Changes in phenolic composition, antioxidant and antidiabetic properties of turmeric as affected by cooking time
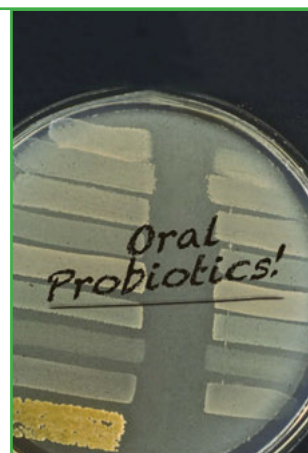
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Abstract

This study sought to assess the effects of cooking time on the phenolic composition, antioxidant and antidiabetic properties of turmeric extracts. Steam cooked turmeric extracts were prepared by cooking 10 g of turmeric for 10 (SCT_{10}) and 20 (SCT_{20}) min, respectively, while the raw sample (RWT) was prepared by soaking 10 g of turmeric in 200 ml of distilled water for 1 h. The total phenol and flavonoid contents of the turmeric extracts were determined and phenolic composition was assessed using high performance liquid chromatography coupled with diode array detection (HPLC-DAD). The ferric reducing antioxidant property, radical [1,1-diphenyl-2 picrylhydrazyl (DPPH), nitric oxide (NO) and hydroxyl (OH)] scavenging abilities and interaction of the extracts with α-amylase and α-glucosidase activities were also investigated. SCT_{20} (4.26 mg/g and 3.96 mg/g) had significantly higher total phenol and flavonoid contents than SCT_{10} (3.58 mg GAE/g and 3.38 mg QE/g) and RWT (2.44 mg/g and 2.38 mg/g), respectively.

SCT_{20} (76.8 mmol/100 g) had the highest reducing property, while RWT (68.2 mmol AAE/100 g) had the lowest. Furthermore, SCT_{20} had significantly ($p<0.05$) higher DPPH, NO and OH radical scavenging ability than SCT_{10} and RWT. The extracts inhibited α-amylase and α-glucosidase in a dose-dependent manner. While RWT had the lowest inhibitory effects, SCT_{20} had the highest. The HPLC analysis revealed the presence of phenolic compounds such as gallic acid, caffeic acid, catechin, quercetin, rutin, luteolin and circumin. The phenolic constituents of the steam cooked extracts were significantly higher than those of the raw samples except for caffeic acid which decreased with increased cooking time. Therefore, steam cooking increased the phenolic composition and antioxidant properties as well as the α-amylase and α-glucosidase inhibitory activities of turmeric.

Introduction

Turmeric is a commonly used spice obtained from *Curcuma longa* L., a rhizomatous herbaceous plant of the ginger family, which is widely cultivated in tropical and subtropical regions of the world [1]. In Ayurvedic and traditional Chinese medicine, turmeric is used for the treatment of inflammatory conditions and as a stimulant, carminative, astringent and diuretic [2-4]. Some reports have revealed that turmeric extracts have cardio-protec-

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tive, hypoglycaemic, anti-amyloidogenic, antimicrobial, anticancer and antioxidant activity [5-10]. Turmeric is known to be a good source of natural antioxidants that can protect cells from oxidative damage [11,12]. Oxidative stress, which is characterized by free radical generation, has been linked to the aetiology and pathophysiology of type 2 diabetes. High blood glucose is a major feature of type 2 diabetes and control of post-prandial blood glucose requires inhibition of carbohydrate hydrolyzing enzymes (α-amylase and α-glucosidase), which is a good therapeutic approach for the management and/or treatment of type 2 diabetes. Phytochemical compounds with strong antioxidant properties are known to be strong inhibitors of these enzymes and it has been demonstrated that turmeric has promising potential [13,14].

Turmeric is commonly consumed in raw or cooked form. Various reports have revealed that heat processing of some spices and vegetables could lead to loss of some bioactive compounds, which can reduce their functional and medicinal properties [15-17]. However, conflicting reports have revealed that some heat processing methods can increase the phenolic content and antioxidant activity of spices, fruits and vegetables [18,19].

Nevertheless, to the best of our knowledge there is little or no information about the effect of cooking on the antioxidant and antidiabetic properties of turmeric. This study therefore sought to investigate the effect of steam cooking on the phenolic composition, antioxidant and antidiabetic properties of turmeric extracts.

Materials and methods

Extract collection and identification
Dried turmeric (*Curcuma longa* L.) rhizomes were purchased at Oja-Oba market in Akure city, Nigeria. Authentication of the sample was carried out in the Department of Biology, Federal University of Technology, Akure, Ondo State, with voucher number FUTA/BIO/0139.

Extract preparation
Turmeric (10 g) was steam cooked for 10 min (SCT_{10}) and 20 min (SCT_{20}), respectively, while 10 g of raw turmeric (RWT) was soaked in 200 ml

of distilled water for 1 h. The samples were allowed to cool and filtered with filter paper. The filtrates from RWT, SCT_{10} and SCT_{20} were kept in a refrigerator until analysis.

Chemicals and reagents
Thiobarbituric acid (TBA), 1,10-phenanthroline, deoxyribose, gallic acid and Folin–Ciocalteau's reagent were procured from Sigma-Aldrich (St Louis, MO), while trichloroacetic acid (TCA) was from Sigma-Aldrich Chemie (Steinheim, Germany). Hydrogen peroxide, methanol, sodium nitroprusside, Griess reagent, acetic acid, hydrochloric acid, sodium carbonate, aluminum chloride, potassium acetate, sodium dodecyl sulphate, iron (II) sulfate, potassium ferricyanide and ferric chloride were from BDH Chemicals (Poole, UK). Unless stated otherwise, all the chemicals and reagents used were of analytical grades and the water was glass distilled.

Determination of total phenol content
Total phenol content was determined according to the method of Singleton et al [20]. Briefly, 0.5 ml extracts were oxidized with 2.5 ml 10 % Folin-Ciocalteau's reagent (v/v) and neutralized with 2.0 ml 7.5 % sodium carbonate. The reaction mixture was incubated for 40 min at 45 °C and the absorbance was measured at 765 nm in a spectrophotometer. The total phenol content was subsequently calculated as gallic acid equivalent.

Determination of total flavonoid content
The total flavonoid content of the extracts was determined using a slightly modified method reported by Meda et al [21]. Briefly, 0.5 ml appropriately diluted extracts (1:10) were mixed with 0.5 ml methanol, 50 μl 10 % $AlCl_3$, 50 μl 1 M potassium acetate and 1.4 ml water and allowed to incubate at room temperature for 30 min. The absorbance of the reaction mixture was subsequently measured at 415 nm and the total flavonoid content calculated as quercetin equivalent.

Quantification of compounds by high performance liquid chromatography coupled with diode array detection (HPLC-DAD)
Reverse phase chromatographic analyses were car-

ried out under gradient conditions using a C_{18} column (4.6 mm × 150 mm) packed with 5 μm diameter particles; the mobile phase was water containing 2 % acetic acid (A) and methanol (B), and the composition gradient was: 5% B until 2 min and 25%, 40%, 50%, 60%, 70% and 100% B at 10, 20, 30, 40, 50 and 80 min, respectively, following the method described by Adefegha et al [22] with slight modifications. RWT, STC10 and SCT20 extracts were analyzed at a concentration of 15 mg/ml. The presence of nine antioxidant compounds was investigated, namely, gallic acid, caffeic acid, catechin, epicatechin, quercetin, quercitrin, rutin, luteolin and curcumin. These compounds were identified by comparing their retention time and UV absorption spectrum with those of the commercial standards. The flow rate was 0.6 ml/min, injection volume 50 μl and the wavelengths were 270 nm for gallic acid, 281 nm for catechin and epicatechin, 327 nm for caffeic acid, 365 nm for quercetin, quercitrin, rutin and luteolin, and 426 nm for curcumin. The extracts and mobile phase were filtered through 0.45 μm membrane filter (Millipore) and then degassed in an ultrasonic bath before use. Stock solutions of reference standards were prepared in the HPLC mobile phase at a concentration range of 0.030–0.200 mg/ml for catechin, epicatechin, quercetin, quercitrin, rutin, curcumin and luteolin, and 0.025–0.300 mg/ml for caffeic and gallic acids. The chromatography peaks were confirmed by comparing their retention times with those of reference standards and by DAD spectra (200–500 nm). Calibration curves were as follows: for catechin: Y = 11965x + 1273.8 (r = 0.9999); epicatechin: Y = 13065x + 1187.6 (r = 0.9996); gallic acid: Y = 11890x + 1193.7 (r = 0.9997); caffeic acid: Y = 12549x + 1307.2 (r = 0.9998); rutin: Y = 12853x + 1238.9 (r = 0.9991); quercetin: Y = 13574x + 1271.9 (r = 0.9995); quercitrin: Y = 11965x + 1358.7 (r = 0.9995); curcumin: Y = 12895x + 1289.8 (r = 0.9999); and luteolin Y = 13509x + 1185.4 (r = 0.9998). All chromatography operations were carried out at ambient temperature and in triplicate. The limit of detection (LOD) and limit of quantification (LOQ) were calculated based on the standard deviation of the responses and the slope using three independent analytical curves; LOD and LOQ were calculated as 3.3 and 10 σ/S, respectively, where σ is the standard deviation of the response and S is the slope of the calibration curve.

Determination of ferric reducing antioxidant property
The reducing property of the extracts was determined by assessing the ability of the extracts to reduce $FeCl_3$ solution as described by Oyaizu [23]. An aliquot (500 μl) of the extracts was mixed with 2.5 ml of 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 10% trichloroacetic acid was added. This mixture was centrifuged at 801 × g for 10 min. A 5 ml aliquot of the supernatant was mixed with an equal volume of water and 1 ml 0.1% ferric chloride. The absorbance was measured at 700 nm and the ferric reducing power was subsequently calculated using ascorbic acid equivalent.

Determination of 1, 1-diphenyl-2-picrylhydrazyl radical scavenging ability
The free radical scavenging ability of the extracts against 1,1-diphenyl-2-picrylhydrazyl (DPPH) radical was evaluated as described by Gyamfi et al [24]. Briefly, an appropriate dilution of the extracts (1 ml) was mixed with 1 ml 0.4 mM DPPH radicals in methanolic solution. The mixture was left in the dark for 30 min, and the absorbance was taken at 516 nm. The control was carried out by using 2 ml DPPH solution without the test extracts. The DPPH radical scavenging ability was subsequently calculated as a percentage of the control:

DPPH radical scavenging ability (%) =
$$[(Abs_{ref} - Abs_{sam})/Abs_{ref}] \times 100$$
where Abs_{ref} represents the absorbance of the reference and Abs_{sam} represents the absorbance of the sample.

Nitric oxide radical scavenging ability
The scavenging effect of the extracts on nitric oxide (NO·) radical was measured according to the method of Susanta et al [25]. Turmeric extract (100–400 μl) was added to the test tubes containing 1 ml of sodium nitroprusside solution (25 mM) and tubes were incubated at 37°C for 2 h. An aliquot (0.5 ml) of the incubation was removed

and diluted with 0.3 ml Griess reagent (1% sulphanilamide in 5% H_3PO_4 and 0.1% naphthlethylene diaminedihydrochloride). The absorbance of the chromophore formed was immediately read at 570 nm against distilled water as blank:

$$NO\ radical\ scavenging\ ability\ (\%) = [(Abs_{ref} - Abs_{sam})/Abs_{ref}] \times 100$$

where Abs_{ref} represents the absorbance of the reference, and Abs_{sam} represents the absorbance of the sample.

OH radical scavenging ability
The ability of the extracts to prevent Fe^{2+}/H_2O_2-induced decomposition of deoxyribose was evaluated using the method of Halliwell and Gutteridge [26]. Briefly, appropriate dilutions of the extracts were added to a reaction mixture containing 120 µl 20 mM deoxyribose, 400 µl 0.1 M phosphate buffer, 40 µl 20 mM hydrogen peroxide and 40 µl 500 µM $FeSO_4$, and the volume made up to 800 µl with distilled water. The reaction mixture was incubated at 37°C for 30 min, and the reaction was stopped by adding 0.5 ml 2.8% trichloroacetic acid (TCA). After this, 0.4 ml 0.6% thiobarbituric acid (TBA) solution was added to the test tubes which were then incubated in a water bath for 20 min. The absorbance was measured at 532 nm using a spectrophotometer. The percentage (%) hydroxyl radical scavenging ability was subsequently calculated:

$$OH\ radical\ scavenging\ ability\ (\%) = [(Abs_{ref} - Abs_{sam})/Abs_{ref}] \times 100$$

where Abs_{ref} represents the absorbance of the reference and Abs_{sam} represents the absorbance of the sample.

α-Amylase inhibition assay
α-Amylase inhibition was determined by the method of Bernfield [27]. The extracts (0–200 µl) and 500 µl of 0.02 M sodium phosphate buffer (pH 6.9 with 0.006 M NaCl) containing porcine pancreatic α-amylase (EC 3.2.1.1) (0.5 mg/ml) were incubated at 25°C for 10 min. Then, 500 µl of 1% starch solution in 0.02 M sodium phosphate buffer (pH 6.9 with 0.006 M NaCl) was added to each tube. The reaction mixture was incubated at 25°C for 10 min and stopped with 1.0 ml of dinitrosalicylic acid colour reagent.

Thereafter, the mixture was incubated in a water bath at 100°C 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 α-amylase inhibitory activity was expressed as percentage inhibition:

$$\%\ Inhibition = [(Abs_{ref} - Abs_{sam})/Abs_{ref}] \times 100$$

where Abs_{ref} is the absorbance without the extracts and Abs_{sam} is the absorbance of the extracts.

α-Glucosidase inhibition assay
The extracts (0–200 µl) and 100 µl α-glucosidase (EC 3.2.1.20) solution in 0.1 M phosphate buffer (pH 6.9) was incubated at 25°C for 10 min. Then, 50 µl 5 mM p-nitrophenyl-α-D-glucopyranoside solution in 0.1 M phosphate buffer (pH 6.9) was added. The mixtures were incubated at 25°C for 5 min, before absorbance was read at 405 nm in the spectrophotometer. The α-glucosidase inhibitory activity was expressed as percentage inhibition [28]:

$$\%\ Inhibition = [(Abs_{ref} - Abs_{sam})/Abs_{ref}] \times 100$$

where Abs_{ref} is the absorbance without the extracts and Abs_{sam} is the absorbance of the extracts.

Data Analysis

The results of three replicates were pooled and expressed as the mean ± standard deviation (SD). Student's *t* test, one-way analysis of variance (ANOVA) and least significance difference (LSD) were performed. Significance was accepted at $p \leq 0.05$. EC_{50} (effective concentration causing 50% inhibition) was determined using linear regression analysis.

Results and Discussion

The total phenol and flavonoid contents of the raw and cooked turmeric extracts were reported as gallic acid equivalents (GAE) and quercetin equivalents (QE), respectively, and are presented in Figs. 1 and 2. The results show that SCT20 (4.26 mg GAE/g and 3.96 mg QE/g) had significantly (p<0.05) higher total phenolic and flavonoid contents than SCT10 (3.58 mg GAE/g and 3.38 mg QE/g) and RWT (2.44 mg GAE/g and 2.38 mg QE/g).

This finding indicates that steam cooking increased the phenolic content of the spice. The result correlates with previous reports where increased cooking time caused an increase in the phenolic content of vegetables [29]. However, there are contradictory reports that thermal processing decreases the phenolic content of fruits and vegetables [30,31], although Hunter and Fletcher [32] reported that the effect of thermal processing on phenolics depends on the type of plant food. The increase in the phenolic content of the cooked turmeric extracts could be attributed to increased levels of free phenolics and flavonoids released by the heat treatment.

The ferric reducing antioxidant properties of the turmeric extracts, reported as ascorbic acid equivalent (AAE) as shown in Fig. 3, revealed that SCT_{20} (76.8 mmol AAE/100 g) had significantly ($p<0.05$) higher reducing power than SCT_{10} (72.4 mmol AAE/100 g) and RWT (68.2 mmol AAE/100 g). This result revealed that as steam cooking time increases, the reducing property of the turmeric extracts increases. The result also agrees with the trend observed in the total phenol and flavonoid contents of the extracts and previous reports where heat processing increased the reducing capacity of vegetables via the reduction of Fe^{3+} to Fe^{2+} [29].

The DPPH radical scavenging abilities of the RWT, SCT_{10} and SCT_{20} extracts are shown in Fig. 4. The extracts scavenged DPPH· in a dose-dependent manner; however, SCT_{20} (EC$_{50}$=2.72 mg/ml) had significantly higher ($p<0.05$) scavenging ability than
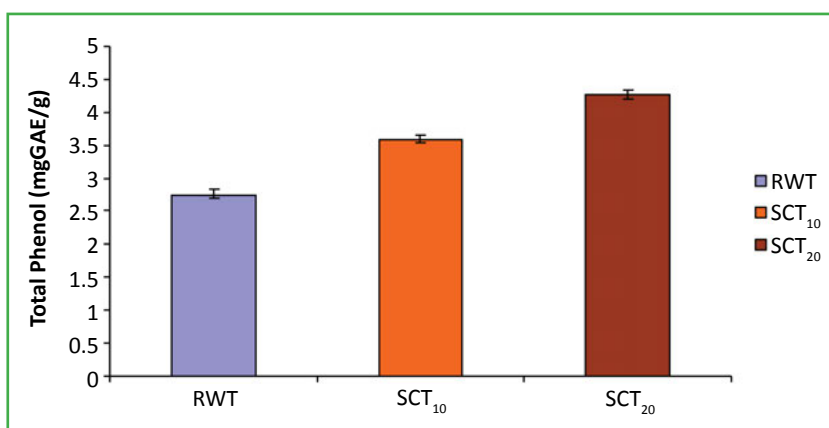

Figure 1 - Total phenol content of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, *SCT₁₀* steam cooked turmeric for 10 min, *SCT₂₀* steam cooked turmeric for 20 min
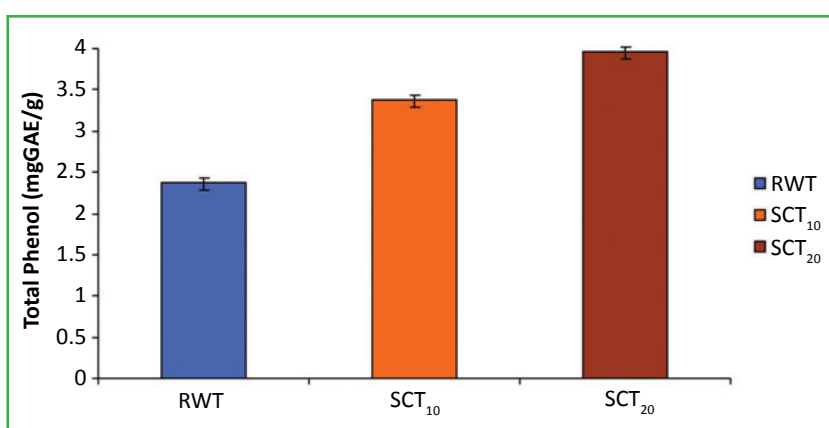

Figure 2 - Total flavonoid content of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, *SCT₁₀* steam cooked turmeric for 10 min, *SCT₂₀* steam cooked turmeric for 20 min
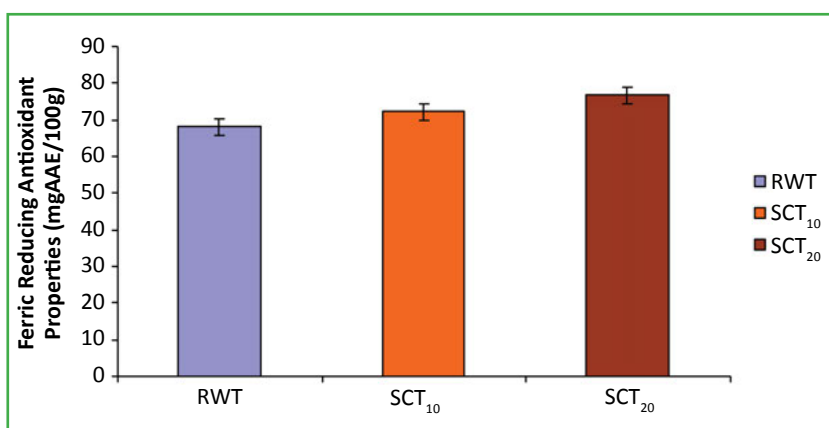

Figure 3 - Ferric reducing antioxidant property of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, *SCT₁₀* steam cooked turmeric for 10 min, *SCT₂₀* steam cooked turmeric for 20 min

SCT_{10} (EC$_{50}$=3.12 mg/ml) and RWT (EC$_{50}$=3.83 mg/ml) as shown in Table 1. Furthermore, the extracts scavenged NO radicals in a dose-dependent manner as shown in Fig. 5.
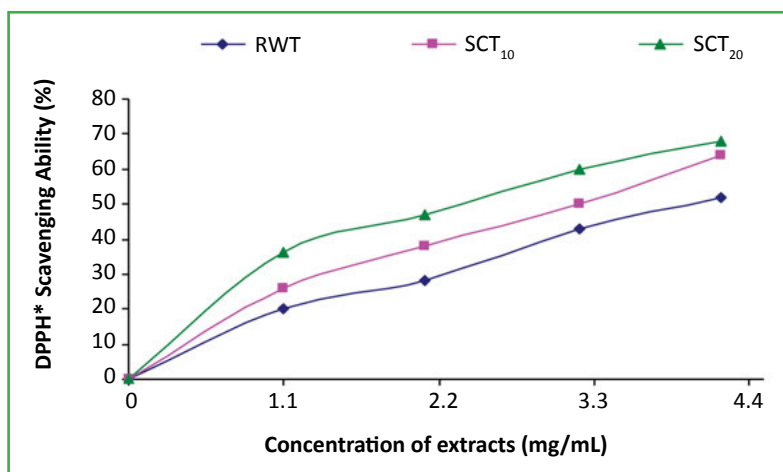
Figure 4 - 1,1-Diphenyl-2 picrylhydrazyl (DPPH) radical scavenging ability of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, *SCT₁₀* steam cooked turmeric for 10 min, *SCT₂₀* steam cooked turmeric for 20 min

Parameter	RWT	SCT₁₀	STC₂₀
Total phenol content (mg/g)	2.44 ± 0.11^a	3.58 ± 0.08^b	4.26 ± 0.10^c
Total flavonoid content (mg/g)	2.38 ± 0.06^a	3.38 ± 0.12^b	3.96 ± 0.09^c
FRAP (mmol/100g)	68.2 ± 1.8^a	72.4 ± 1.2^b	76.8 ± 1.5^c
DPPH· scavenging ability (mg/ml)	3.83 ± 0.10^c	3.12 ± 0.08^b	2.72 ± 0.05^a
NO· scavenging ability (mg/ml)	0.55 ± 0.02^c	0.47 ± 0.03^b	0.13 ± 0.01^a
OH· scavenging ability (mg/ml)	0.18 ± 0.03^b	0.15 ± 0.01^a	0.13 ± 0.02^a
α-Amylase inhibition (mg/ml)	1.13 ± 0.02^c	1.01 ± 0.01^b	0.85 ± 0.02^a
α-Glucosidase inhibition (mg/ml)	1.10 ± 0.01^c	0.97 ± 0.03^b	0.81 ± 0.01^a

Values represent the mean±standard deviation (n=3).
Values with different superscript along the same row are significantly ($p<0.05$) different
DPPH: 1,1-diphenyl-2 picrylhydrazyl - *FRAP*: ferric reducing antioxidant properties
OH: hydroxyl - *NO*: nitric oxide.

Table 1 - Total phenol and flavonoid content, EC₅₀ of inhibition of α-amylase and α-glucosidase activity and radical (DPPH, NO, OH) scavenging ability of raw turmeric (RWT), steam cooked turmeric for 10 min (SCT₁₀) and steam cooked turmeric for 20 min (SCT₂₀)
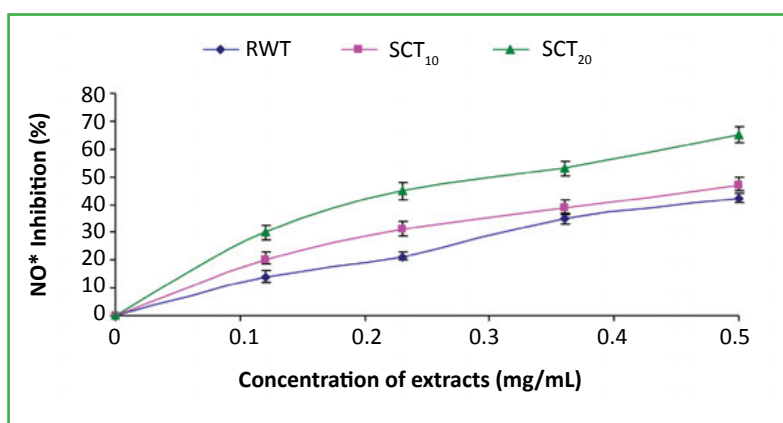

Figure 5 - Nitric oxide (NO) radical scavenging ability of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, *SCT₁₀* steam cooked turmeric for 10 min, *SCT₂₀* steam cooked turmeric for 20 min

The result revealed that the heat treatment increased radical scavenging ability as higher scavenging abilities were observed in SCT₂₀ (EC₅₀=0.13 mg/ml) and SCT₁₀ (EC₅₀=0.47mg/ml) compared to RWT (EC₅₀= 0.55 mg/ml). The results in Fig. 6 and Table 1 also revealed that the turmeric extracts scavenged OH radicals in a dose-dependent manner. SCT₂₀ (EC₅₀=0.13 mg/ml) had a higher scavenging ability compared to SCT₁₀ (EC₅₀=0.15 mg/ml) and RWT (EC₅₀=0.18 mg/ml).

Free radical generation has been associated with hyperglycaemia-induced diabetic complications. Hyperglycaemia stimulates the formation of ROS/reactive nitrogen species (RNS) such as NO and OH radicals. NO radicals can react with superoxide to form peroxynitrite, a powerful oxidant which has been implicated in diabetes [33]. Also, glucose can react with hydrogen peroxide in the presence of iron to form hydroxyl radical which can attack and reduce pancreatic β-cell function [34]. The increased DPPH, NO and OH radical scavenging abilities observed in the SCT extracts can be linked to the increase in the phenolic and flavonoid content as the antioxidative activity of polyphenolic compounds has been attributed to radical scavenging ability [35].

Inhibition of carbohydrate hydrolyzing enzymes is a good therapeutic approach in the management of diabetic conditions. The effect of the raw and cooked extracts of turmeric on α-amylase activity is presented in Fig. 7. The extracts inhibited α-amylase activity in a dose-dependent manner; however, STC₂₀ (EC₅₀=0.85 mg/ml) had a significantly higher ($p<0.05$) inhibitory effect on α-amylase activity than SCT₁₀ (EC₅₀=1.01 mg/

ml) and RWT extract (EC_{50}=1.13 mg/ml) (Table 1). The effect of the turmeric extracts on α-glucosidase activity is presented in Fig. 8 and Table 1. RWT (EC_{50} = 1.16 mg/ml) extract had a significantly (p<0.05) lower α-glucosidase inhibitory activity compared to SCT_{10} (EC_{50}=0.99 mg/ml) and SCT_{20} (EC_{50}=0.86 mg/ml). The inhibitory effects of the extracts could be beneficial in the management and/or treatment of type 2 diabetes as they delay glucose absorption and reduce post-prandial hyperglycaemia.

The result shows that cooking time increased the inhibitory activity of the extracts. This result agrees with the findings of Oboh et al [36] who reported that heat treatment such as roasting and cooking increased the α-amylase and α-glucosidase activities of African pear (*Dacryodes edulis*). The enzyme inhibitory activity of the extracts can be linked to their phenolic constituents such as caffeic acid, curcumin, luteolin, quercetin, rutin, quercitrin and epicatechin which were identified in the turmeric extracts and have been established by previous findings to inhibit α-amylase and α-glucosidase activity [37-40].

The phenolic composition of the turmeric extracts is also presented in Table 2. HPLC analysis (Fig. 9) revealed the presence of phenolic compounds such as curcumin, gallic acid, caffeic acid, catechin and epicatechin, and flavonoids such as rutin, quercetrin, quercitrin and luteolin. The trend observed in these results shows that cooking increased the phenolic constituents of the extracts. SCT_{20} had a significantly (p<0.05) higher amount of curcumin (14.03 mg/g), catechin (5.93 mg/g), epicatechin (1.62 mg/g), rutin (2.09 mg/g), quercetin (11.73 mg/g), quercitrin (6.12 mg/g) and luteolin (10.36 mg/g) than SCT_{10} and RWT.
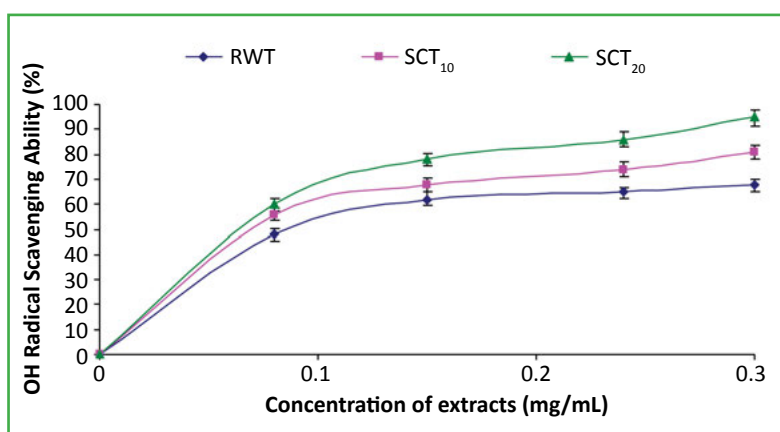

Figure 6 - Hydroxyl (OH) radical scavenging ability of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. RWT raw turmeric, SCT10 steam cooked turmeric for 10 min, SCT20 steam cooked turmeric for 20 min

Figure 7 - α-Amylase inhibitory ability of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. *RWT* raw turmeric, SCT_{10} steam cooked turmeric for 10 min, SCT_{20} steam cooked turmeric for 20 min

Figure 8 - α-Glucosidase inhibitory ability of raw and cooked extracts of turmeric. Values represent the means of triplicate readings. RWT raw turmeric, SCT10 steam cooked turmeric for 10 min, SCT20 steam cooked turmeric for 20 min
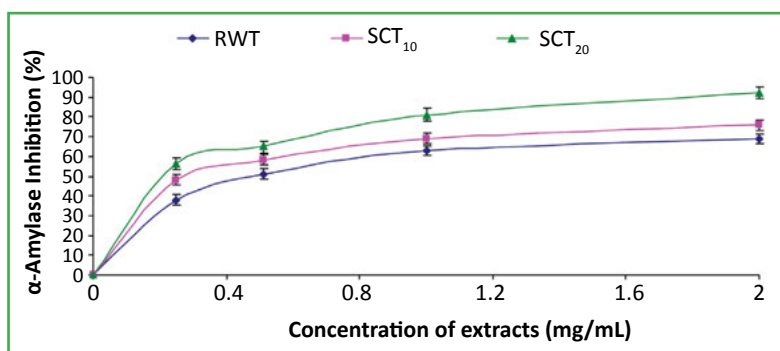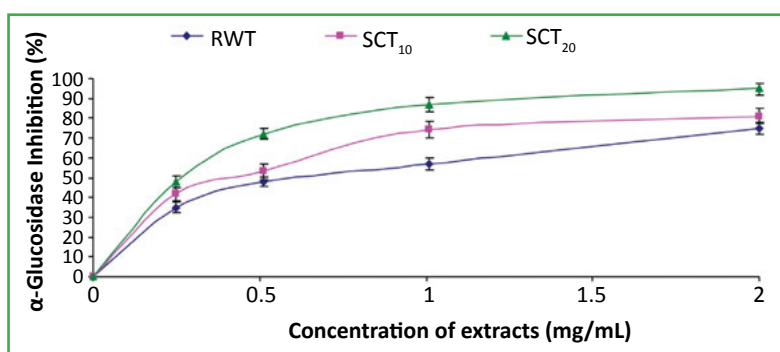
However, SCT_{10} had the highest amount of caffeic acid (5.36 mg/g), while there was no significant (p>0.05) difference in the gallic acid content of the three extracts. This result shows that cooking increased the phenolic constituents of the extracts, although a decrease was observed for gallic acid and caffeic acid in SCT_{20}. This finding shows the same

Compound	RWT (mg/g)	SCT$_{10}$ (mg/g)	SCT$_{20}$ (mg/g)
Gallic acid	1.59 ± 0.03[a]	1.60 ± 0.01[a]	1.58 ± 0.02[a]
Catechin	1.54 ± 0.01[a]	2.81 ± 0.01[b]	5.93 ± 0.01[c]
Caffeic acid	4.27 ± 0.01[b]	5.36 ± 0.02[c]	3.17 ± 0.02[a]
Epicatechin	0.95 ± 0.02[a]	1.54 ± 0.01[b]	1.62 ± 0.01[c]
Rutin	1.14 ± 0.01[c]	1.51 ± 0.03[a]	2.09 ± 0.03[d]
Quercitrin	6.07 ± 0.03[a]	5.09 ± 0.01[b]	6.12 ± 0.02[a]
Quercetin	4.15 ± 0.01[a]	9.13 ± 0.01[b]	11.73 ± 0.01[c]
Luteolin	6.30 ± 0.02[a]	9.26 ± 0.01[b]	10.36 ± 0.02[c]
Curcumin	10.79 ± 0.01[a]	13.81 ± 0.03[b]	14.03 ± 0.02[c]

Results are expressed as the mean ± standard deviations (SD) of three determinations. Values with different superscript along the same row are significantly (*p*<0.05) different using the tukey's test

Table 2 - Phenolic composition of raw turmeric (RWT), steam cooked turmeric for 10 min (SCT$_{10}$) and steam cooked turmeric for 20 min (SCT$_{20}$)

trend for the total phenol and flavonoid contents of the turmeric extracts. The decrease observed in the gallic acid and caffeic acid contents in the SCT$_{20}$ extract could be attributed to the increase

in temperature associated with the cooking time which can lead to the degradation of these phenolic acids. Lemos et al [41] reported that an increase in temperature also decreased gallic acid and caffeic acid content in baru nuts.

Conclusion

Our findings revealed that steam cooking increased the phenolic constituents, antioxidant and antidiabetic properties of turmeric. As cooking time increased, the phenolic composition, radical scavenging abilities and enzyme inhibitory activities of STC$_{10}$ and STC$_{20}$ increased. Therefore, steam cooked turmeric could be more beneficial in the management and/or treatment of diabetes than raw turmeric.

Conflict of interest
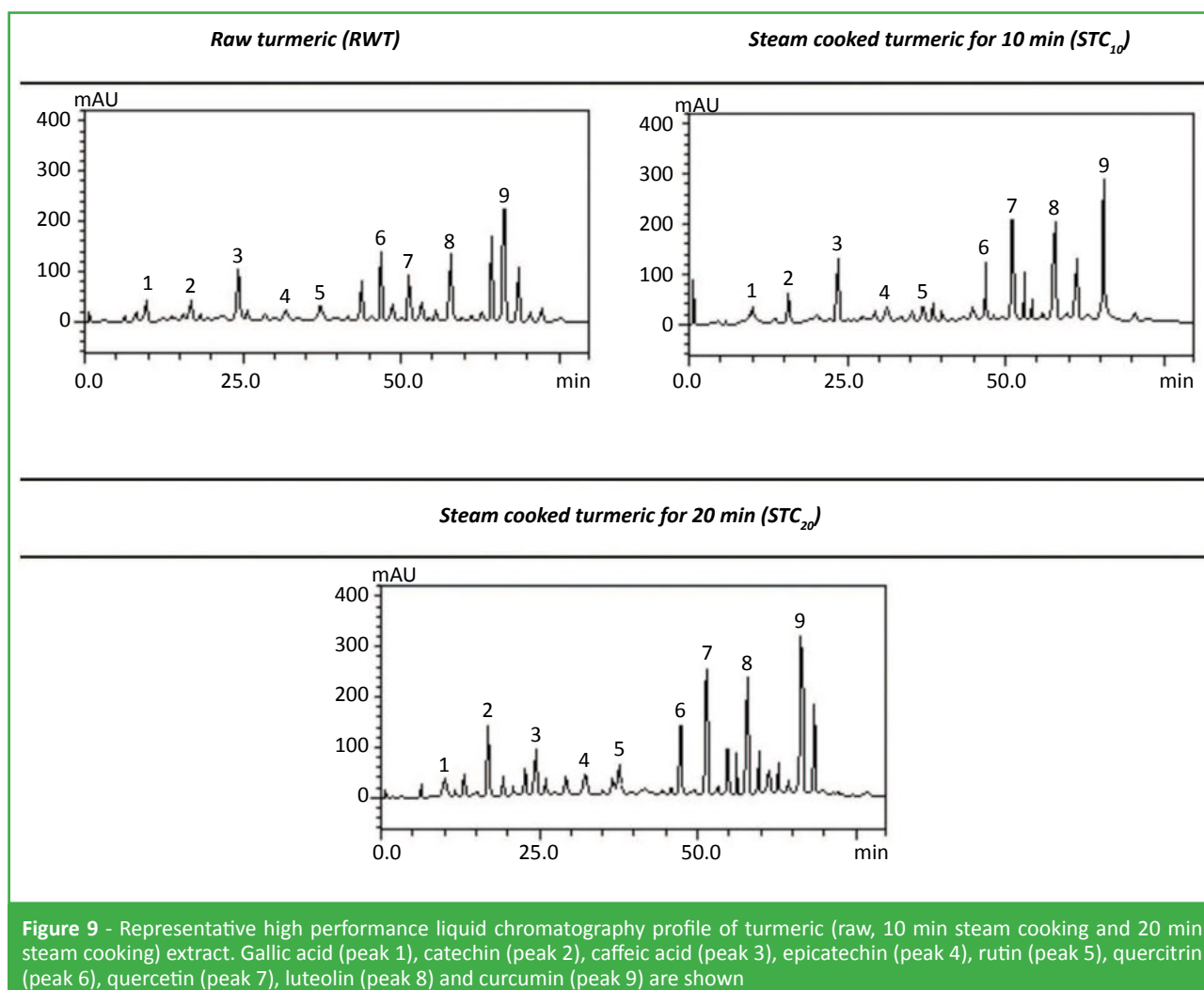Authors declare no conflict of interest

Figure 9 - Representative high performance liquid chromatography profile of turmeric (raw, 10 min steam cooking and 20 min steam cooking) extract. Gallic acid (peak 1), catechin (peak 2), caffeic acid (peak 3), epicatechin (peak 4), rutin (peak 5), quercitrin (peak 6), quercetin (peak 7), luteolin (peak 8) and curcumin (peak 9) are shown

References

1. Li S, Yuan W, Deng G *et al* (2011) Chemical composition and product quality control of turmeric (*Curcuma longa* L.). Pharm Crops 2:28–54

2. Remadevi R, Surendran E, Kimura T (2007) Turmeric in traditional medicine. In: Ravindran PN, Nirmal Babu K, Sivaraman K (eds) Turmeric: the genus Curcuma. CRC Press, Boca Raton, pp 409–436

3. Sasikumar B (2005) Genetic resources of Curcuma: diversity, characterization and utilization. Plant Gen Resour 3:230–251

4. Jurenka JS (2009) Anti-inflammatory properties of curcumin, a major constituent of *Curcuma longa*: a review of preclinical and clinical research. Altern Med Rev 14:141–153

5. Rivera-Espinoza Y, Muriel P (2009) Pharmacological actions of curcumin in liver diseases or damage. Liver Int 29:1457–1466

6. Mohanty I, Singh AD, Dinda A *et al* (2004). Protective effects of *Curcuma longa* on ischemia reperfusion induced myocardial injuries and their mechanisms. Life Sci 75:1701–1711

7. Honda SA, Tanaka H, Kishida H *et al* (2006). Effects of ingested turmeric oleoresin on glucose and lipid metabolisms in obese diabetic mice: a DNA microarray study. J Agric Food Chem 54:9055–9062

8. Kuroda M, Mimaki Y, Nishiyama T (2005) Hypoglycemic effects of turmeric (*Curcuma longa* L. rhizomes) on genetically diabetic KKAy mice. Biol Pharm Bull 28:937–939

9. Shytle RD, Bickford PC, Rezai-zadeh K *et al* (2009) Optimized turmeric extracts have potent antiamyloidogenic effects. Curr Alzheimer Res 6:564–571

10. Cho JY, Choi GJ, Lee SW *et al* (2006) *In vivo* antifungal activity against various plant pathogenic fungi of curcuminoids isolated from the rhizomes of *Curcuma longa*. Plant Path J 22:94–96

11. Carlsen MH, Halvorsen BL, Holte K *et al* (2010) The total antioxidant content of more than 3100 foods, beverages, spices, herbs and supplements used worldwide. Nutr J 9:3

12. Atawodi SE (2005) Antioxidant potential of African medicinal plants. Afr J Biotechnol 4:128–133

13. Kwon YI, Vattem DA, Shetty K (2006) Evaluation of clonal herbs of Lamiaceae species for management of diabetes and hypertension. Asia Pac J Clin Nutr 15:107–118

14. Shobana S, Sreerama YN, Malleshi NG (2009) Composition and enzyme inhibitory properties of finger millet (*Eleusine coracana* L.) seed coat phenolics: mode of inhibition of α-glucosidase and pancreatic amylase. Food Chem 115:1268–1273

15. Srinivasan K, Sambaiah K, Chandrasekhara N (1992) Loss of active principles of common spices during domestic cooking. Food Chem 43:271–274

16. Suresh D, Manjunatha H, Srinivasan K (2007) Effect of heat processing of spices on the concentrations of their bioactive principles: turmeric (*Curcuma longa*), red pepper (*Capsicum annuum*) and black pepper (*Piper nigrum*). J Food Comp Anal 20:346–351

17. Adefegha SA, Oboh G (2012) Inhibition of key enzymes linked to type-2 diabetes and sodium nitroprusside-induced lipid peroxidation in rat pancreas by water extractable phytochemicals from some tropical spices. Pharm Biol 50:857–865

18. Valverdú-Queralt A, Medina-Remón A, Andres-Lacueva C *et al* (2011) Changes in phenolic profile and antioxidant activity during production of diced tomatoes. Food Chem 126:1700–1707

19. Gerard KA, Roberts JS (2004) Microwave heating of apple mash to improve juice yield and quality. Food Sci Technol 37:551–557

20. Singleton VL, Orthofor R, Lamuela-Raventos RM (1999) Analysis of total phenols and oxidation substrates and antioxidants by means of Folin-Ciocaltau reagent. Methods Enzymol 299:152–178

21. Meda A, Lamie C, Romito M *et al* (2005) Determination of the total phenolic, flavonoid and proline contents in Burkina Faso honey, as well as their radical scavenging activity. Food Chem 91:571–577

22. Adefegha SA, Oboh G, Adefegha OM *et al* (2014) Antihyperglycemic, hypolipidemic, hepatoprotective and antioxidative effects of dietary clove (*Syzygium aromaticum*) bud powder in a high-fat diet/streptozotocin-induced diabetes rat model. J Sci Food Agric 94:2726–2737

23. Oyaizu M (1986) Studies on products of browning reaction: antioxidative activity of products of browning reaction prepared from glucosamine. Jpn J Nutr 44:307–315

24. Gyamfi MA, Yonamine M, Aniya Y (1999) Free-radical scavenging action of medicinal herbs from Ghana: *Thonningia sanguinea* on experimentally induced liver injuries. Gen Pharmacol 32:661–667

25. Susanta K, Chakraborty G, Gupta M *et al* (2006) *In vitro* antioxidant activity of Dispyros malabaricakostel bark. India J Exp Biol 44:39–44

26. Halliwell B, Gutteridge JMC (1981) Formation of a thiobarbituric-acid-reactive substance from deoxyribose in the presence of iron salts. FEBS Lett 128:347–352

27. Bernfield P (1951) Enzymes of starch degradation and synthesis. Adv Enzymol 12:379–380

28. Apostolidis E, Kwon YI, Shetty K (2007) Inhibitory potential of herb, fruit, and fungal-enriched cheese against key enzymes linked to type 2 diabetes and hypertension. Food Sci Emerg Technol 8:46–54

29. Adefegha SA, Oboh G (2011) Enhancement of total phenolics and antioxidant properties of some tropical green leafy vegetables by steam cooking. J Food Process Preserv 35:615–622

30. Sultana B, Anwar F, Ashraf M *et al* (2012) Effect of drying techniques on the total phenolic contents and antioxidant activity of selected fruits. J Med Plants Res 6:161–167

31. Chipurura B, Muchuweti M, Manditseraa F (2010) Effects of thermal treatment on the phenolic content and antioxidant activity of some vegetables. Asian J Clin Nutr 2:93–100

32. Hunter KJ, Fletcher JM (2002) The antioxidant activity and composition of fresh, frozen, jarred and canned vegetable. Innovation Food Sci Emerg Technol 3:399–406

33. Zou MH, Shi CM, Cohen RA (2002) Oxidation of the zinc-thiolate complex and uncoupling of endothelial nitric oxide synthase by peroxynitrite. J Clin Invest 109:817–826

34. Valko M, Leibfritz D, Moncol J *et al* (2007) Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol 39:44–84

35. Amic D, Davidovic-Amic D, Beslo D *et al* (2003) Structure radical scavenging activity relationship of flavonoids. Croatia Chem Act 76:55–61

36. Oboh G, Ademosun AO, Olasehinde TA *et al* (2015) Effect of processing methods on the antioxidant properties and inhibition of α-amylase and α-glucosidase by African pear (*Dacryodes edulis*) fruit. Nutrafoods 14:19–26

37. McDougall GJ, Shpiro F, Dobson P *et al* (2005) Different polyphenolic components of soft fruits inhibit α-amylase and α-glucosidase. J Agric Food Chem 53:2760–2766

38. Adefegha SA, Oboh G (2013) Phytochemistry and mode of action of some tropical spices in the management of type-2 diabetes and hypertension. Afr J Pharm Pharmacol 7:332–346

39. Pereira DF, Cazarolli LH, Lavado C *et al* (2011) Effects of flavonoids on α-glucosidase activity: potential targets for glucose homeostasis. Nutrition 27:1161–1167

40. Hasan MD, Uddin N, Monir Hossain MD *et al* (2014) *In vitro* α-amylase inhibitory activity and *in vivo* hypoglycemic effect of ethyl acetate extract of *Mallotus repandus* (Willd.) Muell. stem in rat model. J Coastal Life Med 2:721–726

41. Lemos, MR, Siqueira, EM, Arruda SF *et al* (2012) The effect of roasting on the phenolic compounds and antioxidant potential of baru nuts [*Dipteryx alata* Vog.]. Food Res Int 48:592