Original Article

Phenolic constituents and modulatory effects of Raffia palm leaf (*Raphia hookeri*) extract on carbohydrate hydrolyzing enzymes linked to type-2 diabetes

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ABSTRACT

This study sought to investigate the effects of Raffia palm (*Raphia hookeri*) leaf extract on enzymes linked to type-2 diabetes mellitus (T2DM) and pro-oxidant induced oxidative stress in rat pancreas. The extract was prepared and its α-amylase and α-glucosidase inhibitory effects were determined. Radical [2,2-diphenyl-1-picrylhydrazyl (DPPH)] scavenging and Fe^{2+}-chelating abilities, and inhibition of Fe^{2+}-induced lipid peroxidation in rat pancreas homogenate were assessed. Furthermore, total phenol and flavonoid contents, reducing property, and high performance liquid chromatography diode array detector (HPLC-DAD) fingerprint of the extract were also determined. Our results revealed that the extract inhibited α-amylase (IC_{50} = 110.4 μg/mL) and α-glucosidase (IC_{50} = 99.96 μg/mL) activities in concentration dependent manners which were lower to the effect of acarbose (amylase: IC_{50} = 18.30 μg/mL; glucosidase: IC_{50} = 20.31 μg/mL). The extract also scavenged DPPH radical, chelated Fe^{2+} and inhibited Fe^{2+}-induced lipid peroxidation in rat pancreas all in concentration dependent manners with IC_{50} values of 402.9 μg/mL, 108.9 μg/mL and 367.0 μg/mL respectively. The total phenol and flavonoid contents were 39.73 mg GAE/g and 21.88 mg QAE/g respectively, while the reducing property was 25.62 mg AAE/g. The HPLC analysis revealed the presence of chlorogenic acid (4.17 mg/g) and rutin (5.11 mg/g) as the major phenolic compounds in the extract. Therefore, the ability of the extract to inhibit carbohydrate hydrolyzing enzymes and protect against pancreatic oxidative damage may be an important mechanisms supporting its antidiabetic properties and could make Raffia palm leaf useful in complementary/alternative therapy for management of T2DM. However, further studies such as *in vivo* should be carried out.
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1. Introduction

Type-2 diabetes mellitus (T2DM) is a chronic metabolic disease and the most common form of diabetes. It is characterized by insulin resistance, pancreatic β-cell dysfunction and hyperglycemia.[1] The progression of T2DM has been reported to cause some metabolic complications such as ketoacidosis, hypertension, atherosclerosis, cardiovascular diseases, pancreatitis and so on.[2–4] More so, previous reports have revealed that the development and progression of T2DM is associated with glucolipotoxicity and oxidative stress.[5] Glucolipotoxicity which involves increased levels of plasma glucose and fatty acids have been implicated in the impairment of pancreatic β-cell function.[5,6] The β-cells play major role in the secretion of insulin which regulates the absorption of glucose. However, at the early onset of T2DM, glucolipotoxicity

induces mitochondria β-cell dysfunction, decreased glucose uptake, induce reactive oxygen species (ROS) production and induce hyperglycemia.[6,7] Hyperglycemia has been reported as a major factor in ROS production which can induce tissue damage and pathological conditions such as diabetic neuropathy, nephropathy and retinopathy.[8] The use of synthetic drugs such as acarbose and miglitol in the reduction of blood glucose via the inhibition of activities of carbohydrate hydrolyzing enzymes (α-amylase and α-glucosidase) as therapeutic targets for the management of T2DM has been widely reported.[9] The inhibition of these enzymes delays the rate of carbohydrate hydrolysis in the small intestine and consequently reduces the absorption of glucose concentration into the blood stream, thereby repressing postprandial hyperglycemia and preventing hyperglycemia-induced ROS.[10]

The use of natural compounds for the treatment of diabetes and its complications has gained lots of interests recently. This is due to the fact that the use of synthetic drugs causes several side effects such as flatulence, diarrhea, abdominal dilatation, and so on in the body and hence, the need for an alternative therapy.[9] Previous reports have shown that some medicinal plants and plant foods have blood glucose lowering abilities and could be essential for management of T2DM.[11–13] Phenolic compounds from natural source have been reported to be potent inhibitors of α-amylase and α-glucosidase activities with little or no side effects.[8,14] The use of medicinal plants for the management of T2DM could be more advantageous than synthetic drugs due to their ability to scavenge free radicals which is a major risk factor of diabetic complications, as antidiabetic drugs such as acarbose lack significant antioxidant effects.[8]

Raffia palm [Raphia hookeri Mann and Wendl (Family: Palmaceae)] is a monocotyledon plant, commonly found in West Africa and very abundant in lowlands and swampy areas in Southwest Nigeria where it grows in water to about 1 m deep.[15] Raffia palm is a very tall plant of about 16 m with a single stem, 1–4 suckers, large leaves and trunks which contains palm wine.[15] The oil processed from this plant is used for cooking and making margarine while the pulp is usually consumed with boiled cassava.[16] Apart from its culinary uses, the Raffia palm roots have a variety of medicinal uses. In traditional medicine, the root extract is used as laxative and to alleviate stomach pain in infants.[17] Although, there are many reports on the antidiabetic properties of Raffia palm root and seed extracts[11,16]; however, to the best of our knowledge, there is little or no information on the antidiabetic properties of the leaf. In view of this and in the context of continuous search for safe treatment of T2DM, this study therefore, sought to investigate the effect of aqueous extract of Raffia palm leaf on α-amylase and α-glucosidase activities its antioxidant abilities, and its ability to protect the pancreas from Fe^{2+}-induced oxidative damage.

2. Materials and methods

2.1. Sample collection and preparation

Raffia palm leaves were collected in Akure, Southwest Nigeria and authenticated at the Biology Department, Federal University of Technology, Akure, Nigeria. The leaves were dried at room temperature and pulverized. Ten gram of the pulverized leaves was extracted with 100 mL of distilled water and vigorously shaken using orbital shaker for 6 h. Thereafter, the extract were filtered through Whatman filter paper (No. 1) and further centrifuge to obtain clear supernatant which was freeze-dried into powdery form with the aid of freeze-drier. One gram of dried extract was then re-dissolved into 100 mL of distilled water and kept at 4 °C for subsequent assays. Unless stated otherwise, all other chemicals and reagents used were of analytical grades and the water was glass distilled. A JENWAY UV–visible spectrophotometer (Model 6305; Jenway, Barlo world Scientific, Dunmow, United Kingdom) was used to measure absorbance.

2.2. Determination of total phenol content

The total phenol content of the extract was determined according to the method of Singleton et al.[18] Briefly, appropriate dilutions of the extract were oxidized with 2.5 mL 10% Folin–Ciocalteau's reagent (v/v) and neutralized by 2.0 mL of 7.5% sodium carbonate. The reaction mixture was incubated for 40 min at 45 °C, and the absorbance was measured at 765 nm in the spectrophotometer. The total phenol content was subsequently calculated and expressed as gallic acid equivalent (GAE).

2.3. Determination of total flavonoid content

The total flavonoid content was determined using a slightly modified method.[19] Briefly, 0.5 mL of diluted extract was mixed with 0.5 mL of absolute methanol, 50 μL of 10% $AlCl_3$, 50 μL of 1 M Potassium acetate and 1.4 mL of distilled water, and allowed to incubate at room temperature for 30 min. The absorbance of the reaction mixture was subsequently measured at 415 nm; the total flavonoid content was subsequently calculated and expressed as quercetin equivalent (QAE).

2.4. High performance liquid chromatography analysis (HPLC-DAD)

Raphia hookeri samples at a concentration of 12 mg/mL were injected by means of a model SIL-20A Shimadzu Auto sampler. Separations were carried out using Phenomenex C_{18} column (4.6 mm × 250 mm × 5 μm particle size). The mobile phase was water with 1% formic acid (v/v) (solvent A) and HPLC grade methanol (solvent B) at a flow rate of 0.6 mL/min and injection volume 40 μL. The composition gradient was: 5% solvent B reaching 15% at 10 min; 30% solvent B at 25 min, 65% solvent B at 40 min and 98% solvent B at 45 min, followed by 50 min at isocratic elution until 55 min. At 60 min the gradient reached the initial conditions again, following the method described by Oboh et al.[13] with slight modifications. The sample and mobile phase were filtered through 0.45 μm membrane filter (Millipore) and then degassed by ultrasonic bath prior to use. Stock solutions of standards references were prepared in the methanol: water (1:1, v/v) at a concentration range of 0.025–0.300 mg/mL. Quantifications were carried out by integration of the peaks using the external standard method, at 254 nm for gallic acid, 280 nm for catechin, 327 nm for chlorogenic acid and caffeic acid; and 365 for rutin, apigenin, kaempferol and luteolin. The chromatography peaks were confirmed by comparing its retention time with those of reference standards and by DAD spectra (200–600 nm). All chromatography operations were carried out at ambient temperature and in triplicate.

2.5. Determination of ferric reducing antioxidant power (FRAP)

The reducing power of the extract was determined by assessing the ability of the extract to reduce $FeCl_3$ solution as described by Oyaizu.[20] Briefly, 2.5 mL aliquot of extract was mixed with 2.5 mL 200 mM sodium phosphate buffer (pH 6.6) and 2.5 mL 1% potassium ferricyanide. The mixture was incubated at 50 °C for 20 min and then, 2.5 mL of 10 % trichloroacetic acid was added. This mixture was centrifuged at 650 rpm for 10 min. Thereafter, 5 mL of the supernatant was mixed with an equal volume of water and 1 mL of 0.1% ferric chloride. The absorbance was measured at 700 nm. The ferric reducing antioxidant property was subsequently calculated and expressed as ascorbic acid equivalent (AAE).

2.6. α-Amylase inhibition assay

α-Amylase inhibition was determine by the method of Worthington.[21] The reaction medium consist of the extract (500 μL), and 500 μL of 0.02 M sodium phosphate buffer (pH 6.9 with 0.006 M NaCl) containing 0.5 mg/mL Hog pancreatic α-amylase (EC 3.2.1.1). the reaction was incubated at 25 °C for 10 min, followed by addition of 500 μL of 1% starch solution in 0.02 M sodium phosphate buffer (pH 6.9 with 0.006 M NaCl), and then incubation at 25 °C for 10 min. the reaction was thereafter stopped by addition of 1.0 mL of dinitrosalicylic acid (DNSA). Thereafter, the mixture was incubated in a boiling water bath for 5 min, and cooled to room temperature. The reaction mixture was then diluted by adding 10 mL of distilled water, and absorbance measured at 540 nm. The reference samples included all other reagents and the enzyme with the exception of the test sample. The α-amylase inhibitory activity was expressed as percentage inhibition while acarbose drug was used as standard control.

2.7. α-Glucosidase inhibition assay

The inhibitory effect of the extract on α-glucosidase activity was determined according to the method described by Apostolidis et al.[22] Briefly, the extract (50 μL) was reacted with 100 μL of α-glucosidase solution (EC 3.2.1.20; 1.0 U/mL) in 0.1 M phosphate buffer (pH 6.9), and incubated at 25 °C for 10 min. This was followed by addition of 50 μL of 5 mM p-nitrophenyl-α-D-glucopyranoside solution in 0.1 M phosphate buffer (pH 6.9), and the mixture was incubated at 25 °C for 5 min; the absorbance read at 405 nm in the spectrophotometer. Acarbose drug was used as control and α-glucosidase inhibitory activity was expressed as percentage inhibition.

2.8. Determination of radical scavenging ability

The radical scavenging ability of the extract against 1,1-diphenyl-2-picrylhydrazyl (DPPH) radical was evaluated as described by Gyamfi et al.[23] Briefly, appropriate dilution of the extract (1 mL) was mixed with 1 mL of 0.4 mM DPPH in methanolic solution, the mixture was incubated in the dark for 30 min and the absorbance was measured at 516 nm. The DPPH radical scavenging ability was subsequently calculated and expressed as percentage e of the control, with ascorbic acid used as the standard.

2.9. Fe^{2+} chelation assay

The Fe^{2+} chelating ability of the extract were determined as described by Minotti and Aust[24] and a slightly modified method of Puntel et al.[25] Freshly prepared 500 μM of $FeSO_4$ (150 μL) was added to a reaction mixture containing 168 μL 0.1 M Tris−HCl (pH 7.4), 218 μL of saline and the extract (0−25 μL). The reaction mixture was incubated for 5 min, before the addition of 13 μL of 0.25% 1, 10-phenanthroline (w/v). The absorbance was subsequently measured at 510 nm in a spectrophotometer. The Fe^{2+} chelating ability was subsequently calculated and expressed as percentage of the control, with EDTA used as the standard.

2.10. Lipid peroxidation assay

2.10.1. Preparation of tissue homogenate

Male albino rats weighing 200−210 g were sourced from the breeding colony of Department of Veterinary Medicine, University of Ibadan, Nigeria. Rats were maintained at 25 °C, on a 12 h light/ 12 h dark cycle, with free access to food and water. They were acclimatized under these conditions for one week before the experiment. Rats were immobilized by cervical dislocation and the pancreatic tissue rapidly isolated, rinsed with cold saline and weighed on ice. The tissue was subsequently homogenized in cold saline (1/10 w/v) with about 10-up and -down strokes at approximately 1200 rev/min in a Teflon glass homogenizer (Mexxcare, mc14 362, Aayu-shi Design Pvt. Ltd. India). The homogenate was centrifuged for 10 min at 3000 × g in a refrigerated centrifuge (KX3400C, KENXIN Intl. Co., Hong Kong) at 4 °C to yield a pellet that was discarded, and a low-speed supernatant (LSS), which was kept for lipid peroxidation assay.[26]

2.10.2. Inhibition of lipid peroxidation and thiobarbituric acid reactions

The ability of the extract to inhibit Fe^{2+}-induced lipid peroxidation in pancreas homogenate was determined using thiobarbituric acid reactive species (TBARS) as described by Ohkawa et al.[27] Briefly 100 μL of rat pancreas homogenate was mixed with a reaction mixture containing 30 μL of 0.1 M Tris−HCl buffer (pH 7.4), extract (0−100 μL) and 30 μL of 250 μM freshly prepared $FeSO_4$. The volume was made up to 300 μL by distilled water before incubation at 37 °C for 1 h. The color reaction was developed by adding 300 μL of 8.1% SDS (Sodium dodecyl sulfate), 500 μL of acetic acid/HCl (pH 3.4) mixture and 500 μL of 0.8% TBA (Thiobarbituric acid). This mixture was incubated at 100 °C for 1 h. TBARS produced was measured at 532 nm and expressed using MDA (Malondialdehyde) equivalent.

2.11. Data analysis

The results of three replicates were pooled and expressed as mean ± standard deviation (S.D.). One-way analysis of variance (ANOVA) was used to analyze the mean and the post hoc treatment was performed using Duncan multiple test. Significance was accepted at $P < 0.05$. IC_{50} (extract concentration causing 50% effectiveness) were calculated with GraphPad Prism version 5.00 for Windows.

3. Results

The phenolic content of the Raffia palm leaf extract is presented in Table 1. The total phenol content of the extract reported as gallic acid equivalent (GAE) was 39.73 mg GAE/g, while the total flavonoid content reported as quercetin equivalent was 21.88 mg QE/g. The phenolic composition of the Raffia palm leaf was determined via HPLC-DAD and the chromatogram is presented in Fig. 1. The result revealed the presence of eight phenolic compounds including phenolic acids (such as gallic acid, caffeic acid, chlorogenic acid) and flavonoids such as catechin, rutin, kaempferol, luteolin and apigenin (Table 2), however, rutin (5.11 mg/g) and chlorogenic acid (4.17 mg/g) were the dominant phenolic compounds present in Raffia palm leaves, followed by gallic acid (2.53 mg/g), kaempferol (2.50 mg/g), apigenin (2.47 mg/g), caffeic acid (2.29 mg/g) respectively. Also, the ferric reducing antioxidant property of the extract reported as ascorbic acid equivalent was 25.62 mg AAE/g (Table 1).

As shown in Fig. 2, the extract and acarbose inhibited α-amylase activity at the concentrations tested (50−200 μg/mL). The extract

Table 1
Total phenol, flavonoid and ferric reducing antioxidant property of Raffia palm leaf extract.

Sample	Total phenol (mg GAE/g)	Total flavonoid (mg QE/g)	FRAP (mg AAE/g)
Raffia palm	39.73 ± 1.22	21.88 ± 2.14	25.62 ± 1.09

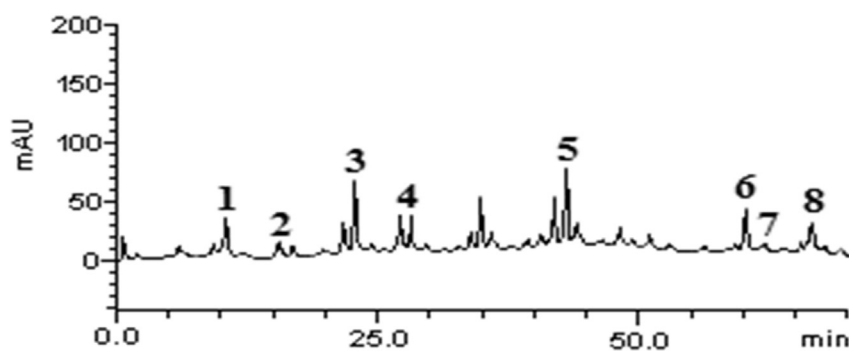Values represent means ± standard deviation of triplicate readings.

Fig. 1. Representative high performance liquid chromatography profile of Raffia palm (*R. Hookeri*) extract. Gallic acid (peak 1), catechin (peak 2), chlorogenic acid (peak 3), caffeic acid (peak 4), rutin (peak 5), kaempferol (peak 6), luteolin (peak 7) and apigenin (peak 8).

Table 2
Phenolic composition of Raffia palm leaf extract.

Composant	mg/g	%
Gallic acid	2.53 ± 0.02	0.25
Catechin	0.86 ± 0.01	0.086
Chlorogenic acid	4.17 ± 0.01	0.42
Caffeic acid	2.29 ± 0.03	0.23
Rutin	5.11 ± 0.04	0.51
Kaempferol	2.50 ± 0.01	0.25
Luteolin	0.18 ± 0.02	0.18
Apigenin	2.47 ± 0.01	0.25

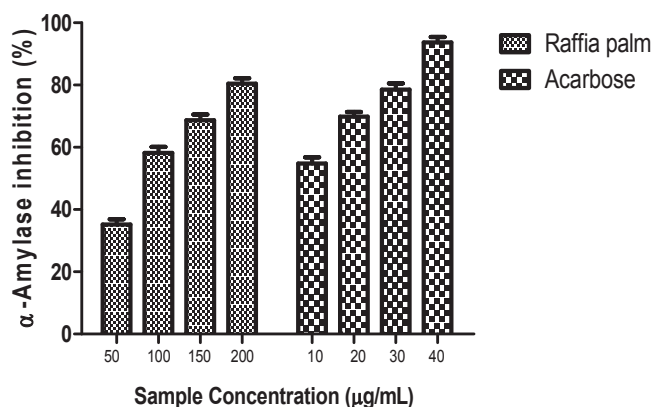Results are expressed as mean $\pm$ standard deviations (SD) of three determinations.

Fig. 2. α-Amylase inhibitory ability of Raffia palm leaf extract and acarbose. The concentrations of the extract used for the plot are 50, 100, 150 and 200 μg/mL. The concentrations of the acarbose used for the plot are 10, 20, 30 and 40 μg/mL. Values represent mean of standard deviation of triplicate readings.

decreased the activity of the enzyme in concentration dependent patterns. Acarbose ($IC_{50} = 18.30$ μg/mL) had higher inhibitory effect on α-amylase than the extract ($IC_{50} = 110.4$ μg/mL). Also, the IC_{50} values listed in Table 3 revealed that the concentration dependent inhibition of α-glucosidase activity of the extract ($IC_{50} = 99.96$ μg/mL) at the concentrations tested (50−200 μg/mL) was lower than that of acarbose ($IC_{50} = 20.31$ μg/mL) (see Fig. 3).

The DPPH free radical scavenging ability of the extract and ascorbic acid is presented in Fig. 4. The result revealed that the extract and ascorbic acid scavenged DPPH radicals in a concentration dependent manner at the concentrations tested (150−600 μg/mL and 10−40 μg/mL respectively). The result revealed that ascorbic acid having the IC_{50} value of 3 19.36 μg/mL had higher DPPH radical scavenging ability than the extract which has the IC_{50} value of 402.9 μg/mL (Table 3). Furthermore, the Fe^{2+} chelating ability of the extract and EDTA is presented in Fig. 5. EDTA had higher chelating ability with IC_{50} value of 51.12 μg/mL compared to that of extract 108.9 μg/mL (Table 3).

Our findings also revealed that incubation of rat pancreas with Fe^{2+} solution caused a significant increase (167.23%) in malondialdehyde (MDA) content; however, the extract (75−300 μg/mL) in turn caused a significant decrease (118.44−81.07%) in the MDA content of the pancreas (Fig. 6).

4. Discussion

Phenolic compounds have diverse biological activities and have been reported to be effective in the treatment and management of degenerative diseases such as diabetes, hypertension and obesity.[28,29] Phenolic compounds have been reported to inhibit α-amylase and α-glucosidase inhibitory activities and exhibit antioxidant properties which are essential in the prevention and management of T2DM.[12] The use of medicinal plants which are rich in polyphenols are often advocated for the management of T2DM

Table 3
IC_{50} values for the inhibition of α-amylase, α-glucosidase, Fe^{2+}-induced lipid peroxidation in rat's pancreas homogenates, DPPH radical scavenging and Fe^{2+}-chelating abilities of aqueous extracts from Raffia palm (*R. hookeri*) leaves (μg/mL).

Parameter	Raffia palm	Acarbose	Ascorbic acid	EDTA
Enzymes inhibition				
α-amylase	110.4 ± 5.62^a	18.30 ± 1.96^b		
α-glucosidase	99.96 ± 4.31^a	20.31 ± 2.14^b		
Radical scavenging and chelating ability				
DPPH	402.9 ± 6.22^a		19.36 ± 1.22^b	
Fe^{2+}-chelation	108.9 ± 4.34^a			51.12 ± 1.14^b
Lipid peroxidation				
Fe^{2+} induced MDA	367.0 ± 5.82			

Values represent means of triplicates; values with the same letter along the same column are not significantly ($P > 0.05$) different.
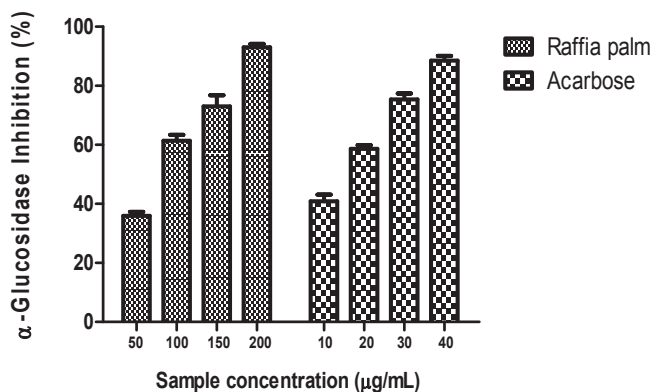
Fig. 3. α-Glucosidase inhibitory ability of Raffia palm leaf extract and acarbose. The concentrations of the extract used for the plot are 50, 100, 150 and 200 μg/mL. The concentrations of the acarbose used for the plot are 10, 20, 30 and 40 μg/mL. Values represent mean of standard deviation of triplicate readings.
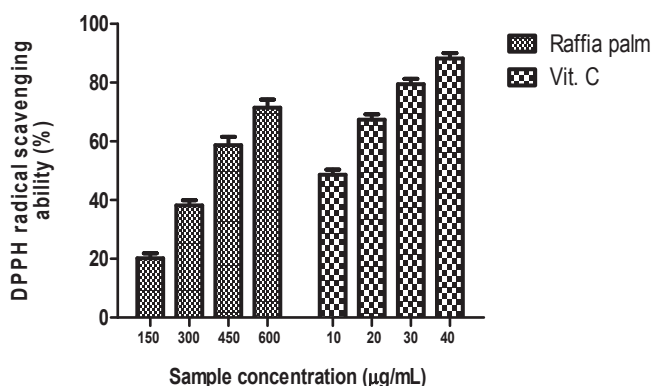

Fig. 4. DPPH radical scavenging ability of Raffia palm leaf extract and Vitamin C. The concentrations of the extract used for the plot are 150, 300, 450 and 600 μg/mL respectively. The concentrations of the Vit. C used for the plot are 10, 20, 30 and 40 μg/mL. Values represent mean of standard deviation of triplicate readings.

owing to their possible lower risk of side effects compared to contemporary antidiabetic drugs such as acarbose.[12] Therefore, the potential antidiabetic properties of Raffia palm leaf extract observed in this study as a result of their inhibitory effect on major carbohydrate catabolizing enzymes as well as their antioxidant effect could be largely linked to their constituent phenolic phytochemicals. Rutin
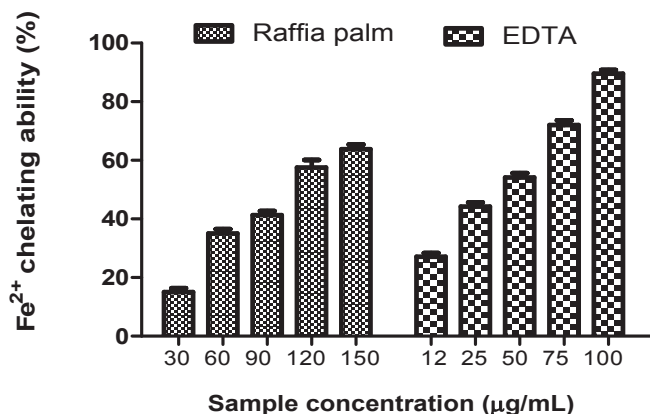

Fig. 5. Fe^{2+} chelating ability of Raffia palm leaf extract and EDTA. The concentrations of the extract used for the plot are 30, 60, 90, 120 and 150 μg/mL. The concentrations of the EDTA used for the plot are 12, 25, 50, 75 and 100 μg/mL. Values represent mean of standard deviation of triplicate readings.
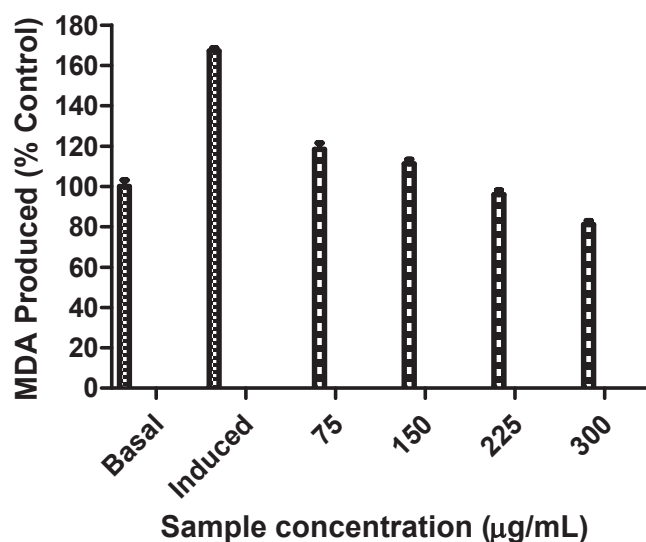

Fig. 6. Inhibition of Fe^{2+}-induced lipid peroxidation in rat pancreas by Raffia palm leaf extract.

and chlorogenic acid were the predominant phenolic compounds that was identified in Raffia leaf. Rutin exerts various pharmacological activities which include anti-inflammatory, antitumor, antibacterial and antimutagenic activities.[30] Furthermore Ahmed et al.[31] reported the anti-hyperglycemic and anti-hyperlipidemic activities of rutin in streptozotocin-induced diabetic rats. Also, Oboh et al.[32] reported that chlorogenic and caffeic acids exhibited significant antioxidant properties and, α-amylase and α-glucosidase inhibitory effects *in vitro*. Caffeic acid showed significantly higher antioxidant and enzyme (α-amylase and α-glucosidase) inhibitory effects than chlorogenic acid. Furthermore, the antioxidant and inhibitory effect of gallic acid on activities of α-amylase and α-glucosidase has also been reported.[8] Therefore, it is suggestive that while the characterized phenolic compounds in Raffia palm leaf exhibited potential antidiabetic properties, possible interactions between individual phenolic compounds contributing to the observed antioxidant and enzyme inhibitory properties of the Raffia palm leaf extract cannot be ruled out.

Synthetic drugs such as acarbose have been linked to higher inhibition of α-amylase activity; however, the effective use of this drug is limited by its side effects such as abdominal distention, flatulence, meteorism, and possibly diarrhea which are induced by excessive inhibition of α-amylase.[8] Previous reports have shown that the mild α-amylase inhibitory activity and stronger α-glucosidase inhibitory activities exhibited by plant phenolic extracts could offer reduced side effects.[33] Therefore, the observed higher inhibitory effect of the extract on α-glucosidase activity than α-amylase, could suggest its use as an alternative anti-hyperglycemic agent with minimal side effects.

Consistent increase in hyperglycemia has been reported to induce the generation of ROS via oxidative phosphorylation, glucose autooxidation and some enzyme catalyzed reactions which could lead to pancreatic cell dysfunction and cell death.[34,35] Previous research has shown that pancreatic β-cells milieu are rich in oxygen, glucose and has weak antioxidant defense system.[36] These factors make the cells susceptible to free radical attack which can cause pancreatic β-cell death and/or impairment in insulin secretion.[37] However, there are reports that antioxidants play a protective role against cell damage by preventing the generation of free radicals and neutralizing and/or scavenging free radicals.[38] The ferric reducing antioxidant property (FRAP) and DPPH radical scavenging assays are common assays used to determine the

antioxidant activity of plant extracts.[39] The studied plant displayed significant antioxidant activity by reduction of Fe^{3+} to Fe^{2+}. The extract also scavenged DPPH radicals with a maximum scavenging ability of 71.4% which indicates that it could serve as a good antioxidant agent. The reducing property and radical scavenging ability of the extract correlates with its high phenolic and flavonoid contents, which agrees with previous reports that high antioxidant activity is associated with high phenolic content.[40–42]

Another approach to prevent pancreatic cell damage involves chelation of transition metal ions such as Fe^{2+}. Fe^{2+} can breakdown hydrogen peroxide produced from normal physiological process via mitochondrial electron transport and metal-induced catalyzed reaction to form highly reactive hydroxyl (OH) radical, which can induce oxidative damage by abstracting an electron from polyunsaturated fatty acids in the β-cells to form a lipid radicals.[7] These lipid radicals can further interact with molecular oxygen or superoxide to form lipid peroxyl radical which can initiate lipid peroxidation reaction and form toxic products, such as malondialdehyde (MDA). The chelating ability and reducing property of the extract indicates that the extract is capable of preventing oxidative stress induced-cell death caused by highly reactive hydroxyl radicals.[43,44] Furthermore, the observed inhibition of Fe^{2+}-induced lipid peroxidation in rat pancreas by the extract, as revealed by the decreased MDA production is also an indication of its protective role against β-cell injury. Previous experimental investigations have revealed that Fe accumulates in the acinar cells and islets of Langerhans in the pancreas, thereby inducing lipid peroxidation which could cause the destruction of β-cell.[45] The significant increase in MDA production could be due to the fact that Fe^{2+} can catalyze electron transfer reactions which can lead to production of OH radicals via decomposition of hydrogen peroxide. Moreover, high level of lipid peroxidation products has been implicated in diabetic subjects.[7] Our findings showed that the extract can inhibit MDA production and invariably prevent pancreatic cell injury and oxidative stress. The inhibition of MDA production by the extract could be linked to its Fe^{2+}-chelating ability which could be attributed to the presence of some phenolic compounds present in the extract. These compounds have the capacity to chelate or form complexes with iron thereby preventing metal-induced lipid peroxidation.[46]

5. Conclusion

This study revealed that aqueous extract from Raffia palm (R. Hookeri) leaves exhibited antioxidant properties and inhibited carbohydrate hydrolyzing enzymes which could be linked to its phenolic constituents. The biological effects exhibited by the extract could be of importance in reducing blood glucose level and attenuating free radical-induced oxidative stress in pancreatic cells which are essential in the management of T2DM. Nevertheless, further in vivo study and clinical trials are highly recommended.

Conflict of interest

The authors declare no conflict of interest.

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