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Review Article

A Comprehensive Review on Medicinal Herbal Plant with Potential Hypolipidemic Activity

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ABSTRACT

Hyperlipidaemia, characterized by elevated blood lipid levels, is a significant risk factor for cardiovascular diseases. While traditional pharmacological treatments are available, their effectiveness and potential side effects highlight the need to explore alternative approaches. Herbal plants have attracted attention due to their variety of bioactive compounds with therapeutic potential. This review aims to provide an overview of the lipid-lowering effects of several herbal plants, discussing their mechanisms of action and available clinical evidence. Notable herbs such as garlic (Allium sativum), ginger (Zingiber officinale) and green tea (Camellia sinensis) have shown potential in reducing lipid levels by modulating enzymes involved in lipid metabolism, inhibiting cholesterol absorption and enhancing lipid excretion. The findings suggest that numerous phytochemicals may offer therapeutic benefits for managing hyperlipidaemia. In addition, an analysis of 83 compounds indicates that 60 adhere to Lipinski's Rule of Five and Veber's Rule, suggesting that these compounds possess optimal physicochemical properties, which could make them viable candidates for future treatments of hyperlipidaemia.

Keywords: Herbal Drug, Herbal Medicine, Hyperlipidaemia Activity, Lipid Herb Drug Interaction, Lipinski's Rule, Physicochemical Properties, Veber's Rule.

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INTRODUCTION

Herbal medicine, also referred to as botanical medicine, is widely practiced across the globe as a common form of treatment. Plant-based products serve purposes beyond food and nutrition; they play a crucial role in the treatment of various disease.[1] Different parts of plants-such as flowers, fruits, seeds, leaves, berries, bark and roots-are utilized in herbal remedies.[2] It is estimated that approximately 80% of the global population relies on herbal medicine for their primary healthcare needs.[3] Herbs are considered a vital part of the diet for many people worldwide. Over time, scientific research in this field has expanded rapidly,[4] with studies showing that about 75% of herbal medicines are derived from research on traditional medicinal plants, while 25% of pharmaceutical drugs are sourced from higher plants.[5]

Since the dawn of human civilization, plants have been one of the most essential sources of medicine.[6] For instance, Mahung, a traditional Chinese medicine, has been used for over 5,000 years to treat various fevers and respiratory conditions. The Cinchona tree was used in Peru as early as 1825, primarily for the treatment of malaria.[7] Despite significant advancements in synthetic medicines and antibiotics, plants continue to be an important resource for both modern and traditional medicine worldwide, with one-third of the global population relying on traditional remedies. Some of the compounds that are commonplace in modern medicine were initially derived from plants in the 19th century. Notable examples include morphine (1803), quinine (1812), atropine (1831), papaverine (1848), cocaine (1860), digoxigenin (1865) and pilocarpine (1875).

Hyperlipidaemia is considered one of the leading risk factors for coronary heart disease, contributing significantly to its prevalence and severity. The incidence of hyperlipidaemia increases with age, with the ratio of affected women to men being 40:37.[8] This condition directly correlates with an increased risk of vascular diseases, including Myocardial Infarction (MI) and

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Cerebrovascular Accidents (CVA). inchona, a plant historically used in Peru since 1825, was primarily utilized for the treatment of malaria. Despite significant advancements in synthetic medicines and antibiotics in the 21st century, plants remain an essential source of medicinal compounds in both modern and traditional medicine across the globe. It is estimated that one-third of the world's population continues to use traditional remedies for self-medication.[9] Many compounds that are now standard in modern pharmaceuticals were originally derived from plants in the 19th century. Notable examples include morphine (1803), quinine (1812), atropine (1831), papaverine (1848), cocaine (1860), digoxigenin (1865) and pilocarpine (1875).[9]

Hyperlipidaemia, a condition characterized by elevated lipid levels in the blood, is a significant risk factor for coronary heart disease and contributes to its growing prevalence and severity. The incidence of hyperlipidaemia increases with age, with a ratio of 40:37 in women to men. This condition directly correlates with an elevated risk of vascular diseases such as Myocardial Infarction (MI) and Cerebrovascular Accidents (CVA). Globally, the prevalence of hyperlipidaemia is estimated at 39%, with higher rates in developed countries (51%) and lower rates in developing nations (26%).

The disorder is characterized by high levels of one or more lipoproteins, with high LDL (Low-Density Lipoprotein) levels being particularly concerning. Elevated LDL levels are associated with an increased risk of atherosclerosis, heart disease and stroke. Conversely, high HDL (High-Density Lipoprotein) levels are considered protective because HDL helps transport cholesterol back to the liver, reducing the risk of cardiovascular conditions. Due to their effects, LDL is often termed "bad cholesterol," while HDL is referred to as "good cholesterol." Extensive research over the past three decades has shown a strong link between low HDL and high LDL levels in the development of coronary artery disease.[10]

Cholesterol

All foods originating from animals contain cholesterol, an odourless, white, wax like matter, available in all food originated from animal but is absent from foods originating from plants.[11] Some cholesterol is said to be healthy. It is always in systemic circulation, where every cell in the body can use it. For instance, cholesterol is used by the liver to produce bile acids and by lymphocytes, adrenal cortex cells, muscle cells and kidney cells to generate cell membranes and steroid hormones.[11] Hypercholesterolemia is the term used to describe an elevated level of cholesterol in the blood.[12]

Lipids

A whole class of fats and molecules that resemble fats in the blood are referred to as lipids.[13] The blood's most significant lipids are TGLs, phospholipids, cholesterol, cholesterol esters and fatty acids. Esters of a long-chain monobasic organic acid that are produced from lipids by hydrolysis are known as fatty acids. Phospholipids (PL) resemble TGs but contain phosphate and a nitrogenous base in place of one fatty acid residue.[14]

Lipoprotein

These are hydrophilic plasma lipid-carrying macromolecular complexes. These are spherical particles composed of hundreds of molecules of proteins and lipids. Cholesterol, TGs and phospholipids are the main lipids that make up lipoproteins. There are five main lipoproteins, each of which has a distinct purpose: chylomicrons, VLDLs, IDLs, LDLs and HDLs.[15]

Soluble lipoproteins are classified into five groups, which are primarily identified by the ratios of cholesterol and triglycerides they contain.

1. Chylomicrons Cytoplasmic lipoproteins are incredibly big. They include 5% cholesterol and 90% triglycerides. Eight to twelve hours after eating, they appear in the bloodstream after being absorbed into the lymphatic system from the GI tract.
2. Very Low-Density Lipoproteins (VLDLs): Triglycerides make up 60% of very-low-density lipoproteins (VLDLs) and cholesterol accounts for 12%. The liver produces fatty acids, which are then carried by VLDLs as triglycerides and stored in adipose tissues. Muscle-rich LDLs and other cells use fatty acids as fuel.[16]
3. Low Density Lipoproteins: Triglycerides make up 6% of Low-Density Lipoproteins (LDLs), which include 65% cholesterol.[17]
4. HDL or high-density lipoproteins: include 50% protein, 25% cholesterol and 5% triglycerides. They prevent cells from absorbing LDLs and extract cholesterol so that it can be returned to the liver for further processing or elimination.[18]
5. Intermediate Density Lipoproteins (IDLs): As TGs are extracted from VLDLs, IDLs are produced. Either the liver directly absorbs the IDLs or they are converted to LDLs. After interacting with LDL to produce a complex that is endocytosed by the cell, IDLs are taken up by the liver.[19]

Normal Dietary Lipid Metabolism in Circulation

Lipid metabolism involves various lipoproteins in both the synthesis (anabolism) and breakdown (catabolism) of lipids (Figures 1 and 2). The primary dietary lipids include Triglycerides (TG), phospholipids and Cholesterol Esters (CE). These lipids, especially TG, are broken down by pancreatic lipases in the intestine. Once hydrolyzed, they are absorbed by the intestinal mucosal cells and transported into the mesenteric lymphatic

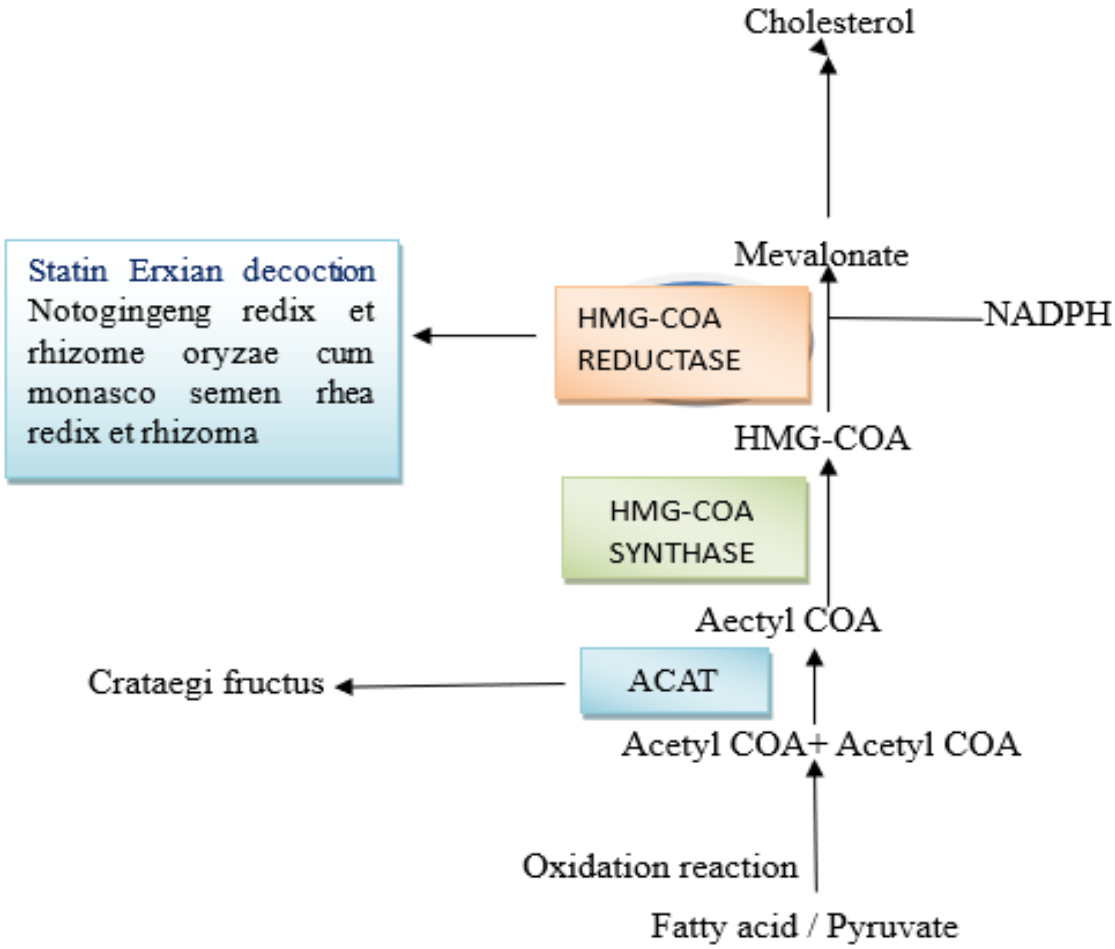


Figure 1: A simplified overview of the Mevalonate pathway involved in cholesterol synthesis. Potential therapeutic interventions, including both conventional drugs and Traditional Chinese Medicines (TCMs), are highlighted. Dotted arrows represent pathways that are bypassed or not shown in this diagram.

vessels as chylomicrons, which contain apoB-48.[20] The TG and CE in chylomicrons are then further hydrolyzed by Lipoprotein Lipase (LPL), resulting in the formation of chylomicron remnants. These remnants are cleared by LDL Receptors (LDLR) and LDLR-related proteins, which direct them to the liver.

In the liver, Very Low-Density Lipoproteins (VLDL) are synthesized, containing apoB-100, apoC-II and apoE. These proteins interact with enzymes or receptors that facilitate the transfer of lipids to various tissues, including arteries, for either storage or further metabolism.[21] ApoB-100 is the key protein that enables the liver to take up LDL. Triglycerides in VLDL are hydrolyzed by LPL, transforming the VLDL into Intermediate-Density Lipoprotein (IDL), which is further converted to LDL. The resulting LDL is then recycled back to the liver or transported to peripheral tissues for additional use.[22]

Diseases Associated with Hyperlipidaemia

An excess of Low-Density Lipoprotein (LDL) cholesterol can lead to atherosclerosis, a condition in which plaque builds up on the walls of the coronary arteries, narrowing them. This can result in the formation of blood clots on the plaque's surface, further obstructing blood flow to the heart, potentially causing a myocardial infarction (heart attack). Symptoms may include dyspnoea (shortness of breath), angina (chest pain), or general discomfort. One of the most severe complications of Coronary Artery Disease (CAD) is a heart attack, which occurs when a blocked coronary artery causes damage to the heart muscle. CAD can also lead to cardiac arrest, with individuals who have significant narrowing of two or more major arteries due to atherosclerosis accounting for 90% of sudden cardiac deaths. Obesity can indirectly increase the risk of CAD through its association with insulin resistance, hyperlipidaemia and hypertension (Tables 1 and 2).[23]

Ginger

Bhandari *et al.* assessed the lipid-lowering and antioxidant properties of an ethanolic extract of adraka, or ginger, *Zingiber officinale* Rosc. in rats with Streptozotocin (STZ)-induced diabetes. In diabetic rats, oral administration of 200 mg/kg of ethanolic ginger extract for 20 days resulted in a significant ($p<0.01$) reduction in hyperglycaemia. Furthermore, as compared to pathogenic diabetic rats, the extract therapy

Table 1: The average percentage of Americans aged 20 and older with abnormal blood lipid levels.

Blood lipid serum	Total blood cholesterol level		LDL-C concentration	HDL-C level
Abnormal level (mg/dL)	≥200	≥240	≥130	≤40
Estimated percentage of Americans*	~42.4% men	~12.8% men	~34.4% men	~29.3% men
	~12.8% women	~13.6% women	~30.3% women	~12.6% women

*Individuals aged 20 years or older from non-Hispanic white, non-Hispanic black and Mexican-American backgrounds are all classified as Americans.

Table 2: An overview of the antihyperlipidemic properties of certain herbal plants and medicines.

Sl. No.	Herbal Medicines	Sources	Active constituent	Biological activity
1	Ginger	Dried Rhizome of the *Zingiber officinale,* family: Zingiberaceae.	1 to 2% volatile oil (gingerol).	Decreasing serum LDL-C and increasing HDL-C.
2	Indian Blackberry	Seed, leaf, stem and bark of *Syzygium cumini*, family: Myrtaceae.	Jambolin, jambosine and ellagic acid.	Lowering triglyceride and LDL level.
3	Apamarga	Whole plant of *Achyranthus aspera,* family: Amaranthaceae.	AchyranthineAchyranthol.	Decreases serum cholesterol, triglycerides and total lipids.
4	Arjuna	Bark of *Terminalia arjuna,* family: Combretaceae.	Arjunolic acid	Significant hypocholesterolaemic effect.
5	Celery	Whole plant of *Apium graveolens*, family: Apiaceae	Phenolic acid (p-coumaric acid and ferulic acid) Flavonoids (apigenin, luteolin and kaempferol).	Decreasing serum triglycerides, total cholesterol, LDL-C and hepatic triglyceride.
6	Garlic	Bulb, stems and leaves of *Allium sativum*.	Allicin, PCSO and MCSO.	Hypocholesterolaemic effect and suppression of LDL.
7	Dandelions	Root of *Taraxacum officinale* L.Family: Asteraceae.	Lutein, esculin, flavonoids, phenolic acids.	Decreasing serum triglycerides, total cholesterol, LDL-C and increasing HDL-C.
8	Green tea	Leaves of *Camellia sinensis*. Family: Theaceae.	Polyphenols 37%, caffeine 3.5%, Theaflavin, epicatechin.	Suppresses adiposity and affects the expression of lipid metabolism genes.
9	Ginseng	Dried root of*Panax ginseng.*	Saponins, AcidicPolysaccharides, PhenolicExtract.	Decreasing serum triglycerides, total cholesterol, LDL-C and increasing HDL-C.
10	Flaxseed	Dried or ripe seed of *Linum usitatissimum.*	Linseed oil	Decreases LDL-cholesterol concentration.

enhanced the levels of HDL cholesterol and decreased the levels of triglycerides and total cholesterol in the serum (p<0.01).[24] In comparison to normal healthy control rats, STZ therapy also resulted in a statistically significant rise (p<0.01) in the levels of lipid peroxide in the liver and pancreas. In comparison to pathogenic diabetic rats, ginger extract treatment reduced the Thiobarbituric Acid Reactive Substances (TBARS) values in the liver and pancreas (p<0.01). The test drug's outcomes were similar to those of Gliclazide (25 mg/kg, orally), a common antihyperglycemic medication. The findings suggest that the tissues can be shielded from lipid peroxidation by an ethanolic ginger extract. In diabetic rats, the extract significantly lowers cholesterol levels as well. This was the initial pilot study to evaluate *Z. officinale's* potential in diabetic dyslipidaemia. There are no known allergic reactions to any form of ginger exposure, if taken at recommended doses.[25]

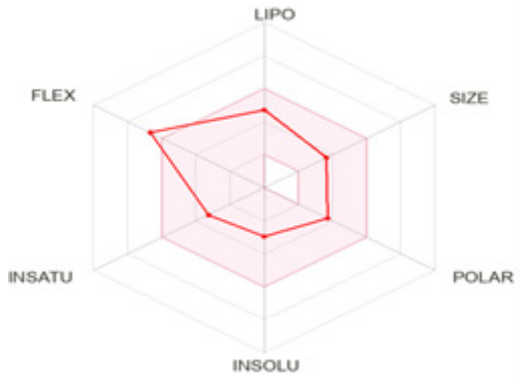


Figure 2: Graphical representation of an optimum range of important physicochemical properties related parameters such as- LIPO; Lipophilicity, POLAR; Polarity, INSOLU; Insolubility, INSATU; Instauration, FLEX; Flexibility.[52]

Indian Blackberry

Seed, leaf, stem and bark of *Syzygium cumini* from family Myrtaceae is responsible for lowering of triglyceride and LDL level. It cuts down on the production of endogenous lipids like Total Cholesterol (TC) and Triglycerides (TG). When overnourished, the human body may produce TC by manufacturing fatty acids.[26] The cytoplasm of the liver, kidney, brain, lung, breast, adipose tissue, etc. was where fatty acids were generated. Liver had the highest rate of fatty acid synthesis, with an ability to synthesise fatty acids that was eight to nine times greater than that of adipose tissue. The liver was where TG was mostly produced. TG and TC were synthesized using acetyl-CoA and Nicotinamide Adenine Dinucleotide Phosphate (NADPH).[27] The primary source of acetyl-CoA was the aerobic oxidation of sugar, while the catalytic process between the cytoplasmic isocitrate dehydrogenase and the pentose phosphate pathway produced NADPH. Several enzymes were implicated in the pathway.[28]

Apamarga

Whole plant of *Achyranthes aspera*, from family Amaranthaceae is responsible for decrease in serum cholesterol, triglycerides and total lipids concentration. In rats with hyperlipidaemia brought on by triton, the alcoholic extract of *Achyranthes aspera* Linn. at a dose of 100 mg/kg reduced serum levels of TC, TG, PL and TL by 60, 51, 33 and 53%, respectively. After giving this medication to normal rats at the same doses for 30 days, the treatment significantly reduced the levels of hepatic lipids and decreased serum TC, PL, TG and TL by 56, 62, 68 and 67%, respectively.[29] Cholic acid and deoxycholic acid excreted in the faeces rose by 24 and 40%, respectively. The quick excretion of bile acids, which results in minimal cholesterol absorption, could be the mechanism of action for *A. aspera*'s potential cholesterol-lowering activity.[30]

Arjuna

105 consecutive patients with Coronary Heart Disease (CHD) who presented to their centre were recruited and using a Latin-square design, the patients were divided into three groups of 35 each. Age, dietary and lifestyle factors, clinical diagnosis and drug treatment status were all matched between the groups. Not a single patient was taking medication to decrease cholesterol.[31] Before the trial started, participants stopped taking supplements of vitamins for a month and everyone received the American Heart Association Step II dietary guidelines. TBARS was used to quantify total cholesterol, triglycerides, HDL and LDL cholesterol and lipid peroxide at baseline. Group II received 400 units of vitamin E per day in capsule form; Group III received 500 mg of finely ground *T. arjuna* tree bark powder daily in capsule form; Group I received placebo capsules. At the 30-day follow-up, lipid and lipid peroxide levels were measured.[32] The response rate ranged from 86 to 91% across different categories. Groups I and II did not exhibit any significant changes in total, HDL, LDL, or triglyceride levels (paired t-test $p>0.05$). Total cholesterol decreased by 9.7+/-12.7% and LDL cholesterol decreased by 15.8+/-25.6% in Group III (paired t-test $p<0.01$). In both treatment groups, there was a significant decrease in lipid peroxide levels ($p<0.01$). Compared to the *T. arjuna* group (-29.3+/-18.9%), the Vitamin E group experienced a greater drop (-36.4+/-17.7%). Consequently, it can be said that the powdered bark of the *T. arjuna* tree exhibits strong antioxidant properties like those of vitamin E. Furthermore, it significantly lowers cholesterol levels.[33]

Celery

One of the annual or perennial medicinal plants that grow in tropical and subtropical areas of Europe, Africa and Asia is celery (*Apium graveolens* L.), a member of the Apiaceae family. The entire plant, seeds and essential oils of celery are frequently employed in food and medicinal. It was also long used in Unani and Ayurvedic treatment.[34] The majority of celery's phytochemicals were phenols such flavonoids, alkaloids and steroids, as well as sugars. The most utilized plant in traditional medicine was celery, owing in part to its phenolic acids and flavonoids. The principal flavonoids are luteolin, kaempferol and apigenin; the principal phenolic acids are caffeic acid, p-coumaric acid and ferulic acid.[35] Research conducted by Al Sa'aidi *et al.* demonstrated that by enhancing lipid peroxidation and its anti-oxidation properties, celery seed (*Apium graveolens*) n-butanol extract could decrease the hypercholesterolemic synthesis of endogenous lipids like TG and TC. A different study also demonstrated that diabetic rats treated with celery n-butanol water extract had increased activity levels of Glutathione (GSH), Alanine aminotransferase (ALT), Catalase (CAT) and Superoxide Dismutase (SOD). It was determined that by altering the synthesis of pyruvate, acetyl-CoA, NADPH and other antioxidant enzymes, celery seed raised the activity of all antioxidant enzymes and changed the level of insulin.[36]

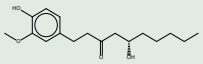
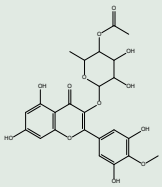
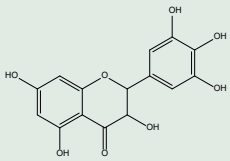
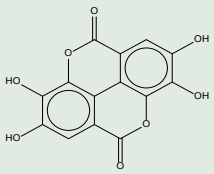
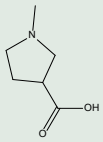
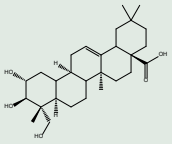
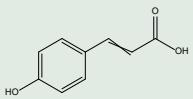
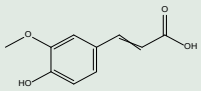
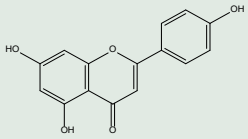
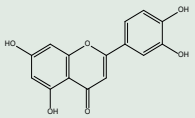
Table 3: An overview some medicinal plant use in the therapeutics activity.

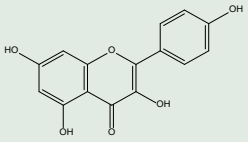
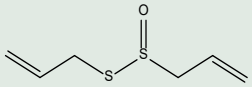
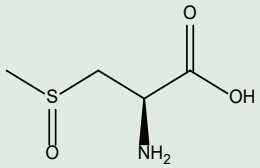
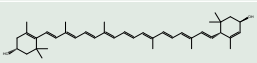
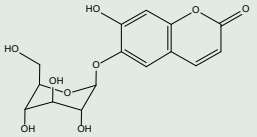
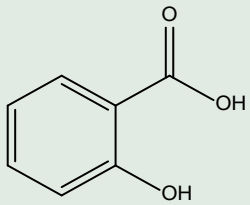
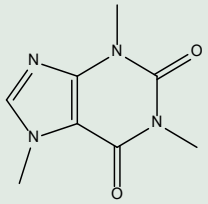
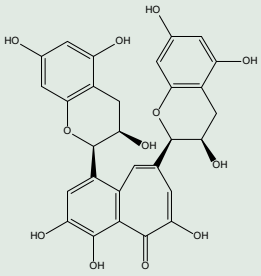
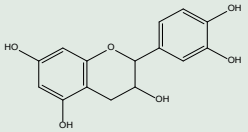
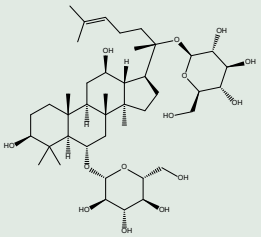
List of medicinal plants Uses therapeutic purpose			
1	Aswagandha	*Withania sominifera*	Solanaceae
2	Vasaka	*Adhatoda vasica*	Acanthaceae
3	Periwinkle	*Catharanthus roseus*	Apocyanaeae
4	Forskohli	*Coloeu forskohlii*	Lamiaceae
5	Guggul	*Commiphora wightii*	Borseraceae
6	Henna	*Lawsonia inermis*	lythraceae
7	Jalbrahmi	*Bacopa monirii*	Plantaginaceae
8	Mint	*Mentha piperita*	Lamiaceae
9	Tulasi	*Occimum sanctum*	Lamiaceae
10	Mexican mint	*Coloeus aeromaticus*	Lamiaceae
11	Sarpagandha/Indian snake root	*Rauwolfia serpentina*	Apocyanaceae
12	Asian pigeon wings/ shankapushpi	*Clitoria ternatea*	Fabaceae
13	Gotu kola/Mandookparni	*Centella asiatica*	Apiaceae
14	Lemon grass	*Cymbopogan flexuosus*	Graminae
15	Golden shower tree	*Cassia fistula*	Fabaceae
16	Basil	*Ocimumbrasilium*	lamiaceae
17	Gurmar	*Gymnema sylvestre*	Apocyanaceae
18	Fringed rue	*Ruta chalepensis*	Rutaceae
19	Shatavari	*Asparagus racemosus*	Asparagaceae
20	Nux vomica	*Strychnus nux-vomica*	Loganiaceae
21	Green chireta	*Andrographis paniculata*	Acanthaceae
22	Coral swirl	*Holarhena antidysentrica*	Apocyanaceae
23	Kasturi benda/Musk mallow	*Abelmoschus moschatus*	Malvaceae
24	Black night shade	*Solanum nigrum*	Solanaceae
25	Bhringraj	*Eclipta alba*	Asteraceae
26	Gaduchi	*Tinospora cordifolia*	Menispermaceae
27	Aloe	*Aloe vera*	Solanaceae
28	Veldt grape	*Cissus quadrangularis*	Vitaceae
29	Gokhru	*Tribulus terrestris*	Loganiaceae
30	Momordica	*Momordica charantia*	Cucurbitaceae
31	Rose	*Rosa sinensis*	Malvaceae

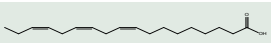
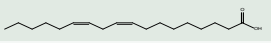
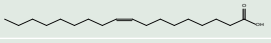
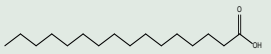
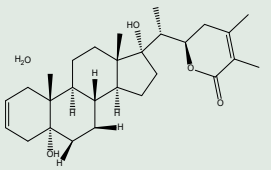
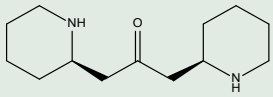
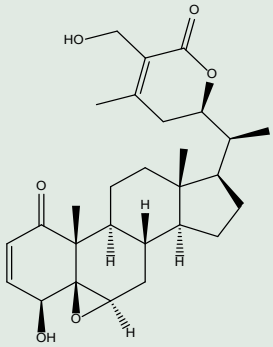
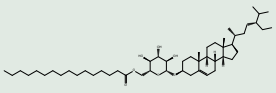
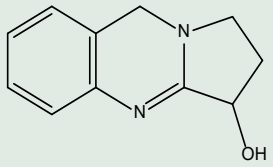
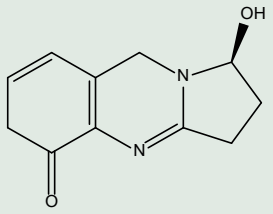
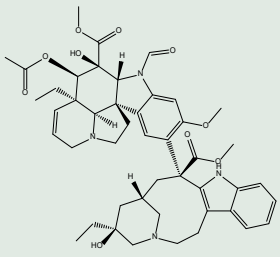
Garlic

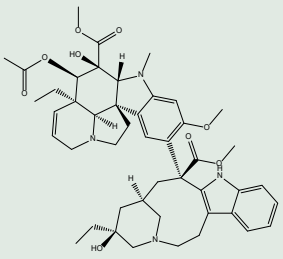
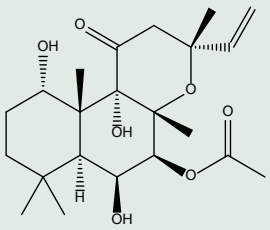
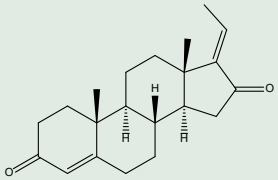
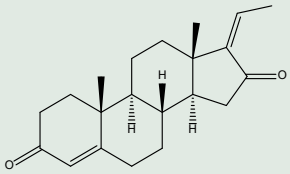
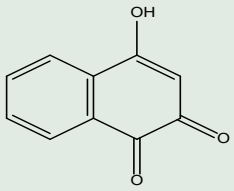
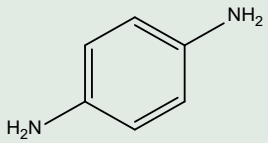
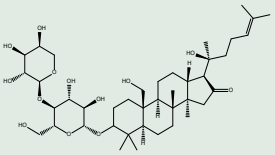
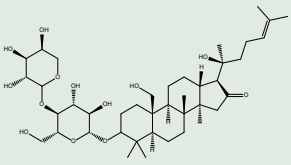
In a double-blind, randomized, placebo-controlled intervention study, 46 patients who had not responded to medication therapy was included. Enteric-coated Australian garlic powder tablets containing 9.6 mg of allicin-releasing potential or corresponding placebo tablets were provided to each participant along with dietary counselling to reduce fat intake.[37] The study shows that when paired with a low-fat diet, enteric-coated garlic powder supplements[38] with a 9.6 mg allicin-releasing potential may be beneficial for patients with mild to moderate hypercholesterolemia. When combined with further data, garlic may be more effective for lipoprotein metabolism if the bioavailability of allicin is increased, maybe by using an enteric-coated dosage form. If this is the case, there's still a chance that a bigger allicin dose will have a stronger hypocholesterolaemic effect.[39] A noteworthy finding in this trial was a little decrease in energy consumption with garlic when compared to a placebo, which was likely caused by a decrease in alcohol, fat and carbohydrate intake. This might have also had an impact on blood lipid levels. According to this study, taking supplements of garlic may have a cholesterol-lowering impact. This effect may be partially attributed to changes in food and nutrient intake as well as direct action of one or more physiologically active components. Human individuals that received short-term garlic supplements showed an increase in

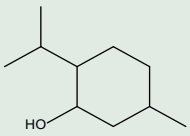
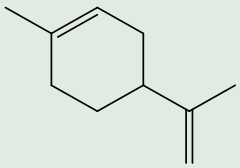
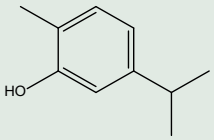
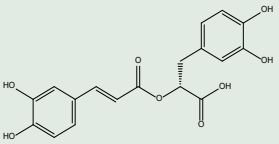
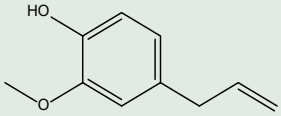
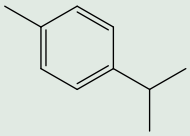
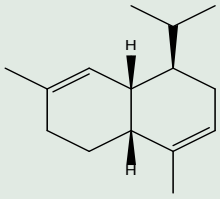
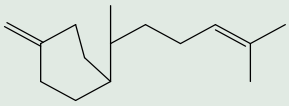
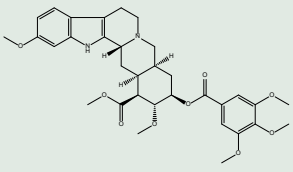
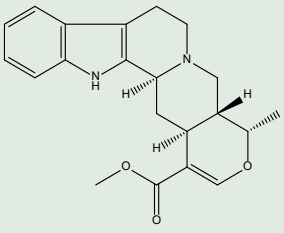
Table 4: Predicted physicochemical parameters of phytoconstituents present in plants having anti-hyperlipidemic activity in various traditional systems of medicine.

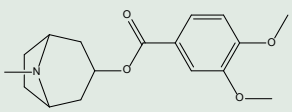
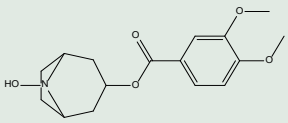
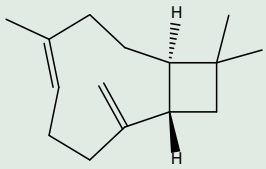
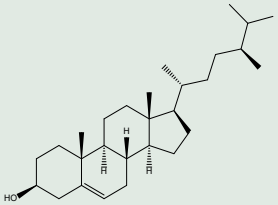
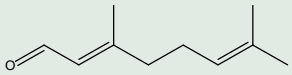
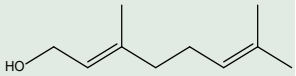
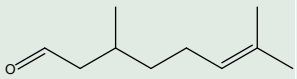
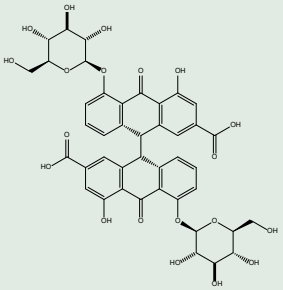
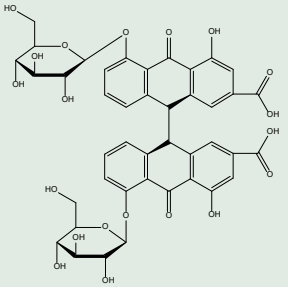
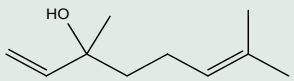
Sl. No.	Name of Phytoconstituents	Chemical Structures	Physiochemical Parameters							
			Lipinski's Rule				Veber's Rules		Genotoxic Carcinogenicity Mutagenicity Rule	PAINS
			Mole. Wt.	Log P	Hydrogen Donor	Hydrogen Acceptor	Total Polar Surface Area Å^2	No. of Rotatable Bonds		
1.	Gingerol		318.1	2.801	0	5.0	80.05	7.0	0	0
2.	Jambolin		520.12	1.095	6	13.0	205.58	6.0	0	0
3.	Jambosine		320.05	0.504	6.0	8.0	147.68	1.0	0	1
4.	Ellagic acid		302.01	0.951	4.0	8.0	141.34	0.0	5	1
5.	Achyranthine		129.08	-2.366	1.0	3.0	40.54	1.0	0	0
6.	Arjunolic acid		460.32	2.712	4.0	5.0	97.99	2.0	0	0
7.	p-coumaric acid		164.05	1.315	2.0	3.0	57.53	2.0	0	0
8.	Ferulic acid		194.06	1.484	2.0	4.0	66.76	3.0	0	0
9.	apigenin		270.05	2.981	3.0	5.0	90.9	1.0	0	0
10.	luteolin		286.05	2.247	4.0	6.0	111.13	1.0	0	1

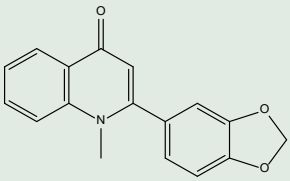
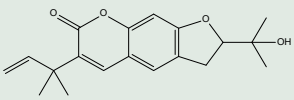
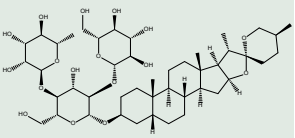
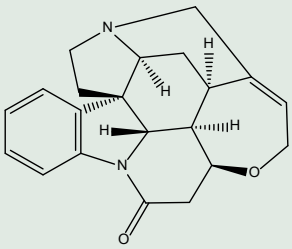
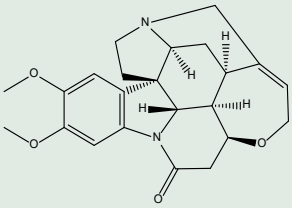
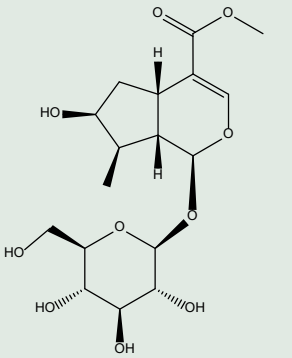
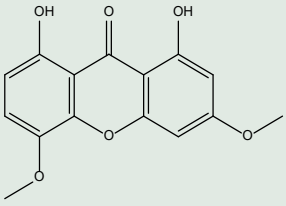
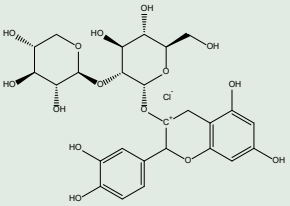
11.	kaempferol		286.05	1.965	4.0	6.0	111.13	1.0	0	0
12.	Allicin		162.02	0.781	0.0	1.0	23.06	5.0	0	0
13.	S-methyl-L-cysteine sulfoxide (MCSO)		151.03	-2.526	3.0	4.0	86.38	3.0	0	0
14.	Lutein		568.43	4.889	2.0	2.0	40.46	10.0	0	0
15.	Esculin		340.08	-1.165	5.0	9.0	149.82	3.0	1	0
16.	Phenolic acids		138.03	2.262	2.0	3.0	57.53	1.0	0	0
17.	caffeine 3.5%,		194.08	0.032	0	6	61.82	0.0	0	0
18.	Theaflavin,		564.13	0.339	9.0	12.0	217.6	2.0	0	1
19.	Epicatechin		290.08	0.684	5.0	6.0	110.38	1.0	0	1
20.	Ginseng		800.49	2.57	10.0	14.0	239.22	10.0	0	0

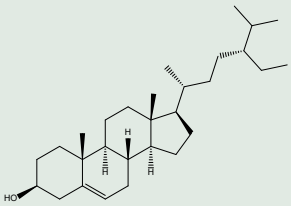
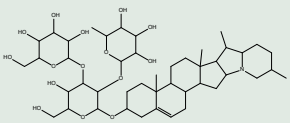
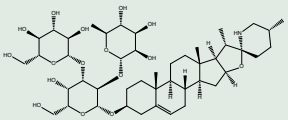
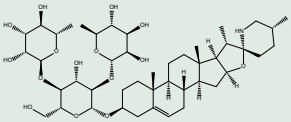
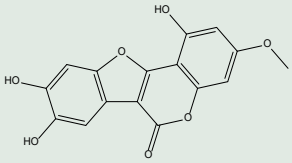
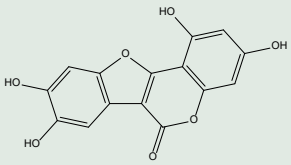
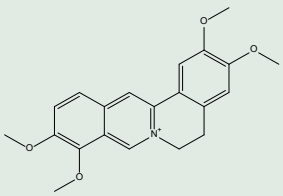
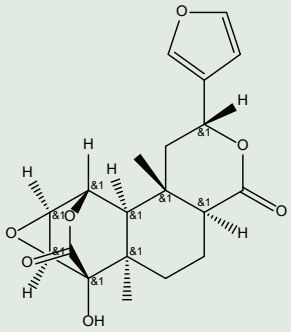
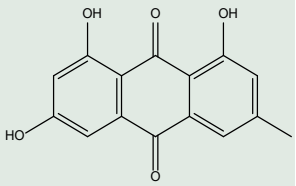
21.	Linolenic acid		278.22	6.597	1.0	2.0	37.3	13.0	0	0
22.	Linoleic		280.24	6.953	1.0	2.0	37.3	14.0	0	0
23.	Oleic acid		282.26	7.063	1.0	2.0	37.3	15.0	0	0
24.	Palmitic acid		256.24	6.648	1.0	2.0	37.3	14.0	0	0
25.	Aswagandh Withanone		442.31	3.63	2.0	4.0	66.76	2.0	1	0
26.	Anaferin		224.19	0.902	2.0	3.0	41.13	4.0	0	0
27.	Withaferin A		470.27	2.923	2.0	6.0	96.36	3.0	7	0
28.	Sitoindoside I		814.67	12.541	3.0	7.0	105.45	25.0	0	0
29.	VasakaVasicine		188.09	0.505	1.0	3.0	35.83	0	0	0
30.	Vasicinone		204.09	-0.766	1.0	4.0	52.9	0.0	1	1
31.	Vincristine		824.4	2.88	3.0	14.0	171.17	11.0	1	0

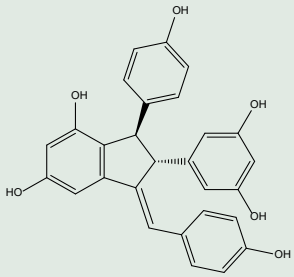
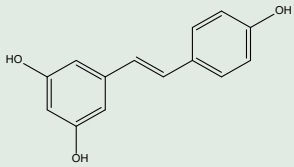
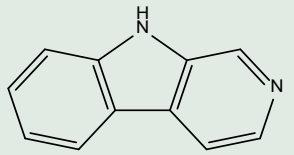
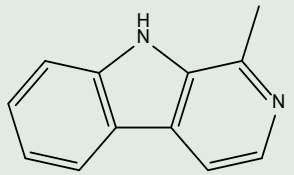
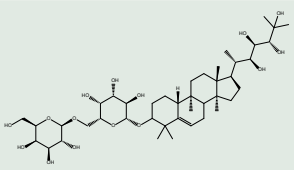
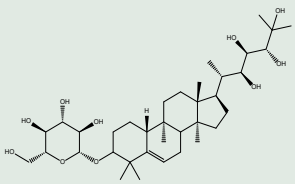
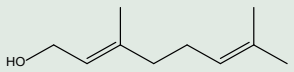
32.	vinblastine		810.42	3.014	3.0	13.0	154.1	10.0	1	0
33.	Forskohlin		410.23	1.418	3.0	7.0	113.29	3.0	0	0
34.	Guggul Guggulsterone E		312.21	2.893	0.0	2.0	34.14	0.0	1	0
35.	Guggulsterone Z		312.21	3.008	0.0	2.0	34.14	0.0	1	0
36.	HennaLawsone		174.03	1.492	1.0	3.0	54.37	0	2	2
37.	p-Phenyldiamine		108.07	-0.259	4.0	2.0	52.04	0.0	5	1
38.	Jalbrahmi Bacoside A		768.47	1.495	8.0	13.0	215.83	10.0	0	0
39.	Bacoside B		768.47	1.52	8.0	13.0	215.83	10.0	0	0

40.	MintMenthol		156.15	3.259	1.0	1.0	20.23	1.0	0	0
41.	Limonene		136.13	4.541	0.0	0.0	0.0	1.0	0	0
42.	Carvacrol		150.1	3.218	1.0	1.0	20.23	1.0	0	0
43.	Rosmarinic acid		360.08	2.005	5.0	8.0	144.52	7.0	1	1
44.	Eugenol		164.08	2.321	1.0	2.0	29.46	3.0	0	0
45.	p-Cymene		134.11	4.347	0.0	0.0	0.0	1.0	0	0
46.	alpha-muurolene		204.19	4.27	0.0	0.0	0.0	1.0	0	0
47.	Bergamotene		206.2	5.366	0.0	0.0	0.0	4.0	0.0	0.0
48.	Reserpine		608.27	2.529	1.0	11.0	117.78	10.0	0	0
49.	Ajmalicine		352.18	2.514	1.0	5.0	54.56	2.0	1	0

50.	Convolamine		305.16	2.103	0.0	5.0	48.0	5.0	0	0
51.	Convoline		307.14	1.81	1.0	6.0	68.23	5.0	2	0
52.	Beta-chariophylen		204.19	4.389	0.0	0.0	0.0	0.0	0	0
53.	Campesterol		400.37	7.448	1.0	1.0	20.23	5.0	0	0
54.	Citral		152.12	3.284	0.0	1.0	17.07	4.0	4.0	0
55.	Geraniol		154.14	3.428	1.0	1.0	20.23	4.0	1	0
56.	Citronellal		154.14	3.037	0.0	1.0	17.07	5.0	0	0
57.	Sennoside A		862.2	0.215	12.0	20.0	347.96	9.0	0	0
58.	Sennoside B		862.2	1.393	12.0	20.0	347.96	9.0	0	0
59.	Linalool		154.14	2.512	1.0	1.0	20.23	4.0	0	0

60.	Graveoline		279.09	3.002	0.0	4.0	40.46	1.0	0	0
61.	Chalepin		314.15	3.488	1.0	4.0	59.67	3.0	1	0
62.	Shatavari Shatavarin IV		886.49	1.899	9.0	17.0	255.91	8.0	0	0
63.	Nux vomica Strychnine		334.17	1.49	0.0	4.0	32.78	0.0	2	0
64.	Brucine		394.19	1.142	0.0	6.0	51.24	2.0	2	0
65.	Loganin		390.15	-1.2	5.0	10.0	155.14	5.0	1	0
66.	Green chiretaSwerchirin		288.06	2.26	2.0	6.0	89.13	2.0	3	0
67.	Kasturi benda/Musk mallowcyanidin-3-sambubioside		583.17	-0.534	10.0	15.0	248.45	6.0	0	1

68.	beta-sitosterol		414.39	8.004	1.0	1.0	20.23	6.0	0	0
69.	solanines		867.5	0.867	9.0	16.0	240.69	8.0	0	0
70.	solasonine		883.49	2.143	10.0	17.0	258.71	8.0	0	0
71.	solamargine		867.5	2.361	9.0	16.0	238.48	7.0	0	0
72.	Bhringraj wedololactone		314.04	2.131	3.0	7.0	113.27	1.0	4	1
73.	demethyl wedelolactone		300.03	1.434	4.0	7.0	124.27	0.0	4	1
74.	Palmatine		352.15	2.893	0	5.0	40.8	4.0	3	0
75.	Tinosporide		374.14	1.195	1.0	7.0	98.5	1.0	5	0
76.	Aloe (Aloe-emodin)		270.050	3.856	3	5	94.830	0	1	1

77.	quadrangularin A		454.140	4.377	6	6	121.380	3	0	0
78.	Resveratrol		228.080	2.542	3	3	60.690	2	0	0
79.	Norharman		168.070	2.481	1	2	28.680	0	3	0
80.	Harman		182.080	2.788	1	2	28.680	0	3	0
81.	Momordicoside A		816.490	2.373	11	15	259.450	11	0	0
82.	Momordicoside B		654.430	3.285	8	10	180.300	8	0	0
83.	Rose (2)		154.140	2.955	1	1	20.230	4	0	0

LDL's resistance to oxidation. These findings imply that one of the potent mechanisms behind garlic's antiatherosclerotic qualities may be decreased LDL oxidation.[40]

Dandelion

The plant *Taraxacum officinale*, commonly referred to as dandelion, has been used in traditional medicine to treat inflammation, hepatic disorders and a number of illnesses affecting women, including uterine and breast cancer. It is also highly regarded in traditional Chinese medicine as a nontoxic plant having remarkable choleretic, diuretic, anti-rheumatic and anti-inflammatory qualities.[41] From the dandelion, several flavonoids have been extracted, including caffeic acid, chlorogenic acid, luteolin and luteolin 7-glucoside.[42] Still, not much research has been done on dandelion root and leaf preventative effects on atherosclerosis.[42] Therefore, the purpose of this study was to assess the possible effects of dandelion root and leaf given orally on the development of atherosclerosis by evaluating lipid profiles and the antioxidant enzyme response in rabbits fed a high-cholesterol diets.[43]

Green Tea

It has been demonstrated that tea (*Camellia sinensis*) inhibits the absorption of exogenous lipids. According to lab research,

green tea inhibits lipid emulsification and absorption, lowers food intake and has a significant impact on fat metabolism. These investigations also shown that green tea could boost energy expenditure by producing heat, oxidising fat and excreting lipids in the faces. Catechin is a distinctive polyphenolic component found in green tea.[44] Epigallocatechin Gallate (EGCG), Epicatechin Augallate (ECG), Epicatechin (EGC) and Epicatechin (EC) were the primary catechins that were presented. The predominant form of tea catechin was EGCG. Reduced lipid absorption may be primarily caused by the strong inhibitory action of EGCG on pancreatic Phospholipase A2 (PLA2). EGCG and the lipid emulsion's surface PC may combine to produce a complex. The compound prevented PLA2 from penetrating the substrate. EGCG may potentially bind directly to the protein of the enzyme, changing its shape and catalytic activity and preventing cells from absorbing lipids. Grove K A, Sae-Tan S, Kennett M J, *et al*. have demonstrated that EGCG administration for six weeks suppressed phospholipid *in vitro* in a noncompetitive manner with respect to substrate concentration, dose-dependently, when compared to a high fat-fed control.[45] It was demonstrated by Koo S. I. and Noh S. K. that EGCG might reduce pancreatic lipase activity. The hydrophilic head of the PC connected with the hydroxyl moiety of EGCG. This interaction may cause the emulsion droplets to enlarge, which would impede the function of pancreatic lipase. According to a different study, green tea extracts also significantly lowered alpha-tocopherol's lymphatic production to 46% and significantly prevented the molecule's absorption. Hepatic lipase, Lipoprotein Lipase (LPL) and Lecithin Cholesterol Acyltransferase (LCAT) were identified as important enzymes in lipoprotein metabolism.[46] These important enzymes supported the transformation of catabolic products of CM and VLDL into high-density lipoproteins, which in turn-controlled lipid metabolism. They also hydrolyzed triglycerides in chylomicrons and very low-density lipoproteins. Lipid metabolism disorders may result from modifications in lipid metabolism brought on by biological activity changes in the enzymes involved in lipid metabolism. Therefore, it may accomplish the goal of improving lipid metabolism by raising the activity or amount of enzymes that affected lipid metabolism.[47]

Ginseng

Since it has been shown to have anti-inflammatory, anti-apoptotic and antioxidant qualities, ginseng has been used traditionally as a natural medication to energize energy, or "Qi." The primary active component of ginseng, ginsenosides, has been demonstrated in numerous studies to have lipid-lowering properties. Nevertheless, rigorous reviews elucidating the molecular processes via which ginsenosides lower blood lipid levels are still lacking, particularly with regard to oxidative stress.[48] A progressive rise in disorders associated with improper lipid metabolism, such as hyperlipidaemia, has been caused by changes in the modern human diet. Increases in plasma levels of Triglycerides (TG), Total Cholesterol (TC) and Low-Density Lipoprotein Cholesterol (LDL-C) and a decrease in serum levels of High-Density Lipoprotein Cholesterol (HDL-C) are the hallmarks of this condition. Atherosclerosis, diabetes, acute myocardial infarction, acute pancreatitis, cerebral infarction and Non-Alcoholic Fatty Liver Disease (NAFLD) are among the conditions for which hyperlipidaemia is a risk factor.[49]

Flaxseed

Flaxseed flour, also known as Linseed (*Linum usitatissimum* Linn.), is widely used in bread and bakery goods. It adds a nutty taste and boosts the nutritional value and health benefits of the finished product. Flaxseed's low-saturated fat content, high polyunsaturated fat and phytosterol content and mucilage content have been shown to lower total and LDL cholesterol concentrations 15, 16.[50] For three months, 15 patients with elevated blood cholesterol levels [>6.2 mmol/L (240 mg/dL)] consumed 15 g of ground flaxseed and 3 slices of bread containing flaxseed daily. This resulted in a 10% decrease in total and LDL cholesterol levels as well as a significant reduction in platelet aggregation, while HDL and tri acylglycerol concentrations remained unchanged (Table 3).[51]

Prediction of physiochemical properties of phytoconstituents from various plants

Prediction of physicochemical properties of phytoconstituents is a crucial step in drug discovery to find out a drug-like lead. Lipinski's rule of five[53] and Veber's rules[54] were used to monitor chemical compounds' drug-likeness. According to the Lipinski rule, compounds having logP<=5, Molecular weight <=500, number of hydrogen bond donors<=5 and number of hydrogen bond acceptors <=10. Lipinski's rules and Veber's rules were predicted by the SWISS ADME server.[55] However, Veber's rule is based on the number of rotatable bonds (No. of rotatable bond<=10) and the Total Polar Surface Area (TPSA<=140 Å2) of compounds. Figure 2 shows the optimum range of compound's physicochemical properties, which means those compounds come under the colored zone having suitable physicochemical space of oral bioavailability. The predicted physicochemical parameters are tabulated in Table 6. The result obtained through an *in silico* study of physicochemical parameters shows that out of 83 screened compounds, only 65 compounds follow Veber's rule and Lipinski's rule of five (Table 4).[56]

CONCLUSION

The anti-hyperlipidemic effects of herbal plants present promising avenues for the management of hyperlipidemia and reduction of cardiovascular risk. Through a multitude of bioactive compounds and diverse mechanisms of action, herbal remedies such as garlic, ginger, green tea and many other demonstrate efficacy in lowering lipid levels, modulating lipid metabolism enzymes and exerting antioxidant and anti-inflammatory

effects. The integration of herbal medicines into conventional therapies offers a complementary approach that may enhance treatment outcomes while potentially mitigating adverse effects associated with synthetic drugs. However, challenges such as variations in bioavailability, standardization of herbal extracts and potential herb-drug interactions underscore the importance of caution and further research in the utilization of herbal remedies for hyperlipidemia management. Rigorous clinical trials and standardized protocols are essential to validate the efficacy, safety and optimal dosages of herbal preparations. Additionally, efforts to bridge traditional knowledge with modern scientific methodologies can enhance our understanding of the mechanisms underlying the therapeutic effects of herbal plants. The physicochemical properties of 83 phytoconstituents from diverse plant sources were investigated and the results demonstrate that only 65 compounds obey Veber's rule and Lipinski's rule, while the remaining compounds violate both laws. The physicochemical parameters obtained throughout this study will aid in the future discovery of novel ligands from natural product research. Overall, the exploration of herbal plants as anti-hyperlipidemic agents holds considerable promise in addressing the global burden of cardiovascular diseases. By harnessing the potential of nature's pharmacy, we can advance towards more holistic, accessible and sustainable approaches to cardiovascular health and wellness.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

LDL: Low-density lipoprotein; **HDL:** High-density lipoprotein; **PL:** Phospholipid **TGs:** Triglycerides; **VLDLs:** Very low-density lipoprotein; **CE:** Cholesterol; **TCMs:** Therapeutic conventional medications; **CAD:** Coronary artery disease; **TBARS:** Thiobarbituric acid reactive substances; **GSH:** Glutathione **ALT:** Alanine aminotransferase **CAT:** Catalase; **SOD:** Superoxide dismutase; **NAFLD:** Non-alcoholic fatty liver disease.

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