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Original Research Article

A comparative study of hypolipidemic effect of atorvastatin and *Coriandrum sativum* in triton-induced hyperlipidemic albino rat model

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ABSTRACT

Background: Hyperlipidemia is the main factor responsible for coronary artery disease. Atorvastatin commonly used, as a competitive inhibitor of HMG-Co-A reductase, is known to reduce LDL and triglyceride levels and increase HDL levels. *Coriandrum sativum* (Coriander) belongs to the Apiaceae family and has hypolipidemic, antidiabetic, and antioxidant properties. Triton WR1339 has been used by several studies to induce hypercholesterolemia in animals which acts on both the synthesis and excretory phase. This study aimed to compare the hypolipidemic effect of *Coriandrum sativum* and atorvastatin in triton-induced hyperlipidemic rats.
Methods: Following approval from the Institutional Animal Ethics committee, 36 Wistar albino rats were divided into 6 groups of 6 animals each. Triton 200mg IP was administered to all the groups except Group 1 (control). Groups 3 and 4 received Coriander extract 300mg/kg and Atorvastatin 80mg/kg orally with Triton. Groups 5 and 6 received Coriander extract and Atorvastatin respectively 22 hours later. Lipid profile was estimated 24 hours before, 24 and 48 hours after administering Triton.
Results: In the synthesis phase, there is a statistically significant increase in the lipid levels in Groups 2, 5, and 6 compared to Groups 3 and, 4 indicating hypolipidemic effects of both Coriander extract and Atorvastatin. In the excretory phase, all groups have shown a decrease in lipid levels but a decrease in total cholesterol level in Group 5 compared to Group 3 was significant.
Conclusions: Atorvastatin and Coriander extract both affect the lipid levels in the synthesis and excretory phase. Even though coriander cannot replace atorvastatin in the management of hyperlipidemia, it can be used as an adjuvant to atorvastatin to reduce the cost effects and adverse reactions caused by allopathic medicines.

Keywords: Atorvastatin, *Coriander sativum*, Hyperlipidemia, Triton

INTRODUCTION

Hyperlipidemia is a significant societal burden, contributing to atherosclerosis and related conditions such as coronary heart disease (CHD), ischemic cerebrovascular disease, and peripheral vascular disease. It is a leading cause of death among middle-aged and older adults, accounting for one-third of all deaths in this demographic.[1] According to the World Health Organization (WHO), high blood cholesterol levels contribute to approximately 56% of cardiovascular disease cases worldwide, resulting in about 4.4 million deaths annually.[2]

Lipids are a class of fats and fat-like substances in the blood, including fatty acids, cholesterol, cholesterol esters, triglycerides, and phospholipids. Lipoproteins are macromolecular complexes composed of lipids and

proteins. There are five major types of lipoproteins: chylomicrons, Very Low-Density Lipoproteins (VLDL), Intermediate Density Lipoproteins (IDL), Low-Density Lipoproteins (LDL), and High-Density Lipoproteins (HDL).[2] Hyperlipidemia, or dyslipidemia, is a condition characterized by elevated levels of serum total cholesterol (TC), LDL, and VLDL, along with decreased levels of HDL.[3] Risk factors for hyperlipidemia include poor dietary habits, cigarette smoking, hypertension, type 2 diabetes mellitus, advancing age, and a family history of premature coronary heart disease (CHD) events in first-degree relatives (men <55 years; women <65 years).[1]

Coronary Artery Disease (CAD) is a serious complication of hyperlipidemia. The severity of CAD risk can be assessed using three ratios: Atherogenic Index of Plasma (AIP), Castelli Risk Index (CRI), and Atherogenic Coefficient (AC). These ratios are calculated as follows:[4]

Atherogenic Index of Plasma (AIP) = log TG/HDLc
Castelli's Risk Index (CRI-I) = TC/HDLc
Castelli's Risk Index (CRI-II) = LDLc/HDLc
Atherogenic Coefficient (AC) = (TC-HDLc)/HDLc

The Atherogenic Index of Plasma (AIP) is considered a strong predictor of atherosclerosis, which can lead to coronary artery disease. The term was proposed by Dobiasova and Frohlich. They found a positive correlation between AIP and the Fractional Esterification Rate of HDL (FERHDL) and an inverse correlation between AIP and LDL particle size. Since FERHDL predicts HDL and LDL particle sizes, which in turn indicate the risk of coronary artery disease, using triglycerides (TG) and HDL as AIP may help assess plasma atherogenicity.[5]

In addition to dietary and lifestyle modifications, pharmacological treatments for hyperlipidemia include statins (atorvastatin, lovastatin, simvastatin), bile acid sequestrants (colestipol, cholestyramine), fibric acid derivatives (fenofibrate, gemfibrozil), niacin, and ezetimibe. Statins have become the standard therapy for hyperlipidemia due to their effectiveness in reducing oxidative stress and vascular inflammation and increasing the stability of atherosclerotic lesions. Atorvastatin is the most effective and best-tolerated statin for treating hyperlipidemia. It is a competitive inhibitor of HMG-CoA reductase, a rate-limiting enzyme in cholesterol biosynthesis. Atorvastatin is administered at a dose of 10-80 mg/day, lowering LDL by 25-40% and increasing HDL by 5-10%. Common side effects include muscle aches and mild gastrointestinal issues, while serious but uncommon side effects include liver failure, myositis, and rhabdomyolysis.[1,6-8]

In India, Ayurveda and traditional literature highlight the use of herbs for enhancing organ function and treating various ailments. Many herbs have demonstrated hypolipidemic properties, in addition to their taste, flavor, color, and preservative qualities. Due to the high cost and potential side effects of pharmaceutical hypolipidemic drugs, researchers have explored herbal alternatives. The hypolipidemic effects of certain plants have been validated in both animal and human studies. Notable plants with lipid-lowering properties include *Allium cepa* (onion), *Trigonella foenum-graecum* (fenugreek), *Zingiber officinale* (ginger), *Momordica charantia* (bitter gourd), *Curcuma longa* (turmeric), *Coriandrum sativum* (coriander), and *Avena sativa* (oat).[9]

Coriandrum sativum, commonly known as coriander, belongs to the Apiaceae (Umbelliferae) family and is an annual, herbaceous plant native to the Mediterranean and Middle Eastern regions. All parts of the plant are used both as a flavoring agent and for treating various disorders. The leaves are rich in Vitamin A and Vitamin C, containing up to 12 mg/100g and 160 mg/100g, respectively. Coriander is composed of 84% water and is low in cholesterol and saturated fat. It is also a good source of thiamine, zinc, and dietary fibers. Additionally, coriander exhibits antioxidant, anti-anxiety, diuretic, and metal detoxification properties.[10,11]

Coriander is beneficial in treating a variety of conditions, including skin inflammation, high cholesterol levels, diarrhea, mouth ulcers, anemia, indigestion, menstrual disorders, smallpox, conjunctivitis, skin disorders, and blood sugar disorders. It also supports eye care. The hypolipidemic effects of coriander are attributed to its ability to enhance bile acid synthesis and promote the degradation of cholesterol into fecal bile acids and neutral sterols. Additionally, coriander reduces the levels of the enzyme HMG-CoA synthase, which helps inhibit cholesterol synthesis.[2]

Triton WR1339, also known as Tyloxapol, is a non-anionic polymeric detergent used in research to induce hypercholesterolemia. It functions by blocking the uptake of lipoproteins from the blood circulation by extrahepatic tissues, leading to elevated levels of circulatory lipoproteins. Triton-induced hyperlipidemia occurs in two phases: the synthesis phase and the excretory phase.

In the synthesis phase, lipid levels rise initially, reaching their peak within 24 hours after Triton administration. Following this peak, the excretory phase begins, during which lipid levels decrease and approach normal levels by the end of 48 hours.[12-14]

Studies have shown that coriander is effective in the biphasic model of Triton-induced hyperlipidemia in rats. At a dose of 1 g/kg body weight, coriander significantly reduced cholesterol and triglyceride levels. In research by Lal et al, coriander's hypolipidemic effects were compared with those of a polyherbal formulation called Liponil. Both coriander and Liponil demonstrated hypolipidemic activity in both the synthesis and excretory phases of Triton-induced hyperlipidemia in rats.[12] Thus, this study aims to evaluate the hypolipidemic effects of coriander and compare them with the standard drug Atorvastatin in Triton-induced hyperlipidemic rats.

METHODS

The study was conducted from 7 March 2016 to 10 March 2016 in the animal house of K S Hegde Medical Academy, Mangalore. A total of 36 albino rats were divided into 6 groups, with 6 animals in each group. They were housed in polypropylene cages and provided standard rodent chow and water ad libitum throughout the experiment. Body weight was recorded regularly. The animals were cared for in accordance with the "Guide for the Care and Use of Laboratory Animals," published by the National Academy of Sciences. The study was conducted following approval from the Institutional Animal Ethics Committee (Reg. No. 115/1999/CPCSEA). The experiment was conducted following the procedure outlined by Vogel and Vogel.[15] Lipid profiles, including total cholesterol, triglycerides, LDL, and HDL levels, were measured in all the rats 24 hours prior to the administration of Triton.

Commercially available ethanolic extract of *Coriandrum sativum* and Atorvastatin tablets were procured for the study. The coriander extract was diluted in gum acacia before oral administration, while the Atorvastatin tablets were powdered and dissolved in distilled water to create a fine suspension. Triton was administered to rats in Groups 2 to 6 following an 18-hour period of starvation. The groups are: Group 1: Basal diet only, Group 2: Triton 200mg/kg body weight intraperitoneally, Group 3: Coriander extract 300mg/kg body weight orally at the time of administration of Triton, Group 4: Atorvastatin 80mg/kg orally, at the time of administration of Triton, Group 5: Coriander extract 300mg/kg body weight Orally 22 hours after administration of Triton, and Group 6: Atorvastatin 80mg/kg orally, 22 hours after administration of Triton.

The lipid profile (total cholesterol, triglycerides, LDL, and HDL) was re-evaluated 24 and 48 hours after Triton administration to compare the values obtained during the synthesis and excretory phases, respectively. This comparison aimed to assess the effects of Coriander and Atorvastatin on lipid profiles in these two phases. Lipid profile tests were conducted using a Star 21 Plus semi-autoanalyzer in the Central Research Laboratory of the institution.

Procedure for blood withdrawal

Ketamine (0.1 to 0.2 ml) was administered intraperitoneally. Blood was collected by puncturing the retro-orbital plexus using a capillary tube after 5 min.

Analysis of the data

The values obtained were analyzed statistically using SPSS software (version 16.0) by performing the descriptive analysis, One-Way ANOVA and Post-hoc Tukey test.

RESULTS

As previously described, 36 rats were divided into 6 groups of 6 rats each, with an average body weight of 202.86±2.92 grams. The lipid profile was measured 24 hours before, 24 hours after, and 48 hours after Triton administration. Table 1 presents the means and standard deviations of Total Cholesterol (TC), High-Density Lipoproteins (HDL), Low-Density Lipoproteins (LDL), and Triglycerides (TG) during the synthesis phase. This evaluation includes Groups 1, 2, 3, and 4.

Table 1: The mean and standard deviation of TC, HDL, LDL and TG during the synthesis phase.

	Group	TC	HDL	LDL	TG
24 hours before triton	1	99.01±30.4	44.59±9.28	33.5±21.7	131.1±43
	2	72.47±12.4	54.75±12.9	20.5±8.4	59.46±12.9
	3	69.92±9.1	44.77±0.4	15.6±9.7	82.75±23.3
	4	64.92±7.17	47.08±5.2	8.85±4.7	94.28±5.2
	P value	*0.013	0.157	*0.042	*0.001
24 hours after triton	1	87.24±15.58	39.27±4.73	22.11±8.8	102.6±25.5
	2	119.26±23.4	39.99±3.17	33.5±7.7	155±14
	3	104.04±4.5	37.72±3.17	35.21±5.5	120.3±5.5
	4	125.8±17.6	37.26±5.19	51.5±15.2	135.6±26.1
	P value	*0.003	0.642	*0.002	*0.001
48 hours after triton	1	95.41±18.6	42.96±5.4	32.3±10.1	100.58±45
	2	108±18.6	43.97±2.37	41.4±12.9	112.7±11.1
	3	80.07±1.21	40.64±3.65	16.6±3.9	113.7±9.4
	4	88.26±2.48	42.07±4.63	24.34±5.27	106.7±7.17
	P value	*0.011	0.57	*0.002	0.77

*Level of significance: p value <0.05

According to the data in Table 1, 24 hours after Triton administration, there was an increase in Total Cholesterol (TC), LDL, and Triglycerides (TG) levels, and a decrease in HDL levels in Groups 2, 3, and 4. Statistical significance was observed for TC, LDL, and TG, with p-values of

0.003, 0.002, and 0.001, respectively. These results were obtained using one-way ANOVA.

Table 2 details the level of significance for TC, HDL, LDL, and TG within Groups 1, 2, 3, and 4 during the synthesis phase.

Triglyceride levels demonstrated statistical significance between the Triton group (Group 2) and the Triton + Coriander group (Group 3). Although there was an increase in Total Cholesterol (TC), LDL, and Triglycerides (TG), and a decrease in HDL, no statistical significance was found among the other groups for these parameters. These findings were confirmed using the Post Hoc Tukey test.

Table 2: The levels of significance (p values) within the four groups for TC, HDL, LDL and TG during the synthesis phase.

	Groups	TC	HDL	LDL	TG
Group 3	Group 1	0.332	0.916	0.198	0.432
	Group 2	0.415	0.782	0.993	*0.03
	Group 4	0.144	0.998	0.077	0.54

*Level of significance: p-value <0.05

Table 3 presents the means and standard deviations of TC, HDL, LDL, and TG during the excretory phase. Statistical comparisons were performed among Groups 1, 2, 5, and 6.

Table 3: The mean and standard deviation of TC, HDL, LDL and TG during the excretory phase.

	Group	TC	HDL	LDL	TG
24 hours before triton	1	99.01±30.47	44.59±9.2	33.5±21.7	131.1±43.5
	2	72.47±12.44	54.75±12.9	20.5±8.4	59.4±12.9
	5	61.99±2.71	54±11.31	8.75±4.9	97.9±21.43
	6	80.57±6.6	47.4±4.67	29.8±2.3	87.6±22.69
	p value	*0.008	0.25	*0.018	*0.002
24 hours after triton	1	87.24±15.58	39.27±4.73	22.11±8.8	102.6±25.5
	2	119.2±23.4	39.99±3.17	33.5±7.7	155±14
	5	126.9±28.1	33.69±3.75	48.81±10.8	120.5±25.05
	6	95.89±4.4	33.23±1.69	22.07±7.16	131.7±42.3
	p value	*0.008	*0.004	-	*0.034
48 hours after triton	1	95.41±18.6	42.96±5.4	32.3±10.1	100.5±45.63
	2	108±18.4	43.9±2.3	41.4±12.9	112.7±11.14
	5	94.97±3.6	38.6±1.46	34.5±2.4	108.8±24.5
	6	89.65±8.2	39.63±6.53	29.2±3.79	103.8±18.54
	p value	0.163	0.14	0.169	0.88

*Level of significance: p value <0.05

Table 4: The levels of significance (p values) within the four groups for TC, HDL, LDL and TG during the excretory phase.

	Groups	TC	HDL	LDL	TG
Group 5	Group 1	1	0.354	0.975	0.95
	Group 2	0.385	0.193	0.587	0.99
	Group 6	0.9	0.978	0.756	0.99

Level of significance: p value <0.05

According to Table 3, the means and standard deviations of Total Cholesterol (TC) and Triglycerides (TG) are reduced in the excretory phase compared to the synthesis phase, while HDL levels have increased. However, no statistical significance was observed for any of the parameters.

Table 4 shows the level of significance for TC, HDL, LDL, and TG among Groups 1, 2, 5, and 6 during the excretory phase.

Although there is a reduction in Total Cholesterol (TC) and Triglycerides (TG) and an increase in HDL levels, no statistical significance was found among the groups (as shown in Table 4).

Table 5 displays the atherogenic indexes for all groups during both the synthesis and excretory phases.

From the data presented in Table 5, it is observed that atherogenic indexes decrease during the excretory phase compared to the synthesis phase. The reduction in the Atherogenic Index of Plasma, Castelli's Risk Index-I, and Atherogenic Coefficient is statistically significant, as detailed in Table 6.

Table 5: The atherogenic indexes of all the groups during both synthesis and excretory phases.

Atherogenic indexes of all the groups during both synthesis and excretory phases							
	Group 1	**Group 2**	**Group 3**	**Group 4**	**Group 5**	**Group 6**	**Mean±SD**
Synthesis phase							
*AIP	0.416	0.587	0.5	0.56	0.55	0.597	0.535±0.67
$CRI-I	2.22	2.98	2.75	3.37	3.76	2.88	2.99±0.52
µCRI-II	0.56	0.83	0.93	1.38	1.48	0.66	0.97±0.37
¥AC	1.22	1.98	1.75	2.37	2.76	1.88	1.99±0.52
Excretory phase							
AIP	0.37	0.41	0.44	0.4	0.45	0.42	0.41±0.02
CRI-I	2.22	2.45	1.97	2.09	2.46	2.26	2.24±0.19
CRI-II	0.75	0.94	0.4	0.57	0.89	0.73	0.71±0.2
AC	1.22	1.45	0.97	1.09	1.46	1.26	1.24±0.19

*AIP: Atherogenic Index of Plasma, $CRI-I: Castelli's Risk Index- I, µCRI-II: Castelli's Risk Index- II, ¥AC: Atherogenic Coefficient

Table 6: The levels of significance for decrease in the atherogenic indexes between the two phases.

	P value
***AIP**	**0.003
$CSR-I	**0.008
µCSR-II	0.168
¥AC	**0.008

**Level of significance: p-value <0.05, *AIP: Atherogenic Index of Plasma, $CRI-I: Castelli's Risk Index-I, µCRI-II: Castelli's Risk Index-II, ¥AC: Atherogenic Coefficient

DISCUSSION

Hyperlipidemia is a significant risk factor for cardiovascular diseases. Historically, medicinal plants have been a major source of therapeutic agents for treating various human ailments. Numerous studies have been conducted to explore the hypolipidemic properties of different medicinal plants.[8,16] The present study was aimed at the screening of natural and chemical hypolipidemic drugs in the biphasic model of Triton-induced hyperlipidemia.

The results of this study indicate that both Atorvastatin and the coriander extract effectively reduced total cholesterol and triglycerides levels while increasing HDL, thus reversing triton-induced hyperlipidemia. The hypolipidemic effects of coriander and Atorvastatin were observed in both the synthesis and excretory phases. The decrease in triglyceride levels may be due to enhanced degradation of total cholesterol through increased lipoprotein lipase activity and decreased hepatic synthesis and secretion of triglycerides. The reduction in Total Cholesterol could be attributed to increased removal of LDL from plasma by boosting LDL receptor activity. These findings are consistent with conclusions drawn by Joshi et al and Lal et al.[12,17]

The atherogenic index is used to predict the risk of cardiovascular diseases. In our study, there was a statistically significant decrease in the Atherogenic Index from the synthesis phase to the excretory phase. This finding aligns with a study by Bharadwaj et al, which reported a reduction in the Atherogenic Index in groups treated for coronary artery disease compared to untreated groups.[1] Another study by Manal et al demonstrated a reduction in Atherogenic Indexes in groups treated with Coriander and Vitamin B6 compared to the positive control group.[18]

Thus, the present study confirms the hypolipidemic effect of *Coriandrum sativum* though not statistically significant when compared to the effect of Atorvastatin.

This study has few limitations. Though *Coriandrum sativum* has shown a decrease in the levels of cholesterol and triglycerides, it cannot be used as an exclusive therapy. However, it can be used as an adjuvant or as a dietary supplement.

CONCLUSION

Herbal medicines are crucial for developing therapeutic agents for various diseases. They offer an alternative to conventional medicine and are significant for pharmacological research and the development of new drugs. Additionally, herbal medicines are often cost-effective and generally have minimal side effects. Our study highlights the beneficial effects of *Coriandrum sativum* on hyperlipidemia. While it may not serve as a primary treatment, it shows promise as an effective adjunct to other hypolipidemic agents. Further research using different animal models beyond rats is needed to elucidate the mode of action and antiatherosclerotic properties of coriander. The promising results support the need for additional studies to explore the hypolipidemic effects of other herbal plants. Although the study was economical and brief, it's important to note that Triton-based animal models are only suitable for preliminary screening and do not fully replicate human hyperlipidemia.

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