

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

5,400

Open access books available

133,000

International authors and editors

165M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Plants with Hypolipidaemic Effects from Nigerian Flora

Ngozi Justina Nwodo, Charles Okeke Nnadi,
Akachukwu Ibezim and Chika John Mbah

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/57181>

1. Introduction

Definition: Hyperlipidemia is a heterogeneous group of disorders characterized by high level of lipids (fats) in the bloodstream. These lipids include cholesterol, cholesterol esters, phospholipids, and triglycerides. Lipids are transported in the blood as large 'lipoproteins'. Alternatively, the disease refers to elevated levels of lipids and cholesterol in the blood, or the manifestations of different disorders of lipoprotein metabolism (dyslipidemia).

Causes: Hyperlipidemia could be caused by: (i) Familial combined hypercholesterolemia (ii) Familial hypertriglyceridemia (iii) other disease states such as *insulin and non-insulin dependent diabetes mellitus*, *hypothyroidism*, *Cushing's syndrome*, *dysproteinemias*, *nephrotic syndrome* and *renal failure*, cholestatic disorders and low thyroid (iv) drugs such as anabolic steroids, beta-blockers, birth control pills and estrogens, corticosteroids, protease inhibitors, retinoids, thiazide diuretics (v) diets like cholesterol intake greater than 300 mg per day, fat intake per total calories greater than 40 %, saturated fat intake per total calories greater than 10 % (vi) life style involving habitual excessive alcohol use, lack of exercise, smoking (vii) risk factors such as advancing age, sex (male), stress and postmenopause.

Classification: Lipoproteins are divided into five major classes, based on density and they include: (i) chylomicrons (ii) very low-density lipoproteins (VLDL) (iii) intermediate-density lipoproteins (IDL) (iv) low-density lipoproteins (LDL) and (v) high-density lipoproteins (HDL). Most triglyceride is transported in chylomicrons or VLDL, while most cholesterol is carried in LDL and HDL. Hyperlipidemia, a major, modifiable risk factor for atherosclerosis and cardiovascular disease, including coronary heart disease (CHD) is classified under (1) Primary hyperlipidemias - are probably genetically based, but the genetic defects are known for only a minority of patients. Examples are (i) primary chylomicronemia- recessive traits of deficiency of lipoprotein lipase or its cofactor (ii) familial hypercholesterolemia- an autosomal

dominant trait, although levels of LDL tend to increase with normal VLDL; familial combined (mixed) hyperlipoproteinemia- elevated levels of VLDL, LDL (iii) familial dysbetalipoproteinemia-increased LDL with increased TG and cholesterol levels (iv) familial hypertriglyceridemia-increased VLDL production with normal or decreased LDL (v) familial mixed hypertriglyceridemia-serum VLDL and chylomicrons are increased. (2) Secondary hyperlipidemia- results from disease states such as Cushing's syndrome, diabetes, liver disorders, renal disorders, thyroid disease, obesity, as well as alcohol consumption, estrogen administration, and other drug-associated changes in lipid metabolism.

Symptoms: Hyperlipidemia usually does not cause symptoms. Very high levels of lipids or triglycerides can cause yellowish nodules of fat in the skin beneath eyes, elbows and knees, and in tendons (xanthomas). Sometimes pain, swelling of organs such as the liver, spleen or pancreas (pancreatitis) or whitish rings around the eye's iris occur. **Diagnosis:** Diagnosis is typically based on medical history, physical examination and most importantly blood test done after overnight fasting. The blood test, measure the levels of lipids in the blood and consist of, a fasting blood test for total cholesterol (TC), LDL (bad cholesterol), HDL (good cholesterol), triglycerides (TG). American Cholesterol Education Program advises that lipids be checked at least once every five years, starting at age 20. However, more frequent or earlier testing is recommended if family history of hyperlipidemia; risk factor or disease that may cause hyperlipidemia; complication that may result from hyperlipidemia exist. Also, the American Academy of Pediatrics recommends lipid screening for children at risk (example, a family history of hyperlipidemia and/or diabetes). Table 1 provides specifications for making a determination.

Cholesterol level	Acceptable	Borderline	High
Total Cholesterol (mg/dl)	<170	170 – 199	≥ 200
LDL Cholesterol (mg/dl)	<110	110 – 129	≥ 130
HDL Cholesterol (mg/dl)	<40	40-59	≥ 60
Total glycerides (mg/dl)	<150	150-200	≥ 200

Table 1. Classification of cholesterol level

Prevalence: (i) A significant percentage of world population has an increased plasma lipid level, resulting in increased risk of coronary heart disease (ii) Ethnic groups adopting a 'western' lifestyle tend to have higher levels of plasma lipids (iii) Men >30 years and women >55 years (in the U.S.) have 10 % rise in fasting triglyceride level >200 mg/dl (iv) Severe hypertriglyceridemia (>2000 mg/dl) higher in diabetic patients or patients suffering alcoholism (v) Lipoprotein lipase deficiency prevalence is much higher in Quebec, Canada. (vi) total C and LDL-C rise steadily about 20% in men aged 20 to 50 years, 30% in women aged 20 to 60 years and younger women have lower levels than men while homozygous familial hypercholesterolemia manifests itself from birth (vii) hyperlipidemia is higher among men than women (gender factor) (viii) total cholesterol and LDL-C levels are similar in whites and blacks,

triglycerides are lower and HDL-C levels tend to be higher in the African-American population. Asian-Indians have the highest risk, Europeans have an intermediate risk while Chinese have the lowest risk (race factor) (ix) familial combined hyperlipidemia inheritance is autosomal dominant and likely to involve one of multiple genetic defects, familial hypertriglyceridemia is most likely inherited as an autosomal dominant defect, lipoprotein lipase deficiency and hepatic lipase deficiency are very rare autosomal recessive conditions hypercholesterolemia in the majority of the general public is attributed to high-fat diets and poorly understood susceptibility and modifier genes (genetics factor). Published data on the prevalence of lipid abnormalities in Nigeria are scanty. This could be attributed to low prevalence of hyperlipidemia in Nigeria prior to occidental lifestyle. Osuji et al, 2012, reported that the current state of dyslipidemia in Nigeria clearly contradicts the previous perceptions. In their report, dyslipidemia was found to be highly prevalent in Nigeria with consistent low HDL-cholesterol and high LDL-c especially amongst the upper social class and people with other risk factors. Other studies reported low HDL-c, with TC/HDL-c to be prevalent in the Northern part of the country while high prevalence of TC, TG and low HDL were observed in the Southern part of the country amongst people of upper social class.

2. Treatment

Dietary intervention: is the primary treatment strategy, but drug therapy may often be added later to augment treatment. The main component of a “heart-healthy” diet is a food pattern that is low in saturated fat and dietary cholesterol and provides adequate energy to support growth and maintain an appropriate weight. Specific dietary recommendations include: (i) decreased intakes of saturated fat- most effective in lowering LDL. Sources include stick margarine, partially hydrogenated oils and fats, hydrogenated peanut butters, commercial bakery products, commercial fried food (e.g., French fries) and high fat animal products (ii) decreased intakes of trans-fatty acids- trans-fatty acids are thought to increase LDL levels nearly as much as saturated fat and appear to lower HDL. (iii) decreased intakes of dietary cholesterol- lead to LDL reduction. Diabetic patients tend to be more sensitive to dietary cholesterol intake, which is only found in animal products (iv) balance the fatty acid composition of diet- polyunsaturated and monounsaturated fatty acids can lower LDL and could be good substitutes for saturated fats (v) increased fiber intakes- soluble fiber can contribute to LDL reduction and is now a formal part of hyperlipidemia dietary recommendations. Common sources of fiber include oats, psyllium, guar gum, pectin, barley, dried beans, fruits, vegetables, cereals, whole grains, and legumes are good sources of soluble fiber (vi) encourage antioxidant food sources such as carotenoids, vitamins C and E and antioxidant-rich foods such as whole grains, citrus fruits, melons, berries and leafy green vegetables rather than supplements (vii) reduce serum homocysteine levels- adequate intakes of folate and vitamins B₆ and B₁₂ as well as total fat restriction may keep homocysteine levels low. Food sources of these nutrients include fruits, dark green and leafy vegetables, fortified cereals, whole grains, lean meats and poultry.

Drug Therapy: Currently, there are many classes of medications that may be utilized in the pharmacologic management of hyperlipidemia. They are (1) HMG-CoA (3-hydroxy-3-methylglutaryl-coenzyme A) reductase inhibitors (statins). The cornerstone of the lipid-lowering therapy in adults has rested with the HMG CoA reductase inhibitors or statins. The use of these drugs has resulted in important reductions in overall cardiovascular morbidity and mortality. Mechanism of action- reduction of cholesterol synthesis in liver; inhibiting the rate-limiting step in endogenous cholesterol synthesis; compensatory increase in synthesis of LDL receptors on hepatic and extra hepatic tissues; increase in hepatic uptake of circulating LDL which decreases plasma LDL cholesterol; increase in HDL, decrease in TGs and vasodilatation and decrease in atherosclerosis. Pharmacological indication: Clinically used in the treatment of all types of hyperlipidemia except those who are homozygous for familial hypercholesterolemia (lack of LDL receptors). Table 2 summarizes the statins and their clinically applications.

Drug	Starting Dose (mg)	FDA-Approved Maximum (mg)	Half-life (hours)	Average Decrease in LDL-C Per Dose (mg:%)
Atorvastatin (lipitor)	10-20	80	14 or 20-30	10:39 20:43 80:60
Fluvastatin (Lescol)	20	80	3	20:22 80:35
Lovastatin (Mevacor)	20	80	2	20:28
Pitavastatin (Livalo)	2	4	12	2:36 4:43
Pravastatin (Pravachol)	40	80	2	40:34 80:37
Rosuvastatin (Crestor)	5-10	40	19	5:45 10:52 40:63
Simvastatin (Zocor)	20	80	4	20:38 80:36-47
Simvastatin/Ezetimibe (Vytorin)	10/10	10/40	22	10/10:45 10/40:55

Table 2. HMG-CoA INHIBITORS

(2) Fibrates (activators of lipoprotein lipase): Mechanism of action- agonists at peroxisome proliferator-activated receptor (PPAR); hydrolysis of VLDL and chylomicrons; decrease in serum TGs; increase clearance of LDL by liver and increase in HDL and expression of genes responsible for increased activity of plasma lipoprotein lipase enzyme. Pharmacological indication: most effective in reduction TGs (hypertriglyceridemia); combined hyperlipidemia (type III) if statins are contraindicated. Typical examples are fenofibrate(prodrug) and gemfibrozil (lipid) (3) Ezetimibe: Mechanism of action- inhibits intestinal cholesterol and

related phytosterol absorption; decrease in concentration of intrahepatic cholesterol; increase in uptake of circulating LDL; decrease in serum LDL cholesterol levels and compensatory increase in LDL receptors. Pharmacological indication: Effective in hypercholesterolemia together with statins and diet regulation; utilization of ezetimibe along with a statin allows for lower doses of the statin to be used, therefore reducing the likelihood of dose-related side effects of the statin. (4) Nicotinic acid; Niacin (Inhibitor of lipolysis): Mechanism of action- a potent inhibitor of lipolysis in adipose tissues; decreases mobilization of FFAs (major precursor of TGs) to the liver; increases HDL levels; decreases LDL, decreases endothelial dysfunction and thrombosis. Pharmacological indication- Used in the treatment of familial hyperlipidemias (type IIB) (increase in VLDL and LDL); combined with fibrates or cholestyramine in the treatment of hypercholesterolemia (5) Bile acids- Sequestrants(resins): The bile acid binding resins have been felt to be preferred in the pediatric age group as they are not systemically absorbed. Mechanism of action- are anion exchange resins; bind bile acids in the intestine forming complex that leads to loss of bile acids in the stools; increase the conversion of cholesterol into bile acids in the liver; compensatory increase in LDL receptors leading to decreased concentration of intrahepatic cholesterol; increase hepatic uptake of circulating LDL and decrease serum LDL cholesterol levels. Pharmacological indication: Effective in the treatment of type IIA and IIB hyperlipidemias (along with statins when response to statins is inadequate or they are contraindicated); treatment of pruritus in biliary obstruction (as rising from increase in bile acids). Typical examples are cholestyramine, colestipol and colesevelam. (6) Lovaza (Omega-3-acid ethyl ester): Mechanism of action: is unclear; however, proposed mechanisms include decreasing lipogenesis in the liver, increasing plasma lipoprotein lipase activity, and increasing mitochondrial and peroxisomal lipase activity. The drug may increase aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels and has also been known to prolong bleeding time. Pharmacological indication: is indicated as adjunct therapy to diet in patients with triglyceride levels greater than or equal to 500 mg/dl (hypertriglyceridemia). It provides significant reduction in triglycerides, of approximately 44.9%, making it an ideal drug choice in patients with high triglycerides (7) Fish oil is another common over-the-counter (OTC) product that provides an alternative to the prescription product Lovaza (8) OTC herbal product: (i) Red yeast rice (RYR)- herbal supplements used for lipid-lowering effects. RYR is obtained by fermenting *Monascus purpureus*, a form of yeast, on rice, which is then dried, pulverized, and encapsulated. This process leads to the formation of 14 monacolins, which are compounds that inhibit HMG-CoA reductase. One of the monacolins, monacolin K (lovastatin or mev-inolin) was the first synthesized HMG-CoA reductase inhibitor. RYR is commercially available in 600-mg capsules (ii) Plant sterols and stanols also assist in the reduction of LDL-C. Plant sterols reduce cholesterol absorption by competing with cholesterol for space within bile salt micelles in the intestinal lumen. The plant stanols, which are the result of the hydrogenation of sterols, are not absorbed as well as sterols. Ingestion of about 2 g per day of plant sterols or stanols, produces LDL-C reduction of 6% to 15%. Prevention: Cardiovascular disease (CVD) is the leading cause of mortality in advanced countries, with hyperlipidemia a common risk factor for CVD, in adults having abnormal cholesterol values and elevated low-density lipoprotein (LDL) cholesterol levels. Prevention could be subdivided

into: Primary prevention- (i) initial treatment is diet/exercise and should be given three to six months on dietary therapy prior to beginning medication and longer if lipids are improving and nearing LDL thresholds (ii) obtain cholesterol tests starting at the age of 20 (iii) eat a diet low in total fat, saturated fat, and cholesterol namely eat poultry without the skin, fish, vegetables, most fruits, whole grains, and skim milk (iv) reduce sugar intake (v) eat foods high in soluble fiber (vi) eat more cold water fish and soy products (vii) avoid cigarette smoking (viii) drink alcohol in moderation (two drinks per day for men, one drink per day for women) (ix) avoid overweight (x) exercise regularly and control blood sugar if diabetes is implicated (xi) increase physical activity (xii) consume a diet that contains adequate potassium, calcium, and magnesium to facilitate blood pressure control. Secondary prevention: Measuring lipids in adolescents that have strong family history of two or more coronary heart disease risk factors. In summary, US National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III in its guidelines has communicated the importance of early identification of risk, lifestyle modification, and pharmacologic treatment as the mainstay of therapy for hyperlipidemia and in the prevention of cardiovascular-related death.

The Promise of Nigeria Natural Products: Since the recognition of hyperlipidemia, a large number of plant species have been identified as having antihyperlipidemic properties and natural products are part of the current therapy for hyperlipidemia. Numerous natural products with antihyperlipidemic effect have been described in the literature. The objective of this chapter is to summarize the role of Nigeria natural products in the treatment and prevention of hyperlipidemia to date and to highlight specific classes of compounds that possess a requisite level of activity that would be considered worthy of further investigation as potential drug candidate.

3. Discussion

3.1. Antilipidemic agents from Nigeria flora

In Nigeria, traditional medicine has been the most popular means of healthcare from the olden days, before the emergence of alternative medicine in the form of synthetic agents. Traditional medicine can be said to be indigenous and a culture handed over to us by our ancestors as a means of surviving from various ailments obvious in every society. Due to high cost of synthetic drugs and side effects, natural products have become the best alternative strategy for the development of safe antilipidemic drugs. Various natural products both crude and isolated components found from plants are effective remedies for hyperlipidemia cases. Several proves are available in nature, indicating the positive effects of many natural product components that can be employed for the treatment of hyperlipidemia. Ibrahim *et al*, 2013 stated that polyphenols as apigenin, genistein and catechins as well as saponins, sterols, stanols polyunsaturated fatty acids, mucilage and carbohydrates are good examples of agents found to exhibit potent hypocholesterolemic activities. Table 3 summarizes the continuous investigations of Nigerian plants used as antihyperlipidemia from ethnopharmacological approach based on the folkloric claims.

Sources	Morphological parts	Comments	References
<i>Persea Americana</i> (Avocado pea)	Leaves, methanolic extract	Hypolipidemic activity at 40 mg/kg	Kolawole <i>et al.</i> , 2012
<i>Garciniakola</i>	Root and seed, normal saline extracts	Hypolipidemic activity at 300-900 mg/kg	Udenze <i>et al.</i> , 2012
<i>Viscumalbum</i>	Plant parts, methanolic extract	Hypolipidemic activity at 50-100 mg/kg	Oluwatosin <i>et al.</i> , 2012
<i>Caricapapaya</i>	Seed, aqueous extract	Hypolipidemic activity at 100-400 mg/kg	Nwangwa and Ekhoje 2013
<i>Emilapraetermissa</i>	Leaves, aqueous extract	Hypolipidemic activity	Anaka <i>et al.</i> , 2013
<i>Cleistopholis patens</i>	Leaves, aqueous extract	Hypolipidemic activity at 400-600 mg/kg	Udem <i>et al.</i> , 2011
<i>Solanumanguivi</i> , <i>S. macrocarpum</i>	Fruit, aqueous extract	Hypolipidemic activity at 20-100 mg/kg	Elekofehiniti <i>et al.</i> 2012; Sodipo <i>et al.</i> , 2011
<i>Annonamuricata</i>	Plant parts, methanolic extract	Hypolipidemic activity	Adeyemi <i>et al.</i> , 2009
<i>Nauclealatifolia</i>	Root and stem bark, ethanolic extract	Hypolipidemic activity at 100-150 mg/kg	Odey <i>et al.</i> , 2013
<i>Acalypha torta</i> <i>A. capitata</i>	Leaves, aqueous extract	Hypolipidemic activity at 100-200 mg/kg	Nnodim <i>et al.</i> , 2011
<i>Scopariadulcis</i>	Plant (herb) parts, methanolic extract	Hypolipidemic activity	Orhue and Nwanze, 2006
<i>Alchorneacordifolia</i>	Leaves, butanolic extract	Hypolipidemic activity at 800 mg/kg	Mohammed <i>et al.</i> , 2012
<i>Vernonia amygdalina</i> <i>Vernonia amygdalina</i>	Plant parts, methanolic extract; Leaves, ethanolic extract; root, normal saline extract	Hypolipidemic activity Hypolipidemic activity at 100-200 mg/kg	Oluwatosin <i>et al.</i> , 2008 Igbakin 2009, Owen <i>et al.</i> , 2011
<i>Moringa oleifera</i>	Leaves, aqueous extract	Hypolipidemic activity at 1 mg/g	Ghasi <i>et al.</i> , 2000
<i>Clerodendrumcapitalum</i>	Leaves, aqueous extract	Hypolipidemic activity at 100-800 mg/kg	Adenaya <i>et al.</i> , 2008
<i>Parkiabiglobosa</i>	Plant parts, methanolic extract	Hypolipidemic activity at 30-60 mg/kg	Odetola <i>et al.</i> , 2006
<i>Citrusparadisi</i>	Seed, methanolic extract	Hypolipidemic activity at 100-600 mg/kg	Adeneye, 2008
<i>Cymbopogoncitrates</i>	Leaves, aqueous extract	Hypolipidemic activity at 125-500 mg/kg	Adeneye & Agbaje, 2007
<i>Catharanthusroseus</i>	Leaves, aqueous extract	Hypolipidemic activity at 1 ml/kg	Antia & Okokon, 2005
<i>Albizziachevalieri</i>	Root, aqueous extract	Hypolipidemic activity at 100-300 mg/kg	Saidu <i>et al.</i> , 2010
<i>Stachytarphelaaugustifolia</i>	Aerial part, methanolic extract	Hypolipidemic activity	Garba <i>et al.</i> , 2013
<i>Vitexdoniana</i>	Leaves, ethanolic extract	Hypolipidemic activity	Oche <i>et al.</i> , 2012

Sources	Morphological parts	Comments	References
<i>Morindamorindoides</i>	Root bark, methanolic extract	Hypolipidemic activity	Olukunle <i>et al.</i> , 2012
<i>Arachishypogaea</i>	Plant parts, aqueous extract	Hypolipidemic activity at 175 mg/kg	Bilbis <i>et al.</i> , 2002
"Ata-Ofa' (polyherbal tea)	Leaves, methanolic extract	Hypolipidemic activity at 50 mg/kg	Atawodi, 2001
<i>Xylopiiaethiopica</i>	Seed, methanolic extract	Hypolipidemic activity at 250 mg/kg	Nwozo <i>et al.</i> , 2011
<i>Parinaripolyandra</i>	Fruit, ethanolic extract	Hypolipidemic activity at 50-250 mg/kg	Abolaji <i>et al.</i> , 2007
<i>Telfairia occidentalis</i>	Plant parts, methanolic extract	Hypolipidemic activity	Adaramoye <i>et al.</i> , 2007
<i>Curcuma longa</i>	methanol extract of the rhizomes	hypoglycemic and hypolipidemic activity 100 mg/kg	Nwozo <i>et al.</i> , 2009.
<i>Spondiamombia</i>	Aqueous leave extract	Lipid lowering effect at the doses of 250, 500and 750 mg/kg	Igwe <i>et al.</i> , 2008
<i>Crotonzambesicus</i>	Ethanolic leaf extract	Lipid lowering effect	Ofusori <i>et al.</i> , 2012.
<i>Momordicacharantia</i> Linn	Methanolic extract of the fruits	Anti-Diabetic and Hypolipidemic Effects at the doses of 200, 400 and 600 mg/kg	Kolawole and Ayankunle, 2012.
<i>Bauhiniathoningii</i>	Aqueous crude extract	Hypoglycemic and lipidemic effecte	Ojezele and Abatan, 2011.
<i>Cajanuscajan</i>	Methanolic leaf extract	Antioxidant and hypolipidemic activity at the dose of 200 mg/kg	Akinloye and Solanke 2011.
<i>Jatrophatanjorensis</i>	Methanolic leaf extract	Serum lipid profile and phytochemical composition at 100, 200 and 500mg/kg dose ranges	Oluwole <i>et al.</i> , 2011.
<i>Melantherascandens</i>	Ethanolic leaf extract	Antidiabetic and hypolipidemic activities at the doses of 37, 74 &111 mg/kg	Akpan <i>et al.</i> , 2012.
<i>Ricinuscommunis</i>	Aqueous root extract	Hypoglycaemic potential, lipid profile effects At a dose of 500mg/kg	Matthew <i>et al.</i> , 2012.

Table 3. Medicinal plants investigated in Nigeria for use as Antihypolipidemic agent

These plants have been identified, authenticated and investigated from Nigeria flora against hyperlipidemia, using pharmacological validated animal models. They all have levels and with some levels of increase in LDL, TC, TG and decrease in HDL. Furthermore, there has been recent interest on the research towards hyperlipidemia due to its obvious relationship with

diabetes and other ailments like cushing's syndrome, renal disorder, pregnancy, polycystic ovary syndrome, underactive thyroid gland etc. Hyperlipidemia arising from high serum triglyceride or total cholesterol concentration or both has been reported in diabetic and hypertensive patients. Diabetics have been reported to be more prone to cardiovascular diseases including hypertension than non-diabetics (Bilbis et al, 2002). An overview of 40 medicinal plant species from Nigerian indigenous plants reported to have hypolipidemic effects are presented. Most of the reported hypolipidemic effects were on crude extracts and active constituents. Above 30 % of the investigated plant parts had effects on both lipid profile and glycaemic index. However, much still needs to be done on several phytoconstituents of these plants, as well as conduct clinical research on active constituents derived from them, especially in the determination of their levels of toxicity. Other Nigerian plants claimed to have positive effects on lipid profile but found to act as soup thickeners are yet to be investigated. The reported Nigerian plants in Table 3 are rich in soluble and dietary fibres (examples, legumes, fruits and vegetables) and if found to have minimal toxicities, can be incorporated into dietary supplements. According to Ibrahim *et al*, (2013), the major advantage of natural hypolipidemic drugs over synthetic drugs is that many natural drugs exhibit their hypolipidemic activity by different mechanisms. Plants are known to have a striking potential in the management of lipid metabolism and providing better therapeutic effects as an alternative medicine.

4. Conclusion

The use of herbal or natural medicines for the treatment of various disorders has a long and extensive history. The reported plants have the potential to act as lipid-lowering agents with minimal side effects (advantage over currently synthetic drugs) and thus could find their way onto the world market as alternatives to prescribed drugs currently available to treat hyperlipidemia. Most of the studies were carried out with crude extract and administered orally. The principal families in which such activity has been reported are Acanthaceae, Apiaceae, Asteraceae, Azoaceae, Combretaceae, Cucurbitaceae, Euphorbiaceae, Fabaceae, Lamiaceae, Liliaceae, Malvaceae, Myrtaceae, Rubiaceae, Rutaceae and Zingiberaceae, Finally, all the plant species appear to be promising as hypolipidemic agents with activity mediated through various mechanisms.

Author details

Ngozi Justina Nwodo*, Charles Okeke Nnadi, Akachukwu Ibezim and Chika John Mbah

*Address all correspondence to: ngozi.nwodo@unn.edu.ng

Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, University of Nigeria, Nsukka Enugu State, Nigeria

References

- [1] Abolaji, A. O, Adebayo, A. H, & Odesanmi, O. S. (2007). Effects of *Parinari polyandra* (Rosaceae) on serum lipid profile and some electrolytes in pregnant rabbits. *Res. J. of Med.Plant* , 1(4), 121-127.
- [2] Adaramoye, O. A, Achem, J, Akintayo, O. O, & Fafunso, M. A. (2007). Hypolipidemic effect of *Telfairia occidentalis* (fluted pumpkin) in rats fed a cholesterol-rich diet. *J Med Food.* , 10(2), 330-6.
- [3] Adeneye, A. A, Adeleke, T. I, & Adeneye, A. K. (2008). Hypoglycemic and hypolipidemic effects of the aqueous fresh leaves extract of *Clerodendrum capitatum* in Wistar rats. *J. Ethnopharmacol* , 116(1), 7-10.
- [4] Adeneye, A. A. (2008). Hypoglycemic and hypolipidemic effects of methanol seed extract of *Citrus paradisi* Macfad (Rutaceae) in alloxan-induced diabetic Wistar rats. *Nig Q J Hosp Med.*; , 18(4), 211-5.
- [5] Adeneye, A. A, & Agbaje, E. O. (2007). Hypoglycemic and hypolipidemic effects of fresh leaf aqueous extract of *Cymbopogon citratus* Stapf. in rats. *J. Ethnopharmacol.* 25;; 112(3), 440-4.
- [6] Adeneye, A. A, & Olagunju, A. J. (2009). Preliminary hypoglycemic and hypolipidemic activities of the aqueous seed extract of *Carica papaya*. *Biology and Medicine* , 1
- [7] Aderamoye, O, Amanlou, M, Habibi-rezaei, M, Pasalar, P, & Ali, M. M. (2012). Methanolic extract of African mistletoes (*Viscum album*) improves carbohydrate metabolism and hyperlipidemia in streptozotocin-induced diabetic rats. *Asian Pac J. Trop. Med.* , 5(6), 427-33.
- [8] Adeyemi, D. O, Komolafe, O. A, Adewole, S. O, & Obuotor, E. M. (2009). Anti-hyperlipidemic activities of *Annona muricata* (Linn). *Int. J. Altern. Med.* DOI:b, 7
- [9] Akinloye, O. A, & Solanke, O. O. (2011). Evaluation of hypolipidemic and potential antioxidant effects of Pigeon pea (*Cajanus cajan* (l) mill sp.) leaves in alloxan-induced hyperglycemic rats. *J. of Med. Plants Res.* , 5, 2521-2524.
- [10] Akpan, E. J, Okokon, J. E, & Offong, E. (2012). Antidiabetic and hypolipidemic activities of ethanolic leaf extract and fractions of *Melanthera scandens*. *Asian Pacific J. of Tropical Biomedicine* , 523-527.
- [11] Akuyam, S. A, Anya, P. O, Isah, H. S, Aliyu, I. S, & Yusuf, R. (2010). Lipid abnormalities: "A Case Study of Ahadu Gello University Hospital, Zaria, Nigeria". *Ann. Nig. Med.* , 4, 10-13.
- [12] Alli Smith YRAdenlawo IG, (2013). Tissue lipid profile of rats administered saponin extract from the root of *bitter kola*. *Adv, in Biochem.*, , 1(1), 1-4.

- [13] American Diabetes Association(2001). Nutrition recommendations and principles for people with diabetes mellitus. *Diabetes Care*. 24:SS47., 44.
- [14] Anaka Ogochukwu NgoziOwolabi Omonkhelin Josephine, EmenikeChinwendu, (2013). Anti-hyperlipidemic effect of aqueous leaf extracts *Emilia praetermissa* niline-redh (Asteraceae) in rats. *Inter J, of Biosciences*. , 3(5), 68-77.
- [15] Antia, B. S, & Okokon, J. E. (2005). Effect of leaf juice of *Catharanthus roseus* Linn on cholesterol, triglyceride and lipoproteins levels in normal rats. *Indian J Pharmacol*. , 37, 401-2.
- [16] Atawodi, S. E. (2011). Evaluation of the hypoglycemic, hypolipidemic and antioxidant effects of methanolic extract of "Ata-Ofa" Polyherbal Tea (A-Polyherbal) in Alloxan-induced diabetic rats. *Drug Invention Today*. , 3(11), 270-276.
- [17] Bilbis, L. S, Shehu, R. A, & Abubakar, M. G. (2002). Hypoglycemic and hypolipidemic effects of aqueous extract of *Arachis hypogaea* in normal and alloxan-induced diabetic rats. *Phytomedicine* , 9(6), 553-5.
- [18] Burkill, H. M. (1997). The useful plants of west tropical Africa. 2. Royia Botanic Gardens, kew., 4
- [19] Chait, A, & Brunzell, J. D. (1990). Acquired hyperlipidemia (secondary dyslipoprotein-emias). *Endocrinol Metab Clin North Am*. , 19, 259-278.
- [20] Dixon, L. B, & Ernst, N. D. (2001). Choose a diet that is low in saturated fat and cholesterol and moderate in total fat:subtle changes to a familiar message. *J. Nutr*. 131(2S-1):510S-526S
- [21] Duplaga, B. A. (1999). Treatment of childhood hypercholesterolemia with HMG-CoA reductase inhibitors. *Ann. Pharmacother*; , 33(11), 1224-1227.
- [22] Elekofehinti, O. O, Adanlawo, I. G, Salin, J. A, & Sodehinde, S. A. (2012). Saponins from *Solanum anguivi* fruits extracts hypolipidemic potential in *Rattusnovergicus*. *Der Pharmacia Lettre*. , 4(3), 811-814.
- [23] Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on DetectionEvaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Jama* (2001). , 285(19), 2486-2497.
- [24] Ezekwesili, C. N, & Obidoa, O. Nwodo OFC, (2008). Effect of ethanol extract of *Acalypha torta* leaves on the lipid profile and serum electrolytes of rabbits. *Nig. J. of Biochem. Mol. Bio.*, , 23(1), 15-21.
- [25] Fletcher, B, Berra, K, Ades, P, et al. (2005). Managing blood lipids: a collaborative approach. *Circulation*., 112, 3184-3209.
- [26] Garba, A, Mada, S. B, & Ibrahim, G. Dauran IA Hamza AB, (2013). Studies on hypoglycemic and hypolipidemic effects of methanolic extract of *Stachytarpheta angustifolia*(mill) in streptozotoin induced diabetic rats. *Asian J. Bio. Sci*. Doi:10.3923/ajbs.2013

- [27] Ghasi, S, Nwobodo, E, & Ofili, J. O. (2000). Hypocholesterolemic effects of crude extract of leaf of *Moringa oleifera* Lam in high-fat diet fed wistar rats. *J. Ethnopharmacol.* , 69, 21-25.
- [28] Gordon, T, Castelli, W. P, Hjortland, M. C, et al. (1977). High density lipoprotein as a protective factor against coronary artery disease: the Framingham Study. *Am J Med.* , 62, 707-14.
- [29] Grundy, S. M. (1998). Hypertriglyceridemia, atherogenic dyslipidemia and the metabolic syndrome. *Am. J. Cardiol.* 81:18B-25B.
- [30] Hepper, F. N. (1963). Emilia in: Hutchinso JJ. and Dalziel JM. (eds) *Flora of West Tropical Africa*, 2nd ed. Crown Agents for Overseas Governments and Administrations, London, Uk. , 2, 244-245.
- [31] Igbakin, A. P. (2009). Comparative studies on hypoglycaemic, hypoproteinaemic, hypocholesterolaemic and hypolipidaemic properties of ethanolic and normal saline extracts of the root of *V. amygdalina* in diabetic rats. *Adv. Enviro. Bio.* , 3(1), 33-38.
- [32] Igwe, C. U, Ojiako, O. A, & Nwaogu, L. A. Onyeze, GOC, (2008). Lipid Lowering Effect of Aqueous Leaf Extract Of *Spondias Mombin* Linn *J. of Pharmacol.* 6 (1) , 10.
- [33] Insull, W. (2006). Clinical utility of bile acid sequestrants in the treatment of dyslipidemia: a scientific review. *South Med J.*; , 99(3), 257-73.
- [34] Knopp, R. H. (1999). Drug treatment of lipid disorders. *N. Engl. J. Med.* , 341, 498-511.
- [35] Kolawole, O. T, Kolawole, S. O, Ayankunle, A. A, & Olaniran, I. O. (2012). Methanolic leaf extract of *Persea americana* protects rats against cholesterol-induced hyperlipidemia. *British J. of Med. Medical Res.*, , 2(2), 235-42.
- [36] Kolawole, O. T, & Ayankunle, A. A. (2012). Seasonal Variation in the Anti-Diabetic and Hypolipidemic Effects of *Momordica charantia* Fruit Extract in Rats *European Journal of Medicinal Plants* , 2(2), 177-185.
- [37] Krauss, R. M. (1982). Regulation of high density lipoprotein levels. *Med. Clin. North. Am.* , 66, 403-30.
- [38] Krauss, R. M, Eckel, R. H, Howard, B, Appel, L. J, Daniels, S. R, Deckelbaum, R. J, et al. (2003). AHA Dietary guidelines: Revision 2000: A statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 2000:102(18):2284-99.
- [39] Lipka, L. Ezetimibe: a first-in-class, novel cholesterol absorption inhibitor. *Cardio-vasc Drug Rev.* , 21(4), 293-312.
- [40] Mckenney, J. (2003). Niacin for dyslipidemia: considerations in product selection. *Am J Health Syst Pharm.*, 60(10), 995-1005.

- [41] Mohammed, R. K, Ibrahim, S, Atawodi, S. E, Eze, E. D, Suleiman, J. B, & Malgwi, I. S. (2012). The study of the effects of n-butanol fraction of *Alchornea cordifolia* leaf extract on lipid profile and liver enzymes in streptozotocin-induced diabetic rats. *Global Journal of medicinal Plant Research.* , 1(1), 1-7.
- [42] Moutzouri, E, Kei, A, Elisaf, M. S, & Milionis, H. J. (2010). Management of dyslipidemias with fibrates, alone and in combination with statins: role of delayed-release fenofibric acid. *Vasc Health Risk Manag.* , 6, 525-539.
- [43] Nwangwa, E. K, & Ekhoye, E. I. (2013). Anti-hyperlipidemic Activity of aqueous extracts of *Carica papaya* seeds in albino rats fed with high fat diet. *Current Trends in Technology and Science, Vol.II*, 2279.
- [44] Nwozo, S, Adaramoye, O, & Ajaiyeoba, E. (2009). Oral Administration of Extract from *Curcuma longa* Lowers Blood Glucose and Attenuates Alloxan-Induced Hyperlipidemia in Diabetic Rabbits, *Pakistan Journal of Nutrition* , 8(5), 625-628.
- [45] Nwozo, S. O. Orojobi BF Adaramoye OA, (2011). Hypolipidemic and antioxidant potentials of *Xylopiya aethiopica* seed extract in hypercholesterolemic rats. *J. Med.Food* 14(1-2) 114-19.
- [46] Odetola, A. A, Akinloye, O, Egunjobi, C, Adekunle, W. A, & Ayoola, A. O. (2006). Possible antidiabetic and antihyperlipidaemic effect of fermented *Parkia biglobosa* (JACQ) extract in alloxan-induced diabetic rats. *Clin. Exp. Pharmacol. Physiol.* , 33(9), 808-12.
- [47] Odey, M. O, Johnson, J. T, Iwara, I. A, Gauje, B, Akpan, N. S, Luke, U. O, Robert, A. E, & Ukpong, K. M. (2012). Effect of anti-hypertensive treatment with root and stem bark extracts on *Nauclea latifolia* on serum lipid profile. *G.J.P. and A. Sc. and Tech.* , 0214, 78-84.
- [48] Ofusori, D. A, Komolafe, O. A, Adewole, O. S, Obuofor, E. M, Fakunle, J. B, & Ayoka, A. O. (2012). Effect of ethanolic leaf extract of *Croton zambesicus* (Mull arg.) on lipid profile in streptozotocin-induced diabetic rat. *Diabetologia croatica.* , 41-2.
- [49] Oguejiofor, O. C, Onwukwe, C. H, & Odenigbo, C. U. (2012). Dyslipidemia in Nigeria: "Prevalence and pattern", (Review). *Ann. African Med.* 11(4), 197-202.
- [50] Ojezele, M. O, & Abatan, O. M. (2011). Hypoglycaemic and coronary risk index lowering effects of *Bauhinia thonningii* in alloxan induced diabetic rats. *Afr. Health Sci.* , 11(1), 85-9.
- [51] Okpe OcheAbdullahi Salman A, Nkeonye Ogechi L, Ilechukwu Chijioke C, Nweke Ogechi, Ihuoma Onyeyirichi, (2012). Hypoglycemic and hypolipidemic Effects of aqueous and ethanolic leaf extracts of *Vitex doniana* (Verbenaceae) in normoglycemic albino rats. *Global Advanced Research Journal of Microbiology* , 1, 173-179.

- [52] Olukunle, J. O, Abatan, M. O, Adenubi, O. T, & Amusan, T. A. (2012). Hypoglycaemic and hypolipidaemic effects of crude extracts and chromatographic fractions of *Morinda morindoides* root bark in diabetic rats. *Acta Vet. Brno.* , 81, 269-74.
- [53] Oluwatosin, A. Adaramoye, Olajumoke Akintayo, Jonah Achem, Michael A, Fafunso, (2008). Lipid-lowering effects of methanolic extracts of *V. amygdalina* leaves in rats fed on high cholesterol diet. *J. Am. Med. Assoc.* , 251, 351-64.
- [54] Oluwole, I. Oyewole¹ and Peter F. Akingbala, (2011). Phytochemical Analysis and Hypolipidemic Properties of *Jatropha tanjorensis* Leaf Extract *Eur. J.1 of Med. Plants*1(4): 180-185,
- [55] OrhueNEJ and Nwanze, EAC, (2006). *Scoparia dulcis* reduces the severity of trypanosome brucei-induced hyperlipidaemia in the rabbit. *Afr. J. of Biotech.* , 5, 883-887.
- [56] Osuji, C. U, Nzerem, B. A, Meludu, S, Dioka, C. E, Nwobodo, E, & Amilo, G. I. (2010). The prevalence of over weight/obesity and dyslipidemia amongst a group of women attending "August" meeting. *Nig. Med. J.* 51(4), 155-159.
- [57] Out, C, Groen, A. K, & Brufau, G. (2012). Bile acid sequestrants: more than simple resins. *Curr Opin. Lipidol.* , 23(1), 43-55.
- [58] Owen, O. J. Amakiri AOA, Karibi-Botoye TA, (2011). Lipid-lowering effects of bitter leaf in boiler chickens fed finishers' mash. *Agr. Bio. J. North Am.* , 2151-7525.
- [59] Patel, S. B. (2004). A novel cholesterol-lowering agent that highlights novel physiologic pathways. *Curr. Cardiol. Rep.* , 6, 439-42.
- [60] Pieper, J. (2003). Overview of niacin formulations: differences in pharmacokinetics, efficacy, and safety. *Am. J. Health Sys. Pharm.* 60(13 suppl 2): SS14., 9.
- [61] Prabhjot, N, Burke, F, Bloesch, A, & Rader, D. (2010). Role of dietary supplements in lowering low-density lipoprotein cholesterol: a review. *J. Clin. Lipidol.* , 4, 248-58.
- [62] Ram, G, & Becker, D. (2011). The role of red yeast rice for the physician. *Curr. Atheroscler. Rep.*, 13, 73-80.
- [63] Sabrin, R. M, Ibrahim, G. A, Mohamed, Z. M, & Banjar, K. M. (2013). Natural antihyperlipidemic agents: Current and future perspectives. *Phytopharmacol.* , 4(3), 492-531.
- [64] Safeer, R. S, & Lacivita, C. L. (2000). Choosing drug therapy for patients with hyperlipidemia. *Am. Fam. Physician* , 61(11), 3371-82.
- [65] Saidu, Y, Nwachukwu, F. C, Bilbis, L. S, Faruk, U. Z, & Abbas, A. Y. (2010). Hypoglycaemic and hypolipidemic Effects of root extracts of *Albizzia chevalieri* in alloxan induced diabetic rats. *Nig. J. Basic and Appl. Sci.* , 18(1), 72-78.
- [66] Shamir, R, & Fisher, E. A. (2000). Dietary therapy for children with hypercholesterolemia. *Am Fam. Physician.* , 61(3), 675-85.

- [67] Sodipo, O A, Abdulrahman, F. I, & Sandabe, U. K. (2011). Total lipid profile, faecal cholesterol, very low density lipoprotein cholesterol (VLDL-C), atherogenic index (A.I) and percent atherosclerosis with aqueous fruit extract of *Solanum macrocarpum* in chronic troton-induced hyperlipidemic albino rats. *Current Res. J.Bio. Sci.* , 4(2), 2026-214.
- [68] Stone, N. J. (1994). Secondary causes of hyperlipidemia. *Med. Clin. North. Am.* , 78, 117-141.
- [69] Stone, N. J. (2001). The optimal dietary strategy to manage risk associated with various dyslipidemias. *Curr. Cardiol. Rep.* , 3, 391-400.
- [70] Tenhola, S, Martikainen, A, Rahiala, E, Herrgard, E, Halonen, P, & Voutilainen, R. (2000). Serum lipid concentrations and growth characteristics in 12-year-old children born small for gestational age. *Pediatr. Res.* ;, 48(5), 623-28.
- [71] Toth, P, Dayspring, T, & Pokrywka, G. (2009). Drug therapy for hypertriglyceridemia: fibrates and omega-3 fatty acids. *Curr. Atheroscler. Rep.*, 11, 71-79.
- [72] Udem, S. C, Ezeonuegbu, U. C, & Obidike, R. I. (2011). Experimental studies on the hypolipidemic and haematological properties of aqueous leaf extract of *Cleistopholis patens* Benth & Diets. (Annonaceae) in hypercholesterolemic rats. *Ann. Med. Health Sci. Res.* (1): 115-21
- [73] Udenze ECCBraide VB, Okwesilieze CN, Akuodor GC, (2012). Pharmacological effects of *Garcinia kola* seed powder on blood sugar, lipid profile and atherogenic index of alloxan-induced diabetes in rats. *Pharmacologia* , 3(12), 693-99.
- [74] Washington, R. L. (1999). Interventions to reduce cardiovascular risk factors in children and adolescents. *Am. Fam. Physician* , 59, 2211-18.
- [75] World Health Organization and International Diabetes Federation in Europe(1989). Diabetes care and research in Europe; Saint Vincent Declaration. *British Med. J.* , 299, 1198-1201.

