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PROCEEDINGS ABSTRACT

Anti-inflammatory and antioxidant effects of medicinal plant extracts: a review of pharmacological evidence and mechanisms

Safa Adnan Mahmood, Qayssar Joudah Fadheel, and Karar M. Jaleel

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Several non-communicable diseases (including cardiovascular disorders, type 2 diabetes mellitus, most neurodegenerative diseases, autoimmune conditions, and cancer) are now known to be directly related to chronic inflammation and oxidative stress. Although such diseases significantly vary in their clinical manifestations, they tend to share common pathophysiological pathways that involve an excessive production of reactive oxygen species (ROS) and a persistent activation of the inflammatory signalling. The excessive ROS generation damages lipids, proteins, and DNA, and activates pro-inflammatory transcription factors such as the nuclear factor-kappa B (NF-κB) and the activator protein-1. The activation of these molecules increases the expression of inflammatory mediators such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6). This creates a self-perpetuating cycle in which oxidative stress promotes inflammation and the latter generates additional ROS, leading to tissue injury and disease progression. In this broad context, medicinal plant extracts have gained considerable attention due to their ability to simultaneously influence multiple biological targets. Plant extracts are known to be rich in bioactive phytochemicals (such as polyphenols, flavonoids, terpenoids, alkaloids, and phenolic acids), and these compounds seem to possess antioxidant and anti-inflammatory properties through several complementary mechanisms. For example, a significant number of phytochemicals can directly counteract free radicals and ROS: they may have the ability to chelate transition metals such as iron and copper, which otherwise catalyse the formation of more free radicals. They can also prevent lipid peroxidation, thereby protecting the cellular membranes from oxidative damage. In addition to these direct effects, plant-derived compounds can strength the body's own defence systems by increasing the activities of endogenous antioxidant enzymes (such as superoxide dismutase, catalase, and glutathione peroxidase). This effect is mainly mediated through the activation of the nuclear factor erythroid 2-related factor 2 / antioxidant response element signalling pathway, which controls the expression of genes involved in antioxidant protection. Additionally, medicinal plant extracts can inhibit key inflammatory pathways: numerous phytochemicals are known to be able to suppress the expression of cyclooxygenase-2 and of the inducible nitric oxide synthase; two enzymes that are strongly associated with inflammatory damage. Some phytochemicals can also prevent the nuclear translocation of NF-κB, thereby lowering the generation of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. Data obtained from cell culture and animal model studies strongly support these positive effects. Furthermore, a growing number of randomized

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controlled trials involving the use of medicinal plants (such as *Curcuma longa*, *Camellia sinensis*, and *Zingiber officinale*) has shown reductions in biomarkers of oxidative stress and inflammation in humans. Despite these promising findings, several limitations hamper clinical application: numerous phytochemicals have poor bioavailability (meaning that only a small amount reaches the bloodstream after administration), while plant extracts often lack standardisation, resulting in variable compositions and potencies. Moreover, large scale clinical trials and long-term safety studies remain limited. Further mechanistic research as well as appropriately designed evidence-based clinical trials are needed before medicinal plant extracts can be systematically integrated into modern pharmacotherapy.

Keywords

chronic inflammation; medicinal plant extracts; NF-κB; oxidative stress; phytochemicals

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Conflicts of interest statement

None to declare.

Affiliation(s)

College of Pharmacy, University of Babylon, Hillah, Iraq *(SAM)* ▪ Department of Clinical Pharmacy, College of Pharmacy, University of Babylon, Hillah, Iraq *(QJF)* ▪ College of Pharmacy, Madenat Alelem University, Baghdad, Iraq *(KMJ)*

Correspondence

Safa Adnan Mahmood
College of Pharmacy
University of Babylon
Hillah, Iraq
pha986.safa.adnan@student.uobabylon.edu.iq