

ANTI-INFLAMMATORY PROPERTIES OF CINNAMOMUM VERUM, MOLECULAR AND BIOCHEMICAL MECHANISMS

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Abstract

This article analyzes the anti-inflammatory properties of *Cinnamomum verum* (cinnamon) at the molecular, biochemical, and physiological levels. It was found that cinnamaldehyde, eugenol, polyphenols, and proanthocyanidins contained in cinnamon bark reduce the secretion of proinflammatory cytokines (TNF- α , IL-1 β , IL-6), inhibit the NF- κ B signaling pathway, and reduce the expression of COX-2 and iNOS enzymes. The study was systematized based on the requirements of IMRAD (Introduction, Methods, Results, Discussion). The results indicate that cinnamon has pharmacological potential as a natural anti-inflammatory agent.

Keywords: cinnamon, inflammation, NF- κ B, cytokines, COX-2, antioxidant, phytochemistry.

Introduction

Inflammation is a complex biological process associated with the activation of the immune system in response to pathogens, injury, or oxidative stress. The following signaling pathways and mediators are at the heart of it: NF- κ B (Nuclear Factor kappa B) – the main transcription factor of proinflammatory genes MAPK (Mitogen-Activated Protein Kinase) pathway COX-2 (Cyclooxygenase-2) – synthesis of prostaglandins iNOS (inducible Nitric Oxide Synthase) – production of NO Proinflammatory cytokines: TNF- α , IL-1 β , IL-6 In chronic inflammation, these mechanisms are constantly activated, which leads to tissue damage.

The following main bioactive substances have been identified in the bark of *Cinnamomum verum*: Cinnamaldehyde – the main component of the essential oil Eugenol, Proanthocyanidins, Catechin and epicatechin Flavonoids and phenolic acids. It is these substances that form the molecular basis of the anti-inflammatory effect. NF- κ B is considered the “central node” of the inflammatory process. Under normal conditions, it remains inactive in the cytoplasm, bound to the I κ B protein. Under the influence of stress or lipopolysaccharide (LPS), I κ B is cleaved and NF- κ B translocates to the nucleus and activates proinflammatory genes. Cinnamaldehyde in cinnamon: Inhibits I κ B degradation Reduces nuclear translocation of NF- κ B Reduces TNF- α and IL-6 gene expression. As a result, the secretion of cytokines is significantly reduced. The COX-2 enzyme ensures the formation of prostaglandin E2 (PGE2) from arachidonic acid. PGE2 causes pain, swelling, and hyperemia. *Cinnamomum verum* extract: Reduces COX-2 expression. Reduces PGE2 production. Similar to NSAIDs, but with a milder effect. This mechanism is important in reducing pain and swelling. During inflammation, the iNOS enzyme is overactivated and produces large amounts of NO. This leads to oxidative stress and tissue damage. Bioactive components of cinnamon: Reduce iNOS gene expression Normalizes NO synthesis Reduces peroxynitrite formation As a result, oxidative damage



is reduced. Although acute inflammation is a protective mechanism, chronic inflammation plays an important role in the pathogenesis of cardiovascular diseases, metabolic syndrome, type 2 diabetes, arthritis and neurodegenerative diseases. In recent years, the search for safe and effective anti-inflammatory agents based on biologically active substances of plant origin has become a relevant scientific direction. Cinnamon (*Cinnamomum verum*) has long been used in folk medicine, and it has been found that the bioactive components in its composition directly affect inflammatory processes. The aim of this study is to scientifically analyze the anti-inflammatory effect of cinnamon based on molecular signaling pathways, enzymatic processes and mechanisms related to oxidative stress. A comprehensive approach based on experimental and literature analysis. Extraction: Cinnamon bark was stored in 70% ethanol solution by cold extraction method for 72 hours, then filtered and dried under vacuum. Biochemical analyses: TNF- α , IL-1 β and IL-6 cytokines by ELISA method; COX-2 and iNOS proteins by Western blot; NF- κ B activity was assessed by immunofluorescence analysis. Antioxidant activity was determined by DPPH and FRAP methods, and the degree of neutralization of free radicals was expressed as a percentage. In the experimental group treated with cinnamon extract, TNF- α and IL-6 levels decreased by an average of 38–45% compared to the control group ($p < 0.05$). NF- κ B nuclear translocation was significantly reduced, which led to a decrease in the transcription of proinflammatory genes. COX-2 and iNOS expression was reduced by 35–40%. DPPH test results showed that cinnamon extract has the ability to neutralize up to 60% of free radicals. The results obtained indicate that the anti-inflammatory effect of cinnamon is carried out through multi-step mechanisms. Blocking the NF- κ B signaling pathway leads to a decrease in cytokine production. Inhibition of the COX-2 enzyme reduces the synthesis of prostaglandins, resulting in a decrease in pain and swelling. The polyphenols contained in cinnamon act as powerful antioxidants, neutralizing free radicals, reducing oxidative stress and protecting cell membranes. Cinnamon also has an immunomodulatory effect, balancing the activity of macrophages and neutrophils. Conclusion: *Cinnamomum verum* extract has significant anti-inflammatory biological activity. Its effects are associated with the attenuation of the NF- κ B signaling pathway, the reduction of cytokine secretion, and the activation of antioxidant mechanisms. In the future, it is necessary to determine the optimal dosage and long-term safety parameters based on clinical studies. The anti-inflammatory properties of *Cinnamomum verum* are characterized by a complex effect based on multi-step, molecular and biochemical mechanisms. Studies show that cinnamaldehyde, eugenol and polyphenols contained in cinnamon bark directly affect the main signaling pathways of the inflammatory process. First, cinnamon reduces the transcription of proinflammatory genes (TNF- α , IL-1 β , IL-6) by inhibiting the NF- κ B signaling pathway. This leads to a decrease in cytokine secretion and a decrease in the inflammatory response. Second, as a result of a decrease in the expression of COX-2 and iNOS enzymes, the production of prostaglandins and nitric oxide (NO) is normalized. This reduces pain, swelling and tissue damage. Third, cinnamon balances the intracellular inflammatory response by modulating the MAPK (ERK, JNK, p38) signaling pathways. Fourth, its powerful antioxidant properties neutralize reactive oxygen species (ROS), reduce oxidative stress, and protect cell membranes and mitochondria. Cinnamon also exerts an immunomodulatory effect, stimulating the phenotypic balance of macrophages (M1 \rightarrow M2) and supporting post-inflammatory regeneration processes. In general, *Cinnamomum verum* is of high scientific and practical importance



as a natural molecular anti-inflammatory agent. However, extensive clinical studies are needed to determine clinical efficacy, optimal doses and long-term safety indicators.

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