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Original Article

Unripe *Musa paradisiaca* (*Linn.*) Aqueous Pulp Extract Mitigates Diabetic-like Phenotypes and Oxidative Stress Markers in *Drosophila melanogaster*

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Abstract

Diabetes mellitus (DM) is a prevalent metabolic condition affecting millions worldwide. In Southwestern Nigeria, unripe plantain (*Musa paradisiaca*) is traditionally consumed as a dietary intervention for DM management. The present study evaluates the effect of unripe *Musa paradisiaca* aqueous pulp extract (UMPAPE) through computational (*in silico*) and experimental (*in vivo*) approaches using *Drosophila melanogaster* as a model organism. Flies were divided into five experimental groups (n=50, five replicates) and treated for 14 days: control, high-sucrose diet (HSD)-induced diabetic untreated, diabetic flies treated with UMPAPE (0.2 g/5 g and 0.4 g/5 g diet), and a standard drug group. Biochemical assays, including glucose, lipid profiles, and antioxidant enzyme activities, were conducted using standard kits. PyRx and Biovia Discovery Studio were used for molecular docking to obtain the interactions between the UMPAPE best binding bioactive compounds (retrieved from PubChem) and the selected diabetic-related protein targets. Aldose reductase (AR) and sorbitol dehydrogenase (SDH) retrieved from the RCSB Protein Data Bank. (*2S, 2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ol (-9.1 kcal/ mol) and 2-trifluoromethylbenzoic acid (-7.2 kcal/ mol) showed strong interactions with AR and SDH. Additionally, UMPAPE administration showed no toxicity in the flies at different doses but significantly ($p < 0.05$) reduced glucose, lipid, and oxidative stress marker levels while enhancing high-density lipoprotein, glutathione, and antioxidant enzyme activities ($p < 0.05$). In conclusion, these findings validate the traditional use of unripe *Musa paradisiaca* pulp in diabetes management and highlight its potential for further translational research.

Keywords: Diabetes Mellitus, Oxidative Stress, *Musa paradisiaca* Pulp, Antioxidant, Lipid Profile, *Drosophila melanogaster*, Molecular Docking

Introduction

Diabetes mellitus (DM) encompasses a range of metabolic disorders characterized by impaired glucose utilization or excessive glucose production, resulting in persistent hyperglycemia due to dysregulated gluconeogenesis and glycogenolysis [1].

According to the International Diabetes Federation (IDF), it is the most prevalent endocrine disorder globally, affecting over 500 million individuals, which corresponds to roughly 10% of the world's adult population [2]. The progression of DM is often associated with mitochondrial function, leading to the generation of reactive oxygen species (ROS)

and the onset of oxidative stress in various tissues, including pancreatic beta cells and blood vessels. Elevated ROS can damage mitochondria as well as critical macromolecules such as proteins, lipids, and nucleic acids, thereby accelerating cellular aging and tissue degeneration [3].

Medicinal foods, particularly fruits and vegetables, have long been recognised as safe and effective sources of bioactive compounds with therapeutic potential [4].

Studies have shown that many medicinal plants are rich in phytochemicals with significant pharmacological activities essential for mitigating metabolic disorders, including DM and its complications [5-7].

Among these compounds are terpenoids, alkaloids, tannins, phenolic acids, flavonoids, anthocyanins, and carotenoids, which exert various protective and regulatory effects [8].

One notable medicinal food is unripe *Musa paradisiaca* (plantain), a widely consumed starchy fruit, belonging to the genus *Musa* and family *Musaceae*. In southwestern Nigeria, it is locally known as Ogede/ Dodo (Yoruba), Ayaba (Hausa), and Ogadejioke (Igbo), and it is predominantly cultivated in the tropical and subtropical regions worldwide [9].

Secondary metabolites extracted from the fruit pulp, peel, seeds, and flower of *Musa paradisiaca* have demonstrated efficacy in managing diarrhoea, dysentery, intestinal lesions in ulcerative colitis, sprue, uremia, nephritis, gout, hypertension, and cardiovascular disorders [10].

Galani [11] further reported that *M. paradisiaca* exhibits a broad spectrum of pharmacological activities, including analgesic, antidepressant, anticonvulsant, central nervous system depressant, antimicrobial, antioxidant, antimalarial, mutagenic, hepatoprotective, and wound healing effects. Animal models have significantly advanced DM studies by allowing detailed exploration of *in vivo* mechanisms and environmental factors that influence disease onset, progression, and complications. *Drosophila melanogaster*, commonly known as the tiny fruit fly and often regarded as the "queen of genetics," displays considerably lower gene redundancy than mammals while sharing homologs for approximately 65-70% of human disease-related genes, including those implicated in diabetes [12].

These features make it an acceptable and reliable model for biochemical and molecular investigations [13].

Accordingly, this study aimed to investigate the therapeutic potential of unripe *Musa paradisiaca* aqueous pulp extract (UMPAPE) using both *in silico* analyses and *in vivo* assessment in *Drosophila melanogaster* model.

Method

Plant Collection, Extraction, and Phytochemical Profiling

Fresh unripe *Musa paradisiaca* fruits were purchased in July 2024 from Iyana-Iba market near LASU, Ojo, Lagos, Nigeria (Lat: 6°28'1.20"N; Long: 3°10'58.80"E) and authenticated by Dr. K. T. Omolokun at the LASU herbarium. The fruits were washed, air-dried at 27 °C, and the pulp homogenized using a Scanfrost blender (SFKAB409). An aqueous extract (100 g pulp/500 mL water) was obtained by maceration for 48 h, filtered (Whatman No.1), concentrated at 40 °C, and stored at 4 °C. Phytochemical profiling was performed using GC–MS and HPLC according to Cheong *et al*. [14] method.

Ligand and Protein Preparation

The 3D structures of phytochemicals identified via GC–MS and HPLC were retrieved from PubChem in .sdf format and converted to .pdb/. mol using Biovia Discovery Studio 2021. SMILES strings were also obtained. Ligands were energy-minimized using the Open Babel MMFF94 force field and converted to AutoDock . pdbqt format. Human AR (PDB:

3S3G) and SDH (PDB: 1PL6) were selected as targets. Grid coordinates were: AR (x = –0.3408, y = –0.6404, z = 15.0471) and SDH (x = 80.0873, y = 62.3478, z = 3.4355). Proteins were purified by removing water and heteroatoms, and chains with complete active sites were retained.

Molecular Docking and ADME/T Profiling

AutoDock Vina was used for docking, with Tolrestat as a reference. Ligands were ranked by binding energy. ADME/T properties of the top ten ligands were predicted using SwissADME and ProTox to assess pharmacokinetics, solubility, druglikeness, and toxicity.

Collection and Culture of Drosophila Melanogaster

Harwich strain flies were obtained from the LASU Biochemistry Drosophila Research Lab. Flies were cultured on a modified diet (52 g cornmeal, 7.9 g agar, 9 g yeast, 0.7 g nipagin) at room temperature under a 12 h light/dark cycle until adulthood [15].

High-Sucrose Diet Induction, Groupings, and Homogenate Preparation

A high-sucrose diet (30% sucrose) was used to induce hyperglycemia and hyperlipidemia in 50 rats (5 g/diet). Flies were randomly divided into five groups: G1: Control (normal diet + 200 µL water); G2: HSD + 200 µL water (untreated); G3: HSD + 0.2 g UMPAPE; G4: HSD + 0.4 g UMPAPE; and G5: HSD + glibenclamide. After 14 days, flies were immobilized on ice, weighed, and homogenized in 0.1 M phosphate buffer (pH 7.4). Homogenates were centrifuged (4000 g, 10 min, 4 °C) using a Sorvall Legend Micro 17R centrifuge (Thermo Scientific, Germany), and supernatants were stored at –20 °C for analysis [16].

Biochemical Assessments

Biochemical parameters were assessed to evaluate the effects of the UMPAPE on HSD-induced fruit flies:

Quantification of Protein, Glucose, and Lipid Concentrations

Protein was measured using the modified Lowry method [17]. Glucose and lipid profiles triglyceride (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-c) were determined using Randox kits (Crumlin, U.K.). In contrast, low-density lipoprotein cholesterol (LDL-c) and very-low-density lipoprotein cholesterol (VLDL-c) were calculated with standard formulas: LDL-c = [TC- (HDL-c + TG/5)], and VLDL-c = TAG/5, respectively [18].

Determination of SOD and GST Activities

Superoxide dismutase (SOD) was assayed using the epinephrine method [19] while glutathione-S-transferase (GST) activity was determined with 1-chloro-2,4-dinitrobenzene (CDNB) substrate [16]. In brief, 0.1 mL of the fly homogenate was added to a mixture of 2.5 mL of 0.05 M phosphate buffer (pH 7.8) and 0.3 mL of adrenaline solution (0.059%). Following this, the absorbance was measured at 750 nm for 90 seconds at 15-second intervals. The SOD activity was calculated and expressed as mmol/mg protein. Conversely, 20 µL of the fly homogenate and 10 µL of CDNB were added to a mixture of 20 mL of 0.25 M phosphate buffer (pH 6.9, containing 2.5 mM EDTA), 10.5 mL of distilled water, and 500 µL of glutathione. The reaction mixture was shaken, and the absorbance was read at 340 nm for 120 seconds (30-second intervals). The GST activity was calculated and expressed as mmol/min/mg protein.

Estimation of Oxidative Stress Marker Levels

Hydrogen peroxide (H_2O_2) was measured using the FOX assay [20]; nitric oxide (NO) levels via Griess reagent [21]; lipid

peroxidation as TBARS [22]; and GSH was determined using the DTNB reaction [23].

Statistical Analysis

Data are presented as Mean ± SEM. Statistical comparisons were made using one-way ANOVA in GraphPad Prism (version 5.0). Differences were considered significant at $p < 0.05$.

Results and Discussion

The current study offers a comprehensive evaluation of the bioactive profile and therapeutic potential of unripe *Musa paradisiaca* aqueous pulp extract, utilising both *in silico* and *in vivo* approaches with the *Drosophila melanogaster* model. The quest for effective DM treatments with minimal adverse effects remains a significant concern, driving a cascade of discoveries regarding various medicinal plants with antidiabetic potential [24-26]. Preliminary quantitative evaluation of the phytochemical profile of the aqueous pulp of unripe *Musa paradisiaca* revealed the presence of important bioactive compounds, including steroids, alkaloids, tannins, flavonoids, saponins, and phenols (Table 1). A similar trend was observed by Uzairu and Kano [27]. Additionally, the evaluation of the extract's polyphenolic content revealed high levels of tannins (7.91 ± 0.39), flavonoids (6.20 ± 0.52), and phenolic acids (4.28 ± 0.08), suggesting a strong antioxidant potential, attributed to the abundance of polyphenols. This finding is consistent with a previous study by Sidhu and Zafar [28], which indicated that quercetin and kaempferol are major constituents of *Musa paradisiaca* and are effective in reducing postprandial hyperglycaemia via enzyme inhibition. Meanwhile, flavonoids [29], tannins [30], phenolic acids [31], and alkaloids have been associated with a role in inflammation [32].

Furthermore, comprehensive phytochemical profiling of the UMPAPE using GC-MS (Table 2) and HPLC (Table 3) techniques confirmed the extract's richness in polyphenols such as syringin (7.933 ppm), kaempferol, quercetin, luteolin, apigenin, beta-sitosterol, capsaicin, caffeic acid, and 20 other compounds that account for 99.98% of the area percentage, including *N*-Hexadecanoic acid, 9,12-octadecadienoic acid, and acora-4(14),8-diene. Previous studies have demonstrated that plant-derived polyphenols, such as those found in UMPAPE, can enhance insulin secretion, improve insulin sensitivity, and reduce hepatic glucose output, ultimately leading to better glucose homeostasis [33-36]. Polyphenols have also been established as a rich source of antioxidants and, thus, may scavenge free radicals in diabetic patients [37]. Among the compounds identified by GC-MS analysis, 9,12-octadecanoic acid and gamma-tocopherol are consistent with findings from other Musa species, emphasizing their potential to inhibit lipid peroxidation and reduce insulin resistance [38,39]. Thus, the results of the phytochemical profiling suggest that UMPAPE could serve as a potential adjunctive therapy in managing diabetes.

Many carbohydrate-metabolising enzymes, such as AR and SDH, are considered effective for treating DM [40].

The application of molecular docking (computational) tools, also known as *in silico* studies, has significantly enhanced drug discovery and development due to their efficiency, reduced labor, and time effectiveness [41].

It has also been important in studying, understanding, and predicting non-covalent interactions between protein targets or receptors and ligands or bioactive compounds (drugs) [42, 43].

Table 1. Preliminary phytochemical profiling of UMPAPE

Sr./No.	Phytochemicals	Qualitative analysis	Polyphenolic content (mg/ 100 g)
1.	Steroids	+	ND
2.	Alkaloids	+	ND
3.	Tannins	+	7.91 ± 0.39
4.	Flavonoids	+	6.20 ± 0.52
5.	Saponins	+	ND
6.	Phenolics	+	4.28 ± 0.08

Key: + = Present, **-** = Absent, and ND = Not determined.

Table 2. GC-MS profiling of UMPAPE

Sr./No.	Compounds	MM (g/mol)	MF	RT	Area (%)
1.	*N*- Hexadecanoic acid	256.00	$C_{16}H_{32}O_2$	14.964	24.01
2.	9,12- Octadecanoic acid	280.00	$C_{18}H_{32}O_2$	15.699	0.84
3.	11,14,17 –Eicosatrienoic acid	320.00	$C_{21}H_{36}O_2$	15.727	0.45
4.	9,12-Octadecanoic acid (*Z, Z*)	280.00	$C_{18}H_{32}O_2$	16.147	36.25
5.	Butanamine,2-methyl-	87.00	$C_5H_{13}N$	17.070	0.12
6.	9- Octadecenamide (*Z*)	281.00	$C_{18}H_{35}NO$	17.565	4.56
7.	Eicosanoic acid	312.00	$C_{18}H_{36}O_2$	17.679	0.43
8.	9,12-Octadecanoic acid (*Z, Z*), 2,3-dihydroxypropyl ester	354.00	$C_{21}H_{38}O_4$	19.882	3.36
9.	Gamma tocopherol	416.00	$C_{28}H_{48}O_2$	22.695	0.71
10.	2-Trifluoromethylbenzoic acid,2,7-dimethyloct-7-en-5-yn-4-yl	324.00	$C_{18}H_{19}F_3O_2$	23.235	0.20
11.	Butyl-9,12,15-octadecatrienoate	334.00	$C_{22}H_{38}O_2$	19.924	1.86
12.	2-Heptyn-1-ol	112.17	$C_7H_{12}O$	6.685	13.62
13.	Acora-4(14),8-diene	204.35	$C_{15}H_{24}$	10.655	2.73
14.	Bergamotene	204.35	$C_{15}H_{24}$	10.747	2.41
15.	Cedrene	204.357	$C_{15}H_{24}$	10.936	0.65
16.	(*2S*)-2-Methyldecanal	170.29	$C_{11}H_{22}O$	17.154	1.12
17.	6-Chloro-1-hexanol	136.00	$C_6H_{13}ClO$	7.771	2.39
18.	Propene	42.08	C_3H_6	12.44	2.55
19.	Pentadecanoic acid,13-methyl-methyl ester	270.45	$C_{17}H_{34}O_2$	15.253	1.38
20.	aR-Turmerone	216.32	$C_{15}H_{20}0$	12.573	0.34

RT= Retention Time; MM= Molar Mass; and MF= Molecular Formular.

Table 3. HPLC profiling of UMPAPE

Sr./No.	Phytochemicals	Concentration (ppm)
1.	Caffeic acid	1.016
2.	Capsaicin	1.400
3.	Syringin	7.933
4.	Kaempferol	6.333
5.	Beta sitosterol	3.483
6.	Apigenin	3.816
7.	Luteolin	5.200
8.	Quercetin	5.516

ppm = Parts per million.

(*2S,2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ols (-9.0 kcal/ mol) and 2-trifluoromethyl-benzoic acid (-7.2 kcal/ mol) (Table 4) showed the best binding energies and affinities for AR and SDH, respectively.

These molecular docking scores confirm the efficacy of UMPAPE-derived phytochemicals in modulating diabetic protein targets, consistent with the report by Kajal and Singh [44], who identified plant compounds with high docking scores comparable to those of synthetic drugs. The formation of Van der Waals forces was observed between (*2S,2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ols and Trp111, Trp219, Asn160, Lys21, Lys77, Lys262, Gln183, Thr19, Asp43, Asp216, Ser210, and Ser214 of AR; pi-sigma interaction with Tyr209; conventional hydrogen bonding with Tyr48 and Trp20; halogen (fluorine) bonding with Ile260 and Gly18; carbon-hydrogen bonding with Gly18; alkyl and pi-alkyl interactions with His110, Cys298, and Val47 (Figure 1).

Conversely, 2-trifluoromethylbenzoic acid formed a Van der Waals force interaction with sorbitol dehydrogenase amino acids: Phe297, Ser46, Glu155, His69, Thr121, and Tyr50; an unfavorable interaction with Phe59; conventional hydrogen bonding with Arg298; alkyl and pi-alkyl interactions with Phe118, Leu274, and Ile56; carbon-hydrogen, and halogen (fluorine) bonding (Figure 2).

These interactions, primarily hydrogen bonds and stacking interactions, stabilise the protein-ligand complexes. Amino acid residues involved in these interactions perform specific biochemical roles, with hydrogen bonds being crucial for the stability of biomolecules and enzymes [45].

Stacking interactions, particularly with aromatic amino acids such as tryptophan, histidine, tyrosine, and phenylalanine, are vital in drug design, as they enhance molecular interactions and binding specificity [46].

Aspartate and glutamate coordinate metal ions, while asparagine and glutamine form hydrogen bonds to aid substrate binding. Histidine, tyrosine, serine, and threonine contribute to catalytic mechanisms and substrate stabilization [47].

The ADME/T profiling, drug likeness, and toxicity properties of the top five ligands showed that they passed Lipinski's rule of five, indicating good oral bioavailability.

Table 4. Binding energies (kcal/mol) of the top ten ligands with sorbitol dehydrogenase and aldose reductase

Sr./No.	Top Ligands	PubChem ID	Protein targets	
-	-	-	AR	SDH
1.	(*2E, 6E*)-7,11-Dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ol	1170889	-9.0	-6.1
2.	Beta sitosterol	222284	-8.7	-6.9
3.	2-Trifluoromethyl benzoic acid	531209	-8.1	-7.2
4.	*Cis*-11,14,17-Eicosatrienoic acid methyl ester	5367326	-8.0	-4.4
5.	Rel –acora	51040470	-7.6	-6.6
*	Tolrestat (STD)	53359	-7.3	-7.0

** Standard drug.*

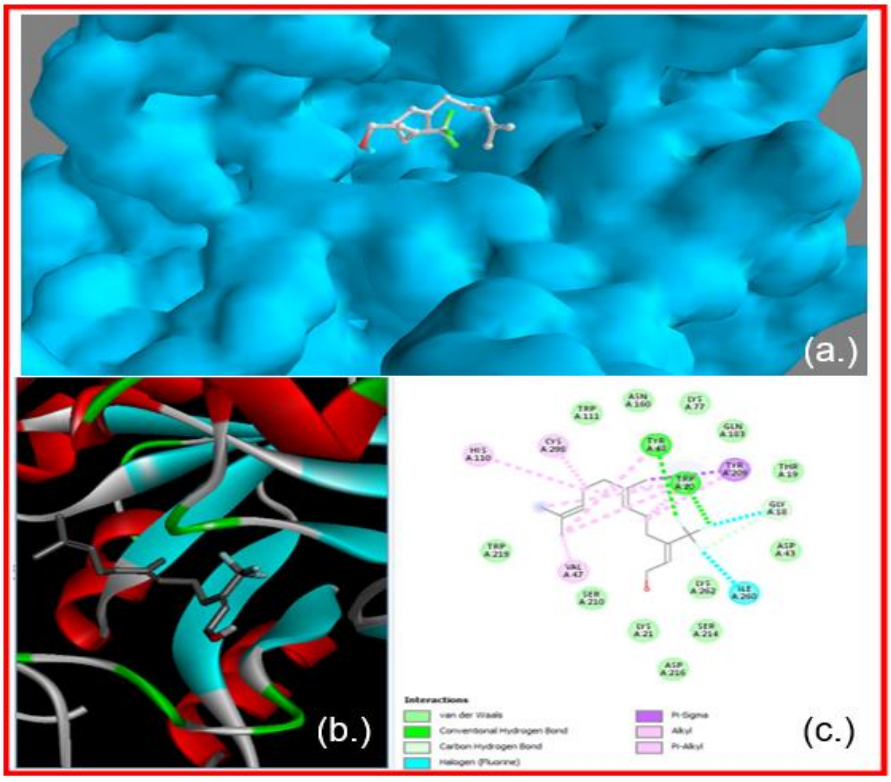

Figure 1. (a) Molecular surface, (b) 3D, and (c) 2D structures of aldose reductase (PDB ID:3S3G) active site interaction with (*2S,2E*) -7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien -1-ols

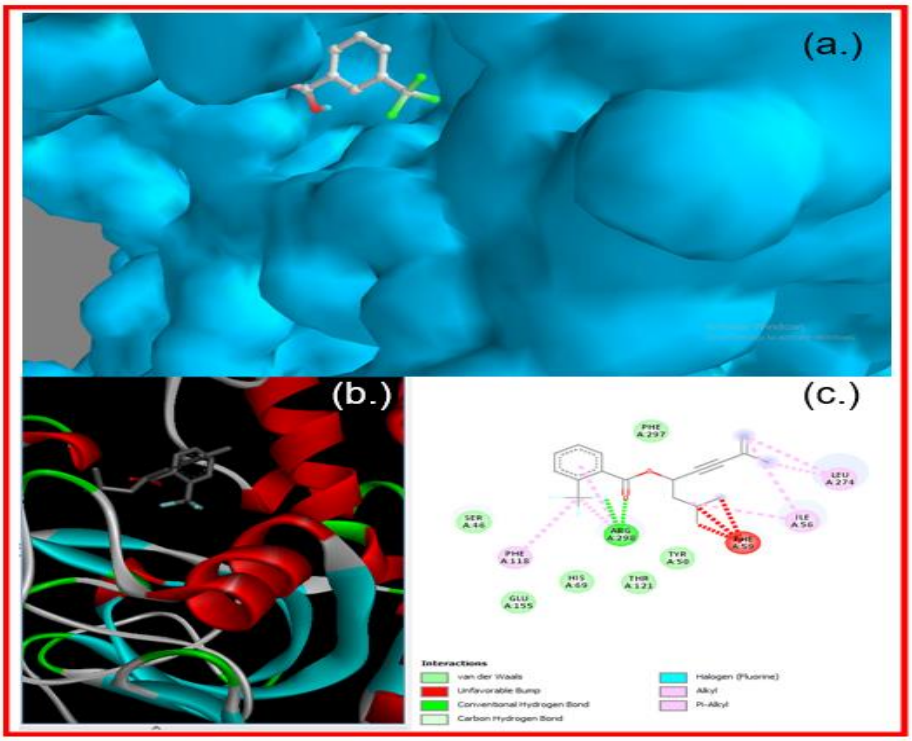

Figure 2. (a) Molecular surface, (b) 3D, and (c) 2D structures of sorbitol dehydrogenase (PDB ID: 1PL6) active site interaction with 2-trifluoromethyl benzoic acid

The overall top ligand, (*2E,6E*)-7,11,14-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ol, exhibited excellent ADMET properties and low toxicity. However, 2-trifluorobenzoic acid was identified as potentially toxic (Table 5).

The physicochemical properties indicate the potential of these key ligands for developing new pharmaceuticals [48].

The ADME/T results demonstrated the safety and drug-likeness potential of UMPAPE, which are crucial for drug development. Similar favourable predictions were observed in the *in silico* evaluations of phytochemicals from *Vernonia amygdalina* [49]. The ADMET predictions for the top five ligands suggest adherence to the Lipinski rule of five (RO5), good bioavailability score, synthetic accessibility, water solubility, and lipophilicity. Ligands with good lipophilicity, bioavailability, and synthetic accessibility scores are expected to exhibit effective membrane penetration, leading to improved metabolism [50], drug-likeness properties [51], and facilitating new drug synthesis [52].

Both (*2S,2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ols and 2-trifluoromethylbenzoic acid) showed a high gastrointestinal absorption, suggesting that they possess favourable pharmacokinetic profiles for oral administration. This high GI absorption indicates efficient permeability across the intestinal epithelium, likely due to their balanced lipophilicity and molecular size [53]. Such characteristics enhance their potential as lead compounds in drug development, particularly for conditions where oral bioavailability is a critical parameter [54]. Furthermore, this property may contribute to optimal systemic exposure and therapeutic efficacy if other ADMET parameters also fall within acceptable ranges [55].

The blood-brain barrier (BBB) permeability showed a positive and negative indication for (*2S, 2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ols and 2-trifluoromethylbenzoic acid, respectively. The positive indication suggests that the compound could be developed for treating central nervous system (CNS) disorders, particularly neurological conditions such as dementia, schizophrenia, Parkinson's disease, and Alzheimer's disease [56].

Notably, both (*2S, 2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ols and 2-trifluoromethylbenzoic acid have high gastrointestinal absorption and are identified as *P*-glycoprotein substrates, suggesting that P-glycoprotein transporters may mediate their cellular efflux. This implies potential limitations in their oral bioavailability and intracellular retention, particularly in tissues with high P-glycoprotein expression such as the intestinal epithelium, blood-brain barrier, and tumour cells [57].

Consequently, co-administration with P-glycoprotein inhibitors or structural modifications to avoid efflux could enhance their therapeutic efficacy and pharmacokinetic profiles [58].

Additionally, the toxicity profile of (*2S, 2E*)-7,11-dimethyl-3-(trifluoromethyl) dodeca-2,6,10-trien-1-ol showed no hepatotoxicity, carcinogenicity, or peroxisome proliferator-activated receptor gamma (PPAR-γ) toxicity. However, 2-trifluoromethylbenzoic acid showed positive toxicity indications for these organs, suggesting that caution should be taken before use and in the development. Hence, the integration of these bioactive compounds for therapeutic use may reduce dependency on synthetic drugs, thereby minimising their adverse side effects and ultimately, death. Treatment with UMPAPE at doses of 0.2 g and 0.4 g significantly ($p < 0.05$) reduced glucose levels and restored protein levels compared to untreated flies that were fed a high-sugar diet. These effects were comparable to those of the standard drug, **g**libenclamide, indicating strong *in vivo* antidiabetic properties (Table 6).

Table 5. ADME/T profiling of the top five (5) ligands

Ligands	(*2E, 6E*)-7,11 Dimethyl -3- (trifluoro methyl) do- deca-2, 6, 10- trien -1-ol	Beta- Sitosterol	2- Trifluoro methyl benzoic acid	*Cis*-11,14,17- Eicosantrienoic acid methyl ester	Rel- acora	Tolrestat*
Molecular weight (g/mol)	255.31	414.71	324.34	320.51	232.32	357.35
Formular	$C_{16}H_{17}NO_2$	$C_{29}H_{50}O$	$C_{18}H_{19}F_3O_2$	$C_{21}H_{36}O_2$	$C_{15}H_{20}O_2$	$C_{16}H_{14}F_3NO_3S$
Hydrogen bond acceptor	2	1	5	2	2	6
Hydrogen bond donor	1	1	0	0	1	1
Rotatable bonds	6	6	6	16	2	6
Log Po/w (iLOGP)	3.14	5.05	3.95	5.07	2.39	4.30
Log Po/w (ESOL)	-5.42	-6.19	-5.14	-5.45	-2.29	-6.11
GI Absorption	High	Low	High	Low	High	High
BBB permeant	Yes	No	No	No	Yes	No
***P*-glycoprotein Substrate**	No	No	No	No	No	No
CYP1A2 Inhibitor	Yes	No	No	Yes	No	Yes
CYP2C19 Inhibitor	Yes	No	No	No	Yes	No
CYP2C9 Inhibitor	Yes	No	Yes	Yes	Yes	No
CYP2D6 Inhibitor	Yes	No	No	No	No	No
CYP3A4 Inhibitor	No	No	No	No	No	No
Bioavailability score	0.55	0.55	0.55	0.55	0.85	0.85
Synthetic Accessibility	2.51	6.30	3.94	3.33	4.61	2.54
Hepatotoxicity	-	+	+	-	+	-
Carcinogenicity	-	-	+	-	-	+
PPAR-γ	-	+	+	+	-	+

**Standard drug incorporated in this study; PPAR-γ= Peroxisome Proliferator-Activated Receptor Gamma.*

Table 6. Effects of UMPAPE treatment on glucose and protein concentrations of HSD-induced diabetic flies

Sr./No.	Groups	Glucose Conc. [mg/dL]	Protein Conc. [mg/mL]
1.	Control	$183.10^a \pm 3.51$	$0.21^a \pm 0.06$
2.	HSD	$272.45^b \pm 8.14$	$0.09^b \pm 0.05$
3.	HSD + UMPAPE 0.2 g	$179.40^a \pm 0.30$	$0.13^b \pm 0.03$
4.	HSD + UMPAPE 0.4 g	$163.20^a \pm 1.31$	$0.20^a \pm 0.09$
5.	HSD + Glb	$171.68^a \pm 0.66$	$0.19^a \pm 0.06$

Different superscript letters (a, b) indicate statistically significant differences between groups ($p < 0.05$).

In addition to a significant (p <0.05) increase in the fly homogenate glucose (hyperglycemia) and protein concentrations following the induction of diabetes using a high-sucrose diet (HSD), a significant (p <0.05) increase in lipids (hyperlipidemia) (Table 7) was also observed in the HSD-induced diabetic flies.

This finding is consistent with the report of Ecker *et al*. [59]. However, treatment with UMPAPE regulated the lipid profile by significantly (p <0.05) reducing total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol levels, while increasing HDL cholesterol levels. The observed effect was found to be dose-dependent and similar to that of glibenclamide. This is in accordance with the report of Asiimwe *et al*. [60].

In contrast, UMPAPE improved oxidative stress biomarkers by significantly (p <0.05) decreasing hydrogen peroxide (H_2O_2), nitric oxide (NO), and malondialdehyde (MDA) levels, indicating a reduction in oxidative stress with UMPAPE treatment. Conversely, non-protein thiol levels significantly (p<0.05) increased (Table 8), reinforcing the defense against oxidative stress (Figure 3).

Table 7. Effects of UMPAPE treatment on lipid profile of HSD-induced diabetic flies

Sr./No.	Groups	Total-Chol [mg/dL]	TRIG [mg/dL]	HDL-Chol [mg/dL]	LDL-Chol [mg/dL]	VLDL-Chol [mg/dL]
1.	**Control**	$383.28^{a} \pm 13.75$	$217.12^{a} \pm 11.89$	$171.57^{a} \pm 10.59$	$168.69^{a} \pm 13.99$	$43.44^{a} \pm 0.39$
2.	**HSD**	$593.49^{b} \pm 11.38$	$311.22^{b} \pm 13.27$	$104.12^{b} \pm 11.72$	$373.66^{b} \pm 11.31$	$116.41^{b} \pm 1.32$
3.	**HSD + UMPAPE 0.2 g**	$563.33^{b} \pm 12.70$	$134.80^{c} \pm 11.83$	$156.12^{a} \pm 11.19$	$380.06^{b} \pm 13.16$	$26.80^{c} \pm 0.38$
4.	**HSD + UMPAPE 0.4 g**	$316.25^{c} \pm 12.29$	$114.2^{c} \pm 12.059$	$182.56^{a} \pm 10.78$	$109.56^{c} \pm 11.53$	$24.21^{c} \pm 1.31$
5.	**HSD + Glb**	$292.51^{c} \pm 13.89$	$260.2^{a} \pm 13.54$	$113.50^{b} \pm 31.74$	$83.55^{d} \pm 17.18$	$49.43^{a} \pm 0.46$

Total-Chol = total cholesterol, HDL = high-density lipoprotein, LDL = low-density lipoprotein, and VLDL = very low-density lipoprotein. Different letters indicate significant differences (p < 0.05).

Table 8. Effects of UMPAPE treatment on oxidative stress markers of HSD-induced diabetic flies

S/N	Groups	H_2O_2 [mmol/ mL]	NO [mmol/ mL]	MDA [nMoles]	GSH [µmol/mg protein]
1.	**Control**	$0.057^{a} \pm 0.03$	$0.461^{a} \pm 0.00$	$0.026^{a} \pm 0.00$	$506.42^{a} \pm 54.88$
2.	**HSD**	$0.114^{b} \pm 0.03$	$0.720^{b} \pm 0.01$	$0.176^{b} \pm 0.07$	$136.30^{b} \pm 0.09$
3.	**HSD + UMPAPE 0.2 g**	$0.074^{a} \pm 0.03$	$0.519^{c} \pm 0.03$	$0.027^{a} \pm 0.01$	$197.11^{c} \pm 10.16$
4.	**HSD + UMPAPE 0.4 g**	$0.036^{a} \pm 0.00$	$0.518^{c} \pm 0.01$	$0.033^{a} \pm 0.02$	$274.50^{d} \pm 13.92$
5.	**HSD + Glb**	$0.057^{a} \pm 0.03$	$0.342^{d} \pm 0.00$	$0.078^{c} \pm 0.01$	$394.68^{e} \pm 4.55$

H_2O_2 = hydrogen peroxide, NO = nitric oxide, MDA = malondialdehyde, and GSH = glutathione. Different letters indicate significant differences (p < 0.05).

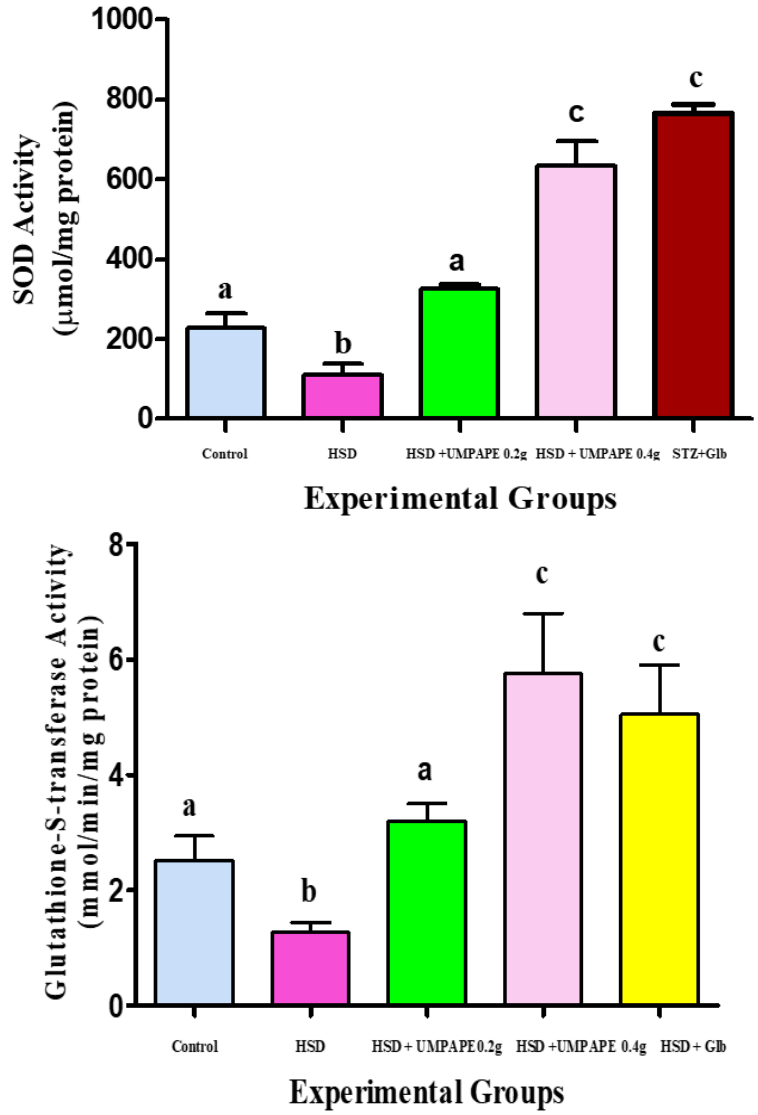

Figure 3. SOD and GST activities of HSD-induced diabetic flies treated with unripe *Musa paradisiaca* aqueous pulp extract (UMPAPE). Each bar represents Mean ± SEM, and bars with different alphabets are statistically significant from each other at $p<0.05$. SOD = superoxide dismutase, GST = glutathione-S-transferase

This further emphasises the extract's potential to reduce oxidative damage associated with diabetes [61].

These findings are especially significant as they indicate that the bioactive compounds present could help restore antioxidant defenses in individuals with diabetes [62].

This study, therefore, contributes to human society by providing insights into natural, sustainable solutions for oxidative stress-related conditions, including DM and dyslipidemia. However, further cross-species investigations and robust model validations may be necessary. SOD and GST are some of the biomarkers of oxidative stress associated with DM [63].

SOD removes excess superoxide radicals (O_2-) by converting them into hydrogen peroxide (H_2O_2) and molecular oxygen (O_2) [64].

On the other hand, GST, a family of eukaryotic and prokaryotic phase II metabolic isozymes, catalysis the conjugation of reduced glutathione (GSH) to xenobiotic substrates in response to oxidative stress for detoxification purposes [16]. These enzymes are usually

inactivated by free radicals, thereby weakening antioxidant defenses [65].

However, treating the HSD-induced diabetic flies with UMPAPE significantly ($p < 0.05$) increased the activities of these antioxidant enzymes. This effect supports the hypothesis that the bioactive compounds in the extract are capable of mitigating oxidative stress, a key contributor to diabetes-induced complications [66].

Conclusion

The results of this study showed that unripe *Musa paradisiaca* aqueous pulp extract is a promising natural product for effective free-radical scavenging activity, as well as for maintaining glucose and lipid homeostasis. Moreover, the data generated from *in vivo* and *in silic*o methodologies have a positive correlation with their traditional use in the management of DM. The *in vivo* model using *D. melanogaster*, while robust, may not fully replicate human metabolic complexity. Additionally, while docking predicts molecular interactions, animal experimental validation and clinical trials will be essential for translation. Therefore, future studies are recommended to test the extract in mammalian models and/or isolate specific active compounds for mechanistic studies.

Conflicts of Interest

The authors declared that they had no financial interest in the subject matter or materials discussed, nor any affiliations or involvement with any entity that may have one.

Consent for Publication

None.

Availability of Data and Materials

All the data are available upon request.

Authors' Contributions

All co-authors participated in all stages of this study.

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Ethical Consent

This study does not require ethical approval as *Drosophila melanogaster* was used as the invertebrate model, which is not subject to ethical review under most institutional and regulatory guidelines. However, all relevant legislation, rules, and regulations required for the study's implementation have been complied with.

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