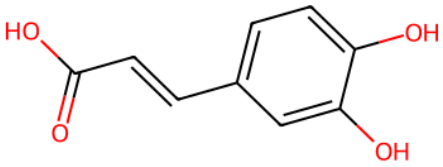
# Caffeic Acid

Main functional characteristics: Antioxidant, anti-inflammatory, glycemia modulator, lipid metabolism regulator

Molecular weight: 180.16 g/mol




## Scientific description

Caffeic acid (3,4-dihydroxycinnamic acid) is a phenolic acid derived from the phenylpropanoid pathway and is present in a wide variety of vegetables, fruits, medicinal herbs, and especially in coffee. Its chemical structure, characterized by a catechol ring and a conjugated double bond with a carboxyl group, provides it with strong electron-donating capacity and radical stabilization by resonance, which underlies its broad-spectrum antioxidant potential.

At the molecular level, caffeic acid exerts its antioxidant function in two major ways: firstly, by directly scavenging reactive oxygen species (ROS) and reactive nitrogen species (RNS); and secondly, by stimulating endogenous signaling pathways such as Nrf2/ARE. Activation of Nrf2 promotes the transcription of phase II enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and heme oxygenase-1 (HO-1), reinforcing cellular defense against chronic oxidative stress. These mechanisms contribute to the prevention of oxidative damage to membranes, proteins, and DNA.

Its anti-inflammatory role is mainly mediated through the inhibition of the NF-κB pathway, a key transcription factor regulating pro-inflammatory genes. Caffeic acid blocks the phosphorylation of IκB kinase (IKKβ), preventing IκB degradation and subsequent NF-κB nuclear translocation. This results in reduced expression of mediators such as COX-2, iNOS, TNF-α, IL-1β, and IL-6. Additionally, it modulates MAPK pathways (ERK, JNK, p38), lowering the production of adhesion molecules (ICAM-1, VCAM-1) and chemokines involved in inflammatory cell recruitment.

Regarding carbohydrate metabolism, caffeic acid and its derivatives inhibit digestive enzymes α-amylase and α-glucosidase, slowing down polysaccharide hydrolysis and glucose absorption in the small intestine. This leads to a reduction in postprandial glycemia. In the liver and skeletal muscle, it activates AMPK, promoting glucose uptake via GLUT4 and suppressing hepatic gluconeogenesis, thereby improving insulin sensitivity.

Concerning lipid metabolism, caffeic acid protects against low-density lipoprotein (LDL) oxidation and reduces lipid peroxidation, key processes in the development of atherosclerosis. These effects are complemented by modulation of nuclear receptors such as PPARγ, which regulate lipid homeostasis. Preclinical studies have shown that its supplementation decreases total cholesterol and triglyceride levels.

Recent investigations suggest a potential role in oncology. Caffeic acid induces apoptosis in tumor cells through the intrinsic mitochondrial pathway, involving loss of mitochondrial membrane potential (ΔΨm), cytochrome c release, and caspase activation. It also interferes with angiogenesis and tumor proliferation, showing antineoplastic properties that justify its growing interest as an adjuvant in cancer therapies.

Overall, the pleiotropic effects of caffeic acid –antioxidant, anti-inflammatory, hypoglycemic, hypolipidemic, and antitumoral– are closely related to its ability to modulate multiple cellular signaling pathways. This makes it a key molecule for the development of standardized extracts of *Leptocarpha rivularis*, with potential applications in nutraceuticals, functional foods, and preventive pharmacology.

## References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