Journal of Applied Pharmaceutical Research ISSN No. 2348 – 0335 www.japtronline.com

ANTIHYPERLIPIDEMIC POTENTIAL OF HERBALS

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One of the most widespread diseases in the world is Coronary Heart Disease (CHD). It is also one of the most preventable. This review explores the management of CHD through changes in dietary modifications, lifestyle, and the use of dietary supplements and botanicals.

Key words: Coronary Heart Disease, antihyperlipidemic

INTRODUCTION

Hyperlipidemia is concerned as the cause for coronary heart diseases [1] and atherosclerosis which results from slow but sure deposition of lipids in arteries is a chief cause of mortality worldwide. [2]Though varieties of synthetic drugs are used in the treatment, still the searches are on for better medicaments especially from the plant kingdom. Many medicinal plants have been studied in this context. [1] WHO has predicted that, by 2030, cardiovascular diseases will affect approximately 23.6 million people around the world [3]. It has been reported that hypercholesterolemia contributes to 45% of heart attacks in Western Europe and 35% of heart attacks in Central and Eastern Europe. [4] The risk of heart attack is three times higher in those with hypercholesterolemia, compared to those who have normal blood lipid profiles. The WHO delineated that unhealthy diets, such as those high in fat, salt, and free sugar and low in complex carbohydrates, fruits, and vegetables, direct to increased risk of cardiovascular diseases. [5] Recent modalities for lowering blood cholesterol levels involve behavior modification, regular exercise, weight management, dietary management, and drug therapy. [6] effective pharmacological agents that reduce cholesterol levels are available in market may be useful in treatment of high cholesterol but they are so expensive and are known to have severe side effects. [7] Epidemiological studies have demonstrated a positive significant relationship between obesity, sex, smoking, hypertension and plasma cholesterol concentration with coronary artery diseases (CAD). [8] Diet is one of the most important factors underlying atherosclerosis. High-cholesterol diets enhance atherosclerosis and vegetarian diets are known to slow down the process. [1] Loss of elasticity of the inner arterial wall plus thickening and hardening of arteries are among the hallmarks of atherosclerosis. This is a progressive condition which starts from childhood and its clinical manifestations face in middle and old age. Three principal biological processes are involved in Atherosclerosis is namely (i) aggregation of smooth muscle cells, macrophages and T lymphocytes, (ii) formation of connective matrix by smooth muscle cells and (iii) accumulation, in cells and in the connective tissue surrounding the cells of lipids which are basically in the form of cholesterol esters and free cholesterol. [9] New medical research evaluating the relationship between plaque formation in arthrosclerosis & high cholesterol food. This section will focus primarily on the use of therapeutic lifestyle changes and the current state of evidence for diet, botanicals, and nutritional supplements that are generally used for the prevention and treatment of coronary heart disease (CHD).

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Lipids

Lipids are a heterogeneous group of substances that are distinguished by their low solubility in water and their high solubility in nonpolar (organic) solvents. They are essential as energy stores and respiratory substrates, as structural components of cells, as vitamins, as hormones, for the protection of internal organs, for heat conservation, for digestion, and for lactation. [10]

The major lipids found in the blood stream are

- Cholesterol,
- Cholesterol ester,
- Triglycerides
- Phospholipids

Cholesterol

Cholesterol is a high molecular weight sterol. Cholesterol is not a fat. (It is fat soluble.) Cholesterol is an important component of the cell membranes, including organelle membranes inside the cell. Cholesterol biosynthesis is a multistep process which takes place mainly in the liver and the intestine, and involves more than twenty enzyme-catalyzed reactions for converting acetate into cholesterol (Figure 1). The rate limiting reaction in cholesterol biosynthesis is catalyzed by 3-hydroxy-3- methylglutaryl-CoA (HMGCoA) reductase, and it is this enzymatic step which has been used as pharmacological target of statin treatments (see below).

Cholesterol is used by the body as raw material for the healing process. This is the reason the injured areas in the arteries (as in atherosclerosis) have cholesterol along with several other components (such as calcium and collagen) in the 'scar' tissue that is formed to heal the 'wound'. Cholesterol move through the blood with transporters, so-called lipoproteins like LDL and HDL. To perform its many important functions in the body, cholesterol is transported from the liver to the cells, tissues, and glands on low density lipoprotein carriers (LDL's). Reverse cholesterol transport (from the cells and tissues to the liver) is via high density lipoprotein carriers (HDL's). [11]

Low-Density Lipoprotein (LDL). This lipoprotein maintains a large amount of cholesterol. The protein layer allows the tissues to use this cholesterol, LDL receptors on these tissues that make this interaction possible. In the tissues such as those of the liver and the inner layer of the arterial wall, cholesterol is taken away from low-density lipoproteins. Free radicals in the body are very reactive and oxidative compounds that can oxidize low-density lipoprotein cholesterol and help atherosclerotic plaque to form in the arteries so LDL is considered as Bad cholesterol. [12]

High-Density Lipoprotein (HDL). The liver also produces another type of lipoprotein, named high-density lipoprotein. High-density lipoprotein collects the surplus cholesterol that cholesterol metabolizing cells cannot utilize. Lecithin-cholesterol acyltransferase (LCAT) is an enzyme that is responsible for transporting surplus cholesterol back to HDL molecules. Unused cholesterol from arteries, liver, and other tissues is absorbed by HDL cholesterol. There is evidence that even some oxidized LDL can be removed by the LCAT and HDL cholesterol. So HDL is considered as good cholesterol. [13] HDL circulates in the body and collects the cholesterol from tissues, it becomes mature and goes back to the liver. There, it is identified by its lipoprotein covering and is lodged in the liver's cholesterol pool.

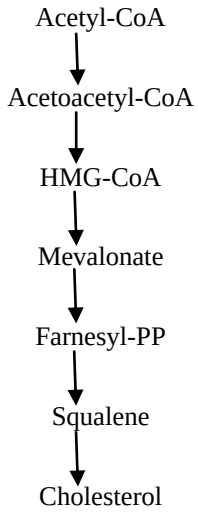

Fig.1 Process of cholesterol synthesis

Cholesterol Esters

Cholesterol is transported throughout the body in the form of cholesterol esters. Excess cholesterol is also stored intracellulary as cholesterol esters. The enzyme cholesterol esterase controls the hydrolysis of these stored cholesterol esters yielding bioavailable cholesterol and fatty acids. Cholesterol esterase hydrolyzes longchain and unsaturated fatty acid esters at a greater rate than short chain saturated fatty acids. Cholesterol esterase also contributes to the incorporation of cholesterol into mixed micelles and aids in the transport of free cholesterol into the enterocyte.[14]

Triglycerides

The fat in the food we eat is mostly in the form of triglycerides. Triglycerides are the main type of fat transported by our body. After a meal, your body digests the fats of food and repackages the fat as triglycerides, which are released into our bloodstream. The blood carries the triglycerides throughout our body to give energy or to be stored as fat. Our liver also produces triglycerides and changes some into cholesterol. Our liver can change any source of excess calories into triglycerides. [15] But when your body makes too many triglycerides, the result can contribute to hyperlipidemia, a clinical word that describes the presence of too much "bad" fat in our bloodstream. [16]

Phospholipids

Phospholipids (PLs) are amphiphilic lipids found in all plant and animal cell membranes, arranged as lipid bilayers. The cell membranes are basically containing glycerol-phospholipids (GPLs), which consist of fatty acids (FAs) esterified to a glycerol backbone, a phosphate group and a hydrophilic residue (e.g. choline, resulting in phosphatidylcholine or lecithin). The backbone of a PL can also be the long chain amino-alcohol sphingosin instead of glycerol. These PL are classified as sphingophospholipids, the most representative being sphingomyelin, found in high quantities in brain and neural tissue, consisting of sphingosin esterified to one FA and phosphocholine.

Besides glycerol phospholipids (GPLs) and sphingomyelin (SPM), biological membranes are also made up of glycolipids and cholesterol, as well as of integral and peripheral membrane proteins..[17] PLs extracted from food products (e.g. soybeans, egg yolk, milk, or marine organisms like fish, roe or krill) are defined as "dietary GPLs". They can be ingested either with normal diet or as supplements. In a normal diet, the daily intake of PC is approximately 2-8 grams. [18]

Dyslipidemia

Dyslipidemia refers to a disruption of lipid metabolism with exceeding serum levels of cholesterol (TC), triglyceride (TG), low-density lipoprotein-cholesterol (LDL-C), and/or lower level of high-density lipoprotein-cholesterol (HDL-C). Serum levels of lipids and lipoprotein lipids are among the most potent and best substantiated risk factors for atherosclerotic diseases, particularly coronary heart disease (CHD). [24]

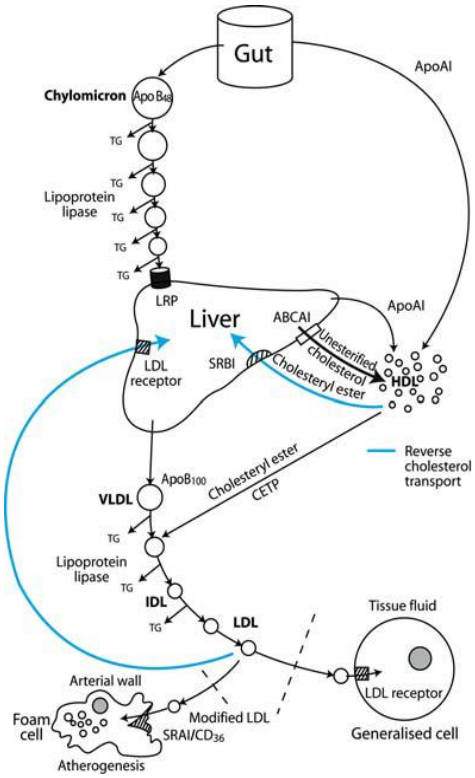

Figure 2. Lipoprotein metabolism[19]

Abbreviations: apoB48, apolipoprotein B48; apo A1, apolipoprotein A1; apoB100, apolipoprotein B100; TG, Triglyceride; LRP, LDL receptor-like protein; ABCA1, ATP-binding cassette A1; SRA1, SRB1, CD36, 3 members of the scavenger receptor family; CETP, cholesteryl ester transfer protein; VLDL, very low density lipoprotein; IDL, intermediate density lipoprotein; LDL, low density lipoprotein; and HDL, high density lipoprotein

Blocking Absorption of Dietary Cholesterol

Dietary cholesterol is obtained from foods derived from animal sources that are rich in fat content. A healthy adult only needs to ingest about 30% of the daily cholesterol requirement. Obtaining more than this

amount from dietary cholesterol can lead to increased cholesterol levels and serious health risks. Dietary cholesterol is absorbed within the lumen of the small intestine. Bile salts produced from cholesterol in the liver interact with phospholipids to produce a biliary micelle that is transported via bile into the lumen. Dietary cholesterol in the lumen is easily incorporated into these micelles and together with the already present biliary cholesterol can now be absorbed into the enterocytes that make up the walls of the lumen. The micelles enter the cell by a channel know as Niemann-Pick C1 like 1 protein (NPC1L1). Once in the cells the cholesterol can either be pumped back out into the lumen or it can be esterified for transport within chylomicrons. Preventing the absorption of this dietary cholesterol has become a key area in cholesterol related research. Plant sterols and stanols have been shown to be effective inhibitors of cholesterol absorption. Ingested as part of a normal diet, plant sterols and stanols are very similar in structure to cholesterol. They actually have a stronger binding affinity than cholesterol to the biliary micelles that aid in absorption. Because of this the sterols and stanols can displace cholesterol from the micelles thus preventing its absorption.

Recently, inhibitors that block the absorption of the biliary micelles into the enterocytes have also been used to block the uptake of dietary cholesterol. [25]

Dietary Intervention

Heart healthy diets should be considered one of the primary therapeutic lifestyle interventions for patients with any level of risk for CHD. Prevention always trumps treatment. [26],[27] LDL cholesterol level can be reduced by limiting consumption of saturated fats (chees, red meat and whole-fat dairy products), increasing physical exercise and weight reduction. Increased consumption of monounsaturated fats (olive oil, fish fats and nuts) has been shown to reduce the LDL cholesterol & increases HDL cholesterol thus lowering CAD risk.The quest for finding the new safe & effective drug for dyslipidemia is going to be a continueous process among the scientific fraternity.herbs have been used as food and medicine for centuries.researchers showed intresthas focused on various herbs that possess antihyperlipidemic activity that may be useful adjuncts in helping reduce the risk of cardiovascular disease.A herbal diet with exercise is an effective prescription for anyone with elevated risk of cardiovascular disease.In addition there are a few herbs available that provide some help for persons with hyperlipidemia or other cardiovascular diseases.[28]

Some pharmacological and clinical studies reported on ayurvedic and other herbs are described below:

1) Sphaeranthus indicus [29]

Tanpe et al studied the antihyperlipidemic property of aqueous extract of Sphaeranthus indicus in rats) (3oomg/kg/day,i.p) against dexamethasone (10mg/kg/day,s.c)induced changes in lipid profile in rat. Treatment with dexamethasone for eight days showed marked increase in the level of serum TC,TG and LDL,VLDL level where as HDL remain unchanged. Treatment with S. indicus (Asteraceae) showed significant decrease in serum TC,TG and LDL,VLDL & there was no significant changes in the level of HDL. Atherogenic index also reduced significantly after S. indicus treatment.

2) Panax notoginseng[30]

Maintenance of normal lipid levels has implicated the involvement of genes induced by liver X receptor alpha (LXR alpha) and Farnesoid X receptor (FXR).This study was designed to evaluate the hypolipidemic effects of n-butanol extract (NE3) of Panax notoginseng root on lipid homeostasis and investigate the possible mechanisms in animal experiments. In the transactivation assays,NE3 was identified as a dual FXR/LXR alpha agonist. Subsequently, Sprague-Dawley male rats on a high –fat diet were treated orally with NE3 or vehicle alone.as expected,the concentration of serum TC,TG and LDL-C in rats treated with various concentrations of NE3 showed significant (Pless than 0.01) and dose –dependent decrease, respectively, accompanied with a significant (Pless than 0.01) and

dose –dependent decrease in concentration of hepatic TC& TG. Express-level analysis indicated that both LXR alpha target genes including ABCA1,ABCG5,ABCG8and FXR target genes including ApoCII and SHP were significantly induced by NE (Pless than 0.01). Interestingly LDLR mRNA level was significantly higher by NE3 (Pless than 0.01) accompanied with decreased expression levels of CYP7A1, ApoCIII and SREBPIc genes (Pless than 0.01).Based on these results, it can be concluded that NE3 as a dual FXR/LXRalphaagonist largely prevented the accumulation of abnormal lipid in the hyperlipidemic rats.

3) Panicum miliare [31]

Hypolipidemic potential of samai (Panicum miliare , Gramineae) was evaluated on selected fifty cardiovascular patients by Radha R. et al. for a period of sixty days (25 control; 25 supplemented with samai). Lipid profiles were analyzed before and after supplementation period for both control and experimental groups. It was noted that all the lipid values with the exception of HDL-cholesterol had reduced after supplementation with Samai and the reductions were statistically significant at 1 percent level. There was a mild increase in the HDL cholesterol level, which was desirable.

4) Murraya koenigii [32]

The powdered leaf of curry *Murraya koenigii* , Rutaceae was experimented in normal and alloxan induced diabetic rats to explore its cholesterol,triglyceride,blood urea nitrogen and alanine transaminase level.the result indicated significant reduction in the serum cholesterol and serum triglyceride level in the treated group**.**

5) Camellia sinensis [33]

To evaluate the antihyperlipidemic effect of **Camellia sinensis** (CS) **leaves** Theaceae was administered at a dose of 200 microg/Kg (p.o.)to Triton induced hyperlipidemic rats by saravana kumar et al . Fenofibrate was used as reference standard. CS shows a significant decrease in the levels of serum cholesterol, phospholipid, triglyceride, LDL,VLDL and significant increase in the level of serum HDL at the dose of 200 microg/Kg (p.o.) against Triton induced hyperlipidemic rats. Aqueous extract fraction decreased serum level of total cholesterol by 69.72 percent and increased the serum HDL cholesterpol level by 24.11 perccent. The decrease in LDL cholesterol level by aqueous extract was 30 percent.

6) Straw Mushroom [34]

A study was conducted by Darwin christdhas Henry et al to investigate the effect of paddy **straw Mushroom** Volvariella volvacea at the rate of 2.5 and 5.0 percent dosage on cholesterol accumulation in blood and liver of weaning male white Albino wistar rats was assessed. The serum total cholesterol level of rats fed with 5 percent decreased when compared to control over a period of 30,60 and 90 days. The reduction of cholesterol was due to the decreased cholesterol content in LDL. There was no significant difference found in serum HDL concentration. It is suggested that prolonged exposure to V. volvacea administrations reduced lipid and lipoprotein concentration significantly in rats.

7) Ginko [35]

It has been found in one of the animal studies that Maidenhair tree, Ginko biloba Linn. Extract (EGB761) inhibits beta amyloid production by lowering the free cholesterol.

8) Gardenia jasminoides [36]

The pancreatic lipase inhibitors were isolated from from the fructus of Gardenia jasminoides (Rubiaceae) and their antihyperlipidemic activities were measured by Lee I. A. et al. The water extract of Gardenia fruits (GF) inhibited pancreatic lipase activity. Crocetin and crocin were isolated from GI water extract as inhibitors of pancreatic lipase with an Ic50 value of 2.1 and 2.6mg/ml (triolein as a substrate) Crocetin and crocin significantly inhibited the increase of serum TG level in

corn oil feeding induced triglyceridemic mice,as well as that of serum triglyceride and total LDL cholesterol levels in triton WR-1339-induced hyperlipidemic mice. These compounds also showed hypolipidemic activity in hyperlipidemic mice induced by high cholesterol high fat or high carbohydrate diets for 5 weeks.The results suggesting that the hypolipidemic activity of GF and its components crosin may be due to the inhibition of pancreatic lipase and crosin and its metabolite.crocetin can improve hyperlipidemia.

9) Roselle[37]

The effect of administrating of dried calyx extracts of roselle at 500 and 1000 mg/kg together in continuous cholesterol feeding to hyperholesterolemic rats for 6 weeks significantly decreased serum cholesterol level by 22 percent & 26 percent respectively serum triglycerides level by 33 percent &28 percent respectively, serum triglycerides level by 22 percent & 32 percent respectively six-week treatment with 250,500 &1000 mg/kg of the extracts significantly decreased thiobarbituric acid reactive substances (TBARs) formation while the formation of conjugated dienes during the oxidation of LDL induced by CuSO4 was reduced. but not significantly different. These lines of evidence suggest that the aqueous extracts from the dried calyx Hibiscus sabdariffa L.(roselle) possess both antioxidant effects against LDL oxidation and hypolipidemic effects in vivo.

10) Bottle gourd [38]

In one of the intresting studies on juice of Bottle gourd (Lagenaria siceraria, Cucurbitaceae) was obtained by crushing the the fresh fruits in the juicer and was subsequently dried in the oven at 40-50˚C. The parent dried juice extract was than fractionated by using the solvent according to polarity in ascending order. Each fraction was dried in oven at 40-50˚C. Thin layer chromatography (TLC) used active fraction obtained by column chromatography for further isolation. The solvent system developed on trial and error basis was n-butanol : methanol: water(6:2:2). Four spots were obtained and were named as LSN-I, LSN-II, LSN-III & LSN-IV. Isolated spots were collected by using preparative TLC the isolated compounds were tested for antihyperlipidemic activity & compounds LSN-I, LSN-II, LSN-III has shown significant results. The study exhibited that elevated level of blood cholesterol, triglycerides, LDL, were significantly reduced & decreased HDL was significantly increased by the administration of fraction of Lagenaria siceraria fruits juice.

11) Bellis perennis [39]

The methanolic extract and its saponin fraction (methanol-eluted fraction) of the flowers of Bellis perennis (Asteraceae) were found to suppress serum triglyceride elevation in olive oil –treated mice. From the saponin fraction, seven new triterpene saponins, perennisosides I(1), II (2), III (3), IV (4), V (5), VI (6) & VII (7), were isolated together with four known saponins, bellidioside A (8), asterbatanoside D (9), bernardioside B2 (10), and bellissaponin BS6 (11).The stereostructures of 1-7 were elucidated on the basis of chemical and spectroscopic evidence. Among these saponins, perennisosides I(1)and II(2) showed inhibitory effects on serum triglyceride elevation at doses of 25-50 mg/kg, po.

12) Terminalia arjuna [40]

The study was carried out to investigate the antihyperlipidemic action of Terminalia arjuna in fructose rich-high fat diet (HFD) fed hamsters. Bark powder of Terminalia arjuna was extracted with petroleum ether (fraction-A), solvent ether (fraction-B) and alcohol (fraction-C) and fed orally at a dose of 250 mg/kg to HFD fed hamsters. At the end of ten days experiment, blood was withdrawn from each hamster to evaluate total cholesterol (TC) serum cholesterol, triglyceride (TG), HDL, glucose (Glu), glycerol (Gly) and free fatty acids (FFA) in plasma by standard spectrophotometric methods. Feeding with fructose rich HFD produced marked dyslipidemia in hamsters as the level of TC, TG, HD-TC, Glu, Gly and FFA was found

increased by 300,106,30,54,87 and 30 percent respectively. Treatment with fractions to dyaslipidemic hamsters caused reversal in the level of plasma lipid and glucose. The efficacy of different fraction of Terminalia arjuna was found, fraction-C more than fraction –B more than fraction-A.

13) Helicteres isora [41]

The aqueous extract of the bark of Helicteres isora (sterculiaceae), at 100 and 200 mg/kg dose for 21 days lowered serum and tissue cholesterol, phospholipids,free fatty acids and triglycerides in STZ diabetic rats. Administration of this bark extract of Helicteres isora in addition to that, significant (p less than 0.05) decrease in LDL whereas significant increase (p less than 0.05) HDL were observed in STZ diabetic rats, which were normalized after 21 days of bark extract treatment. The bark extract at a dose of 200 mg/kg b.w. showed much significant hypolipidaemic effect than at dose of 100 mg/kg b.w.

14) Petrocarpus marsupium [42]

Petrocarpus marsupium (Fabaceae) is used by traditional medicine practitioner of India for its hypolipidemic & antihyperlipidemic action. The alcoholic extract of plant was used for assessing its activity in albino rats when compared to standard drugs. The activity was assessed by studying the lipid profile in serum of the control and drug treated animals. The results lend support to the traditional use of Petrocarpus marsupium in the treatment of Hyperlipidemia.

15) Karela [43]

Effects of three Japanese cultivars (Koimdori, Powerful-Reishi and Hyakunari of bitter melon (Momordica charania linn.) and those of methanol fraction extract of CV, Koimidori on serum and liver triacyglycerides were studied in rats by senanayake and others. Feeding of diets containing either bitter melon or various fractions isolated by organic solvents caused no adverse effects on food intake or growth of rats. when the effect of three different varieties of bitter melon was compared,the cv. koimidori was found to be the most effective in lowering hepatic triglyceride levels as compared to the other two cultivars, suggesting a variety- dependent difference in their activity. Furthermore, the active component(s) responsible for the liver triglyceride lowering activity of cv. koimidori was assumed to be concentrated in the methanol fraction, but not in other fraction, but not in other fractions such as the n-hexane, the acetone or the residual fraction. The triglyceride lowerining activity was furthermore confirmed by the dose-dependent reduction of hepatic triglyceride, the lowest level in rats fed 3.0% supplementation was observed. In these experiments, the effects on serum lipids were marginal. The results of these studies clearly show that bitter melon, especially cv. Koimidori exhibits a potent liver triglyceride lowering activity.

16) Curcuma longa [44]

Dou X. and others studied the effect of curcumin, an active component of the rhizome of Curcuma longa (Zinziberaceae) were examined on LDL-R expression and its molecular mechanism in HepG2 cells. curcumin increased LDL-R expression (mRNA and protein) and the resultant uptake of Dil-LDL in a dose and time dependent manner. Using a GFP receptor system in a tranfected HepG2 /SRE-GFP cell line curcumin was found to activate the sterol regulatory element of the LDL-R promoter. In HepG2/Insig2 cells, curcumin reversed the inhibition of LDL-R expression induced by Insig2 over expression. The data demonstrate that curcumin increases LDL-R protein expression and uptake activity via the SREBPs pathway. These findings suggested the hypocholesterolemic and antiatherosclerotic effect of curcumin.

17) Soyabean [45] [46]

Convincing evidence shows that soyabean (Glycine max Merrill) protein intake has beneficial effects on lipid changes, but it is unclear which components of soy protein are responsible. Researchers have conducted a meta analysis to identify and quantify the effect of soy

protein with isoflavones intact was associated with significant decrease in serum total cholesterol (by 0.22 mmol/L,or 3.77%),LDL cholesterol (by 0.21 mmol/L,or 5.25%) and triacylglycerol (by 0.10 mmol/L,or 7.27%) and significant increases in serum HDL-cholesterol (by 0.04 mmol/L,or 3.03%).the reduction in total and LDL cholesterol were larger in man then in women. Initial total cholesterol concentration had a powerful effect on changes in total and HDL-cholesterol, especially in subjects with hypercholesterolaemia. Studies with intake>80mg showed better effects on the lipid profile. The strongest lowering effects of soy protein containing isoflavones on total cholesterol, LDL cholesterol and triacylglycerol occurred within the short initial period of intervention, whereas improvements in HDL—cholesterol were only observed in HDL-cholesterol were only observed in studies of >12 week duration. Tables containing extracted soy isoflavones did not have a significant effect on total cholesterol reduction. Thus, Soy protein containing isoflavones significantly reduce serum total cholesterol, LDL-cholesterol and triacylglycerol and increased HDL cholesterol,but the changes are related to the level and duration of intake and the sex and initial serum lipid concentrations of the subjects. One more study on a peptide which was isolated by HPLC from the pepsin hydrolysate of 11S-globulin isolated from the defatted soy (Glycine species, leguminosae). Its molecular weight (755.2 Da) and amino –acid sequence (Ile-Ala-Val-Pro-Gly-Glu-Val-Ala) were established. The hypocholesterolemic activity was determined by analysis of bile-acid and the percent inhibition of 3- hydroxyl-3-methyglutaryl coenzyme A reductase in vitro. The lowering of the cholesterol content is explained by bile acids bound to hydrolysate peptides shielding them from reabsorption & stimulating the transformation of cholesterol in blood plasma.

18) Cucurbita pepo.[47]

Dietary plants and herbal preparations have been traditionally used as medicine in developing countries and obtained a resurgence of use in resurgence of use in the various countries. Research carried out in last few decades has validated several such claims of use of traditional medicinal medicine plants. Popularity of pumpkin in various systems of traditional medicine for several aliments with antihypercholesterolemic activity focused the investigators attention on this plant. Considerable evidence from several epidemiological studies concerning bioactivities leads have stimulated and clinical trials designed to test this pharmacological actions.

Conclusion

In ayurveda, lipids can be equated with Medodhatu. Therefore, any drugs that will be working against kapha dosha or Medodhatu might act as lipid lowering agents. Also, there is a description of group of drugs such as Medoghna (the one which decreases the Medodhatu), Lekhana (the one which helps in reducing the body mass), Kaphaghna gana(the one which acts against which might demonstrate the antilipidemic activity. The commonest them will be the contents of Triphala i.e. three myrobalans, viz. Amalki (Emblica officinalis Gaertn. Syn. Phyllanthus emblica Linn.), Haritaki(Terminalia chebula Retz.) and Bibhitaki (Terminalia bellerica Roxb.) and Trimada i.e. Vidang (Embelia ribes), Musta (cyperus rotundus Linn.), Chitrak (Plumbago zeylanica Linnn.) and many more herbs given in a dosage form of Medohar guggul pills with a base of Guggul. Thus, we need to focus on our rich heritage of Ayurvedic Medicine and should adopt the accepted Golden triangle approach (a research collaboration of Traditional Medicine of India i.e. Ayurveda with Modern Basic Sciences and Modern Medical Science, so that we can approach more real solution on this front of lipid lowering drugs.

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Received 31st December 2013
Revised 11th January 2014
Accepted 19th January 2014
J. App. Pharm. Res., 2 (1); 2014: 7 – 17