

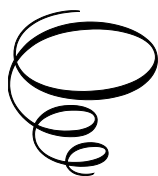
The Potential Role of Flavonoids in Mitigating Inflammatory Disorders

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Edited by

Anuradha Mishra, Narhari N. Palei,
Sumel Ashique and Satya Prakash Singh

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CHAPTER 6

ROLE OF FLAVONOIDS IN INFLAMMATORY LUNG DISORDERS

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Abstract

Inflammatory lung disorders include chronic obstructive pulmonary disease, asthma, and acute respiratory distress syndrome. Due to their chronic nature, these conditions are a huge burden to the global health environment. These conditions have fewer options for therapy. Flavonoids, a diverse group of polyphenolic compounds found in fruits, vegetables, and medicinal plants, have emerged as promising agents in modulating inflammatory pathways associated with these disorders. This chapter covers the biochemical mechanisms involved in flavonoid activity, including its antioxidant, anti-inflammatory, and immunomodulatory action. Through inhibition of main inflammatory mediators, such as cytokines, ROS, and NF- κ B, flavonoids may decrease inflammatory and oxidative stress reactions within the lungs, thereby preventing damage in lung tissue. The work reviews clinical and preclinical studies conducted on the effectiveness of specific flavonoids, including quercetin, hesperidin, and naringin, that have shown potential in diminishing symptoms and improving lung condition. Finally, challenges in the translation of flavonoid research into clinical practice, bioavailability and standardization among them, are discussed. This comprehensive analysis underlines the potential of flavonoids as adjunctive therapies in inflammatory lung disorders, offering new avenues for treatment and prevention strategies.

Keywords: Flavonoids, Inflammatory Lung Disorders, Oxidative Stress, Asthma, Chronic Obstructive Pulmonary Disease (COPD)

1. Introduction

The term "inflammatory lung disorders" refers to a collection of illnesses that affect respiratory function by causing inflammation of the lung tissues, airways, or alveoli. These disorders may be brought on by hypersensitivity reactions, autoimmune reactions, infections, or environmental exposures. Redness, swelling, heat, discomfort, and loss of function are macroscopic signs of inflammation. Under a microscope, it manifests as inflammatory cell infiltration, vasodilation, and enhanced vascular permeability. The purpose of inflammatory reactions is to wall off and restrict the harmful substances, as well as to eliminate and eliminate them. In addition, inflammation boosts the immune system to aid in healing. Different kinds of inflammatory cells are activated during inflammation. To alter the actions of other inflammatory cells, each produces mediators and cytokines. Inflammation advances as a result of the coordination of various cells and chemicals. Asthma, pneumonia, TB, pulmonary fibrosis, hypersensitivity pneumonitis, acute respiratory distress syndrome (ARDS), and chronic obstructive pulmonary disease (COPD) are examples of common inflammatory lung conditions (Aghasafari, 2019, 59-74).

1.1 Defense mechanism in the lung

The epithelium is where inhaled substances first come into contact with the airway. Among the substances released by its epithelial cells that nonspecifically defend the respiratory system against microbial attack are mucins, defensins, lysozyme, lactoferrin, and nitric oxide (Oliveira, 2007, 578-591). In order to attract inflammatory cells to the site of inflammation, it also generate a variety of mediators, including oxidative stress, pro-inflammatory cytokines like tumor necrosis factor alpha (TNF- α), interleukin 1 beta (IL-1 β), granulocyte/macrophage colony-stimulating factor (GM-CSF), and platelet-activating factor. The release of arachidonic acid from membrane lipids is stimulated by the cytokines, which results in the synthesis of eicosanoids, which in turn promote tissue inflammation and mucus secretion by goblet cells (KB, 1994, 128-145). In addition to preventing microbial adhesion to the epithelial surface, immunoglobulin A (IgA), which is released by plasma cells, also suppresses certain viral infections (such as rotavirus and influenza) by interacting with its assembly processes. Additionally, it binds to antigen, resulting in antibody-dependent