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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 platue 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 maladies ². Even though, etiology of hypertension is very composite but renin-angiotensin system is largely apprehensive in the pathogenesis of hypertension ³. Moreover, hyperlipidemia manifested by elevation of the serum total cholesterol, lowdensity lipoprotein (LDL) cholesterol and triglyceride concentrations along with a decrease in the high density lipoprotein (HDL) cholesterol level. So, hyperlipidemia is turmoil of lipoprotein metabolism ⁴. 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 treatment ³.

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 effective 5 but allied with many side effects like hyperuricemia, diarrhoea, myositis, hepatotoxicity, abdominal disturbances, heamoroids, generalized flushing, sensation of warmath, nausea, vomiting, impotence, myalgia (muscle cramping and aching), increased or decreased angina, cardiac arrhythmias, exhaustion and rashes ⁶. Notably statins are chiefly enzyme inhibitors, so it is probable that they may be inhibiting other critical enzymes in the body ³. 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 things 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 patients 8. 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 aspect ⁹. 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 care 3. 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 medicines ⁴. 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 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 analysis ¹². Diverse studies have reported that fructose feeding increases fasting plasma level of leptins in rats. The increase of plasma leptin level is indicative of resitance that result in hypertriglyceridemia in rats. It is therefore established that dietry fructose intake, hypertryglycridemia with weight gain has a direct liaison with hypertriglyceridimia ¹³.

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 phopholipid, free and total cholesterol and triglycrides. All these changes are coupled with coagulative and fibrinolytic actions in rats with hyperlipidemia

Cyclosporin A-Induced Hyperlipidemia

Etiquette adopted for this, cyclosporine is injected to male Wistar rats intraperitonaly 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 efects 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 efects of hyperlipidemia 16.

Poloxomer 407 Induced Hypelipidemia

In this practice Poloxomer 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. Poloxomer 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 level ¹⁷. 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-dependent ¹⁸.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 μ g/kg) and oxotremorine (20-40 μ g/kg) intravenously in a dosereliant manner increase BP. A direct start of central cholinergic mechanism and peripherally sympathetic nervous system has been occurred after injection that ultimately increased B.P 11 .

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 weeks ¹⁸. The amplified level of an angiotensin II boost sympathetic activity, Na and Cl reabsorption, water retension 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 peritonealy 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	Unartancian and	Urnarlinidamia
Table 1. Medicinal	piants for treating	Tryper tension and	i Hypernpiuenna

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

Eugenia cumini ⁴⁴	Myrtaceae	Seeds	
Eclipta prostrate ⁴⁵	Asteraceae	Leaves	
Eugenia jambolana ⁴⁶	Myrtaceae	Kernels	
Ficus racemosa ⁴⁷	Moraceae	Bark	
Glycyrrhiza glabra ⁴⁸	Leguminoceae	Root	
Hibiscus cannabinus ⁴⁹	Malvaceae	Fresh mature Leaves	
Habiscus rosasinesis ⁵⁰	Malvaceae	Root	
Ipomoea aquatic ⁵¹	Convovulaceae	Root	
Lagenaria siceraria ⁵²	Cucurbitaceae	Whole plant	
Lycium barbarum ⁵³	Solanaceae	Fruit	
Luffa aegyptiaca ⁵⁴	Cuccurbitaceae	Fruit	
Morinda citrifolia ⁵⁵	Rubiaceae	Fruit	
Moringa oleifera ⁵⁶	Moringaceae	Fruit	
Nelumbo nuficera ⁵⁷	Nulumbonaceae	Leaves	
Ougeinia oojeinensis ⁵⁸	Fabaceae	Bark	
Oscium basilium ⁵⁷	Labiateae	Whole plant	
Pterocarpus marsupium ⁵⁷	Fabaceae	Wood and bark	
Pithecellobium Dulce 59	Leguminosae	Fresh leaves	
Piliostigma Thonningii ⁶⁰		Leaves	
Psidium guaiava ⁶¹	Myrtaceae	Leaves	
Randia dumetorum ⁶²	Rubiaceae	Fruit	
Rhinacanthus nasutus ⁶³	Acanthaceae	Whole plant	
Salvodora persica ⁶⁴	Salvadoraceae		
Sapindus emarginatus ⁶⁴	Sapindeae	Pericarps	
Spergularia purpurea ⁶⁵	Caryophyllaceae		
Sphaeranthus indicus ⁶⁶	Asteraceae	Flower heads	
Sesbania grandiflora ⁶⁷	Fabaceae	Leaves	
Terminalia arjuna ⁶⁸	Combritaceae	Fruit	
Tribulus alaltus ⁶⁹	Zygophyllaceae	Aerial parts	
Tinospora cardifolia ⁷⁰		Stem	
Terminalia chebula ⁵⁷	Combritaceae		
Urtica diocia ⁷¹	Urticaceal		
Vernonia anthalmintica ⁵¹	Zingiberaceae	Flower,stem	
Withania somnifera ³⁹	Solanaceae	Roots,leaf	
Zingiber officinale 72	Zingiberaceae	Rhizome	

Botanical name	Family	Part used	
Agastache mexicana ¹	Lamiacea	Bark	
Aronia mitchurnii ⁷³			
Allium sativa ⁷⁴	Rosaceae	Fruit	
	Amaryllidaceae	Fruit	
Astragalus complanatus ¹	Febaceae	Seeds	
Averrhoa carambola ⁷⁵	Oxalidaceae	Leaves	
Agathosma betulina ⁷⁶	Rutaceae		
Annona muricata ⁷⁷	Annonaceae	Leaf	
Apium graveolens ⁷⁴	Apiaceae		
Blond psyllium ⁷⁶	Plantaginaceae		
Borago officinalis ⁷⁸	Boraginaceae	Leaves	
Buddleja crispa ⁸	Buddlejaceae	Leaves	
Coriandrum sativum ⁷⁹	Apiaceae	Fruit	
Cuscuta japonica ⁸⁰	Convulvulaceae	Leaves	
Cocos nucifera 81	Arecaceae	Seed	
Citrus limetta ⁸²	Asteraceae	Whole plant	
Centella asiatica ⁸³	Apiaceae	Whole plant	
Daucus carota ⁸⁴	Umbelliferae	Aerial parts	
Desmodium styracifolium ⁸⁵	Leguminosae	Leaves,Stem	
Fuchsia magellanica ⁸⁶	Onagraceae	Leaf	
Gynura procumbens ⁸⁷	Asteraceae	Leaves	
Glycine max ⁸⁴	Fabaceae	Beans	
Gossypium barbadens ⁸⁵	Malvaceae	Leaves,	
Geum japonicum ⁸⁸	Rosaceae	Leaves	
Globimetula cupulata85	Loranthaceae	Leaves	
Graptopetalum paraguayense ⁸⁹	Crassulaceae	Leaves	
Ginko biloba90	Ginkgoaceae	Leaves	
Harpephyllum caffrum Bernh ¹	Anacardiaceae	Stem, bark	
Hibiscus sabdariffa ²⁸	Malvaceae	Leaves, calyx, and Corolla	
Hyptis fruticosa ⁹¹	Lamiaceae	Leaves	
Jacaranda mimosaefolia ⁸	Bignoniaceae	Leaves	
Jatropha gossypiifolia ⁹²	Euphorbiaceae	Leaves	
Leonurus cardiaca ¹	Lamiaceae	Aerial parts	
Loranthus ferrugineus ⁸	Lorantheaceae	Leaves	
Lavandula stoecha ¹	Lamiaceae		
Lepidium latifolium ⁸⁴	Cruciferae	Leaves	
Lumnitzera racemosa ⁸	Combretaceae	Whole plant	

Lycopersicon esculentum ⁹³	Solanaceae	Fruit
Moringa oleifera ⁹⁴	Moringaceae	Leaves
Musanga cecropiodes ⁸	Cecropiaceae	Stem bark
Memordica charantia ⁹⁵	Cucurbitaceae	Whole plant
Melothria maderaspatana ⁹⁶	Cucurbitaceae	Leaves
Mesona procumbens ⁸	Lamiaceae	Seed
Matricaria recutita ⁹⁷	Asteraceae	Flower
Nigella sativa ⁹⁸	Ranunculaceae	Seeds
Oleo europpaea ¹	Oleaceae	Leaves
Opuntia dillenii cladodes ⁹⁹	Cactaceae	Leaves
Peganum harmala ⁸	Nitrariaceae	
Phyllanthus amarus ¹⁰⁰	Euphorbiaceae	Leaves
Pinus pinaster ¹⁰¹	Pinaceae	Bark
Phyllanthus urinaria ¹⁰²	Phyllanthaceae	Fruit ,leaf ,flower
Passiflora nepalensis ¹⁰³	Passifloraceae	Petals
Panax ginseng 104	Araliaceae	Root
Panax quinquefolius ¹⁰⁵	Araliaceae	Root
Periploca laevigata ¹	Apocynaceae	Bark, root
Persea americana mill 106	Lauraceae	Leaves
Raphanus sativus ⁸³	Cruciferae	
Rhaptopetalum coriaceum oliver ⁸	Scytopetalaceae	Stem bark
Retama raetam forssk ¹	Fabaceae	Leaves
Sesamum indicum ⁸	Pedaliaceae	Seeds
Solanum sisymbriifolium ¹⁰⁷	Solanaceae	Root
Salvia cinnabarina ¹	Lamiaceae	Leaves
Solanum torvum ¹⁰⁸	Solanaceae	Fruits
Saururus chinensis ¹⁰⁹	Saururaceae	Root
Theobroma cacao ¹¹⁰	Malvaceae	
Tanacetum vulgare ¹¹¹	Asteraceae	Leaf
Tropaeolum majus ¹¹²	Tropaeolaceae	Seed, leaf, flower
Ulmus macrocarpus ¹	Ulmaceae .	Root bark
Uncaria rhynchophylla ¹¹³	Rubiaceae	
Vitex donian ¹	Verbenaceae	Plant
Zingiber officinale 114	Zingiberaceae	Rhizome
Achillea wilhemsii ¹¹⁵	Astreaceae	Leaves, Flowers
Lepeechinia caulescens ¹¹⁶	Lamiaceae	Leaves
Achillea millefolium ¹¹⁷	Asteraceae	Leaves
Clerodendron trichotomum 118	Lamiaceae	Stem
Crataegus oxyacantha 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
Poloxomer 407 induced hypelipidemia	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 hyperten	

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 cogulative and fibrinolitic activities
Cyclosporin A-induced Hyperlipidemia:	Rats	10mg/kg	i.p	2 weeks	Obstruction bile, bile flow, bilirubin
Cholesterol-diet induced hyperlipidemia	Rats	2% cholestrol	Oraly feed	6 weeks	Metabolic changes
Poloxomer 407 induced hypelipidemia	Rats	1 g/kg	i.p	15/24 h	3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductas, 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

		12 egg yolk			
High salt intake induced hypertension	Rats, Chiks, Rabbit	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, vasopresor 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	1 mg/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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