Review Article

# A Comprehensive Review on Antihyperlipidemic Activity of Various Medicinal Plants

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#### ABSTRACT

Hyperlipidemia is the greatest hazard factor of coronary heart disease. At present allopathic antihyperlipidemic drugs have been associated with large number of side effects. Herbal treatment for hyperlipidemia has no side effects and is relatively contemptible and locally available. Medicinal plants are the "backbone" of traditional medicine so considered as good source of life for all people due to its wealthy therapeutic properties and being 100% natural. Medicinal plants are extensively used by majority of populations to treat various diseases and have high impact on the world's economy. Traditional therapeutic systems which mainly rely on plants, herbs and shrubs always played a fundamental role in the global health system. Natural products are generally less toxic, have less side effects and easily available so the requirement for herbal drugs is rising. The review article is undertaken to investigate the herbal Plants for antihyperlipidemic activity and various models use in this investigation. This review is specified on the anti- hyperlipidemic activity of the most recognizable therapeutic plants of medicine.

Keywords: Hyperlipidemia, Medicinal plants, Coronary heart disease.

#### INTRODUCTION

Hyperlipidemia is a disarray of lipid metabolism produced by elevation of plasma concentration of the diverse lipid and lipoprotein fractions, which are the source of cardiac disease. It is define as increase serum TC, TG, VLDL, LDL and HDL which are responsible for different complications like: heart attack, coronary artery syndrome, stroke, atherosclerosis, myocardial infarction and pancreatitis. Hyperlipidemia can be either primary or secondary type, the primary syndrome may be treated by hypolipidemic drugs, but secondary induced by diabetes, hypothyroidism or renal lipid nephrosis which treated by treating the original disease moderately than hyperlipidemia<sup>1</sup>.Genetic disorders and lifestyle diet rich in calories, fat, and cholesterol play a vital role to cause dyslipidemia around the world<sup>2</sup>. The main factor which are responsible for hyperlipidemia includes changes in life style habits in which risk factor is mainly poor diet i.e. fat intake greater than 40 percent of total calories, saturated fat ingestion more than 10 percent of total calories; and cholesterol ingestion larger than 300 milligrams per day<sup>3</sup>. For hyperlipidemia large number of synthetic drugs available, not a bit is helpful for all lipoprotein disorders, and each drugs are linked with a number of adverse effects. Therefore, now a day other materials are search from natural sources with the intention of less toxic, less expensive, and provide superior safety and efficacy on a long term practice. Natural products from plants are a rich source of medicine used for centuries to treat various diseases4.

Hyperlipidemia

Hyperlipidemia is a medical state characterized by an elevation of any or all lipid profile or lipoproteins in the blood<sup>5</sup>. The lipid metabolism is synchronized in many different ways. Enzymes are most important regulators of lipid metabolism. 3-Hydroxy-3-methylglutaryl coenzyme A reductase enzyme responsible for cholesterol biosynthesis<sup>6</sup>.

While elevated low density lipoprotein cholesterol (LDL) is thought to be the best gauge of atherosclerosis. dyslipidemia (abnormal amount of lipids in the blood) can also express prominent total cholesterol (TC) or triglycerides (TG), or low levels of high density lipoprotein cholesterol (HDL<sup>5</sup>. Hyperlipidemia is a medical as well as social problem, especially associated with diabetes mellitus leading to increasing morbidity and mortality. The chief risk factors of hyperlipidemia are associated with atherosclerosis which predispose ischemic heart disease and cerebrovascular disease<sup>7</sup>.

Many allopathic hypolipidemic drugs like statins are available in the market, but they cause many side effects like hyperuricemia, diarrhoea, myositis, hepatotoxicity, etc. As they are mainly enzyme inhibitors, so they may be inhibit other grave enzymes in the body. Moreover, statins are intake on a long-term basis so it cause chronic toxic effects over a life time use Therefore attention is now rewarded much to investigate natural hypolipidemic agents from plant sources<sup>8</sup>.

#### Classification of hyperlipidemia

Hyperlipidemia may be classified as either familial (also called primary) caused by definite genetic abnormalities, or acquired (also called secondary) that leads to change in Diagnosis<sup>3</sup>

S.No	Test name	Normal values	Indicators
1	Total	Total Cholesterol: < 200	200-239  mg/dL = Borderline High (borderline
	Cholesterol	mg/dL (desirable) (< 180	risk for coronary heart disease > 240 mg/Dl
		optimal)	Hypercholesterolemia
2	Total Cholesterol for children	< 180 mg/Dl	> 180 mg/dL may lead to Atherosclerosis
3	Triglyceride Levels	Less than 150 mg/dl	150-199 mg/dL is Border line High 200-499
			mg/dL is High 500 mg/dL or above isVery
			High.
4	VLDL cholesterol	The VLDL normal range	>40 suggest increase the risk of developing
		is between 0-40 mg/dL	heart disease
		and the suggested	
		optimum range is between	
		0-30 mg/dL	
5	C-reactive Protein (CRP)	CRP< 1 mg/dl	CRP> 1mg/dl (> 10mg/dl suggests
			inflammation
6	LDL Cholesterol	< 100 mg/dL (optimal)	130-159Mg/dL Borderline
		100-129 mg/dL (near	High 160-189 Mg/dL High ≥190 Mg/dL Very
		/above optimal)	High
7	HDL Cholesterol	> 60 mg/dl is enviable	HDL levels < 40 Mg/dL increases risk for
			CHD. women with levels $< 47 \text{ mg/dL}$ and men
			< 37 mg/dL have increased risk.

Hyperlipoproteinemia		Occurrence	Imperfection	Elevated Symptoms Lipoprotein		Appearance of Serum		
Туре І	А	Very rare	Reduce lipoprotein lipase	Chylomicron	ns	Stomach ache, retinalis, eruptive skin xanthomas	Creamy laver	top
	В	Very rare	Distorted Apo c2			hepatosplenomegaly		
	С	Very rare	LPL inhibitor in blood					
Type II	А	Less ordinary	LDL receptor	LDL		Xanthelasma, tendon xanthomas	Apparent	
	В	Usual	Reduce LDL receptor & augmented Apo B	LDL a VLDL	and		Lucid	
Type III		Atypical	Imperfection in Apo E 2 synthesis	IDL			Opaque	
Type IV		Average	2	VLDL			Cloudy	
Type V		Ordinary		VLDL a Chylomicron	and ns		Creamy layer	top

plasma lipid and lipoprotein metabolism.

*Familial (primary):* Familial hyperlipidemia are classified as:

Type I - Raised cholesterol with high triglyceride

Type II - High cholesterol with normal level of triglyceride

Type III - High cholesterol and triglycerides

Type IV - Raised triglycerides, and raised uric acid

Type V - Raised triglycerides

Acquired (secondary)

Acquired hyperlipidemias (also called secondary dyslipoproteinemias) in which increased risk of atherosclerosis, when associated with marked hypertriglyceridemia, may cause pancreatitis and various complications of the chylomicronemia disease.

Most ordinary causes of acquired hyperlipidemia are:

Diabetes Mellitus (Type 2)

Use of drugs such as diuretics, beta blockers, and estrogens etc.

Animal models for evaluation of antihyperlipidemic activity<sup>9</sup>

High Cholesterol diet induced method High Fructose diet induced method

Triton induced hyperlipidemic method

Streptozotocin induced diabetic method

Alloxan induced diabetic method

Tylaxapol induced hyperlipidemic method

High fat diet induced hyperlipidemic method

Hydrocortisone induced hyperlipidemic method Atherogenic diet induced hyperlipidemic method The other models which can be used<sup>10</sup>:

S no	Plant name	Family	Part used	Dose	Models used	Reference
5. 110.	Abalomosahu	Ганнгу	Whole	Dose	Tulayanal induced	Kelefence
1	s asoulantus	Malvaceae	plants	300mg/kg	mothod	13
	s esculentus Achyranthus		Whole		Allovan induced	
2	aaspara Lipp	Amaranthaceae	plant	250-500mg/kg	method	14
			plant		Oil fad hyperlinidamia	
3	Aegie	Rutaceae	Leaf	250 mg/kg	On red hyperhipideniic	15
	marmelos		W/11-		rat Strents - stanin in durad	
4	Ajuga iva	Labiatea	whole	10mg/kg	Streptozotochi induced	16
	A 11:		Frach		Triton V 100 induced	
5	Allium	Alliaceae	Fresh	10mg/kg	Intona 100 induced	17
	sativum		iruits		metnod	
6	Alpinia	Zingiberaceae	Rhizome	200& 400mg/kg	Triton induced method	18
	Galangal L.	-		100 200 400		
7	Alstonia	Apocynaceae	Leaves	100,200,400mg/	Streptozocin induced	19
	Scholarin	1 2		kg	diabetic rat	
8	Amaranthus	Amaranthaceae	Leaves	200,400mg/kg	Streptozocin induced	20
	Viriais				diabetic rat	
9	Andrographis	Acanthaceae	Leaves	25mg/kg	Tyloxapol induced	21
	paniculata			0 0	method	
10	Anethum	Apiaceae	Essential	45,90,180mg/kg	High cholesterol diet	22
	Graveolens	1	01l		induced method	
11	Anogeissus	Combretaceae	Fresh	250,500,750mg/	Atherogenic diet	23
	Latifolia		gum	kg	induced method	
12	Anthocephalu	Rubiaceae	Roots	500mg/kg	Tyloxapol induced	24
	s Indicus	100100000	10005	0001118/118	method	
13	Apium	Aniaceae	Seed	213 425mg/kg	High cholesterol diet	25
15	Graveolens	ripideede	Beed	213, 123 mg/ kg	induced method	23
14	Asparagus	Liliaceae	Roots	150mg/kg	Alloxan induced	26
11	Racemosus	Linuccue	Roots	1501116/166	method	20
15	Amaranthus	Amaranthaceae	Leaves	200-400mg/kg	Triton induced method	27
15	<i>caudatus</i> L.	7 marantinaceae	Leaves	200 1001115/115	The made a method	27
	Rauhinia		Leaves &		Tylaxapol induced	
16	nurnurea	Fabaceaae	unripe	300mg/kg	method	28
	purpurcu		fruits		method	
	Bauhinia		Roots &			
17	variegate	Ceasalpiniaceae	Stem	200 &400mg/kg	Triton induced method	29
	Linn.		Stelli			
18	Commiphora	Burseraceaae	Resin	250mg/kg	High fat diet induced	30
10	mukul	Duiscideedde	part	2501115/185	method	50
19	Caesearia	Flacourtiaceae	Leaves	300mg/kg	Streptozocin induced	31
17	sylvestris	1 lacourtiaceae	Leaves	500mg/kg	method	51
20	Capparis	Capparidaceae	Bark,Flo	500mg/kg	Streptozocin induced	30
20	Deciduas	Cappandaceae	wer,Fruit	Jooning/Kg	method	52
21	Capparis	Capparidaceae	Fruite	200& 100mg/kg	Tylaxapol induced	33
21	spinosa	Cappandaceae	Tuns	200 <b>&amp;</b> 400111g/kg	method	55
22	Carica	Caricacaaa	Seed,Lea	100 /00mg/kg	Alloxan induced	34
22	рарауа	Cancaccac	ves	100-400ing/kg	method	54
23	Cassia fistula	Fabaceae	Legume	100,250,500mg/	High cholesterol diet	35
				kg	induced method	
24	Catharanthus	Aconthecese	Loover	150mg/kg	Streptozocin induced	36
24	roseus Linn	Acanthaceae	Leaves	1JUIIIg/Kg	method	50
25	Celastrus	Calastrasses	Saad	65 m a /le a	High fat diet induced	27
23	paniculatus	Celastraceae	Seed	oomg/kg	method	51
26	Curcuma	Zingiharaaaa	Dhigoma	200mg/ka	Streptozocin induced	29
20	longa	Zingiberaceae	Knizome	SUUIIIg/Kg	method	30
27	Cymbopogon	Graminassas	Loover	100 & 200m ~ /1	Dexamethasone	20
<i>∠1</i>	citrates	Grannnaceae	Leaves	100a200ing/kg	induced method	37

# Medicinal plants with hypolipidemic activity

28	Coccinia indica	Cucurbitaceae	Leaf	200 mg/kg/b.w	Alloxan induced	40
29	Cassia	Caesalpiniaceae	Flowers	150,300,450mg/	Tylaxapol induced	41
30	auriculata Cynara	Asteraceae	Leaver	kg bw 150,300,600mg/	Cholesterol diet	42
30	scolymus Eclipta	Asteraceae	Leaves	kg	induced method	42
31	prostate (L.)	Asteraceae	Leaves	100&200mg/kg	Atherogenic diet induced method	43
32	Elaeis guineensis	Arecaceae	Root	250&500mg/kg	Olive oil induced method	44
33	Eugenia Jambolana	Myrtaceae	Seed Kernel	100mg/kg bw	Streptozocin induced method	45
34	<i>Ficus racemosa</i> Linn.	Moraceae	Bark	100-500mg/kg bw	Alloxan induced diabetic rat	46
35	Garcinia cambogia	Guttiferae	Peel of matured fruits	400mg/kg bw/day	High fat diet induced method	47
36	Glyccyrrhiza glabra	Fabaceae	Rhizome	250-500mg/kg	High fat diet induced method	48
37	Gymnena sylvestre	Asclepiadaceae	Leaf	200mg/kg	High cholesterol diet induced method	49
38	Hibiscus rosa sinesis	Malvaceae	Root	500mg/kg/day	Tylaxopol induced method	50
39	<i>Hibiscus</i> Sabdariffa Linn.	Malvaceae	Leaves & Calyces	500mg/kg/ day	High cholesterol diet induced method	51
40	Icacina senegalensis	Icacinaceae	Root	100,200& 400mg/kg	Alloxan induced method	52
41	Lagenaria siceraria Mol	Cucurbitaceae	Fruits	200&400mg/kg bw	Triton induced method	53
42	Luffa acutangula	Cucurbitaceae	Fruit	200-400mg/kg	Streptozocin along with nicotinamide	54
43	Lycium barbarum	solanaceae	Fruits	250&500mg/kg	Alloxan induced method	55
44	Morinda Citrifolia	Rubiaceae	Fruits	0.25-1.00g/kg	Streptozocin induced diabetic rat	56
45	Moringa oleifera	Moringaceae	Leaf	100mg/kg/ bw	Cadmium exposed rat	57
46	Melothria Maderaspata na	Cucurbitaceae	Aerial parts	100&200mg/kg b.w	Streptozocin induced method	58
47	Morus alba	Moraceae	Leaves	30mg/kg	Tylaxopol induced method	59
48	<i>Morus indica</i> L.	Moraceae	Leaves	500mg/kg	Streptozocin induced method	60
49	Mucuna Prurines	Leguminoseae	Leaves	200mg/kg	Alloxan induced method	61
50	Nelumbo Nuficera	Nelumbonaceae	Fruit	100-1000mg/kg	Poloxamer407 induced method	62
51	Ocimum basilicum	Lamiacea	Whole plant	20mg/kg	Streptozocin induced method	63
52	Ocimum Tenuiflorum	Lamiaceae	Leaves	250-500mg/kg	Streptozocin+nicotina mide induced method	64
53	Pipper longum	Piperaceae	Root	200mg/kg	Streptozocin induced method	27
54	Psidium guajava linn	myrtaceae	leaves	200& 400mg/kg	Cholestrol diet induced method	27

55	Piliastigma thonningii	Musecea	leaf	50-200mg/kg	Serum lipid profile of male albino rat	27
57	Peucedanum pastinacifoliu m Boiss.	Apiaceae	Aerial parts	125,250,500mg/ kg	High cholesterol diet induced method	65
58	Plumeria rubra L.	Apocynaceae	Fresh flowers	250mg/kg b.w	Alloxan induced method	66
59	Pterocarpus marsupium	Fabaceae	Wood & bark	150-300mg/kg	Alloxan hydrate inducde method	27
60	Rosa laevigata Michx.	Rosaceae	Fruits	25and50mg/kg	High fat diet induced method	67
61	Randia dumetorum	Rubiaceae	Fruit	200-400mg/kg	Streptozocin & nicotinamide induced method	68
62	Sphaeranthus indicus	Asteraceae	Flower head	500mg/kg/day	Atherogenic diet induced method	69
63	Sesbania grandiflora	Fabaceae	Leaves	200mg/kg	Tylaxapol induced method	70
64	Stevia rebaudiana	Asteraceae	Leaves	150mg/kg/ bw	Alloxan induced method	71
65	Salvodora persica	Salvadoraceae	Root	250-500mg/kg	Streptozocin induced method	72
66	Spergularia purpurea	Caryophyllaceae	Whole plant	10mg/kg	Streptozocin induced diabetic rat	27
67	Salvadora oleoides	Salvadoraceae	Aerial Parts	1g,2g/kg	Alloxan induced method	73
68	Syzigium alternifolium	Myrtaceae	Bark	100,200mg/kg	High fat diet &Dexamethasone	74
69	Terminalia arjuna	Combretaceae	Bark	10-50mg/kg	High fat diet induced method	75
70	Terminalia chebula	Combretaceae	Pericarp fruit	1.05 ,2.10mg/kg	Atherogenic diet induced method	76
71	Trianthum portulacastru m	Azoaceae	Whole plant	100,200mg/kg	High fat diet induced method	77
72	Urtica dioica	Urticaceal	Leaves	50mg/kg	Alloxan induced method	78
73	Withania somnifera	Solanaceae	Roots and Leaf	100,200mg/kg	Alloxan induced method	79
74	Zingiber Officinale	Zingiberaceae	Rhizome	500mg/kg	Streptozocin induced method	80

Hereditary hypercholesterolemia in experimental animals like rats.

Hereditary hyperlipidemia in rabbits:

Transgenic animals- apoprotein E knock out model

Fructose induced hypertriglyceridemia in laboratory animals rats

Pathophysiology of hyperlipidemia

The pathophysiology of hyperlipidemia is deliberate beneath the two basic classifications of hyperlipidemia, i.e., primary and secondary hyperlipidemia.

Primary hyperlipidemia involve the hyperchylomicronemia in which defect in lipid metabolism lead to hypertriglyceridemia and hyperchylomicronemia cause by a imperfection in lipoprotein lipase activity or the lack of surface apoprotein CII. Further, in primary hyperlipidemia, the LDL cholesterol is elevated.

In secondary hyperlipidemia, absorption of chylomicrons from the G.I tract within a 30-60 min, after ingestion of a meal containing fat that may enhance serum triglycerides for 3-10 hours. The diabetes mellitus patients have been noted to acquire low LPL activity which caused high synthesis of VLDL cholesterol by the liver leading to hyperlipidemia. Moreover, hypothyroidism-induced low LPL activity and lipolytic activity responsible to reduce hepatic degradation of cholesterol to bile acids. Moreover, hyperadrenocorticism enlarged the synthesis of VLDL by the liver cause hypercholesterolemia and hypertriglyceridemia. Liver disease hypercholesterolemia caused by reduced seepage of cholesterol in the bile. Moreover, in nephritic syndrome, the common pathway

for albumin and cholesterol causes low pressure leading to improved cholesterol synthesis<sup>11</sup>.

Primary disorders are classified into six categories. Lipoprotein elevations include the following: I (chylomicrons), IIa (LDL), IIb (LDL + VLDL), III (intermediate-density lipoprotein, or HDL); IV (VLDL), and V (VLDL + chylomicrons). Secondary hyperlipidemia also be present and various drugs may increase lipid levels (e.g., progestins, thiazide, glucocorticoids, protease inhibitors, cyclosporine, mirtazapine,). Primary defect in hypercholesterolemia is the inability to bind LDL to LDL receptor (LDL-R) or, a defect of LDL-R complex into the cell after binding. This leads to lack of LDL deprivation by cells and unfettered biosynthesis of cholesterol, with total cholesterol and low density lipoprotein being inversely proportional to the insufficiency in low density lipoprotein receptors<sup>5</sup>.

*Etiology/Causes of Hyperlipidemia* Acute intermittent porphyria Acromegaly Obesity

Anorexia nervosa

Autoimmune disease

Hypothyroidism and

Cushing's disease

Hepatitis<sup>12</sup>

Diabetes mellitus (type 2)

Glucocorticoids

Monoclonal gammopathies

Nephrotic syndrome

Other factors may include medications (eg, beta blockers and oral contraceptives, thiazide diuretics, glucocorticoids)<sup>3</sup>.

Treatment<sup>5</sup>

Treatment therapy consist of two approaches, which are Non-pharmacological therapy and Pharmacological therapy.

Non pharmacological therapy

The aim of non pharmacological therapy is decrease the ingestion of total fat, saturated fatty acids and cholesterol. This therapy involves;

Decreased saturated fat intake to 7 percent of daily calories Decreased total fat intake to 25 to 35 percent of daily calories

Inadequate dietary cholesterol less than 200 mg per day Consumption of 20 to 30g of soluble fiber, which is found in oats, peas, beans, and certain fruits; and Increased ingestion of plant sterols, substances found in nuts, vegetable oils, corn and rice, to 2 to 3 g daily. Other foods that can assist to control cholesterol consist of cold-water fish, for example mackerel, sardines, and salmon. Soybeans found in soy nuts and many meat substitutes restrain a powerful antioxidant that can decrease LDL level.

Pharmacological therapy

HMG-CoA reductase inhibitors (Statins): Lovastatin, Simvastatin, Pravastatin, Atorvastin, Rosuvastin.

Bile acid sequestrants (Resins): Cholestyramine, Colestipol.

Activate lipoprotein lipase (Fibric acid derivatives): Clofibrate, Gemfibrozil,Benzafibrate and Fenofibrate. Inhibit lipolysis and triglyceride synthesis: Nicotinic acid. Others: Ezetimibe, Gugulipid

## CONCLUSION

Hyperlipidemia is a crtical condition of elevated lipid levels in the body that ultimately lead to the development and progression of various CVDs. The link between hyperlipidemia and occurance of CVDs has already been established, the problem of enchanced cholesterol levels in blood is still prevailing and is being a cause for many coronary disorders. Studies reveal that an increase in HDL cholesterol and decrease in TC, LDL cholesterol and TG is associated with a decrease in the risk of ischemic heart diseases. Though many drugs are available to treat Hyperlipidemia. The antihyperlipidaemic activity of plants plays an important role in the reduction of CVD. Plant parts or plant extract are sometimes even more potent than hypolipidemic known drugs. Currently used hypolipidemic drugs are associated with so many adverse effects and withdrawal is associated with rebound phenomenon which is not seen with herbal preparations.

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