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Review Article

Ginger: From the Roots to the Rhythm

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ABSTRACT

Cardiovascular diseases (CVDs) remain the leading cause of global mortality despite advances in pharmacotherapy. Interest in nutraceuticals with multitarget mechanisms has intensified, particularly those supported by both traditional medicine and modern molecular evidence. Zingiber officinale (ginger) has a long history of use in Ayurvedic and traditional medical systems and is increasingly supported by experimental and clinical research. This review integrates pharmacognostic, phytochemical, molecular, and clinical evidence, with special emphasis on the detailed mechanisms of action of gingerols and shogaols. Mechanistic insights derived from recent signaling-pathway–focused studies are incorporated into an existing cardiovascular review framework. Ginger exerts cardioprotective effects through antioxidant, anti-inflammatory, antihypertensive, antihyperlipidemic, antiplatelet, and metabolic regulatory pathways involving Nrf2/ARE, NF-κB, MAPK, AMPK, PI3K/Akt, eNOS, COX, and PPAR signaling. Human trials and meta-analyses demonstrate modest but clinically relevant reductions in blood pressure, lipids, glycemia, and inflammatory markers. Collectively, ginger represents a safe, low-cost adjunctive strategy for cardiovascular prevention and risk reduction, although long-term standardized trials with hard endpoints remain necessary.

INTRODUCTION

Cardiovascular diseases account for approximately one-third of global deaths and are driven by interconnected mechanisms including oxidative stress, chronic inflammation, endothelial dysfunction, dyslipidemia, hypertension, and thrombosis. While conventional therapies reduce mortality, residual cardiovascular risk persists, prompting interest in complementary interventions with multitarget activity and favorable safety profiles. Ginger (Zingiber officinale Roscoe), a widely consumed culinary spice and medicinal rhizome, has emerged as a promising candidate due to its rich phytochemistry and pleiotropic biological effects.

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2. PHARMACOGNOSTIC AND ETHNOMEDICAL OVERVIEW

2.1 Botanical identity and quality standards

Ginger is a perennial herb of the family Zingiberaceae. The medicinal rhizome contains oleoresin cells, starch granules, and volatile oils. Pharmacopoeial standards require ≥1.0% volatile oil and standardized levels of marker compounds such as [6]-gingerol and [6]shogaol.

2.2 Traditional cardiovascular relevance

In Ayurveda, ginger (Śuṇṭhī) is classified as hṛidya (cardiotonic), dīpana (metabolic stimulant), and śothahara (anti-inflammatory). These concepts parallel modern interpretations of enhanced circulation, metabolic regulation, and vascular protection.

3. PHYTOCHEMICAL CONSTITUENTS RELEVANT TO CARDIOVASCULAR HEALTH

Ginger's biological activity is primarily attributed to phenolic ketones, notably gingerols ([6]-, [8]-, [10]-gingerol) and their dehydration products shogaols ([6]-, [8]-, [10]-shogaol).

Additional contributors include diarylheptanoids, flavonoids, and volatile terpenes.

Structural differences between gingerols and shogaols confer distinct potency and signaling behavior, with shogaols generally exhibiting stronger electrophilic and antioxidant effects.

4. INTEGRATED MOLECULAR MECHANISMS OF ACTION IN CARDIOVASCULAR PROTECTION

The figure illustrates the multitarget cardioprotective actions of ginger bioactives (gingerols and shogaols). Gingerols and shogaols activate antioxidant pathways (Nrf2/ARE), suppress pro-inflammatory signaling (NF-κB, MAPK), modulate metabolic and vascular regulators (AMPK, PI3K/Akt, PPAR-γ), enhance endothelial nitric oxide synthase (eNOS) activity, and inhibit cyclooxygenase (COX)-mediated thromboxane synthesis. Collectively, these pathways converge to reduce oxidative stress, inflammation, endothelial dysfunction, hypertension, atherosclerosis, thrombosis, and metabolic syndrome.

4.1 Antioxidant and redox-regulating mechanisms

Oxidative stress is central to endothelial dysfunction and atherogenesis. Gingerols and shogaols directly scavenge reactive oxygen species and modulate redox-sensitive signaling pathways. Shogaols, due to their α,β-unsaturated carbonyl structure, interact with Keap1, promoting dissociation and nuclear translocation of Nrf2. Activated Nrf2 binds antioxidant response elements (ARE), inducing expression of heme oxygenase-1, superoxide dismutase, catalase, glutathione peroxidase, and related enzymes. Gingerols also enhance antioxidant enzyme activity, likely via indirect MAPK- and ERK-dependent Nrf2 activation. These mechanisms reduce lipid peroxidation, preserve nitric oxide bioavailability, and attenuate oxidative vascular injury.

4.2 Anti-inflammatory signaling pathways

Chronic vascular inflammation underlies plaque formation and instability. Gingerols and shogaols suppress inflammatory cascades by inhibiting NF-κB activation and downstream transcription of COX-2, iNOS, TNF-α, IL-6, and IL-1β. Gingerols inhibit MAPK and PI3K/Akt signaling, while shogaols additionally activate PPAR-γ,

contributing to macrophage polarization toward an anti-inflammatory phenotype. Crosstalk between Nrf2 and NF-κB further links antioxidant and anti-inflammatory effects, producing sustained vascular protection.

4.3 Endothelial function and antihypertensive mechanisms

Hypertension is strongly associated with endothelial dysfunction and calcium dysregulation. Gingerols increase endothelial nitric oxide synthase phosphorylation via AMPK and PPARδ signaling, enhancing nitric oxide release and vasodilation. Both gingerols and shogaols exhibit calcium-channel–blocking activity, reduce angiotensin II receptor expression, and activate cGMP-dependent potassium channels. These combined actions result in reduced peripheral resistance and clinically measurable blood pressure reductions.

4.4 Lipid metabolism and anti-atherogenic effects

Ginger improves lipid profiles by inhibiting hepatic HMG-CoA reductase, enhancing LDL receptor expression, and promoting bile acid excretion. Activation of AMPK and PPAR-γ suppresses lipogenesis and adipocyte lipid accumulation. Gingerols additionally inhibit pancreatic lipase and intestinal lipid absorption. These mechanisms reduce LDL-cholesterol, triglycerides, and foam-cell formation, thereby attenuating atherosclerotic plaque development.

4.5 Antiplatelet and antithrombotic activity

Thromboxane-mediated platelet aggregation is a key event in myocardial infarction and stroke. Gingerols and shogaols inhibit arachidonic acid metabolism by modulating COX-1/COX-2 activity and reducing thromboxane B_2 formation. The net effect favors a prostacyclin-dominant environment, reducing platelet aggregation without substantial bleeding risk at dietary doses.

4.7 Metabolic and glycemic regulation relevant to CVD risk

Insulin resistance and hyperglycemia exacerbate cardiovascular risk. Gingerols activate AMPK-GLUT4 signaling to enhance glucose uptake in skeletal muscle and suppress hepatic gluconeogenesis. Shogaols additionally modulate TRPV1-mediated calcium signaling and improve insulin sensitivity independent of insulin secretion. These metabolic effects indirectly reduce endothelial damage and vascular inflammation.

5. EXPERIMENTAL AND CLINICAL EVIDENCE

Preclinical models consistently demonstrate reductions in oxidative stress, blood pressure, lipid accumulation, thrombosis, and myocardial injury following ginger administration. Human randomized controlled trials and recent meta-analyses report modest but significant reductions in systolic blood pressure, LDL-cholesterol, triglycerides, fasting glucose, and inflammatory biomarkers with daily doses of 1–3 g ginger or standardized extracts.

6. Safety, Dosage, and Drug Interactions

Ginger is well tolerated at dietary and supplemental doses up to 4 g/day. Mild gastrointestinal symptoms are the most common adverse effects. Due to antiplatelet activity, caution is advised when combined with anticoagulants, although clinically significant bleeding is rare.

7. CONCLUSION

Integrating traditional knowledge with contemporary molecular pharmacology reveals ginger as a multitarget cardioprotective agent. The complementary actions of gingerols and shogaols on oxidative stress, inflammation, vascular tone, lipid metabolism, thrombosis, and glucose regulation provide mechanistic plausibility for observed clinical benefits. Ginger may serve as an effective adjunct in cardiovascular prevention strategies, pending confirmation from long-term, standardized clinical trials.

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