Chapter 7
Ethnopharmacology and Toxicological Profiles of Anti-Inflammatory and Analgesic Herbs

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ABSTRACT

Inflammation in the body might be viewed as a cascade (driven by the immune, vascular, and signaling systems) whenever the body is affected by some foreign agents. This cascade boils and terminates at activated repair mechanisms when the host detects the offending stimulus. The persistence of states of drug resistance and toxicological effects in the human body stimulates the search for new therapeutic agents that can combat inflammation and analgesia. Ethnopharmacological research of plants with traditional uses as anti-inflammatory and analgesic agents demonstrates an extensive variety of bioactive constituents that have been utilized traditionally by various tribal communities to alleviate pain and restrain the inflammatory processes. Phytochemical analysis of these ethnomedicinal herbs has revealed that be contain secondary metabolites such as curcumin, ursolic acid, escin, withaferin A, etc., often showing the ability to inhibit important inflammatory mediators, viz., cyclooxygenase-2 (COX-2), nuclear factor-κB (NF-κB), and pro-inflammatory cytokines, e.g., TNF-α and IL-1β.

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INTRODUCTION

Inflammation can be defined as an instinctive response to injury, infection, irritants, or harmful stimuli. Foreign antigen exposure triggers an integrated cascade involving the immune, vascular, and signaling systems that culminates in host-initiated repair mechanisms upon stimulus recognition. Symptoms such as pain, increased body temperature, localized swelling, and organ dysfunction can result from the dilation of venules and the subsequent leakage of plasma into the affected tissue, which is facilitated by specific biological mediators, including histamine, prostaglandins, and cytokines. At the same time, the immune cells (i.e., WBCs) are deputed to the injured location to restore the tissues (*Figure 1*). A prolonged reaction (chronic) could continue for up to a year or more. The neutrophils and macrophages release mediators, i.e., histamine, bradykinin, PGE_2, LTB_4, TNF-α, IL-1β, and IL-6 to perform phagocytosis. Ultimately, the resolution takes place via *a*) debris clearance, *b*) collagen deposition, or can progress to chronic inflammation. Although acute inflammation transpires decisively and endures for a brief duration due to a wound or infection, chronic inflammation is typically triggered by the immune system striking the infected tissues, which may ultimately lead to abnormalities such as tumors, cardiovascular disease, type 2 diabetes, asthma, and arthritis, such as rheumatoid arthritis etc.

Figure 1. Diagrammatic representation of the inflammation mechanism. Initiation leads to leukocyte adhesion and migration. The neutrophils and macrophages release mediators, i.e., histamine, bradykinin, PGE_2, LTB_4, TNF-α, IL-1β, and IL-6 to perform phagocytosis. Resolution takes place via a) debris clearance, b) collagen deposition, or can progress to chronic inflammation