8], catalase (CAT)[9], and glutathione peroxidase (GPx)[10] and for malondialdehyde (MDA) concentration as a marker of lipid peroxidation[11], using standard colorimetric methods and spectrophotometry.

***In vitro* DPPH radical scavenging assay:**

The antioxidant activity of the individual and combined extracts was assessed using the 2, 2-diphenyl-1-picrylhydrazyl (DPPH) radical scavenging method, following the procedure outlined by reference 12. In brief, a 0.1 mM methanolic DPPH solution was combined with different concentrations of turmeric extract, ginger extract, and their mixture. The samples were incubated in the dark at room temperature for 30 minutes, after which absorbance was recorded at 517 nm using a UV-Vis spectrophotometer. The percentage of DPPH radical scavenging was calculated, and IC_{50} values were determined to evaluate the antioxidant strength of each extract.

Statistical Analysis

Results are expressed as mean ± standard deviation (SD). Statistical significance was assessed using one-way ANOVA followed by Duncan's post hoc test, with *p*-values less than 0.05 considered significant.

RESULTS

This figure illustrates the antioxidant activity of turmeric, measured based on its capacity to neutralize DPPH (2, 2-diphenyl-1-picrylhydrazyl) free radicals. Greater scavenging activity indicates stronger antioxidant potential.

Table 1: Effect of curcumin and gingerol combinations on oxidative stress biomarkers in experimental groups.

Group	SOD (IU/g protein)	MDA (nano moles/g protein)	Catalase (kU/g protein)	Glutathione (µg/L)
Normal control	5.95±0.83^{c}	33.23±0.62^{c}	5.41±0.89^{bc}	2.22±0.33^{b}
Negative control	5.73±0.43^{c}	37.65±2.14^{c}	3.51±0.83^{c}	2.05±0.12^{b}
Metformin, 100 mg/kg	7.75±0.39^{c}	36.18±0.77^{c}	4.83±1.59^{bc}	2.21±0.33^{b}
Curcumin, 100 mg/kg + Gingerol 100 mg/kg	9.48±0.96^{bc}	27.63±0.76^{c}	4.69±0.42^{bc}	1.84±0.16^{b}
Curcumin, 200 mg/kg + Gingerol 200 mg/kg	29.80±0.19^{a}	135.35±8.51^{a}	16.90±2.59^{a}	1.35±0.29^{b}
Curcumin, 400 mg/kg + Gingerol 400 mg/kg	24.49±6.85^{a}	75.05±24.76^{b}	19.02±1.76^{a}	5.31±1.26^{a}

The results likely show a concentration-dependent increase in radical scavenging. This figure shows the Free radical scavenging capacity of ginger extracts assessed by DPPH assay. Similar to turmeric, higher scavenging percentages reflect stronger antioxidant properties. The data can be used to compare the antioxidant strength of ginger with that of turmeric. This figure presents the FRAP assay results for turmeric. FRAP measures the extract's ability to reduce Fe^{3+} to Fe^{2+}, indicating reducing power. A higher FRAP value indicates a stronger antioxidant capacity.

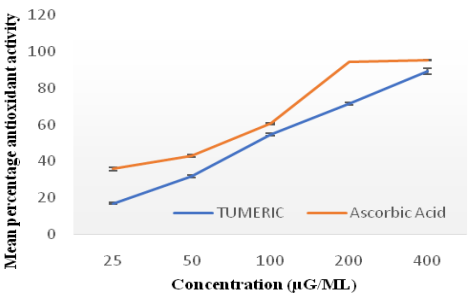

Figure 1: DPPH radical scavenging activity of turmeric extracts.

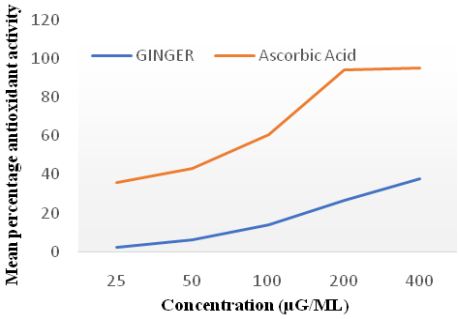

Figure 2: DPPH radical scavenging activity of ginger extracts.

This figure displays the FRAP values for ginger, assessing its antioxidant capacity through ferric ion reduction. Comparing this with turmeric helps evaluate which has stronger reducing (antioxidant) properties. FRAP of Ginger.

DISCUSSION

The MDA reduction, although not reaching levels in normal control, indicates partial mitigation of lipid

peroxidation but at high dose, the MDA offset still remained above baseline, implying potential oxidative challenges or dose-linked pro-oxidant effects that warrant closer dose optimization. This finding concurs with the research of He *et al.*[13], and Hamdy *et al.*[14], who reported similar dose-dependent antioxidant responses and cautioned about the biphasic effects of curcumin and related phytochemicals on oxidative stress markers. The marked increase in glutathione, often depleted in diabetic conditions, suggests restored redox buffering capacity, consistent with Nrf2-mediated upregulation of antioxidant genes observed in curcumin and gingerol treatment studies[13-15].

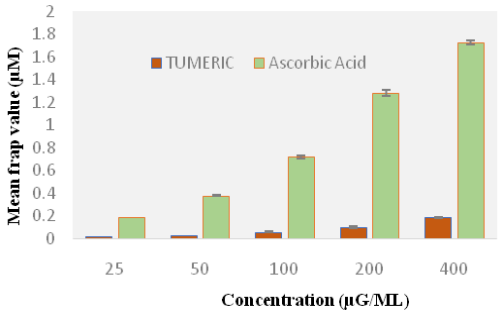

Figure 3: Ferric reducing antioxidant power of turmeric extracts.

The combination's ability to outperform metformin in boosting endogenous defences (SOD, CAT, GSH) highlights its promise as an adjunct or complementary strategy, although clinical relevance remains to be established. This is supported by research demonstrating curcumin's potent Stimulation of the Nrf2–ARE signaling cascade, leading to the induction of HO-1, NQO-1, and enzymes such as catalase and SOD[16,17]. Similarly, gingerol has been reported to stimulate Nrf2 signaling and antioxidant enzyme expression in diabetic models[18,19].

The high dose (400 mg/kg) produced the strongest antioxidant response, but also a relatively elevated MDA level compared to normal controls, indicating the need to identify the optimal therapeutic window. Such biphasic dose responses have been documented in phytochemical research[13,14], highlighting the importance of balancing efficacy with potential pro-oxidant effects at supraphysiologic doses. Future studies should incorporate histopathological assessments and explore markers of tissue oxidative damage or repair. Additionally, evaluating gene expression (e.g., Nrf2, HO-1, NF-κB) would elucidate mechanistic pathways of synergy beyond enzyme activation[20].

The combination of curcumin and gingerol at high doses (curcumin 400 mg/kg + gingerol 400 mg/kg) produced substantial improvements in oxidative stress biomarkers: SOD activity increased to 24.49 ± 6.85 IU/g protein, catalase to 19.02 ± 1.76 kU/g protein, and glutathione reached 5.31 ± 1.26 µg/L, significantly higher than both negative controls and metformin groups ($p < 0.05$). MDA, an index of lipid peroxidation, was also reduced compared to the 200 mg/kg group (75.05 vs. 135.35 nmol/g protein), albeit not to baseline levels. These findings suggest a powerful induction of endogenous antioxidant defences in a dose-dependent manner (Table 1), consistent with previous reports on curcumin's dose-dependent antioxidant enzyme induction[13,16].

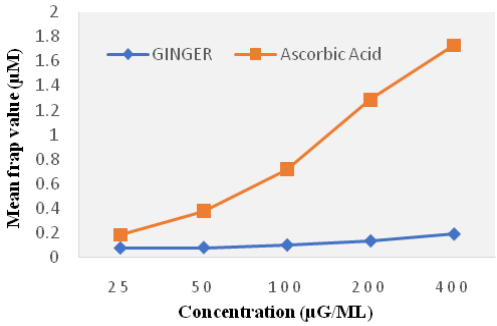

Figure 4: Ferric reducing antioxidant power of ginger extracts.

In particular, the high-dose combination elevated SOD and catalase far above metformin-treated and normal control levels, highlighting its antioxidant enzyme induction potential. This aligns with prior evidence Curcumin stimulates the Keap1–Nrf2–ARE pathway, enhancing the expression of antioxidant genes like HO-1, SOD, CAT, and glutathione peroxidase in diabetic models[16]. Similarly, gingerol-based interventions in diabetic rat studies have demonstrated elevated SOD, CAT, and glutathione levels, alongside reduced MDA in brain and renal tissues, reflecting systemic antioxidant protection[18,19].

Both *in vitro* assays (Figure 1 - Figure 4) supporting concentration-dependent radical scavenging activity (DPPH) and ferric reducing antioxidant power (FRAP) for turmeric and ginger extracts reinforce the plausibility of their *in vivo* synergy. Such antioxidant capacity has been documented for both turmeric and ginger extracts individually, with several studies noting enhanced scavenging activities correlating with improved oxidative stress biomarkers in animal models[21,22].

This study is novel in demonstrating dose-dependent antioxidant synergy of combined curcumin and gingerol, with 400 mg/kg outperforming lower doses and metformin in antioxidant enzyme activity. Biomarkers evaluate multiple Oxidative stress indicators such as SOD, CAT, GSH, and MDA systematically in a combination-treated diabetic model, extending beyond single-agent studies that typically measure only one or two biomarkers[13,14]. Including metformin as a standard comparator allows direct assessment of how herbal synergy measures against established pharmaceutical therapy[16]. Correlating *in vitro* radical-scavenging potency with *in vivo* enzymatic and molecular outcomes provides mechanistic insight into systemic antioxidant modulation[21,23].

Limitation of the study

Although antioxidant enzyme activities and oxidative stress markers were evaluated, the study did not extensively investigate the molecular signaling pathways involved in the turmeric-ginger synergy. The antioxidant activities observed *in vitro* (e.g., DPPH and FRAP assays) may not fully represent the complex interactions and metabolism of these compounds in living organisms.

CONCLUSION

This study contributes significantly by demonstrating, for the first time in a systematic manner, the dose-responsive synergistic antioxidant potential of curcumin and gingerol combination in diabetic oxidative stress management. The findings offer mechanistic insights grounded in robust *in vitro*/*in vivo* correlation and establish groundwork for future translational research.

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AUTHORS' CONTRIBUTIONS

Alaebo PO: conceptualization, supervision, writing the original draft. **Egbuonu ACC:** supervision, methodology, writing, review. **Okpara PU:** investigation, editing. **Njoku JC:** investigation, formal analysis, data collection. **Nwede CA:** Investigation, Resources, Animal Handling. **Abalihe CN:** laboratory analysis, validation. **Ugboaja TC:** data collection, investigation. **Chukwu LC:** writing, review, editing. **Chukwuka EW:** literature review. **Uchegbusi JC:** software, data entry. **Ataka KI:** literature review. Final manuscript was checked and approved by all authors.

DATA AVAILABILITY

Data will be made available on request.

CONFLICT OF INTEREST

None to declare.

REFERENCES

1. Alaebo PO, Njoku GC, Udensi GC, *et al*. Hypoglycemic and hypolipidemic effect of *Dialum guineense* (ICHEKU) fresh leaves on alloxan-induced diabetes in male albino rats. Asia J Res Cardi Dis 2022;4(4):1-7.
2. Panahi Y, Sahebkar A, Amiri M, *et al*. Improvement of sulfur mustard-induced chronic pruritus, quality of life, and antioxidant status by curcumin: Results of a randomized, double blind, placebo controlled trial. Brit J Nutr 2012;108(7):1272-1279. *https://doi.org/10.1017/S0007114511006544*
3. Zhou X, Afzal S, Wohlmuth H, *et al*. Synergistic anti inflammatory activity of ginger and turmeric extracts in inhibiting lipopolysaccharide and interferon γ induced proinflammatory mediators. Mole 2022;27(12):3877. *https://doi.org/10.3390/molecules27123877*
4. Uhuo EN, Aaduaka EG, Chinedu NP, *et al*. Studies of lipid profile and oxidative stress markers in doxorubicin-induced cardio toxicity in rats administered *Adansonia digitata* leaf extract. Lond J Med Health Res 2023;22(5):9-18.
5. Achuba FI, Ja-anni MO. Effect of abattoir wastewater on metabolic and antioxidant profiles of cowpea seedlings grown in crude oil-contaminated soil. Inter J Recyc Orga Waste Agri 2018;7:59-66. *https://doi.org/10.1007/s40093-017-0190-6*
6. Joffe AR, Bara M, Anton N. The ethics of animal research: A survey of the public and scientists in North America. BMC Med Ethics 2016;17(1):17-22. *https://doi.org/10.1186/s12910-016-0100-x*
7. World Medical Association. WMA statement on guiding principles for the use of telehealth for the provision of health care (S-2009-04-2009).
8. Misra HP, Fridovich I. The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. J Bio Chem 1972;247(10):3170-3175. *https://doi.org/10.1016/S0021-9258(19)45228-9*
9. Aebi H. Catalase *in-vitro*. Meth Enzym 1984;105:121-126. *https://doi.org/10.1016/S0076-6879(84)05016-3*
10. Rotruck JT, Pope AL, Ganther HE, *et al*. Selenium: Biochemical role as a component of glutathione peroxidase. Sci 1973;179(4073): 588-590. *https://doi.org/10.1126/science.179.4073.588*
11. Ohkawa H, Ohishi N, Yagi K. Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Ana Biochem 1979; 95(2):351-358. *https://doi.org/10.1016/0003-2697(79)90738-3*
12. Cuvelier ME, Berset C. Use of a free radical method to evaluate antioxidant activity. Food Sci Tech 1995;28(1):25-30. *https://doi.org/10.1016/S0023-6438(95)80008-5*
13. He HJ, Wang GY, Gao Y, *et al*. Curcumin attenuates Nrf2 signaling defect, oxidative stress in muscle and glucose intolerance in high fat diet-fed mice. World J Diab 2012;3(5):94-104. *https://doi.org/10.4239/wjd.v3.i5.94*
14. Hamdy AA, El-Nashar HM, Ibrahim AH. *et al*. Research progress on the mechanism of curcumin anti-oxidative stress based on signaling pathway. Front Pharma 2024;15:1548073.
15. Yang F, Tan X, Wang H, *et al*. Mechanisms of Nrf2-mediated antioxidant response: ARE-driven gene expression. Free Rad Bio Med 2019;134:169-183.
16. Jiang Z, Liu Q, Chen S. *et al*. Curcumin alleviates liver oxidative stress in type 1 diabetic rats via activation of Keap1-Nrf2-ARE pathway. Oxid Med Cell Long 2012;605178.
17. Balogun E, Hoque M, Gong P, *et al*. Curcumin activates the haem oxygenase-1 gene via regulation of Nrf2 and the antioxidant-responsive element. Biochem J 2003;371(3):887-895. *https://doi.org/10.1042/bj20021619*
18. Srinivasan K. Role of ginger in the prevention of oxidative stress and diabetic complications. Phyto Res 2017;31(1):58-65.
19. Wu S, Zhu J, Wu G, *et al*. 6-Gingerol alleviates ferroptosis and inflammation of diabetic cardiomyopathy via the Nrf2/HO-1 pathway. Oxid Med Cell Long 2022,30275. *https://doi.org/10.1155/2022/3027514*
20. Rao JR, Rao PV, Rao LJ. Effect of turmeric and curcumin on oxidative stress and antioxidant enzymes in streptozotocin-induced diabetic rat. J Ethno 2008;116(1):1-7.
21. Nagpal M, Sood S. The synergistic effect of turmeric and ginger extracts on antioxidant potential: An *in-vitro* study. J Herbal Med 2020; 22:100-108.
22. Singh G, Aggarwal BB. Antioxidant properties of turmeric and ginger: A comparative review. Phyto Res 2019; 33(12): 3249-3265.
23. Alaebo PO, Egbuonu ACC,Achi NK, *et al*. *In-vivo* antidiabetic potentials and toxicological effect of *Dennettia tripetala* seeds on alloxan-induced diabetic male albino rats. Universal J Pharm Res 2024; 9(2): 27-33. *http://doi.org/10.22270/ujpr.v9i2.1082*