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## An Overview of Hyperlipidemia

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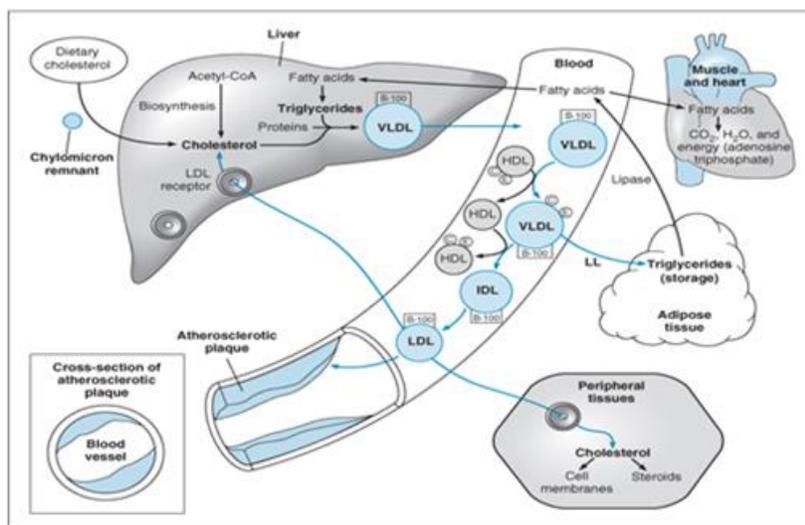
### ABSTRACT

Hyperlipidemia is a broad term which is also called hyperlipoproteinemia, is a common disorder in developed countries and is the major cause for coronary heart diseases. It results from abnormalities in lipid metabolism or plasma lipid transport or as a disorder in the synthesis and degradation of plasma lipoproteins. The term “dyslipidemia” now a days is increasingly being used to describe abnormal changes in lipid profile, replacing the old term hyperlipidemia. Hyperlipidemia means abnormal increase in fat levels of blood. These fats include cholesterol and triglycerides. These are important for our body to function, but when their levels are high they, can cause heart disorders. Hyperlipidemia is manifested as hypercholesterolemia and hypertriglyceridemia. Hypercholesterolemia is the most common hyperlipidemia. The lipids that are involved in hypercholesterolemia are cholesterol, an essential component of cell membrane and a precursor of steroid hormone synthesis and triglycerides are important energy source, they are transported in blood as lipoproteins. The consequence of hyperlipidemia is to cause atherosclerosis, leading to the risk of coronary heart diseases and strokes. Thus the risk of heart diseases depends on factors such as levels of cholesterol, blood vessels and blood circulation.

**Keywords:** Hyperlipidemia, Hypercholesterolemia, Lipid profile, Diabetes.

### INTRODUCTION

Hyperlipidemia is a heterogeneous disorder commonly characterized by elevated serum total cholesterol, low density and very low-density lipoprotein cholesterol, triglycerides, and decreased high-density lipoprotein levels [1]. Liver synthesizes two-third of the total cholesterol in the body. The rate limiting enzyme is 3-hydroxy-3-methylglutaryl (HMG)-Co A reductase and provides feedback regulation by controlling the cholesterol concentrations in cells. Treatment of hyperlipidemia involves diet control, exercise, and the use of lipid-lowering diets and drugs [2]. The most commonly employed drugs for treatment of hyperlipidemia include hydroxymethylglutarate coenzyme A (HMG-CoA) reductase inhibitors, also called as statins. Other drugs employed for treatment of hyperlipidemia include bile acid sequestrants (anion-exchange resins) such as cholestyramine and colestipol; fibrates such as clofibrate, gemfibrozil, fenofibrate, ciprofibrate, bezafibrate; niacin; cholesterol absorption inhibitors such as ezetimibe; and omega-3-fatty acids [3,4] (Figure 1).



**Figure 1:** Cross section of atherosclerotic plaque.

### **SCHEME OF CHOLESTEROL FORMATION**

Hypolipidemic agents, or antihyperlipidemic agents, are a diverse group of pharmaceuticals that are used in the treatment of high levels of fats (lipids), such as cholesterol, in the blood (hyperlipidemia). They are called lipid-lowering drugs.

Hyperlipidemia is typically asymptomatic and is frequently detected during routine screening. Hyperlipidemia often results from delayed or defective clearance, or overproduction of VLDL by the liver, which is subsequently transformed into LDL. Hypercholesterolemia involves defective hepatic and nonhepatic LDL receptors. Excess intake of saturated fats increases the liver's production of VLDL and triglycerides *via* a molecular mechanism involving protein activators [5]. Saturated fats found in animal products, such as meat, whole milk dairy products (milk, cream, cheese), and butter, and tropical oils (palm, palm kernel, and coconut).

High concentrations of total and LDL cholesterol and low levels of high density lipoprotein (HDL) cholesterol, predicts cardiovascular risk in both men and women. High triglyceride levels have been associated with greater risk in women only. The risk of cardiovascular disease increases by an average of 2%, for each corresponding 1% rise in total cholesterol. Adolescents with high TC or LDL may have a genetic disorder of lipid metabolism such as familial hypercholesterolemia or familial combined hypercholesterolemia. Those with homozygous forms of these disorders can experience myocardial infarction or other events during childhood or early adolescence. Familial hypercholesterolemia is often diagnosed in adolescence and is characterized by high LDL levels that can be refractory to dietary treatment. These patients can present clinically with xanthomas or xanthelasma— cholesterol deposits under the skin on the hands, elbows, knees, heel or eyelids [6].

There are several secondary causes of abnormal lipids that may occur in adolescence. Children, infants and geriatrics have been shown to have higher levels of cholesterol, especially those who exhibit poor catch-up growth [7]. The starved state that occurs in anorexia and the use of anabolic steroids are both associated with abnormal lipids. Certain medications for acne, seizure disorders, immunosuppression, and contraception can adversely affect lipids as can a high carbohydrate diet or a ketogenic diet sometimes prescribed for refractory epilepsy. Adolescents with a history of a transplant also tend to have an abnormal lipoprotein panel despite a TC in the normal range [8]. Although high cholesterol can be inherited, it's more often the result of unhealthy lifestyle choices [9].

### **LIPID PROFILE**

Lipid profiles are commonly used in the routine evaluation of cardiovascular risk, given the high correlations of hypercholesterolemia and hypertriglyceridemia and cardiovascular risk. A standard lipid profile includes determination of serum or plasma total cholesterol (TC),

high-density lipoprotein-associated cholesterol (HDL-C), low-density lipoprotein-associated cholesterol (LDL-C), and total triglycerides (TG). In many laboratories, LDL-C is calculated from the Friedewald equation ( $LDL-C = TC - HDL-C - TG/5$ ), which correlates well with gold standard determinations of LDL-C by ultracentrifugation when TG are less than 400 mg/dL. This calculation is based on the following assumptions: The vast majority of cholesterol is transported by LDL, HDL, and very low-density lipoproteins (VLDL) in fasting plasma [10] most TG are associated with VLDL in fasting specimens; and the normal ratio of TG to cholesterol in VLDL is. However, these assumptions are not always true.

### ***Fasting versus non fasting lipid profiles***

Cardiovascular risk assessment and monitoring of lipid-lowering therapy based on LDL-C and TG should be performed in specimens collected after a period of 12–14 hr of overnight fasting without any dietary intake except for water and medication because TG (predominantly in the form of chylomicrons) remain elevated after meals for several hours. In nonfasting specimens and other conditions with elevated TG, non-VLDL particles carrying TG change the ratio of TG to cholesterol and invalidate the Friedewald formula, usually leading to overestimation of LDL-C. In these cases, a direct assay for LDL-C can be used, although direct LDL-C results in nonfasting specimens may not be as predictive of cardiovascular events in women. Recently, emphasis has been placed on non-HDL-C ( $TC - HDL-C$ ) as a secondary target of therapy because it includes not only LDL-C but also other atherogenic lipoproteins, such as intermediate-density lipoproteins. Importantly, TC, HDL-C, and non-HDL-C can be accurately determined in nonfasting specimens. Some studies also indicate that nonfasting TG, which peak approximately 4 or 5 hr postprandially, may be better predictors of cardiovascular events, but the exact cutoffs and postprandial intervals have not been well-defined. Together with determination of apolipoproteins A1 and B100, it is possible that in the future, nonfasting lipid profiles will be acceptable for cardiovascular risk assessment.

### **OTHER PRE-ANALYTICAL CONSIDERATIONS**

Although cholesterol and triglycerides are considered small molecules, they circulate as components of large lipoprotein complexes; therefore, plasma levels are subject to dilutional variation in protein concentration induced by a change to the upright position (~7%-16% increase) and prolonged tourniquet application (5%-20% higher). Pregnancy is associated with a decrease of cholesterol and triglycerides in the first trimester but a gradual increase afterwards. The nephrotic syndrome and hypothyroidism are associated with elevated TC, predominantly due to LDL-C. Stress, inflammation, infections, and other acute disorders are associated with increases in triglycerides and cholesterol, whereas acute coronary syndromes result in decreases of TC and LDL-C and 20%-30% increases in TG within 24-48 hr, leading to the recommendation that lipid profiles should be avoided after 24 hr of hospitalization or initiation of an acute event. A study using fasting samples indicated that the magnitude of change after an acute myocardial infarction is small, with a mean decrease in TC and LDL-C of 2% at 24 hr of admission and a subsequent increase of 6% in the next 2 days, whereas TG and HDL-C showed even smaller changes. However, other studies showed larger reductions in TC, non-HDL-C, direct LDL-C, TG, and apolipoprotein A1 in the first 18-42 hr postadmission, whereas apolipoprotein B100 and HDL-C did not change significantly. The best recommendation is to perform lipid profiles at admission of patients with suspected acute coronary syndromes [9] (Figure 2).

<b>Lipid profile Test - Reference Values</b>				
	Unit	Optimal	Intermediate	High
<b>Total Cholesterol (calculated)</b>	mg/dl	<200	200-239	>239
	mmol/L	<5.2	5.3-6.2	>6.2
<b>LDL Cholesterol (calculated)</b>	mg/dl	<130	130-159	>159
	mmol/L	<3.36	3.36 - 4.11	>4.11
<b>HDL Cholesterol</b>	mg/dl	>60	40 - 60	<40
	mmol/L	>1.55	1.03 - 1.55	<1.03
<b>Triglycerides</b>	mg/dl	<150	150 - 199	>199
	mmol/L	<1.69	1.69 - 2.25	>2.25
<b>Non-HDL-C (calculated)</b>	mg/dl	<130	130 - 159	>159
	mmol/L	<3.3	3.4 - 4.1	>4.1
<b>TG to HDL Ratio (calculated)</b>	mg/dl	<3	3.1 - 3.8	>3.8
	mmol/L	<1.33	1.34 - 1.68	>1.68

**Figure 2:** Lipid profile test.

**Normal lipid profile**

**Vascular complications:** While hyperlipidemia runs in some families, still there are choices you can make every day to favorably influence your blood cholesterol, and ultimately, your heart health.

**Body weight:** If your Body Mass Index (BMI) is greater than 30%, then it's highly likely that you are at increased risk of developing high cholesterol, as well as cardiovascular heart disease.

**Physical activity:** A lack of physical activity causes many health-related issues. Since exercise helps to increase the high density lipoprotein (HDL) cholesterol that is heart-protective, and helps to lower low density lipoproteins (LDL), which is one type of blood cholesterol that increases your heart disease risk.

**Diet:** Food choices can improve blood cholesterol or can cause harmful plaque. Heart healthy diet is one that is high in dietary fiber and heart healthy fats. Important dietary fiber food includes vegetables, beans, legumes, fruits and whole grains. These foods mainly made with white flour cause a rise in triglycerides, another type of blood cholesterol that is directly related to heart disease. The 2nd category that affects heart health risk is saturated fats. The types of artery-clogging foods to skip include red meat, butter, and baked/prepared foods containing trans fats or lard.

**Smoking:** Smoke damages blood vessels, and damaged blood vessels are more prone to accumulation of fatty deposits there by narrowing the blood vessels and potentially causing cardiovascular issues. Also, lower HDL Cholesterol.

**High blood pressure:** This also goes along with heart health. High blood pressure can damage the walls of your blood vessels, making them more likely to accumulate fatty deposits that can lead to cardiovascular problems.

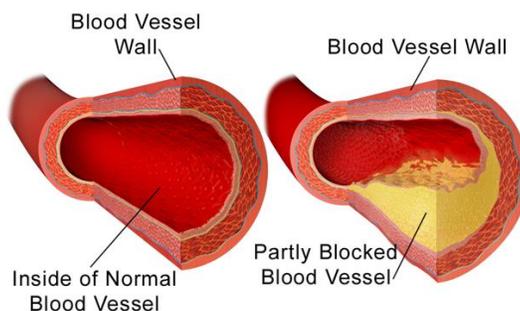
**Type 2 diabetes:** Routinely high blood glucose levels damage blood vessels, damaged blood vessels are more likely to develop plaque the fatty deposits. Also, type 2 diabetes raises your LDL cholesterol levels (the bad cholesterol) and lowers your HDL cholesterol levels (the good cholesterol) [11].

**Atherosclerosis:** Hardening and narrowing of the arteries silently and slowly blocks arteries, putting blood flow at risk. It's the usual cause of heart attacks, strokes, and peripheral vascular disease.

**Coronary heart disease:** The main risk associated with high cholesterol is coronary heart disease (CHD). High cholesterol builds up on the walls of arteries. Over time, this buildup is known as atherosclerosis. This condition causes arteries to become narrowed, and the narrowed blood vessels reduce blood flow to the heart. This can result in angina (chest pain) a condition in which not enough blood flows to the heart, or a heart attack in cases when a blood vessel is blocked completely and the heart muscle begins to die.

**Stroke:** A stroke occurs when a blood vessel that carries oxygen and nutrients to the brain becomes blocked or bursts. A stroke can result if the blood supply to the brain is reduced. When stroke occurs, part of the brain cannot get the blood and oxygen it needs, so it starts to die.

**Peripheral arterial disease:** High cholesterol also has been linked to peripheral arterial disease (PAD), which refers to diseases of blood vessels that are outside the heart and brain. In PAD, fatty deposits build up along artery walls and affect blood circulation, mainly in arteries leading to the legs and feet. The arteries of the kidney can also be affected (Figure 3).



**Figure 3:** Theory of blood vessel.

## NEEDS FOR ANTI HYPOLIPIDEMIC AGENTS

### *Lipid-lowering drugs*

Such as statins and niacin, that reduces cholesterol and triglycerides in blood.

### *Antithrombotic drugs*

such as warfarin, low-dose aspirin, clopidogrel and prasugrel which, by thinning the blood, prevent further plaque accumulation, mitigate injuries from blood clots caused by atherosclerosis and treat heart disease.

FDA approved new anti-hyperlipidemia drugs [12,13].

Food and drug administration (FDA), U.S. approved some new drugs for the treatment of hyperlipidemia associated diseases. They are:

I. Livalo (Pitavastatin)

II. Juxtapid (Lomitapide)

III. Kynamro (Mipomersen)

IV. Vascepa (Icosapent ethyl)

## LDL-LOWERING STRATEGIES

The first lifestyle change recommended by the NCEP is to reduce intake of saturated fat and cholesterol. Saturated fat should be less than 7% of total calories per day, and dietary cholesterol should be less than 200 mg per day.

Saturated fat is found mostly in foods that come from animals: meat, poultry fat, lard, butter, cheese, and other dairy products. It is also found in foods from tropical plants such as coconut oil, palm oil, and cocoa butter. Saturated fat is solid at room temperature. Such as the fatty strips in bacon or the fat found under chicken skin.

Reducing the amount of saturated fat, trans fat, and cholesterol in the diet does not mean that it is an extremely low-fat diet. On the contrary, for persons with the metabolic syndrome, lipid disorders, and diabetes, total fat should make up 30% to 35% of total calories. In addition, saturated fat and trans fat should be replaced with monounsaturated fat (up to 20% of calories) and polyunsaturated fat (up to 10% of total calories). Evidence suggests that replacing saturated fat with unsaturated fat is more effective in lowering the risk of coronary heart disease than reducing total fat consumption.

The two types of unsaturated fat in the diet monounsaturated and polyunsaturated are found in a variety of foods. Monounsaturated fat is found in avocados, almonds, olives, peanuts, and olive, peanut, and canola oils. Polyunsaturated fat can be divided into two groups: omega-6 polyunsaturated fat, which is found mainly in seeds and vegetable oils such as corn and soy oils, and omega-3 polyunsaturated fat, which is found in fish, flaxseeds, and walnuts. Consuming omega-3 fatty acids from fish and plant sources may lower risk of coronary heart disease. Replacing saturated fat with monounsaturated or polyunsaturated fat can be as simple as putting avocado on sandwich instead of cheese, having nuts for a snack instead of chips or baked goods, and using salad dressing made with olive oil or canola oil instead of using a creamy salad dressing.

## GOING EVEN LOWER

By reducing intake of saturated fat, cholesterol, and trans fat does not help in maintenance of LDL. For maintenance of LDL first strategy is to include plant or animal sterols in our diet. These are derived from natural plant components such as soy. They reduce the absorption of cholesterol in the digestive tract, resulting in lower total and LDL cholesterol levels. They can be found in some soft margarine products, and orange juice is preferred then margarine products. Plant sterol intake of 2-3 grams per day has been found to lower LDL cholesterol by 6% to 15%.

A second strategy is to add more foods high in soluble fiber in the diet. A total fiber intake of 20-30 grams per day is part of the diet recommended by the NCEP. By increasing soluble fiber intake by only 5 to 10 grams per day, most people can reduce their LDL cholesterol by about 5%. Managing weight and engaging in regular physical activity are also fundamental components of the NCEP-recommended changes. Strategies must be developed for weight maintenance, and anticipate high-risk occasions for possible weight gain (for example,

times of significant stress). If overweight, start with a weight-loss goal of 10% through dietary changes, portion control, and daily physical activity. Any increase in physical activity should be taken into consideration with current heart health, age, and physician's assessment

### **TRIGLYCERIDES**

Triglycerides in the blood come from the foods we eat. Calories that are not immediately used by the body are converted into triglycerides and transported to the fat cells to be stored. Like LDL cholesterol, excess triglycerides in the blood are linked to a higher risk of coronary artery disease.

If triglycerides remain elevated LDL cholesterol is reduced. Overweight and lack of physical activity are strong contributors to high triglycerides. Hence it is necessary to start a program of weight loss and daily exercise that could lead to an improved triglyceride level.

In addition to reducing calorie intake and exercising, there are some other changes that can lower triglyceride level. First, intake of refined or processed carbohydrates, such as candy, table sugar, white flour, and baked goods made with white flour must be reduced as these foods can promote an increase in triglycerides. Excessive intake of these foods may also increase blood glucose, and it is very common to see high blood triglycerides when diabetes is not well controlled.

Foods rich in monounsaturated and polyunsaturated fats must be taken instead of those that contain refined sugar. Substituting fish rich in omega-3 fatty acids (found in fatty fish such as salmon, sardines, and tuna) may also lower triglycerides. Omega-3 fish oil capsules may be therapeutic in some cases, but over-the-counter fish oil supplements have been found to contain varying amounts of fish oil, so only prescription supplements are recommended at this time. High doses can cause excessive bleeding in some people. Even small amounts of alcohol can increase triglycerides. So if high triglycerides are a problem it would be a good idea to cut down on alcohol as much as possible.

### **HDL CHOLESTEROL**

HDL cholesterol is called "good" cholesterol because higher levels protect against coronary heart disease by carrying cholesterol away from the arteries and back to the liver where it is disposed off. A low level of HDL cholesterol is associated with an increased risk of coronary heart disease.

When triglyceride levels are elevated, HDL cholesterol levels begin to fall. Any precautions taken to improve triglycerides may also lead to an improvement in HDL Cholesterol level. However, low HDL cholesterol may also be observed in the absence of high triglycerides. This is called isolated low HDL cholesterol and is attributed to many of the same factors that may promote high triglycerides, including overweight and obesity, physical inactivity, cigarette smoking, very high carbohydrate intake (more than 60% of calories), Type 2 diabetes, and genetic factors.

Losing weight or maintaining a healthy weight, being physically active and not smoking can increase HDL cholesterol. In addition, maintaining a low intake of saturated fat and trans fat, avoiding processed carbohydrates, and consuming an adequate amount of monounsaturated and polyunsaturated fats all promote a healthy level of HDL cholesterol.

### **NEED FOR HERBAL THERAPY**

Plants have been used for medicinal purposes since ancient times. Primitive men observed and appreciated the great diversity of plants available to them. Plants provide food, clothing, shelter, and medicine. Much of the medicinal use of plants seems to be developed through observations of wild animals, and by trial and error. As time went on, each tribe added the medicinal power of herbs in their area to its knowledge base. They methodically collected information on herbs and developed well-defined herbal pharmacopoeias [14,15].

Many drugs listed as conventional medications were originally derived from plants. Salicylic acid, a precursor of aspirin, was originally derived from white willow bark and the meadowsweet plant. Cinchona bark is the source of malaria-fighting quinine. The opium poppy yields morphine, codeine and paregoric, a remedy for diarrhoea. Laudanum, a tincture of the opium poppy, was the favored tranquilizer in Victorian times. Even today, morphine the most important alkaloid of the opium poppy remains the standard against which new synthetic pain relievers is measured. Similarly, tetrahydrocannabinol (THC), the component of *Cannabis sativa* responsible for the CNS effect, has also been found to reduce nausea associated with cancer chemotherapy. Another therapeutic area where natural products have had a major impact on longevity and quality of life is in the treatment of cancer. In fact, most of the major anticancer drugs are natural products either

from plants or micro-organisms. Examples include important anticancer drugs such as Bleomycin, Doxorubicin, Vincristine, Vinblastine, and now the recent addition of Paclitaxel (Taxol), Irinotecan (a camptothecin derivative) and Etoposide and Teniposide (Podophyllotoxin derivatives).

Some of the most exciting natural products discovered in the recent years are the cholesterol-lowering agents derived from fungi.

These drugs act by inhibiting 3-hydroxy-3-methylglutaryl co-enzyme A reductase (HMG-CoA reductase), an enzyme in the biosynthesis of cholesterol. The first of the HMG-CoA reductase inhibitors were isolated from *Penicillium sp* [4]. Substances derived from the plants remain the basis for a large proportion of the commercial medications used today for the treatment of heart diseases, high blood pressure, pain, asthma, cancer and other problems [16]. Plants contain a number of metabolites, only a small percentage has been investigated phytochemically and some fractions of them have been submitted for biological screening. The process of evaluation of plants for various pharmacological activities is a much time consuming process. So also is the process of isolation of active components present in the plants. Hence, it requires multi-disciplinary collaboration. [17,18] Natural products have proven to be the richest source of medicinal compounds. Screening the marine flora and fauna, soil samples, fungi and microbes is conducted either to discover a new drug or a lead structure. A lead is a prototype compound for a given biological activity. For example for anti-tumour activity, a natural product lead structure is subjected to chemical modification or scaffolds to arrive at the therapeutically important molecular fragment, the pharmacophore. Only a few natural products are directly used as drugs, but in many cases the chemical scaffolds of the lead structure provides a more potent synthetic or semi-synthetic analogs.

Globally, there has been an unparalleled growth in the plant-derived medicinally useful formulations, drugs and health-care products. It has a market covering more than 60% products derived from plant origin. India exhibits remarkable outlook in modern medicines that are based on natural products besides traditional system of Indian medicines. Almost, 70% of the modern medicines in India are derived from natural products.

Medicinal plants play a central role not only as traditional medicines but also as trade commodities, meeting the demand of distant markets. Ironically, India has a very small share (1.6%) of this ever-growing global market.

To compete with the growing market, there is urgency to expeditiously utilize and scientifically validate more medicinally useful plants while conserving these species, which seems a difficult task ahead [7].

### MEDICINAL AND AROMATIC PLANTS

India has 2.4% of world's area with 8% of global bio-diversity. It is one of the 12 mega-diversity hot-spot regions of the world. Other countries being Brazil, Colombia, China, South Africa, Mexico, Venezuela, Indonesia, Ecuador, Peru, USA and Bolivia. Across the country, the forests of India are estimated to harbour 90% of India's medicinal plant diversity. Only about 10% of the known medicinal plants of India are restricted to non-forest habitats. The estimated numbers of plant species and those used for medicinal purpose vary. According to Schippmann, (2002), one fifth of all the plants found in India are used for medicinal purpose. The world average stands at 12.5% while India has 20% plant species of medicinal value.

But according to Hamilton (2003) [19], India has about 44% of flora, which is used medicinally. Although it is difficult to estimate the number of medicinal and aromatic plants present worldwide, the fact remains true that India with its rich biodiversity ranks first. The existence of traditional medicine depends on plant species diversity and the related knowledge of their use as herbal medicine. Both plant species and traditional knowledge are important to the herbal medicine trade and the pharmaceutical industry where plants provide raw materials and the traditional prerequisite information [20].

India has one of the richest plant medical traditions in the world. It is the tradition that is of remarkable contemporary relevance for ensuring health security to the teeming millions. There are estimated to be around 25,000 effective plant-based formulations, used in folk medicine and are known to rural communities in India. There are over 1.5 million practitioners of traditional medicinal system using medicinal plants in preventive, promotional and curative applications. It is estimated that there are over 7800 medicinal drug-manufacturing units in India, which consume about 2000 tons of herbs annually two of the largest users of medicinal plants are China and India [21].

### HERBAL MEDICINE

Herb has various meanings, but in simplest form, it refers to “crude drugs of vegetable origin utilized for the treatment of diseases, often of a chronic nature, or to attain or maintain a condition of improved health”. Herbal medicine, sometimes referred to as Herbalism or Botanical Medicine, is the use of herbs for their therapeutic or medicinal value. An herb is a plant or plant part valued for its medicinal, aromatic or savory qualities. Herbs plants produce and contain a variety of chemical substances that act upon the body. Herbal preparations called “phytopharmaceuticals”, “phytomedicinal” or “phytomedicine”, are preparations made from different parts of herbs or plants. They come in different formulations and dosage forms including tablets, capsules, elixir, powder, extract, tincture, cream and parenteral preparations. A single isolate or active principle derived from plants such as digoxin or reserpine tablet is not considered herbal medicine [22-26].

### HERBAL REMEDIES

The effectiveness of herbal remedies, their easy availability, low cost and comparatively being devoid of serum toxic effects popularized them and due to their eco-friendly-nature and literature evidences it is inferred plant-based medicines could be beneficial in the management of Heart diseases, cancer and diabetes. This prompted us to select and evaluate a herbal supplement in reducing hypolipidemia a major cause leading to heart diseases. Development of such scientifically validate herbal remedy could contribute significantly to the human society in preventing heart diseases. A literature survey was conducted with a view to select common plants with hypolipidemic potential and to validate scientifically the hypolipidemic activity.

### CONCLUSION

These are important for our body to function, but when their levels are high they, can cause heart disorders. Hyperlipidemia is manifested as hypercholesterolemia and hypertriglycerlomia. Hypercholesterolemia is the most common hyperlipidemia. The lipids that are involved in hypercholesterolemia are cholesterol, an essential component of cell membrane and a precursor of steroid hormone synthesis and triglycerides are important energy source, they are transported in blood as lipoproteins. Treatment of hyperlipidemia involves diet control, exercise, and the use of lipid-lowering diets and drugs. The most commonly employed drugs for treatment of hyperlipidemia include hydroxymethylglutarate coenzyme A (HMG-CoA) reductase inhibitors, also called as statins. Other drugs employed for treatment of hyperlipidemia include bile acid sequestrants (anion-exchange resins) such as cholestyramine and colestipol; fibrates such as clofibrate, gemfibrozil, fenofibrate, ciprofibrate, bezafibrate; niacin; cholesterol absorption inhibitors such as ezetimibe; and omega-3-fatty acids.

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### REFERENCES

- [1] [Akhtar MS, Iqbal J., \*J Ethnopharmacol\*, 1991, 31\(1\):49-57.](#)
- [2] [Vogel HG, Vogel WH, Scholkens BA, et al., \*Springer\*, 2002, 2: 1103-1107.](#)
- [3] [Schurr PE, Schultz JR, Parkinson TM, \*Lipids\*, 1971, 7\(1\):68-73](#)
- [4] [Buccolo G, David H, \*Clinical Chemistry\*, 1973, 19\(5\): 476-482](#)
- [5] [Werner M, Gabrielson DG, Eastman, \*J Clinical Chemistry\*, 1981, 7\(2\): 268-271](#)
- [6] [Allain CC, Poon LS, Chan CS, et al., \*Clinical Chemistry\*, 1974, 20\(4\): 470-475](#)
- [7] [Kumar V, Khan MM, Khanna AK, et al., \*Evid Based Complement Alternat Med\*, 2010,7\(3\):317-322](#)
- [8] [Health line \*Hyperlipidemia\*. 2016.](#)
- [9] [biochemistrygenetics-and-molecular-biology/lipid-profile. 2018.](#)
- [10] [Walker KA \*Cholesterol risk factors\*, 2018](#)
- [11] [High cholesterol diseases: conditions and outcomes 2020.](#)
- [12] [Scott G, Carver M, Mckeen M, et al \*Endocrinology\*, 1997, 138\(4\)](#)
- [13] [Leenen R, Vander kooy K, Meyboom S, et al. \*J Lipid Res\*, 1993,34\(12\):2183](#)

- [14] [American Association of Neuropathic Physicians. 2020.](#)
- [15] [Petrovska B., \*Pharmacogn Rev\*, 2012, 6\(11\): 1-5.](#)
- [16] [Ebadi.M., \*CRC Press\*, 2002.](#)
- [17] [Petrovska B., \*Pharmacogn Rev\*, 2012, 6\(11\):1-5.](#)
- [18] [Manjulatha K., \*Thesis: Gulbarga University\*, 2006, 2.](#)
- [19] [Gilani A.H. Shaheen f, zaman m, \*phytotherapy res\*. 1999, 13 \(8\): 665-669.](#)
- [20] [Ram Gopal M.D., \*Thesis: Gulbarga University\*, 2006.](#)
- [21] [\*Schippmann U, Leaman DJ, Cunningham AB., Biodiversity and the Ecosystem Approach in Agriculture, Forestry and Fisheries\*, 2002.](#)
- [22] [Tabuti, JRS, Lye KA, Dhillon SS., \*J Ethnopharmacology\*, 2003, 88 \(1\): 19-44.](#)
- [23] [Ramakrishnappa K., \*Biodiversity and the Ecosystem Approach in Agriculture, Forestry and Fisheries\*, 2002.](#)
- [24] [Masood E., \*Nature\*, 1997, 385\(6617\):570.](#)
- [25] [Wang ZG, Ren J., \*Trends Pharmacol Sci\*, 2002, 23 \(8\): 347-348.](#)
- [26] [Thot.TT., \*Ind J Pharma Edu\*. 1998, 32 \(2\): 104-106.](#)