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Preliminary phytochemicals and evaluation of the hypolipidemic effect of a saponin from *Asparagus officinalis* L. roots in hyperlipidemic rats

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Abstract

Hyperlipidemia, a condition of elevated lipids in the blood, is a major risk factor for atherosclerosis and subsequently cardiovascular disease (CVD), a leading cause of mortality worldwide. This study aimed to evaluate the hypolipidemic properties of the saponin extracted from the roots of *A. officinalis* (AOe) against Swiss albino Wistar rats. The root extract of *A. officinalis* was screened for its phytochemical investigation. Initial phytochemical analysis confirmed the presence of saponins. The root extract of *A. officinalis* underwent Soxhlet extraction with a solvent, followed by fractionation with n-butanol to isolate the saoponin-rich fraction. The separated fraction was orally administered to Triton-induced hyperlipidemic Wistar rats for 28 days at increasing doses of 100, 200, and 300 mg/kg body weight. At the end of treatment, serum lipid profiles, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL), were assessed and compared with those of a conventional hypolipidemic agent (atorvastatin). The present study found that the saponin-rich fraction therapy from *A. officinalis* significantly ($p<0.001$) attenuated the elevation of total cholesterol (TC), triglycerides, low-density lipoprotein (LDL) levels, and very low-density lipoprotein (VLDL), coupled with a concurrent improvement in high-density lipoprotein (HDL) cholesterol, demonstrating pronounced hypolipidemic efficacy. This study demonstrates the hypolipidemic efficacy of the saponin fraction from *A. officinalis* in rats, exhibiting a reduction in bad cholesterol and an increase in good cholesterol. These findings suggest it could serve as a viable alternative for managing hyperlipidemia.

Keywords: Hyperlipidemia, *A. officinalis,* Triton, Soxhlet, total cholesterol, hypolipidemic

1. INTRODUCTION

Hyperlipidemia is a major risk factor for the development of heart and coronary diseases and atherosclerosis [1, 2]. Hyperlipidemia is a major modifiable risk factor for cardiovascular diseases (CVDs), which remain the leading cause of death globally [3]. Cardiovascular diseases are associated with several cardio-metabolic risk factors, such as hypercholesterolemia, diabetes mellitus, hypertension, obesity, and physical inactivity [4]. Hyperlipidemia is known as an imbalance in blood cholesterol levels characterised by elevated low-density lipoprotein cholesterol and lower levels of high-density lipoprotein cholesterol (HDL-C). Types of hyperlipidemias include pure hypercholesterolemia, mixed hyperlipidemia, which is characterised by increased levels of both triglycerides and cholesterol, and hypertriglyceridemia [5]. Hyperlipidemia causes more than four million premature deaths per year [4]. It severely impacts health by precipitating cardiovascular disorders such as angina, myocardial infarction, hypertension, and heart failure [6]. Atherosclerosis of the coronary arteries is generally a disease of an arterial network called a dynamic and silent killer, described by the production of abrasions known as atherosclerotic plaques inside the walls of coronary arteries, which cuts down blood flow to the heart and is called CAD (coronary artery disease) [7]. Hyperlipidemia is divided into two types: primary hyperlipidemia (familial hyperlipidemia), which is caused by specific genetic problems. The other type of hyperlipidemia is secondary hyperlipidemia (acquired hyperlipidemia), which is caused by some disorders leading to abnormalities in plasma lipids and lipoprotein metabolism [6]. Dyslipidemia, or lipidemia, is a metabolic dysfunction identified by abnormalities in blood lipid concentrations, including elevated total cholesterol (TC) and triglyceride (TG) levels. These changes may encompass increased triglyceride levels, diminished high-density lipoprotein (HDL), or elevated low-density lipoprotein (LDL) [2]. Dyslipidemia originates from diverse etiologies, including deleterious lifestyle factors like obesity, sedentary lifestyles, and poor nutrition. It can also emanate from underlying pathologies such as diabetes mellitus or hypothyroidism, or possess a

hereditary component, exemplified by familial hypercholesterolemia. Given its frequently asymptomatic presentation during early stages, timely detection through systematic cholesterol screening is imperative to mitigate long-term sequelae, particularly adverse cardiovascular events [5, 8]. Despite progress in combating high cholesterol, its prevalence and associated risks continue to rise, underscoring the critical importance of treatment and prevention. Conventional therapies like statins, fibrates, and nicotinic acids, while effective, carry risks of long-term side effects, including hepatic and renal damage. This has accelerated the search for natural, cost-effective alternatives. Cholesterol-lowering plants offer a safer, holistic approach to managing dyslipidemia, potentially complementing traditional pharmacotherapy and promoting overall wellness [9].

Herbal medicine has a protective role in hyperlipidemia, with a high curative effect and fewer side-effect events [10]. Herbs play a potential role in controlling blood lipids and reducing the risk of cardiovascular events via excretory function enhancement, cardiovascular system improvement, and tonic effect reinforcement [11]. *A. officinalis* L., usually termed asparagus, is a perennial flowering plant esteemed for its medicinal and culinary uses. As a member of the Asparagaceae family, it is native to Europe, Northern Africa, and Western Asia. Esteemed for its subtle flavour and abundant nutritional benefits, asparagus has been grown as a vegetable for millennia [12]. Asparagus includes over 250 species, of which *A. officinalis* L. is the only cultivated species. However, several wild species are traditionally collected for consumption and medicinal purposes in the Mediterranean Basin, such as *A. acutifolius* L. and *A. horridus* L. (syn. *A. stipularis* Forssk) [13]. Asparagus is a diclinous plant with a yellowish-green male flower and a less conspicuous female flower on a separate plant. Asparagus can be simply classified into green asparagus, white asparagus, purple-green asparagus, purple-blue asparagus, and pink asparagus based on the colour differences [14]. The roots of *A. officinalis* and other asparagus species were recently reviewed for various pharmacological activities, such as aphrodisiac, anti-ageing, antiepileptic, antidepressant, antiparasitic, antimicrobial, anti-inflammatory, diuretic, and antioxidant attributes. The medicinal potential is ascribed to a varied phytochemical makeup, encompassing flavonoids, saponins, ascorbic acid, and amino acids such as asparagine [15]. The saponin content of the roots of *A. officinalis* has been extensively studied. In addition, the shoots of this plant have also been found to contain significant amounts of saponins, which are structurally similar to those found in the roots [15]. In this content, the present study aimed to evaluate the hypolipidemic activity of the saponin fraction from *A. officinalis* L. (AOe) on Wistar rats with hyperlipidemia induced by Triton.

2. MATERIAL AND METHODS

2.1 Chemicals

M/s. Sigma Chemical Company, St. Louis, MO, USA, provided the diethyl nitrosamine (DEN). Additional analytical-grade chemicals were procured for this investigation from Hi-media Laboratories located in Mumbai, India.

2.2 Collection and authentication of plant

The *A. officinalis* was collected from a local vegetable market of Prayagraj, Uttar Pradesh (India). The authenticated process was confirmed by Prof. Satya Narain from *Duthie* Herbarium, Botany Department, University of Allahabad (Prayagraj, India).

2.3 Extraction process

The *A. officinalis* was washed with distilled water three times and placed in the shade to dry at room temperature. A mechanical blender was employed to pulverise the desiccated plant. After sieving, the powdered substances were inserted into the thimble of filter paper. The filled thimble was placed into the primary chamber of the Soxhlet extractor, and 200 mL of petroleum ether was introduced into a round-bottom flask. The water bath is utilised to provide heat to the flask until the solvent reaches its boiling point. Subsequently, replicated this technique utilising ethanol [16]. The preparation of the solvent extract of *A. officinalis* is outlined in **Figure 1.**

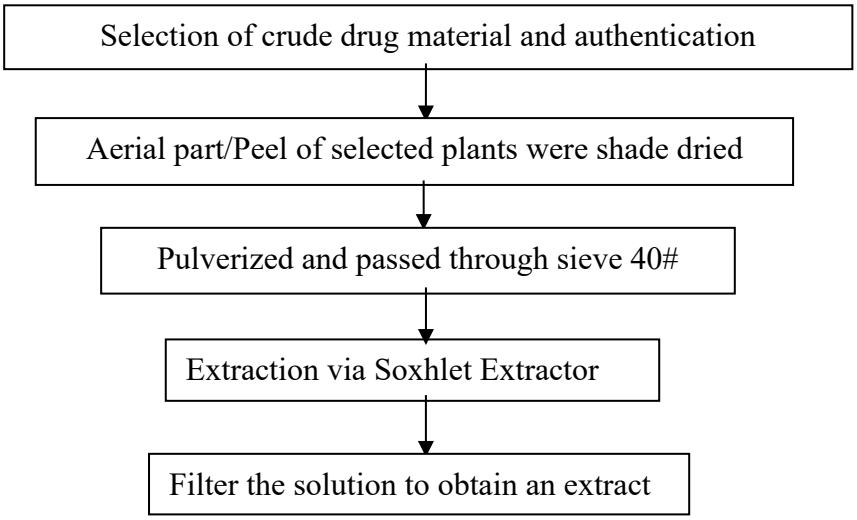

Figure 1: Schematic representation of the extraction process

2.4 Preliminary phytochemical analysis

The plant extracts were subjected to qualitative phytochemical screening for identification of various classes of active compounds using established methods [17, 18].

Test for alkaloids

Potassium mercuric iodide solution, or Mayer's reagent, was combined with 1 ml of extract. The precipitate's growth, which turned white or cream, demonstrated the presence of alkaloids in colour.

Test for glycosides

The plant extract was mixed with 1 ml of concentrated sulfuric acid and 1 ml of glacial acetic acid, which contains traces of ferric chloride. This resulted in a reddish-brown colour at the liquid-liquid junction, which is indicative of the existence of glycosides.

Test for flavonoids

After dissolving the extract in water, a few zinc fragments were added, along with 1 mL of concentrated HCl is added to the solution, and the solution turns reddish brown, indicating the presence.

Test for sterols

A few drops of chloroform were mixed with one gram of the extract. When two mL of strong sulfuric acid were added, a yellow ring formed, indicating that the extract contained sterols.

Test for saponins

1 ml of extract was combined with a few drops of sodium bicarbonate, and the mixture was thoroughly agitated. The presence of foam indicates a successful saponin test, which resembles a honeycomb.

Test for carbohydrates

1 mL of extract was combined with 1 mL of Fehling's Solution A and B (copper sulphate in alkaline conditions) and heated over a water bath. Carbohydrates are indicated by the formation of brick-red precipitates.

Test for Tannins and Phenolic Compounds

The presence of tannins and phenolic compounds in the extract is confirmed by the precipitate that forms when one ml of a 5% lead acetate solution is mixed with the aqueous extract.

Test for Proteins and Amino Acids

When 5-6 drops of Million's reagent mixture of nitrous acid and mercury nitrate are applied to two ml of the aqueous extract, the formation of red precipitate confirms the presence of proteins and free amino acids.

Test for coumarins

A test tube containing a small amount of material was covered with filter paper that had previously been soaked in a diluted NaOH solution. This test tube was submerged in water and brought to a boil. When the filter paper was eventually taken off and exposed to UV light, it produced green fluorescence, indicating the presence of coumarins.

2.5 Isolation of saponins (Liquid-Liquid Extraction)

Isolation of saponin from *A. officinalis* root extract was performed with slight modifications. In brief, the concentrated extract was dissolved in a small volume of distilled water. The reaction mixture was transferred to a separating funnel and extracted with n-butanol in multiple portions. Collect the *n*-butanol layer (upper layer containing saponins). Wash the *n*-butanol layer with a small amount of 5% NaCl solution to remove impurities. Evaporate the *n*-butanol under reduced pressure to obtain crude saponin extract [19].

2.6 In vivo Pharmacological activity

The saponin fraction from *A. officinalis* root extract was subjected to *in vivo* hypolipidemic evaluation in hypercholesterolemic Swiss albino rats.

2.6.1 Animal and ethics

The study utilized either sex of Swiss albino Wistar rats with an initial body weight ranging from 100 to 200 grams for the experiment. A minimum of three animals (n=4) were housed together per group in standard polypropylene cages equipped with suitable bedding. The animals were maintained in a controlled environment with a 12-hour light/dark cycle, a constant temperature of 22 ± 2°C, and a relative humidity of 50-60%. All animals underwent a minimum acclimatization period of seven days to this environment. Animals were maintained on a standard laboratory diet and water *ad libitum* [20].

2.6.2 *Induction of hyperlipidemia*

Hyperlipidemia was induced using Triton WR-1339, also known as Tyloxapol. The agent was administered via a single intraperitoneal injection at a dose of 400 mg per kg of body weight[21]. Blood samples were collected for analysis 18-24 hours after the injection to confirm the hyperlipidemic state. Hyperlipidemic conditions were created by monitoring blood lipid levels in Wistar rats with levels ranging from 107 to 207 mg/dL [22].

2.6.3 Experimental design

Group I: Normal control: fed with a normal diet and vehicle (0.5% saline)

Group II: Hyperlipidemic control: received Triton (i.p.)

Group III: Hyperlipidemic + AOe (100 mg/kg b.w., p.o)

Group IV: Hyperlipidemic + AOe (200 mg/kg b.w., p.o)

Group V: Hyperlipidemic + AOe (300 mg/kg b.w., p.o)

Group VI: Positive control: received atorvastatin (400 mg/kg b.w., p.o)

2.6.4 Measurement of lipid profile

After the end of the experiment, all the rats were subjected to overnight fasting and later euthanized under mild anesthesia. Serum from blood was collected and centrifuged at 10,000 rpm for 10 minutes. Serum total cholesterol (TC), triglyceride (TG), and HDL-C were estimated using standard diagnostic kits (Robonik) in a biochemical analyzer (Robonik India Pvt. Ltd.). LDL

cholesterol (LDL-C) and very LDL cholesterol (VLDL-C) were calculated according to Friedewald *et al.* (1972) [20].

VLDL=TG/5

LDL=TC (HDL + VLDL)

Atherogenic index (AI) was calculated using the formula:

AI = TC/HDL

2.7 Statistical Analysis

All data are expressed as the mean ± SEM, and statistical significance was determined using a one-way ANOVA followed by Dunnett's post-test, with a p-value of less than 0.05 considered significant.

3. RESULTS

3.1 Phytochemical profile studies

The phytochemical analysis of the alcoholic and aqueous extracts of plant samples demonstrated the presence of alkaloids, saponins, flavonoids, steroids/triterpenoids, phenols/tannins, glycosides, Carbohydrates, and proteins/amino acids. The result of preliminary phytochemical identification in *A. officinalis* is shown in **Table 1.**

Table 1: Phytochemistry of a fraction of *A. officinalis* roots.

Phytochemical Group	Result
Alkaloids	+
Saponins	++
Flavonoids	++
Steroids/Triterpenoids	++
Phenols/Tannins	++
Glycosides	+
Carbohydrates	++
Proteins/Amino Acids	++

weakly positive (+); positive (++)

3.2 Proximate analysis

The moisture content is found to be 7.2%, and the loss on drying at 110°C resulted in a total ash value of 5.12%, with acid-insoluble ash at 1.14%, acid-soluble ash at 13.95%, water-soluble ash at 15.43%, and sulphated ash at 1.62%, as shown in **Table 2.**

Table 2: Proximate analysis (moisture and ash content) of *A. officinalis*

Proximate analysis					
Moisture content	Total Ash content				
	Ash content	Acid-insoluble Ash	Acid-soluble Ash	Water-soluble ash	Sulphate Ash
7.2%	5.12 %	1.14 %	13.95%	15.43%	1.62%

3.3 Effect of saponin fraction from *A. officinalis* on serum lipid profiles

After the study, the Triton-induced hyperlipidemic rats treated with saponin fractioned from AOe root extracts exhibited a significant reduction and dose-dependent decrease in total cholesterol (TC) level. TC decreased from 122.20 ± 0.76 mg/dl to 88.56 ± 0.43 mg/dl, at a dose of 100 mg/kg, 78.18 ± 0.52 mg/dl at a dose of 200 mg/kg, and 74.23±0.36 mg/dl at a dose of 300 mg/kg, leading to a decrease of 27.52 %, 36.02% and 39.25% respectively compared to normolipidemic control **(Table 3)**. Triglycerides decreased from 171.40 ± 1.00 mg/dl to 117.90 ± 0.51 at a dose of 100 mg/kg, 102.20 ± 0.6 at a dose of 200 mg/kg, and 89.57 ± 0.43 at a dose of 300 mg/kg, leading to a decrease of 31.21%, 40.37% and 47.74% compared to normolipidemic control **(Table 3)**.LDL level decrease from 31.13 ± 0.51 mg/dL with the cholesterol control to 20.14±0.52 mg/dL or the rats treated with Atorvastatin (35.30%), and to 26.38 ± 0.36 mg/dl, 23.69±0.20 mg/dl and 21.35 ± 0.46 mg/dl, leading to decrease of 15.25%, 23.89% and 31.41% for the rats treated with saponin fraction from AOe at the doses of 100, 200 and 300 mg/kg, respectively, compared to hyperlipidemic group **(Table 3)**. VLDL level decreased from 34.28 ± 0.87 with the cholesterol control to 16.28 ± 0.84 mg/dL (35.30%) or the rats treated with Atorvastatin (52.50 %), and to 23.58 ± 0.61, 20.44 ± 0.92 and 17.91 ± 0.49 leading to decrease of 31.21%, 40.37 % and 47.74 % for the rats treated with saponin fraction from AOe at the doses of 100, 200 and 300 mg/kg, respectively, compared to hyperlipidemic group **(Table 3)**.

Moreover, the decreased level of HDL-C was significantly restored in the groups treated with saponin fraction AOe roots. After the treatment, Rats treated with Atorvastatin (standard drug) showed a significant increase in HDL level compared to the Hyperlipidemic (Cholesterol) group from 12.17 ± 0.44 to 23.51 ± 0.37, leading to an increase of 48 %. Treatment of AOe extract on rats showed a significant improvement and dose-dependent of HDL-C levels, compared to the Hyperlipidemic (cholesterol) group, from 17.46 ±0.30, 19.32 ± 0.15 and 21.60±0.24, leading to an increase of 30.29 %, 37 % and 43.65 %, respectively, at the doses of 100, 200 and 300 mg/kg compared to the cholesterol group **(Table 3)**.

The Athrogenic Index (AI) and LDL/HDL ratio for both the Cholesterol control and AOe administered group are presented in **Table 3,** and the graphs are presented in

Figure 2. The AI of hyperlipidemic groups was significantly increased from 8.70 ± 0.24 fold when compared to the normolipidemic group with 0.64 ± 0.4, demonstrating a statistically significant ($p < 0.001$). Treatment with the saponin fraction of AOe root decreased the AI significantly ($p < 0.001$) to 5.54 ± 0.20, 4.20 ± 0.10, and 3.23 ± 0.11, leading to a decrease of 36.32 %, 51.7 % and 62.87% at 100, 200, and 300 mg/kg b.w., respectively, when compared to hyperlipidemic rats. Similarly, AI remarkably declined ($p < 0.001$) to 2.21±0.19 (74 %) fold with atorvastatin.

Other cardiac risk factors, such as LDL/HDL cholesterol ratio, were markedly increased in significantly hyperlipidemic controls by 2.55-fold when compared to the normal group ($p < 0.001$). The administration of saponin fraction of AOe roots at 100, 200, and 300 mg/kg b.w., and atorvastatin decreased significantly to 1.51, 1.22, 0.98, and 0.85, respectively, when compared with the hyperlipidemic (Cholesterol group)

The findings from the *in vivo* study indicated that the mean effective doses of saponin fraction from AOe were examined at doses of 100-300 mg/kg b.w., with no recorded mortality or observed manifestations of toxicity after 28-day studies.

Table 3. Treatment groups showing the effect of various doses of AOe on cholesterol control

Groups	Total Cholesterol	Triglycerides	HDL	LDL	VLDL	AI	LDL/ HDL
Group 1	54.70±0.22	71.83±0.51	31.59±0.54	16.37±0.33	14.36±0.99	0.64±0.04	0.51
Group 2	122.20±0.76***	171.40±1.00***	12.17±0.44***	31.13±0.51***	34.28±0.87***	8.70±0.24***	2.55
Group 3	66.42±0.34***	81.44±0.56***	23.51±0.37***	20.14±0.52***	16.28±0.84***	2.21±0.19***	0.85
Group 4	88.56±0.43***	117.90±0.51***	17.46±0.30***	26.38±0.36***	23.58±0.61***	5.54±0.20***	1.51
Group 5	78.18±0.52***	102.20±0.61***	19.32±0.15***	23.69±0.20***	20.44±0.92***	4.20±0.10***	1.22
Group 6	74.23±0.36***	89.57±0.43***	21.60±0.24***	21.35±0.46***	17.91±0.49***	3.23±0.11***	0.98

Values are mean ± SEM, n=3.

Group 1, normal control; Group 2, Cholesterol Control (negative control); Group 3, Standard control (atorvastatin, 400 mg kg^{-1} b.w.); Group 4, AOe (100 mg kg^{-1} b.w.) and hyperlipidemic; Group 5, AOe (200 mg kg^{-1} b.w.) and hyperlipidemic; Group 6, AOe (300 mg kg^{-1} b.w.) and hyperlipidemic $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ compared to cholesterol groups.

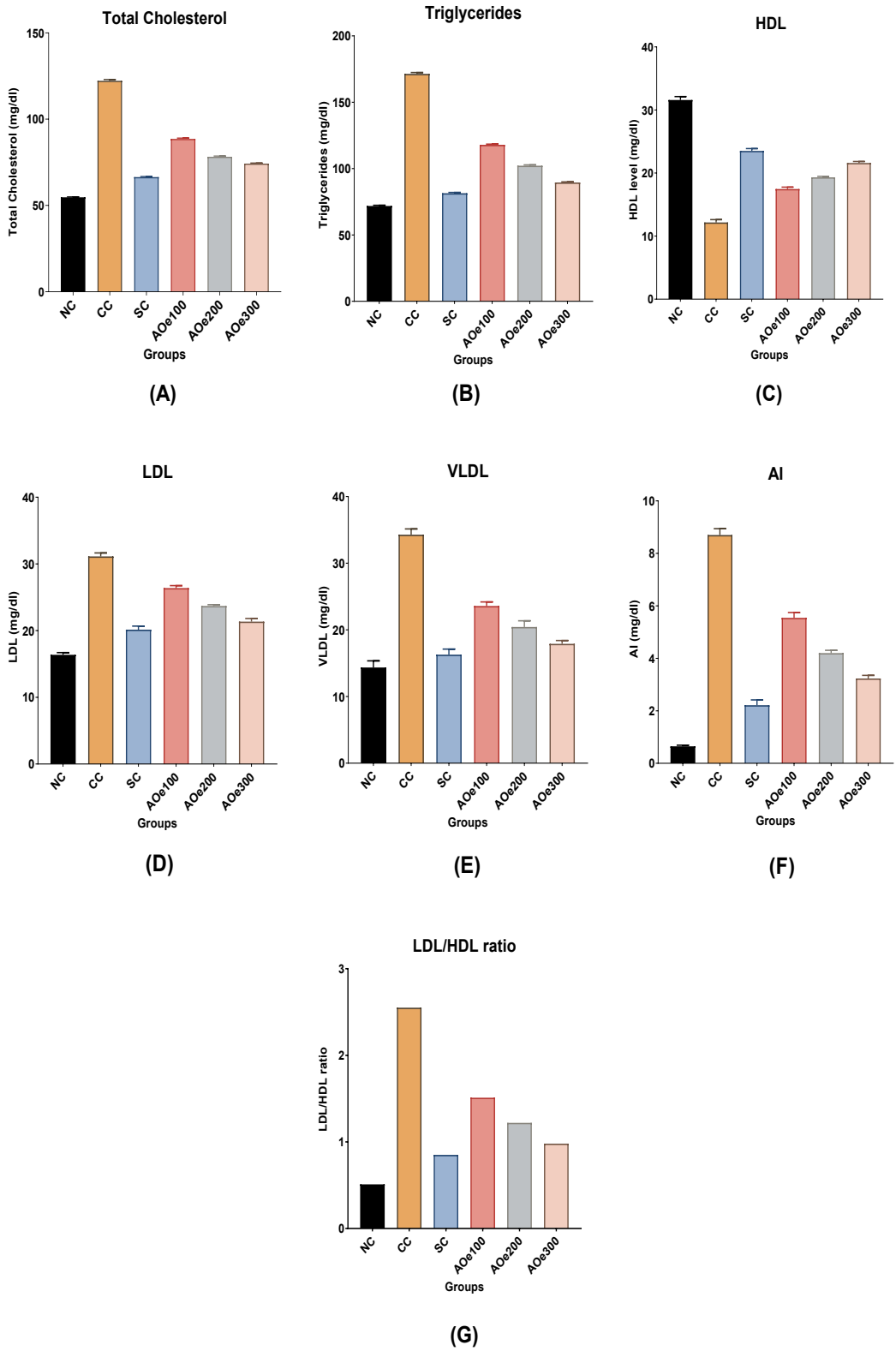

Figure 2: Total profile variation among the groups of test organisms after saponin fraction AOe root administration. (A) Total cholesterol, (B) Triglycerides, (C) HDL, (D) LDL, (E) VLDL, (F) AI, and (G) LDL/HDL ratio

4. DISCUSSION

Unhealthy lifestyles (high-fat diet, smoking, alcohol consumption, too little exercise, etc.) in the current society are prone to cause lipid metabolism disorders affecting the health of the organism and inducing the occurrence of diseases [21]. Total lipid profile, such as Cholesterol, triglycerides (TG), and high-density lipoproteins (HDL-C), is an essential component of the human biological system. Cholesterol is an unsaturated alcohol belonging to the steroid family, which maintains the normal physiological functions of animal cells, and is a necessary element of cell membrane activity. Cholesterol is important for the synthesis of the adrenaline hormone and gonadal steroidal hormones. Triglycerides (TG) are fatty acid esters of glycerol, which act as fat depots in humans [22].

This study aimed to evaluate the hypolipidemic activities of the saponin fraction from *A. officinalis* L. root related to both human health conditions and interest in the development of new drugs. In our experiment, Triton induced hyperlipidemic animals treated with the saponin fraction from *A. officinalis* root extract. Administration of Triton in normolipidemic rats induces hyperlipidemia, resulting in the elevation of cholesterol and triglyceride, LDL and VLDL, and concurrently decreases HDL compared with the untreated rats of the normal control.

The current study revealed that the biphasic character of Triton-induced hyperlipidemia in rats is a suitable model for understanding the mechanism of hypolipidemic drugs from natural products. Generally, lipid-lowering drugs interfere with the lipid biosynthesis pathway, lipid excretion, and the lipid metabolism pathway of the excretory phase [23].

Currently available antihyperlipidemia drugs, such as statins, inhibit the HMG-CoA reductase enzyme, which is responsible for the synthesis of cholesterol in the liver. Atorvastatin competitively inhibits the catalytic activity of 3-OH-3-methylglutarylcoenzyme A (HMG-CoA) to a mevalonate compound, which is the rate-limiting step in hepatic cholesterol synthesis. Inhibition of the HMG-CoA reductase leads to decreases in the synthesis of cholesterol in the liver, and decreases the LDL-cholesterol in the blood circulation [24].

In this context, treatment with saponin fraction from AOe roots (100 mg/kg, 200 mg/kg and 300 mg/kg) in Triton (400 mg/kg b.w.) induced lipidemic rats exhibited a significant reduction in cholesterol, triglycerides, VLDL, LDL and an increase in HDL, compared with the cholesterol group (hyperlipidemic control) **(Figure 2)**. The atherogenic protection percentage of the saponin fraction AOe treated groups (100 mg/ kg, 200 mg/kg and 300 mg/kg) were detected as a dose dependent protective effect, and standard drug atorvastatin (400 mg/kg) treated group was found effective, when compared to cholesterol group **(Figure 2)**, which could confirm that saponin fraction at the 100mg/kg, 200 mg/kg and 300 mg/kg of dose had a significant protective effect against hyperlipidemic activity in rats.

The ratios of atherogenicity/LDL of dyslipidemic rats treated with the extract of saponin fraction *A. officinalis* L. roots were significantly reduced. These results reflect a lipidic profile antiatherogenic, and suggest a protective effect of the extract with respect to the hypercholesterolemia induced by the mode enriched out of cholesterol [25].

Saponins are compounds composed of lipophilic aglycones linked to hydrophilic sugars. Natural saponins are isolated from plants and some Marine organisms. As important cholesterol-lowering drugs, natural saponins have attracted wide attention for their therapeutic potential in a variety of cholesterol-related metabolic diseases [26]. Due to the amphiphilic nature of the saponin itself, it is generally an amorphous white powder, but also exists as hederagenin, which is similar to needle-like crystals, primarily bitter, hygroscopic, and a natural surfactant, and mixes with water to produce a stable foam that is commonly used in cleaning products [21]. Saponins are highly active plant compounds reported to increase faecal cholesterol excretion and also increase the lipoprotein lipase activity (LPL), which helps in faster removal of free fatty acid from circulation, consequently decreasing total cholesterol [27]. Several possible mechanisms of saponins-mediated hypolipidemic action are potentially mediated by the AMP-activated protein kinase (AMPK) signalling pathway and related transcriptional regulators such as peroxisome-proliferator-activated receptors (PPAR), CCAAT/enhancer-binding proteins (C/EBP), sterol-regulatory element binding proteins (SREBP), and adipokines such as adiponectin [21].

These data were corroborated with earlier observations on Zhu *et al.* (2011), which revealed that treatment of rats with n-butanol extract (BEA) from asparagus by-products in mice fed a high-fat diet (HFD), significantly decreased the levels of body weight gain, serum total cholesterol and low density lipoprotein cholesterol, it dramatically increased the high density lipoprotein level when administered at three different doses (40, 80 or 160 mg/kg body weight) for 8 weeks in hyperlipidemic mice [16]. In another study, García *et al*. (2011) observed that freeze-dried asparagus at 125, 250, and 500 mg/Kg of body weight/day was able to significantly reduce total cholesterol, LDL cholesterol levels, and Atherogenic index (AI) in a dose-dependent manner [28].

In light of earlier reports, it is presumed from the present findings that the hypolipidemic exhibited by *A. officinalis* L. could be due to the presence of the saponins. Further, the synergistic action of its metabolite production may be more plausible for the beneficial effects exerted, exhibiting hypolipidemic activity. These findings may offer useful insight into the treatment of diseases such as atherosclerosis.

CONCLUSION

The saponin fraction of *A. officinalis* was found to possess potent antihypercholesterolemic and hypotriglyceridemic activities. Administration of atorvastatin resulted in significant decreases in LDL-C and triglycerides, alongside an increase in HDL-C. Additionally, its diuretic effect provides a mechanistic rationale for its application in atherosclerosis management. Further toxicological evaluation is required to ensure safe use.

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