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Changes in blood pressure indices in normotensive adults after the consumption of lemongrass tea

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ABSTRACT

Objective: To accurately evaluate the effect of lemongrasses tea (LGT) on blood pressure (BP) indices in normal humans.

Methods: A total of 105 participants were sub-divided into 3 groups with 35 in each group and they were administered with LGT prepared from 2, 4 or 8 g of the lemongrass leaf powder (LP) for 30 days, respectively. They were evaluated for various BP indices and other clinical and biochemical parameters at days 0, 10 and 30 after the administration of LGT using standard methods.

Results: At day 10, systolic blood pressure and diastolic blood pressure (DBP) were lower than baseline levels. The mean arterial pressure was slightly reduced, while pulse pressure and heart rate (HR) significantly increased in subjects administered with LGT prepared from 4 or 8 g of the LP. At day 30, systolic blood pressure and DBP remained decreased in participants administered with LGT prepared from 4 g of the LP. DBP normalized in participants administered with LGT prepared from 4 g of the LP. The mean arterial pressure and HR decreased further in participants administered with LGT prepared from 8 g of the LP, but HR normalized in subjects treated with LGT prepared from 4 g of the LP. Pulse pressure almost returned to baseline level.

Conclusions: Ingestion of LGT may be associated with decreased BP indices in normotensive humans due to its varied bioactive constituents and their activities.

1. Introduction

Lemongrass (*Cymbopogon citratus*) is an aromatic tropical plant of the family *Poaceae* with long, slender and green leaves, which is originally native to Southeast Asia. Lemongrass is now grown around the world, including North and South America[1] and Africa. It has diverse chemical constituents including crude protein, moisture, ash, crude fibre, fat and carbohydrates and it is rich in minerals, vitamins, phytochemicals and anti-nutrients[2-5]. Among all the constituents of lemongrass, its essential oil appears to be the most pharmacologically potent and physiologically important due to its citral content.

A research indicates that lemongrass is used in herbal medicine worldwide for a wide range of applications, including for its anti-inflammatory, cardioprotective, antiprotozoal, antioxidant, antifungal, anti-carcinogenic, antiplatelet aggregation and antibacterial[6-13]. It has also been used to treat dyslipidemia, gastrointestinal disturbances[14,15], diabetes[16], flu, fever, pneumonia[15], anxiety[17] and malaria[18]. Accordingly, lemongrasses tea (LGT) is increasingly being used to prevent and treat cardiovascular disorders due to its vasodilatory and diuretic properties. For instance, in Brazil and Cuba, some hypertensive patients drink LGT daily for its hypotensive effects[19-21], while in some communities in India, the majority of hypertensive individuals take LGT to lower their blood pressure (BP), particularly when experiencing symptoms presumed to be associated with increased BP. Anecdotal evidence suggests that this use has been a traditional practice among the residents of these communities[22]. Furthermore, a recent study reported transient hypotension and bradycardia in rats treated with citral-rich essential oil obtained from lemongrass[20]. Similarly, Shiina *et al.*[23] reported the cardiovascular effects of lemongrass essential oil in humans that included improvements in coronary flow, hypotension and bradycardia.

Public awareness of the health and nutritional benefits of lemongrass has increased the interest in the consumption of the plant by the general population. It is most frequently consumed for

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therapeutic and recreational purposes, much like green, black and red teas, herbal tea blends and coffee[24]. Lemongrass is also added to non-alcoholic beverages and baked foods in prepared foods as preservative[15,25] due to its distinctive taste, lemony smell, colour, strength, intensity and anti-microbial effects[26]. Accumulated evidences have shown that consumption of some plants and plant products could result in several physiological consequences including hemodynamic and biochemical changes, diuresis, electrolyte and mineral wasting as well as gastrointestinal disturbances. The consumption of this plant by healthy individuals raised the question that whether there could be associated hemodynamic effects in normal humans. Plant food-based nutritional studies are required for the evaluation of the effect of the plant on our health and wellbeing. The aim of this study was to accurately evaluate the hemodynamic changes after the ingestion of LGT by normal humans.

2. Materials and methods

2.1. Study design and selection of subjects

One hundred and five participants took part in this study which was carried out in the Department of Physiology at the University of Uyo, Nigeria in May 2012. The research work was preceded by a pre-survey lecture delivered by the research coordinator to inform the participants about the aim and possible outcomes of the study and a signed informed consent was obtained from each participant before data collection. This study was approved by the Institutional Research Ethics Committee and carried out at the University of Uyo, Nigeria, according to the guidelines set forth in the Declaration of Helsinki governing the conduct of human research.

Subject's eligibility was determined by medical history, physical examination and screening laboratory tests that included determinations of pre-survey glycaemic level, complete blood count, and serum biochemical markers of kidney and liver function.

The exclusion criteria included allergy to lemongrass constituents, inappropriate age (< 18 or > 35 years), pregnancy, lactation, evidence of past or present renal or hepatic disease and unfavorable pre-survey assessment data. Participants were instructed to maintain their routine diet, activity status and avoid intake of medications during the survey period.

2.2. Dosing

The subjects were sub-divided into 3 groups (n = 35/group) and administered with LGT prepared from 2, 4 or 8 g of the lemongrass leaf powder (LP) respectively in 150 mL of hot water once daily for 30 days. The choice of the experimental dose was guided by the results of a preceding pilot survey on 10 volunteers using LGT prepared from 2, 4, 8, and 10 g of lemongrass LP in 150 mL of hot water with no complication recorded. The LGT was also prepared to reflect the manner in which it is prepared by the population[27].

According to Leite *et al.*[27], LGT is usually prepared by pouring 150 mL of boiling water on 2–3 g of fresh or dried lemongrass leaves and it is commonly used as a traditional therapy for nervous disturbances such as insomnia, and anxiety[27]. We doubled and quadrupled the starting dose to produce the experimental dose range of 2, 4 or 8 g LGT used in the present. This was to enable the assessment of dose effect.

2.3. Measurement

The harvesting, identification and phytochemical analysis of lemongrass leaf extract were performed accordingly using standard methods[28,29]. Trained personnel measured the BP in the morning after the consumption of the LGT and after 5 min of rest in a sitting position[30]. A mercury sphygmomanometer (Acosin 300, Dekamet Ltd. England) with the appropriate cuff size was used, with the hand (right) being supported and positioned such that the middle of the cuff was on the upper arm at the level of the right atrium. Participants were instructed to relax and not to talk while BP was being measured. Systolic blood pressure (SBP) was indicated by the onset of Korotkoff sounds and diastolic blood pressure (DBP) was indicated by the disappearance of sounds. SBP and DBP were recorded as the mean of two consecutive BP measurements.

Measurements were categorized according to the European Society of Hypertension Guidelines[31]. Normotension was considered as a SBP less than 140 mmHg and a DBP less than 90 mmHg. Above these values, BP was considered to be in the hypertensive range[32]. The mean arterial pressure (MAP) was defined as DBP + 1/3(SBP – DBP), and pulse pressure (PP) was calculated as the difference between SBP and DBP. Heart rate (HR) was measured using the auscultatory method and was defined as the number of heart beats per minute.

2.4. Statistical analysis

Statistical analysis was performed using SPSS version 20.0. Data (mean ± SD) were analyzed using One-way ANOVA. Differences were considered statistically significant at $P < 0.05$.

3. Results

Preliminary analysis of the lemongrass leaf extract for its bioactive natural constituents showed the presence of high concentrations of saponins, moderate levels of tannins, flavonoids and phenols and relatively low levels of anthraquinones, alkaloids and deoxy-sugars. Also, the amounts of moisture, total ash, fat, crude proteins, crude fibre and carbohydrate composition in the extracts were 2.4, 4.2, 0.7, 2.7, 3.0 and 36.0 g, respectively. Additionally, the amounts of K^+, Na^+, Ca^{2+}, Fe^{2+}, Cu, Zn, Mg^{2+}, Mn and vitamin C in the leaf extract were 475.0, 8.0, 56.0, 15.6, 0.4, 2.1, 46.2, 8.3, 12.3 and 3.8 mg, respectively.

The socio-demographic and clinical data of the study subjects at baseline showed that a greater number of them (52%) were males with ages between 18 and 35 years and of Ibibio ethnicity. The mean weight, body mass index, SBP, DBP, MAP, and HR were (60.70 ± 1.93) kg, (23.50 ± 0.75) kg/m^2, (120.50 ± 1.89) mmHg, (74.60 ± 1.60) mmHg, (85.70 ± 1.13) mmHg and (77.70 ± 1.99) beats/min, respectively, whereas the mean PP, mean respiratory rate and estimated glomerular filtration rate were 45.9 ± 1.04, 18.6 ± 1.52 per minute and (99.9 ± 1.52) mL/min, respectively.

Table 1 shows the effects of the LGT on the 12-hour urinary volume, frequency of urination, urinary electrolytes (K^+, Na^+, Ca^{2+} and Cl^-) and urinary specific gravity (USG) at day 10 (acute) and 30 (sub-chronic), respectively. By day 10, the 12-hour urinary volume significantly increased in all treatment groups ($P < 0.05$). After 30 days of treatment with LGT, the elevated urinary volume was

maintained in all subjects, but the increase was only significant among participants administered LGT prepared from 8 g of the LP ($P < 0.05$). A significant increase in the frequency of urination (per 24 h) was also observed in all groups except for those administered LGT prepared from 2 g of the LP for 30 days.

The results of urinary electrolytes assessments revealed that at day 10, the urinary K^+ level was increased in all treatment groups. However, by day 30, the urinary K^+ levels had decreased below pre-treatment level. At day 10, the urinary Na^+ level was significantly increased ($P < 0.05$) in all groups except for those administered with LGT prepared from 8 g of lemongrass LP, where there was a non-significant increase. By day 30, the urinary Na^+ level returned to pre-treatment level in the subjects who received the LGT prepared from 4 g of lemongrass LP but was significantly lower in the groups administered with LGT prepared from 2 or 8 g of the LP. The urinary Ca^{2+} levels increased significantly ($P < 0.05$) in all groups at day 10 and 30 except for groups administered with the LGT prepared from 8 g of the LP for 10 days. There was also a significant increase urinary Cl^- levels at days 10 and 30.

Additionally, the peak, diuretic action and the natriuretic effect were achieved at day 10 in subjects who were treated with the LGT prepared from 2 or 4 g of the LP. At day 30, these effects were decreased in participants administered LGT prepared from 8 g of the LP. Similar effects were obtained for the kaliuretic, chloruretic and calciuretic indices (Table 2).

The assessment of the cardiovascular indices (MAP, DBP, SBP, and HR) indicated that at day 10, MAP was significantly lower in the participants who were administered with LGT prepared from 4 or 8 g of the LP ($P < 0.05$). MAP decreased further after 30 days in the participants receiving LGT prepared from 8 g, but not 4 g of the LP (Figure 1A).

By day 10, SBP was slightly lower in the participants who received the LGT prepared from 2, 4 or 8 g of the LP, and by day 30, SBP remained decreased in all treatment groups but only significantly in the participants receiving the LGT prepared from 4 g of the LP (Figure 1B). Conversely, DBP was lower in all treatment groups by day 10. At day 30, DBP remained low in the participants receiving LGT prepared from 4 g of the LP, but partially normalized in those receiving the LGT prepared from 8 g of the LP (Figure 1C). HR was significantly increased at day 10 in the subjects who received LGT prepared from either 4 or 8 g of the LP ($P < 0.05$), but it eventually returned to baseline by day 30 in those receiving the LGT prepared from 4 g of the LP. In the participants receiving the LGT prepared from 8 g of the LP, HR was lower than that at day 10, but was still higher than the baseline. HR was unchanged in the subjects administered with LGT prepared from 2 g of the LP (Figure 2A). At day 10, PP tended to increase in all groups but was only significantly increased in the participants who received the LGT prepared from 4 or 8 g of the LP. At day 30, PP had almost returned to baseline values in all groups (Figure 2B). Figure 2C shows that saluretic action peaks at day 10 in participants administered with LGT prepared from 2 or 4 g of the LP, and subsequently decreased at day 30 in participants administered with LGT prepared from 8 g of the LP.

Table 1

Acute and sub-chronic effects of infusions prepared from 2, 4 or 8g lemongrass powder on urine parameters.

Dosing	Mean 12 h urine volume (L)	Mean 24 h urination frequency	K^+ (mmol/L)	Na^+ (mmol/L)	Ca^{2+} (mmol/L)	Cl^- (mmol/L)	USG
Baseline							
2 g	0.80 ± 0.13	6.42 ± 0.24	40.83 ± 1.87	160.63 ± 5.57	11.78 ± 3.87	130.34 ± 6.37	1.010 00 ± 0.000 01
4 g	0.71 ± 0.04	5.06 ± 0.20	39.40 ± 1.37	117.94 ± 8.56	16.25 ± 2.97	147.80 ± 6.44	1.010 00 ± 0.000 01
8 g	0.86 ± 0.05	6.02 ± 0.28	37.71 ± 1.68	135.63 ± 8.20	11.95 ± 2.84	137.23 ± 7.46	1.010 00 ± 0.000 01
Acute							
2 g	0.90 ± 0.04[a]	7.08 ± 0.22[a]	58.57 ± 3.18[a]	170.37 ± 8.02[a*]	13.69 ± 1.83[a]	198.80 ± 8.47[a]	1.010 00 ± 0.000 01
4 g	1.10 ± 0.02[ab]	7.04 ± 0.37[a]	52.87 ± 2.87[a]	141.89 ± 5.53[ab]	20.38 ± 2.30[ab]	206.97 ± 3.35[a]	1.011 00 ± 0.000 01
8 g	1.01 ± 0.04[ab]	7.02 ± 0.34[a]	44.06 ± 3.06[abc]	140.06 ± 7.20[b]	12.86 ± 1.86[c]	181.51 ± 11.72[ac]	1.014 00 ± 0.000 12
Sub-chronic							
2 g	0.88 ± 0.01	6.56 ± 0.25	37.67 ± 0.39	127.14 ± 8.21[a]	17.13 ± 2.26[a]	190.23 ± 7.43[a]	1.013 00 ± 0.000 02
4 g	0.80 ± 0.06	6.74 ± 0.31[a]	37.40 ± 1.85	116.00 ± 6.73	20.43 ± 2.99[a]	174.60 ± 8.44[a]	1.011 00 ± 0.000 01
8 g	0.96 ± 0.03[abc]	8.00 ± 0.22[abc]	36.54 ± 0.65	126.11 ± 7.70[a]	17.05 ± 2.54[a]	162.94 ± 8.16[ac]	1.014 00 ± 0.000 01

[a]: Significantly different from baseline ($P < 0.05$); [b]: Significantly different from 2 g ($P < 0.05$); [c]: Significantly different from 4 g ($P < 0.05$); Values reported as mean ± SEM.

Table 2

Acute (10 days) and Sub-chronic (30 days) effects of infusions prepared from 2, 4 or 8 g lemongrass powder on diuretic indices.

Dosing	Na^+/Cl^-	Na^+ test/ Na^+control	K^+test/K^+control	Ca^{2+}test/ Ca^{2+}control	Cl^- test/Cl^- control	Urine output in test group/ urine output in control group	$Na^+ + Cl^-$
Baseline							
2 g	1.230 ± 0.450	-	-	-	-	-	290.970 ± 8.220
4 g	0.80 ± 0.060	-	-	-	-	-	252.740 ± 6.250
8 g	0.99 ± 0.040	-	-	-	-	-	272.800 ± 9.520
Acute		-	-	-	-	-	
2 g	0.860 ± 0.025	1.060 ± 0.012	1.430 ± 0.031	1.160 ± 0.120	1.530 ± 0.060	1.125 ± 0.030	369.170 ± 5.220[a]
4 g	0.690 ± 0.021	1.200 ± 0.011[b]	1.340 ± 0.035	1.250 ± 0.240	1.400 ± 0.052	1.550 ± 0.050[b]	348.860 ± 10.050[a]
8 g	0.770 ± 0.042[a]	1.030 ± 0.022[c]	1.170 ± 0.055[bc]	1.080 ± 0.010[c]	1.320 ± 0.070[b]	1.170 ± 0.080[c]	321.570 ± 7.580[abc]
Sub-chronic							
2 g	0.670 ± 0.011	0.790 ± 0.01	0.920 ± 0.020	1.450 ± 0.040	1.460 ± 0.050	1.100 ± 0.080	317.370 ± 10.570[a]
4 g	0.660 ± 0.010	0.980 ± 0.03[b]	0.950 ± 0.010	1.260 ± 0.036[b]	1.180 ± 0.023	1.130 ± 0.120	290.600 ± 12.080[a]
8 g	0.770 ± 0.021	0.930 ± 0.01[b]	0.970 ± 0.011	1.430 ± 0.021[c]	1.190 ± 0.016	1.120 ± 0.060	289.050 ± 15.860[ab]

2 g (410 mg yield); 4 g (810 mg yield); 8 g (1 570 mg yield); [a]: Significantly different from baseline ($P < 0.05$); [b]: Significantly different from 2 g ($P < 0.05$); [c]: Significantly different from 4 g ($P < 0.05$); Values reported as mean ± SEM.

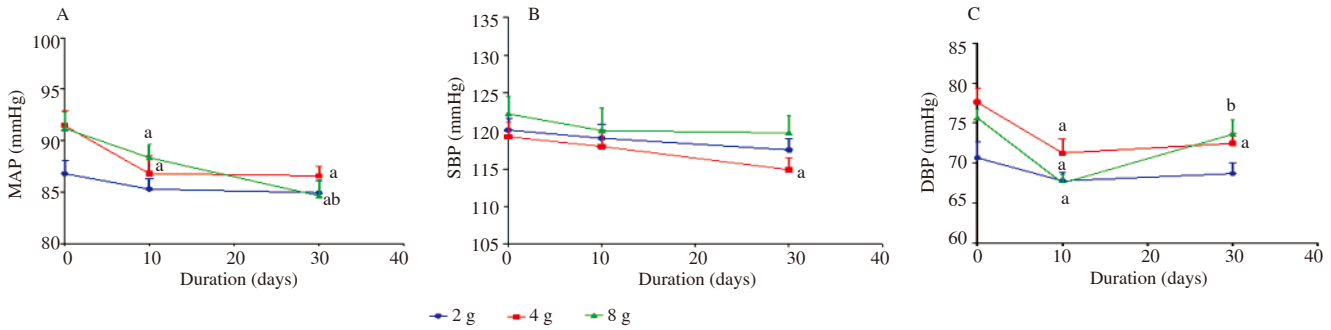

Figure 1. Acute and sub-chronic effects of infusions prepared from 2, 4, or 8 g of lemongrass LP on MAP (A), SBP (B), and DBP (C) of study participants. a: $P < 0.05$ *vs.* baseline; b: $P < 0.05$ *vs.* 10 days; c: $P < 0.05$ *vs.* 30 days. Values were reported as mean ± SD.

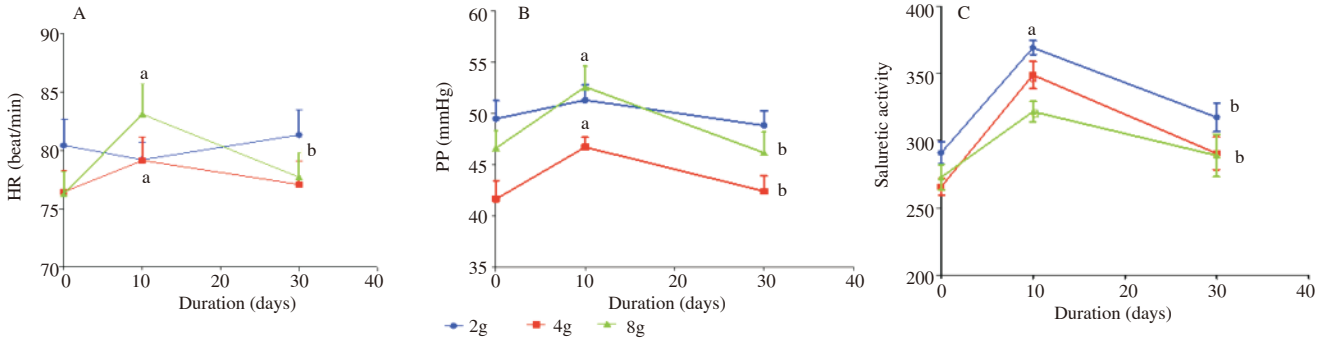

Figure 2. Acute and sub-chronic effects of infusions prepared from 2, 4, or 8 g of lemongrass LP on HR (A), PP (B) and saluretic activity (C) indices of study participants.
a: $P < 0.05$ *vs.* baseline; b: $P < 0.05$ *vs.* 10 days; c: $P < 0.05$ *vs.* 30 days; Values were reported as mean ± SD.

4. Discussion

The ingestion of LGT prepared from lemongrass LP, especially those containing higher concentrations of the powder, decreased MAP. This decrease was largely due to a drop in DBP at day 10 and by drops in both DBP and SBP at day 30. There was a concomitant increase in HR, urinary volume, urination frequency and electrolyte concentrations and the renal fractional excretion of substances, in addition to fluctuations in PP and non-significant changes in USG. There were also increases in other indices of diuresis, including the natriuretic, saluretic and diuretic actions. DBP partially normalised in some groups during the sub-chronic phase of the study.

The aforementioned clinical and biochemical changes observed in the present study following the ingestion of LGT are consistent with those previously observed in individuals on standard diuretic therapy[33] and confirm the diuretic and natriuretic effects of LGT that have been previously reported[19,22]. Studies by others[34-36] have shown that diuresis, whether in hypertensive or normotensive human subjects or in animal models, can cause changes in haemodynamic parameters, including a reduction in total blood and/or plasma volume, cardiac output and BP, regardless of the chemical category of the diuretic agent and its site of action. It is plausible that the BP-lowering effects of LGT observed in the present study were, at least in part, related to volume depletion and plasma contraction, which could have occurred due to the acute diuretic and natriuretic effects of the extract. This assertion is supported by similar findings in both animal and human studies[14,22]. Although the mechanisms by which LGT induces diuresis and hence hypotension have not yet been clearly elucidated[19,22], the results of biochemical analyses of the extract in the present and previous studies[2,37] indicated the presence of high levels of saponins, polyphenol-compounds (tannins, flavonoids and phenols) proteins, carbohydrates, electrolytes and vitamins. Accumulating evidences suggest that some of these bioactive constituents (saponins, phenolic compounds and K^+) can induce diuresis, saluresis and natriuresis and hence hypotension[38] by interfering, either individually or synergistically, with the re-absorption of electrolytes (Na^+ and Cl^-) and water in the renal tubules[39]. Previous studies have shown that some of these substances can induce diuresis and natriuresis through inhibition of Na^+/K^+-ATPase activity in the kidney[40,41] by directly binding the enzyme, impairing its activity or causing changes in membrane fluidity[42]. Others have postulated that some of these substances can also regulate interactions between membrane phospholipids and Na^+/K^+-ATPase pumps. de Souza *et al.*[43] reported that saponins inhibited Na^+/K^+-ATPase in a similar manner but stronger than furosemide, a standard loop diuretic. Rhiouani *et al.*[44] assessed the effects of saponins from *Herniaria glabra* on BP and renal function in rats and concluded that chronic oral administration of saponins decreased the arterial BP and affected salt and water transport in renal tubules. Similarly, Jouad *et al.*[45] reported that flavonoids significantly increased the urinary Na^+, K^+ and $2Cl^-$ concentrations in a manner similar to that of furosemide. In a similar study, Tarkang *et al.*[46] found increased blood urea and nitrogen and mild tubular distortion in the kidney after the administration of ethanol extract of lemongrass for 28 days. Compared to the control, serum creatinine levels suggested a non-renal cause of the derangement, which further implicated the action of lemongrass phytoactives including saponins and flavonoids which are known for their adverse metabolic effects.

Taking the above consistent empirical evidences into account, it can be concluded that the hemodynamic and biochemical changes observed in the present study are partly attributable to the effects of the phytochemical constituents in the LGT.

Furthermore, inhibition of the renin-angiotensin-aldosterone system, a key regulator of BP and body fluid volume in humans, is another plausible mechanism by which lemongrass phytonutrients (including its high flavonoids and protein contents) can lower BP[47,48]. Several studies have shown that angiotensin-converting enzyme (ACE) inhibitory peptides that are derived from foods (particularly those with high protein content such as lemongrass) can act as potential physiological modulators of BP as well as other cardiovascular functions by preventing the angiotensin II-mediated secretion of aldosterone from the adrenal cortex and by suppressing the metabolism of bradykinins (a vasodilator). The effects of these actions are as follows: decreases in water and Na^+ reabsorption, depletion of extracellular fluid volume, plasma contraction and the associated vasodilation and decrease in BP[49,50]. Thus, the peptides derived from the high lemongrass protein content could have contributed to the hemodynamic changes observed in the present study by their ACE inhibitory, diuretic and vasodilatory actions. Moreover, an existing research indicates that some polyphenolic compounds including flavonoids and tannins are effective natural bioactive ACE inhibitors, and possess a high potentiality to inhibit ACE *in vitro*[51-53]. In addition to the indirect vasodilatory actions of LGT, evidence in the literature also supports the view that some biologically active substances in lemongrass extract, such as the flavonoids, tannins and electrolytes, also have direct vasodilatory effects[54,55]. These substances have also been shown to exert calcium channel-blocking effects[56,57], a known BP-lowering action. As previously documented[3], in the present study we detected antioxidants in the lemongrass extract. These antioxidants included flavonoids, tannins, saponins, alkaloids and vitamin C. Given the role of oxidative stress in the pathogenesis of cardiovascular pathologies, such antioxidants could also have contributed to the hemodynamic modulatory effects of the LGT.

Previous studies have described plausible mechanisms by which antioxidants modulate cardiovascular indices. It has been suggested that antioxidants reduce oxidative stress by directly scavenging free radicals, such as reactive oxygen species (ROS), which are known to inhibit nitric oxide synthesis. This inhibition occurs through the inhibitory actions of ROS on dimethylarginine dimethyl-aminohydrolase, which is an enzyme known to degrade methyl arginine, a nitric oxide synthase inhibitor. Thus, when dimethylarginine dimethyl-aminohydrolase is inhibited, methyl arginine accumulates, causing inhibition of nitric oxide synthase and the resultant vasoconstriction[58]. Antioxidants reverse this action by increasing the production of a reduced form of tetrahydrobiopterin and also by increasing the activity of extracellular superoxide dismutase. These actions are known to block further production of ROS and increase endothelial NO production, subsequently triggering the formation of cyclic guanosine monophosphate and leading to vasodilation. Furthermore, some dietary antioxidants lower BP by inhibiting ACE. Increasing evidences indicate that ACE inhibition increases the levels of NO[59], thereby decreasing oxidative stress.

Other bioactive constituents found in lemongrass extract with well-known BP-lowering potential include electrolytes such as K^+, Mg^{2+} and Ca^{2+}. The intake of high levels of these electrolytes in a natural form has been shown to be effective in reducing BP through various mechanisms. For example, potassium modulates BP through its vasodilatory effects on smooth muscle cells, whereas the BP-lowering effects of Mg^{2+} salt are ascribed to its ability to modulate vascular tone and reactivity[60]. This action occurs via alterations of cellular Mg^{2+}/Ca^{2+} interactions in vascular smooth muscle cells and thus improves endothelial function. Mg^{2+} competes with Ca^{2+} for membrane-binding sites, thus lowering the levels of intracellular Ca^{2+} and leading to vasodilation[61]. Their ability to replace Na^+ is another plausible mechanism because salt sensitivity is a key factor in the pathogenesis of hypertension[62]. Therefore, the diuretic, natriuretic and saluretic activities underlying the hypotensive effects of LGT observed in the present study may be due to synergistic actions between the active principles and the K^+ and Mg^{2+} salts presenting in the LGT. The observed increase in HR, particularly at day 10 and in the subjects treated with the LGT prepared from 4 or 8 g of the LP, may be a physiological response to compensate for the decrease in BP and other haemodynamic changes resulting from the effect of the LGT. As previously documented[63], a significant BP decreasing in a normotensive subject could initiate a series of compensatory mechanisms aiming at preventing a further decrease while it is attempting to restore the normal level. These mechanisms include activation of the vasomotor centre, subsequent activation of the adrenergic nervous system and decreased parasympathetic activity and then activation of the sympathetic nervous system. The latter results in increases in the HR, strength of myocardial contraction, cardiac output and a return of BP back to normal levels. These complementary actions of the sympathetic and parasympathetic nervous system to BP changes explain the fluctuations in other cardiovascular parameters (including the acute significant increase in the HR and PP and the return to near baseline levels during the sub-chronic phase) observed in the present study. Additionally, the natriuretic effects of the leaf infusions observed during the acute phase of the study could have triggered compensatory hyperaldosteronism, a plausible explanation for the return to near baseline levels in the clinical (BP, HR and PP) and biochemical indices during the sub chronic phase of the study.

The results of the present study suggest that the consumption of LGT may be associated with decreases in BP indices in normotensive humans due to its varied bioactive constituents and their activities. Further studies to assess the effect of LGT on hypertensive subjects are recommended.

Conflict of interest statement

We declare that we have no conflict of interest.

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