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Review Article

Hyperlipidemia: Mechanisms, Experimental Models, and Herbal Therapeutic Strategies – A Comprehensive Review

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Abstract: Hyperlipidemia, characterized by elevated lipid levels in the blood, is a critical risk factor for cardiovascular, cerebrovascular, and peripheral vascular diseases. This review provides a comprehensive overview of hyperlipidemia, including its classification and lipid transport mechanisms. Various experimental models—diet-induced, genetic, and chemical—used to replicate hyperlipidemia in animals are discussed, highlighting their mechanisms and outcomes. Furthermore, the review explores the role of medicinal plants with antihyperlipidemic potential, including *Cynara cardunculus*, *Medicago sativa*, *Allium sativum*, *Ginger*, and *Flax seeds*. These herbs exhibit lipid-lowering effects via multiple mechanisms such as antioxidant activity, inhibition of lipid synthesis, and enhancement of cholesterol excretion. Their efficacy, often comparable to standard pharmaceuticals like statins, positions them as promising alternatives or adjuncts in hyperlipidemia management. The review underscores the potential of integrating traditional medicine with modern therapeutic approaches to combat hyperlipidemia effectively.

Keywords: Hyperlipedemia, *Cynara Cardunculus, Medicago Sativa, Achyranthes Aspera*, *Hibiscus Sabdariffa* L.

INTRODUCTION

Hyperlipidemia is a major global health issue caused by disruptions in fat metabolism or its transport within the body. It is mainly defined by an increase in blood lipid levels, including total lipids, fatty acids, and detrimental lipoproteins, while protective lipoproteins are decreased. This condition significantly contributes to the onset and advancement of cardiovascular, peripheral vascular, and cerebrovascular diseases [1].

Fredrickson /WHO Classification of Hyperlipoproteinemias

Hyperlipidemia is broadly classified into primary (familial) and secondary types, depending on its root causes.

Table No 1: Classification for hyperlipidemia [2]

Sl. No	Disorder	Deficiency	Elevated lipoproteins	Manifestations	Therapy
I	Familial hyperchylomicronemia Or Primary hyperlipoproteinemia	ApoC2 alteration or Lipoprotein lipase deficiency	Chylomicrons	Lipemia retinalis, xanthomas, hepatosplenomegaly, and acute pancreatitis	Diet management
IIa	Familial hypercholesterolemia or Polygenic hypercholesterolemia	LDL receptors deficiency	LDL	Arcus senilis, xanthelasma, and tendon xanthomas	Statins, bile acid sequestrants, and niacin

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Sl. No	Disorder	Deficiency	Elevated lipoproteins	Manifestations	Therapy
IIb	Familial combined hyperlipidemia	Reduced LDL receptors and elevated ApoB	LDL and VLDL		Statins, niacin, and fibrates
III	Familial dysbetalipoprotenemia	Impairment in ApoE-2 synthesis	IDL	Tuberous xanthomas and palmar xanthomas	Fibrate, statins
IV	Familial hypertriglyceridemia	Elevated VLDL production and reduced excretion	LDL	High triglyceride levels can lead to pancreatitis.	Fibrate, niacin, statins
V	Endogenous hypertriglyceridemia	Elevated VLDL production and reduced lipoprotein lipase (LPL) activity	Chylomicrons & VLDL		Niacin and fibrates

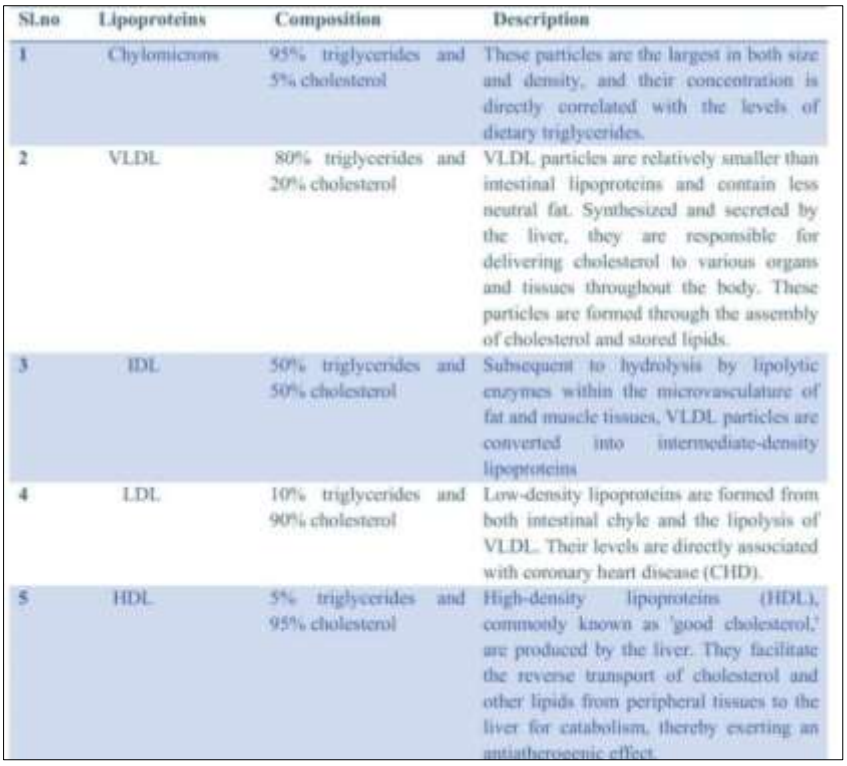
Table No 2: Secondary Form of Hyperlipoproteinemia [3]

Sl.no	Disorder	Plasma lipid	Mechanism
1	Diabetes Mellitus	Occasionally elevated LDL levels and raised VLDL levels	Enhanced VLDL secretion and decreased catabolism
2	Alcoholic Hyperlipedemia	Elevated VLDL	Increased secretion of VLDL
3	Uremia	Elevated VLDL	Reduced VLDL catabolism
4	Beta blockers	Elevated VLDL and decreased HDL	Reduced activation of lipoprotein lipase
5	Nephrotic syndrome	Elevated VLDL and LDL	Partial inhibition of lipoprotein lipase as a compensatory response to urinary albumin loss
6	Corticosteroids	Elevated VLDL & LDL	Suppressed lipoprotein lipase
7	Hypothyroidism	Increased LDL and intermittent VLDL	Lowered LDL receptor levels

LIPOPROTEINS [4]

These are large spherical particles composed of a nonpolar lipid core, containing stored forms of cholesterol and neutral fats, surrounded by a polar surface layer consisting of amphipathic lipids, unesterified cholesterol, and structural proteins.

CLASSIFICATION OF LIPOPROTEINS



Sl.no	Lipoproteins	Composition	Description
1	Chylomicrons	95% triglycerides and 5% cholesterol	These particles are the largest in both size and density, and their concentration is directly correlated with the levels of dietary triglycerides.
2	VLDL	80% triglycerides and 20% cholesterol	VLDL particles are relatively smaller than intestinal lipoproteins and contain less neutral fat. Synthesized and secreted by the liver, they are responsible for delivering cholesterol to various organs and tissues throughout the body. These particles are formed through the assembly of cholesterol and stored lipids.
3	IDL	50% triglycerides and 50% cholesterol	Subsequent to hydrolysis by lipolytic enzymes within the microvasculature of fat and muscle tissues, VLDL particles are converted into intermediate-density lipoproteins
4	LDL	10% triglycerides and 90% cholesterol	Low-density lipoproteins are formed from both intestinal chyle and the lipolysis of VLDL. Their levels are directly associated with coronary heart disease (CHD).
5	HDL	5% triglycerides and 95% cholesterol	High-density lipoproteins (HDL), commonly known as 'good cholesterol,' are produced by the liver. They facilitate the reverse transport of cholesterol and other lipids from peripheral tissues to the liver for catabolism, thereby exerting an antiatherogenic effect.

PATHWAYS OF LIPID TRANSPORT [5]

Cholesterol is absorbed in the intestine and delivered to the liver via chylomicron remnants, which are internalized through LDL receptor-related proteins (LRP). The liver secretes cholesterol as VLDL, which is converted to remnant lipoproteins following triglyceride removal by lipoprotein lipase. These remnants are either cleared by LDL receptors or further processed into LDL for clearance. HDL mediates reverse cholesterol transport from peripheral tissues to the liver, where cholesterol is either recycled via CETP or taken up by hepatic lipase and ultimately excreted in bile.

ENZYMES MODULATING LIPOPROTEIN METABOLISM [6]

Enzymes	Functions
Lipoprotein lipase	Lipoprotein lipase (LPL), located on endothelial cells in tissues like muscle, heart, and adipose, hydrolyzes triglycerides into free fatty acids and monoacylglycerol. It is essential for lipid metabolism and energy delivery.
Hepatic lipase	Hepatic lipase (HL), produced by the liver and found in the adrenal glands and ovaries, hydrolyzes phospholipids and triglycerides in lipoproteins, aiding their uptake by cells through receptor and proteoglycan interactions.
Lecithin cholesterol acyl transferase	Lecithin-cholesterol acyltransferase (LCAT) is essential for HDL metabolism, converting free cholesterol into cholesteryl esters that are incorporated into the lipoprotein core, aiding HDL maturation.
Cholesteryl ester transfer protein	Cholesteryl ester transfer protein (CETP) is a plasma glycoprotein that transfers cholesterol esters from HDL to chylomicrons, VLDL, and LDL in exchange for triglycerides. CETP deficiency is associated with higher HDL and lower LDL levels.
Microsomal triglyceride protein	Microsomal triglyceride transfer protein (MTP) transfers neutral lipids between microsomal membranes in the liver and intestines. It is crucial for assembling apolipoprotein B-containing lipoproteins and regulating lipid biosynthesis.
Acyl Co-A transferase	Acyl-CoA transferase (ACAT) is a membrane-bound enzyme that synthesizes cholesteryl esters from fatty acyl-CoA and cholesterol, helping maintain cholesterol balance and supporting the assembly of apolipoprotein B-containing lipoproteins in the liver and intestines.

Animal Models of Hyperlipedemia [7, 8]

1. Diet-Induced Hyperlipidemic Models

Cholesterol Fed Animals

Animals were subjected to various high-cholesterol dietary regimens to induce hyperlipidemia (HL), a key factor in the development of cardiac disorders such as atherosclerosis and ischemic heart disease. Diets included: an 8-week regimen with 2% added cholesterol; a high-lipid diet containing 10% lard, 1% cholesterol, 1% sodium tauroglycocholate, 5% egg yolk, and 0.2% propylthiouracil; a 12-week high-fat diet with 0.5 g each of cholesterol, coconut oil, and cholestyramine per kg of body weight per day; and a 40-day diet with 1.3% cholesterol and 3% saturated fat.

Methionine Induced Hyperlipidemia

Methionine, an essential amino acid vital for protein synthesis and sulfate production, may support memory and reduce heart disease risk. However, studies show that oral administration of methionine (1 g/kg) for 30 days significantly raises total cholesterol, triglycerides, and LDL levels while lowering HDL, making it effective for inducing hyperlipidemia (HL).

2. Genetic Models

Apoe Knock-Out Mice

The critical role of apoE in lipoprotein metabolism is evident in apoE-deficient mice, developed through homologous recombination in embryonic stem cells. Independently created by three research groups, these knockout models lack apoE expression, leading to severely elevated serum cholesterol due to impaired remnant lipoprotein clearance. When fed a high-fat, high-cholesterol diet, the mice accumulate cholesterol-rich, VLDL-sized lipoproteins.

3. Chemical Models

Triton Induced Hyperlipidemia

Triton X-100 (TX) is an example of a non-ionic detergent or surfactant. It is an octylphenol polyethoxylate with a hydrophilic polyethylene oxide chain. Both Triton X-100 and Triton WR-1339 have been identified as distinct forms of Triton that can be used to induce hyperlipidemia (HL).

Triton wr-1339 Induced Hyperlipidemia

Administration of Triton WR-1339, dissolved in normal saline at a 4% concentration and given

intravenously at a dose of 400 mg/kg, effectively induces hyperlipidemia. In treated rats, this results in a marked elevation of serum lipids, with triglycerides increasing nearly sevenfold, total cholesterol about fivefold, and LDL cholesterol approximately fourfold.

Glucocorticoids Induced Hyperlipidemia

Glucocorticoids, which bind to receptors in most vertebrate cells, regulate immune responses by reducing inflammation. They enhance anti-inflammatory protein production in the nucleus and inhibit pro-inflammatory proteins in the cytoplasm. Low-dose triamcinolone (0.5 mg/kg for 5 days) doubles triglyceride and VLDL levels without affecting HDL or total cholesterol. In contrast, high-dose treatment lowers VLDL and triglycerides but doubles HDL and cholesterol levels.

MEDICINAL PLANTS TO TREAT HYPERLIPEDEMIA

Cynara Cardunculus

In the early 1900s, researchers in France identified artichoke leaves as effective stimulants for both liver function and bile production. These leaves were used as a diuretic to enhance kidney function and as a bile stimulant to increase bile flow from the liver. Italian researchers later recommended cynarin, a key compound in artichokes, to stimulate liver activity and bile production, as well as manage high cholesterol levels. While artichoke leaves are known to promote bile production and may help alleviate dyspepsia, there is limited evidence supporting their direct treatment of these digestive issues. However, animal studies suggest that artichoke leaves can reduce cholesterol production in hepatocytes and safeguard the liver from harm caused by toxic substances.

Medicago Sativa

In traditional medicine, Medicago sativa is used as a natural remedy, known for its antidiabetic, antihyperlipidemic, and immune-boosting effects. It also treats conditions such as irregular menstruation, digestive issues, kidney and urinary conditions, skin burns, and inflammation of the arteries. The plant is prized for its high content of beta-carotene and vitamins B, C, E, and K. Research has demonstrated that Medicago sativa seeds can lower blood cholesterol levels in laboratory animals. A study in which monkeys were given these seeds for a year showed no side effects and a marked decrease in cholesterol levels in the bloodstream [9].

Achyranthes Aspera

In Siddha medicine, Achyranthes aspera is used to treat conditions such as leucorrhoea, haemorrhoids, obesity, oligomenorrhoea, and gastric ulcers. A paste of its leaf juice is applied to syphilitic ulcers, and the herb helps regulate menstruation. The root is also used for dental caries. Research indicates that an alcoholic extract of Achyranthes aspera, at 100 mg/kg, significantly lowers serum cholesterol and phospholipid levels in normal rats over 30 days.

Bauhinia Purpurea

In Siddha medicine, the herb Bauhinia purpurea is utilized to treat skin diseases, alleviate constipation, address eye disorders, and manage menorrhagia. Additionally, a preparation known as Mantharai ver chooranam, made from the root, is used to reduce excess body mass. The ethanol extract of unripe pods and leaves of Bauhinia purpurea has been assessed for its anti-hyperlipidemic effects in rat models with cholesterol-induced hyperlipidemia. The ethanol extract of both unripe pods and leaves was administered daily at a dose of 300 mg/kg orally for one month. At the end of the study, changes in body mass and serum lipid levels were evaluated. The results indicated a notable decrease in body mass, along with reductions in overall cholesterol, fats, LDL, and VLDL levels.

Garcinia cambogia In Siddha medicine, Garcinia cambogia is primarily used to address obesity and as an ingredient in cooking. The fruit contains L-hydroxy citric acid, which has lipid-lowering properties and is therefore utilized in managing obesity. Crude ethanolicextracts from Garcinia cambogia seeds have demonstrated a dose-dependent decreased in plasma levels of VLDL and an increase in chylomicrons in adult male rats. The extract effectively minimized weight gain, visceral fat buildup, blood and liver lipid levels, along with plasma insulin and leptin levels in a high-fat diet (HFD)-induced obesity mouse model [10].

Allium Sativum

This study found that extracts from the aerial parts of garlic (Allium sativum), typically discarded as waste, significantly improved lipid profiles and liver function in hyperlipidemic mice. Among the tested extracts, the n-butanol extract (NBE) and petroleum ether extract (PEE) were the most effective. They notably reduced total cholesterol (TC), triglycerides (TG), LDL cholesterol, and liver enzymes (ALT, AST), while increasing HDL cholesterol and liver antioxidant activity (SOD), and lowering oxidative stress markers like malondialdehyde (MDA). NBE was rich in flavonoids (e.g., kaempferol, quercetin), organic acids, and vitamins such as riboflavin—compounds known for their antioxidant, anti-inflammatory, and lipid-lowering effects. PEE contained polyunsaturated fatty acids (PUFAs) and phytosterols, which enhance lipid metabolism and block cholesterol absorption. Overall, these findings highlight the potential of garlic's aerial parts as a valuable source of bioactive compounds for managing hyperlipidemia [11].

Fenugreek

Fenugreek (Trigonella foenum-graecum) has shown significant lipid-lowering effects, especially in newly diagnosed type II diabetics. In a study, patients taking 25 mg of fenugreek seed powder twice daily for

one month saw notable improvements: total cholesterol dropped by 13.6%, triglycerides by 23.5%, LDL-C by 23.4%, and HDL-C increased by 21.7%, all statistically significant. These benefits are attributed to fenugreek's high content of fiber, saponins, and estrogen-like compounds, which enhance bile acid and cholesterol excretion, reduce reabsorption, and slow glucose and fat absorption—supporting its use as a natural therapy for hyperlipidemia [12].

Artemisia Iwayomogi Kitamura & Curcuma Longa

The herbal combination of *Artemisia iwayomogi* and *Curcuma longa* (ACE) has shown strong potential in treating hyperlipidemia, particularly in apoE-deficient mice fed a high-fat diet. ACE significantly reduced elevated total cholesterol, LDL, triglycerides, and the TC/HDL ratio, while also lowering oxidative stress and inflammation. Its benefits stem from anti-inflammatory and antioxidant effects, including reduced cytokine and ROS levels and restored liver antioxidant enzymes. ACE also modulated lipid metabolism genes by downregulating those for lipid synthesis (SREBP-1c, FAS, SCD-1) and upregulating genes for fatty acid oxidation (PPAR-α, CPT-1), helping to normalize lipid balance and prevent atherosclerosis [13].

Ginger

Ginger (Zingiber officinale) significantly reduces hyperlipidemia, a key risk factor for coronary artery disease. In rats fed a high-fat diet (HFD), those treated with ginger powder (500 mg/kg) showed marked reductions in total cholesterol (30.6%), triglycerides (37.9%), and LDL cholesterol (43.3%) compared to untreated controls. While HDL levels did not increase significantly, they showed a positive trend. Ginger's lipid-lowering effects are linked to multiple mechanisms: inhibition of HMG-CoA reductase (similar to statins), increased cholesterol conversion to bile acids via 7-alpha-hydroxylase, and reduced intestinal cholesterol absorption. These actions are supported by its bioactive compounds—gingerol, shogaol, and zingerone—which also provide antioxidant and anti-inflammatory benefits [14].

Flax seeds

A study on Wistar albino rats demonstrated that flaxseed (Linum usitatissimum) effectively reduces hyperlipidemia and early-stage atherosclerosis. Rats fed a high-cholesterol diet (HCD) and supplemented with flaxseed (7.5 g/kg/day for 90 days) showed significantly lower levels of total cholesterol, triglycerides, LDL-C, and VLDL-C compared to the HCD-only group. Although HDL-C levels improved, the change was not statistically significant. Flaxseed's lipid-lowering effects are attributed to its high content of alpha-linolenic acid (omega-3), soluble fiber, and lignans such as secoisolariciresinol diglucoside (SDG). These compounds reduce cholesterol absorption, enhance bile acid excretion, and inhibit liver lipid synthesis. Additionally, flaxseed boosted antioxidant enzyme activity (SOD, catalase, GPx, GR, and GST), reducing oxidative stress and inflammation—key factors in lipid peroxidation and vascular damage. Histological findings supported these results, showing reduced fatty liver changes and prevention of early atherosclerotic lesions in the aorta, including foam cell formation and intimal thickening. Notably, flaxseed also promoted endothelial cell proliferation and new vascular channel formation, indicating protective cardiovascular effects [15].

Caralluma edulis & Verbena Officinalis

This study evaluated the anti-hyperlipidemic effects of Caralluma edulis and Verbena officinalis in mice with high-fat diet-induced hyperlipidemia. Both plant extracts, administered at 500 and 1000 mg/kg, significantly reduced total cholesterol, triglycerides, LDL, and VLDL levels while increasing HDL levels, with effects comparable to atorvastatin. Phytochemical analysis revealed the presence of flavonoids, phenols, saponins, and other bioactive compounds known for their antioxidant properties. Histological examination showed reduced liver fat accumulation, especially with Verbena officinalis, which also preserved normal liver architecture. While Caralluma edulis additionally reduced body weight, Verbena officinalis did not. These findings suggest both plants have promising lipid-lowering and hepatoprotective potential, warranting further investigation into their active constituents and mechanisms of action [16].

Hibiscus Sabdariffa L

This study on Hibiscus sabdariffa L. leaves demonstrates its significant hypolipidemic activity in cholesterol-induced hyperlipidemic Wistar rats. The ethanolic extract of the leaves (HSEE) was tested at doses of 100, 200, and 300 mg/kg for four weeks. The extract at 200 mg/kg and 300 mg/kg showed a dose-dependent reduction in key lipid parameters: total cholesterol (by 18.5% and 22%), triglycerides (by 15.6% and 20.6%), LDL (by 24% and 30%), and VLDL (by 15.5% and 20.5%), with no significant effect on HDL levels. These effects were significant ($p < 0.05$) and the 300 mg/kg dose was nearly as effective as the standard drug atorvastatin (10 mg/kg). The hypolipidemic action is attributed to the presence of bioactive compounds such as hibiscus acid, flavonoids (quercetin, kaempferol), sitosterol-β-D-galactoside, and Pectin. These constituents may act by inhibiting lipid absorption, reducing lipogenesis, and enhancing antioxidant defenses, suggesting that Hibiscus sabdariffa leaves could serve as a promising natural agent in managing hyperlipidemia [17].

Camellia Sinensis

The study on Camellia sinensis (green tea) reveals its significant role in managing hyperlipidemia. The aqueous extract of Camellia sinensis leaves was tested in Triton WR-1339-induced hyperlipidemic albino rats at a dose of 200 mg/kg. The treatment produced a substantial reduction in total cholesterol (by ~65%), LDL

(by ~80%), VLDL, triglycerides, and phospholipids, and notably increased HDL levels both in serum and liver tissues. The antihyperlipidemic effect is attributed to the plant's rich content of polyphenolic compounds, especially flavonoids, tannins, proanthocyanidins, and saponins, which exhibit antioxidant and lipid-lowering properties. These compounds help modulate lipid metabolism by enhancing LDL catabolism through hepatic receptors and inhibiting lipid absorption and biosynthesis [18].

CONCLUSION

Hyperlipidemia remains a significant contributor to the global burden of cardiovascular diseases. Understanding its classification, pathophysiology, and experimental modeling is vital for developing targeted treatments. This review emphasizes the efficacy of various herbal remedies that demonstrate significant lipid-lowering, antioxidant, and hepatoprotective effects in preclinical models. Plants such as *Allium sativum*, *Garcinia cambogia*, *Hibiscus sabdariffa*, and *Camellia sinensis* have shown promising results comparable to conventional lipid-lowering agents. Integrating these natural therapies into clinical practice may offer safer and more holistic management strategies. However, further clinical studies are needed to validate these findings and elucidate precise mechanisms of action.

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