



Review Article

HYPERLIPIDEMIA AND HYPERTENSION; CARDIOVASCULAR RISK FACTORS, VARIOUS INDUCTION METHODS AND THEIR MANAGEMENT BY ETHNOMEDICINES

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ABSTRACT

Fundamental to cardiovascular diseases are two peril factors, hyperlipidemia and hypertension. These are leading cause of deaths in both developing as well urbanized countries and treated with many synthetic medicines that may attain their plateau in effectiveness and having many side effects. So, therefore utmost need to seek out safe and well tolerated medicinal plants for management with scientific appraisal by using various animal models. Hence there is an agreement from many scientific facts that medicinal plants have less side effects and proven beneficial in lowering hyperlipidemia and hypertension. Hyperlipidemia causes atherosclerosis, which ultimately leads to cardiovascular diseases while in case of hypertension many factors contribute towards development of cardiovascular diseases, most importantly renin angiotensin system and sympathetic system activation. Many synthetic medicines available for treatment of these factors but allied with side effects that overcome by use of medicinal plants that are made available for human use by conducting first experiments in animal models by inducing hypertension and hyperlipidemia by various techniques and after that treating with medicinal plants. Hence review elaborated some methods of induction and various plants having hypolipidemic and hypotensive effects.

Key words: Hyperlipidemia, Hypertension, Medicinal plants, Induction methods

INTRODUCTION

Despite of many peril factors, hyperlipidemia and hypertension have been largely associated with cardiovascular diseases. Major cardiovascular diseases are coronary heart diseases (CHD) along with cerebrovascular diseases (generally known as stroke). Hypertension being a grave facet for various heart diseases and stroke can also cause kidney damage as well as other health problems. Furthermore, hypertension is the foremost source of death in both mounting as well as urbanized countries and impinges on both sex's male and female¹. Conversely, hyperlipidemia leads to the advancement of atherosclerosis which eventually results in cardiovascular maladies². Even though, etiology of hypertension is very composite but renin-angiotensin system is largely apprehensive in the pathogenesis of hypertension³. Moreover, hyperlipidemia manifested by elevation of the serum total cholesterol, low-density lipoprotein (LDL) cholesterol and triglyceride concentrations along with a decrease in the high density lipoprotein (HDL) cholesterol level. So, hyperlipidemia is turmoil of lipoprotein metabolism⁴. Recently, field of pharmacology in union with modern medicine, chemistry and biochemistry have mutually been making endeavor to figure out the etiological facets, mechanisms and pathways of derangements in the normal body functions accountable for cardiovascular risk factors with intent to get better prevention, diagnosis and treatment³.

Ethnomedicines for Hypertension and Hyperlipidemia

Current allopathic anti-hyperlipidemic drugs comprising of bile acid sequestrants, HMG-coA reductase inhibitors, fibric acid

derivatives and niacin are effective⁵ but allied with many side effects like hyperuricemia, diarrhoea, myositis, hepatotoxicity, abdominal disturbances, hemorrhoids, generalized flushing, sensation of warmth, nausea, vomiting, impotence, myalgia (muscle cramping and aching), increased or decreased angina, cardiac arrhythmias, exhaustion and rashes⁶. Notably statins are chiefly enzyme inhibitors, so it is probable that they may be inhibiting other critical enzymes in the body³. Likewise, several synthetic drugs have been developed for the treatment of hypertension and most of these drugs have established better effectiveness but possess a number of disagreeable things⁷. Hypotensive therapy consisting of angiotensin converting enzyme inhibitors, angiotensin II receptor antagonists, alpha blockers, beta blockers, calcium channel blockers, diuretics, direct rennin inhibitors and vasodilators cause dry cough, dizziness, swollen ankles, tiredness, insomnia, impotence, depression, palpitations, slow heartbeat, loss of taste, constipation, headache, gout and kidney damage (rare). Hence, there is a enormous agreement of scientific facts to recommend that the use of cautiously chosen herbal remedies and dietary supplements can be helpful to lower blood pressure, besides improve on the whole functioning of heart, arteries, and entire cardiovascular system. The conventional antihypertensive therapies including diuretics, b-blockers, and calcium channel blockers are erratically flourishing in achieving the exigent target blood pressure values in hypertensive patients⁸. The renin-angiotensin-aldosterone system antagonists (angiotensin converting enzyme inhibitors and receptor blockers, renin inhibitors, and mineralocorticoid receptor blockers), attain a great success in this aspect⁹. But owing to high cost and adverse effects of available hypotensive and hypolipidemic drugs, use of natural substances has been increasing now-a-days. Improved

cultural adequacy, improved compatibility with the human body and lesser side effects reported, proven herbal medicine as the mainstay of about 75-80% of the world population, chiefly in the developing countries, for primary health care³. Therefore majority of plants have been reported in ayurvedic literature used for the treatment of hyperlipidemia and hypertension free of toxic contour and well tolerated. Thus, natural herbal treatment has following advantages over the synthetic medicine as natural remedy is somewhat more economical, easily accessible, free from unwanted or undesired effects, help in revitalizing and invigorating the human health and treat the root cause thus, helps in terminating health disorder permanently as compare to modern medicines⁴. Hence, there is utmost need of the day to seek out natural herbal hypotensive and hypolipidemic agents for treating cardiovascular diseases or to eliminate its root cause¹⁰. Medicinal plants used in the treatment of hypertension and hypelipidemia are given in (table 1). In addition, a vast number of plants listed in the traditional medicine are yet to receive scientific documentation.

Induction Methods in Animal Models

It is indispensable to scientifically appraise the plants thus supporting their traditional use. As these valuable plants might be source of new phytotherapeutic entities that can be developed into newer, safer and economical hypolipidemic or hypotensive drugs. Therefore, to assess the herbal plants various techniques and methods using a array of animal species like rats, mice, rabbits etc are being utilized³. Many textures of these animal models are akin to human diseases which mainly contribute in disease state. Nearly all of the animal models are developed from first to last by number of etiologic factors responsible for development of hypertension¹¹ and hyperlipidemia³ as summarized in Table 2, 3. Several new Animal models of hypertension and hyperlipidemia are being developed as new insights in to the pathogenesis of hypertension and hyperlipidemia and importantly to know how to treat cardiovascular risk factors.

Fructose Induced Hypertriglyceridemia

In this method the rats are given 25 % fructose in drinking water for 21 days, daily. Then rats develop hypertriglyceridemia can be assessed by taking blood sample of rats and conducting laboratory analysis¹². Diverse studies have reported that fructose feeding increases fasting plasma level of leptins in rats. The increase of plasma leptin level is indicative of resistance that result in hypertriglyceridemia in rats. It is therefore established that dietary fructose intake, hypertriglyceridemia with weight gain has a direct liaison with hypertriglyceridemia¹³.

Triton-Induced Hyperlipidemia

Triton WR (isooctyl-polyoxyethylene phenol) injected to mice or rats 200mg/kg intravenously and serum cholesterol levels increase piercingly 2–3 times following to 24 h. Subsequent to injection, there is increase in level of phospholipid, free and total cholesterol and triglycerides. All these changes are coupled with coagulative and fibrinolytic actions in rats with hyperlipidemia¹⁴.

Cyclosporin A-Induced Hyperlipidemia

Etiquette adopted for this, cyclosporine is injected to male Wistar rats intraperitoneally 10mg/kg daily for two weeks. After two weeks cholesterol, bilirubin, bile flow and biliary excretion can be measured by insertion of fistula into rats. From blood or serum samples concentration of these parameters is firmed¹⁵.

Cyclosporin A (CsA) is basically an immunosuppressant drug widely used in organ transplant recipients and patients with auto-immune disorders. CsA is allied with hyperlipidemia and an increased risk of atherosclerosis after long term deployment³.

Cholesterol-Diet Induced Hyperlipidemia and Atherosclerosis

The way, 2% cholesterol diet is given to rats for six weeks. After six weeks, plasma cholesterol and triglyceride levels become increased. The essential molecular mechanisms of the direct effects of cholesterol diet-induced hyperlipidemia reticence of the mevalonate pathway, decrease in NO bioavailability and cGMP metabolism, increase in free radical and peroxynitrite formation, inhibition of heat shock response, and expression of oxidized low-density lipoprotein receptors which induces apoptosis, have been shown to play a role in the effects of hyperlipidemia¹⁶.

Poloxamer 407 Induced Hypelipidemia

In this practice Poloxamer 407 is injected intraperitoneally to rats at the dose of 1 g/kg. After 15 and 24 hrs, blood sample is collected and examined for cholesterol parameters. Before this, for 26 days food and tested drug daily specified to animals. Poloxamer 407 is non ionic hydrophobic surfactant. P-407 causes hypercholesterolemia by targeting the enzyme in cholesterolgenesis, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase which is a rate limiting enzyme in cholesterol synthesis².

Glucose Induced Hypertension

In rats, glucose induced elevation in blood pressure has been found contemporaneous to the elevation of sympathetic nervous system, rennin angiotensin system activation and enhanced angiotensin II level¹⁷. In this practice, 10 % glucose solution instead of tap water is given daily for 21 consecutive days⁷. Blood pressure is measured on week 0, 1, 2, and 3.

Egg Feed Induced Hypertension

Egg feed diet, comprising of 12 egg yolk mixed with 500 g of normal rat diet is given for 21 uninterrupted days with the aim of producing cholesterol induced hypertension. All animals received saline instead of tap water *ad libitum*. Deliberate, B.P on week 0, 1, 2 and 3 by the instrument⁷.

High Salt Intake Induced Hypertension

Replacement of drinking water with 1-2% sodium chloride for 9-12 months produces hypertension in rats, chicks and rabbits. Normal kidney has the ability to seep salt and B.P remains in limits. But high salt intake cause physiologic changes and kidney not capable to expel all salt properly, ultimately results in hypertension in animals¹⁸.

DOCA Induced Hypertension

Hypertension, in this course of action is induced by replacing rat's drinking water with 2% sodium chloride solution *ad libitum*. After that DOCA (deoxycorticosterone acetate) dissolved in sesame oil given at a dose of 10 mg/kg, twice weekly for 43 days. DOCA induces hypertension by increasing salt and water absorption, that in due course increase blood volume, vasopressin with ion secretion and increase vasoconstriction. RAAS related bustle is also distorted by administration of DOCA in rats. Other animals like pigs and

dogs also prone to induction of hypertension by DOCA administration¹¹.

Stress Induced Hypertension

BHRs (borderline hypertensive rats) have been used for stress induced hypertension that bare to daily sessions of either short (20 min) or long (120 min) interval air-jet stimulation. Hypertension is induced in rats in contrast to home cage controls rats after two weeks. With 120 min long phase; rats develop high systolic blood pressure than that of 20 min short phase. The Stress-induced hypertension is linked with either normal or concealed PRA (plasma rennin activity) values, signifying that the hypertension in these animals is not renin-dependent¹⁸. The inhibition of prostaglandin (PG) synthesis by the release of corticoids in the stress response is promising method for stress-induced hypertension¹⁹.

Cholinomimetic Agents Induced Hypertension

A cholinesterase inhibitor, physostigmine and a direct muscarinic cholinergic agonist, oxotremorine have been worn for induction of hypertension in rats. Physostigmine (10-80 µg/kg) and oxotremorine (20-40 µg/kg) intravenously in a dose-reliant manner increase BP. A direct start of central cholinergic mechanism and peripherally sympathetic nervous system has been occurred after injection that ultimately increased B.P¹¹.

Angiotensin-II Induced Hypertension

Angiotensin-II, an important vasoconstrictor at a dose of (0.7 mg/kg/day) subcutaneous infusion using mini pump elicits

hypertension in rats in 4-8 weeks¹⁸. The amplified level of an angiotensin II boost sympathetic activity, Na and Cl reabsorption, water retention and K-excretion, vasoconstriction of arterioles, increase ADH secretion, increase aldosterone secretion. All these alteration result in increased circulating volume along with water retention, which in turn increase blood pressure²⁰.

Cadmium Induced Hypertension

Chronic administration of Cadmium chloride (CdCl) at a dose of (1 mg/kg) daily intra peritoneally for 2 weeks induces hypertension in rats. CdCl-induced hypertension and create a undeviating contractile effect on vascular smooth muscle which is linked to the fact that the metal ion might mimic Ca²⁺ ion as a partial agonist and produce a hypertension¹⁸.

Chronic Nitric Oxide Inhibition-Induced Hypertension

A nonselective nitric oxide synthase inhibitor, N- nitro-L-argininemethyl ester (L-NAME) is given to rats by oral administration (40 mg/kg/day) by dissolving in drinking water daily for 4 weeks¹¹. As a result hypertension produced is estimated by measuring different markers in plasma and tissue of rats. Markers comprise plasma nitric oxide metabolites (NOx), lipid peroxidative products such as thiobarbituric acid reactive substances, lipid hydroperoxides, conjugated dienes, and antioxidants such as superoxide dismutase, catalase, glutathione peroxidase, vitamin C, vitamin E, and reduced glutathione level²¹.

Table 1: Medicinal plants for treating Hypertension and Hyperlipidemia

Botanical name	Family	Part used
<i>Achyranthus aspera</i> ²²	Amarantaceae	Whole Plant
<i>Aegle marmelos</i> ³	Rutaceae	Fruits
<i>Agave veracruz</i> ²³	Amaryllidaceae	Roots, leaves, gum
<i>Allium cepa</i> ²⁴	Liliaceae	Bulbs
<i>Aloe barbadensis</i> ³	Liliaceae	Leaves
<i>Amaranthus Spinosa</i> ²⁵	Amaranthaceae	Leaves
<i>Boswellia ovalifoliolata</i> ⁶	Bursereaceae	Gum resin
<i>Bambusa arundunacea</i> ³	Graminae	Leaves
<i>Boswellia serrata</i> ²⁶	Burserraceae	Gum
<i>Brassica vercapitata</i> ³	Cruciferae	Oil
<i>Butea monosperma</i> Lam. ²⁷	Fabaceae	Leaves
<i>Bauhinia purpurea</i> ²⁸	Fabaceae	Leaves
<i>Commiphora mukul</i> ²⁹	Burseraceae	Resin
<i>Cissus quadrangularis</i> ²⁸	Vitaceae	Stem
<i>Cinnamomum tamala</i> ³⁰	Lauraceae	Leaves
<i>Cassia fistula</i> L. ³¹	Fabaceae	Legume
<i>Capparis dedicua</i> ³²	Capparidaceae	Leaves, fruits and stems
<i>Capparis spinosa</i> ²²	Capparidaceae	Fruits
<i>Capsicum frutescens</i> ³³	Solanaceae	Fruits
<i>Carum capaticum</i> ³	Umbelliferae	Fruits, roots
<i>Celastrus paniculatus</i> ³⁴	Celastraceae	Seed oil, barks, roots and fruits
<i>Curcuma amada</i> ³	Zingiberaceae	Rhizomes
<i>Curcuma longa</i> ²²	Zingiberaceae	Rhizomes
<i>Cyamopsis tetragonoloba</i> ²²	Leguminosae	Seeds
<i>Carum carvi</i> ¹²	Apiaceae	Seeds
<i>Chlorophytum borivilianum</i> ³⁵	Liliaceae	Root
<i>Carica papaya</i> ²²	Cariaceae	Seed
<i>Fritillaria ussuriensis maxim</i> ³⁶	Liliaceae	Bulb
<i>Crategus aronica</i> ³⁷	Rosaceae	Fruits
<i>Crotalaria juncea</i> ³⁸	Fabaceae	Leaves
<i>Chlorophytum Borivilianum</i> ³⁹	Liliaceae	Root
<i>Dalbergia latifolia</i> ⁴⁰	Fabaceae	Bark
<i>Dracocephalum kotschy</i> ⁴¹	Lamiaceae	Leaves
<i>Eclipta alba</i> L. ⁴²	Asteraceae	-----
<i>Emblia officinalis</i> ⁴³	Euphorbiaceae	Dried fruits, Seeds, leaves

<i>Eugenia cumini</i> ⁴⁴	Myrtaceae	Seeds
<i>Eclipta prostrata</i> ⁴³	Asteraceae	Leaves
<i>Eugenia jambolana</i> ⁴⁶	Myrtaceae	Kernels
<i>Ficus racemosa</i> ⁴⁷	Moraceae	Bark
<i>Glycyrrhiza glabra</i> ⁴⁸	Leguminosae	Root
<i>Hibiscus cannabinus</i> ⁴⁹	Malvaceae	Fresh mature Leaves
<i>Hibiscus rosasinesis</i> ⁵⁰	Malvaceae	Root
<i>Ipomoea aquatic</i> ⁵¹	Convolvulaceae	Root
<i>Lagenaria siceraria</i> ⁵²	Cucurbitaceae	Whole plant
<i>Lycium barbarum</i> ⁵³	Solanaceae	Fruit
<i>Luffa aegyptiaca</i> ⁵⁴	Cucurbitaceae	Fruit
<i>Morinda citrifolia</i> ⁵⁵	Rubiaceae	Fruit
<i>Moringa oleifera</i> ⁵⁶	Moringaceae	Fruit
<i>Nelumbo nucifera</i> ⁵⁷	Nelumbonaceae	Leaves
<i>Ougeinia oojeinensis</i> ⁵⁸	Fabaceae	Bark
<i>Oscium basilium</i> ⁵⁷	Labiatae	Whole plant
<i>Pterocarpus marsupium</i> ⁵⁷	Fabaceae	Wood and bark
<i>Pithecellobium Dulce</i> ⁵⁹	Leguminosae	Fresh leaves
<i>Piliostigma Thonningii</i> ⁶⁰		Leaves
<i>Psidium guajava</i> ⁶¹	Myrtaceae	Leaves
<i>Randia dumetorum</i> ⁶²	Rubiaceae	Fruit
<i>Rhinacanthus nasutus</i> ⁶³	Acanthaceae	Whole plant
<i>Salvadora persica</i> ⁶⁴	Salvadoraceae	-----
<i>Sapindus emarginatus</i> ⁶⁴	Sapindeae	Pericarps
<i>Spergularia purpurea</i> ⁶⁵	Caryophyllaceae	-----
<i>Sphaeranthus indicus</i> ⁶⁶	Asteraceae	Flower heads
<i>Sesbania grandiflora</i> ⁶⁷	Fabaceae	Leaves
<i>Terminalia arjuna</i> ⁶⁸	Combretaceae	Fruit
<i>Tribulus alatus</i> ⁶⁹	Zygophyllaceae	Aerial parts
<i>Tinospora cardifolia</i> ⁷⁰		Stem
<i>Terminalia chebula</i> ⁷¹	Combretaceae	-----
<i>Urtica dioica</i> ⁷¹	Urticaceae	-----
<i>Vernonia anthalmintica</i> ⁷¹	Zingiberaceae	Flower, stem
<i>Withania somnifera</i> ³⁹	Solanaceae	Roots, leaf
<i>Zingiber officinale</i> ⁷²	Zingiberaceae	Rhizome

Botanical name	Family	Part used
<i>Agastache mexicana</i> ¹	Lamiaceae	Bark
<i>Aronia mitchurnii</i> ⁷³	Rosaceae	Fruit
<i>Allium sativa</i> ⁷⁴	Amaryllidaceae	Fruit
<i>Astragalus complanatus</i> ¹	Fabaceae	Seeds
<i>Averrhoa carambola</i> ⁷⁵	Oxalidaceae	Leaves
<i>Agathosma betulina</i> ⁷⁶	Rutaceae	-----
<i>Annona muricata</i> ⁷⁷	Annonaceae	Leaf
<i>Apium graveolens</i> ⁷⁴	Apiaceae	-----
<i>Blond psyllium</i> ⁷⁶	Plantaginaceae	-----
<i>Borago officinalis</i> ⁷⁸	Boraginaceae	Leaves
<i>Buddleja crispa</i> ⁸	Buddlejaceae	Leaves
<i>Coriandrum sativum</i> ⁷⁹	Apiaceae	Fruit
<i>Cuscuta japonica</i> ⁸⁰	Convolvulaceae	Leaves
<i>Cocos nucifera</i> ⁸¹	Arecaceae	Seed
<i>Citrus limetta</i> ⁸²	Asteraceae	Whole plant
<i>Centella asiatica</i> ⁸³	Apiaceae	Whole plant
<i>Daucus carota</i> ⁸⁴	Umbelliferae	Aerial parts
<i>Desmodium styracifolium</i> ⁸⁵	Leguminosae	Leaves, Stem
<i>Fuchsia magellanica</i> ⁸⁶	Onagraceae	Leaf
<i>Gynura procumbens</i> ⁸⁷	Asteraceae	Leaves
<i>Glycine max</i> ⁸⁴	Fabaceae	Beans
<i>Gossypium barbadense</i> ⁸⁵	Malvaceae	Leaves,
<i>Geum japonicum</i> ⁸⁸	Rosaceae	Leaves
<i>Globimetula cupulata</i> ⁸⁵	Loranthaceae	Leaves
<i>Graptopetalum paraguayense</i> ⁸⁹	Crassulaceae	Leaves
<i>Ginkgo biloba</i> ⁹⁰	Ginkgoaceae	Leaves
<i>Harpephyllum caffrum</i> Bernh ¹	Anacardiaceae	Stem, bark
<i>Hibiscus sabdariffa</i> ²⁸	Malvaceae	Leaves, calyx, and Corolla
<i>Hyptis fruticosa</i> ⁹¹	Lamiaceae	Leaves
<i>Jacaranda mimosaeifolia</i> ⁸	Bignoniaceae	Leaves
<i>Jatropha gossypifolia</i> ⁹²	Euphorbiaceae	Leaves
<i>Leonurus cardiaca</i> ¹	Lamiaceae	Aerial parts
<i>Loranthus ferrugineus</i> ⁸	Loranthaceae	Leaves
<i>Lavandula stoecha</i> ¹	Lamiaceae	----
<i>Lepidium latifolium</i> ⁸⁴	Cruciferae	Leaves
<i>Lummitzera racemosa</i> ⁸	Combretaceae	Whole plant

<i>Lycopersicon esculentum</i> ⁹³	Solanaceae	Fruit
<i>Moringa oleifera</i> ⁹⁴	Moringaceae	Leaves
<i>Musanga cecropioides</i> ⁸	Cecropiaceae	Stem bark
<i>Memordica charantia</i> ⁹⁵	Cucurbitaceae	Whole plant
<i>Melothria maderaspatana</i> ⁹⁶	Cucurbitaceae	Leaves
<i>Mesona procumbens</i> ⁸	Lamiaceae	Seed
<i>Matricaria recutita</i> ⁹⁷	Asteraceae	Flower
<i>Nigella sativa</i> ⁹⁸	Ranunculaceae	Seeds
<i>Oleo europaea</i> ¹	Oleaceae	Leaves
<i>Opuntia dillenii cladodes</i> ⁹⁹	Cactaceae	Leaves
<i>Peganum harmala</i> ⁸	Nitrariaceae	-----
<i>Phyllanthus amarus</i> ¹⁰⁰	Euphorbiaceae	Leaves
<i>Pinus pinaster</i> ¹⁰¹	Pinaceae	Bark
<i>Phyllanthus urinaria</i> ¹⁰²	Phyllanthaceae	Fruit ,leaf ,flower
<i>Passiflora nepalensis</i> ¹⁰³	Passifloraceae	Petals
<i>Panax ginseng</i> ¹⁰⁴	Araliaceae	Root
<i>Panax quinquefolius</i> ¹⁰⁵	Araliaceae	Root
<i>Periploca laevigata</i> ¹	Apocynaceae	Bark, root
<i>Persea americana mill</i> ¹⁰⁶	Lauraceae	Leaves
<i>Raphanus sativus</i> ⁸³	Cruciferae	----
<i>Rhaptopetalum coriaceum oliver</i> ⁸	Scytotopetalaceae	Stem bark
<i>Retama raetam forssk</i> ¹	Fabaceae	Leaves
<i>Sesamum indicum</i> ⁸	Pedaliaceae	Seeds
<i>Solanum sisymbriifolium</i> ¹⁰⁷	Solanaceae	Root
<i>Salvia cinnabarina</i> ¹	Lamiaceae	Leaves
<i>Solanum torvum</i> ¹⁰⁸	Solanaceae	Fruits
<i>Saururus chinensis</i> ¹⁰⁹	Saururaceae	Root
<i>Theobroma cacao</i> ¹¹⁰	Malvaceae	
<i>Tanacetum vulgare</i> ¹¹¹	Asteraceae	Leaf
<i>Tropaeolum majus</i> ¹¹²	Tropaeolaceae	Seed, leaf ,flower
<i>Ulmus macrocarpus</i> ¹	Ulmaceae .	Root bark
<i>Uncaria rhynchophylla</i> ¹¹³	Rubiaceae	----
<i>Vitex donian</i> ¹	Verbenaceae	Plant
<i>Zingiber officinale</i> ¹¹⁴	Zingiberaceae	Rhizome
<i>Achillea wilhemsii</i> ¹¹⁵	Astaceae	Leaves, Flowers
<i>Lepechinia caulescens</i> ¹¹⁶	Lamiaceae	Leaves
<i>Achillea millefolium</i> ¹¹⁷	Asteraceae	Leaves
<i>Clerodendron trichotomum</i> ¹¹⁸	Lamiaceae	Stem
<i>Crataegus oxyacantha</i> ¹¹⁹	Rosaceae	Whole plant

Table 2: Animal models of Hyperlipidemia and Hypertension

Induction methods for Hyperlipidemia	Induction method for hypertension
Fructose induced hypertriglyceridemia in rats	Glucose induced hypertension
Triton-induced hyperlipidemia	Egg-feed induced hypertensive rats
Cyclosporin A-induced Hyperlipidemia	High salt intake induced hypertension
Cholesterol-diet induced hyperlipidemia	DOCA induced hypertension
Poloxamer 407 induced hyperlipidemia	Stress induced hypertension
Transgenic animals	Cholinomimetic induced hypertension
Hereditary hypercholesterolemia in rats	Angiotensin induced hypertension
Hereditary hyperlipemia in rabbits	Cadmium induced hypertension
	Chronic nitric oxide inhibition induced hypertension

Table 3: Animal models of cardiovascular risk factors

Animal model	Animals used	Dose	Route	Duration	Mechanism of induction
Fructose induced hypertriglyceridemia in rats:	Rats, (Mouse, guinea pig ,hamster)	25 % fructose	Orally, by drinking water	21 days	Leptin resistance
Triton-induced hyperlipidemia:	Mouse and rats	200mg/kg	i.v	24 h	Increase phospholipids, associated coagulative and fibrinolytic activities
Cyclosporin A-induced Hyperlipidemia:	Rats	10mg/kg	i.p	2 weeks	Obstruction bile, bile flow, bilirubin
Cholesterol-diet induced hyperlipidemia	Rats	2% cholesterol	Orally feed	6 weeks	Metabolic changes
Poloxamer 407 induced hyperlipidemia	Rats	1g/kg	i.p	15/24 h	3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, a targeted enzyme
Glucose induced hypertension	Rats	10 % glucose	Orally, in drinking water	21 days	Elevation of SNS, activation of RAAS, increase level of angiotensin II
Egg-feed induced hypertensive rats	Rats	500g of normal diet along with	Orally in diet	21 days	Increase level of cholesterol

High salt intake induced hypertension	Rats, Chiks, Rabbit	12 egg yolk 1-2 5 NaCl	Orally in drinking water	9-12 months	Kidney inability to excrete all salts
DOCA induced hypertension	Rats	2 5 NaCl+10mg/kg DOCA in sea same oil	Orally in diet	Twice weekly for 43 days	Increasing salt and water absorption, that ultimately increases blood volume, vasopressor ion secretion and increase vasoconstriction. RAAS related activity increase
Stress induced hypertension	Borderline hypertensive rats	Air jet stimulation		60mints/120 mints	Usual or concealed PRA values, indicative of that, the hypertension in these animals is not renin-dependent.
Cholinomimetic induced hypertension	Rats	20-40 µg/kg	i.v		Direct stimulation of SNS, Cholinergic mechanism
Angiotensin induced hypertension	Rats	(0.7 mg/kg/day)	s.c	4-8 weeks	Increase angiotensin level,, a vasoconstrictor
Cadmium induced hypertension	Rats	1mg/kg	Peritoneal	2 weeks	Ca ions express contractile effect on vascular smooth muscle
Chronic nitric oxide inhibition induced hypertension	Rats	40mg/kg	Orally, drinking water	4 weeks	Plasma nitric oxide metabolites (NOx), lipid peroxidative

CONCLUSION

In a nutshell hyperlipidemia and hypertension are deep-seated threat factors to cardiovascular diseases and have been managed with medicinal plants that are harmless and well tolerated with scientific evaluation by using various animal models. Further studies should be conducted towards the purification and characterization of biological active compounds that might serve as novel lead molecules for the synthesis of newer and safer therapeutic agents for cardiovascular diseases allied with hypertension and hyperlipidemia.

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