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Original Research Article

Preliminary Evaluation of Cinnamon Oil for Cardio-protection and Lipid Regulation

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Abstract: Hyperlipidaemia is an escalating public-health concern; prevalence estimates in the USA approach 30 million, and current projections suggest that, if trends persist, up to one in three adults may have diabetes by 2050. The present study investigated the anti-hyperlipidaemic and cardioprotective potential of cinnamon oil administered orally at 25 and 50 mg/kg. We employed a dexamethasone-induced hyperlipidaemia model to evaluate lipid-lowering activity. Cardioprotective effects were assessed using a doxorubicin-induced cardiotoxicity model. Plasma lipid parameters, including total cholesterol, triglycerides, high-density lipoprotein and low-density lipoprotein, together with cardiac biomarkers lactate dehydrogenase and creatine kinase-MB (CK-MB), were measured to quantify metabolic and myocardial responses. Dexamethasone administration produced marked hyperlipidaemia, while doxorubicin treatment resulted in biochemical evidence of cardiac injury, reflected by elevated LDH and CK-MB. Treatment with cinnamon oil produced dose-dependent improvements in lipid profiles and attenuation of cardiac biomarker elevation; the 50 mg/kg dose exhibited greater efficacy than 25 mg/kg in correcting dyslipidaemia. Overall, cinnamon oil at both tested doses demonstrated significant anti-hyperlipidaemic and cardioprotective effects in validated rodent models. These results support further examinations of cinnamon oil as a potential therapeutic agent for dyslipidaemia and chemotherapy-related cardiotoxicity, including mechanistic studies and dose- optimization before clinical evaluation.

Keywords: Cinnamon Oil, Cardioprotective, Hyperlipidaemic, Biomarkers elevation.

INTRODUCTION

High levels containing total cholesterol, low levels containing lipoprotein, and very low levels containing lipoprotein in the blood, together with low levels of high-density lipoprotein, are the hallmarks of hyperlipidaemia [1]. Hyperlipidaemia is characterized by an abnormally high level of the lipid's cholesterol and triglycerides in the blood, which increases the likelihood of cardiovascular disease and its consequences, such as atherosclerosis. Some of the most common medical issues associated with hyperlipidaemia are xanthomas, gallstones, pancreatitis, hypertension, coronary artery disease, and myocardial infarction. For instance, coronary artery disease is a major killer worldwide, and nations like India may anticipate an increase in CAD cases by 2020 as a result of the increasing frequency of hyperlipidaemia [2-3]. A persistent inflammatory response initiated by an excess of lipids in the arterial walls is a common component in the pathophysiology of coronary artery disease [4]. *Cinnamon* (*Cinnamomum zeylanicum and Cinnamomum cassia*), a well-known spice from the Lauraceae family, has garnered attention for its multifaceted therapeutic properties, demonstrating free radical scavenging, inflammatory pathway modulation, glycemic regulation, pathogen inhibition, and cardiovascular system protection. Cinnamon's bioactive components, such as *cinnamaldehyde, cinnamic acid*, and *cinnamate*, contribute to its lipid-lowering and cardiovascular benefits [5]. These compounds have shown promise in alleviating hyperlipidemia and mitigating the risk of heart disease by reducing lipid levels in the bloodstream and improving vascular health. Studies also suggest that *cinnamon's* essential oils contain cinnamaldehyde, which possesses anti-tyrosinase activity, further highlighting its therapeutic potential [6]. Additionally, procyanidins, including both A-type and B-type linkages, present in cinnamon, exhibit antioxidant properties, further supporting its role in combating oxidative stress and inflammatory processes associated with hyperlipidemia and CVD. Beyond its role in cardiovascular health, cinnamon has demonstrated efficacy as an antimicrobial, antifungal, and anticancer agent. It has been traditionally used to treat various ailments, including toothaches and oral health issues, owing to its beneficial effects on oral microbiota [7-8].

MATERIALS AND METHODS

Experimental Rodents:

The study used Wistar albino rats, both male and female, with a weight range of 150-200 g. Thanks to the approval of the preclinical procedure by the Institutional Animal Ethics Committee, the rodents were obtained from the institute's animal facility. Using regulated environmental conditions. The rats were kept in conventional polypropylene cages, three animals per cage. They had unlimited access to drinkable water that had not been distilled and were given a regular meal of Hindustan Lever chow pellets. A specific time of fasting was observed before the experimental procedures began. We followed the IAEC's ethical rules for animal care and all of our experimental techniques.

Anti-Hyperlipidaemic activity of Dexamethasone–induced hyperlipidaemia in rats:

Using dexamethasone, a glucocorticoid, will increase hyperlipidaemia because it is known to cause a rise in plasma lipids. To induce hyperlipidaemia, Wistar rats were given dexamethasone (10 milligrams/kilogram /day, subcutaneous) for eight days. The rodents were divided into five groups, with six (n=6) Wistar rodents in each group.

Group-Normal control - Administered normal saline solution

Group-Hyperlipidemic control – Administered normal saline solution

Group- Standard group – Gemfibrozil 10 Milligram/kilogram/ day suspended in gum acacia in water

Group-Test group-I – Cinnamon Oil 25 Milligram/kilogram orally

Group-5 Test group- II-Cinnamon Oil 50 Milligram/kilogram orally

Hyperlipidemia was induced in all groups by subcutaneous administration of dexamethasone (10 milligrams/kilogram /day) for eight days. During this period, the hyperlipidemic control group received saline. Group III received gemfibrozil (10 Milligram/kilogram /day, i.p.) in gum acacia. Groups IV and V were treated orally with cinnamon oil at doses of 25 and 50 milligrams/kilogram/day, respectively. Following an overnight fast, animals were anesthetized with light ether, and plasma samples were taken for serum lipid profile investigation [2-3].

Myocardial toxicity induced by doxorubicin administration in rats:

Rodents: Albino rodents of both sexes. Weight: 150-200 grams

Group I (Control): Normal saline (5 mL/kg, i.p.).

Group II (Doxorubicin): Doxorubicin (2.5 Milligram/kilogram,i.p.), 6 alternate-day doses over 2 weeks (cumulative dose 15 Milligram/kilogram).

Group III (Cinnamon Oil 25): Cinnamon oil (25 milligrams/kilogram, p.o.) daily for 2 weeks, followed by vehicle for 2 weeks.

Group IV (Cinnamon Oil + Doxorubicin):

Cinnamon oil (50 milligrams/kilogram, p.o.) for two weeks (pretreatment), followed by doxorubicin as in Group II. In order to estimate the levels of cardiac biomarkers CPK and LDH, as well as total cholesterol, triglycerides, and LDL, blood samples were taken from the retro-orbital plexus under mild ether anesthesia using heparinized microcapillaries 36 hours after the previous therapy [9].

U/L = 9683 x DA 340nm/min; Where DA = Change in absorbance.

Biochemical Estimation of Blood Serum:
Plasma lipid levels were measured using Qualigens Diagnostics kits on a semi-automatic analyzer.

Statistical Analysis:
Data were analyzed with GraphPad Prism 5.0; results are presented as mean ± SEM. Group differences were assessed by ANOVA with Dunnett's post hoc test; $P < 0.05$ was considered significant.

RESULTS

The study evaluated the anti-hyperlipidemic effect of cinnamon oil at 25 and 50 milligrams/kilogram. Results are presented below.

Changes in total cholesterol and triglyceride levels due to hyperlipidemia caused by dexamethasone

Total cholesterol was elevated in the hyperlipidaemia-induced group (117.71 ± 1.33 milligrams/ deciliter) versus controls (64.43 ± 0.93 milligrams/ deciliter), indicating hypercholesterolemia. Cinnamon oil (25 and 50 milligrams/kilogram) reduced TC to 84.23 ± 1.05 milligrams/ deciliter ($P < 0.001$) and 82.35 ± 0.89 milligrams/ deciliter ($P < 0.0001$), respectively; gemfibrozil reduced TC to 73.70 ± 0.79 milligrams/ deciliter ($P < 0.001$). Triglycerides increased to 150.71 ± 0.52 mg/dL in the induced group versus 63.75 ± 0.51 mg/dL in controls. Cinnamon oil (25 and 50 milligrams/kilogram) lowered TG to 79.50 ± 0.53 milligrams/ deciliter ($P < 0.001$) and 75.25 ± 0.64 milligrams/ deciliter ($P < 0.0001$), respectively; gemfibrozil lowered TG to 68.33 ± 0.57 milligrams/ deciliter ($P < 0.001$).

Dexamethasone affected the levels of good cholesterol in the body:

HDL cholesterol (HDL-C) was reduced in the dexamethasone-induced group (24.75 ± 0.41 milligrams/ deciliter) vs controls (40.68 ± 0.71 milligrams/ deciliter). Cinnamon oil (25 and 50 milligrams/kilogram) yielded HDL-C of 25.79 ± 0.60 milligrams/kilogram ($P < 0.001$) and 28.40 ± 0.52 milligrams/ deciliter ($P < 0.0001$), respectively; gemfibrozil increased HDL-C to 34.50 ± 0.67 milligrams/ deciliter ($P < 0.001$).

Dexamethasone affected the levels of low levels containing lipoprotein, and very low levels containing lipoprotein in the body:

Versus controls (14.59 ± 0.50 Milligram/ deciliter). Cinnamon oil (25 and 50 Milligram/kilogram) reduced LDL-C to 33.67 ± 0.61 Milligram/ deciliter ($P < 0.001$) and 26.73 ± 0.55 Milligram/ deciliter ($P < 0.0001$), respectively; gemfibrozil reduced LDL-C to 23.35 ± 0.56 Milligram/ deciliter ($P < 0.001$). VLDL-C rose to 38.42 ± 0.65 Milligram/ deciliter in the induced group versus 13.42 ± 0.46 mg/dL in controls. Cinnamon oil (25 and 50 Milligram/kilogram) lowered VLDL-C to 30.60 ± 0.44 Milligram/ deciliter ($P < 0.001$) and 25.80 ± 0.51 Milligram/ deciliter ($P < 0.0001$), respectively; gemfibrozil lowered VLDL-C to 19.75 ± 0.53 Milligram/ deciliter ($P < 0.001$).

Atherogenic results:

The atherogenic index in the group of rats treated with dexamethasone for hyperlipidemia increased to 4.89 compared to the normal rat group's 1.58. Significantly lower values of 3.26 and 2.89, respectively, were seen in the group that was treated with Cinnamon Oil at dosages of 25 Milligram/kilogram and 50 Milligram/kilogram. The values of 2.13 in Table 1 have been significantly reduced with gemfibrozil.

Table 1. Impact of Cinnamon Oil on Hyperlipidemia Induced by Dexamethasone Injection

The atherogenic index increased to 4.89 in the dexamethasone-induced hyperlipidemia group versus 1.58 in controls. Cinnamon oil (25 and 50 Milligram/kilogram) reduced the index to 3.26 and 2.89, respectively; gemfibrozil reduced it to 2.13.

Groups	Preventive/dose	Total cholesterol	TG	Good Cholesterol	Low levels containing lipoprotein	Very low levels containing lipoprotein	Atherogenic results
I	Normal-group	64.43 ± 0.9	63.75 ± 0.711	40.68 ±0.79	14.59±0.495	13.42 ±0.45	1.58

II	Normal-control group	117.71 ± 1.3	150.71 ± 0.518	24.75 ±0.410	56.32 ±0.8	38.42 ±0.6	4.89
III	Standard group Gemfibrozil (10 Milligram/kilogram)	73.70 ± 0.79**	68.33 ± 0.572**	34.50 ±0.665**	23.35 ±0.56**	19.75 ±0.527**	2.13
IV	Test group-I Cinnamon Oil (25 Milligram/kilogram) –I	84.23 ± 1.04**	79.50 ± 0.526**	25.79 ±0.602**	33.67±0.60**	30.60 ±0.441**	3.26
V	Test group-II Cinnamon oil (50 Milligram/kilogram)	82.35 ± 0.8***	75.25 ±0.641***	28.40 ±0.517***	26.73 ±0.55***	25.80 ±0.50***	2.89

Data are presented as mean ± SEM. Statistical analysis was performed by one-way ANOVA with Dunnett's multiple comparisons; P < 0.05 was considered significant. ***P < 0.0001 versus control.

Doxorubicin induced myocardial infraction:

Chronic doxorubicin administration induced cardiotoxicity, evidenced by significant increases in cardiac biomarkers (CPK, LDH) and elevated cholesterol and triglycerides versus control (Group 1), with minimal change in HDL. Cinnamon oil treatment (Groups 3 and 4) significantly reduced cholesterol and triglycerides and increased HDL versus the doxorubicin group (Group 2), and attenuated CPK and LDH elevations.

Table.2 Cinnamon oils impact on serum TC, TG, HDL, LDH, LDL, and CK-MB levels in experimental and control rats

Groups	Preventions	Total cholesterol levels	Total cholesterol	Good Cholesterol	CK-MB	LDH	Low levels containing lipoprotein
1	Control	60.99±1.51***	106.01±1.01***	30.38±1.79*	19.99±0.67***	149.14±2.91***	28.92***
2	Doxorubicin induced (2.5 Milligram/kilogram)	107.06±2.21	297.81±1.53	22.15±1.03	34.61±1.33	205.04±2.31	72.47
3	Test group-I Cinnamon Oil (25 Milligram/kilogram –I	79.83±2.61***	183.30±1.67***	36.38±1.23^{ns}	26.83±1.16***	177.59±1.24***	46.93***
4	Test group-II Cinnamon oil (50 Milligram/kilogram)	72.44±1.40**	159.69±1.11***	35.9±3.41^{ns}	21.97±0.54^{ns}	165.34±1.82***	38.42***

Values are mean ± SEM (n = 6). Data were analyzed by one-way ANOVA with Dunnett's post hoc test. *P < 0.05, **P < 0.01, ***P < 0.001 versus Group II.

CONCLUSION

The present pharmacological study investigated cinnamon oil's (25 and 50 Milligram/kilogram) potential antihyperlipidemic and cardioprotective effects. Reduced levels of triglyceride cholesterol, Low levels containing lipoprotein, very low levels containing lipoprotein, and increased levels of HDL

are the results of cinnamon oil's anti-cholesterol effects. When given orally, cinnamon oil significantly reduces hyperlipidaemia in Wistar rats. The treatment of dyslipidaemia can thus make use of a medication derived from cinnamon oil. Acute hyperlipidaemia was induced in Wistar rats by administering 10 Milligram/kilogram intraperitoneally for Eight days. In Dexamethasone induced hyperlipidaemia models, the elevation of TC, total glyceride, high-density-containing lipid protein, low-thickness-containing lipid protein, and very-low-density-containing lipid protein was successfully prevented (Vasu et al., 2009; Yu Z et al., 2017). The presence of higher levels of marker enzymes, such as LDH and CK-MB, in rats demonstrating cardioprotective effects of doxorubicin was evident. Results indicated that both doses of cinnamon oil extract inhibited doxorubicin-induced CK-MB and LDH release in rat serum. Overall, the study's results indicate that, when it comes to hyperlipidaemia, 50 milligrams per kilogramme of cinnamon oil is more beneficial than 25 milligrams per kilogramme. Cinnamon oil has significant anti-hyperlipidaemic and cardioprotective potential, according to preliminary test findings (25 and 50 Milligram/kilogram, respectively).

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