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## A COMPREHENSIVE REVIEW ON PLANTS WITH POTENTIAL ANTIHYPERLIPIDEMIC ACTIVITY

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### ABSTRACT

Hyperlipidaemia is considered as one of the major risk factors contributing to the development coronary heart diseases. This is a medical condition where abnormally high levels of plasma lipids including triglycerides, cholesterol, cholesterol esters, and phospholipids are found in the blood. Anti-hyperlipidemic drugs such as statins and fibrates are extensively used in the treatment of elevated plasma lipids. But these drugs are cursed with side effects. In the last few years, there has been a rapid growth in the use of medicinal plant and gaining popularity on both developing and developed countries as it possesses minimal side effects. Medicinal plants carry various bioactive secondary metabolites and these metabolites are responsible for showing different properties useful for medicinal purposes. This mini-review tries to describe few plant species having potential anti-hyperlipidemic property.

**KEYWORDS:** Hyperlipidemia, Herbal medicine, Heart diseases.

### INTRODUCTION

Hyperlipidemia is also called hyperlipoproteinemia is the major cause of coronary heart disease. It occurs due to abnormalities in lipid metabolism or plasma lipid transport or disorder in the synthesis & degradation of plasma lipoproteins. High plasma level of cholesterol along with generation of reactive oxygen species (ROS) play key role in the development of coronary artery diseases (CAD) and atherosclerosis. Oxidative stress is currently suggested a mechanism underlying hypercholesterolemia (*Gamal A.et al., 2014*). Epidemiologic data reported that around 12 million people die of cardiovascular diseases and cerebral poplexy each year all over world. Therefore, it is very important to pay attention to early stage prevention and control of hyperlipidemia in a comprehensive way. The generally suggested measure for the treatment of hyperlipidemia associated diseases is through restriction of caloric intake or increased caloric expenditure and/or use of lipid lowering drugs. Based on the data analysis of BMI (Body Mass Index) in 1997, the World Health Organization (WHO) described hyperlipidemia as an epidemic hazard worldwide. Causes of hyperlipidemia involve genes, metabolism, diet, physical activity, lifestyle modification & socio-cultural environments (*Neeraj Kumar Sharma et al.,2013*). Hyperlipidemia also

caused due to medical diseases are diabetes, kidney disease, pregnancy, and an under active thyroid gland (*Khaled Mohamed Mohamed Koriem et al.,2014*). Many allopathic anti-hyperlipidemic drugs are available in the market but the side effects like hyperuricemia, diarrhea, myositis, hepatotoxicity, etc. were reported.

Although statins were found to be effective in the lowering of serum low-density lipoprotein (LDL) as well as cholesterol level, they have been found to cause many side effect. As they are basically enzyme inhibitors, so it is likely that they may be inhibiting other critical enzymes in the body. Moreover, statins are ingested on a long term, so there may be a risk of chronic toxic effects such as carcinogenicity, teratogenicity and mutagenicity over a life time of use (*Dhaliya Salam. A et al., 2013*). In the last few decades there has been tremendous growth in the field of herbal medicine due to lesser side effects (*Ankur Rohilla et al., 2012*). Herbs have been used as food & for medicinal purposes for centuries. Research interest has been focused on various herbs possessing hypolipidemic property to reduce risk of cardio vascular diseases (*SagarPetal.,2012*).

### Prevalence

In most of the developed countries, hyperlipidemia and atherosclerosis are the leading cause of cardiac illness and death (*A. Misra et al., 2004*). According to WHO, nearly 60% of Indians will contribute the world cardiac demographic class amounting about 100 million patients. Moreover, a WHO survey by the year reveals that India is predicted to have large number of mortalities due to CAD (*P.K. Patel et al., 2009*). Hyperlipidemia is an important risk factor in the initiation and progression of atherosclerotic impasse. Therefore, prime consideration in the therapy for hyperlipidemia and atherosclerosis is to attenuate the elevated blood serum/plasma levels of lipids (*R.P. Dikshit et al., 2009*). In these complex disease groups hypolipidemic drugs are of crucial importance for preventing further disease risk and ameliorating the quality of everyday life (*B.K. Mayes et al., 2003*).

### Pathophysiology of Hyperlipidemia

The pathophysiology of hyperlipidemia can be studied under two headings, i.e., primary hyperlipidemia and secondary hyperlipidemia.

1. Primary Hyperlipidemia: The pathophysiology of primary hyperlipidemia involve that the idiopathic

hyperchylomicronemia defect in lipid metabolism leads to hypertriglyceridemia and hyperchylomicronemia which is caused by a defect in lipoprotein lipase activity or the absence of the surface apoprotein (*Bassam Abdul et al., 2013*).

2. Secondary Hyperlipidemia: In secondary hyperlipidemia, the postprandial absorption of chylomicrons from the gastrointestinal tract occurs 30-60 min after ingestion of a meal containing fat that may increase serum triglycerides for 3-10 hrs. The diabetes mellitus patients have been noted to possess low LPL activity which further caused high synthesis of VLDL cholesterol by the liver ultimately leading to hyperlipidemia (*Jama2001*).

### Mechanism of Lipid Transport

Lipid transported around the body as lipoproteins because these are insoluble in water. There are two sources from which lipids originate: endogenous lipids, synthesized in the liver, and exogenous lipids, ingested and processed in the intestine.

Near about 7% of body's cholesterol circulates in plasma in the form of low density lipoproteins (*K D Tripathi 1985*).

**Table 2.1: Classification of Hyperlipidemia (Sagar P. Mahamuni et al., 2012).**

Type	Total cholesterol	LDL cholesterol	Plasma TGs	Lipoprotein abnormality	Primary cause	Secondary cause
I	Elevated	Low or normal	Elevated	Excess chylomicrons	Lipoprotein lipase deficiency, apoC-II deficiency	Systemic lupus erythematosus
IIa	Elevated or normal	Elevated	Normal	Excess LDL	Familial hypercholesterolemia	Hypothyroidism
IIb	Elevated	Elevated	Elevated	Excess LDL and VLDL	Familial combined hyperlipidemia	Nephrotic syndrome, Diabetes, Anorexia nervosa
III	Elevated	Low or normal	Elevated	Excess chylomicron remnants and intermediate density lipoprotein	Familial type III Hyperlipoproteinemia	Hypothyroidism, diabetes, Obesity
IV	Elevated or normal	Normal	Elevated	Excess VLDL	Familial combined hyperlipidemia Familial Hypertriglyceridemia	Diabetes, chronic renal diseases
V	Elevated	Normal	Elevated	Excess chylomicrons and VLDL	Familial hypertriglyceridemia, apoC-II deficiency	Alcohol, diuretics, $\beta$ blockers, oral

apoC-II = apolipoprotein-C II; LDL = Low density lipoprotein ; TG = Triglyceride; VLDL = Very low density lipoprotein

### Signs and Symptoms of Hyperlipidemia

Hyperlipidemia usually has no noticeable symptoms and tends to be discovered during routine examination or evaluation for atherosclerotic cardiovascular disease (*Bhatnagar D et al., 2008; Grundy SM et al., 1998*).

Xanthoma, Xanthelasma of eyelid, Chest Pain, Abdominal Pain, Enlarged Spleen, Liver Enlarged, High cholesterol or triglyceride levels, Heart attacks, Higher rate of obesity and glucoseintolerance, Pimple like

lesions across body, Atheromatous plaques in the arteries, Arcus senilis, Xanthomata.

### Causes of Hyperlipidemia

The main cause of hyperlipidemia is changes in lifestyle habits including poor diet i.e. with a fat intake greater than 40% of total calories, saturated fat intake greater than 10% of total calories and cholesterol intake greater than 300 milligrams per day (*Dhaliya Salam et al., 2013*). In some degree of cases it occurs due to genetic factors, medical reasons or psychiatric illness. Recent increases of obesity including insufficient sleep, endocrine disruptors, Pregnancy at a later age, natural selection of higher BMI and assortative mating leading to increased concentration of obesity risk factors. The predisposing factors associated with hyperlipidemia constitute.

1. Elevated low density lipoprotein-cholesterol (LDL-C) levels and decreased high density lipoprotein-cholesterol (HDL-C) levels
2. Age (male > 45 years; female > 35 years)
3. Family history of premature death
4. Diet rich in saturated fats and cholesterol
5. Diabetes Mellitus
6. Hypertension
7. Hypothyroidism
8. Cigarette smoking and alcohol abuse
9. Physical inactivity
10. Obesity or overweight
11. Overactive adrenal gland
12. Increased levels of c-reactive proteins
13. Increased Lipoprotein (a) levels
14. Liver and kidney problems (*Keane WF et al., 1992*).

### Herbs used in the management of Hyperlipidemia

#### *Achyranthes aspera*



Fig. 2.8: Aerial part of *Achyranthes aspera*.

It belongs to the family *Amaranthaceae*. It is an annual or perennial herb. It is found throughout tropical Asia, Africa, Australia and America. The plant is widely used in traditional medicinal system of India and has been reported to possess hepatoprotective, anti-inflammatory, antitussive, antifungal and also used to check wounds healing, hypoglycemic, antipyretic and antibacterial properties. The alcoholic extract of this plant at 100mg/kg dose, lowered total serum cholesterol (TC) and phospholipid (PL), triglyceride (TG) and total lipid (TL) levels by 60, 51, 33 and 53 percent, respectively in triton-induced hyperlipidemic rats. Same dosage to

normal rats for 30 days lowered serum TC, PL, TG and TL by 56, 62, 68 and 67 %, respectively followed by significant reduction in the levels of hepatic lipids. This cholesterol lowering activity might be due to rapid excretion of bile acids causing low absorption of cholesterol (*Khanna A.K et al., 1992*). The methanolic leaf extract at 1g/kg showed significant lowering of serum lipids such as total cholesterol, triglyceride, HDL, LDL. (*Workineh Shibeshi et al., 2006*).

#### *Allium sativum*



Fig 2.9: *Allium sativum*.

It belongs to the family *Liliaceae*. It is a small herb. It is a native of central Asia and occurs all over India. S-allyl cysteine sulfoxide is the active principle found in this plant. It is considered a warm, bitter herb with particular effects on the Large Intestine, Spleen and Stomach meridians. It is used to lower blood pressure, to treat parasitic infections, food poisoning and tumors, and as a mild anticoagulant. It is traditionally contraindicated in patients with a yin deficiency and tuberculosis (*Bensky D et al., 1993*). It showed inhibitory effects on transaminases, alkaline phosphatase lipogenic enzymes and HMG CoA reductase and stimulatory effects on plasma lecithin cholesterol acyl transferase lipolytic enzymes and fecal excretion of sterols and bile acids. Further studies showed that the S-allyl cysteine sulfoxide treatment also reversed the lipid peroxidation and decreased reduced glutathione levels, superoxide dismutase and catalase activities in cholesterol fed rats (*Sheela CG et al., 2005*). Garlic protein (16% of diet) and garlic oil (100mg/kg/day) exhibited significant lipid lowering effects in rats fed with cholesterol diet. The hypolipidemic action is primarily due to a decrease in hepatic cholesterologenesis in the treated rats.

#### *Acorus calamus*



Fig 2.10: Rhizome of *Acorus calamus*.

It belongs to the family *Araceae*. It is a semi-aquatic, perennial, aromatic herb with creeping rhizomes. It is found throughout India in damp marshy places. In Ayurvedic system the *Acorus calamus* has been used as a magical root which cures asthma, fevers, bronchitis and all over it is a sedative. The paste of the *Acorus calamus* is applied externally on the inflamed joints, rheumatism and in rheumatic fever alleviates the pain and swelling. Administration through nasal route is salutary in headache, heaviness, epilepsy and hysteria (*Rupali Singh et al., 2011*). Investigations revealed that the presence of tannins in this plant is responsible for hypolipidemic activity. Tannins from this plant at 10mg/kg dose decreased the serum cholesterol and triglyceride level in rats fed with atherogenic diet. At a dose of 20mg/kg both triglycerides and serum cholesterol were brought back to the baseline. But HDL cholesterol levels were raised after treatment. Saponins at either of the doses (10mg/kg, p.o. and 20mg/kg p.o.) were unable to bring the serum cholesterol level and triglycerides back to the baseline value. However, a dose dependant effect was seen with respect to HDL cholesterol (*Snehalata et al., 1999*). Aqueous and hydro alcoholic extracts prepared from the roots of *A. calamus* showed hypolipidemic activity in rats fed with an atherogenic diet (*Reshma, P et al., 2011*).

#### *Cissus quadrangularis*



**Fig 2.11: Succulent vine of *Cissus quadrangularis*.**

It belongs to the family *Vitaceae*. It is a succulent vine. It occurs throughout India. It is useful in asthma. The stem is bitter, laxative, aphrodisiac, tonic analgesic. It uses to cure pile, chronic ulcer. *Cissus quadrangularis* is used for obesity, diabetes, a cluster of heart disease risk factors called metabolic syndrome (*Akshada Kakade et al., 2014*), and high cholesterol. Proprietary extract of this plant (CQR-300) at a dose of 300 mg daily and proprietary formulation containing CQR-300 (CORE) at a dose of 1028 mg daily showed significant ( $p < 0.001$ ) reductions in plasma TBARS and carbonyls. They also brought significant reductions in weight, body fat, total cholesterol, LDL-cholesterol, triglycerides, and fasting blood glucose levels over the respective study periods in a double-blind placebo controlled design, involving initially 168 overweight and obese persons. These changes were accompanied by a significant increase in HDL-cholesterol levels, plasma 5-HT, and creatinine (*Oben J et al., 2006*). The loss in weight in the above study was comparable to that observed with cissus studies, sibutramine for one year (*Smith IG et al., 2001*), and orlistat for 6 months or 1 year (*Kelly DE et al., 2002*).

2002). A reduction of fasting blood glucose levels as well as MDA levels have been previously reported to accompany weight loss in obese subjects (*Yesilbursa D et al., 2005*).

#### *Garcinia cambogia*



**Fig 2.12: Fruit of *Garcinia cambogia*.**

It belongs to the family *Guttiferae*. It is a small or medium-sized tree with a. It occurs in the evergreen and shola forests of Western Ghats in India. (-)-Hydroxycitric acid is the active ingredient of the fruit and the rind of this plant. The fruit juice possesses anti-scorbutic, anthelmintic and cardiotonic properties. Hence it finds application in the treatment of piles, dysentery, tumors, pains and heart complaints (*Verghese J, 1991*). The decoction of the fruit rind is given in rheumatism and bowel complaints. It competitively inhibits the extra mitochondrial enzyme adenosine triphosphate-citrate (pro-3S)-lyase (*Mahendran, P et al., 2000*). This enzyme ensures the supply of acetyl-CoA, which is used by acetyl-CoA carboxylase, the regulatory enzyme of lipogenesis in the liver. This is also important in the regulation of biosynthesis of endogenous lipids, levels of plasma lipoproteins VLDL (very-low-density lipoprotein), LDL (low-density lipoprotein) and HDL (high-density lipoprotein) (*Goto, Jr et al., 1987*). And the distribution of the lipid in extra hepatic tissues, especially adipocytes, the storage site of body fats. Crude ethanolic extracts of *G. cambogia* (bitter kola) seeds showed dose-dependent decrease in the plasma level of very-low-density lipoprotein and a dose-dependent increase of chylomicrons in adult male rats. There was a slight, but significant, decrease in the level of high-density lipoprotein and a significant increase in the level of LDL (low-density lipoprotein). Significant dose dependent decrease in the TAG (triacylglycerol) pool of adipose tissue and the liver, but a significant increase in the TAG pool of the gastrointestinal system was observed (*Kayode Alaba et al., 2007*). *G. Cambogia* extract effectively lowered the body weight gain, visceral fat accumulation, blood and hepatic lipid concentrations, and plasma insulin and leptin levels in a high-fat diet (HFD)-induced obesity mouse model (C57BL/6J Mice). It reversed the HFD-induced changes in the expression pattern of such epididymal adipose tissue genes as adipocyte protein aP2 (aP2), sterol regulatory element-binding factor 1c (SREBP1c), peroxisome proliferator-activated receptor2 (PPAR $\gamma$ 2), and CCAT/enhancer-binding protein  $\alpha$  (C/EBP  $\alpha$ ).

***Gymnemasylvestre*****Fig 2.13: Leaves of *Gymnemasylvestre*.**

It belongs to the family *Asclepiadaceae*. It is a woody climber. It is found in the Deccan Peninsula and western India. It is reported to be bitter, astringent, acrid, thermogenic, anti-inflammatory, anodyne, digestive, liver tonic emetic, diuretic, stomachic, stimulant, anthelmintics, laxative, cardiotonic, expectorant, antipyretic and uterine tonic. It is useful in dyspepsia, constipation and jaundice, haemorrhoids. (Ankit Saneja et al., 2010). The active compound of this plant is a group of acids termed as gymnemic acids. It has been observed that there could be a possible link between obesity, Gymnemic acids and diabetes (Parijat Kanetkar et al., 2007). leaf extract at 25-100 mg/kg when orally administered to experimentally induced hyperlipidaemic rats for 2 weeks, reduced the elevated serum triglyceride (TG), total cholesterol (TC), very low-density lipoprotein (VLDL)-and low-density lipoprotein (LDL)-cholesterol in a dose-dependent manner. The decreased serum high-density lipoprotein (HDL)-cholesterol and antiatherogenic index (AAI) in hyperlipidaemia were also reversed towards normalization. The ability of this extract (at 100 mg/kg) to lower TG and TC in serum and its antiatherosclerotic potential were almost similar to that of a standard lipid lowering agent-clofibrate (Anupam Bishayee et al., 1987). Gymnemate promoted weight loss in Otsuka Long-Evans Tokushima Fatty (OLETF) rat, a genetic multifactor syndrome model which exhibits progressive overweight, hyperlipidemia and hyperglycemia. The total cholesterol was decreased about 1/3, moreover LDL+VLDL (low-density and very-low-density lipoprotein) cholesterol decreased about 1/2. The proportion of HDL (high-density lipoprotein) cholesterol to the total cholesterol was increased. The serum triglyceride was decreased to the 1/4. There were no significant difference in levels of serum cholesterol and triglyceride (Hong Luo Akiko Kashiwag et al., 2006).

***Hibiscus sabdariffa*****Fig 2.14: *Hibiscus sabdariffa*.**

It belongs to the family *Malvaceae*. It is an annual/Perennial shrub. It is native of West Indies and is now cultivated in Uttar Pradesh, Andrapradesh, West Bengal, Bihar, Punjab, Assam and Tamilnadu. Hibiscus acid and its 6-methyl ester were respectively isolated as active principles from the 50% methanol and acetone extracts of rosella tea (Chanida Hansawasdi et al., 2000). It is used as a beverage that helps to lower the body temperature, to treat cardiac conditions, spasmolytic, antibacterial, cholagogic, diuretic and anthelmentic (Mahadevan N et al., 2009). And as a diuretic Aqueous extract of dried calyces of *H. sabdariffa* at 0.8 ml/kg body weight showed significant decrease in plasma glucose and cholesterol in rats fed with 99% growers mash and 1% cholesterol. Same results were obtained when rats were subjected to an aqueous extract of Hibiscus sabdariffa and zingier officinale at 1ml/Kg body weight. Extracts of *H. sabdariffa Z. officinale*. Apart from being hypocholesterolemic and hypoglycemic, they control blood sugar especially in those prone to diabetes mellitus (Agoreyo F. O et al., 2008). Hibiscus extract inhibited significantly the lipid droplet accumulation by MDI (isobutyl methylxanthine dexamethasone, and insulin) in a dose-dependent manner and attenuated dramatically the protein and mRNA expressions of adipogenic transcriptional factors, C/EBPa and PPAR $\gamma$ , during adipogenesis in 3T3-L1 preadipocytes. The increase of phosphorylation and expression of PI3-K/Akt during adipocytic differentiation was markedly inhibited by treatment with hibiscus extract or PI3-K inhibitors. This suggests that hibiscus extract inhibits the adipocyte differentiation through the modulation of PI3-K/Akt and ERK pathway that play pivotal roles during adipogenesis (Jin-Kyung Kim et al., 2003).

***Momordica charantia*****Fig 2.15: Fruit of *Momordica charantia*.**

It belongs to the family *Cucurbitaceae*. It is a slender, climbing annual vine. It is cultivated all over India. 5 % lyophilised Bitter melon (*M. charantia*; BM). The fruit is considered as tonic, stomachic, stimulant, emetic, antibilious, laxative and alterative. The fruit is useful in gout, rheumatism and subacute cases of the spleen and liver diseases. Leaves act as galactagogue. Root is astringent (Sathish Kumar D et al., 2010). Powder showed decreased adipose tissue mass, TAG content and glycerol-3-phosphate dehydrogenase activity when supplemented with high-fat diet for rats. This implies

that BM might reduce lipogenesis in adipose tissue. Rats also showed significantly lower mRNA levels of fatty acid synthase, acetyl-CoA carboxylase-1, lipoprotein lipase and adipocyte fatty acid-binding protein implying that BM can reduce insulin resistance effectively. Thus BM can suppress the visceral fat accumulation and inhibit adipocyte hypertrophy, which may be associated with markedly down regulated expressions of lipogenic genes in the adipose (Huang et al., 2008). Oral administration of fruit extract at (150 mg/kg & 300 mg/kg) to old obese rats showed significant reduction in total cholesterol, triglyceride and HDL-CH, LDL- CH and VLDL-CH levels in serum samples. Subchronic study of this fruit extract in alloxan induced diabetic rats showed significant antihyperglycemic activity by lowering blood glucose and percent glycosylated haemoglobin (GHb%,). Pattern of glucose tolerance curve was also altered significantly. This treatment enhanced uptake of glucose by hemidiaphragm and inhibited glycogenolysis in liver slices in vitro.

#### *Panax japonicas*



Fig 2.16: Rhizome of *Panax japonicas*.

It belongs to the family Aralioideae. It is perennial herb. It is also known as Japanese ginseng and is cultivated in Japan. Chikusetsusaponins are the active principle found in this plant. The rhizomes of *Panax japonicus* are used as a folk medicine for treatment of lifestyle related diseases such as arteriosclerosis, hyperlipidemia, hypertension and noninsulin dependent diabetes mellitus as a substitute for ginseng roots in China and Japan. It has been used as a substitute for Ginseng roots (Han KH et al., 1998).

#### *Solanum melongena*



Fig 2.17: Fruit of *Solanum melongena*.

It belongs to the family Solanaceae. It is a small tropical perennial. It is a native of Africa and Asia. Flavonoids are

the active principles in this plant. Flavonoids extracted from the fruits of *S. melongena* (Brinjal) at a dose of 1mg/100g BW/day showed significant hypolipidemic action in normal and cholesterol fed rats (Sudheesh, S et al., 1997). The fresh, ripe fruits of *Solanum melongena* and *Solanum gilo* significantly reduced serum total cholesterol by 65.40% and 52.69% respectively, triglyceride by 47.7 and 27%, LDL cholesterol by 85 and 83% respectively. They also increased significantly serum HDL by 24.7 and 25% respectively leading to increased HDL/LDL cholesterol ratio (3.37 and 3.25 respectively) in hypercholesterolemia induced New Zealand white rabbits by feeding the animals with normal diet supplemented with 1% cholesterol and groundnut oil for 3 weeks. This trend was also similar with liver lipid levels. Histopathological examination of the liver and aorta paraffin section stained with Haematoxylin and Eosine showed fewer lesions. These observations demonstrated that they have strong hypolipidemic effect and is an indication of the possible use of this fruit in the treatment of diseases associated with hyperlipidemia and arteriosclerosis (Odetola, A.A et al., 2004).

#### *Tamarindus indica*



Fig 2.18: Fruit of *Tamarindus indica*.

It belongs to the family Fabaceae. It is a large tropical tree. It is cultivated and naturalized in the tropics throughout the world. Oral administration of aqueous pulp extract of this plant resulted in a dose dependent decrease in body weight of rats. The decrease in body weight may be attributed to the reduction in food and water intake caused by chemicals that affect brain centers involved in satiety and hunger or could have inhibited digestive enzymes or decreased bioavailability of nutrient caused by anti nutritional factors present in plant extract. Dose dependent decrease in body weight could also be attributed to the presence of anti nutritional factors like saponins in the extract. Though the rats were fed with diet with adequate protein, the plant extract might not have allowed proper absorption of protein which could account for the decreased body weight. The aqueous pulp extract of the plant at 2700-4500mg/kg dose had lowered body weight, serum cholesterol and low density lipoprotein. It had significantly increased triglycerides and high density lipoproteins (Ukwuani, A.N et al., 2008).

***Zingiber officinale*****Fig 2.19:** Rhizome of *Zingiber officinale*.

It belongs to the family *Zingiberaceae*. It is a rhizomatous perennial herb. This is a native of tropical Asia, but is now grown as a commercial in Latin America and Africa as well as South East Asia. Aqueous extract of *Z. officinale* at 0.4 ml/kg body weight showed significant decrease in plasma glucose and cholesterol in rats fed with 99% growers mash and 1% cholesterol. Same results were obtained when rats were subjected to an aqueous extract of *Hibiscus sabdariffa* and *Z. Officinale* at 1ml/Kg body weight. Ethanolic extract of ginger (200mg/kg) lowered serum triglycerides, lipoproteins, phospholipids as well as serum and tissue cholesterol. In addition rats receiving ginger extract with cholesterol showed a lower degree of atherosclerosis. Ginger extract consumption reduced plasma cholesterol, inhibited LDL oxidation and attenuated development of atherosclerosis in atherosclerotic, apolipoprotein E-deficient mice (*Fuhrman B et al., 2000*).

***Cucurbita moschata*****Fig 2.21:** Whole plant of *Cucurbita moschata*.

*Cucurbita moschata* commonly known as Pumpkin and winter squash belongs to family *Cucurbitaceae*. During the screening of a variety of plant sources for their anti-obesity activity, it was found that a water-soluble extract, named PG105, prepared from stem parts of *Cucurbita moschata*, contains potent anti-obesity activities in a high fat diet-induced obesity mouse model. In this animal model, increases in body weight and fat storage were suppressed by 8-week oral administration of PG105 at 500 mg/kg, while the overall amount of food intake was not affected. Furthermore, PG105 protected the development of fatty liver and increased the hepatic  $\beta$ -oxidation activity. Results from blood analysis showed that the levels of triglyceride and cholesterol were significantly lowered by PG105 administration, and also that the level of leptin was reduced.

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