Effect of lemongrass (*Cymbopogon citratus* Stapf) tea in a type 2 diabetes rat model

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Abstract

Background: Lemongrass (*Cymbopogon citratus* Stapf) tea is a widely consumed beverage for nourishment and the remedy of diabetes mellitus (DM) in Africa locally. The aim of the present study was to investigate the antidiabetic action of lemongrass tea (LGT) in a type 2 diabetes (T2D) model of rats.

Methods: The fructose-streptozotocin (STZ) animal model for T2D was used and the LGT was prepared by boiling for 10 min in water, allowed to cool and administered at 0.25 or 0.5% (ad libitum), for 4 weeks to the T2D rats.

Results: The LGT showed higher phytochemical contents compared to the cold-water extract. The diabetic untreated animals exhibited significantly ($p < 0.05$) higher serum glucose and lipids, insulin resistance (HOMA-IR) index with a significantly lower ($p < 0.05$) levels of serum insulin, β-cell function (HOMA-β) and liver glycogen compared to the normal animals. Oral supplemented of LGT for 4 weeks improved these changes comparable to the metformin treated group.

Conclusion: The data suggests that LGT intake had excellent antidiabetic effect in a T2D model of rats attributed to the higher content of the ingredients.

Keywords: Antidiabetic, Lemongrass, Tea, Type 2 diabetes, Rats

Introduction

Diabetes mellitus (DM) is a metabolic derangement associated with sustained hyperglycemia due to the defective insulin secretion and/or insulin action, resulting to changes in the generation of energy [1]. Recent data has shown that DM affected over 500 million people globally in 2018, and this number is likely to double by 2045 [2]. Type 1 (T1D) and type 2 (T2D) are the major classes of DM with T2D being the most prevalent of all diabetes cases. T2D is marked with insulin resistance and partially dysfunctional pancreatic β-cells to adequately secrete insulin in response to hyperglycemia [3]. Considerable evidence from preclinical and clinical studies has supported the significance of nutraceuticals, functional foods, and dietary patterns in the treatment of T2D [4–6]. In recent time, diet and food-based

therapies for T2D are receiving much attention due to the fear of insulin injection and intake of oral hypoglycemic drugs. In addition, the perceived short- and/or long-term side effects associated with conventional oral hypoglycemic drugs have greatly influence the shift to food-based therapy [7]. These have prompted the increasing search for potent food-derived ingredients as possible alternative therapies for T2D and *Cymbopogon citratus* Stapf (Poacae) is among the promising functional food with various medicinal and nutritional potential.

The *C. citratus* commonly known as lemongrass is native from Asia, Africa, and the Americas, but is widely cultivated in temperate and tropical regions. It is an aromatic grass-like plant, with long slender green leaves and is widely distributed in the tropical and subtropical countries [8]. For several decades, lemongrass has been reported to be extensively used for a number of folkloric, cosmetic, and nutritional purposes. In Nigeria, Egypt, South Africa and Tanzania, lemongrass tea (LGT) is

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consumed for the treatment of DM and other related disorders such as hypertension and obesity [9–12]. Major active ingredients of LGT were phenolics, flavonoids and terpenoids of which the contents were reported to be higher compared to cold water extract [13].

In some previous studies aqueous extract of lemongrass was shown to reduce blood glucose and lipid profiles levels in non-diabetic [11, 14] and type 1 diabetic rats [15]. The major limitation to these studies was poor extraction method as most of active ingredients of lemongrass such as citral, limonene and linalool, were volatiles and cold extraction may not properly extract the bioactive compounds. Furthermore, boiling has been the widely method used for the preparation of lemongrass as beverage for nourishment and for the traditional treatment of diabetes across most parts of the world. Interestingly, Bharti et al. [16] have reported the hypoglycemic, hypolipidemic and antioxidant potential of lemongrass essential oils in hyperlipidemic rats. The main constrains to this study were lack of using T2D animal model and essential oils may not contain some polar component of lemongrass such as the flavonoids. Thus, to the best of our knowledge, the scientific evidence to validate the use of the LGT in the treatment of diabetes, precisely T2D has not been investigated, despite its consumption for medicinal and nutritional purposes. To further support the selection of LGT, Oboh et al. [13] have reported higher antioxidant property of the LGT than the cold-water extract due to the higher phytochemical content. Therefore, our present study is designed to investigate the antidiabetic potential of the LGT on T2D rat model.

Materials and methods
Plant material
Fresh lemongrass was collected in January, 2018 from Zaria, Kaduna State, Nigeria. The sample was authenticated at the herbarium unit of the Biological Science Department, Ahmadu Bello University, Zaria, Nigeria and a voucher specimen number 1882 was deposited accordingly. The leaf sample was immediately washed and shade-dried for 2 weeks to constant weight and was ground to a fine powder.

Tea preparation
The lemongrass tea (LGT) preparation was prepared by mimicking the preparation protocols used locally for the treatment of diabetes or consumed for nourishment and was in accordance with Islam [17] method. Briefly, the fine powdered samples; 0.25 g/100 ml and 0.5 g/100 ml were prepared by boiling for 10 min in water, allowed to cool to room temperature and filtered through Whatmann filter paper (No. 1). The filtrates were supplied to the animals during the 4 weeks intervention period ad libitum. For the phytochemical analysis, the filtrate was evaporated using a rotary evaporator to obtain the extract.

Phytochemical analysis
Flavonoid content
The method of Bohm and Koupai-Abyazani [18] was used for the quantification of flavonoids. Briefly, 10 g of the samples were repeatedly extracted in 100 ml of 80% methanol. The mixtures were filtered using Whatman filter paper No. 42 (125 mm). The filtrates were transferred into crucible and evaporated into dryness in water bath and weighed to constant weight.

Total phenolic content
Folin-Ciocalteu method of Chang et al. [19] was used to quantify phenolic content of the sample. Five grams (5 g) of the samples were boiled in 50 ml of ether for 15 min and then 5 ml of the extracts were pipetted into 50 ml flask. 2 mL of ammonium hydroxide solution and 5 ml of amyl alcohol were also added to the samples and made up to the mark. It was left to react for 30 min for colour development; the absorbance was measured at 550 nm. Gallic acid was used for calibration of a standard curve. The results are expressed as mg gallic acid equivalents (mgGAE)/g dry weight of the plant tissue.

Alkaloid content
The Naili et al. [20] method was used to determine the alkaloid content. 5 g of the samples were weighed into a 250 ml beaker and 200 mL of 10% acetic acid in ethanol was added and they were covered and allowed to stand for 4 h. The mixtures were filtered and the extracts were concentrated on a water bath to one-quarter of the original volume. Concentrated ammonium hydroxide was added dropwise to each of the extracts until the precipitation was complete. The whole solutions were allowed to settle and the precipitates were collected and washed with dilute ammonium hydroxide and then filtered. The residues were weighed and recorded as alkaloid content.

Saponin content
The method of Obadoni and Ochuko [21] was used to determine the saponin content. 20 g of the samples were put into a conical flask and 100 mL of 20% aqueous ethanol was added. The mixtures were heated over a hot water bath for 4 h with continuous stirring at about 55 °C. The mixtures were filtered and the residues reextracted with another 200 mL 20% ethanol. The extract was reduced to 40 mL over water bath at about 90 °C. The concentrates were transferred into a 250 mL separatory funnel and 20 mL of diethyl ether was added and they were shaken vigorously. The aqueous layer was recovered while the ether layer was discarded. The

purification process was repeated. A volume of 60 mL of n-butanol was added. The combined n-butanol extracts were washed twice with 10 mL of 5% aqueous sodium chloride. The remaining solutions were heated in a water bath. After evaporation the samples were dried in the oven to constant weight.

Tannin content
The tannin content was assayed according to the AOAC [22]. Aqueous solutions (25 mL) of the preparations were transferred into1 L conical flask, then 25 ml of indigo solution and 750 ml distilled deionised water were added. 0.1 N aqueous solution of $KMnO_4$ was used for titration until the blue coloured solution changes to green colour. Then few drops at time until solution becomes golden yellow. Standard solution of Indigo carmine was prepared The blank tests by titration of a mixture of 25 mL Indigocarmine solution and 750 mL distilled water were carried out.

Experimental animals and grouping
Forty-two (42) male Wistar rats were obtained from the Department of Pharmacology, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, Zaria, Nigeria with initial mean bw of 211.01 ± 17.05 g. The handling and use of the animals were in accordance with the National Institutes for Health Guide for the Care and Use of Laboratory Animals. The handling and use of the experimental animals were approved by the Animal Research Ethical Committee of the Ahmadu Bello University, Zaria, Nigeria. Animals were housed in transparent cages (3 or 4 animals/cage) with a 12 h light-dark cycle and supplied with a standard rat pellet diet ad libitum during the entire experimental period. The animals were randomly divided into six groups of seven animals namely; NC: Normal Control, DBC: Diabetic Control, DLTL, Diabetic + low dose (0.25%) of LGT, DLTH, Diabetic + high dose (0.5%) of LGT, DMF: Diabetic + metformin (150 mg/kg bw), NLTH: Non-diabetic + high dose (0.5%) of LGT. The animals were allowed to acclimatize for 1 week before starting the experiment. The selection of the dosages used for the LGT and standard metformin were based on trial study and available literature as well [23, 24].

Induction of type 2 diabetes (T2D)
In order to induce the two major pathogeneses of T2D, insulin resistance and partial pancreatic β-cell dysfunction, the method of Wilson and Islam [25] was adopted. Briefly, in the initial first 2 weeks of the experiment, the animals in the DBC, DLTL, DLTH and DMF groups were supplied with a 10% fructose solution (ad libitum) for the induction of insulin resistance while the animals in the NC and NLTH groups were supplied with normal

drinking water. After this period and an overnight fast, a low dose of streptozotocin (STZ) at 40 mg/kg bw dissolved in citrate buffer (pH 4.5) were intraperitoneally injected to the animals in the DBC, DLTL, DLTH and DMF groups to induce partial pancreatic β-cell dysfunction, whereas the animals in the NC and NLTH groups were injected with a similar volume of the vehicle buffer. One week after the STZ injection, the fasting blood glucose (FBG) of all animals were measured in the blood collected from the tail vein by using a portable glucometer (Glucoplus Inc., Accucheck). Animals with a FBG level ≥ 200 mg/dl were considered as diabetic [26] while the animals with a FBG level < 200 mg/dl were excluded from the study.

Intervention period
After the confirmation of diabetes, a respective concentration of the LGT was supplied to the animals for 4 weeks ad libitum in DLTL and DTLH and NLTH groups while the animals in controls (NC and DBC) and DMF groups were treated with similar volume of the vehicle and metformin, respectively. Throughout the experimental period, feed and fluid intake were measured every morning by subtracting the remaining amount of feed and fluid respectively, from the amount given on the previous morning. Moreover, the weekly body weight and FBG levels were measured in all animal groups during the entire intervention period.

Oral glucose tolerance test (OGTT)
To measure the glucose tolerance ability of each animal, the OGTT was performed in the last week of the 4-week intervention period. A single dose of glucose solution (2 g/kg bw) was orally administered to each animal and the subsequent levels of blood glucose were measured at 0 (just before glucose ingestion), 30, 60, 90 and 120 min after the ingestion of glucose.

Collection of blood and organs
At the end of the experimental period, animals were sacrificed by anesthesia and the blood and liver were collected. The whole blood of each animal was preserved immediately after collection and centrifuged at 3000 rpm for 15 min. The serum from each blood sample was separated and preserved in refrigerator for further analysis. The liver was collected from each animal, washed with normal saline, wiped with filter paper, weighed and preserved in a refrigerator until subsequent analysis.

Analytical methods
The serum insulin concentration was measured by an enzyme-linked immunosorbent assay (ELISA) method using an ultrasensitive rat insulin ELISA kit. The serum lipid profile, albumin, total protein concentrations as

well as liver function enzymes; aspartate and alanine transaminases (AST and ALT) and alkaline phosphatase (ALP) were measured using commercially available Randox kits, according to the manufactures guide. Homeostatic model assessment (HOMA-IR and HOMA-β) scores were calculated at the end of the intervention period according to the following formula:

$$HOMA-IR = [(\text{Fasting serum insulin in U/l} \\ \text{x Fasting blood glucose in mmol/l})/22.5]$$

$$HOMA-\beta = (\text{Fasting serum insulin in U/l} \\ \text{x 20/Fasting blood glucose in mmol/l}-3.5)$$

$$\text{Conversion factor}: \text{insulin } (1 \text{ U/l} = 7.174 \text{ pmol/l}).$$

LDL-cholesterol was calculated according to Friedewald et al. [27] equation as shown below:

$$LDL-Cholesterol \text{ (mg/dl)} = [TC-HDL-(TG/5)]$$

Liver glycogen concentrations were measured by phenol-sulfuric acid method as described by Good et al. [28]. Liver tissue (1.0 g) was placed in 4.0 ml of KOH (30%) then heated in boiling water for 10 min. After cooling, 0.2 ml of Na_2SO_4 (20%) and 5.0 ml of ethanol (95%) were added and kept at 20 °C for 5 min. After precipitation was completed, the mixture was centrifuged at 3000 x g for 10 min and decanted. The packed glycogen in the tube was dissolved by addition of 5.0 ml distilled water with gentle warming. Exactly 5.0 ml of HCl (1.2 mol/L) was added to 5.0 ml of the sample in test tubes then neutralized by the addition of 2 drops of 0.5 mol/L NaOH and a drop of phenol red as indicator then cooled. Glucose in the sample was measured separately. Briefly, a reagent blank was prepared by pipetting 1.0 ml of distilled water into a clean test tube. Exact 1.0 mL of sample and standard glucose solution (0.5 mg/mL of glucose), was pipetted into a similar tube. Then, 5.0 mL of anthrone reagent was delivered into each tube and tightly capped. The test tubes were placed in a cold-water bath. After all tubes have reached the temperature of the cold water, they were immersed in a boiling water bath to a depth little above the level of the liquid in the tubes for 15 min and was then removed and placed in a cold-water bath and cooled to room temperature. Absorbance of test and standard were read against the reagent blank at 620 nm.

Glycogen (mg/g liver tissue) = (Optical density unknown 0.5 **x** Vol. of sample × 100 × 0.9)/.

Optical density x g of the tissue.

Statistical analysis
All data are presented as the mean ± SD of seven animals. Data were analyzed by using the analysis of variance (ANOVA) (SPSS for Windows, version 22, IBM Corporation, NY, USA) using Tukey's-HSD multiple range post-hoc test. Values were considered significantly different at $p < 0.05$.

Results
Quantitative phytochemical content of LGT
The amount of the phytochemicals presents in the lemongrass tea (LGT) and the cold-water extract is presented in Table 1. With the exception of saponins, all the ingredients quantified were higher in LGT compared to the cold-water extract, though not statistically ($p > 0.05$) different from each other. However, the amount of total phenolics in LGT was significantly ($p < 0.05$) higher compared to the cold-water extract (Table 1).

Effect of LGT on feed and fluid intakes and mean body weight change in T2D rats
The data of the feed and fluid intakes and mean body weight change, are presented in Figs. 1 and 2, respectively. It was observed that induction of T2D significantly ($p < 0.05$) elevated the feed and fluid intakes and reduced the body weight in the diabetic groups compared to NC. After the 4-week LGT intervention, there was a significant ($p < 0.05$) improvement in the feed and fluid intakes of the treated animals (Fig. 1), though the effect on body weight (Fig. 2) was not statistically significant ($p > 0.05$) compared to DBC group as well as within the treated diabetic groups. Additionally, there was no significant ($p > 0.05$) effect within the groups treated with the LGT and standard metformin (Figs. 1 and 2).

Effect of LGT on weekly blood glucose and oral glucose tolerance test in T2D rats
The result of the weekly blood glucose level is presented in Fig. 3. From the data, after the induction of T2D, there was a significant ($p < 0.05$) elevation of fasting blood glucose (FBG) level in the DBC group compared to NC group. Consumption of LGT significant ($p < 0.05$) reduced blood glucose level. In the first and second weeks of LGT treatment, there was significantly ($p < 0.05$) reduction of FBG in DLTH compared to DLTL. However, after 4-week intervention, no significant ($p < 0.05$) effect was observed within the LGT treated groups and were comparable to metformin treated group, when no effect was observed in NLTH group (Fig. 3). Moreover, it was evident from the results of the OGTT that T2D induction significantly ($p < 0.05$) affected the glucose utilization of the DBC group compared to the NC group (Fig. 4). On the other hand, significantly ($p < 0.05$) better glucose tolerance and utilization was observed in the DLTH and DLTL groups compared to the DBC group and was comparable to the DMF group, which

Table 1 Phytochemical contents of lemongrass tea and cold-water extract

Phytochemicals	Lemongrass tea	Lemongrass cold-water extract
Alkaloids (mg/g)	38.00 ± 2.65[a]	32.00 ± 1.73[a]
Flavonoids (mg/g)	68.00 ± 7.55[a]	58.05 ± 1.64[a]
Saponins (mg/g)	22.00 ± 2.65[a]	24.00 ± 5.29[a]
Tannins (mg/g)	69.00 ± 4.58[a]	51.00 ± 2.00[a]
Total phenolics (mgGAE/g)	111.00 ± 5.57[a]	88.00 ± 3.61[b]

Data are shown as mean ± SD. Values with different letters across the rows for a given parameter are significantly different from each other

again was more prominent in DLTH than DLTL and DMF groups (Fig. 4).

Effect of LGT on serum insulin and the calculated HOMA-IR and HOMA-β indices and other biochemical parameters in T2D rats

The results of serum insulin and the calculated HOMA-IR and HOMA-β indices are presented in Table 2. According to the data, the serum insulin level and the calculated HOMA-β index were decreased significantly ($p < 0.05$) when HOMA-IR index was increased significantly ($p < 0.05$) in the DBC group compared to the NC group (Table 2). After 4-week intervention of LGT, there was significant ($p < 0.05$) and dose-dependent increase in serum insulin levels and the calculated HOMA-β index with a concomitant decrease of HOMA-IR index. These parameters were not altered in the NLTH compared to NC groups (Table 2). It was also observed that the liver glycogen content, serum AST and ALP levels were significantly ($p < 0.05$) decreased whereas serum ALT, total proteins and albumin were elevated in the DBC group compared to the NC group (Table 3). Treatment with LGT modulated these alterations by reverting

to near normal (Table 3). In addition, despite no significant ($p > 0.05$) difference was observed within the treated groups in liver glycogen content, serum ALT, AST, ALP, and albumin levels, the effects were more pronounced in DLTH than DLTL groups (Table 3).

Effect of LGT on serum lipid profiles in T2D rats

The serum total cholesterol (TC), triglycerides (TG) and low-density lipoprotein (LDL) cholesterol were significantly ($p < 0.05$) decreased in the DBC group compared to the NC group (Fig. 5). Consumption of LGT to the diabetic animals significantly ($p < 0.05$) decreased serum TC, TG and LDL-cholesterol levels compared to the DBC group. Moreover, HDL-cholesterol level in DBC group increased but not significantly ($p < 0.05$) different compared to the NC group. Treatment of LGT further reduced HDL-cholesterol, though not statistically significant ($p < 0.05$) compared to the DBC group Additionally, the levels of the above-mentioned parameters were not affected in NLTH group at the end of the intervention period (Fig. 5).
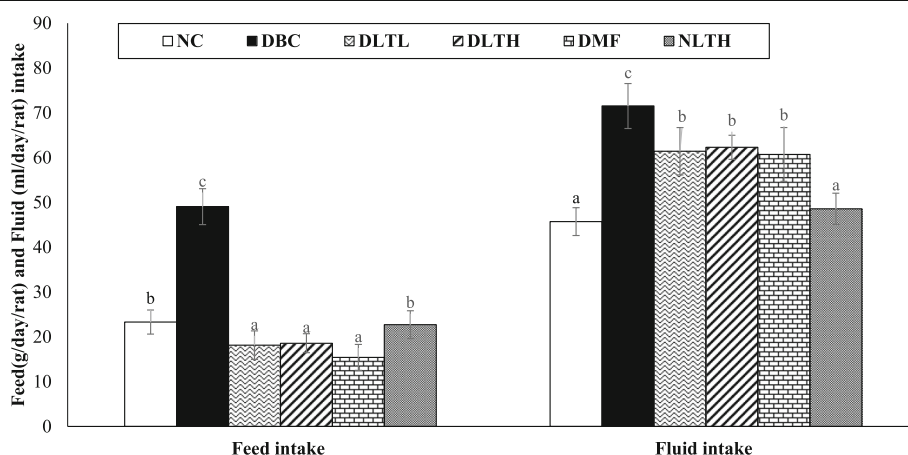
Fig. 1 Feed and fluid intake in different animal groups during the experimental period. Data are presented as the mean ± SD of 7 animals. [a–c]Values with different letters over the bars for a given parameter are significantly different from each other group of animals (Tukey's-HSD multiple range post hoc test, $p < 0.05$). NC- Normal control; DBC- Diabetic control; DLTL- Diabetic lemongrass tea low; DLTH- Diabetic lemongrass tea high; DMF- Diabetic metformin; NLTH- Normal lemongrass tea high
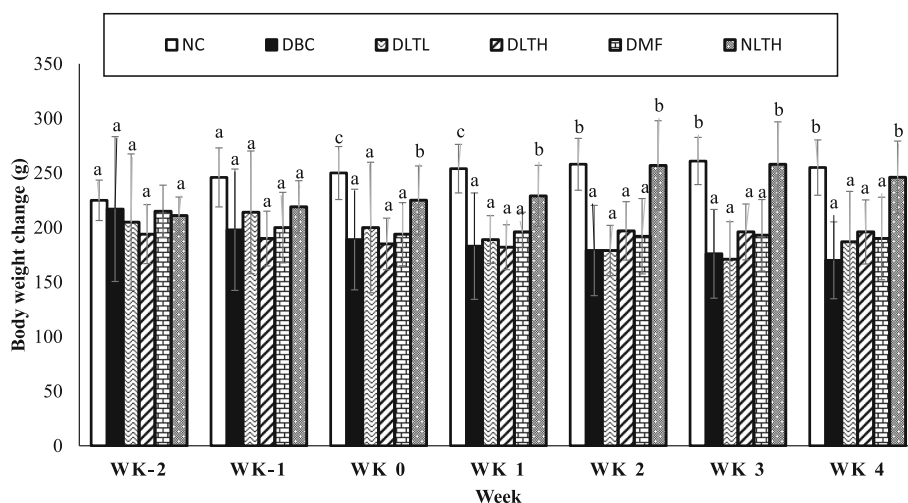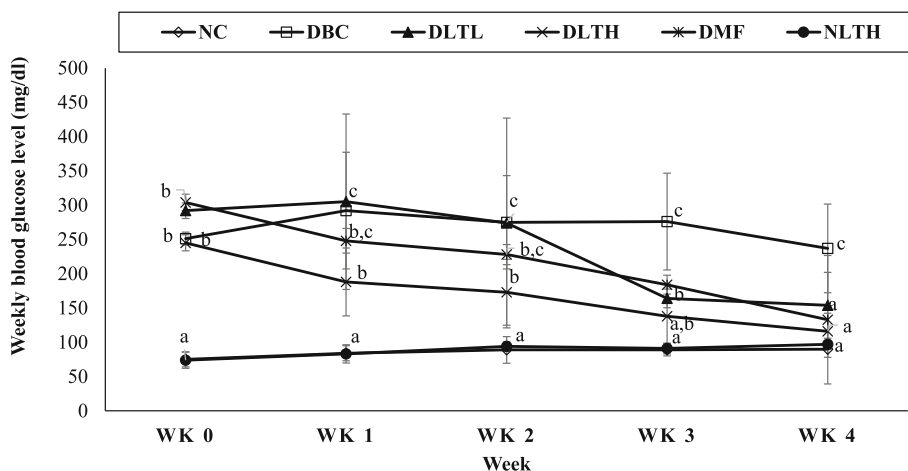
Fig. 2 Mean body weight changes in different animal groups during the experimental period. Data are presented as the mean ± SD of 7 animals. [a,c]Values with different letters over the bars for a given parameter are significantly different from each other group of animals (Tukey's-HSD multiple range post hoc test, $p < 0.05$). NC- Normal control; DBC- Diabetic control; DLTL- Diabetic lemongrass tea low; DLTH- Diabetic lemongrass tea high; DMF- Diabetic metformin; NLTH- Normal lemongrass tea high

Discussion

Our present study showed that the LGT consumption for 4 weeks at both dosages reduced blood glucose levels, improved postprandial glucose utilization, ameliorated insulin resistance, hyperlidemia and alterations in some biochemical parameters in T2D rat model. This could be the first report that adequately reported the antidiabetic potential of LGT in T2D rat model. The LGT had higher content of the phytochemicals compared to the cold-water extract, with phenolics having the highest content (Table 1). This accord to several previous studies that shows dramatic increase in total phenolics, tannins and alkaloids contents of fruits and vegetables when boiled

for less than 30 min than the cold-water extracts, though reason remains speculative [29–31]. It has been proposed that boiling improves the contact of the phytochemicals with water molecules which subsequently enhances extraction efficiency and in turn greater content of the ingredients in the boiled extract [32]. Interestingly, Oboh et al. [13] have shown an increased antioxidant potential of LGT compared to the cold-water extract due to the higher content of the ingredients in LGT. This again supports the selection of LGT for the present study in addition to the widely acclaimed health benefits in the treatment of several diseases including T2D.

Fig. 3 Weekly blood glucose levels of different animal groups during the experimental period. Data are presented as the mean ± SD of 7 animals. [a–c]Values with different letters over the bars for a given parameter are significantly different from each other group of animals (Tukey's-HSD multiple range post hoc test, $p < 0.05$). NC- Normal control; DBC- Diabetic control; DLTL- Diabetic lemongrass tea low; DLTH- Diabetic lemongrass tea high; DMF- Diabetic metformin; NLTH- Normal lemongrass tea high
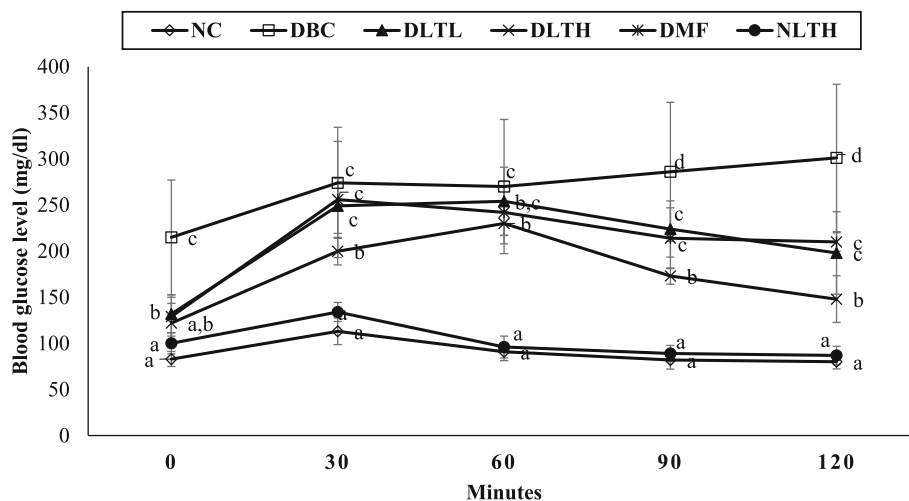
Fig. 4 OGTT of different animal groups during the experimental period. Data are presented as the mean ± SD of 6 animals. [a–c]Values with different letters over the bars for a given parameter are significantly different from each other group of animals (Tukey's-HSD multiple range post hoc test, $p < 0.05$). NC- Normal control; DBC- Diabetic control; DLTL- Diabetic lemongrass tea low; DLTH- Diabetic lemongrass tea high; DMF- Diabetic metformin; NLTH- Normal lemongrass tea high

Our data showed that induction of T2D decreased the mean body weight, caused polyphagia and polydipsia in the diabetic untreated animals. These, might be linked to the increased energy expenditure, excessive fluid retention and increased eating habit to compensate for the body weight loss in uncontrolled T2D condition [33]. Consumption of LGT for 4-weeks ameliorated these alterations, signifying possible recovery from the diabetic state. Our results are in line with the previous studies that used cold-water extract [11, 14] which have been further supported by the significant reduction in hyperglycemia in LGT treated groups.

Successful reduction of either fasting or postprandial hyperglycemia remains a prime target in the prevention and treatment of T2D and its associated complications [34]. In the present study, induction of T2D elevated the blood glucose levels and was sustained throughout the study period, indicating the success of the induction. Administration of LGT to the diabetic animals reverted the alteration to near normal (Fig. 3). In some previous studies, treatment of lemongrass ethanol and aqueous extracts to non-diabetic animals reduced blood glucose by 21.3% and 19.8%, respectively [11, 14]. Similarly, administration of essential oil or aqueous extract reduced blood glucose by about 47.3% and 42.4% in

hyperlipidemic and diabetic animals, respectively [15, 16]. However, in our present study, consumption of LGT reduced blood glucose by 60.3% indicating greater efficacy, attributed to the higher phytochemical contents compared to other extracts in the literature. Our finding was further supported by higher glucose tolerance ability of the LGT diabetic treated compared to the untreated diabetic rats (Fig. 4). This again could partly support the potent inhibitory ability of the lemongrass on the activities of α-amylase and α-glucosidase action reported previously [10]. Similarly, citral, limonene and linalool, active ingredients of LGT were shown to reduced hyperglycemia and attenuated diabetes-associated complications [35–37]. Therefore, the antihyperglycemic activity observed in our present study could be linked to the individual or combined action of these phytochemicals.

Studies have shown that chronic uncontrolled T2D may led to reduction of circulating insulin levels, and alter the pancreatic integrity and function [38, 39], which were observed in our present study. In addition, the pancreatic β-cell function was greatly tempered with the appearance of insulin resistance in untreated diabetic rats. Consumption of LGT improved the pancreatic integrity via increased serum insulin level, improving pancreatic β-cell function and attenuation of insulin

Table 2 Serum insulin level, HOMA-IR and HOMA-β scores of type 2 diabetic rats given lemongrass tea ad libitum

Groups	NC	DBC	DLTL	DLTH	DMF	NLTH
Insulin (pmol/l)	251.60 ± 50.10[d]	147.40 ± 80.90[a]	208.10 ± 80.80[b]	214.70 ± 21.00[b,c]	225.30 ± 24.40[c]	268.90 ± 57.90[d]
HOMA-IR	55.90 ± 11.10[a]	86.30 ± 47.30[c]	76.40 ± 28.60[b]	61.50 ± 6.00[a]	74.00 ± 8.00[b]	64.40 ± 13.90[a]
HOMA-β	15.90 ± 3.18[d]	3.55 ± 1.95[a]	7.72 ± 3.00[b]	10.60 ± 1.03[c]	9.68 ± 1.04[c]	15.40 ± 3.24[d]

Data are shown as mean ± SD. Values with different letters across the rows for a given parameter are significantly different from each other. *NC* normal control, *DBC* diabetic control, *DLTL* diabetic lemongrass tea low, *DLTH* diabetic lemongrass tea high, *DMF* diabetic metformin, *NLTH* normal lemongrass tea high

Table 3 Serum biochemical parameters and liver glycogen content of type 2 diabetic rats given lemongrass tea ad libitum

|	NC	DBC	DLTL	DLTH	DMF	NLTH
ALT (U/l)	11.60 ± 1.52[a]	19.40 ± 2.30[c]	15.40 ± 2.30[b]	14.80 ± 2.59[b]	18.40 ± 1.67[c]	11.00 ± 2.71[a]
AST (U/l)	183.00 ± 22.90[e]	87.60 ± 14.35[a]	138.40 ± 21.56[b,c]	145.4 ± 23.14[c]	136.40 ± 21.02[b]	173.75 ± 23.00[d]
ALP (U/l)	10.72 ± 2.88[b]	6.70 ± 0.76[a]	8.50 ± 1.46[a,b]	7.99 ± 0.92[a]	9.00 ± 0.59[b]	10.80 ± 3.00[b]
Albumin (g/dl)	2.49 ± 0.58[a]	3.24 ± 0.20[b]	2.90 ± 0.51[a,b]	3.14 ± 0.31[b]	3.48 ± 0.20[b]	2.30 ± 0.34[a]
Total Protein (g/dl)	4.00 ± 1.34[a]	4.87 ± 0.91[b]	4.15 ± 0.95[a]	5.78 ± 0.97[c]	4.59 ± 0.89[b]	4.06 ± 0.40[a]
Glycogen (mg/g tissue)	10.72 ± 1.07[c]	3.68 ± 0.29[a]	6.32 ± 1.29[b]	6.66 ± 0.64[b]	5.78 ± 1.28[b]	8.82 ± 2.75[c]

Data are shown as mean ± SD. Values with different letters across the rows for a given parameter are significantly different from each other. NC normal control, DBC diabetic control, DLTL diabetic lemongrass tea low, DLTH diabetic lemongrass tea high, DMF diabetic metformin, NLTH normal lemongrass tea high

resistance (Table 2). This could explain the drastic reduction of the blood glucose level observed as elevated circulating insulin and improved β-cell function facilitated the movement of glucose in to the cells for the energy production.

Consumption of high-fructose diet has been associated with increased hepatic and muscle lipid deposition and accumulation in T2D, via stimulation of lipogenesis [40]. Previously, oral treatment of lemongrass alcoholic or aqueous extracts (200–1000 mg/kg bw) to non-diabetic, hyperlipidemic and diabetic animals exhibited antihyperlipidemic action [11, 14, 41]. This accord with our present data and further confirms the potential of lemongrass in reducing hyperlipidemia associated with diabetes. The elevated HDL-cholesterol in DBC group although not significantly ($p > 0.05$) different with the NC could be due to the ability of body system to produce more HDL-cholesterol to neutralize the negative effect of higher levels of TC, TG and LDL-cholesterol.

On the other hand, treatment of the lemongrass alcoholic extract for 2 weeks showed no effect on the serum total protein, albumin, ALT, AST, ALP and urea [41]. However, in our present study, there was reduction of the alterations after LGT administration (Table 3), signifying better effect of the LGT compared to the alcoholic extract. This could be attributed to the greater amount of the phytochemicals and the longer study period. The lower glycogen content of the DBC group in our study could be linked to the stimulation of the hepatic glycogen phosphorylase, which apparently may increase glucose output and eventually complicates the hyperglycemia in diabetic condition. Interestingly, treatment of LGT ameliorated the alteration and further shows the ability of the LGT to reverse this alteration associated with diabetes. Phenolics and terpenes which are active ingredients of LGT were shown to increase hepatic glucokinase activity, which augments glucose utilization to promote energy storage in the form of glycogen [42]. Hence, the increased glycogen content observed in the treated groups could be attributed to the inhibition of glucokinase activity by the major active ingredients. This probably could be the mechanism of antidiabetic effect of LGT.
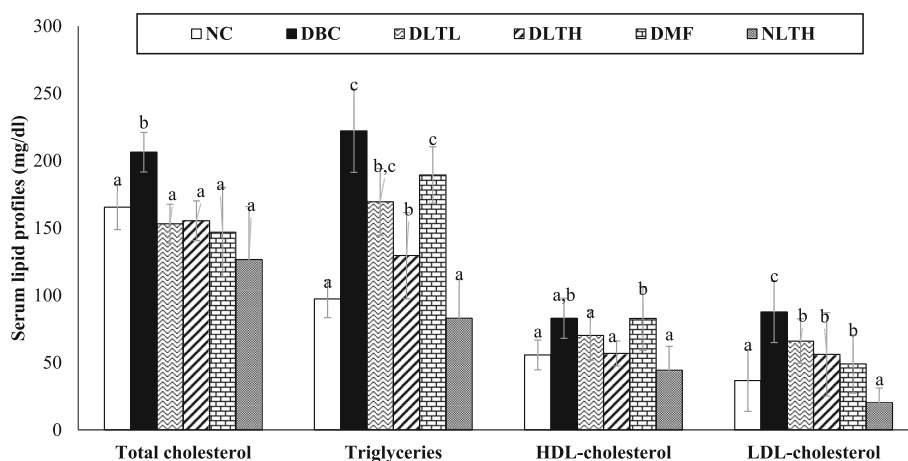
Fig. 5 Lipid profile levels of different animal groups during the experimental period. Data are presented as the mean ± SD of 7 animals. [a–c]Values with different letters over the bars for a given parameter are significantly different from each other group of animals (Tukey's-HSD multiple range post hoc test, $p < 0.05$). NC- Normal control; DBC- Diabetic control; DLTL- Diabetic lemongrass tea low; DLTH- Diabetic lemongrass tea high; DMF- Diabetic metformin; NLTH- Normal lemongrass tea high

Conclusion

In conclusion, oral intervention of LGT demonstrated antidiabetic actions via improving body weight gain, reducing food and fluid intake and hyperglycemia, improving glucose tolerance ability, insulin sensitivity, β-cell functions and dyslipidemia in T2D model of rats. Hence, our findings suggest that consumption of LGT may provide a good management option for T2D patients with no considerable side effects which also supports the antidiabetic claims of the tea. Further clinical study is required to confirm the effects in human subjects and specific active ingredient responsible for the observed action.

Abbreviations
ALP: Alkaline phosphatase; ALT: Alanine transaminase; AST: Aspartate transaminase; DBC: Diabetic control; DMF: Diabetic metformin; DLTH: Diabetic lemongrass tea high dose; DLTL: Diabetic lemongrass tea low dose; ELISA: Enzyme-linked immunosorbent assay; HOMA-β: Homeostatic model assessment-β cell function; HOMA-IR: Homeostatic model assessment-insulin resistance; LGT: Lemongrass tea; NC: Normal control; NFBG: Non-fasting blood glucose; NLTH: Normal lemongrass tea high dose; OGTT: Oral glucose tolerance test; T2D: Type 2 diabetes; STZ: Streptozotocin

Authors' contributions
H.A Garba performed experiments and provided equipment and re-agents; A. Mohammed conducted the statistical analysis and wrote the manuscript; M. A Ibrahim and M. N Shuaibu made manuscript revisions. The authors read and approved the final manuscript.

Authors' information
Not applicable.

Availability of data and materials
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate
The animal study was conducted in strict compliance with the Animal Research Ethical Committee guide of the Ahmadu Bello University, Zaria, Nigeria.

Consent for publication
Not applicable.

Competing interests
We declare that we have no competing interest within this article.

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