JOURNAL OF PHARMA INSIGHTS AND RESEARCH ISSN NO. 3048-5428

REVIEW ARTICLE

Dietary Bioactives for Modulation of Blood Pressure and Mitigation of Cardiovascular Risk

Anusha B[1], Sunitha Kamidi*[2]

[1]*UG Scholar, Department of Pharmaceutical Analysis, Koringa College of Pharmacy, Korangi, Kakinada, Andhra Pradesh, India*
[2]*Assistant Professor, Department of Pharmaceutical Analysis, Koringa College of Pharmacy, Korangi, Kakinada, Andhra Pradesh, India*

Publication history: Received on 7th July 2025; Revised on 16th Aug 2025; Accepted on 23rd August 2025

Article DOI: 10.69613/qdt28023

Abstract: Cardiovascular disease (CVD) is one of the major causes of global morbidity and mortality, with systemic arterial hypertension constituting its most significant modifiable risk factor. Elevated blood pressure drives pathophysiological processes like vascular remodeling, endothelial dysfunction, and chronic inflammation, culminating in myocardial infarction and stroke. Dietary nutraceuticals, which are bioactive compounds derived from functional foods and plant extracts, offer compelling, accessible avenues for mitigating these underlying pathologies. The main compounds, including omega-3 fatty acids, organosulfur compounds (from *Allium sativum*), catechins (from *Camellia sinensis*), and curcuminoids (from *Curcuma longa*), show notable antihypertensive and cardioprotective effects. These beneficial actions are primarily mediated through enhanced nitric oxide bioavailability, modulation of the Renin-Angiotensin-Aldosterone System (RAAS), and reduction of systemic oxidative stress. Moreover, specific phytochemicals like anthocyanins (found in berries) and nutrients such as potassium (high in kiwifruit) contribute positively to improving vascular compliance and maintaining critical electrolyte balance. The existing evidence, derived from epidemiological data and randomized controlled trials, suggests a supportive role for these agents. However, rigorous future investigation is required to establish standardized dosages, optimal formulations, and long-term clinical outcomes. These dietary components are powerful, accessible complements to conventional therapeutic options for primary and secondary CVD prevention.

Keywords: Hypertension; Nutraceuticals; Endothelial Function; Oxidative Stress; Cardiovascular Disease

1. Introduction

Cardiovascular disease (CVD), encompassing conditions like coronary artery disease, stroke, and heart failure, accounts for over 18 million deaths annually, solidifying its position as the leading global health crisis [1]. A major contributor to this epidemic is arterial hypertension, a persistently elevated systemic blood pressure that accelerates atherosclerosis and organ damage across the renal, cerebral, and cardiac systems. Hypertension is highly prevalent, particularly in adults over the age of sixty [2].

Effective management of this condition is paramount for minimizing the global burden of CVD. The therapeutic landscape for hypertension traditionally relies on pharmacological agents such as diuretics, Angiotensin-Converting Enzyme (ACE) inhibitors, and calcium channel blockers. However, lifestyle modification, particularly through optimized nutrition, serves as the foundational component of both prevention and treatment.

In recent decades, the focus has shifted towards isolating and utilizing specific dietary compounds that confer health benefits beyond basic nutrition. The term nutraceutical was introduced to describe a food or food component that provides medical or health benefits, including disease prevention and treatment [3]. These substances bridge the gap between nutrition and pharmaceuticals and include a wide variety of bioactive molecules such as polyphenols, carotenoids, and polyunsaturated fatty acids.

Nutraceuticals differ from conventional functional foods, which are generally enriched in their original matrix (e.g., fortified yogurt); nutraceuticals are typically consumed in supplement forms, such as capsules, tablets, or powders, and are utilized either as preventative measures or as supportive adjuncts alongside conventional medicine [4, 5]. The growing interest in these natural compounds stems from their established safety profiles and their potential to target multiple pathophysiological pathways associated with chronic diseases, including hypertension, oxidative stress, and dyslipidemia [6].

* Corresponding author: Sunitha Kamidi

2. Pathophysiology

Hypertension is clinically defined by a sustained elevation in systolic blood pressure (SBP) greater than 130 mmHg or diastolic blood pressure (DBP) greater than 80 mmHg. Its etiology is complex and multifactorial, involving a delicate interplay of genetic predispositions, environmental factors, and lifestyle choices.

2.1. Mechanisms Driving Elevated Blood Pressure

The regulation of systemic blood pressure is dictated by two primary physiological variables: cardiac output and total peripheral resistance [7]. Hypertension arises when an imbalance in these factors occurs, driven by several key mechanisms:

2.1.1. Vascular Dysfunction

The healthy endothelium releases powerful vasodilators, notably nitric oxide (NO), which relaxes smooth muscle and maintains low peripheral resistance. In hypertension, endothelial dysfunction leads to decreased NO bioavailability, often due to oxidative stress, resulting in sustained vasoconstriction and increased vascular stiffness [8].

2.1.2. Renin-Angiotensin-Aldosterone System (RAAS) Overactivity

The RAAS plays a crucial role in regulating blood volume and vascular tone. Overactivation of this cascade leads to increased production of Angiotensin II, a potent vasoconstrictor and stimulator of aldosterone release, which promotes renal sodium and water retention, thereby raising blood pressure [9].

2.1.3. Sympathetic Nervous System (SNS) Hyperactivity

Chronic stress or physiological anomalies can lead to excessive sympathetic outflow, causing increased heart rate and peripheral vasoconstriction, contributing significantly to essential hypertension [10].

2.1.4. Renal Sodium Handling

Impaired pressure natriuresis, where the kidneys fail to adequately excrete sodium in response to pressure changes, contributes to volume expansion and chronic blood pressure elevation.

Table 1. Pathophysiological Targets for Nutraceutical Intervention

Pathophysiological Mechanism	Consequence in Hypertension	Nutraceutical Target Examples
Endothelial Dysfunction (↓ NO)	Sustained vasoconstriction, increased peripheral resistance, arterial stiffness.	L-Arginine, Polyphenols (EGCG, Anthocyanins), Organosulfur Compounds H_2S.
Oxidative Stress (↑ ROS)	Degradation of NO, vascular inflammation, LDL oxidation.	Curcumin, Green Tea Catechins, Vitamin C (Kiwifruit), Anthocyanins.
RAAS Overactivity (↑ Angiotensin II)	Enhanced vasoconstriction, aldosterone release, salt/water retention.	Peptides (e.g., from milk proteins), Green Tea EGCG (ACE inhibition).
Electrolyte Imbalance (↑ Na, ↓ K/Mg)	Volume expansion, increased sympathetic tone, vascular smooth muscle tension.	Potassium (Kiwifruit), Magnesium, Dietary Fiber (indirectly).

2.2. Etiological Classifications

Hypertension is primarily classified into two distinct types based on its underlying cause:

2.2.1. Essential (Primary) Hypertension

This is the most common form, accounting for approximately 90-95% of cases, where no single, identifiable cause can be determined [11]. Its development is slow and progressive, strongly correlating with lifestyle risk factors such as high dietary sodium intake, insufficient potassium and magnesium consumption, physical inactivity, obesity, chronic alcohol intake, and aging.

2.2.2. Secondary Hypertension

This type is caused by a specific underlying medical condition. It is typically more severe and abrupt in onset. Common causes include chronic kidney disease, adrenal gland disorders (which may over-secrete hormones like aldosterone or cortisol), thyroid pathologies, and obstructive sleep apnea [12]. Identifying and treating the underlying pathology is crucial for resolving secondary hypertension.

2.3. Conventional Therapeutic Options

Effective management necessitates a stepwise approach, beginning with fundamental lifestyle modifications (e.g., following a low-sodium, potassium-rich diet like the DASH diet, achieving a healthy weight, and engaging in regular exercise). When lifestyle changes are insufficient to reach the target blood pressure goal—typically below 130/80 mmHg for high-risk individuals—pharmacological intervention is initiated. First-line agents often include thiazide diuretics, ACE inhibitors, Angiotensin II Receptor Blockers (ARBs), or calcium channel blockers. Combination therapy, involving two or more distinct drug classes, is often required to achieve optimal blood pressure control [13]

3. Therapeutic Potential of Dietary Bioactives

Nutraceuticals are gaining prominence as valuable adjunctive agents that can mitigate hypertension-related risk factors through complementary mechanisms to pharmacological treatments. The compounds discussed below have shown antihypertensive, anti-inflammatory, and anti-atherogenic properties.

3.1. Omega-3 Polyunsaturated Fatty Acids (PUFAs)

Omega-3 fatty acids, specifically eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are key dietary components primarily sourced from marine oils [14]. Their benefits in cardiovascular health extend beyond traditional lipid lowering, impacting blood pressure regulation directly.

3.1.1. Mechanistic Actions in Vascular Tissue

EPA and DHA contribute to vasodilation by increasing the synthesis of local eicosanoids, such as prostacyclin PGI-2, which acts as a powerful vasodilator [15]. Additionally, omega-3 PUFAs are integrated into cell membranes, altering their fluidity and directly affecting the function of ion channels and signal transduction pathways involved in vascular smooth muscle contraction. They also exert a significant anti-inflammatory action by shifting the balance away from pro-inflammatory mediators (derived from omega-6 fatty acids) toward less inflammatory or anti-inflammatory mediators, thereby preserving endothelial function, which is critical for blood pressure control [16].

Table 2. Primary Antihypertensive Nutraceuticals and Mechanisms of Action

Nutraceutical Source	Bioactive Compound(s)	Mechanism of Action	Antihypertensive Efficacy
Allium Sativum (Garlic)	Allicin, Organosulfur Compounds	H_2S generation, potent vasorelaxation, ACE inhibition activity.	Modest to Significant (dose-dependent)
Camellia Sinensis (Green Tea)	Epigallocatechin gallate (EGCG)	Enhances eNOS activity, ACE inhibition, strong anti-oxidation.	Modest
Omega-3 Fatty Acids (Fish Oil)	EPA, DHA	Increases prostacyclin PGI_2 synthesis, anti-inflammation, membrane fluidity effects.	Modest to Significant (especially DBP)
Curcuma Longa (Turmeric)	Curcuminoids	Suppresses NF-kappa-B inflammatory pathway, improves endothelial function.	Modest
Berries (Blueberries, etc.)	Anthocyanins	Increases NO bioavailability, reduces arterial stiffness, scavenges ROS.	Modest

3.2. Antioxidant and Anti-inflammatory Phytochemicals

Oxidative stress, characterized by an overproduction of reactive oxygen species (ROS), is a central driver of hypertension, primarily by damaging the endothelium and degrading nitric oxide [17]. Various plant-derived phytochemicals act as potent antioxidants and anti-inflammatory agents to counteract this effect.

3.2.1. Allium Sativum (Garlic)

Garlic has a long history in traditional medicine for treating various cardiovascular conditions. Its antihypertensive activity is attributed mainly to its organosulfur compounds, such as allicin and its derivatives. The primary proposed mechanism involves the conversion of garlic-derived organic polysulfides by red blood cells into hydrogen sulfide [18]. H_2S is a gaseous signaling molecule that relaxes vascular smooth muscle, induces vasodilation, and significantly reduces blood pressure. Clinical studies have shown that supplementation with aged garlic extract can reduce SBP by 7–16 mmHg and DBP by 5–9 mmHg in patients with uncontrolled hypertension, indicating a clinically relevant hypotensive effect [19]. Moreover, these sulfur compounds interfere with Angiotensin II-mediated signaling and reduce platelet aggregation, contributing to overall cardioprotection [20].

3.2.2. Camellia Sinensis (Green Tea)

Green tea is rich in polyphenols, particularly catechins, with the most pharmacologically active being epigallocatechin gallate (EGCG). These compounds possess strong antioxidant properties that scavenge free radicals, protecting the endothelium from damage and enhancing NO bioavailability. EGCG specifically influences several pathways relevant to hypertension [21]. It can inhibit key enzymes involved in vasoconstriction, such as ACE, effectively mimicking the action of pharmacological ACE inhibitors. EGCG improves vascular elasticity and compliance by promoting endothelial NO production, leading to reduced peripheral resistance and a modest but significant lowering of blood pressure, particularly in individuals with pre-hypertension or early-stage hypertension [22]

3.2.3. Curcuma Longa (Turmeric)

The primary bioactive components of turmeric are the curcuminoids, with curcumin being the most studied. Chronic, low-grade inflammation is intrinsically linked to the development of hypertension, as inflammatory cytokines contribute to arterial stiffness and vascular remodeling [23]. Curcumin's therapeutic potential in hypertension lies in its ability to suppress inflammatory pathways, such as NF-kappa B signaling, and its powerful antioxidant activity [24]. Crucially, curcumin is known to improve endothelial function, comparable to that achieved through exercise or certain medications [25]. It enables better NO-mediated vasodilation, thereby promoting blood pressure regulation and reducing arterial stiffness, a significant predictor of future cardiovascular events by restoring the integrity and function of the endothelial lining.

3.3. Fruit-Derived Bioactives

Fruits provide essential micronutrients and phytochemicals that directly support vascular health and help maintain fluid and electrolyte balance, which is fundamental to blood pressure regulation.

3.3.1. Berry Anthocyanins

Berries, such as blueberries and strawberries, are characterized by high concentrations of anthocyanins, which are responsible for their vibrant color and potent antioxidant profile [26]. Anthocyanins exert their antihypertensive effects via two main mechanisms: improved endothelial function and direct arterial stiffness reduction. These flavonoids increase the expression of endothelial NO synthase (eNOS), leading to greater NO production [27]. Moreover, the regular consumption of berry bioactives has been correlated with reduced total peripheral resistance and improvements in blood pressure and arterial stiffness, particularly in high-risk populations like postmenopausal women with metabolic syndrome [28].

3.3.2. Actinidia Deliciosa (Kiwifruit)

Kiwifruit provides significant levels of antioxidants (e.g., Vitamin C) and is an excellent source of potassium [29]. The inverse relationship between dietary potassium intake and blood pressure is well established.

Potassium facilitates the renal excretion of sodium and directly contributes to relaxing the smooth muscle in blood vessel walls, thereby decreasing systemic blood pressure and counteracting the pressor effects of high dietary sodium [30]. The high fiber and bioactive enzyme (actinidin) content also contribute indirectly to metabolic health and digestion, which are essential components of overall cardiovascular wellness [31].

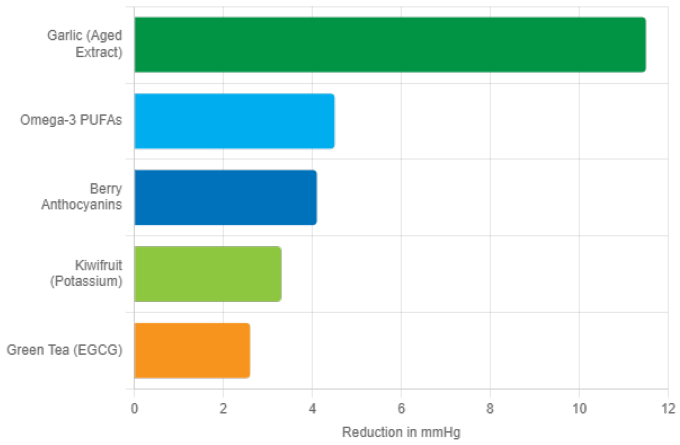

Figure 1. Comparative Efficacy of Nutraceuticals on Systolic Blood Pressure (SBP) [14-18]

4. Lipid and Glucose Modulating Nutraceuticals

Hypertension frequently coexists with other metabolic disorders, most notably dyslipidemia (abnormal cholesterol/triglyceride levels) and glucose intolerance, forming the cluster of risk factors termed Metabolic Syndrome. Addressing these co-morbidities with nutraceuticals provides an integrated strategy for reducing overall cardiovascular risk [32]

4.1. Cholesterol-Lowering Agents

High levels of low-density lipoprotein cholesterol (LDL-C), particularly oxidized LDL-C, contribute directly to endothelial damage and atherosclerotic plaque formation, which aggravates hypertension.

4.1.1. Plant Sterols and Stanols

Plant sterols and stanols are structurally similar to cholesterol and compete with dietary and biliary cholesterol for incorporation into micelles in the small intestine [33]. This competition significantly reduces the absorption of cholesterol, leading to a compensatory uptake of LDL-C from the bloodstream by the liver. Consumption of approximately 2 grams per day can reduce LDL-C levels by 5% to 15%, offering a valuable non-pharmacological means of managing hypercholesterolemia, a critical step in slowing the progression of atherosclerosis and vascular stiffness [34]

4.1.2. Dietary Fiber (Soluble Fiber)

Soluble fibers, found abundantly in oats, barley, and psyllium husks, decrease LDL-C by two mechanisms: binding bile acids in the intestine, forcing the liver to synthesize new bile acids from cholesterol, and through the production of short-chain fatty acids (SCFAs) via microbial fermentation. SCFAs, such as propionate, may inhibit hepatic cholesterol synthesis [35]. While the direct effect on blood pressure is modest, the pronounced impact on total and LDL cholesterol is vital for reducing the vascular burden associated with hypertension.

4.2. Glucose Homeostasis Modulators

Type 2 diabetes mellitus is a major risk factor for hypertension. Bioactives that improve insulin sensitivity and glucose control contribute indirectly to blood pressure regulation by reducing chronic inflammation and vascular stiffening associated with hyperglycemia.

4.2.1. Cinnamon and Berberine

Cinnamon-derived polyphenols and the alkaloid berberine have been widely studied for their insulin-sensitizing effects. Berberine, in particular, has been shown to activate AMP-activated protein kinase (AMPK), a metabolic master switch that improves insulin signaling and glucose utilization [36]. Improved glucose control lessens the production of advanced glycation end products (AGEs), which are key drivers of vascular stiffness and endothelial dysfunction in hypertensive diabetic patients [37]

Table 3. Dual-Action Nutraceuticals for Metabolic Syndromes

Nutraceutical Agent	Effects on Cardiovascular System	Effect on Metabolism	Primary Clinical Target
Berberine	Improves endothelial function, reduces vascular inflammation.	Activates AMPK, lowers fasting glucose and HbA1c, reduces dyslipidemia.	Type 2 Diabetes & Hypertension
Plant Sterols/Stanols	Reduces LDL-C, slowing atherosclerosis and vascular stiffening.	Competitively inhibits intestinal cholesterol absorption.	Hypercholesterolemia
Soluble Fiber	Indirectly reduces vascular burden by lowering cholesterol.	Binds bile acids, promotes SCFA production (e.g., Propionate), lowers LDL-C.	Dyslipidemia & Atherosclerosis Risk
Omega-3 Fatty Acids	Vasodilation, reduced inflammation, modest BP lowering.	Dramatically lowers circulating plasma triglycerides.	Hypertriglyceridemia

5. Synergistic Effects and Combinatorial Strategies

The multifactorial nature of hypertension, which involves chronic inflammation, oxidative stress, RAAS overactivity, and metabolic dysregulation, suggests that a single nutraceutical agent may offer limited efficacy in complex cases. Therefore, strategies that combine multiple bioactives targeting different pathological pathways simultaneously are becoming increasingly relevant.

5.1. Polyphenol-Omega-3 Synergism

The combination of highly bioavailable omega-3 fatty acids (EPA/DHA) and antioxidant polyphenols (e.g., from green tea or grapes) demonstrates greater efficacy than either component alone [38]. Omega-3s exert anti-inflammatory effects by modulating eicosanoid pathways, while polyphenols protect the vascular endothelium from oxidative damage. The synergistic action involves polyphenols preserving the integrity of the omega-3 molecules themselves, preventing their oxidation, and collectively leading to enhanced NO bioavailability and better vascular health [39]

5.2. Combination with Conventional Therapy

Nutraceuticals should be viewed as complements, not substitutes, for conventional antihypertensive medications. For instance, in patients taking ACE inhibitors, garlic supplementation may offer an additive benefit by activating a parallel, H_2S-mediated vasodilatory pathway, further reducing peripheral resistance. Similarly, coenzyme Q10, a crucial component of mitochondrial energy production and a powerful antioxidant, is often depleted by statin drugs used to treat co-morbid dyslipidemia. Supplementing CoQ10 in such patients not only addresses the drug-induced deficiency but also supports myocardial energy requirements and reduces oxidative stress on the heart [40]

5.3. Role in Atypical Cardiovascular Manifestations

The scope of nutraceutical intervention extends beyond primary hypertension to address complications:

5.3.1. Management of Cardiac Arrhythmias

Electrolyte imbalances, particularly of potassium and magnesium, are strongly linked to the risk of cardiac arrhythmias. Magnesium, which is essential for over 300 enzymatic reactions, plays a role in regulating ion channels in the myocardium. Deficiency can increase myocardial irritability and the risk of atrial fibrillation and ventricular ectopy [41]. Supplementation with magnesium and maintaining adequate potassium intake (often achieved through high-potassium foods like kiwifruit) provides a crucial protective mechanism against these hypertension-associated complications.

5.3.2. Support in Heart Failure

Coenzyme Q10 and L-carnitine have been researched as supportive agents in heart failure (HF), a common endpoint of chronic uncontrolled hypertension. CoQ10 improves energy production within compromised heart muscle cells, while L-carnitine enhances fatty acid metabolism in the myocardium, potentially improving cardiac output and quality of life in HF patients [42]. These are adjuncts that address the metabolic consequences of the heart muscle damage caused by chronic pressure overload.

Table 4. Synergistic Approaches to Cardiovascular Risk Reduction

Combined Agents	Rationale	Combined Effect on CVD Markers
Polyphenols + Omega-3s	Polyphenols protect fragile PUFAs from oxidation; combined anti-inflammatory pathways.	Enhanced NO production, greater reduction in CRP and vascular markers.
Garlic + ACE Inhibitors	Garlic acts via H2-vasodilation (parallel pathway); ACE inhibitor blocks Angiotensin II.	Additive blood pressure lowering, dual pathway modulation.
CoQ10 + Statin Therapy	CoQ10 replenishes levels depleted by statins; improves mitochondrial function.	Reduces statin-induced myopathy risk; supports myocardial energy output.
Magnesium + Potassium	Magnesium regulates ion channels; Potassium excretes sodium and relaxes vessels.	Stabilizes heart rhythm, optimizes electrolyte balance, facilitates natriuresis.

6. Conclusion

Natural bioactive compounds, or nutraceuticals, sourced from agents like garlic, green tea, turmeric, and fruits such as berries and kiwifruit, offer compelling, evidence-based support for cardiovascular health. These agents function by targeting the fundamental mechanisms of hypertension, including improving endothelial NO bioavailability, modulating the RAAS cascade, and aggressively reducing systemic oxidative stress and inflammation. Moreover, agents targeting related metabolic disorders like dyslipidemia (plant

sterols, fiber) and glucose intolerance (berberine) enhance the overall strategy. While the observed benefits—such as reduced blood pressure, improved lipid profiles, and enhanced vascular function—are promising and well-supported by several studies, it is crucial to recognize that these substances serve as complementary interventions. Future clinical research must focus on establishing robust, large-scale randomized controlled trials to determine optimal dosages, standardization parameters, and the long-term impact of these dietary bioactives on hard clinical endpoints (e.g., incidence of stroke and myocardial infarction.

References

[1] Roth GA, Huffman MD, Moran AE, Feigin V, Mensah GA, Naghavi M, et al. Global and Regional Patterns in Cardiovascular Mortality From 1990 to 2013. Circulation. 2015;132(19):1667-78.

[2] Qureshi M, Hassan M. Blood Pressure and Hypertension Prevalence among Different Age Groups: A Cross-Sectional Investigation. Int J Med Sci Clin Res. 2023;5(11):625-33.

[3] DeFelice SL. The nutraceutical revolution: its impact on food and healthcare. Trends Food Sci Technol. 1995;6(2):59-61.

[4] Granato D, Barba FJ, Bursac Kovacevic D, Lorenzo JM, Cruz AG, Putnik P. Functional Foods: Product Development, Technological Trends, Efficacy Testing, and Safety. Annu Rev Food Sci Technol. 2020;11:93-118.

[5] Roberfroid MB. Concepts and benefits of functional foods. Nutr Rev. 1999;57(5 Pt 2):S1-12.

[6] Nasri H, Baradaran A, Shirzad H, Rafieian-Kopaei M. New concepts in nutraceuticals as alternative treatments for cardiovascular diseases. Int J Prev Med. 2014;5(12):1487-99.

[7] Guyton AC, Hall JE. Textbook of Medical Physiology. 14th ed. Philadelphia: Elsevier; 2020.

[8] Schiffrin EL. Vascular remodeling and endothelial function in hypertension. Can J Cardiol. 2018;34(5):S41-5.

[9] Crowley SD, Gurley SB, Herrera MJ, Ruiz P, Griffiths R, Mondoñedo JR, et al. Angiotensin II–dependent hypertension in Bmpr1a conditional knockout mice. J Clin Invest. 2005;115(7):2216-23.

[10] Grassi G, Mark A, Esler M. The sympathetic nervous system in hypertension and heart failure. Circ Res. 2015;116(2):325-36.

[11] Carey RM, Whelton PK, Casey DE Jr, Cohn JN, Curtis LH, Fendrick SB, et al. Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2018;71(6):e13-115.

[12] Williams TA, Reincke M. Secondary hypertension: prevalence, diagnosis, and treatment. Lancet. 2022;400(10349):319-32.

[13] Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Himmelfarb CD, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2018;71(19):e127-248.

[14] Lavie CJ, Milani RV, Mehra MR, Ventura HO. Omega-3 polyunsaturated fatty acids and cardiovascular diseases. J Am Coll Cardiol. 2009;54(7):585-94.

[15] Simopoulos AP. Omega-3 fatty acids in health and disease and in growth and development. Am J Clin Nutr. 1991;54(3):438-63.

[16] Harris WS, Mozaffarian D, Rimm E, Kris-Etherton P. Optimal serum concentrations of EPA and DHA for cardiovascular risk reduction: an update to the scientific statement from the American Heart Association. Circulation. 2020;142(21):e208-20.

[17] Touyz RM. Oxidative stress and vascular damage in hypertension. Curr Hypertens Rep. 2007;9(4):315-9.

[18] Benavides GA, Squadrito GI, Mills RW, Patel HD, Musah RA, Ravi K, et al. Hydrogen sulfide as an H2 donating vasodilator in vitro and in vivo. Cardiovasc Res. 2007;75(2):290-300.

[19] Ried K, Travica N, Sali A. The effect of aged garlic extract on blood pressure and other cardiovascular risk factors: a systematic review and meta-analysis. Front Nutr. 2019;6:146.

[20] Al-Qattan KK, Hanif J, Al-Saleh E, Al-Amer MM. Garlic (Allium sativum) as a potential inhibitor of angiotensin II-induced vasoconstriction in isolated rat thoracic aorta. Int J Clin Exp Med. 2013;6(7):563-71.

[21] Cooper KA, Morre DM, Morre DJ. Medicinal benefits of green tea: Part I. Review of noncancer applications. J Altern Complement Med. 2205;11(3):521-8.

[22] Khalesi S, Sun D, Buys N, Jamshidi R, Nikbakht E, Khosravi M, et al. Green tea catechins and blood pressure: a systematic review and meta-analysis of randomised controlled trials. Eur J Nutr. 2017;56(3):1291-302.

[23] Gotsis E, Tsioufis C, Tousoulis D, Stefanadis C. Inflammation in the vascular wall: current perspectives in hypertension. J Hypertens. 2011;29(9):1657-65.

[24] Menon VP, Sudheer AR. Antioxidant and anti-inflammatory properties of curcumin. Adv Exp Med Biol. 2007;595:105-25.

[25] DiSilvestro RA, Joseph E, Zhao S, Broderick T. Diverse effects of curcumin on the cardiovascular system. Nutr J. 2012;11:78.

[26] Kalt W, Cassidy A, Howard LR, Lydia K, Joseph A, Lila MA, et al. The potential health benefits of red grapes and blueberries: a review of the mechanisms and the clinical evidence. J Agric Food Chem. 2020;68(32):8599-605.

[27] Wang X, Yang Y, Zhang F, Niu Z, Wang S. Anthocyanin-rich extract from black soybean seed coat prevents endothelial dysfunction in high-fructose diet-induced hypertension. J Nutr Biochem. 2014;25(11):1114-23.

[28] Johnson SA, Figueroa A, Navaei N, Wong A, Kalfon R, Ormsbee MJ, et al. Daily blueberry consumption improves blood pressure and arterial stiffness in postmenopausal women with pre- and stage 1 hypertension: a randomized, double-blind, placebo-controlled clinical trial. J Acad Nutr Diet. 2015;115(3):369-77.

[29] Korpelainen R, Korpelainen H, Korpelainen K. Kiwifruit and cardiovascular health. Adv Food Nutr Res. 2022;102:183-210.

[30] Aburto NJ, Hanson S, Gutierrez H, Hooper L, Elliott P, Zhao W, et al. Effect of increased potassium intake on cardiovascular risk factors and disease: systematic review and meta-analyses. BMJ. 2013;346:f1378.

[31] Stonehouse W, Gammon CS, Beck KL, Conlon CA, von Hurst PR, Kruger R. Kiwifruit and the gastrointestinal tract: a review of the evidence. Adv Food Nutr Res. 2013;68:21-33.

[32] Sowers JR, Epstein M, Frohlich ED. Diabetes mellitus and hypertension. An update. Hypertension. 2001;37(4):1053-9.

[33] Katan MB, Grundy SM, Jones P, Law M, Miettinen T, Paoletti R. Efficacy and safety of plant stanols and sterols in the management of blood cholesterol levels. Mayo Clin Proc. 2003;78(8):965-72.

[34] Ras RT, Geleijnse JM, Trautwein EA. LDL-cholesterol-lowering effect of plant sterols and stanols is not subject to a threshold: a systematic review and meta-analysis. Crit Rev Food Sci Nutr. 2014;54(1):24-35.

[35] Theuwissen E, Mensink RP. Soluble fiber slows the release of bile acids from the small intestine, potentially increasing enterohepatic recirculation: a random-order, crossover study in healthy humans. J Nutr. 2011;141(12):2243-8.

[36] Kong W, Zhang H, Song DQ, Xue R, Zhao S, Wei J, et al. Berberine activates AMPK and improves endothelial function in diabetic rats. J Cardiovasc Pharmacol. 2009;53(4):316-22.

[37] Schalkwijk CG, Stehouwer CDA, van Hinsbergh VWM. AGEs, AGE receptors, and vascular dysfunction in diabetes mellitus. Cardiovasc Res. 2004;63(4):599-609.

[38] Ciccocioppo F, Binda E, Cannella S, Lazzarino G, Tana M, Cifani C, et al. Antioxidants and omega-3 fatty acids: a combined approach for cardiovascular protection. Nutrients. 2018;10(9):1243.

[39] Poudyal H, Panchal SK, Brown L. PPAR alpha activation attenuates cardiovascular structural and functional changes in the metabolic syndrome. J Lipid Res. 2010;51(11):3214-24.

[40] Deichmann R, Lavie C, Andrews S. Coenzyme Q10 and statin-induced myopathy. South Med J. 2007;100(3):286-90.

[41] Khan AM, Lubinski V, Al-Khatib SM. Magnesium and the heart: the role of magnesium in disorders of the heart. Am Heart J. 2007;153(3):575-80.

[42] Kalra J, Bajaj J, Kaur S, Kaur G, Kumar R. Role of L-carnitine and coenzyme Q10 in heart failure. J Cardiovasc Dis Res. 2012;3(2):64-8.