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Cardioprotective Effects of Phytochemicals: Mechanisms, Key Compounds and Translational Prospects

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Abstract : *Cardiovascular disease remains the leading cause of death worldwide, and the long-term burden of conventional drug therapy, including side effects and cost, has renewed interest in plant-derived compounds as protective agents. Phytochemicals, especially polyphenols and flavonoids, are abundant in fruits, vegetables, herbs and medicinal plants and have been linked with lower cardiovascular risk. This review summarises evidence on the cardioprotective effects of phytochemicals, with attention to their classes, molecular mechanisms, specific cardiovascular conditions and practical concerns. The principal mechanisms are scavenging of reactive oxygen species, activation of the Nrf2 antioxidant pathway, suppression of inflammation, improvement of endothelial function, inhibition of lipid oxidation and atherogenesis, and limitation of cell death after ischaemia and reperfusion. Protective effects have been reported for atherosclerosis, myocardial ischaemia-reperfusion injury, heart failure, hypertension and doxorubicin-induced cardiotoxicity, with quercetin, resveratrol, curcumin, apigenin, silibinin and Ginkgo biloba flavonoids among the best studied compounds, and newer plant extracts and isolated polyphenols are being identified. Poor bioavailability, extensive metabolism, limited toxicological data and a shortage of large clinical trials restrict clinical use. Better formulation, standardisation, mechanistic and sex-specific studies, and rigorous trials are needed before phytochemicals can be recommended as cardioprotective therapy..*

Keywords: Phytochemicals; cardioprotection; polyphenols; flavonoids; oxidative stress; Nrf2; ischaemia-reperfusion injury; quercetin; resveratrol; curcumin.

I. INTRODUCTION

1.1 Cardiovascular disease and the case for phytochemicals

Cardiovascular diseases (CVDs) include coronary artery disease, myocardial infarction, heart failure, hypertension, stroke and arrhythmia, and together they are the main cause of death and disability across the world. Established treatments such as statins, antihypertensives, antiplatelet drugs and beta-blockers have reduced mortality, but they must be taken for many years, are not equally effective in all patients and may cause adverse effects. Many patients also look for dietary and herbal approaches that can supplement prescribed drugs. Population studies have long associated diets rich in fruits, vegetables, tea, wine and spices with lower cardiovascular risk, and attention has turned to the non-nutrient plant compounds, or phytochemicals, that these foods contain [1,2,5,12].

Phytochemicals are secondary metabolites made by plants to defend against ultraviolet light, pathogens and oxidative stress. In humans, many of them display antioxidant, anti-inflammatory, vasodilatory and lipid-modifying activity, which are the properties most relevant to the heart and blood vessels [2,12]. A series of reviews has examined

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polyphenols, flavonoids and other phytochemicals in cardiovascular disease [1,2,3,5,6,12], and experimental studies keep adding new compounds and sources [8,10,13,15,16,17]. Yet these reports are spread across many journals and differ in model, dose and quality, so an organised account that links compounds to mechanisms and to specific cardiac conditions is useful.

1.2 Oxidative stress and inflammation in cardiovascular pathology

Much of the logic of phytochemical cardioprotection rests on the role of oxidative stress. Reactive oxygen species (ROS) are normal by-products of metabolism, but when production exceeds antioxidant defence, they damage lipids, proteins and DNA, uncouple endothelial nitric oxide synthase, activate inflammatory signalling and trigger cell death. Each major CVD has an oxidative component.

Atherosclerosis. Oxidation of low-density lipoprotein (LDL) in the vessel wall promotes foam-cell formation, endothelial dysfunction and plaque growth, so antioxidants that prevent LDL oxidation are expected to slow the disease [4].

Ischaemia-reperfusion (I/R) injury. Restoring blood flow after an infarct brings a burst of ROS, calcium overload and inflammation that kills further myocardium, a injury that is itself a target for antioxidants [8,10].

Heart failure. Chronic oxidative stress and inflammation contribute to remodelling and contractile dysfunction, and the response may differ between men and women [7].

Drug-induced cardiotoxicity. Anticancer anthracyclines such as doxorubicin generate ROS and damage mitochondria in cardiac cells, limiting their use [13].

The body's central defence against such stress is the Nrf2 (nuclear factor erythroid 2-related factor 2) pathway, which switches on genes for antioxidant and detoxifying enzymes. Natural products that activate Nrf2 are therefore of great interest in CVD [9].

1.3 Classes of cardioprotective phytochemicals

Several phytochemical classes have been connected with heart health. By far the largest group in the reviewed literature is the polyphenols, which are characterised by multiple phenolic hydroxyl groups and include flavonoids, phenolic acids, stilbenes and curcuminoids [1,3,5,6].

Table 1. Representative cardioprotective phytochemicals and their sources.

Class	Representative compounds	Typical dietary or medicinal sources	Ref.
Flavonols	Quercetin	Onions, apples, berries, tea	[3,10,15]
Flavones	Apigenin	Parsley, celery, chamomile	[15]
Flavonolignans	Silibinin	Milk thistle	[15]
Flavonoids from medicinal extracts	Ginkgo biloba flavonoids	Ginkgo leaf	[14]
Stilbenes	Resveratrol	Grapes, red wine, berries	[10]
Curcuminoids	Curcumin	Turmeric	[8]
Other polyphenols	Polyphenols from Geum japonicum var. chinense	Medicinal plant	[17]
Mixed extracts	Hydroethanolic extract of Cucumis melo kernels	Melon seed kernels	[16]

Besides these, reviews mention terpenoids, saponins, alkaloids and sulphur-containing compounds as contributors to the cardiovascular effects of herbal medicines, although the polyphenols have received the most experimental attention [2,12]. A recurring theme is that whole extracts contain many compounds acting together, whereas isolated compounds allow precise dosing and mechanistic study.

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1.4 Mechanisms of cardioprotection

Phytochemicals do not act through a single target. The reviewed literature points to several complementary mechanisms (Table 2).

Table 2. Principal cardioprotective mechanisms of phytochemicals.

Mechanism	How phytochemicals act	Cardiovascular consequence	Ref.
Direct antioxidant action	Donate hydrogen or electrons to ROS, chelate metals, prevent lipid peroxidation	Less LDL oxidation, less tissue damage	[3,4,6]
Nrf2 pathway activation	Induce antioxidant response element genes, such as heme oxygenase-1 and glutathione enzymes	Stronger endogenous defence	[9]
Anti-inflammatory action	Reduce NF-κB signalling and cytokine release	Slower atherosclerosis; smaller infarct response	[1,5,8]
Endothelial protection	Preserve nitric oxide availability and vasodilation	Lower blood pressure, better vessel function	[3,5,16]
Anti-apoptotic and mitochondrial protection	Stabilise mitochondria, limit caspase activation	Fewer cardiomyocytes lost in I/R and drug toxicity	[10,13]
Lipid and platelet effects	Lower lipid oxidation; may reduce platelet aggregation	Reduced plaque formation and thrombosis risk	[4,5,12]
Metabolic and signalling modulation	Alter metabolic pathways revealed by metabolomics	Improved cardiac energy handling	[14,16]

Two observations deserve emphasis. First, the direct radical-scavenging ability of polyphenols, though easy to measure in a test tube, may be less important in the body than their ability to switch on cellular defences such as Nrf2, since plasma concentrations of the parent compounds are usually low [6,9]. Second, anti-inflammatory and antioxidant actions are interlinked, as ROS activate inflammatory pathways and inflammation generates ROS, so interventions at one point often influence both.

1.5 Flavonoids and cardiovascular health

Flavonoids form the largest and most investigated group of cardioprotective phytochemicals. Reviews describe how they influence endothelial function, blood pressure, lipid metabolism, platelet activity and inflammation, and they link flavonoid-rich diets with a lower risk of CVD [3,6]. An early and influential line of reasoning presents flavonoids as antioxidants against atherosclerosis: by protecting LDL from oxidation and reducing vascular oxidative stress, they may delay the formation of plaque [4]. More recent reviews expand the picture to include the regulation of signalling pathways, gene expression and the gut microbiota, and caution that the therapeutic and toxicological features of these compounds, including their pharmacokinetics, need more study [6].

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Three individual flavonoids illustrate the field. Quercetin, abundant in onions, apples and tea, is among the most studied; together with resveratrol it has shown antioxidant protection against myocardial reperfusion injury in experimental models [10]. Apigenin, quercetin and silibinin have been reviewed with respect to their isolation, identification and pharmacological actions in the heart, with evidence of antioxidant, anti-inflammatory and anti-apoptotic effects [15]. Flavonoids of Ginkgo biloba have been explored in cardiovascular disease with the help of pharmacokinetic, metabolism and metabolomic approaches that aim to clarify their mechanism of action [14].

1.6 Stilbenes and curcuminoids

Resveratrol, a stilbene found in grapes and red wine, has attracted attention because of the so-called French paradox, in which relatively high fat consumption coexists with low rates of coronary disease in populations that drink red wine. Experimental work discussed in reviews shows that resveratrol, like quercetin, can protect the heart against reperfusion injury through antioxidant effects and modulation of survival signalling [10].

Curcumin, the main yellow pigment of turmeric, has anti-inflammatory and antioxidant properties and has been studied in myocardial I/R injury. A systematic review and meta-analysis that combined preclinical and clinical studies evaluated its protective effect, and the results supported a beneficial action in this setting while underlining the limits of the available data [8]. Curcumin is also known for poor absorption and rapid metabolism, a problem that is considered in Section 1.10.