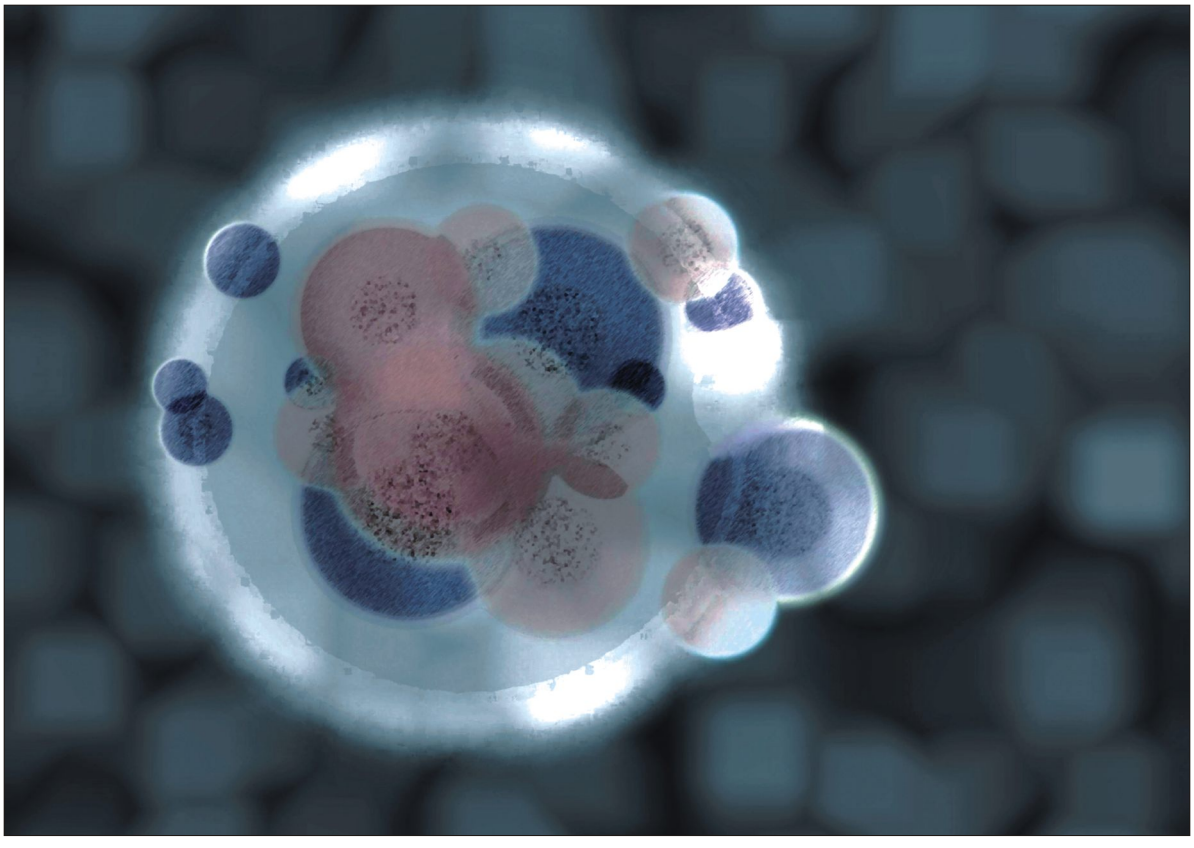

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Research Article
Protective Effects of Methanolic Stem Bark Extract of *Commiphora kerstingii* Engl. on Pancytopenia and Hyperlipidemia Induced with $CdCl_2$ in Male Wistar Rats

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Abstract

Background and Objective: *Commiphora kerstingii* (Burseraceae) is a medicinal plant with extensive health benefits and is used traditionally to cure arrow poisons. Scientific studies have reported its antimicrobial, hypoglycemic, anticonvulsant and antioxidant properties. However, its protective effect against Cadmium compounds induced hyperlipidemia and pancytopenia are scanty in literature. The study investigated the protective effect of methanolic stem bark extract of *Commiphora kerstingii* (MSBECK) on pancytopenia and hyperlipidemia induced with Cadmium chloride in males Wistar rats. **Materials and Methods:** Thirty-five male Wistar rats were divided into 5 groups, containing seven rats each. Group A was given distilled water, while group B [5 mg/kg/b.wt. of Cadmium chloride ($CdCl_2$)], group C [5 mg/kg/b.wt. of $CdCl_2$+200 mg/kg/b.wt. of MSBECK], group D [5 mg/kg/b.wt. of $CdCl_2$+400 mg/kg/b.wt. of MSBECK], while group E received 400 mg/kg/b.wt. of MSBECK only. All administration was done orally for 14 days. **Results:** The administration of Cadmium chloride caused a significant ($p<0.05$) reduction in blood cells and HDL-cholesterol levels. In contrast, it created a considerable increase in the concentration of serum total cholesterol, LDL-cholesterol, triglycerides, cardiac risk indices. However, the co-administration of MSBECK caused a significant ($p<0.05$) dose-dependent improvement in blood cells and restored total serum cholesterol, triglycerides, LDL-cholesterol and cardiac risk indices to normal ranges. **Conclusion:** The study showed that methanolic stem bark extract of *Commiphora kerstingii* protected against a reduction in all of the formed elements of blood and high lipid in the blood induced by the administration of Cadmium chloride in male Wistar rats.

Key words: Cadmium chloride, medicinal plant, *Commiphora kerstingii*, hyperlipidemia, pan

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

INTRODUCTION

There are enormous environmental agents involved in the causes, symptoms and course of human diseases. Some heavy environmental metals emitted naturally or through anthropogenic means affect many body systems, including the blood and immune system, the nervous system and the cardiovascular system, by disrupting the healthy metabolism[1]. Cadmium and related compounds occur in sources where they can easily have contact with humans such as in industries, commercially used in laser, paints, batteries and other products, including vegetables and cereals[1]. Cadmium is on the list of the ten chemicals that are of interest to public health as published by the World Health Organization[2]. Smokers and industrial workers are at risk of Cadmium toxicity[1], their exposure destroys the liver and the kidney[3] and also have carcinogenic properties in humans[4,5].

Exposure to Cadmium is related to dyslipidemia prevalence and cardiovascular diseases[6]. Cadmium exposure leads to its accumulation in cardiovascular tissues and in particular, leads to endothelial anomalies, the thickness of carotid intima-media[7] and atherosclerotic plaques formation[8]. The loss of endothelial function that ensues may give rise to the creation of thrombus and then myocardial infarction[9]. The mechanism of toxicity of Cadmium also involves endothelial nitric oxide inhibition and suppression of acetylcholine-induced vascular relaxation which may result in high blood pressure, with free radical production, implicated as a general mechanism[10]. Long-time exposure increases the risk of peripheral arterial diseases[11].

The blood is not only a medium for the transportation of absorbed substances but also a target for its toxicity[12,13]. Cadmium may induce anaemia by three mechanisms which are: the advancement of blood destruction, building up of iron in the body and inadequate production of erythropoietin[14]. The search for an effective and generally acceptable treatment for Cadmium toxicity is a work in progress. EDTA widely used in the procedure may lead to kidney damage[15]. However, studies have shown that EDTA is more effective when used together with antioxidants[16]. Therefore, since plants are a good source of antioxidants, they could be an alternative way of managing Cadmium toxicity.

The use of herbal remedies for treatment has been an age-long practice. Herbs are available in different forms like powders, tinctures, teas and other types[17]. One of such herbs of medicinal value is *Commiphora kerstingii*, a member of the Burseraceae family, classified as a useful plant in West Africa[18]. *Commiphora kerstingii* bark is used traditionally as protection against fire, as a cure for arrow poison[18] and to treat diabetes[19]. Screening of methanolic extract of *Commiphora kerstingii* stem bark presents metabolites such as alkaloids, anthraquinones, cardiac glycosides, flavonoids, saponins and tannins[20]. Scientific studies from previous researches have shown that *Commiphora kerstingii* possesses antimicrobial[21] glucose reducing[19] anticonvulsant[22] and antioxidant properties[20]. Therefore, this study investigated the ability of methanolic extract of *Commiphora kerstingii* stem bark to attenuate hyperlipidemia and pancytopenia induced by the administration of Cadmium chloride in male Wistar rats, to provide an alternative for the prevention of Cadmium chloride toxicity in case of exposure.

MATERIALS AND METHODS

Study area: The study was carried out between October, 2020 and February, 2021 at the Animal House of the Department of Physiology Ladoke Akintola University of Technology, Ogbomoso, Nigeria.

Chemicals/reagents: The kits for assaying lipid profiles are products of Randox Laboratories, United Kingdom. Other chemicals used are of analytical grade procured from Sigma Aldrich, UK.

Identification and extraction of plant materials: Collection of *Commiphora kerstingii* took place at Oja-Igbo, Ogbomoso, Nigeria. Plant identification by Dr. K.A. Adeniji, a botanist in the Forestry Research Institute, Ibadan, a copy of the authenticated sample is available at the Forest Herbarium, Ibadan. The voucher number is No. FHL. 110153. The plant's name was also checked with www.theplantlist.org and the data are available at http://www.ipni.org/ipni/idPlantNameSearch.do?id=127 699-1. The dried stem bark of *Commiphora kerstingii* was pulverized to obtain a considerable small particle size and then soaked in methanol for 24 hrs. After this, the sieving of the mixture took place to get the solution of dissolved active ingredients from the plant materials and the shaft (residue) removed. The concentration of the solution followed by removing the excess methanol using the Soxhlet extraction method. The concentrated sample of the stem bark extract of *Commiphora kerstingii* was dried and granulated to powder in an oven at 40°C. The powdered methanolic extract was kept in a dry place before, during and after use. The weight of the extract was taken and used in preparing a stock solution for separate doses of the extract to be administered to the rats.

Preparation of plant extracts for determination of phytochemicals and DPPH radical scavenging activity: The 50 mL of methanol and 5 g of a dried sample was mixed in a separate conical flask. Aluminium foil for covering of the container and the solution were allowed to stand for 48 hrs for extraction to take place. The extracts were filtered with a Whatman filter paper number 1 and evaporated at 70°C using a rotary evaporator. The extracts were collected and weighed. The final stock solution concentration was 50 mg mL^{-1}.

Alkaloid determination: Five grams of the sample was weighed into a 250 mL beaker and 200 mL of 20% acetic acid in solvent ethanol were mixed and then sheltered while standing for 4 hrs. The resultant solution underwent filtration and the extract was concentrated with the aid of a water-bath to a quarter of the initial quantity to the extracted sample until complete condensation occurred. The entire solution stayed for a while and the precipitate was collected after that by filtration and then weighed[23].

Determination of flavonoid: Five grams each of the plant samples were extracted continually with 100 mL of 80% aqueous methyl alcohol at ambient temperature. The entire solution was filtered using Whatman filter paper No. 42 (125 mm). The filtrate was then conveyed into a melting pot then vaporized to dryness in a water bath with the weight recorded[23,24].

Determination of saponin: Five grams of the plant sample spread in 200 mL of 20% ethanol. The resultant solution was put over a hot water bath for 4 hrs with constant stirring at about 55°C. The mixture's filtrate obtained stood separately and the residue was after that, extracted again with an additional 200 mL of 20% ethanol. The extractions were condensed to 40 mL over a water bath at 90°C and the concentrate was turned over into a 250 mL funnel for separation with 20 mL of Diethyl ether included and shaken thoroughly. The water layer recovered in a container, while the ether layer was separated and jettisoned. Another purification procedure was carried out with 60 mL of n-butanol added. The mixed n-butanol extracts were washed two times with 10 mL of 5% hydrous sodium chloride and the remained solution was heated in a water bath. The samples were desiccated in the oven to a steady weight after evaporation and the saponin content was calculated in percentage[23-25].

Determination of tannin: Five grams of the sample was measured into a 100 mL plastic bottle together with filtered water of 50 mL by volume. It was agitated for 1 hr using a mechanical shaker, which was later filtered into a 50 mL flask for volume analysis and made up to the mark with water. Then 5 mL of the filtrate was piped out with a pipette into a tube and agitated with 3 mL of 0.1 M $FeCl_3$ together with 0.1N HCl and 0.008 M potassium ferrocyanide. A photometer measured the absorption of light at a wavelength of 120 nm for 10 min. A fresh specimen was made available and the colour change that developed gave the reading of tannin acid in 100 ppm content[23].

Determination of phenolic component: The sizeable fine granules of the sample with 50 mL of ether underwent boiling for 15 min. The 5 mL of the extract was pipetted inside a 50 mL flask with 10 mL of distilled water added. Afterwards, 2 mL of ammonium hydroxide solution and 5 mL of concentrated amyl alcohol were mixed, which was allowed to stay for 30 min to develop colour. The recording of absorbance reading of the solution employs the use of a spectrophotometer at a wavelength of 505 nm wavelength[23].

DPPH radical scavenging activity: According to the method by Hsu *et al.*[26], about 0.1 mL of the concentrated extract was agitated with 1.9 mL of 0.1 mM DPPH methanolic solution. The mixture was shaken vigorously and left to stand for 30 min at room temperature. The absorbing capacity measurement at 517 nm against a blank happened using ascorbic acid as a positive control.

The DPPH radical scavenging activity was determined using the following Eq.:

$$\text{DPPH radical scavenging activity (\%)} = \frac{A_0 - A_1}{A_0} \times 100$$

Where:

A_0 = Absorbance of the control (without extract)

A_1 = Absorbance of the sample

Experimental animals and design

Experimental animals: Thirty-five male Wistar rats, weighing between 160-180 g, procured from the Department of Physiology Animal House Ladoke Akintola University of Technology, Ogbomoso, Nigeria were used in this research. They were experimental animals that stayed in ventilated

cages at room temperature, 12 hrs light/dark cycle with pelletized diet and water were given *ad libitum*. The rats underwent 2 weeks of acclimatization before the administration commenced.

Ethical statement: All the animals received humane care in line with the guidelines outlined in the National Institute of Health Submission for the management of Laboratory Animals. Ethical approval was obtained from an ethical review committee of the Oyo State Ministry of Health with reference number (AD 13/479/867) before the commencement of the study.

Experimental design: Thirty-five Wister rats were allocated into five groups containing seven rats each. The group A rats received distilled water, while group B rats were given 5 mg/kg/b.wt. of Cadmium chloride ($CdCl_2$)[27], group C rats received 5 mg/kg/b.wt. of $CdCl_2$+200 mg/kg/b.wt. of methanolic stem bark extract of *Commiphora kerstingii*, group D rats were administered 5 mg/kg/b.wt. of $CdCl_2$+400 mg/kg/b.wt. of methanolic stem bark extract of *Commiphora kerstingii,* while, group E rats received 400 mg/kg/b.wt. of methanolic stem bark extract of *Commiphora kerstingii* only. Administrations using oral cannula took place daily for 14 days.

Collection of blood samples: At the end of the experiment, the rats went through euthanasia and whole blood samples were collected through cardiac puncture into labelled EDTA bottles to avoid clotting. The serum used for lipid profiling was collected into containers without anticoagulant and allowed to clot and then centrifuged at 4000 rpm for 30 min to obtain the serum stored in a refrigerator at -40°C.

Assessment of haematological variables: The determination of Packed Cell Volume (PCV) followed the microhematocrit method. Haemoglobin (Hb) concentration was carried out according to the cyano-methaemoglobin procedure[28]. Red Blood Cell (RBC) count and White Blood Cell (WBC) were counted by the improved Neubauer haemocytometer method[29]. The mean corpuscles volume, mean corpuscles haemoglobin and mean corpuscular haemoglobin concentration was calculated by previous formula and protocolss[29,30].

Determination of serum lipid profile: Total cholesterol, HDL-cholesterol and triglyceride were determined by an enzymatic method with Randox kits using Camspec M106 UV spectrophotometer manufactured by Ohaus Corporation Pine Brook USA, as described by Ajayi *et al.*[31]. LDL-cholesterol was determined using the formula. The atherogenic indices include Cardiac Risk Ratio (CRR), Atherogenic Coefficient (AC) and Atherogenic Index of Plasma (AIP). Cardiac Risk Ratio (CRR) was determined by finding the ratio of total cholesterol concentration to that of HDL cholesterol. The atherogenic coefficient is the difference between total cholesterol and HDL cholesterol divided with HDL cholesterol while calculating the Atherogenic Index of Plasma (AIP) by using as previously described Eq.[32]:

$$\text{Log} = \frac{\text{TG}}{\text{HDL-C}}$$

Statistical analysis: Data from this study were in Mean±Standard deviation (Mean±SD) and one-way analysis of variance (ANOVA) was made use of through Graph Prism software. The student's t-test was used to compared the differences between sets of data and the value of $p<0.05$ was statistically significant.

RESULTS

Phytochemical analysis on the stem bark of *Commiphora kerstingii* showed the presence of alkaloids (40.00 mg g^{-1}), saponin (27.00 mg g^{-1}), flavonoids (0.02 mg g^{-1}), tannins (2.50 mg g^{-1}), cardiac glycoside (12.50 mg g^{-1}) and anthraquinone (17.01 mg g^{-1}). At the same time, the total phenolic compound was 00.04 mg GAE/100 g and DPPH radical scavenging capacity was 17.35% inhibition.

Table 1 showed the blood variables in rats given Cadmium chloride and methanolic extract of *Commiphora kerstingii* stem bark. Rats given Cadmium chloride without treatment with extract showed a significant decrease in blood parameters; Hb content (8.80±0.40 g dL^{-1}), Haematocrit value (39.20±1.63%), red blood cell (4.85±0.44×10^{12} L^{-1}), white blood cell (4.54±0.31×10^{9} L^{-1}), platelets count (360.20±5.61×10^{9} L^{-1}), MCHC (22.45±0.52 g dL^{-1}) and lymphocyte count (38.40±1.30%). These blood parameters were all increased in a dose-dependent manner after administration of *Commiphora kerstingii* to rats given Cadmium chloride, with 400 mg/kg/b.wt. of *Commiphora kerstingii* treatment showing a more significant increase: Hb content (14.80±0.88 g dL^{-1}), haematocrit value (47.80±1.53%), red blood cell (7.84±0.06×10^{12} L^{-1}), white

blood cell ($8.36\pm0.25\times10^{9}$ L^{-1}), platelets count ($490.40\pm6.90\times10^{9}$ L^{-1}), mchc (30.96 ± 0.72 g dL^{-1}) and lymphocyte count ($56.20\pm0.73\%$). There was also a significant increase in Hb content (15.06 ± 0.68 g dL^{-1}), haematocrit value ($50.20\pm0.99\%$), red blood cell ($7.90\pm0.61\times10^{12}$ L^{-1}), white blood cell ($8.40\pm0.38\times10^{9}$ L^{-1}), platelets count ($500.20\pm3.56\times10^{9}$ L^{-1}), MCHC (30.00 ± 1.85 g dL^{-1}) and lymphocyte count ($56.20\pm1.67\%$) in the group that was administered *Commiphora kerstingii* extract only compared with Cadmium chloride only group.

But MCV (80.82 ± 1.76 fl) and neutrophil count ($61.60\pm1.24\%$) significantly increased in the Cadmium chloride the only group, while treatment with 400 mg kg^{-1} body weight of *Commiphora kerstingii* extract reduced the values significantly: MCV (63.54 ± 1.50 fl) and neutrophil count ($42.00\pm0.80\%$). In the same pattern, the neutrophil: lymphocyte ratio increased significantly in the Cadmium chloride only group (1.60 ± 0.04) and the values decreased in a dose-dependent manner in the extract-treated groups with 400 mg/kg/b.wt. of *Commiphora kerstingii* extract having 0.75 ± 0.03 ratio.

Table 2 showed the serum lipid profile of the experimental rats. There was a significant increase in total cholesterol (156.6 ± 0.99), triglyceride (134.40 ± 2.10 mg dL^{-1}) and low-density lipoprotein (89.00 ± 0.92 mg L^{-1}) in the Cadmium chloride only treated group while the HDL cholesterol value reduced significantly (30.50 ± 0.79 mg dL^{-1}) in Cadmium chloride only treated group compared to other groups. These values were, however, reversed in a dose-dependent fashion after treatment with *Commiphora kerstingii* extract, 400 mg/kg/b.wt. of the extract decreased the, total cholesterol (113.1 ± 1.78 mg dL^{-1}), triglyceride (70.80 ± 0.58 mg dL^{-1}) and low-density lipoprotein (52.60 ± 0.76 mg dL^{-1}) value significantly, while the HDL cholesterol value increased significantly (47.00 ± 1.06 mg dL^{-1}) compared to the Cadmium chloride the only group. The extract only group (Group E) has values comparable with that of the control group.

Figure 1 showed the Cardiac Risk Ratio (CRR) in the experimental rats. CRR increased significantly in group B (5.13 ± 1.25) given Cadmium chloride and significantly decreased from groups C (3.35 ± 1.05), D (2.41 ± 1.68) to E (1.92 ± 0.39) in a dose-dependent pattern following administration of *Commiphora kerstingii* extract.

Figure 2 showed the Atherogenic Coefficient (AC) in experimental rats. Rats that received Cadmium chloride alone (group B) had a significant increase in atherogenic coefficient (4.13 ± 0.68). The effect was reduced in a dose-dependent manner after treatment with methanolic extract of *Commiphora kerstingii* stem bark from groups C (2.35 ± 0.61), D (1.41 ± 0.25) to E (0.92 ± 0.05).

Figure 3 showed the effects of methanolic extract of *Commiphora kerstingii* stem bark and Cadmium chloride on the Atherogenic Index of Plasma (AIP) in the experimental rats. Atherogenic Index of Plasma (AIP) showed an increased effect in group B then decreased significantly in group C, then group D and at last group E following *Commiphora kerstingii* administration.

Table 1: Effects of methanolic stem bark extract of *Commiphora kerstingii* on haematological parameters in Cadmium chloride-induced male Wistar rats

Variables	Group A	Group B	Group C	Group D	Group E
Hb concentration (g dL^{-1})	14.55 ± 0.48	8.80 ± 0.40*	$12.20\pm1.23^{\#}$	$14.80\pm0.88^{\#}$	$15.06\pm0.68^{\#}$
PCV (%)	48.80 ± 1.16	39.20 ± 1.63*	42.20 ± 0.65*	$47.80\pm1.53^{\#\S}$	$50.20\pm0.99^{\#\S}$
RBC (10^{12} L^{-1})	7.80 ± 0.51	4.85 ± 0.44*	$7.17\pm0.33^{\#}$	$7.84\pm0.06^{\#}$	$7.90\pm0.61^{\#}$
Total WBC (10^{9} L^{-1})	8.20 ± 0.26	4.54 ± 0.31*	$7.00\pm0.65^{\#}$	$8.36\pm0.25^{\#}$	$8.40\pm0.38^{\#}$
Platelets count (10^{9} L^{-1})	502.40 ± 3.08	360.20 ± 5.61*	390.20 ± 16.05*	$490.40\pm6.90^{\#\S}$	$500.20\pm3.56^{\#\S}$
MCV (fl)	62.56 ± 1.18	80.82 ± 1.76*	$58.86\pm1.08^{\#}$	$60.97\pm1.01^{\#}$	$63.54\pm1.50^{\#}$
MCH (pg)	18.65 ± 0.38	18.14 ± 0.71	17.02 ± 0.57	18.86 ± 0.56	19.06 ± 1.01
MCHC (g dL^{-1})	29.82 ± 1.89	22.45 ± 0.52*	$28.91\pm0.60^{\#}$	$30.96\pm0.72^{\#}$	$30.00\pm1.85^{\#}$
Lymphocytes count (%)	54.00 ± 1.35	38.40 ± 1.30*	$45.60\pm1.78^{*\#}$	$56.20\pm0.73^{\#\S}$	$56.20\pm1.67^{\#\S}$
Neutrophil count (%)	45.60 ± 1.02	61.60 ± 1.24*	$52.40\pm1.54^{*\#}$	$42.40\pm0.59^{\#\S}$	$42.00\pm0.80^{\#\S}$
Neutrophil: Lymphocyte ratio	0.84 ± 0.02	1.60 ± 0.04*	$1.12\pm0.04^{*\#}$	$0.75\pm0.03^{\#\S}$	$0.75\pm0.07^{\#\S}$

*$p<0.05$, when juxtapose with the control, $^{\#}p<0.05$, when compared with the $CdCl_2$ only group and $^{\S}p<0.05$, compared with $CdCl_2$+200 mg/kg/b.wt.

Table 2: Effects of methanolic extract of *Commiphora kerstingii* stem bark on lipid profile in Cadmium chloride induced male Wistar rats

Variables	Group A	Group B	Group C	Group D	Group E
Total cholesterol (mg dL^{-1})	101.4 ± 2.84	156.6 ± 0.99*	$129.2\pm0.77^{*\#}$	$113.1\pm1.78^{*\#\S}$	$97.13\pm1.75^{\#\S}$
Triglyceride (m dL^{-1})	66.33 ± 0.65	134.40 ± 2.10*	$88.80\pm1.33^{*\#}$	$70.80\pm0.58^{\#\S}$	$68.40\pm2.29^{\#\S}$
Low density lipoprotein (mg L^{-1})	48.80 ± 1.05	89.00 ± 0.92*	$67.60\pm1.58^{*\#}$	$52.60\pm0.76^{\#\S}$	$46.40\pm0.55^{\#\S}$
High density lipoprotein (mg dL^{-1})	49.20 ± 1.33	30.50 ± 0.79*	$38.60\pm1.97^{*\#}$	$47.00\pm1.06^{\#\S}$	$50.60\pm1.67^{\#\S}$

*$p<0.05$, when juxtapose with the control, $^{\#}p<0.05$, when compared with the $CdCl_2$ only group and $^{\S}p<0.05$, compared with $CdCl_2$+200 mg/kg/b.wt.

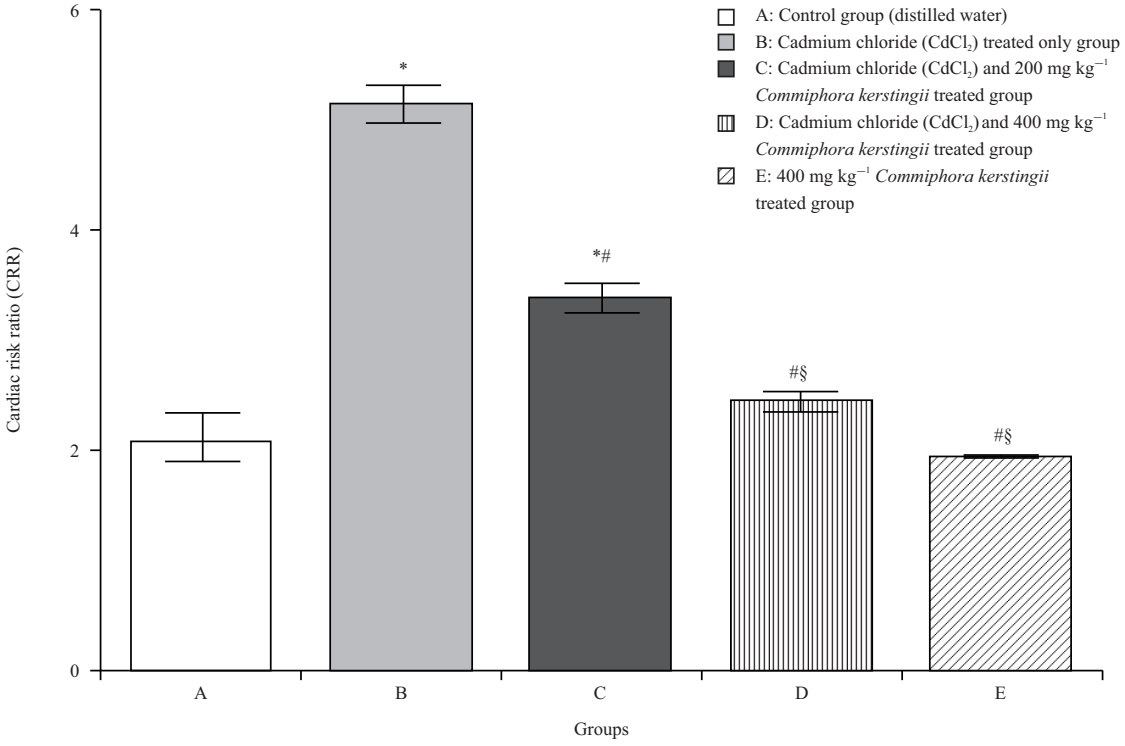

Fig. 1: Effects of methanolic extract of *Commiphora kerstingii* stem bark on Cardiac Risk Ratio (CRR) in Cadmium chloride-induced male Wistar rats

*p<0.05, when juxtapose with the control, #p<0.05, when compared with the $CdCl_2$ only group and § p<0.05, compared with $CdCl_2$+200 mg/kg/b.wt.

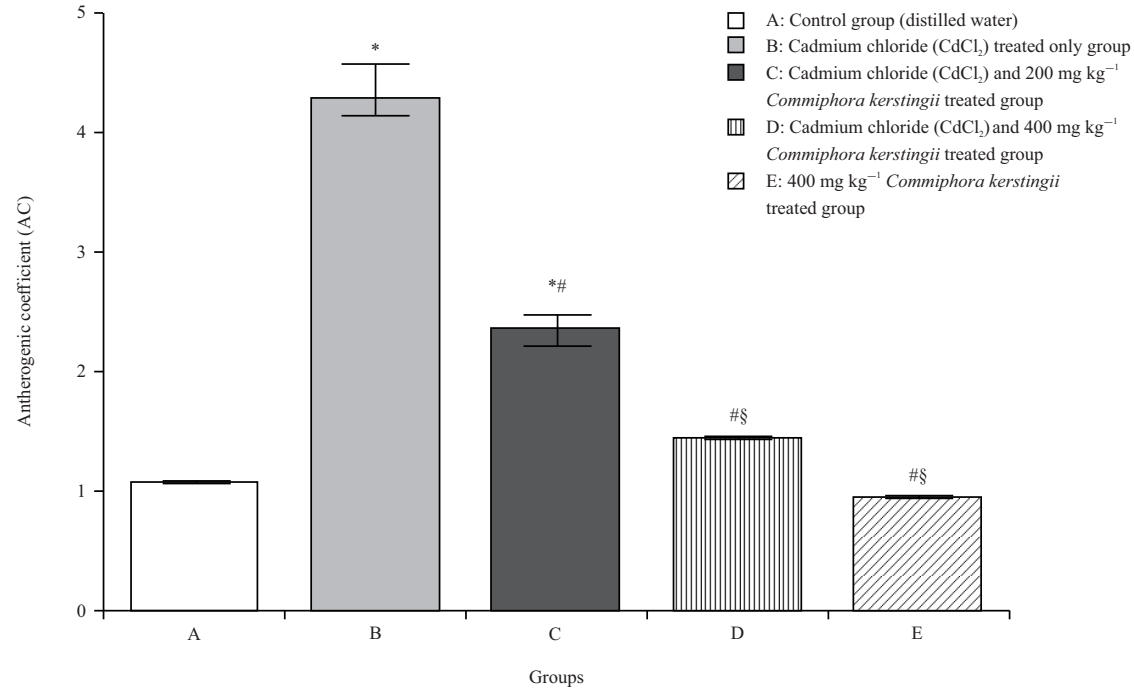

Fig. 2: Effects of methanolic extract of *Commiphora kerstingii* stem bark on Atherogenic Coefficient (AC) in Cadmium chloride-induced male Wistar rats

*p<0.05, when juxtapose with the control, #p<0.05, when compared with the $CdCl_2$ only group and § p<0.05, compared with $CdCl_2$+200 mg/kg/b.wt.

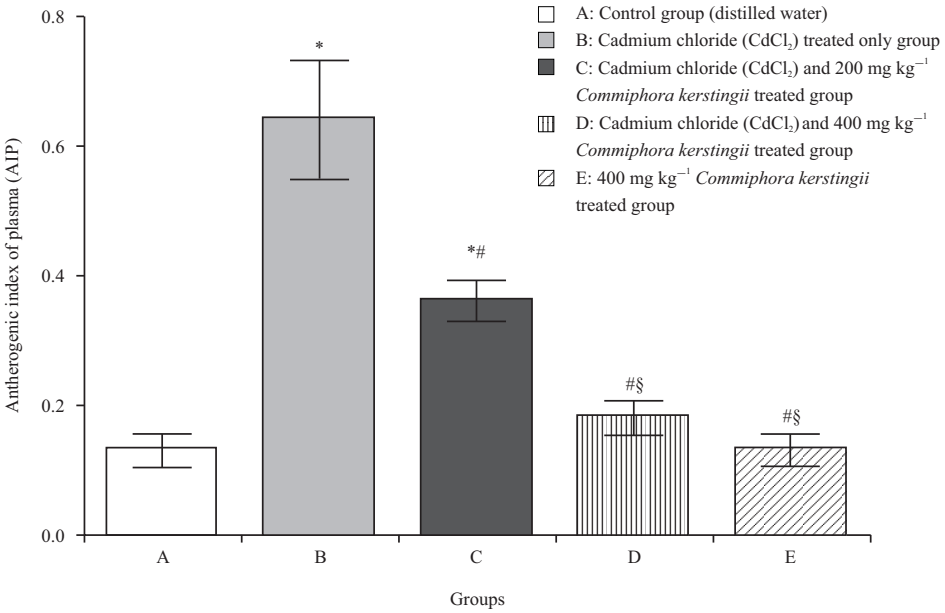

Fig. 3: Effects of methanolic extract of *Commiphora kerstingii* stem bark on Atherogenic Index of Plasma (AIP) in Cadmium chloride-induced male Wistar rats

*p<0.05, when juxtapose with the control, #p<0.05, when compared with the $CdCl_2$ only group and § p<0.05, compared with $CdCl_2$+200 mg/kg/b.wt.

DISCUSSION

Screening of MSBECK showed the presence of alkaloids, saponin, flavonoids, tannins, cardiac glycoside and anthraquinone. In a study by Gan *et al.*[33], there is a significant positive correlation between total alkaloid concentration and antioxidant capacities (ferric reducing antioxidant potential, hydrogen radical scavenging ability and lipid peroxidation inhibition ability). Saponin, flavonoids and tannins exhibit some antioxidant activities which corroborated the result of the DPPH radical scavenging capacity of the plant, showing that the plant has an antioxidant capacity as earlier reported by Ibrahim *et al.*[21].

The most sensitive system to determine the toxicity of any compound is the haemopoietic system[34]. Cadmium underwent intestinal absorption following administration orally and after that, transported through the blood, from where the distribution through the red blood cells and albumin occur[35].

The result of haematological variables in this study depicts that rats exposed to Cadmium chloride developed pancytopenia, which is a medical condition associated with generally reduced blood cell components[27]. The result could be due to the ability of Cadmium chloride to induce toxicity on hematopoietic processes, in which stress plays an important role[36]. Cadmium could also reduce blood cell components through reduced production as a result of reduced erythropoietin secretion. Also, other mechanisms could be increased haemolysis and consequent accumulation of iron in the body or movement of blood cells from the periphery into the tissues, which could also be responsible for the result obtained after Cadmium chloride administration[14,37].

Administration of *Commiphora kerstingii* restored PCV, RBC, WBC, Hemoglobin, Platelets, MCV, MCHC, lymphocyte and neutrophil to the normal range as the control rats. The result suggested that *Commiphora kerstingii* has a beneficial effect on the blood production process, which is consistent with the finding by Maidala and Abdullahi[38], who reported *Commiphora kerstingii* as an effective supplement that improved the haematological indices in broiler chickens. The dose-dependent increase in total WBC count, neutrophil and lymphocyte count by the extract suggests that the extract can improve the immune function, as reported for garlic extract[39]. The neutrophil and lymphocyte ratio reduced with the administration of the extract which suggested that the extract reduced inflammation since the neutrophil and lymphocyte ratio is a marker of the degree of inflammation[40]. *Commiphora kerstingii* also restored platelet counts to values comparable to those seen in the control group. A similar result was obtained for Carica papaya leaf extract on platelet count, too[41]. The beneficial and therapeutic effect of this extract on blood cells suggested that it could protect against Cadmium-induced toxicity on the haemopoietic system and this could be that the extract contains bioactive plant substances that

can stimulate the production of blood cells. Saponins and alkaloids screened from the MSBECK could work synergistically with the antioxidant potential as indicated by the DPPH inhibition capacity. The antioxidant potential could be responsible for its medicinal properties and influence on the blood[21].

Results from this research indicated that administration of Cadmium chloride caused a significant increase of serum total cholesterol, triglycerides, low-density lipoprotein, cardiac risk ratio, atherogenic coefficient and the Atherogenic Index of Plasma (AIP). A noticeable decrease in the level of High-Density Lipoprotein (HDL) in rats treated with Cadmium chloride compared to the control rats occurred. The results could be associated with the ability of Cadmium to cause peroxidation and modification of cellular structure, an event usually connected with the high amount of LDL cholesterol (bad cholesterol) as opposed to the low level of HDL cholesterol (Good cholesterol)[42,43]. HDL cholesterol is good because of its ability to remove cholesterol from within arteries and return them to the liver for excretion[44]. A high level of HDL cholesterol is negatively correlated with cardiovascular diseases while the low level of HDL correlates positively with a high rate of heart diseases[45].

Commiphora kerstingii extract showed lipid-lowering potentials as it significantly decreased TG, total cholesterol, LDL, Cardiac risk ratio, atherogenic coefficient and the atherogenic index of plasma and increased HDL in rat's serum. The result depicted the extract as a potential candidate for the treatment of hyperlipidemia and cardiovascular diseases[46]. The hypolipidemic and blood-boosting actions of the extract may be due to its phytochemical components like alkaloids, saponins, anthraquinones, cardiac glycosides, tannins and flavonoids as earlier reported[20] and confirmed by this study. Various phytochemicals possess beneficial physiological actions on the production of blood cells and lipid metabolism in man and animals. Reports showed that saponin has the cholesterol-lowering capacity by excreting it as bile acid[47] and therefore prevent cardiovascular diseases[48]. Since atherogenic indices are indicators of the risk of developing heart disease, an increase in the index is directly proportional to the risk of developing heart diseases[49-51]. Therefore, the reduced indices as a result of the administration of *Commiphora kerstingii* indicate that the stem bark extract of this plant may have a protective capacity against coronary heart diseases.

CONCLUSION

Results from this study revealed that methanolic extract of *Commiphora kerstingii* stem bark protects against the development of pancytopenia and hyperlipidemia with the potential of preventing coronary heart diseases due to its constituent phytochemicals and antioxidant capacity. This study, therefore, justifies the use of *Commiphora kerstingii* stem bark as a therapy for boosting blood and in the treatment of cardiovascular diseases.

SIGNIFICANCE STATEMENT

This study discovered the potentials of *Commiphora kerstingii* stem bark in protecting against the development of pancytopenia and hyperlipidemia that can be beneficial for the prevention and treatment of coronary heart diseases. Therefore, this study will help researchers to uncover the critical areas of development of novel and alternative therapy for cardiovascular diseases that many researchers were not able to explore. Thus a new theory on the use of *Commiphora kerstingii* stem bark in the management of cardiovascular diseases may be arrived at.

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