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STUDYING THE ANTI-INFLAMMATORY PROPERTIES OF CERTAIN MEDICINAL PLANTS

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Annotation: Nowadays, new types of inflammation are emerging. Each of them is related to the activity of individual organs and organ systems. In general, all diseases are actually "Inflammation." Their development varies depending on acute and chronic conditions. This article presents information about their molecular mechanisms and some of the plants used in folk medicine to combat them.

Keywords. Inflammation, molecular mechanism, chemical composition, cyclooxygenase (COX), Prostaglandins, Rosa L, Sinapis arvensis, Amygdalus communis L.

Аннотация: В настоящее время появляются новые виды воспалений. Каждый из них связан с деятельностью отдельных органов и систем органов. В общем, все болезни на самом деле являются «воспалениями». Их развитие варьирует в зависимости от острых и хронических состояний. В данной статье представлена информация об их молекулярных механизмах и некоторых растениях, используемых в народной медицине для борьбы с ними.

Ключевые слова. Воспаление, молекулярный механизм, химический состав, циклооксигеназа (ЦОГ), простагландины, Rosa L, Sinapis arvensis, Amygdalus communis L. Hozirgi vaqtda yallig'lanishning yangi turlari paydo bo'lmoqda. Ularning har biri alohida organlar va organ tizimlarining faoliyati bilan bog'liq. Umuman olganda, barcha kasalliklar aslida "yallig'lanish" dir. Ularning rivojlanishi o'tkir va surunkali holatlarga qarab farq qiladi. Ushbu maqolada ularning molekulyar mexanizmlari va ularga qarshi kurashish uchun xalq tabobatida qo'llaniladigan ba'zi o'simliklar haqida ma'lumot berilgan.

Kalit so‘zlar. Yallig'lanish, molekulyar mexanizm, kimyoviy tarkibi, siklooksigenaza (COX), Prostaglandinlar, Rosa L, Sinapis arvensis, Amygdalus communis L.

Inflammation is often a complex process associated with pain, involving phenomena such as increased vascular permeability, enhanced protein denaturation, and membrane changes. When body cells are damaged by microbes, physical factors, or chemical agents, the damage manifests as stress. Tissue inflammation occurs as a response to stress, characterized by redness, pain, heat, swelling, and loss of function in the affected area. Loss of function depends on the location and extent of the damage. Since inflammation is one of the body's non-specific defense mechanisms, the reaction of randomly cut tissues is similar to other types of tissue damage caused by heat, radiation, bacterial, or viral invasion. Prostaglandins are hormone-like substances synthesized in almost all tissues of the body, including blood vessel walls. They regulate blood pressure, uterine contractions, and various other physiological processes.

Prostaglandins are small molecules belonging to a group of lipid-like substances called eicosanoids. This group also includes chemically similar compounds like leukotrienes, involved in inflammation and allergic reactions, and thromboxanes, involved in blood clotting. All eicosanoids are derived from arachidonic acid, an unsaturated fatty acid, and are synthesized from another fatty acid, linolenic acid, which enters the human body with food.

Molecular Mechanisms of Inflammation

Cyclooxygenases (COX) are enzymes involved in synthesizing prostanoids such as prostaglandins, prostacyclins, and thromboxanes. Pharmacological inhibition of cyclooxygenases reduces inflammation and pain, with aspirin and ibuprofen being examples of such inhibitors. The terms "prostaglandin synthase" and "prostaglandin synthetase" are sometimes used to refer to cyclooxygenases. Detailed studies of COX revealed their presence in various tissues and their different sensitivity spectra to aspirin-like drugs, suggesting the existence of enzyme isoforms. In humans, two genes encode COX: COX-1 and COX-2. The alternative splicing of the first gene's product results in two forms of the enzyme. Cyclooxygenases catalyze the conversion of arachidonic acid to prostaglandin H2 (PGH2), a precursor of other prostaglandins, prostacyclins, and thromboxane A2.**Inflammation is one of the central processes required to protect animal cells from injuries or microbial infections [1,2]. Nevertheless, inflammation is regularly acute [3] or chronic [1]. Chronic inflammation leads to various diseases, including neurodegenerative disorders, cancer, and cardiovascular diseases [4]. The inflammation mechanism represents a chain of coordinated, dynamic responses, including specific humoral secretions with cellular and vascular events. These pathways involve the physical alteration of white blood cell locations (monocytes, basophils, eosinophils, and neutrophils), plasma, and fluids to the inflamed site [5]. A group of latent mediators and other signaling molecules (such as histamine, prostaglandins, leukotrienes, oxygen and nitrogen-derived free radicals, and serotonin) is primarily released by immune defense cells in the inflammatory mechanism [6]. Regardless, the inflammatory response is triggered in two stages: (a) acute and (b) chronic, each

mediated by different mechanisms [3]. The acute inflammation involves the participation of immune responses vascular and cellular [7]. Responses occurring in microvasculature typically emerge within minutes of tissue injury or microbial infection, involving inflammatory stimuli known as vascular events [7]. This process quickly leads to vasodilation and subsequently makes the vessels more permeable. These processes allow the entry of inflammatory mediators and produce interstitial edema [8]. During inflammation, the infiltration of white blood cells from the circulatory system is crucial [9,10]. A group of chemotactic agents, such as microbial endotoxins with amino-terminal N-formyl methionyl groups, C5a complement fragments, and interleukins, along with platelet-activating factors like histamine and leukotriene B, can stimulate leukocytes to swim within minutes [11,12]. Among leukocytes, neutrophils are the first inflammatory cells recruited to the acute inflammation site [13]. The infiltration of immune cells is triggered by a complex mechanism where white blood cells work together with endothelial cells in post-capillary venules [14]. The cellular events encompass a sequence involving capture, trundling, and adhesion to microvascular endothelium [15]. These events are regulated by the mobilization of cell adhesion molecules (CAMs). These CAMs include intracellular adhesion molecules (ICAM)-1, ICAM-2, integrins, and selectins. The selectin group of CAMs includes three families: P-selectin and E-selectin produced by endothelial cells and L-selectin produced by white blood cells [16]. High-affinity binding between integrins (CD11/CD18) and adhesion molecules (CAM-1 and CAM-2) on white blood cells and endothelial cells mediates the adhesion of white blood cells to endothelium [17]. After the stationary adhesion period, white blood cells can exit post-capillary venules by extending pseudopodia between endothelial cells and reaching the subendothelial space. This complex event is often called white blood cell extravasation and transendothelial migration [18]. Chronic inflammatory events are characterized by mononuclear cell infiltration (such as monocytes and lymphocytes), fibroblast proliferation, collagen fibers, and connective tissue formation, eventually leading to granuloma formation [19]. The degeneration of tissues in chronic inflammation is usually mediated by nitrogen species, proteases, and other reactive oxygen species released by infiltrated inflammatory cells [20]. Indeed, genomic changes in p53 have been confirmed as a cause of most chronic inflammatory diseases (such as inflammatory bowel disease and rheumatoid arthritis) and cancer [21-23]. The novelty of this review is that it provides a summary of recent knowledge on the involvement of mediators in inflammation and addresses some misconceptions and facts about inflammatory processes. This review aims to highlight the knowledge gap about inflammation processes, including the addition of the latest and most relevant issues concerning this phenomenon.

Inflammation is a crucial mechanism for human health and disease. The first description of inflammation by Roman Cornelius Celsus in the 1st century identified the clinical signs of

inflammatory diseases. Four main signs of inflammation were identified: rubor et tumor cum calore et dolore (redness and heat with swelling and pain). The development of the disease was defined by these four cardinal signs [24,25]. In 1858, the inclusion of an additional cardinal sign, functio laesa (loss of function), was proposed by Rudolf Virchow's study of the cellular basis of pathology. Subsequent research in the late 19th century included the microbial theory of disease, with microorganisms identified as the primary inducers of the acute inflammatory response by Robert Koch and Louis Pasteur. Metchnikoff discovered that acute inflammation is resolved when neutrophils are engulfed by tissue macrophages. Recently, advanced cellular and molecular mechanisms controlling the fate of inflammation have been identified. Acute inflammation is considered a physiological response to protect vascularized tissues and maintain homeostasis. Inflammation begins as a protective response to problems related to pathogens or foreign bodies or injuries experienced by host tissues. This process is characterized by blood vessel dilation, increased capillary permeability, enhanced blood flow, and the recruitment of leukocytes. Among the first leukocytes to accumulate at the inflamed site are polymorphonuclear neutrophils. These cells are crucial as the first line of defense for the innate immune system due to their phagocytic and microbicidal functions. Subsequently, mononuclear cells, monocytes, and macrophages enter the inflammatory site and clear cellular debris and apoptotic polymorphonuclear neutrophils through nonphlogistic (non-heat or fever-producing) phagocytosis, avoiding the extension of inflammation [26].

While the inflammatory reaction is protective, the failure to clear harmful materials produced by neutrophils through phagocytosis, the non-clearance of apoptotic inflammatory cells, and the delay of apoptosis lead to chronic and pathological lesions. Complete removal of leukocytes from the lesion is observed in sensitive individuals; acute inflammation is not resolved and chronic disease and fibrosis develop [27]. Accordingly, the failure to resolve and restore tissue homeostasis through neutrophil-mediated clearance leads to chronic inflammation [28]. This is a primary cause of human inflammatory pathologies, including arthritis, asthma, cancer, cardiovascular diseases, and periodontal diseases. From the time of Celsus, we have thought about inflammation in terms of induction. In recent years, we have identified the molecular mediators of inflammation induction (cytokines and chemokines) as our understanding of the process at the molecular level has evolved. We have never paid attention to attenuation; how is inflammation turned off? We have always assumed this was a passive process due to the cessation or breakdown of inducers. To maintain a healthy state, both the initiation and resolution of acute inflammation must be effective. The loss of resolution and the failure to restore tissue homeostasis due to neutrophil-mediated clearance leads to chronic inflammation [28]. This is a primary cause of human inflammatory pathologies, including arthritis, asthma, cancer, cardiovascular diseases, and periodontal diseases. From the time of Celsus, we have thought about

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Studying Anti-Inflammatory Properties of Plants:

1. Take 5 ml of blood and wash it three times with a 1:1 ratio of saline solution. Prepare the final blood clot using saline solution at a 10% concentration.

2. Prepare 1 ml of the extract under study at various concentrations (1000, 800, 600, 400, and 200 μg/ml) and dissolve it in distilled water (it should also be soluble in DMSO). Aspirin is used as a positive control.

3. Add 1 ml of the extract to the test tube, add 1 ml of the 10% blood solution, and incubate the mixture in a water bath at 56°C for 30 minutes. Then cool to room temperature. Centrifuge the mixture at 2500 rpm for 5 minutes, and measure the optical density of the supernatant at 560 nm using a spectrophotocolorimeter (the solvent itself is used as a control).

Results of Studied Plants:

Local Name	Scientific Name	Extract Source	Biomass Obtained (gr)	Extract Obtained (mg)	Anti-Inflammatory Activity (%)
Ordinary Reed	Typha orientalis	Leaf	5.15	50	87
Ordinary Pigweed	Chenopodium vulgaris	Leaf, Seed, Stem	13.8	320	80
Dandelion	Dandelion officinalis	Leaf, Seed, Flower	13.13	60	96
Peppermint	Mentha piperita	Flower	20.86	840	0
American Bugle	Lycopus virginicus	Leaf, Stem	20.87	840	7.03
Field Bindweed	Convolvulus L.	Leaf, Stem	5.3	250	86
Common Blackberry	Caesius L.	Leaf, Stem	5.09	370	86

Rosa L (common rosehip): The fruit contains up to 4-6%, sometimes up to 15% vitamin C, vitamins B2, P, E, and K, 12-27 mg% carotene, up to 29% organic acids (citric, malic, etc.), up to 18% sugars, up to 3.7% pectin, and up to 4.5% tannins. The seeds and other parts contain active compounds. The fruit is rich in multivitamins and is used as a natural food concentrate to treat vitamin deficiency diseases. High-vitamin varieties (Begger and Fedchenko rosehips) are used to treat and prevent vitamin deficiency diseases. The oil extracted from the seeds and the oily extract from the fruit pulp are used to treat burns, trophic ulcers, eczema, skin diseases, ultraviolet burns, and ulcerative colitis.

Sinapis arvensis (wild mustard): The seeds contain the glycoside sinigrin (up to 15% in powder form). Sinigrin is broken down into glucose, potassium bisulfate, and allyl isothiocyanate (mustard essential oil) by the enzyme myrosinase. Mustard essential oil can be obtained from fermented seeds through steam distillation. The seeds contain 1.17-2.89% essential oil, composed of 40% allyl mustard oil, 50% crotonyl mustard oil, and trace amounts of dimethyl sulfide, carbon disulfide, and other compounds. The seeds also contain 23-47% oil and up to 26% protein. Mustard preparations are used for inflammatory diseases, myositis, bronchitis, and rheumatic diseases.

Amygdalus communis L (common almond): Both types of almond seeds contain 20-60% oil. They contain the enzyme emulsin (β-glucosidase) and 3% of the cyanogenic glycoside amygdalin. The oil is used as a solvent for drugs. The

residue from the oil extraction is used to obtain bitter almond water. Sweet almond residue is used in cosmetics. Bitter almond seeds are toxic, and consuming 5-10 seeds can be fatal for children. Bitter almond water is prepared by hydrolyzing the residue in warm water for several hours. Sweet almond kernels contain oil (up to 40-60%), proteins (about 30%), mucilage, vitamins, pigments, carotenoids, lycopene, and traces of essential oil (0.5-0.8%), which gives them their distinctive almond scent. The oil contains oleic (80%) and linoleic (15%) acid glycerides. Sweet almond oil from dehulled seeds contains small amounts of linolenic and myristic acids, absent in oil from hulled seeds. Wild bitter almonds are toxic due to amygdalin, which releases hydrocyanic acid, benzaldehyde, and glucose upon hydrolysis. Whole bitter almonds are odorless but release a characteristic almond scent when sliced due to benzaldehyde.

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