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Research Article
Effect of Cinnamon (*Cinnamomum cassia*) on Blood Sugar, Lipid Profile and Liver Function of Male Wistar Rats

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Abstract

Background and Objective: Cinnamon is one of the most important spices that are highly valued for their health benefits. Thus, the study aimed at determining the effect of cinnamon on blood sugar, lipid profile and liver function of male wistar rats. **Materials and Methods:** Cinnamon sticks were sorted, washed, dried at 60°C and milled to powdery form. Fifteen male wistar rats were grouped into three. Group A (control) was fed regular rat feed, while group B was fed regular rat feed with 5% concentration of cinnamon and group C was given regular rat feed with 2.5% cinnamon. The weight and fasting blood sugar level of the rats were taken at baseline. Weekly weight gain, daily feed and water intake were recorded. Fasting blood sugar was determined every two weeks during the experiment. At the end of the experiment at fourth week, blood samples were collected for determination of the plasma glucose concentration, glycosylated haemoglobin (HbA1c), liver function and for lipid profile test. Liver samples were harvested for histopathology. **Results:** Cinnamon had blood sugar lowering effect at both concentrations of 2.5 and 5%. However, there was no significant difference in glycosylated haemoglobin, while plasma glucose concentration was lower in rats fed cinnamon. Cinnamon increased the high density lipoprotein (HDL) level but had negligible effect on other lipid profile parameters. There was no significant effect on aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Also the liver histopathology of rats fed 2.5% cinnamon and that of the control were comparable while that of rats fed 5% cinnamon revealed a deposition of plagues, enlarged sinusoids and hepatocyte with mild necrotic features. **Conclusion:** Cinnamon powder at the concentrations used, had lowering effect on the blood sugar with positive effect on the lipid profile and mild to adverse effect on the liver of normal wistar rats.

Key words: Blood sugar, cinnamon, lipid profile, liver, non-communicable diseases

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Non-communicable diseases (NCDs) such as diabetes, obesity and cardiovascular diseases now account for most of the global burden of diseases and an increasing burden has been forecasted in the decades ahead, particularly in low and middle income countries[1]. Non-communicable diseases kill about 41 million people each year, which is equivalent to 71% of deaths globally and recent reports show that 80% of NCD deaths occur in low and middle income countries[2-4].

Cardiovascular diseases account for most NCD deaths causing about 17.9 million deaths annually, followed by cancers causing 9 million deaths, respiratory diseases 3.9 million deaths and diabetes causing 1.6 million deaths[5]. In order to reduce the risk of these NCDs and the risk associated with the conventional drugs use for their management, a lot of plant materials are being exploited for their medicinal properties. Also, people who are not diagnosed of any physiological disorder also use these plant materials (spice, tree bark, herbs etc.) in different ways as a kind of preventive measure.

Cinnamon is a common spice that has been used for several centuries by different cultures around the world. It is obtained from the inner bark of trees from the genus Cinnamomum, a tropical evergreen plant that has two varieties; *Cinnamomum zeylanicum* and *Cinnamomum cassia*. Almost every part of the cinnamon tree including the bark, leaves, flowers, fruit and roots, have some medicinal or culinary use.

Infact, high levels of antioxidants are present in cinnamon and are likely to contribute to its health benefits. The current in vitro and in vivo studies involving cinnamon suggest that it could be used to treat and prevent chronic diseases, such as obesity and diabetes mellitus which are prevalent in the developed and developing world[6,7]. *Cinnamomum cassia* bark contains bioactive components which includes; cinnamic aldehyde, cinnamyl aldehyde, tannins and carbohydrates. These bioactive compounds have also been found to have anti-microbial, anti-inflammation and anti-tumour activity[8,9].

Similarly, cinnamon extracts are reported to have beneficial effects on people with normal and impaired glucose tolerance, metabolic syndrome, type 2 diabetes, insulin sensitivity and insulin resistance[10]. Whole cinnamon and/or aqueous extracts of cinnamon also have beneficial effects on lipids, antioxidant status, blood pressure, lean body mass and gastric emptying. However, not all studies have shown positive effects of cinnamon, as well as the type of subject is likely to affect the response to cinnamon[8].

Therefore, this study was conducted to ascertain if cinnamon use in the absence of any diagnosed physiological disorder is beneficial. This study is also expected to provide information on the possible alternative for pharmacological therapies used in the management of non-communicable diseases such as diabetes, obesity and hyperlipidemia. In addition, information on the possible toxic effect of cinnamon powder to the liver will also be provided.

MATERIALS AND METHODS

Materials: Cinnamon (*Cinnamomum cassia*) dried bark was obtained from Agege market in Lagos State, Nigeria. Male wistar rats weighing 110-130 g and aged 9-10 weeks were obtained from Animal Research Facility, Babcock University. Other materials used include; cotton wool, nose mask, hand sanitizer, glucometer, test tubes, sample bottles, bowls, plain bottles, gloves, syringes, weighing scale, dissecting set, measuring cylinder, distilled water, mini metabolic cages, water bottles and ethanol.

Processing of cinnamon: Cinnamon dried barks were sorted, washed and dried at 60°C before being reduced to powder in a mill. The powdered cinnamon was preserved in an air-tight container until it was required.

Animal studies: Ethical approval for the study was obtained from Babcock University Health Research Ethics Committee. The experiment was carried out at the Animal Facility Babcock University, Ilishan-Remo, Ogun State Nigeria. A total of 15 male wistar rats were housed in cages at a temperature of 28±2°C and 12 h artificial light period for 7 days for acclimatization and the same condition was maintained during the experiment. During the acclimatization period, the rats were given free access to standard rat feed and water.

Rats were randomly divided into 3 groups (n = 5) consisting of one control group and two treatment groups. The control group A was given standard rat feed only, while treatment group B was given standard rat feed containing 5% concentration of cinnamon powder and treatment group C was given standard rat feed in which 2.5% concentration of cinnamon powder was incorporated[11]. Fasting blood sugar level was determined at the beginning and every two weeks during the period of the experiment using Accu-chek glucometer and weekly body weight gain was recorded. Water and daily feed intake of the rats were determined and duration of the experiment was four weeks.

Sample collection and preservation: At the end of the experiment, animals were deprived of food for 12 h and blood samples were collected for the determination of the plasma glucose concentration, the glycosylated hemoglobin, the blood lipid level and the liver function. The blood samples were kept in both heparin bottles and fluoride oxalate bottles. The blood was centrifuged and the supernatant (serum) was separated and kept in plain bottles which were refrigerated until it was required for use.

The rats were rendered unconscious through cervical dislocation and their liver was harvested in order to determine the weight. The rat livers were afterwards preserved in 10% formalin solution for histopathology.

Biochemical analyses

Fasting blood sugar: The Accu-chek glucometer was used to determine fasting blood sugar. The rats were fasted overnight for 12 h and were brought out of the cages one after the other. Scissors was sterilized using methylated spirit and the tail of the rat was also cleaned with methylated spirit. The tip of the rat tail was snipped using scissors in order to let out blood from the tail vein. Blood from the rat tail was dropped on the strip in the glucometer and the blood sugar reading was noted and recorded[12].

Plasma glucose concentration: All reagents previously refrigerated were brought to room temperature. The content of reagent R1b was reconstituted with a portion of buffer R1a to form R1.Clean and dried test tubes were properly labelled and placed in a test tube rack. Reagent R1(1000 µL) was pipetted into all the test tubes and the test tube labelled 'blank' had only reagent R1 in it. Reference standard solution (10 uL) was added to the test tube labelled 'standard' and 10 µL of the various blood samples was pipetted into their corresponding test tubes. The various solutions were properly mixed and left to incubate at 37°C for 10 min. The absorbance of the standard ($A_{standard}$) and the sample (A_{sample}) was measured against the reagent blank within 60 min. The test was carried out using the GOD-PAP method described in the Randox laboratory manual[13] for determination of plasma glucose concentration.

$$\text{Glucose concentration mmol L}^{-1} = \frac{A_{sample}}{A_{standard}} \times \text{standard concentration (mmol L}^{-1})$$

$$\text{Mg dL}^{-1} = \frac{A_{sample}}{A_{standard}} \times \text{standard concentration (mg dL}^{-1})$$

Glycosylated haemoglobin: The determination of glycosylated haemoglobin involve the preparation of hemosylate, the separation of glycosylated hemoglobin and the determination of total hemoglobin fraction. To prepare hemosylate, lysing reagent (0.5 mL) was measured into tubes labelled control (C) and test (T). The reconstituted control (0.1 mL) and the well mixed blood samples were added to the appropriately labelled tubes. The solution was mixed until complete lysis was evident and the mixture was allowed to stand for 5 min.

In the separation of glycosylated hemoglobin (GHb), the caps of the ion-exchange tubes were removed and tubes were labelled as control (C) and test (T). Hemosylate (0.1 mL) was added to the appropriately labelled ion exchange tube. A resin separator was inserted into each tube so that the rubber sleeve is approximately 1cm above the liquid level of the resin suspension and the tubes were mixed on a rotator continuously for 5 min. The resin was allowed to settle and then the resin separator was pushed into the tubes until the resin was firmly packed. The supernatant fluid was poured directly into a cuvette and absorbance of each sample in a spectrophotometer at 546 nm was measured against distilled water.

Five millilitre of distilled water was dispensed into tubes labelled 'T' and 'C' and 0.02 mL of the hemolysate was added into the appropriately labelled tubes. The solution was properly mixed and each absorbance was read against distilled water for total hemoglobin fraction. This method is as described in the Spectrum glycosylated hemoglobin kit manual[14].

$$\text{Ratio of control } (R_C) = \frac{\text{Abs. test GHb}}{\text{Abs. test THb}}$$

$$\text{Ratio of test } (R_T) = \frac{\text{Abs. test GHb}}{\text{Abs. test THb}}$$

$$\text{GHb (\%)} = \frac{\text{Ratio of test (RT)}}{\text{Ratio of control (Rc)}} \times 10 \text{ (value of control)}$$

Lipid profile test

Serum cholesterol: Clean and dried test tubes were properly labelled as 'tests', 'standard' and 'blank'. The test tubes were placed in a rack and 1000 µL of the cholesterol reagent was dispensed into all the test tubes. Added to test tube labelled blank was 10 µL of distilled water, while10 µL of the reference standard was pipetted into the test tube labelled standard and 10 µL of the blood sample was added to the other test tubes excluding the standard and the blank test tubes. The

content of the various test tubes were properly mixed and then incubated for 10 min at 25°C. Thereafter, the content of each test tube was poured into cuvette and the absorbance (at 546 nm) of the samples was measured against the reagent blank within 60 min[13].

$$\text{Concentration of cholesterol in sample} = \frac{\Delta A_{sample}}{\Delta A_{standard}} \times \text{Concentration of standard}$$

Serum HDL cholesterol: Properly labelled, clean and dried centrifuge tubes were placed on rack and 500 µL of the diluted precipitant R1 was pipetted into all the test tubes. Standard solution (200 µL) was pipetted into the test tube labelled as 'standard' and 200 µL of the blood samples was also dispensed into their respective test tubes. The content of each test tube was mixed and allowed to stay for 10 min at room temperature. They were then centrifuged for 10 min at 4,000 rpm (revolution per minute). The clear supernatant fluid was separated and cholesterol content was determined using the CHOD-PAP method.

Cholesterol reagent (1000 µL) was pipetted into clean, dry and properly labelled test tubes and 1000 µL of the standard supernatant was added to the content of the test tube labelled 'standard'. Also, 100 µL of the samples supernatant was pipetted into their correspondingly labelled test tubes and 100 µL of distilled water was dispensed into the test tube labelled 'blank'. The content of the various test tubes was mixed and incubated for 10 min at 25°C. The absorbance of the sample (A_{sample}) and standard ($A_{standard}$) was measured against the reagent blank within 60 min[13].

$$\text{Concentration of HDL cholesterol in supernatant (mg dL}^{-1}) = \frac{\Delta A_{sample}}{\Delta A_{standard}} \times \text{conc. of standard}$$

Calculation of LDL cholesterol: The calculation of LDL cholesterol was according to the Randox laboratory manual for the determination of serum LDL cholesterol[13]:

$$\text{LDL cholesterol (mg dL}^{-1}) = \text{Total chelesterol} \frac{\text{Triglycerides}}{2.2} - \text{HDL cholestrol}$$

Serum triglycerides: The test was carried out using the CHOD-PAP method for determination of serum triglyceride[13]. The content of reagent R1b was reconstituted with a portion of buffer R1a to form R1. The entire content of bottle R1b was transferred to R1a, rinsing vial R1b several times. Clean and dried test tubes labelled 'standard', 'blank' and 'tests' (for blood samples) were arranged in a test tube rack. Reagent R1 (1000 µL) was pipetted into all the test tubes followed by addition of 10 µL of the standard solution into the test tube labelled standard and 10 µL of the various blood samples into the rest of the test tubes excluding the test tubes labelled 'blank' and 'standard'. The content of each test tube was properly mixed and incubated at 25°C for 10 min. The absorbance (546 nm) of the sample (A_{sample}) and standard ($A_{standard}$) was measured against the reagent blank within 60 min.

$$\text{Triglyceride concentration (mg dL}^{-1}) = \frac{A_{sample}}{A_{standard}} \times \text{standard conc. (mg dL}^{-1})$$

Liver function test

Alanine aminotransferase and Aspartate aminotransferase: The reagents for the analysis were maintained at room temperature. Clean and dried test tubes were properly labelled as 'blank' and 'samples' and 0.5 mL of solution R1 was pipetted into all the test tubes. Distilled water (0.1 mL) was pipetted into the test tube labelled blank and 0.1 mL of the various blood samples was also dispensed into their appropriate test tube. The content of the various test tubes was properly mixed and incubated for exactly 30 min at 37°C. After incubation, 0.5 mL of solution R2 was added to all the test tubes. The content of the various test tubes was mixed and allowed to stand for exactly 20 min at 25°C.

Five millilitre of sodium hydroxide was dispensed into all the test tubes and the content of the test tube was mixed. The absorbance of the sample (Asample) was read against the reagent blank after 5 min using a spectrophotometer and the activity of serum Alanine aminotransferase (ALT) and As partate aminotransferase (AST) was obtained from the table for determination of ALT and AST in serum[13].

Liver histopathology: Histological test used was Hematoxylin and Eosin Staining Procedure[15]. Deparaffinise slides were placed in xylene solution which was changed three times within 3 min. Tissue sections were hydrated by subjecting them to decreasing concentration of alcohol (100% alcohol and 95% alcohol) with two changes in 3 min each. Afterwards they were rinsed in distilled water until ripples disappeared from the slides. The slides were then placed in hematoxylin for staining for 15 min after which the slides were washed under running water till the water was clear.

The slides were immersed in 1% hydrochloric acid alcohol differentiation liquid solution with 6 quick dips for 30 sec until the slides were red. It was then rinsed in water for about 15 min till a section could be seen to be blue. Afterwards, the differentiation was checked microscopically to see the nuclei that should be distinct and the cytoplasm which should be uncoloured. The tissue sections were dipped in alkaline solution (Bluing Agent) for about 5 min and were washed in lukewarm water thereafter.

The same tissues were stained in eosin for 2 min and were dehydrated in increasing concentration of alcohol (95 and 100%) with three changes in 2 min. The tissues were then cleared in 3 changes of xylene for 2 min each and the cover glass was mounted.

Statistical analysis: All analyses were carried out in triplicates and results were recorded as Mean±SD. Difference between the groups was determined by subjecting the data to analysis of variance and means were separated using Duncan multiple range test (SPSS Version 20.0).

RESULTS

Mean weight gain, feed and water consumption: The mean weight gain, mean feed and water consumed are presented in Table 1. Group B (fed 5% cinnamon) had the least mean weight gain (31.40±14.47) while group A (control) had the highest mean weight gain (39.40±9.10).

At week 1, the Group B (fed 5% cinnamon) consumed the highest amount of feed (113.14±9.61) which was statistically different from that of the other groups. There was no significant difference in the means of feed consumed in the subsequent weeks.

At week 1, there was no significant difference in the means of water consumed by all the groups. But in the following weeks, the Group B (fed 5% cinnamon) consumed the highest amount of water.

Fasting blood sugar: Figure 1 shows the result of fasting blood sugar over the period of four weeks. The baseline blood sugar for Group A, B and C was 91.2, 90.6 and 88.8 mg dL^{-1} respectively. By the second week, the blood sugar of all the groups dropped. However, that of Group B was more drastic (dropped from 90.6-60.2 mg dL^{-1}). At the expiration of the fourth week, Group B and C had close blood sugar level of 65.6 and 66.6 mg dL^{-1} respectively.

Plasma glucose concentration and glycosylated hemoglobin (HbA1c): Table 2 presents the results of plasma glucose concentration and glycosylated hemoglobin of the test rats. Group B (fed 5% cinnamon) had the lowest average plasma glucose concentration (80.84 mg dL^{-1}), followed by Group C (110.5 mg dL^{-1}) and the control group (122.29 mg dL^{-1}). The results of mean HbA1c ranged from 4.97±0.13 for Group A, to 4.85±0.13 for Group C and 4.77±0.23 for Group B.

Lipid profile of the test rats: Figure 2 shows the results of the lipid profile of the rats. Group C (fed 2.5% cinnamon) had the highest means of serum total cholesterol (100.48 mg dL^{-1}). This was followed by the mean serum total cholesterol of Group A (control) which was 92.48 mg dL^{-1} and Group B (fed 5% cinnamon) had the least serum total cholesterol of 91.23 mg dL^{-1}.

Group B (fed5% cinnamon) had the highest mean serum HDL of 30.78 mg dL^{-1}, followed by Group C (2.5% cinnamon) with mean serum HDL of 29.41mg dL^{-1} and Group A (control) had the least serum HDL of 27.83 mg dL^{-1}.

Group C (fed 2.5% cinnamon) had the highest mean serum LDL 31.94 mg dL^{-1}, followed by Group A

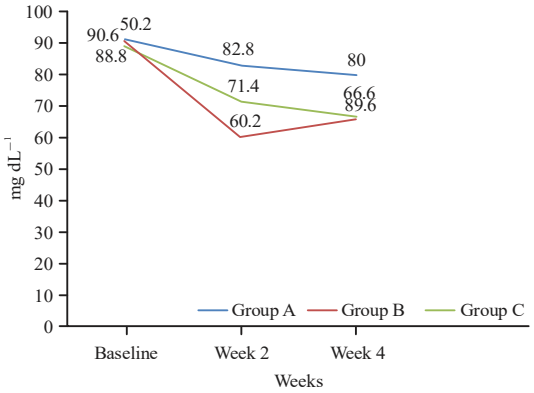

Fig. 1: Blood sugar of the test rats over four weeks
Group A: Control, Group B: 5% Cinnamon, Group C: 2.5% Cinnamon

Table 1: Mean feed and water consumed by rats per day and overall weight gain

	Group A	Group B	Group C
Weight gain (g)	39.40±9.10[a]	31.40±14.47[a]	32.40±6.58[a]
Feed intake (g)			
Week 1	88.86±24.75[a]	113.14±9.61[b]	95.28±23.65[ab]
Week 2	104.00±19.98[a]	106.57±13.66[a]	98.86±25.53[a]
Week 3	106.86±18.95[a]	107.14±14.24[a]	111.57±13.15[a]
Week 4	99.57±26.01[a]	105.00±17.32[a]	90.57±18.82[a]
Water intake (mL)			
Week 1	197.86±57.55[a]	242.71±61.00[a]	196.29±39.14[a]
Week 2	168.43±23.68[a]	269.43±55.73[b]	203.29±56.50[a]
Week 3	163.29±31.61[a]	231.57±71.03[b]	193.29±41.76[ab]
Week 4	176.14±39.25[a]	285.00±37.53[b]	214.43±63.57[a]

Means in the same row with the same superscript are not significantly different ($p \leq 0.05$), Group A: Control, Group B: 5% Cinnamon, Group C: 2.5% Cinnamon

Table 2: Mean plasma glucose concentration and glycosylated hemoglobin (HbA1c)

Parameters	Group A	Group B	Group C
Plasma glucose	122.29±8.9[a]	80.84±8.42[b]	110.5±15.93[a]
HbA1c	4.97±0.13[a]	4.77±0.23[a]	4.85±0.13[a]

Means in the same row with the same superscript are not significantly different ($p \leq 0.05$), Group A: Control, Group B: 5% Cinnamon, Group C: 2.5% Cinnamon

Table 3: Liver function and weight of the liver

Parameters	Group A	Group B	Group C
AST (IU L^{-1})	60.00±0.83[a]	60.21±1.07[a]	62.10±2.84[a]
ALT (IU L^{-1})	6.14±0.74[a]	5.85±0.65[a]	7.30±0.34[b]
Liver weight (g)	5.12±0.66[a]	5.50±0.70[a]	5.34±0.44[a]

Means in the same row with the same superscript are not significantly different ($p \leq 0.05$), Group A: Control, Group B: 5% Cinnamon, Group C: 2.5% Cinnamon, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

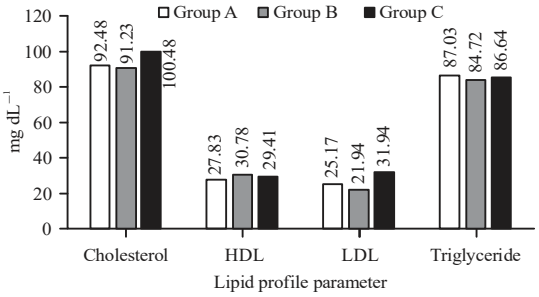

Fig. 2: Lipid profile of the test rats

Group A: Control, Group B: 5% Cinnamon, Group C: 2.5% Cinnamon

(control) with value of 25.17 mg dL^{-1} and Group B (fed 5% cinnamon) had the least mean serum LDL of 21.94 mg dL^{-1}.

Mean serum triglyceride ranged from 87.03 mg dL^{-1} for Group A (control) to 86.04 mg dL^{-1} for Group C (fed 2.5% cinnamon) which was followed by 84.72 mg dL^{-1} for Group B (fed 5% cinnamon).

Liver function test and liver weight: Table 3 shows the level of liver function enzymes activity; aspartate aminotransferase (AST) and alanine aminotransferase (ALT) and also the liver weight of the test rats. The level of enzyme AST ranged from 60.00±0.83 to 62.10±2.84. The value for ALT of group C was higher (7.30±0.34) than that of the control group (6.14±0.74) and group B (5.85±0.65). The mean liver weight for groups A, B and C was 5.12±0.66, 5.50±0.70 and 5.34±0.44 respectively.

Liver histopathology: Figure 3 shows the photomicrograph of the liver. The control group reveals a better histoarchitecture except for few cases of enlarged sinusoids. The group B showed some depositions of plagues and mild necrotic features of the hepatocytes at magnification of ×400. In addition sinusoidal enlargement can also be noticed. Group C also present a near to normal histology when compared with the control group.

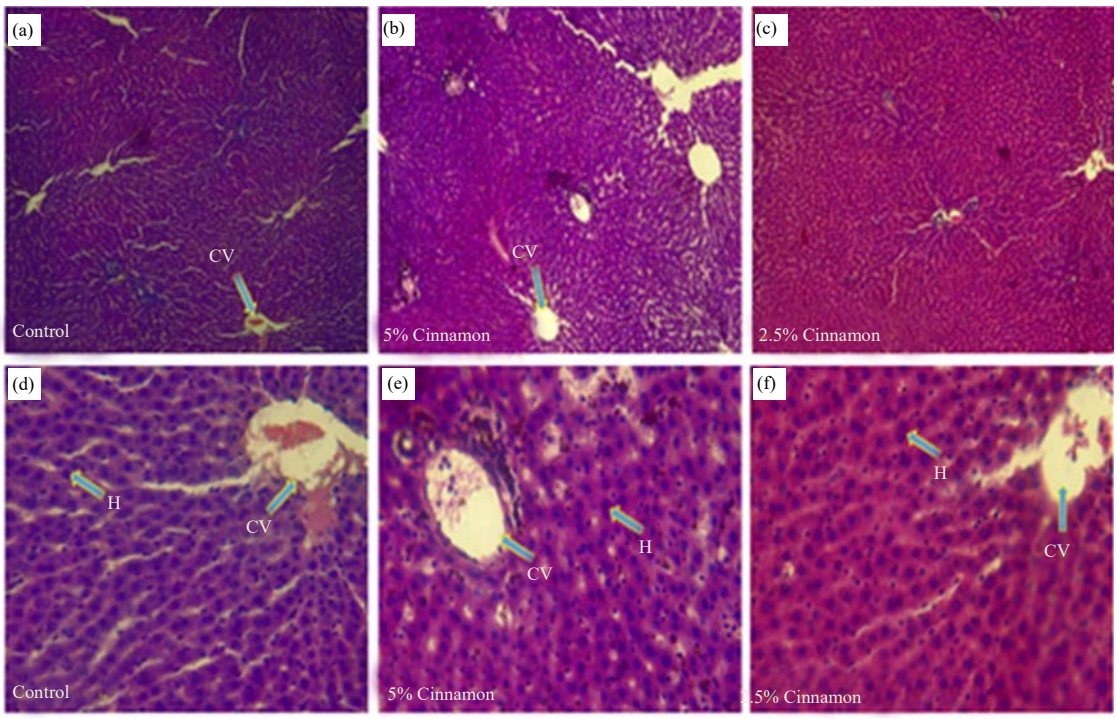

Fig. 3(a-f): Liver histopathology

CV: Central vein, H: Hepatocyte, H and E×100 above and ×400 below

DISCUSSION

Effect of cinnamon, a popular spice used in cooking, was determined in normal wistar rats in order to further substantiate information about its use for medicinal purposes. The average concentrations of cinnamon ingested by the test rats during the experiment was calculated to be 8.5, 6.7, 6.6, 6.3 mg g^{-1} body weight for rats fed 5% cinnamon and 3.7, 3.4, 3.2, 2.6 mg g^{-1} body weight for rats fed 2.5% cinnamon for the first, second, third and fourth week respectively. The Ingestion of these concentrations of cinnamon powder did not cause any behavioural changes and mortality in the test animals.

Table 1 shows the mean feed and water intake including the corresponding mean weight gain of the test animals. There was significant difference in the means of feed intake of the animals in the first week of the experiment, while for the rest of the weeks the feed intake was not significantly different. However, water intake of the test rats in the different groups in the first week was comparable but varied for the remaining period of the experiment. This finding is supported by Ahmad *et al.*[16] who reported that cinnamon extract did not significantly affect water and feed intake of rats. Also, there was no significant difference in the weight gain of the rats which is consistent with the findings of Ahmad *et al.*[16] and Beji *et al.*[11] who reported that non-diabetic induced rats showed normal increase in weight and therefore, cinnamon supplementation in normal rats has no effect on weight gain. It is worth mentioning that the concentration used in this study is comparable to what was reported by Beji *et al.*[11] and Bernado *et al.*[17].

Cinnamon administration caused a significant difference ($p \leq 0.05$) in the blood sugar as the blood sugar of rats of Group C (fed with 2.5% cinnamon) and B (fed with 5% cinnamon) reduced. At the end of the cinnamon administration, there was reduction in the blood sugar means from 90.6±12.05 to 65.6±8.40 for Group B and from 88.8±9.17 to 66.6±7.92 for Group C. However, this is in contradict with the findings of Beji *et al.*[11] who reported that cinnamon did not alter the blood sugar levels of normal rats treated with cinnamon while report of Ranasinghe *et al.* (2012) substantiate the finding of this research. However, there was an unexplainable slight reduction in the blood sugar of the rats in the control group. The administration of cinnamon powder caused a reduction in the plasma glucose concentration of the rats treated with both 5 and 2.5% cinnamon, when compared to the control group. This result of plasma glucose concentration substantiate the observation made in the blood sugar level determined with glucometer and proves that cinnamon powder can improve the uptake and utilization of glucose even in the tissue of normal rats. Soonham *et al.*[18] reported a similar result for Streptozotocin induced diabetic rats. There was no significant difference ($p \geq 0.05$) in the glycosylated hemoglobin (HbA1c) of all the groups. This finding is supported by Vanschoonbeek *et al.*[19] who studied the effect of cinnamon supplementation on glycemic control in post-menopausal diabetes patients. The very similar values of HbA1c for control group (4.97±0.13), Group B (4.77±0.23) and Group C (4.85±0.13) could be attributed to the short duration of the study as glycosylated hemoglobin measures the average exposure of the red blood cells to glucose (life span of red blood cell is around 120 days) within a period of 2-3 months. However, the low values of HbA1c obtained for cinnamon groups may likely be due to the effect of cinnamon as reported by Ranasinghe *et al.*[6].

The indicators of lipid profiles (serum cholesterol, high density lipoprotein, low density lipoprotein and triglycerides) slightly reduced except high density lipoprotein (HDL) in the group B (fed 5% cinnamon) when compared with the control group. These changes were not statistically significant except for the value of HDL for the group B (30.78±0.64) which was higher and significantly different from the control group (29.41±0.14). The rats in Group C had the highest total cholesterol and LDL cholesterol but the values remained within the normal range. These findings are partially supported by Rahman *et al.*[20] who observed a slight change in all the lipid profile parameters. These findings are also supported by Ranasinghe *et al.*[6] and Bodeker and Kronenberg[21] who reported the ability of cinnamon to reduce cholesterol and LDL with increase in HDL.

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) values remained within normal reference range for AST and ALT for rats, that is 50 to 150U L^{-1} and <40U L^{-1} respectively[22]. It was also reported by Anand *et al.*,[23] who studied high dose effect of *Cinnamomum zeylanicum* in healthy wistar rats. Statistically, no significant difference was observed in the liver weight of the control and treatment groups[23].

The histopathology showed alteration in the structure of the liver of test animals fed cinnamon compared to the control. There were enlarged sinusoids, deposition of plagues in the liver which could have been due to remnant of the detoxification process by the liver and mild necrotic feature of the hepatocytes. This observation is in agreement with the findings of Anand *et al.*[23] who also reported incidence of sinusoids defect and other abnormalities in the liver of rats administered cinnamon extract.

CONCLUSION

Cinnamon powder had blood sugar lowering properties and also reduced glycosylated hemoglobin at both concentrations of 2.5 and 5%. It increased HDL level with

minimal effect on other lipid profile indicators. Cinnamon used at a concentration of 5% had deleterious effect on the liver though liver enzymes AST and ALT were not significantly affected.

SIGNIFICANCE STATEMENT

This study discovered that regular consumption of cinnamon in normal rats was beneficial and increasing the HDL and reduced the glycosylated haemoglobin level. But cinnamon consumption at the concentrations used in this study may also be detrimental due to possible lowering of the blood sugar below the normal range if continued for a period of time. Also there could be deposition of plague in the liver at high concentration. Thus, this study will help researchers to uncover critical area of determining safe dosage, duration and mode of administration of cinnamon in normal subject for attainment of maximum health benefit.

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